Exhibit 160

1 IN THE UNITED STATES DISTRICT COURT
2 FOR THE EASTERN DISTRICT OF NORTH CAROLINA
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4 - - - - - - - - - - - - - - - - - x
5 IN RE: : Case No.:
6 CAMP LEJEUNE WATER LITIGATION : 7:23-CV-00897
7 This Document Relates To: :
8 ALL CASES : Pages 1-342
9 - - - - - - - - - - - - - - - - - x
10
Baltimore, MD
11
Wednesday, April 23, 2025
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13
Videotaped deposition of:
14
15 LUKASZ GONDEK, M.D., Ph.D.
16 called for oral examination by counsel for the
17 Defendant, pursuant to notice, before Sherry L.
18 Brooks, Certified LiveNote Reporter, of Golkow
19 Litigation Services, a Notary Public in and for the
20 State of Maryland, beginning at 10:04 a.m., when were
21 present on behalf of respective parties:
22

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P R O C E E D I N G S

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THE VIDEOGRAPHER: Here begins the video recorded deposition of Dr. Lukasz Gondek taken in the matter In Re: Camp Lejeune Water Litigation in the U.S. District Court for the Eastern District of North Carolina, Case No. 7:23-CV-00897.

Today's date is April 23rd, 2025. The time is 10:04 a.m. This deposition is being held at 36 South Charles Street, Baltimore, Maryland.

The court reporter is Sherry Brooks. The video camera operator is Kim Johnson. Both are on behalf of Golkow.

All attorneys present will appear on the written record. So if the court reporter will please swear in our witness, we can proceed.

* * * * *

WHEREUPON,

LUKASZ GONDEK, M.D., PH.D.,

called as a witness, and having been first duly sworn, was examined and testified as follows:

- - -

EXAMINATION

BY MR. TUBIN:

Q. Dr. Gondek, before we get started, I just have to make a record of something, so bear with me.

MR. TUBIN: So this is government Exhibit 1. This is the joint status report filed April 21st, 2025, copies available here.

(Exhibit Number 1 was marked for identification and was attached to the deposition.)

The top of page 11 was an issue that was addressed by the United States, DOJ's late supplementation of materials considered noting that on several occasions before depositions in phase 2 there have been supplemental materials disclosed several days or on the eve before deposition.

And I just want to mark as government Exhibit 2 an email I received yesterday at 1:59 p.m. from PLG (sic) attaching Dr. Gondek's supplemental materials considered list, which includes one recent study related to leukemia and benzene exposure, and the study itself was also attached.

(Exhibit Number 2 was marked for

identification and was attached to the deposition.)

MR. TELAN: I would only put on the record as to Exhibit 2 that that study that is referenced was, I think, first published for preprint on April 9th of 2025. And Dr. Gondek can talk to you about when he found it, but we just received it yesterday. So we produced it on the day that we received it.

BY MR. TUBIN:

Q. Okay. Dr. Gondek, how are you doing today, sir?

A. Very good. How are you?

Q. Good. Thank you. You understand you're under the same oath today as if you were testifying in court, correct?

A. Yes, I do.

Q. There's nothing preventing you from thinking clearly today, is there?

A. No.

Q. Nothing preventing you from testifying truthfully today?

A. No.

Q. I'm going to assume you understand my

question unless you tell me otherwise. Okay?

A. Okay.

Q. If you need a break, at any point, just let me know.

A. I will.

Q. For clarity on the record, I'm going to refer to trichloroethylene as TCE, as in Thomas, Charlie, echo, and I'll refer to tetrachlorethylene as PCE, as in Paul, Charlie, echo.

Did you bring any materials with you today?

A. Just my testimony, I think my fee schedule, invoices, and the list of my prior depositions.

Q. When you say your testimony, do you mean the report you submitted or --

A. The list of my prior --

Q. Oh, your prior testimony, correct?

A. -- prior testimony.

Q. Okay. To your knowledge, all of that material was already disclosed to the United States with your report?

A. That's my understanding, yes.
Q. There's nothing new in there that you considered for your opinions in your report?
A. No.
Q. Okay. Have you formed any new opinions since your expert report in this case?
A. No.
Q. So nothing new from that date until today?
A. No.
Q. Okay. Now, lymphoma and leukemia are different diseases, correct?
A. That is correct.
Q. They each have distinct subtypes, correct?
A. That is correct.
Q. If you were evaluating whether a chemical can cause lymphoma or leukemia, you would perform a separate analysis for each disease, correct?
A. Yes, with one comment, if I may.
Q. Please.
A. They both originate from the same tissue, which is bone marrow, so I would take that into consideration --

Q. Okay.
A. -- because it is biologically important.
Q. But you would not combine lymphomas and leukemias into one group for this analysis?
A. I would not.
Q. Okay. Combining them would make it difficult to tell which disease the chemical can or cannot cause, correct?
A. That is correct.
Q. Okay. Any other reasons you would not do this?
A. No.
Q. Okay. The statement TCE causes hematopoietic cancers is too broad to tell you whether TCE causes a certain type of NHL or a certain type of leukemia, correct?
MR. TELAN: Object to the form.
A. That says that it's associated with hematological malignancies, but there are various types of hematological malignancies.
BY MR. TUBIN:
Q. Right. So that statement alone, TCE can

cause hematopoietic cancers does not tell you if TCE causes acute myeloid leukemia or acute lymphocytic leukemia, correct?

A. Correct.

Q. It also doesn't tell you if it causes a particular subtype of NHL, correct?

A. That is correct.

Q. Now, when were you first asked to serve as an expert witness in this case?

A. I think it's been probably one or two years ago.

Q. Okay. Can you be more specific?

A. Specific? I think it was probably the beginning of 2023, which is two years ago.

Q. Okay. Who contacted you about serving as an expert witness in this case?

A. So I was contacted by Veramedica.

Q. And when were you actually retained in this case?

A. I think it was around the same time.

Q. Beginning 2023?

A. Beginning of 2023.

Q. Do you have a month?
A. I don't -- it was the beginning of the year. I don't quite remember. It might have been February.
Q. Okay. Is it reflected in any of the invoices or any documents?
A. It may be.
Q. Did you receive an assignment after you were retained?
A. I was asked to provide my opinion about benzene PCE, TCE, vinyl chlorides, and association with leukemias.
Q. All leukemias or any in specific?
A. Leukemias in general.
Q. Who gave you that assignment?
MR. TELAN: Object to the form.
A. Well, Pat Telan and the law firm asked me to provide my opinion about that.
BY MR. TUBIN:
Q. When you say "the law firm," was there anyone else other than Mr. Telan?
A. No, him and his law firm.

Q. Did you have any questions about the assignment?

MR. TELAN: Object to the form.

A. Not particularly. Obviously, I had questions about the case and what it implied but not to the merit of the assignment.

BY MR. TUBIN:

Q. So they asked you to give this opinion and you had no questions for them in return about the opinion?

MR. TELAN: Object to the form.

A. About my opinion or about the case in general?

BY MR. TUBIN:

Q. About what you were going to do.

A. So I had a question about the case.

Q. What was your question?

A. So I wanted to know more about the case. And I was familiar with the Camp Lejeune case, but I didn't know the specifics and I wanted to know -- like they wanted my expertise as a physician and concert (sic) biologist to provide my opinion on

these agents and incidents of leukemia.

Q. Did you modify the assignment that you were given in any way?

MR. TELAN: I'm going to object to the form and I'm going to instruct him not to answer because you're outside of the scope of Rule 26 now which doesn't allow for questions or conversations that occur between us prior to the finalization of his report.

MR. TUBIN: I'm not asking about any of the content of the conversations.

MR. TELAN: Well, you are going to the substance of it, so I'm going to instruct him not to answer. And if you want to take it up with the court, we can do that later.

BY MR. TUBIN:

Q. Are you following your attorney's instruction not to answer the question?

A. Yes.

Q. Did you ask your attorneys for any information to help with the assignment?

MR. TELAN: Same objection.

MR. TUBIN: I'm not asking for content, just whether he asked you for help with the assignment. There's no substance there. It's just whether he asked you for help or not.

MR. TELAN: I'll allow him to answer that.

MR. TUBIN: Thank you.

A. Can you specify exactly what you mean by help with the assignment?

BY MR. TUBIN:

Q. No, because I think I will be getting into substance at that point. I just want to know if there was some back-and-forth about the assignment with you and your attorneys?

A. It was back-and-forth about the case?

Q. About what you were assigned to do, if you had any comments, if you had any --

MR. TELAN: Object to form of the question.

You can answer that question.

A. I don't think I do have any comments.

BY MR. TUBIN:

Q. Okay. Was it your idea to opine on

leukemia generally instead of specific subtypes?

A. I was asked to do it on leukemia specifically because this is the -- well, my research is focused on mostly on myeloid leukemia.

So they wanted to make sure that my expertise aligned with my opinion.

Q. Do you know the subtypes of leukemia alleged in this case?

A. Yes.

Q. And what are they?

A. Well, they're acute leukemias and chronic leukemias.

(Simultaneously speaking addressed by Madam Reporter.)

BY MR. TUBIN:

Q. Acute myeloid leukemia, correct?

A. Acute myeloid leukemia is one of them. There is also acute lymphoblastic leukemia, ALL. So those are acute leukemias and there are myelodysplastic syndromes, which is a form of myeloid leukemias.

Q. Is that MDS?

A. It is MDS.

Q. Is it your understanding the plaintiff is alleging MDS in this case?

MR. TELAN: Object to the form.

A. I'm not entirely sure.

BY MR. TUBIN:

Q. You did not provide an opinion on causation related to MDS in your report, correct?

A. On myeloid malignancies. The distinction sometimes between MDS and acute leukemia is really semantics. It is -- as most MDS will eventually become leukemia.

Q. Right, but it's not leukemia when it's MDS, correct?

A. It is hematological malignancy, myeloid hematological malignancy. It's -- it's not acute leukemia in the understanding of acute myeloid leukemia, but it is a preleukemia state. Biologically and clinically it's considered a similar entity.

Q. Right. But if you diagnosed a patient with MDS, you wouldn't tell one of your colleagues

they have AML, correct?

A. Again, semantics. There's some high risk MDS that we treat as acute leukemia and they behave like acute leukemia, and most of the time the difference is what we call blast percentage.

So to give you an example, if someone has myeloid cancer with 19 percent blasts in a bone marrow, you call it MDS. If the patient has very similar disease with 21 percent of blasts in the bone marrow, you call it acute myeloid leukemia.

Q. Okay. So there is a distinction between the two that's more than just semantics, correct?

A. Well, if --

Q. Based on what percentage of blasts?

A. Like in my opinion difference between 19 percent and 21 is negligible, but we have to set the threshold. It was more important historically than in this right now.

Q. Okay. When diagnosing and treating patients, though, you pay close attention to the specific subtype of leukemia they have, correct?

A. That is correct.

Q. You treat them differently, correct?
A. That is correct.
Q. Your research is specific to leukemia subtypes, correct?
A. Not necessarily. Mostly it's myeloid malignancies which, again, is a spectrum of diseases. What's common about this disease is they arise from what we call myeloid cells in the bone marrow.
Q. But you do have papers that are specific to, let's say, APL or ALL, correct?
A. Correct.
Q. And I believe there's one paper about identifying distinct features of a subtype and improving how you can distinguish one subtype from another, correct?
A. That is correct.
Q. So there is a degree of attention -- special attention paid to the distinctions between subtypes, correct?
A. Yes.
Q. Okay. Veramedica -- what is Veramedica?
A. Well, it's the company that provides

administrative support for me. So they deal with arranging meetings and schedules with the lawyers, getting documents together, sharing documents. This is --

Q. Is it just for litigation?

A. It's just for litigation.

Q. And how did you become affiliated with Veramedica?

A. A friend of mine who did some work with them recommended me.

Q. Who is your friend?

A. It was Dr. Doug Smith.

Q. Martyn Smith?

A. Doug.

Q. Oh, Doug Smith.

A. Um-hum.

Q. And how long ago was that?

A. It must have been four years.

Q. So it -- to your knowledge, Veramedica is -- they profit off of expert witness services that they provide, correct?

MR. TELAN: Object to form.

A. They charge the fee for their administrative services.

BY MR. TUBIN:

Q. They charge that to you?

A. That's included in my fee, is their charge. I think we have a fee schedule available to you.

Q. Yeah. Do you know who owns Veramedica?

A. I don't know who the owner is.

Q. Who is the point of contact for you at Veramedica?

A. So two people. I usually talk to Kelley and Maddie.

Q. Do you have last names?

A. I don't remember.

Q. Are they attorneys?

A. No. They are assistants.

Q. Are there attorneys that work at Veramedica?

A. No.

Q. Do you have any financial investment or interest in Veramedica?

A. No.
Q. Does anyone you know?
A. No.
Q. And I think you answered this. But there are no lawyers who are a part of Veramedica?
MR. TELAN: Object to the form.
A. There are no lawyers that are employed by Veramedica, yes.
BY MR. TUBIN:
Q. Are there lawyers Veramedica works very closely with?
MR. TELAN: Object to form.
A. I don't know what "very closely" means. I think they do work with lawyers.
BY MR. TUBIN:
Q. So is the expert work they provide only plaintiffs work or have --
MR. TELAN: Object to the form.
A. I don't think they provide expert's work. There are administrative support for experts. I don't think they're specific to plaintiffs -- actually, I do know because they also work with

defense.

BY MR. TUBIN:

Q. If you had to quantify it percentagewise between plaintiff and defense, what would be --

MR. TELAN: Object to the form.

A. I don't know.

BY MR. TUBIN:

Q. How many cases have you testified on behalf of plaintiffs while with Veramedica?

A. Two.

Q. What about defense?

A. I did not testify. I provided reports.

Q. Is that the Johnson & Johnson case, talcum?

A. Yes.

Q. And what was your opinion there?

A. So they asked me about my opinion about biology of cancer in the context of exposure to different carcinogens.

Q. And what was the conclusion?

MR. TELAN: Object to the form.

A. So specifically they asked about

association between talcum exposure and mesothelioma, and my opinion was to provide a kind of biological understanding of carcinogenesis and what goes into carcinogenesis.

BY MR. TUBIN:

Q. Okay. So it was the mechanistic and biological function between asbestos and mesothelioma?

A. It was more between talcum and mesothelioma. I think the association and causation between asbestos and mesothelioma is very well-established. It was more about whether talcum itself was associated with mesothelioma.

Q. Did you find it was?

A. So based on the literature search that I did it was unclear whether the exposure to the talcum product was associated with mesothelioma.

Q. Was it unclear because of a dose issue or --

MR. TELAN: Object to form.

A. My understanding wasn't (sic) that it wasn't (sic) unclear because it's not clear whether

talcum was contaminated with the type of asbestos that causes mesothelioma.

BY MR. TUBIN:

Q. Okay. So it was more akin to the type of chemical substance, not how much or how little of it?

A. Correct.

Q. In 2023 you attended a meeting of experts in Washington, DC on Camp Lejeune, correct?

A. That's correct.

Q. And that was before you were retained in this case, correct?

A. I think it may have been after.

Q. Veramedica sent you to that meeting; is that correct?

MR. TELAN: Object to form.

A. They -- well, they didn't send me. They, I think, helped organize the meeting.

BY MR. TUBIN:

Q. So they organized this meeting with experts in Washington DC on Camp Lejeune in 2023?

MR. TELAN: Object to form.

A. I think -- I don't know who organized it.

I think they were probably helping with organizing the meeting.

MR. TUBIN: Okay. Exhibit No. 3.

(Exhibit Number 3 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recall being deposed in a Case Patricia Godfrey versus Union Pacific Railroad Company in July of 2024?

A. Yes.

Q. And you were under oath in that case, correct?

A. Yes.

Q. And you had counsel representing you there, correct?

A. Yes.

Q. I'm going to mark this as Exhibit 3. It's the transcript from that deposition. So if you turn to page 16, Dr. Gondek -- it's page 5 -- like the little small pages, page 16.

At the top line 7 to 9:

"ANSWER: So I worked with Veramedica,

which is the firm that brings expert (sic) together, so that was my involvement with Camp Lejeune."

So does that jog your memory at all as to whether Veramedica organized this meeting?

MR. TELAN: Object to form of the question.

A. I don't know who organized this, who was the organizer. I learned about this meeting from Veramedica.

BY MR. TUBIN:

Q. Right. So they told you about the meeting?

MR. TELAN: Object to form of the question.

A. Yes.

BY MR. TUBIN:

Q. Okay. And you went and attended this meeting, correct?

A. Correct.

Q. Do you recall what month this meeting was?

A. The end of March, beginning of April.

Q. 2023?

A. Yes.

Q. So when you said on page 16:

"ANSWER: So I work with Veramedica, which is the firm that brings expert (sic) together, so that was my involvement with Camp Lejeune" -- so, I mean, you said you believe you were retained in the Camp Lejeune litigation in February 2023?

A. I believe so. I don't remember exactly.

Q. But you don't mention here that you were --

MR. TELAN: Hold on. I don't know if he was finished, but it seems like you were talking over each other a little bit.

MR. TUBIN: My mistake.

MR. TELAN: Go ahead.

BY MR. TUBIN:

Q. From this testimony it seems that you're not mentioning that you were retained yet in the Camp Lejeune litigation?

MR. TELAN: Hold on one second. Object to form. It's improper. This wasn't asked in this deposition.

But you can answer the question.

A. I'm not sure if they asked me whether I was retained. I think they asked me about other cases I was involved with.

BY MR. TUBIN:

Q. Okay. So the meeting was -- in Washington, DC at Camp Lejeune was to brainstorm why there was an increased risk of certain cancers at Camp Lejeune, correct?

A. Correct.

MR. TELAN: Object to form.

BY MR. TUBIN:

Q. And what was the evidence that was discussed or that -- on there being an increased risk of cancer at Camp Lejeune?

MR. TELAN: Object to the form of the question.

A. There was general discussion about the experts on the contaminations, the level of contaminations, type of modeling used, ATSDR reports.

BY MR. TUBIN:

Q. Besides the ATSDR reports, were there any

other documents or materials discussed on Camp Lejeune?

MR. TELAN: Object to form.

A. I don't remember.

BY MR. TUBIN:

Q. What other experts attended this meeting?

MR. TELAN: Object to the form.

Could we go off the record for a second? I need to talk to counsel about this particular issue, if you don't mind, before you answer the question.

THE VIDEOGRAPHER: Off the record at 10:29.

(Discussion held off the record.)

THE VIDEOGRAPHER: Back on the record at 10:33.

MR. TUBIN: So there was a question pending. We thought maybe you had -- Counsel, you had a privilege objection or a concern you wanted to discuss. So the next time we'll just have the question answered and then take the break.

MR. TELAN: Understood.

MR. TUBIN: Madam Court Reporter, can you please read back the question that was asked before the break?

(Whereupon, Madam Reporter read back the question.)

A. I don't think I remember all the names. I didn't know them. I know there were physicians there, industrial hygienists, toxicologists, but I don't think I remember all the names of the people present there.

BY MR. TUBIN:

Q. Was Dr. Dean Felsher there?

A. I don't remember.

Q. Dr. Kathleen Gilbert?

A. I don't remember.

Q. Dr. Timothy Mallon?

A. I don't remember.

Q. That's fine. I'm just trying to jog your memory. Dr. Paul Rosenfeld?

A. I think I remember Dr. Rosenfeld, yes.

Q. What did you speak with Dr. Rosenfeld about at the meeting?

A. I didn't really talk to the experts. I was more listening to what they had to say.

Q. He was like a panelist?

A. Yes.

Q. And what did he have to say?

A. He was talking about modeling strategies for what are contaminations, not my area of expertise.

Q. Do you recall any details about these strategies?

A. Not really.

Q. You say "not really." Anything at all?

A. No. Like I said, it wasn't -- it's not my area of expertise, so I don't recall much from his presentation.

Q. After you met Dr. Paul Rosenfeld, he connected you to an attorney named Steve Monroe, correct?

A. That is correct.

Q. And then Steve Monroe retained you to opine that --

(Whereupon, Madam Reporter asked for

clarification from Counsel.)

BY MR. TUBIN:

Q. Steve Monroe then retained you to opine that benzene and diesel exhaust caused the plaintiff's cancer in Bonar versus Norfolk Southern Railway, correct?

MR. TELAN: Object to the form.

A. Yes. It was an opinion about benzene as a causative agent in mythologic cancers, yes.

BY MR. TUBIN:

Q. Multiple myeloma I believe it was?

A. Yes.

Q. So your testimony, as I understand this, is you were retained in this case in the beginning of 2023 in February?

A. Around that time, I believe.

Q. And then in March, towards the end of March, you attended this meeting with other experts who you don't remember in Washington, DC on Camp Lejeune?

A. That is correct.

Q. And do you know if any of those other

experts were retained in this litigation at that time?

MR. TELAN: Object to form.

A. I don't know.

BY MR. TUBIN:

Q. Did you discuss your retention with anyone at that meeting?

A. No.

Q. Did you talk to anyone there about Camp Lejeune?

A. Yes. That's what the meeting was about.

Q. Right. So who did you talk to about Camp Lejeune with?

A. Well, there was a panelist discussion. People shared their opinions or their expertise with other panelists. I didn't have a formal presentation at that meeting, so I was more a participant rather than a panelist.

Q. So I want to know what you said at that meeting.

MR. TELAN: Object to the form.

A. So I was talking about the mechanism of

carcinogenesis and how DNA mutations contribute to development of cancer.

BY MR. TUBIN:

Q. Did you talk about anything else?

A. No.

Q. Do you recall the -- any specific topics the panelists spoke on?

A. Not in addition to what I already mentioned.

Q. Okay. Were there attorneys at this meeting?

A. Yes.

Q. Okay. What attorneys were there?

A. So Mr. Telan was there. I think I recall Mr. Bell was there. I don't remember the other names.

Q. Okay. Had they asked you to go to this meeting?

A. Well, I don't know who invited me. It was -- like I said, I was contacted by Veramedica. I presume it was Mr. Telan.

Q. Who asked Veramedica to ask you to go?

A. I don't know.
MR. TELAN: Object to form.
BY MR. TUBIN:
Q. You didn't ask. You just got the invitation and went?
MR. TELAN: Object to the form.
A. Well, I don't know who asked them specifically. I knew Mr. Telan was involved, so I presume he asked them to invite me.
BY MR. TUBIN:
Q. Okay. Other than Mr. Telan and Mr. Bell, were there any other attorneys there?
A. There were. I don't remember their names, though.
Q. Any attorneys involved in this case?
MR. TELAN: Object to the form.
A. I'm not sure who is involved in this case from the list of attorneys.
BY MR. TUBIN:
Q. Do you know if those attorneys -- any of them represented plaintiffs?
A. I presume they represented the plaintiffs.

Q. What makes you presume that?
A. Well, I know Mr. Telan represents plaintiffs. So does Mr. Bell.
Q. And was the meeting -- the tone of the meeting on behalf of plaintiffs?
A. Yes.
MR. TELAN: Object to the form.
BY MR. TUBIN:
Q. Okay. So there was no discussion of defense strategies or anything at this meeting?
MR. TELAN: Object to the form.
A. I don't recall.
BY MR. TUBIN:
Q. Were there discussions of how to -- were there any -- strike that.
Were there any discussions about expert strategy in the Camp Lejeune litigation?
A. I don't remember.
Q. You performed a literature review in this case, correct?
A. Yes.
Q. That's the search that you described in

your report?

A. That is correct.

MR. TUBIN: Okay. We're going to talk about this, so we might as well mark this report here.

(Exhibit Number 4 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. So page 4 of your report -- let me back up a moment.

You recognize this document, correct?

A. Yes.

Q. That's your signature on the front page?

A. Yes.

Q. And what do you recognize it to be?

A. I'm sorry. Can you repeat the question?

Q. What do you recognize it as?

A. That's my expert report on general causation.

Q. Okay. Thank you. So the top of page 4 are the search terms you used in this case, correct?

A. That is correct.

Q. And these are all the search terms you used, correct?

A. That is correct.

Q. Did you perform any other searches?

A. I did look at other documents that are not part of the PubMed Medline database.

Q. You got them from Medline database?

A. No. I'm talking more about certain reports that are not part of the Medline database like ATSDR reports and the --

Q. How did you get those documents?

A. They were made available to me by -- or Veramedica.

Q. How did Veramedica get them?

A. I presume they got it from Mr. Telan or his law group.

Q. So how does this work? Veramedica just brings you documents about a case you're working on, and you don't ask any questions about where they come from?

MR. TELAN: Object to form. Argumentative.

A. I guess I know where they come from.
BY MR. TUBIN:
Q. Where do they come from?
MR. TELAN: Object to the form. Overbroad.
A. They probably come from the people I work with, which are attorneys.
BY MR. TUBIN:
Q. So what were those additional documents that came from the attorneys?
A. The ATSDR reports.
Q. Any others?
A. I don't recall any others.
Q. What about the Frank Bove mortality and incidence studies?
A. Well, they're published manuscripts available in PubMed.
Q. Right.
A. I found them through the PubMed search.
Q. Did you also get them from Veramedica?
A. No.
Q. So the only documents Veramedica gave you

1 were the ATSDR reports?
2 A. Correct.
3 Q. Did you get documents in any other way,
4 other than Veramedica or PubMed Medline?
5 A. No, Internet search and -- yeah.
6 Q. Okay. Did you ask Veramedica or
7 plaintiffs' counsel for any materials?
8 A. I don't recall asking for any materials.
9 Q. Do you have any reason to think you did?
10 A. No.
11 Q. Did you talk to any other experts in this
12 case?
13 A. Not -- not outside of the meeting in
14 Washington, DC, not since.
15 Q. And you can't remember any of those
16 experts' names, correct?
17 A. I don't recall their names.
18 Q. Okay. Except for Dr. Rosenfeld, correct?
19 A. Correct.
20 MR. TELAN: Object to the form.
21 BY MR. TUBIN:
22 Q. Did you ask to speak to anyone else, any

other experts?
A. No.
Q. When did you analyze this literature?
A. I think I performed a search on May 15th, 2024.
Q. And when did you do your analysis?
A. After May 15th, 2024.
Q. Do you know when after?
A. Well, it took several months.
Q. So did you start right away?
A. Probably.
Q. When did you complete your analysis of the literature?
MR. TELAN: Object to the form.
A. I presume it took several months, so it must have been September or October.
BY MR. TUBIN:
Q. So October at the latest?
MR. TELAN: Object to the form.
A. Probably.
BY MR. TUBIN:
Q. When did you first form the opinion that

benzene, TCE, and PCE together have an additive or synergistic effect?

MR. TELAN: Object to the form.

A. When did I form this opinion?

BY MR. TUBIN:

Q. Correct.

A. Well, I suggested it may have synergistic effect. I don't think I specifically stated that that's the case, but it was through my literature search and what I know about carcinogens and how they work in the initiation of the tumor.

Q. So you said you suggested this, correct?

A. For synergistic effect, perhaps there may be a synergistic effect.

Q. So in your report the opinion is that perhaps there may be or it's suggested that it could be synergistic effect?

MR. TELAN: Object to the form.

A. Correct.

BY MR. TUBIN:

Q. And when did you first communicate this opinion to plaintiffs' counsel?

MR. TELAN: Object to the form.
A. I think it was finalized in December of 2024, so it was around that time.
BY MR. TUBIN:
Q. So your report is dated December 9th, 2024, correct?
A. Correct.
Q. You didn't mention this opinion at any time before that?
MR. TELAN: Object to the form of the question.
A. Well, I provided my written opinion. I don't think I discussed the -- I discussed the subject of my opinion before.
BY MR. TUBIN:
Q. You did not, is your testimony, correct?
MR. TELAN: Object to the form.
A. That is correct. I provided my opinion in December of 2024.
BY MR. TUBIN:
Q. And before that you didn't have prior communications about it?

MR. TELAN: Object to the form. Again, I'm going to go off and just say that it's not allowed under Rule 26.

MR. TUBIN: Whether he communicated with you?

MR. TELAN: No, the subject.

MR. TUBIN: I didn't ask about subject.

MR. TELAN: You did.

MR. TUBIN: I asked whether he communicated with you --

MR. TELAN: About the subject.

MR. TUBIN: Nothing in the report.

MR. TELAN: Can you read back the question again?

(Whereupon, Madam Reporter read back the question.)

MR. TELAN: And "it" -- is there a question before?

MR. TUBIN: I can't ask him whether he discussed his opinion with you before December 9th?

MR. TELAN: The substance of the opinion.

MR. TUBIN: But I'm not asking about the

substance.

MR. TELAN: I think you did. I think it's back before that.

MR. TUBIN: I mean, I'm happy to -- I can rephrase it then. We'll do it that way.

MR. TELAN: Yeah.

THE WITNESS: Yes, please.

BY MR. TUBIN:

Q. Before December 9th when this report was served, did you discuss the report with your attorneys?

A. I -- we had communication between me and attorneys.

Q. How frequently?

A. Maybe once every two weeks.

Q. And did they ask you to make any changes to your report?

MR. TELAN: Object to the form of the question. That's outside the scope. So --

MR. TUBIN: Why is that outside the scope?

MR. TELAN: Rule 26 allows for you to find out what his opinions are but not about modifications

leading up to the final report.

MR. TUBIN: But I didn't ask him what the changes are, just if the changes were made.

MR. TELAN: Whether there are changes or not --

MR. TUBIN: That's in Rule 26.

MR. TELAN: I think it's implied.

MR. TUBIN: Are you instructing him not to answer?

MR. TELAN: No. I objected to the form. You asked for clarification. So that's why I'm explaining. I didn't instruct him not to answer.

MR. TUBIN: Okay. Understood.

BY MR. TUBIN:

Q. You can answer the question.

A. Can you repeat the question, please?

MR. TUBIN: Could you read back the question?

(Whereupon, Madam Reporter read back the question.)

A. Well, I provided this report on December 9, 2024. So after I provided the report there were

no changes to the report.

BY MR. TUBIN:

Q. I mean --

A. I didn't share the report with the attorney prior to.

Q. Right. You said you had spoken with them about the report maybe once every two weeks. During those conversations, was there ever an instruction to change something in your opinion?

MR. TELAN: Object to the form of the question.

A. I don't recall any.

BY MR. TUBIN:

Q. Now, you received millions of dollars in research funding from the Federal Government through NIH, correct?

A. That is correct.

Q. Does the source of funding for your research ever bias your research?

A. No.

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. Statistical significance gives you confidence that a result is not due to chance, correct?

MR. TELAN: Form objection.

A. Not necessarily. There is still a chance, but is it the chance that scientific community agrees to?

BY MR. TUBIN:

Q. But it gives you more confidence that it's not due to chance than if you didn't do significance testing, correct?

A. So let me make sure that I understand your question. So the significance that the scientific community agrees upon is on average -- well, it's a P-value of 0.05.

What that means that -- as a scientific community, we acknowledge and we accept the -- there's a 5 percent chance that the finding may be just by chance. So that's what "statistical significance" means.

Q. Okay. Now, you performed statistical significance testing in your published work, correct?

A. Yes.
Q. And why do you do that?
A. Because it's a well-established thing to do, to minimize or to reach the level that we all as a scientific community agreed upon.
Q. The level of confidence or the level of assurance --
A. The level of observation being just by chance.
(Simultaneously speaking addressed by Madam Reporter.)
BY MR. TUBIN:
Q. Are you familiar with a measurement called a "confidence interval ratio"?
A. I am familiar with the term "confidence interval."
Q. Yes.
A. Yes.
Q. But "confidence interval ratio"?
A. Not really with the ratio.
Q. When you say "not really," have you ever used this before?

A. I haven't used the confidence interval ratio before.

Q. Have you ever seen it used before?

A. I've heard about it. I haven't used it myself.

Q. Where did you hear about it?

A. In the scientific literature.

Q. What -- what studies?

A. I don't remember exactly which studies -- epidemiological studies because that's where we come up with confidence intervals.

Q. So you can't identify for me which studies you've seen this used in?

A. No.

Q. And you've not used confidence interval ratios in interpreting statistical results from a study, correct?

A. Not ratios, I used confidence interval and point estimates.

Q. Got you. Leukemia is a rare disease, correct?

A. Yes.

Q. When studying rare diseases it's necessary to perform statistical significance testing, correct?

A. I think it's true for all diseases, not only rare diseases.

Q. The 2014 Camp Lejeune Mortality Studies by Frank Bove studied leukemia, correct?

A. That's correct.

Q. They did not use statistical significance testing to interpret findings, correct?

A. They did use statistical tests. I don't think I understand your question.

Q. They did not use statistical significance testing to interpret findings, correct?

A. I still don't think I understand your question. Can you clarify what "statistical significance testing" means?

Q. I'll pull up the study and we'll do it that way.

(Exhibit Number 5 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recognize this document, Dr.

Gondek?

A. Yes.

Q. It's the 2014 "Evaluation of Mortality Among Marines and Navy Personnel Exposed to Contaminated Drinking Water at USMC Base Camp Lejeune: A Retrospective Cohort Study," correct?

A. Correct.

Q. Now, if you turn to page 6 of 14 --

A. Yes.

Q. And the top right part of the page, that first full sentence, can you read that?

A. The results and discussion?

Q. Above that.

A. "We did not use statistical significance testing to interpret findings."

Q. Correct. So would you agree this paper did not use statistical significance testing to interpret findings?

A. Yes, clearly if it says so.

Q. And leukemia is, again, one of the diseases that were looked at in this study, correct?

A. That is correct.

Q. And when studying rare diseases like leukemia it's necessary to perform statistical significance testing, correct?

MR. TELAN: Object to the form.

A. Well, I don't quite understand what statistical significance testing to interpret findings means. The reference is provided 24 to 28. I will have to look them up and know exactly what they mean by that.

BY MR. TUBIN:

Q. So when someone says "statistical significance," what does that mean to you?

A. Statistical significance, meaning that you run the statistical tests to find whether the results are significant or not.

Q. And what does that mean, "significant or not"?

A. So significant is the value that we all agree upon that meets the statistical significance criteria.

Q. So they didn't do that in this study?

MR. TELAN: Object to the form.

A. Well, they did statistical testing because you have provided hazard ratios, mortality ratios, confidence intervals, which means that statistical testing was done.

BY MR. TUBIN:

Q. Right. But not for significance, not for whether the null hypothesis was included in the confidence interval or not, correct?

MR. TELAN: Object to the form.

A. Well, that's the matter (sic) of the interpretation of the results.

BY MR. TUBIN:

Q. But Dr. Bove is specifically stating here, is he not: "We did not use statistical significance testing to interpret findings"?

A. So I'll have to review the references he provided after this statement to know exactly what that means.

Q. So when you were reviewing this paper for your report and you saw that, you didn't think to go look it up until now?

MR. TELAN: Object to the form.

A. I didn't look up all the references cited in the paper. I looked at the results of the statistical analysis they performed.

BY MR. TUBIN:

Q. Right. But you yourself do statistical significance testing in your own work, correct?

A. I don't do statistical significance testing because, like I said, I don't quite understand what it means in this context. I do statistical testing, which provides certain numbers and whether something is significant or not.

So in these terms -- that may be an epidemiological term for certain analysis. I have to make sure that I review the references to know exactly what that means.

Q. Okay. So it's possible that statistical significance testing here means something different than statistical significance testing in your published work?

MR. TELAN: Object to form.

A. I think we're confusing the terms. There is a term that's statistical significance --

BY MR. TUBIN:

Q. Right.

A. -- right? And what it means is that whether the results we have in front of us are statistically significant or not.

The statistical significance testing I have a feeling means something different. I cannot tell you on (sic) the top of my head what it means until I review the references that are provided.

Q. Okay. You want to take a look at the references real quick and tell me if any of them you're familiar with? I believe it's 24 to 28.

A. The epidemiologic papers are probably describing this matter that we're talking about. I don't recognize any of those. I will have to look them up, pull up the manuscript, and review them.

Q. Right. You said they're epidemiological papers, right?

A. Correct.

Q. Now, in epidemiology what does statistical significance testing mean? Can you answer that?

A. I'm not an epidemiologist. So I don't

want to give you the false answer.

Q. Okay. You have no -- I mean, you've interpreted epidemiology studies as part of your report, correct?

A. I interpret statistical significance. The statistical significance testing appears to be a different term than statistical significance.

Q. Right. My question is: You've reviewed and interpreted epidemiological studies for your report?

A. Correct.

Q. So when the term "statistical significance" is used in a epidemiology study, what does that mean for you?

A. Statistical significance, not statistical significance testing.

Q. Right.

A. It's two different things.

Q. Adding this word "testing," you're not sure what that is?

A. I'm not exactly sure in this context what that is.

Q. Okay.

A. But every time you -- I'm sorry -- every time you have numbers provided -- so if you look at Table 4 of the paper. When you have SMR and confidence interval it means that the testing was performed, right?

Whether it's statistically significant or not, then you can deduct from the numbers that are provided. So this is statistical significance.

Q. Are you aware anywhere in this paper whether they talk about these results being significant or not?

MR. TELAN: Object to the form.

A. I don't remember on (sic) the top of my head right now.

BY MR. TUBIN:

Q. In your papers you will highlight if a result is statistically significant, correct?

A. Correct.

Q. In the 2024 Camp Lejeune studies, do you recall if they used statistical significance testing to interpret findings there?

A. I don't recall statistical significance testing. I recall some study being statistically significant.

Q. Okay.

A. And can you also clarify which 2024 paper you're talking about?

Q. Let's look at the incidence study.

A. The most recent one?

Q. Yes.

A. Yes.

(Exhibit Number 6 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. If you go to page 4 -- it's dash 4 -- page 107008-4. And above Results, that first full paragraph, can you read the first sentence?

A. "Statistical significance testing was not used."

Q. Right. Now --

A. "Findings were interpreted based on the magnitude of the hazard ratio, its precision using the ratio of the upper and lower limits of the 95

1 percent confidence interval."
2 Q. So that's the confidence interval ratio
3 that we spoke about earlier, correct?
4 A. Um-hum -- yes.
5 Q. You don't use that in your research,
6 correct?
7 A. I look at the confidence interval not the
8 ratio.
9 Q. Right. But here they're talking about
10 using the ratio, correct?
11 A. Yes, they do.
12 Q. Okay. All right. Let's go to page 27 of
13 your report, Dr. Gondek.
14 A. Page 27 of my report?
15 Q. Yes, please.
16 A. Yes.
17 Q. So the last paragraph, second sentence
18 states: "Research specifically examining Camp
19 Lejeune residents has demonstrated a statistically
20 significant increase in the incidents of blood
21 cancers, including leukemia, among individuals
22 exposed to the contaminated water," correct?

A. Correct.
Q. You don't identify here the leukemia subtypes alleged in this case, correct?
A. Correct.
Q. The research being referred to are the 2014 and 2024 studies by Dr. Bove; is that correct?
MR. TELAN: Object to the form.
A. Most -- yes, I reviewed all of them. The statistical significance was observed in the most recent one looking at the incidence of cancer.
BY MR. TUBIN:
Q. I just want to confirm when this sentence says "research specifically examining Camp Lejeune residents," that research is the 2014 and 2024 studies, correct?
A. Yes.
Q. Okay. Now, the 2024 studies provided data on leukemia subtypes, correct?
A. Some of them, yes.
Q. From your statement on page 27, the one I just read, would leukemia include acute myeloid leukemia?

A. Yes.
Q. And it would also include acute lymphoblastic leukemia?
A. Yes.
Q. Did you intend leukemia here to refer to any other subtypes besides those two?
A. No. I focused on acute -- myeloid acute lymphoid leukemia.
Q. All right. So it's fair to say your opinions on leukemia in this report are limited to acute myeloid leukemia and acute lymphoblastic leukemia, correct?
MR. TELAN: Object to the form.
A. That is correct.
BY MR. TUBIN:
Q. Now, I want to take a look at the 2014 evaluation of mortality among Marines and Navy personnel. I believe you have that --
A. That's Exhibit 5?
Q. I think so.
A. Okay.
Q. Thank you. And I'm also going to have

your report in front of me here.

MR. TELAN: Marcus, what page did you say on Exhibit 5?

MR. TUBIN: So I haven't turned to a page yet on Exhibit 5. I'm on page 23 of his report.

MR. TELAN: Okay. Thank you.

BY MR. TUBIN:

Q. Let's give your counsel a second. So the second paragraph under subsection F. Camp Lejeune Studies, the one starting with, "The first study was a retrospective cohort mortality study," that's referring to the 2014 evaluation of mortality among Marines and Navy personnel, correct?

A. That is correct.

Q. Now, you list mortality results from that study that, I believe, came from Table 5; is that correct? It should be on page 8 of the study.

If you look at the hazard ratio for all cancers, it's the one represented here, and the same for kidney cancer.

So would you agree that this is a table that these results came from?

A. Yes.
Q. Okay. Now, none of these results you listed are statistically significant, correct?
MR. TELAN: Object to the form.
A. It depends how you define statistical significance.
BY MR. TUBIN:
Q. Okay. I can give you a definition. So -- that the confidence interval includes one.
A. That is accepted definition of statistical significance, yes.
Q. So none of these would be statistically significant based on that definition?
A. Based on the definition, yes, this is correct.
Q. Okay. Now, you report kidney cancer results in this section, correct?
A. Yes.
Q. You report liver cancer?
A. Yes.
Q. Esophageal cancer?
A. Yes.

Q. Cervical cancer?
A. Yes.
Q. But you do not list the results for leukemias, correct?
A. Correct.
Q. Okay. Why did you report those other cancers?
A. Why did I report those other cancers?
Q. Yes.
A. Well, I think they were part of the studies, probably that's why.
Q. But your report is on leukemia, not kidney cancer, correct?
MR. TELAN: Object to the form of the question.
A. So leukemia cases -- so specific leukemia cases were not studies. It was leukemias in general studies. That's what I said in my last sentence.
Q. Right. But wouldn't it be more relevant to at least put the result for leukemias instead of liver cancer?
MR. TELAN: Object to the form.

A. I don't know if it would be more relevant.
BY MR. TUBIN:
Q. Well, your report is on leukemia, correct?
A. On myeloid leukemias.
Q. Right. And aren't leukemias more relevant to that than liver cancer?
A. To my report, probably it is.
Q. Okay. Table 5 lists leukemias as 1.11 with a confidence interval of .75 to 1.62, correct?
A. Correct.
Q. And would you consider that statistically significant?
A. By confidence interval they don't meet statistical significance.
Q. Do they meet statistical significance in any other metric?
A. So when I look at the study -- there are two things with hazard ratio and confidence interval that we look at. One is point estimate, which is a hazard ratio.
Q. The 1.11?
A. The 1.11. The other one is confidence

intervals. The confidence intervals is simply an interpretation of the size of the cohort and number of cases.

So, in general, if you have very few cases the confidence intervals are usually very wide, even the point estimate may suggest that there is an association.

If there is a large number of cases usually the confidence intervals are very tight and they would meet what we normally call statistical significance.

So when I interpret the results in the rare diseases like leukemia, I don't only look at confidence intervals but I also look at point estimates because the fact that the point estimates are wide and, quote/unquote, statistically non-significant doesn't mean that the findings are not biologically significant.

Q. Okay. But I'm not talking about biological significance here. I'm not saying it's not important.

Have you ever in your research or your

publications identified a result as statistically significant when it included one in the confidence interval?

A. It doesn't meet the criteria for statistical significance, but the interpretation of the results may vary based on certain aspects of the study, most of the time number of cases that are included in the study.

Q. I understand. I just -- for me to have a clear record, you have never identified a result as statistically significant that included 1 in the confidence interval, correct?

A. In the sense of stricter (sic) meaning of statistical significance, no.

Q. Now, from this table you also don't list the results for hematopoietic cancers, correct?

A. Correct.

Q. And are all leukemias a form of hematopoietic cancers?

A. All leukemias are a form of hematopoietic cancers, yes.

Q. Okay. Let's turn to Table 7 of this same

study. It's on page 10. And let's look down at leukemia -- leukemias. For TCE there's no dose response reported there, correct?

A. What do you mean by "no dose response"? You're looking at Table 7?

Q. Table 7, subsection, I guess, C, leukemias, TCE at low exposure the risk is 2.0?

A. Yes.

Q. At medium exposure it goes down to 1.54?

A. Um-hum.

Q. And then high exposure it goes up to 1.81?

A. Yes.

Q. From low exposure at 2 to high exposure at 1.81 there's a decrease in risk, correct?

A. Yes. That's correct.

Q. And isn't that inconsistent with the dose response?

MR. TELAN: Object to the form.

A. Well, you just said the results are not statistically significant, so I'm not entirely sure what kind of conclusion you can draw from it.

BY MR. TUBIN:

Q. So if the results are not statistically significant you don't pay attention to the dose response?

A. I do.

Q. Okay. So how would you characterize this dose response?

A. So there's very little you can say with the number of cases that were studied. So if you just look at the numbers, yes, they don't increase. But it's hard to make the interpretation from dose response with such small numbers.

Q. Right. The numbers for leukemia here are the highest group out of kidney cancer, Hodgkin lymphoma, or ALS in this table, correct?

A. Um-hum.

Q. So they have the largest sample size here, correct?

A. Um-hum. The other reason why I'm saying that, there's unquestionable association between benzene and acute myeloid leukemia or leukemias. And you kind of see a similar trend suggesting that the reason why you see those results is just the low

number of cases rather than lack of dose response.

Q. Now, you believe the association between benzene and acute myeloid leukemia is unquestionable, correct?

A. Correct.

Q. And you also believe that it's a linear relationship, correct?

MR. TELAN: Object to the form.

A. Not necessarily. We can talk about biology and the dose response curve in benzene.

BY MR. TUBIN:

Q. Right. We will. I just -- for now --

A. You want to leave it for later?

Q. Yeah. For right now in these results the low exposure numbers for benzene are higher than the medium exposure risk and the high exposure risk, correct?

A. Um-hum.

Q. Is that something you would expect to see?

A. No.

Q. You would expect the high exposure to have the highest risk, correct?

A. Correct.
Q. Okay. Now -- and we have the same trend for total volatile organic compounds, correct?
A. Correct.
Q. And PCE was not included here, correct?
A. Correct.
Q. And that's one of the contaminants you've opined on, correct?
A. It was not included in assessment of the leukemia risk.
Q. In this table?
A. In this table. It was included in the assessment of other diseases.
Q. Correct. But for this table we only have TCE, benzene, and total volatile organic compounds?
A. Correct.
Q. PCE is in kidney cancer, correct?
A. Correct.
Q. And for ALS at the bottom?
A. Um-hum -- yes.
Q. Did you review any of the additional files noted at the end of this study?

A. No.

MR. TUBIN: I'm going to mark this as Exhibit 7.

(Exhibit Number 7 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. So, Dr. Gondek, have you seen this document before?

A. This is the supplemental information. I don't believe I reviewed that.

Q. So I want to represent to you this is the additional file to the 2014 Evaluation of Mortality Among Marines and Navy Personnel Exposed to Contaminated Drinking Water at USMC Base Camp Lejeune.

Now, if you look on page -- look on page 1, this is for total volatile organic compounds. And you see that 2.5 for low exposure, 1.33 risk for medium, and 2.33 for high, correct?

A. Correct.

Q. And that's also here on Table 7, correct?

A. Correct.

Q. Okay. Now, if you go to page 9 we have cumulative exposure results for PCE, the contaminant that was left out of Table 7, correct?

A. Correct.

Q. Okay. If you go down under underlying cause of death to leukemia, low exposure, 1.95 has a ratio, medium exposure drops to 1.42, high exposure drops further to 1.36.

This is the complete opposite of a dose response, correct?

A. Yes, but you also have to look at confidence intervals. They're getting wider just because the number of cases went from 16 to 11. So I don't think you can make any definitive statement about hazard ratio going down. That's the reason why confidence intervals are getting higher, just a few number of cases.

Q. So you're looking at how wide the confidence intervals were there, correct?

A. Yes. It reflects the variability of your results that can be anything from .66 to 307, for example, in medium exposure.

Q. Okay. Let's go to your report for a second on page 22. And the second to last paragraph that starts, "Finally an extended follow-up," if you look at the last sentence of this paragraph: "Leukemia mortality significantly increased during 1984 to 1989."

You give a risk ratio or standard mortality ratio of 6.06. Then your confidence interval is 1.24 to 17.53. That is far wider than any of the confidence intervals in this table we just looked at for PCE, correct?

A. Yes.

Q. And then it says: "And reached a peak during 1985 to 1990," and the confidence interval is 2 to 19?

A. Correct.

Q. So you're using these very wide confidence intervals in your report at times, correct?

A. Correct.

Q. I want to look at the 2014 civilian mortality now --

MR. TELAN: Have you already marked that?

MR. TUBIN: Not yet, I don't believe so.

(Exhibit Number 8 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Okay. And if you could have page 23 of your report open, too. So the paragraph -- third paragraph down stating, "In the second study elevated hazard ratios in the Camp Lejeune cohort" -- it's on page 22 of your report.

This is the 2014 civilian mortality study, correct?

A. Correct.

Q. Okay. And here you did list the mortality results for leukemias, correct?

A. Correct.

Q. They were 1.59 with the confidence interval of .66 to 3.84, correct?

A. Correct.

Q. And in your non-litigation work you would not call that statistically significant, correct?

A. Correct.

Q. Now, looking at Table 5, which is on the

next page --

A. Wait. I'm sorry. What --

Q. The same -- this is Exhibit 8. It's Table 5, page 9.

A. Okay.

Q. So this gives hazard ratios for tertiles of maximum cumulative exposure and coefficients for continuous cumulative exposure, correct?

A. Correct.

Q. Now, if we look at leukemias and then look down at benzene, neither the medium nor the high cumulative benzene exposures were close to statistical significance, correct?

A. Correct. You should also note that in benzene there's only one case.

Q. Um-hum, for medium exposure, and there's 8 in high exposure?

A. Correct.

Q. And for all the other contaminants, there's two in medium exposure.

A. Um-hum.

Q. Do you know why there's that discrepancy?

A. Between?

Q. Between the benzene numbers for medium exposure is 1, and the medium exposure number is 2 for all the other categories.

A. This -- you mean in hazard ratio?

Q. No, in the number of subjects.

A. I'm not exactly sure. Perhaps the 1 was not able to be assessed.

Q. All right. So there are only nine cases reported here for benzene and leukemia, correct?

A. Correct.

Q. Just less than all the others which had ten, correct?

A. Yes.

MR. TUBIN: All right. We can put that study away for now. I want to look at the 2024 mortality study.

MR. TELAN: When you get to a stopping point, we have been going about an hour and 15.

MR. TUBIN: We can stop now. Sure.

THE VIDEOGRAPHER: Off the record at 11:26.

(A break was taken.)

THE VIDEOGRAPHER: We are back on the record at 11:35.

MR. TUBIN: I'm going to mark as Exhibit 9 the 2024 Evaluation Mortality Among Marines, Navy Personnel, and Civilian Workers.

(Exhibit Number 9 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Dr. Gondek, do you have page 23 of your report still up?

A. Yes.

Q. So the paragraph that starts, "To address the short follow-up which was the major limitation of the previous studies, Bove and colleagues reported an updated retrospective cohort study" -- that's referring to this 2024 evaluation, right?

A. That's correct.

Q. Okay. Now, for this study you list results for kidney cancer, esophagus cancer, female breast cancer, Parkinson's disease, MDS, testicular cancer, cervical cancer, and ovarian cancer, correct?

A. Correct.
Q. And these results came from Table 4 on page 8, I believe, if you could confirm that.
MR. TELAN: You said page 8, Marc?
MR. TUBIN: Table 4 on page 8.
BY MR. TUBIN:
Q. Is that correct? That's where these results came from?
A. Yes.
Q. Okay. Now, you, again, do not mention the result for leukemias, correct?
A. Correct.
Q. And here we have acute leukemias studied and data for them, correct?
A. Correct.
Q. Now, the risk for Marines with leukemia of 1.1 comparing Camp Lejeune with Camp Pendleton is shown there in Table 4, correct?
A. Correct.
Q. This is not a result you would consider statistically significant, correct?
A. Correct.

Q. And, again, we have subtype data here, right, for leukemias?

A. We do.

Q. And you do not mention this subtype data in your report, correct?

A. Correct.

Q. For ALL it's .85 with a confidence interval of .47 to 1.53, correct?

A. Correct.

Q. Based on the hazard ratio that indicates 15 percent fewer deaths from ALL at Camp Lejeune than Camp Pendleton, correct?

A. Correct, which is like I mentioned, not statistically significant.

Q. For CLL it's .89, correct?

A. Correct.

Q. For AML it's 1.11 and not statistically significant, correct?

A. Correct.

Q. All right. Let's turn to Table 5 on page 10. This is comparison of Camp Lejeune and Camp Pendleton civilian workers underlying cause of death.

Now, the leukemias adjusted hazard ratio was 1 with the confidence interval of .61 to 1.64, correct?

A. Correct.

Q. Now, based on that hazard ratio it indicates no effect was observed, correct?

A. Correct.

Q. And you did not mention this result in your report, correct?

A. Correct.

Q. Again, we have subtype data here, correct --

A. Yes.

Q. -- including for acute myeloid leukemia? For CLL there was a 15 percent less risk at Camp Lejeune versus Camp Pendleton based on the hazard ratio, correct?

MR. TELAN: Object to the form.

A. Correct, just to clarify, CLL is part of the lymphoid malignancy.

BY MR. TUBIN:

Q. You're not opining on CLL on this report,

correct?

A. Correct.

Q. But your report -- you're opining on acute myeloid leukemia, and based on the hazard ratio here the Camp Lejeune residents had a 3 percent less chance of AML versus Camp Pendleton?

MR. TELAN: Object to form.

BY MR. TUBIN:

Q. Is that correct?

A. Yes, not statistically significant, but if you're interpreting the hazard ratios.

Q. And just to be clear, I said for residents. This is the civilian workers cohort. So just to be accurate on that.

Now, do you recall testifying that if a study fails to show a statistical association between benzene exposure and AML there must be some issue with the study?

MR. TELAN: Object to the form.

A. Well, if I testified issue, I mean, yes, the study should show significance between benzene exposure and AML because that's well-established.

BY MR. TUBIN:

Q. When you say "significance," do you mean a confidence interval that doesn't include the null?

MR. TELAN: Object to the form.

A. Not necessarily, because I think we discussed before the statistical significance and biological significance may be different, and it's all related to the number of cases.

So I think every epidemiological study should be interpreted in a context of what we know about biology, and sometimes point estimate even in a study that confidence interval include one may be informative in the right context.

BY MR. TUBIN:

Q. Understood. So looking at the result for AML here, this is a moderately wide confidence interval, correct?

A. Yes, I would say so.

Q. And the risk ratio is below 1. So would you consider this an indicator that there's something wrong with the study?

MR. TELAN: Object to the form.

A. Not necessarily. I think when you go to my report, page 23, the last sentence of the paragraph that we're talking about, "The major limitation of cancer mortality studies is that they may underestimate the risk for curable cancers such as leukemias."

BY MR. TUBIN:

Q. And what is that statement based off of?

A. Clinical and biological and epidemiological knowledge. So let me explain that to you.

Q. Before you do, maybe this can short-circuit things.

Do you have a particular reference or treatise that would represent that?

MR. TELAN: Let me interject because he's entitled to explain the answer to the question before you ask the next question. So I'd ask that he be allowed to explain.

MR. TUBIN: That's fair.

A. There's probably many references in the epidemiological study why that may be the case.

In general if you look at the diseases that are potentially curable, the incidence study is probably more informative than the cancer mortality study, and I can explain why.

So if you're dealing with a curable disease or a highly curable disease, what we care about in this case is the association between agent and diagnosis.

If you look at mortality and you say you cure 50 percent of the patients and they die of heart attack, in the mortality study you wouldn't see those as cases because they were cured. So automatically you underestimate the association for disease that may be cured.

What is the go-to and the gold standard and probably more relevant is the diagnosis with the disease. So whether the exposure to the agent (sic) is associated with the diagnosis of disease, whether you cure the disease or not, is you take this out of the equation.

So that's the main limitation of this study, is for -- cancer very likely leads to death,

let's say pancreatic cancer. That is incurable, 90 percent death rate due to pancreatic cancer. I think there is less of an issue.

The other one, the issue with mortality studies is what you have on death certificate, and we know the death certificate are not always reliable because people may die with the cancer of a heart attack.

BY MR. TUBIN:

Q. Is there any way to test this limitation or is this just you thinking through this rationally?

A. No. It's a very well-known phenomenon in epidemiology.

Q. Can it be tested to see if it's present or not?

A. If you don't have data -- unless you have data on both. So it can be tested.

If your study includes incidence data and mortality data, you can compare two of them.

Q. What if it just has mortality data?

A. You can't because you don't know what the true incidence is.

Q. In this study did they have mortality and incidence data or just mortality --

A. Just mortality.

Q. So there's no way to test whether this limitation was present here?

A. There is no way, but that's why there is a subsequent study looking at incidence, which is more informative than the study of looking at the death rate.

Q. How do you know that's the reason that was the subsequent study?

MR. TELAN: Object to form.

A. Well, I don't know if that was the reason, but as a cancer biologist and clinician that's what I would like to know.

BY MR. TUBIN:

Q. So you're speculating that that was the reason?

MR. TELAN: Object to the form.

A. I cannot speculate. You probably can ask Dr. Bove what was the reason, as he published the study. But if I have two studies, one is mortality,

one is incidence, I rely more on the incidence study than mortality.

BY MR. TUBIN:

Q. So you don't -- you can't say for certain the reason the incident study was done is because of its limitation?

A. No. That's one of the limitations.

Q. Now, you said there's probably many epidemiological references. Can you identify any for me that would support your explanation just now of this limitation?

A. Well, that's a well-known thing in common science, so I don't -- I cannot provide on (sic) the top of my head any epidemiological book will say that.

Q. So can you name an epidemiological book where it would say that?

A. No, I can't right now.

Q. Did you review the supplemental tables for the 2024 mortality study?

A. I did not review supplemental information.

Q. Were you aware that supplemental

information existed?

A. I probably was aware. I don't recall reviewing them.

Q. So you weren't curious?

MR. TELAN: Object to the form.

A. I don't -- I don't think it would provide significant information beyond what was available in the main manuscript.

BY MR. TUBIN:

Q. Why is that?

A. Because usually supplemental information are not necessary to make conclusion from the published manuscript. The published manuscript relies on supplemental information. So normally when we publish the paper the message should be very clear from the body of the paper.

Supplemental information is something extra that's not going to change the conclusion of the study and the main information.

Q. Okay. For the 2014 study where we looked at the additional table earlier today -- you recall that, right? --

A. Yes.
Q. -- PCE was not listed under the leukemia section, correct?
A. Um-hum.
Q. But it was in the supplemental table, correct?
A. Correct.
Q. So there can be information that you would miss if you didn't look at the supplemental tables, correct?
MR. TELAN: Object to the form.
A. It's not -- it would not -- the information if it's only in the supplemental, obviously, you're not going to see it when it's not in the main manuscript.
You're going to miss it if the conclusion changes based on reviewing additional information, and that's not the case in many manuscripts.
BY MR. TUBIN:
Q. But in that 2014 study there wasn't even a conclusion given for PCE in Table 7, correct?
A. Yeah, which means it wasn't significant.

That's why usually the studies -- if there is a significant difference, the study would emphasize that part.

Q. How do you know the study authors didn't think PCE was significant in that study?

MR. TELAN: Object to the form.

A. Why do I know --

BY MR. TUBIN:

Q. You said it's because it's not significant, that's why it's not there. How do you know that?

MR. TELAN: Object to the form.

A. Well, based on the statistical test that they run. I think we just discussed that reviewing the tables the studies -- the results were not statistically significant.

BY MR. TUBIN:

Q. But they were not statistically significant for the results in Table 7 either.

MR. TELAN: Is that a question?

MR. TUBIN: Yeah.

BY MR. TUBIN:

Q. Were the results statistically significant in Table 7 of the 2014 paper?

A. Which ones?

Q. Let's go back to it. The Marines 2014 mortality --

A. Exhibit 8?

Q. Yes. It should be Exhibit 8 -- no.

MR. TELAN: Exhibit 8?

MR. TUBIN: Yeah, page 10, Exhibit 8.

A. Table 7?

BY MR. TUBIN:

Q. Correct. So just -- I want to make sure I --

A. I want to make sure -- you said Exhibit 8?

MR. TELAN: You said Marine. Did you mean civilian?

MR. TUBIN: 2014 Evaluation of Mortality Among Marines and Navy Personnel --

MR. TELAN: 8 is civilians.

THE WITNESS: It's 9.

(Simultaneously speaking.)

MR. TELAN: So we're on 8?

MR. TUBIN: I'm on the 2014 Evaluation of Mortality Among Marines and Navy Personnel.

THE WITNESS: Okay. This one?

MR. TUBIN: That's 9.

THE WITNESS: Oh, you're on 8?

MR. TUBIN: No. No. No. I think that's -- that's 2024, though, right?

A. Yes.

BY MR. TUBIN:

Q. Okay. I'm talking about 2014, the one with the blue bar. That's civilians --

A. Mortality Study of Civilian Employees Exposed to Contaminated Drinking Water --

MR. TELAN: So that's 5.

MR. TUBIN: Yeah, 5.

A. Oh, yes. I got it.

MR. TELAN: So page 10, Number 5?

MR. TUBIN: Correct.

THE WITNESS: Table 7.

BY MR. TUBIN:

Q. I'm sorry?

A. Table 7?

Q. Correct. So just so I am clear because I don't want to be misunderstanding you, you said PCE was not listed here. And the reason you gave is because it was not statistically significant?

A. Well, I'm not sure what the reason of not listing it here was, but, yeah, it wasn't statistically significant.

Q. Okay. Well, for TCE none of those results are statistically significant, correct?

A. If you -- yes, that is technically correct.

Q. For benzene the medium exposure and the high exposure, neither of those are statistically significant, correct?

A. Correct.

Q. And for total volatile organic compounds, neither the medium -- the medium exposure is not statistically significant, correct?

A. Correct.

Q. Okay. I pray this will be less painful going back. Okay. I was talking about supplemental files to the 2024 mortality.

(Exhibit Number 10 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Okay. Dr. Gondek, table S2 gives standardized mortality ratios for Camp Lejeune and Camp Pendleton, correct?

A. Correct.

Q. This is a comparison to the general population, correct, SMR?

A. To the population at Camp Pendleton?

Q. Well, that's included in the third column --

A. Right.

Q. -- but the SMR in the first and second column is Camp Lejeune versus the general population, right?

A. Yes. That is correct, yes.

Q. So for leukemias the SMR is below 1, correct?

A. Correct.

Q. It's .92. That indicates 8 percent decrease in risk of leukemia at Camp Lejeune versus

the general population, correct?

MR. TELAN: Object to the form.

A. Correct.

BY MR. TUBIN:

Q. And that result was adjusted for age, race, and sex, correct?

A. Yes.

Q. I'll go to table S6 and it's on -- go to the second page of table S6 so we can look at leukemias. Now, this table compared risk by duration of exposure at Camp Lejeune versus Camp Pendleton, correct?

A. Correct.

Q. And for leukemias we had no dose response here, correct?

A. You mean there's no increase in incidence or hazard ratio if higher doses?

Q. Correct.

A. Yes.

Q. And, in fact, out of low duration, medium duration, and high duration, the lowest hazard ratio was for the high duration category, correct?

A. For which one?
Q. For leukemias.
A. Yes.
Q. For ALL there was also no increase in risk as dose increased, correct?
A. Correct.
Q. And, again, the lowest hazard ratio was for the highest duration, correct?
A. Correct.
Q. And acute myeloid leukemia also did not show an increase in risk between low duration to high duration, correct?
A. Correct.
Q. In fact, low duration for acute myeloid leukemia had the highest hazard ratio, correct?
A. Correct.
Q. Now, you didn't mention any of these results in your report, correct?
A. I did not mention these specific results. That is correct.
Q. Okay. So that major limitation you referred to, the cancer incidence study would not

have that limitation, correct?

A. That is correct.

Q. The major limitation of cancer mortality studies -- I should be specific -- would not have -- I'm referring to the sentence you pointed out, "The major limitation of cancer" --

A. Yes. I just compared the limitation of mortality compared to incidence.

Q. Right.

A. Yes.

Q. So this limitation is not something the cancer incidence study would have?

A. That is correct.

Q. You reviewed Dr. Frank Bove's deposition transcript, correct?

A. I did not.

Q. I believe it's in your materials considered list.

MR. TUBIN: Exhibit No. 11.

(Exhibit Number 11 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. So if you can turn to page 3 of Exhibit 11, can you confirm this is your materials considered list?

A. My materials considered list, right.

Q. Page 3, number 28 says: "Deposition of Frank Bove," correct?

A. That's correct.

Q. So do you recall reviewing that deposition?

A. I don't recall reviewing it. It was probably available to me, but I don't recall reviewing it.

Q. Does this mean, though, that you did, in fact, review it?

A. It means it was on my list of materials. I don't recall reviewing it. I may have looked at it, but I don't quite recall what was it all about.

Q. So there are materials that are listed on here that you don't look at?

MR. TELAN: Object to the form.

A. I do look at it. I just don't recall the deposition of Dr. Bove.

BY MR. TUBIN:

Q. Okay. But you're confident you reviewed it?

MR. TELAN: Object to the form.

A. I assume I did look at it.

BY MR. TUBIN:

Q. Because it's on this list, correct?

A. That is correct.

Q. All right.

MR. TUBIN: It should be Exhibit 12.

BY MR. TUBIN:

Q. Have you seen the preprint of the 2024 cancer incidence study?

A. Dr. Bove's study?

Q. Correct. It was Exhibit 7 to his deposition.

A. I don't recall whether it was a preprint or not.

MR. TUBIN: Can you mark this as 12?

(Exhibit Number 12 was marked for identification and was attached to the deposition.)

MR. TUBIN: It says 7 on it because it's

from that deposition, but this is 12 now.

MR. TELAN: Okay.

BY MR. TUBIN:

Q. Have you seen this document before, Dr. Gondek?

A. This particular form?

Q. This --

A. This preprint, no.

Q. Do you recall any of the questioning in Dr. Bove's deposition about this preprint?

A. No.

Q. All right. Let's turn to Table 6. It should be -- the page number on the bottom right is 60158.

And this table is titled "Duration Station at Camp Lejeune (Camp Pendleton as a reference): Marines/Navy Personnel Subgroup." It has a ratio and 95 percent confidence intervals, correct?

A. Yes.

Q. Now, if you turn to page 60160 we have results for leukemias, correct?

A. Correct.

Q. And here for leukemias as duration increased the risk ratio decreased, correct?

A. Correct.

Q. And we have subtype data for the specific leukemia --

A. Sorry. That's not totally true.

Q. Explain.

A. The one hazard ratio at low duration is 1.67. High duration is 1.68.

Q. Well, you're looking at acute myeloid leukemias?

A. That is correct.

Q. I'm just talking about leukemias right now.

A. Yes. That is correct.

Q. And as you probably surmise from that, we have subtype data here in this table. True?

A. Yes.

Q. Now, ALL risk decreased as exposure increased, correct?

A. Well, I wouldn't say it was consistent, but on average you can say that. So actually .75

medium duration and .96 in medium/high duration. But they were lower than low duration. I agree.

Q. Understood. And then from the medium/high duration to high duration it went down, correct?

A. Correct.

Q. And the high duration risk is 28 points lower than the low duration risk, correct?

MR. TELAN: Object to the form.

A. The hazard ratio difference is between 1.18 and .9, yes.

BY MR. TUBIN:

Q. Right. It went from 1.18 in the lowest duration down to .9 at the highest duration?

A. Correct.

Q. Okay. Now, for AML the hazard ratio didn't change really at all from low duration to high duration, correct?

MR. TELAN: Object to the form.

A. Well, if you want to look at it, it did go down in medium duration and medium/high duration. It went up again in high duration.

BY MR. TUBIN:

Q. My question was not well-phrased.
Low duration was 1.67 for the hazard ratio?
A. Um-hum.
Q. High duration was 1.68. So there's really no change between those two?
MR. TELAN: Object to the form.
A. Yeah. There's minimal change.
BY MR. TUBIN:
Q. .01, is that substantial to you in any way?
MR. TELAN: Object to the form.
A. Minimal.
BY MR. TUBIN:
Q. Let's look at Table 7. 60162 is the Bates. This is Duration Employed at Camp Lejeune, October 1972 to December 1975 (Camp Pendleton as reference) for Civilian Workers this time, correct?
A. Correct.
Q. All right. Go to the next page, 60163. For leukemias low duration, medium duration, and high duration all had decreased risk, correct, meaning the

1 risk ratio was below 1 for all?
2 A. Yeah. They increased, though, but they
3 are all 1. Yes, I agree.
4 Q. So even at the highest duration it didn't
5 exceed 1.0, correct?
6 A. That is correct.
7 Q. Which indicates that even the high
8 duration there's a decreased risk?
9 MR. TELAN: Object to the form.
10 A. Well, I wouldn't interpret the results in
11 this way, but they're not significant. So I'm not
12 sure if you can --
13 BY MR. TUBIN:
14 Q. I understand.
15 A. Just by looking at hazard ratio, that is
16 -- that's an interpretation of the results, yes.
17 Q. So the results being statistically
18 significant would be very important for you to be
19 able to interpret this, correct?
20 MR. TELAN: Object to the form.
21 A. No. I think the magnitude would be
22 important, and every time you review epidemiological

studies you have to review it in the context of many things including disease, biology, number of cases, and so on and so forth.

So looking at the isolated hazard ratio from that study has a very limited significance.

BY MR. TUBIN:

Q. The criticism you just had is that these are not statistically significant?

MR. TELAN: Object to the form.

A. I wouldn't say it's a major criticism. It is one of the -- they are not significant, but I wouldn't interpret the results as being at Camp Lejeune for a higher duration prevents leukemia from happening, if that's what you're implying.

BY MR. TUBIN:

Q. No. I'm not implying anything. I'm just asking if the risk result at high duration was below 1?

A. Yes, it is.

Q. And for AML there's minimal change in the risk ratio from medium duration to high duration, correct?

A. From medium to high, yes. You can call it minimum, but it's three times as high as low duration.

Q. Well, okay. Fair. The -- and, again, here these results are not statistically significant?

A. That is correct.

Q. Okay. All right. I want to go to Table 2, 60148.

MR. TELAN: Which table did you say?

MR. TUBIN: Table 2, 60148 is the base.

MR. TELAN: Thank you.

BY MR. TUBIN:

Q. So these are standardized incidence rates comparing Camp Lejeune and Camp Pendleton to the general population and then Camp Lejeune and Camp Pendleton, correct?

A. Yes.

Q. So for leukemias Camp Lejeune had 13 percent less incidence risk than the general population, correct?

A. Total leukemias?

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. Total leukemias, yes.

A. Can you repeat that, please?

Q. So the leukemia SIR is .87, correct?

A. Yes.

Q. So you say that's a 13 percent decrease in risk of leukemias versus the general population?

A. Yes, you can say that.

Q. Okay. And then down for ALL it's a 16 percent decrease in risk versus general population?

A. Yes.

Q. And for AML it's only a 5 percent increase, correct?

A. Correct.

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. And it's not statistically significant?

A. That is correct.

Q. All right.

A. And here -- I'm sorry. Oh, I thought we're going to compare it to Camp Pendleton. I guess we're not?

Q. No. I was just curious about the general population.

A. Okay.

(Whereupon, there was an interruption by the videographer.)

MR. TELAN: I apologize.

BY MR. TUBIN:

Q. Can we turn back to the 2024 cancer incidence study? Do you have it there, Dr. Gondek, not the preprint, the final 2024 incidence study?

A. That was exhibit --

Q. I don't recall off the top of my head.

A. I guess I have it somewhere. I don't think I -- oh, yes I do. It's Exhibit 6.

Q. Okay. Thank you. Real quickly, if you turn to Table 5 -- it's on page 10. So you see there are three exposure groups here: Low duration, medium duration, and high duration, correct?

A. Correct.

Q. And in the preprint we looked at the table for duration station at Camp Lejeune versus Hamilton Marines Navy personnel subgroup had low duration,

medium duration, medium/high, and high duration, correct?

A. Yes.

Q. So the final study omitted one category of exposure data, correct?

MR. TELAN: Object to the form.

A. Correct.

BY MR. TUBIN:

Q. So page 23 of your report lists several results from Table 3 of the 2024 cancer incidence study, correct?

A. Yes.

Q. And that's comparison of cancer outcomes at Camp Lejeune versus Camp Pendleton among the Marine and Navy personnel subgroup.

You do not list the results for leukemias, correct, just some subtypes?

A. That is correct.

Q. Okay. The leukemia results adjusted as a ratio was 1.07 and it was not statistically significant, correct?

MR. TELAN: Object to the form.

A. Yes.

BY MR. TUBIN:

Q. So looking at these four studies and these four different paragraphs on your report, page 23, when the hazard ratio for leukemias is 1.59, as it is in the second study, you identify it.

But when it was 1.1 in the 2024 mortality it's not identified on this page, or when it was 1.11 in the 2014 Marine mortality, that first paragraph, it's not identified, or when it's 1.07 in this 2024 cancer incidence study, correct?

MR. TELAN: Object to the form.

A. It's not listed as a result, that is correct.

BY MR. TUBIN:

Q. You only listed it as a result for that second study, correct?

A. I mentioned the results in the other two studies. I mentioned leukemia, but I didn't list specifically hazard ratios. That is correct.

BY MR. TUBIN:

Q. You also did not list the results for

acute lymphocytic leukemia from Table 3 from the cancer incidence study in your paragraph on this study here on page 23, correct?

A. That is correct.

Q. And that result was 0.94 with a confidence interval of .53 to 1.67, correct?

A. Correct.

Q. Flip to Table 4 just the civilian cohort results, you don't mention these results in your report, correct?

A. That is correct.

Q. And for leukemias the risk ratio was again below 1, correct?

A. Correct.

Q. It was 0.86. ALL had no adjusted hazard ratio, but the unadjusted hazard ratio was 0.27, correct?

A. I'm trying to find it.

Q. It's spelled out, so it's right above chronic lymphocytic leukemia.

A. Yes. I see that.

Q. And the result for AML --

A. Well, one case of ALL.
Q. Right. But the result for acute myeloid leukemia was not statistically significant, correct?
A. Correct.
Q. Let's turn to the next table, Table 5. For leukemias none of the results here are statistically significant, correct?
A. Yes.
Q. Just the broad category?
MR. TELAN: Object to the form.
A. Just the broad category if you look at hazard ratio of AML and low durations 1.36. But, yes, confidence interval include 1.
BY MR. TUBIN:
Q. And then it went down to 1.11 for medium duration, correct?
A. Um-hum, and up to 1.9, which was statistically significant for high duration.
Q. Correct.
A. Yes.
Q. For ALL the risk ratio never exceeded 1, correct?

A. That is correct.
Q. It was below 1 in all three duration categories, correct?
A. Correct.
Q. In fact, the risk decreased from .96 to .93 to .79, correct?
A. Numerically it did, yes.
Q. Well, Table 5's note, the footnote at the end -- it's on page 11 -- says that to qualify for the high duration category the Marine had to be present at Camp Lejeune for more than ten quarters, correct?
A. Yes.
Q. And that's 2.5 years, correct?
A. Correct.
Q. So for AML that 1.9 risk ratio that was statistically significant, that is -- only applies to someone that was there for 2 1/2 years or longer, correct?
A. That is correct.
Q. We're done for now with the Camp Lejeune studies.

A. Okay.

Q. Can you look at page 21 in your report, please --

MR. TELAN: 21, did you say?

MR. TUBIN: 21, yes.

BY MR. TUBIN:

Q. So the second to last paragraph above PCE and leukemia, the last sentence: "Given that almost all epidemiological studies looking at the association between TCE and leukemia are occupational studies, the Cohn New Jersey study stands as the most similar to the exposures found at Camp Lejeune," correct?

A. Yes.

Q. Now, Cohn had no data on long-term residents, correct?

(Whereupon, Madam Reporter asked for clarification from Counsel.)

BY MR. TUBIN:

Q. Cohn had no data on long-term residents, correct?

A. That must be correct. I don't remember

exactly the design.

Q. The 2024 studies of Camp Lejeune use duration of residents as a proxy for exposure, correct?

A. Correct.

Q. You would agree the type of study is critical in evaluating associations in epidemiological, correct?

MR. TELAN: Object to the form.

A. Can you repeat the question, please?

BY MR. TUBIN:

Q. The type of study is critical in evaluating associations, correct?

MR. TELAN: Same objection.

A. The type of study is critical? It is important. I wouldn't say it's critical, but it is important.

BY MR. TUBIN:

Q. If you could turn back to the Godfrey transcript and go to page 119 of the transcript.

So if you see on page 119, lines 18 to 21 -- and if you want to read the whole answer, you can,

1 but I'll just read it on the record.
2 "ANSWER: Suggesting that it's really the
3 type of the study and quality of the data and measure
4 of exposure that's critical in evaluating the
5 association. So that was another one."
6 Would you agree that the type of the study
7 is critical in evaluating association?
8 A. Yes.
9 MR. TELAN: I'll object to the form of the
10 question because it doesn't include the context.
11 BY MR. TUBIN:
12 Q. Okay. Cohn was an ecological study,
13 correct?
14 A. That is correct.
15 Q. The 2014 and 2024 mortality and incidence
16 studies were cohort studies, correct?
17 A. That is correct.
18 Q. In ecological studies information about
19 individuals is generally not gathered, correct?
20 A. That is correct.
21 Q. These studies may be useful for
22 identifying associations and areas for further

research, correct?

A. That is correct.

Q. But they rarely provide definitive causal answers, correct?

A. None of the epidemiological studies alone provides a definitive causative answer.

Q. If an epidemiological study lacks a precise estimation of exposure level, it's really difficult to draw a conclusion of association, correct?

MR. TELAN: Object to the form.

A. That is correct.

BY MR. TUBIN:

Q. The best approach to selecting literature is to pick studies that have really precise or relatively precise measures of exposure, correct?

A. That is correct.

Q. You agree that the quality of the data and measure of exposure are critical in evaluating an association in the study, correct?

MR. TELAN: Object to the form.

A. When available, that is correct.

BY MR. TUBIN:

Q. Now, ecological studies do not have exposure data, correct?

A. They have populational level data, correct.

Q. Cohn had no individual data on water consumption or inhalation or volatile organic compounds, correct?

A. That is correct.

Q. So if there was a confounding source of high VOCs being inhaled by the population, the study would not have picked up on that, correct?

MR. TELAN: Object to form.

A. That is correct, and it's probably true for all non-randomized clinical trials.

BY MR. TUBIN:

Q. You don't mention this limitation in your report, correct?

A. I did not mention that. I don't believe so, yes.

Q. Cohn concluded that his results only suggest a link between TCE, PCE, and leukemia/NHL,

correct?

A. Correct.

Q. But Cohn authors noted their conclusions are limited by potential misclassification of exposure, correct?

A. Yes.

Q. You do not mention the limitation in your report, correct?

A. Correct.

MR. TUBIN: Exhibit No. 13.

(Exhibit Number 13 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. If you could turn to the last page of this study, Doctor. So the study concludes with: "The results of this study, if corroborated by follow-up work, would support the policy of setting standards at levels that reflect protective public health assumptions," correct?

A. Yes.

Q. Was there any follow-up work done for this study?

MR. TELAN: Object to the form.
A. I don't believe so.
BY MR. TUBIN:
Q. You didn't -- you don't recall reviewing any work that was couched as a follow-up to this study?
A. I don't remember reviewing any work.
Q. Do you agree that with rare exceptions ecological studies are a weak strategy for inferring cause and effect relationships?
MR. TELAN: Object to the form.
A. Compared to what studies, in general?
BY MR. TUBIN:
Q. In general.
A. In general they have their limitations, yes.
Q. Would you agree they're a weak strategy for inferring cause and effect relationships?
MR. TELAN: Object to the form.
A. They have limitations.
BY MR. TUBIN:
Q. I understand. All studies have

limitations. But are ecological studies a weak strategy for inferring cause and effect relationships?

MR. TELAN: Object to the form.

A. Weaker -- I would agree that they're weaker in the study that provides individual level exposure data.

BY MR. TUBIN:

Q. Are they weaker than cohort studies?

MR. TELAN: Form objection.

A. In that regard, yes.

BY MR. TUBIN:

Q. Weaker than case control studies?

MR. TELAN: Form.

A. Yes, as long as the case control studies and cohort studies provide reliable individual level exposure data.

BY MR. TUBIN:

Q. Are ecological studies among the weakest studies for determining cause relationships?

A. No.

Q. What's weaker?

A. Case reports, case series, case reports, anecdotal evidence.

Q. Are those studies or more just individual reports on patients?

A. They're usual individual reports or case series, but they're definitely weaker than ecological studies.

Q. Do you agree that ecological studies are usually a method only for determining whether more rigorous studies are warranted?

MR. TELAN: Object to the form.

A. I don't think I would say that. Sometimes the more rigorous follow-up studies are simply impossible to do because the data don't exist, and we're limited to the data we have and it may be populational level exposure data.

BY MR. TUBIN:

Q. When did you first form the opinion that benzene can cause leukemia?

MR. TELAN: Object to the form of the question.

A. Well, that's been a well-known -- through

my medical training that's the one association that was very clear between benzene and myeloid malignancies and myeloid diseases that included acute myeloid leukemia.

BY MR. TUBIN:

Q. So in medical school?

MR. TELAN: Object to the form.

A. From my training as a medical student, internal medicine resident oncologist, and through my training in biology of cancer and acute myeloid leukemia.

BY MR. TUBIN:

Q. So more than ten years ago you formed this opinion?

A. I wouldn't say I formed this opinion. This data were available and taught in med school and oncology curriculum.

Q. Um-hum. So you would have agreed with that statement at least ten years ago?

A. Correct.

Q. Okay. And leukemia there would include acute myeloid leukemia and acute lymphocytic

leukemia?

A. It was very clear in myeloid diseases, so acute myeloid leukemia, MDS, and the other entity called aplastic anemia, which is not a cancer. It's bone marrow failure syndrome.

Q. Leukemias including AML and ALL are hematological malignancies, correct?

A. That is correct.

Q. Turn back to the Godfrey transcript, page 65. It's really the first -- the last line of 64.

A. Page 64?

Q. Um-hum. This is Exhibit 3, I believe. Can you read your answer that begins on line 25 of page 64 and ends on line 3 of page 65?

MR. TELAN: Can we get the -- can he get the opportunity to read the question as well?

MR. TUBIN: Sure.

MR. TELAN: Where are you at? You said page 65 --

MR. TUBIN: 64, 23 is where the question starts.

BY MR. TUBIN:

Q. Would you mind reading it out loud, Doctor?

A. So the question was -- maybe we can go with the question before that. Okay. The question was:

"QUESTION: Would you agree that's not enough for causality, or is it in your view?"

"ANSWER: Well, like I mentioned earlier, not all association is causal. In terms of benzene, there isn't enough evidence to suggest that benzene is a causal agent for hematological malignancies."

Q. And as you said, leukemia is including --

A. Well -- I'm sorry.

(Simultaneously speaking.)

MR. TELAN: Let him answer -- let him ask the question first.

A. I think there must be a typo or maybe I was -- misunderstood. I would never say that there is no -- enough evidence to suggest benzene being associated.

BY MR. TUBIN:

Q. You reviewed this transcript?

MR. TELAN: Hold on. You cut him off again. Let him answer the question.

A. It must have been either I didn't catch that, but based on the report that I provided and the rest of the transcript for Godfrey, if you can surmise that I would never say that there is not evidence to suggest that benzene is not associated with hematological malignancies.

That would contradict my entire report and testimony.

BY MR. TUBIN:

Q. If you could turn to page 142.

A. Yes.

Q. Who is Gogel in this case?

A. An attorney.

Q. He was the attorney representing you?

A. Yes.

Q. So on page 142:

"MR. GOGEL: Yeah. Dr. Gondek, sometimes the court reporter does a good job taking this down -- I'm not saying she didn't do a good job in this case -- and we don't have the witness read it over.

"In this case, I would prefer that you read it over, make any sort of corrections. We'll go over all of that. If you're okay with that, we'll read and sign."

"THE WITNESS: "Yes, I'm okay with that."

Did I read that correctly?

A. Yes, you did.

Q. Now, page 27 of your report at the top, the last sentence says: "For the comparison the level of benzene concentration in water at Camp Lejeune" --

A. I'm sorry. Where are we?

Q. Page 27.

A. And what part of page 27?

Q. At the top, the first, I guess -- the last sentence of that paragraph at the top.

MR. TELAN: The last sentence, first paragraph?

MR. TUBIN: Yeah. It's maybe a continuation from the prior page, the paragraphs -- I don't want to say complete paragraphs.

A. Yes, okay.

(Simultaneously speaking.)
BY MR. TUBIN:
Q. "For the comparison the level of benzene concentration in water at Camp Lejeune was around 10 ug/l (equals to .01 ppm)," correct?
A. Correct.
Q. Where did you get this number from?
A. I think I got it from the models of concentration of benzene TCE and PCE at Camp Lejeune.
Q. Which models?
A. I don't remember. I don't think I listed that.
Q. You didn't list the source of the contamination levels?
A. I think I forgot to list the source of the table.
Q. You said you think --
A. Well, I didn't list the source of the table, but I did review the model and the level of contamination at Camp Lejeune at different locations throughout the years. I just don't have that listed. It was my oversight.

Q. What materials is this modeling data listing that you reviewed, which documents?

A. I don't remember the title of the document right now, but I can definitely provide that.

Q. Do you have your reference list?

A. Um-hum.

Q. I don't want to belabor this, but if you can flip through it in a minute --

A. Sure. Let me check. I'm not entirely sure right now, but I can definitely double-check on that. I'm not sure if it's listed in materials considered.

Q. It seems it would be important to put in materials considered where you got that number, correct?

MR. TELAN: Object to the form. Argumentative.

A. I would agree.

BY MR. TUBIN:

Q. What literature shows that exposure to this level of benzene can cause leukemia in human beings?

A. We can pull up the -- so there were numerous reports suggesting that the levels went under one part per million was associated with -- or biologically significantly associated with DNA damage and the risk of leukemia.

Q. So I'm not -- DNA damage does not always lead to leukemia, correct?

A. That is correct.

Q. So I'm not talking about just damage to DNA. I'm talking about a study that shows .01 ppm is sufficient to cause leukemia in human beings?

A. So since I wrote my report that was kind of a notion based on looking at biological relevance and significance of low-level benzene exposure.

If you can pull up the manuscript that was just published on April 9 -- I don't know if you have access to that? --

Q. That's the one that was provided to us yesterday?

A. Yes -- we can review the data in this manuscript, or we can do it later, if you'd like.

Q. You wrote this report before you had the

manuscript?

A. Yes, and that was based on the data that I discuss in the paragraph -- page 10 starts with -- I think top of the page -- I think it starts on page 9, "In addition," and goes through the first, second, and third paragraph, suggesting that there's no such thing -- or there may be no such thing as a minimal threshold to harm in benzene based on the data and biological effect of benzene with less than 1 part per million.

So if you interpret -- back then when I interpreted the results, the DNA damage you mentioned correctly not always leads to leukemia but increases the risk of leukemia because leukemia is truly a genetic disease in terms of DNA damage that causes leukemia.

So surmising from the available data would suggest that levels way under 1 part per million may be biologically significant and increase the risk of leukemia.

Q. You say surmising from the data. Where is the study that shows .11 ppm is sufficient to cause

leukemia in human beings?

A. There's no study with this particular level. I was saying that those levels are biologically significant and cause DNA damage which may lead to leukemia.

Q. So cause DNA damage which may lead to leukemia?

A. Correct.

Q. DNA damage is not sufficient for leukemia?

MR. TELAN: Object to the form.

A. It is necessary.

BY MR. TUBIN:

Q. Right, but it's not sufficient?

A. That is true.

Q. Okay. So 1 ppm is 1,000 parts per billion, correct?

A. That's correct.

Q. So these studies --

MR. TELAN: P.

BY MR. TUBIN:

Q. 1 ppm is 1,000 parts per billion?

A. That is correct.

MR. TELAN: Oh, I'm sorry. I thought you said BPM.

MR. TUBIN: No, no, no -- maybe I did.

(Simultaneously speaking.)

BY MR. TUBIN:

Q. So these studies that are talking about concentration levels below 1 part per million (sic), we don't know how far below 1 part per million (sic) these effects are at?

A. We don't.

Q. So we can't say if it's at 10 parts per billion, 500 parts per billion, or even 900 parts per billion. There's no way to know that, correct?

MR. TELAN: Object to the form.

A. No, it is.

BY MR. TUBIN:

Q. Not based on this data, in your report?

A. Not based on this data, but based on what we know about benzene metabolism, how it's -- the range of the exposure matters in terms of how the benzene is metabolized. We can talk about it. It's supra linear exposure curve.

It would suggest that the levels under 1 part per million -- we don't know the minimum thresholds, but -- are biologically significant.

Q. Has there been a study that's shown a level of benzene exposure below 1 part per million (sic) causes leukemia in human beings?

A. Under 1 part per million, but like I said and you mentioned, there's no minimum level. It wasn't back in December of last year.

Q. So you're saying there is a study that shows below 1 part per million exposure to benzene can cause leukemia?

A. Right now, yes.

Q. What's that study?

A. The one that was just published recently, and I think you -- we added this to the documents.

MR. TUBIN: Exhibit No. 14.

(Exhibit Number 14 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. This is the study you're referring to?

A. Yes.

Q. So on that first page, second paragraph from the bottom, it states: "Please note that, during the production process, errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain," correct?

A. That is correct.

Q. This study looked at long-term exposure to low-level ambient benzene, toluene, ethylbenzene, and xylene, correct?

A. Correct.

Q. Do you know what they mean by "long-term exposure"?

A. Well, I would assume it's years of exposure. This study was a longitudinal cohort study that followed patients for decades. So we're talking about the years of exposure, and obviously it depends on the time of enrollment. I don't quite know. We have to look back at what the median follow-up was.

Q. So you're assuming decades of exposure?

MR. TELAN: Object to the form.

A. I'm assuming years, probably not decades

because it started in 2006. So years --

BY MR. TUBIN:

Q. So it would be like a minimal amount of years?

MR. TELAN: And, Marc -- I'm sorry -- but you continue to kind of talk over him a little bit, if you would let him just finish his answer.

MR. TUBIN: Understood.

A. One second. So if you go to Methods -- there's no pages unfortunately, but there's a Methods section, Study population.

So there were participants in the study that was enrolled between 2006 and 2010. So I think -- I presume that was less than 20 years' worth of data.

BY MR. TUBIN:

Q. So is it my understanding four years --

A. The study enrolled participants and observed these participants until now. The study is still ongoing.

Q. So what would be the lowest number of years that the study looked at for exposure to a

given participant?
A. That's hard to tell. I don't think we can tell what the minimum length of exposure would be here.
Q. Can you tell if it would be more than five years?
A. In some cases, probably a little bit more than five years.
Q. Would it ever be less than five years?
MR. TELAN: Object to the form.
A. It's possible that it would be less than five years.
BY MR. TUBIN:
Q. Possible it would be less than three years?
A. So -- perhaps.
Q. And what are you basing that off of?
A. Just the time of enrollment and the study follow-up, the fact that new cases are added to the study.
Q. Okay. Above Methods on that page, Methods Study population you pointed me to, Number 3 says:

"Individual exposure assessment is not available due to lack of monitoring data."

A. That's correct.

Q. That's a limitation on this study, correct?

A. Yes.

Q. There was no quantitative exposure data in this study, correct?

A. There was quantitative exposure data based on the regions, and I think they defined the regions as one-by-one kilometer because that was the resolution of the measurement.

Q. The exposure data was modeled, though, correct?

A. And verified by on-ground measurement of the levels, yes. So in the Methods -- they said when they -- in Europe, they measure the level of different toxins in different places.

So they verified their model based on actual measurements, and I think it was plus/minus 25 percent.

Q. And so they assigned annual average

exposure levels to each participant's residence, correct?

A. Correct.

Q. It wasn't measured data from each participant's residence?

A. That is correct.

Q. And if we look at -- again, because there's no page numbers on this, it's the page that has results at the bottom --

A. Yes.

Q. -- they use statistical significance testing here, correct, or --

A. Yes.

Q. -- they interpreted the results using statistical significance with a P-value of less than .05, correct?

A. That is correct.

Q. Now, on the next page on results, 67 percent of the subjects were overweight or obese, correct?

A. That is correct.

Q. Obesity is a risk factor for acute myeloid

leukemia, correct?

A. That is correct. It's not very -- yes.

Q. Compared to non-cases -- this is two sentences down -- incident cancer cases during follow-up tended to be older, overweight, or obese, correct?

A. That is correct.

Q. Now, on the Discussion page at the bottom -- it just says Discussion -- the first full paragraph, the double-spaced paragraph there, states that the associations between long-term benzene exposure and overall cancer was nonsignificant below a threshold of .18 parts per billion, correct?

A. Correct.

Q. All right. And then two pages later -- I don't have a way to mark it -- there's a footnote at the bottom, 40. It says -- do you see that? There's a footnote at the bottom, Number 40?

A. No.

Q. There's no other way for me to --

A. Can you tell me what the paragraph starts with?

Q. "Similarly, in the present study, we reported a significant" --

A. Yes, I see that.

Q. Okay. The last sentence on that page: "Future investigations based on individual exposure are warranted to confirm our findings."

A. Yes.

Q. And now two pages later it's a paragraph that states -- well, that begins, "The mechanisms underlying."

Do you see that?

A. Two pages later, you said?

Q. I believe it was two pages later, yeah.

A. "The mechanism," yes.

Q. "The mechanisms underlying the carcinogenicity of benzene, toluene, ethylbenzene, and xylene are largely unknown."

Do you agree with that?

MR. TELAN: Object to the form.

A. Well, I think while they are mentioning four of them at the same time, I think for some of them -- I don't know about toluene and other agents.

1 For benzene it is known.
2 BY MR. TUBIN:
3 Q. You say they're mentioning four of them at
4 the same time. Does that mean the study only looked
5 at these four together?
6 A. No, individually as well.
7 Q. So you don't interpret this to be
8 referring to them individually?
9 MR. TELAN: Object to the form.
10 A. I don't because the mechanism underlying
11 the carcinogenicity of benzene is very well-known.
12 BY MR. TUBIN:
13 Q. But there's no way to know from this
14 statement if they're referring to them individually
15 or as a group?
16 MR. TELAN: Object to the form.
17 A. I can't tell what they're referring to.
18 BY MR. TUBIN:
19 Q. If you go to the next page: "Moreover,
20 despite the adjustment of a series of confounders, we
21 could not rule out residual confounding by other
22 unmeasured factors that might affect the exposure and

cancer incidence."

MR. TELAN: I'm sorry, Marc. Where are you?

MR. TUBIN: The last page where the Conclusion is.

BY MR. TUBIN:

Q. I read that correctly, right, Dr. Gondek?

A. Yes.

Q. Then: "Finally, indoor emissions are an important source of BTEX. The lack of data on individual indoor exposure is a common limitation in environmental epidemiological research, and the results should be interpreted with caution," correct?

A. That is true.

Q. And at the end, the last sentence: "Our findings carry significant public health implications and highlight the need for further research to corroborate these associations and contribute to the evidence of carcinogenic potential BTEX," correct?

A. That is correct.

Q. So I'm going to ask the question again: What literature demonstrates a causal association

between benzene at .01 ppm and leukemia?

A. Well, can we go over the results of the strips?

Q. Sure.

A. So I have a couple comments, if I may. Because you brought up obesity and age, I just want to mention that the analysis that was done adjusted for all the known confounders, including obesity, smoking status, alcohol consumption, social levels, non-carcinogenic exposures, activity levels.

Everything you can think about that may confound the results were adjusted for in the multivaried analysis.

So that is true. Leukemia is associated with age and it's associated with obesity. This way we adjust for those confounders to see if the exposure to the toxin that you are studying is not confounded by these other things that are associated with the disease.

So if you look at I think it's Table 1, that's the first table after references.

Q. Okay.

A. I know it's a small print. So you can look at various cancers, including hematological malignancies, and we're focusing on benzene, obviously.

So if you look at leukemia -- or maybe we can start with lymphoma and hematopoietic tissues with a hazard ratio of 2.03 with confidence interval 1.91 to 2.17.

And just as an example on the comparison how I think about interpreting the data, you can kind of appreciate what happens when you study half a million individuals with 60,000 cancer cases.

The confidence intervals are super tight, which suggests that the variability of the data is very, very small. Unlike the studies that we discussed moments ago with the confidence intervals from .5 to 4.

So that tells you what the numbers mean in rare diseases like leukemia. You just need to have a high number of cases and population to have significant results and tight intervals.

So you can tell for leukemia non-Hodgkin's

lymphoma the results are highly significant with the hazard ratio of 2, above 2, and very tight confidence intervals.

Like you mentioned, this is an ecological study. You have to interpret the study in the complex of knowledge.

Q. This is an ecological study, you said?

A. It is because you assess the populational level of exposure, not individual level of exposure.

Q. Right.

A. The other thing that's also important -- you can look at the next figure, benzene, and you have the hazard ratio as a function of exposure level.

And exposure level is in parts per billion. So you can tell there is no increase in hazard ratio up until 0.18 part per billion, which, by the way, is 0.00018 part per million.

And then you can see it is almost logarithmic increasing hazard ratio with exposure over 1.8 part per billion. And the reason why, you don't see the same thing for other agents like

toluene or xylene, but it's very clear for benzene.

So these results from these studies would suggest that the levels above 1 point -- .18 part per billion increase the risk of cancer, including hematopoietic cancers.

Q. Can you say that -- the levels above what, 1. --

A. .18 part per billion.

Q. .18 part per billion?

A. Correct, which is the ambient level of exposure.

Q. Um-hum. And, again, there is no individual exposure data in this study, correct?

A. That is correct.

Q. And this is an ecological study, correct?

A. That is correct.

Q. So this is -- this is the sole study that you can point to that exposures of .01 ppm can cause leukemia --

MR. TELAN: Object to the form.

(Whereupon, Madam Reporter asked for clarification from Counsel.)

BY MR. TUBIN:

Q. This is the sole study that you can point to that exposures of .01 ppm benzene --

A. PPB.

Q. Well, I was looking at .01 ppm from your report --

A. Oh, I'm sorry. My report, yes.

Q. -- can cause leukemia in human beings?

A. With this particular level of .01 ppm, yes. There were studies looking at level under -- we discussed it -- under 1 part per million that were associated with increased risk, but we didn't know the minimum safe level.

This study gives us a little bit more granularity in terms of what this minimal level may be.

Q. When you say those other studies show increased risk, increased risk for DNA damage, though, correct, not --

A. As a biomarker, yes.

Q. But those studies were not showing leukemia cases?

A. That is correct.

Q. Okay. So this is the only study that you're pointing to as demonstrating .01 ppm can cause leukemia in human beings?

A. Correct. And if that's true now you can think about whether control cases are truly control cases because that's another issue with agents that may increase the risk of leukemia at such a low level that what you think are the controls that are non-exposed may, in fact, be people who are exposed to biologically significant levels.

MR. TELAN: Marc, just so you know, there's a time -- I'm not sure --

BY MR. TUBIN:

Q. Let me ask a similar question for ALL. What literature shows that exposure to benzene of .01 ppm can cause ALL in human beings?

A. Well, the same study that we just discussed and what we -- there is no epidemiological study that showed this level being associated with leukemia, which is understandable because most of the studies that were done decades ago didn't assess the

1 benzene level under 1 ppm.
2 There's very few studies that give us the
3 range that goes down to 1 ppm. But what we know
4 about, again, every time we -- we cannot analyze epi
5 studies. They're not in a vacuum -- epidemiological
6 studies in a vacuum.
7 We have to analyze the results in the
8 context of what we know about the disease, the
9 natural history of the disease, the mechanism by
10 which the disease occurs, and the mechanism by which
11 these toxins can cause leukemia.
12 So we did discuss this. The epi studies
13 alone are not sufficient to determine or disprove
14 causality. They may point to association, but what
15 we know right now in 2025 and what we know about
16 cancer is that they have to be interpreted in the
17 context of what we know the biology of the disease is
18 and the mechanism of action of the carcinogens.
19 Q. This Yu study had no data on leukemia
20 subtypes, correct?
21 A. They didn't include specific subtypes of
22 leukemia. They distinguish between lymphoid and

1 myeloid like non-Hodgkin's lymphoma and multiple
2 myeloma and leukemia.
3 (Whereupon, Madam Reporter asked for
4 clarification from the witness.)
5 A. They did not have any information of
6 subtypes of leukemia. They distinguished between
7 leukemia and lymphoid diseases which is non-Hodgkin's
8 lymphoma and myeloma.
9 But what was important in the study is
10 that it wasn't limited to leukemia. It was cancer
11 overall that was significantly increased with
12 exposure to an ambient-level of benzene.
13 MR. TUBIN: Take a break.
14 MR. TELAN: Sure.
15 THE VIDEOGRAPHER: Off the record at 1:05.
16 (Whereupon, at 1:05 p.m., a
17 luncheon recess was taken.)
18 - - -
19 A F T E R N O O N S E S S I O N
20 (1:49 p.m.)
21 Whereupon,
22 LUKASZ GONDEK, M.D., PH.D.

was called for continued examination, and having been previously duly sworn was examined and testified further as follows:

THE VIDEOGRAPHER: Okay. We're back on the record at 1:49.

EXAMINATION BY COUNSEL FOR DEFENDANTS CONTINUED

BY MR. TUBIN:

Q. Dr. Gondek, when did you first form the opinion that TCE can cause leukemia?

MR. TELAN: Object to the form.

A. After reviewing the literature. So that was probably sometime in the second half of 2024.

BY MR. TUBIN:

Q. And that includes AML and ALL?

A. Yes.

Q. And when did you communicate this opinion to plaintiffs?

MR. TELAN: Object to the form.

A. I think in the written form it was on December 9. And, obviously, we had communication before.

BY MR. TUBIN:

Q. And when did you first form the opinion that PCE can cause leukemia?

MR. TELAN: Same objection.

A. The same time.

BY MR. TUBIN:

Q. So you had never previously examined whether TCE can cause leukemia?

A. Before I got involved in this case?

Q. Um-hum.

A. That is correct, no.

Q. And the same for PCE?

A. That is correct.

MR. TUBIN: Exhibit No. 15.

(Exhibit Number 15 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Dr. Gondek, do you recognize Exhibit 15?

A. Yes.

Q. And what is Exhibit 15?

A. That's my expert report on benzene in association with acute myeloid leukemia.

Q. And that's your signature on page 20, correct?
A. That is correct.
Q. Turn to page 8 to 9 -- turn to page 8. It's a table that goes from 8 to 9.
A. Yes.
Q. And this table is titled Accepted risk factors for Acute Myeloid Leukemia, correct?
A. That is correct.
Q. And this report is dated February 12th, 2024, correct?
A. That is correct.
Q. Now, in the table you identify carcinogenic agents with sufficient evidence in humans, correct?
A. That is correct.
Q. And you list benzene here, correct?
A. Yes.
Q. But you do not list TCE, correct?
A. I did not.
Q. You do not list PCE, correct?
A. Correct.

Q. You do not list vinyl chloride, correct?
A. That is correct.
Q. The next column is for agents with only limited evidence of carcinogenicity in humans, correct?
A. In acute myeloid leukemia?
Q. Yes.
A. Yes, that is correct.
Q. And, again, neither TCE, PCE, nor vinyl chloride are listed, correct?
A. That is correct.
Q. And you did not provide a table similar to this in your report for this case, correct?
A. That is correct.
Q. And, again, this was an acute myeloid leukemia case; is that correct?
A. That is correct.
Q. And acute myeloid leukemia is one of the leukemia subtypes you're opining on in this case, correct?
A. That is correct.
Q. Page 27 of your report --

A. I don't have -- on my current report?
Q. Yeah, on your report in this case -- sorry -- yes. The last sentence of the second paragraph lists maximum levels of TCE and PCE at Camp Lejeune as 1,400 parts per billion and 39 parts per billion respectively, correct?
A. That is correct.
Q. What literature shows that 1,400 parts per billion of TCE exposure can cause leukemia in human beings?
A. So there was one study that we'll probably discuss from New Jersey that had levels of TCE and PCE assessed in the study, and the levels were -- the levels were, as I recall, comparable to these levels.
Q. That's the Cohn study; is that correct?
A. That's correct.
Q. And that's an ecological study, correct?
A. That is correct.
Q. And there was no individual exposure data in that study, correct?
A. That is correct.
Q. And there was no accounting for the

confounding factor of volatile organic compounds, correct?

A. As we discussed before, yes.

Q. What literature shows that 39 parts per billion of PCE exposure can cause leukemia in human beings?

A. The similar answer to the previous one.

Q. Again, the New Jersey Cohn study?

A. Yes.

Q. Any others?

A. I'm not aware of any others.

Q. Are there any others beside the Cohn for the PCE?

A. I'm not aware of any.

Q. Would you agree that the etiological window for benzene-induced AML is ten to 15 years?

MR. TELAN: Object to the form.

A. Not necessarily, no.

BY MR. TUBIN:

Q. In your report you cite to the Linet 2019 paper; is that correct?

A. That is correct.

MR. TUBIN: Exhibit No. 16.

(Exhibit Number 16 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. And do you recognize this as that study?

A. Yes.

Q. And do you believe this is a reliable authority?

MR. TELAN: Object to the form.

A. Are you referring to the authors or the study?

BY MR. TUBIN:

Q. The study itself.

MR. TELAN: Same objection.

A. I don't think I would call it authority. I think it's a reliable study.

BY MR. TUBIN:

Q. Okay. What's the difference between a reliable authority and a reliable study when you say that? How do you draw the distinction?

MR. TELAN: Object to the form.

A. I think it depends on the type of the

study, quality of the study, quality of assessment, granularity of the data that went into determining the assessment and outcome.

So there's a lot of components that goes into an evaluation of the study and whether the results you would consider reliable or not.

BY MR. TUBIN:

Q. But this study was sufficiently reliable for you to rely on it in your report in this case?

A. To include it in my report, yes. There are studies that are more or less reliable when designed. Sometimes there's studies that -- well, the -- there's only one study available and we have to review the study whether it's ideal or not ideal.

Q. Okay.

A. But if we have multiple studies on a subject, obviously, you would put more weight on the study that's well-designed with the granular level of information.

Q. So looking in the abstract section in this paper under results -- it's kind of the middle of the paragraph -- the study says: "There was little

evidence of exposure response after at least ten years regardless of age at first exposure," correct?

A. Yes.

Q. Now, does that indicate a ten-year etiological window?

MR. TELAN: Object to the form.

A. Not necessarily. And I can tell you what I based my opinion on in terms of latency. I think that's what you're referring to?

BY MR. TUBIN:

Q. I'm referring to time since last exposure to diagnosis.

A. Yeah, based on this study? Or in general?

Q. Just -- whether that -- that sentence, this study, you know, listing this in their results supports a ten-year etiological window.

MR. TELAN: Object to the form.

A. In this particular study, yes.

BY MR. TUBIN:

Q. All right. I'm going to give you another study.

(Exhibit Number 17 was marked for

identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recognize this study?

A. Yes.

Q. You cite this study in your report, correct?

A. That is correct.

Q. Would you call this a reliable study?

A. Yes, I would call it a reliable study.

Q. Okay. Let's turn to page 85 and the middle paragraph, first sentence states: "We showed that the risk of leukemia is associated with exposure within 15 years of diagnosis. The association with exposure prior to this period is weak," correct?

A. Correct.

Q. Now, it's your opinion that there's no safe level of exposure to benzene, correct?

A. Well, I think we have more information right now that I had when I submitted my opinion based on the paper that we recently discussed. So if I had to say right now I would say it's .18 part per billion.

Q. Just so I'm clear, so your opinion has changed based on the Yu paper from there being no safe level of exposure to benzene to .18 part per billion being a safe level of benzene?

MR. TELAN: Object to the form.

A. I think we have to be reasonable, right. So I'm not claiming that exposure to one molecule of benzene causes cancer. I'm just saying that there is no known safe level of benzene exposure or at least we didn't know what the minimum safe level was. I think now we have a better idea of what it is.

BY MR. TUBIN:

Q. Based on that Yu study?

A. Based on that paper, yes.

Q. Is it your opinion that there's no safe level of exposure to TCE?

MR. TELAN: Object to the form.

A. I don't think we have data to say what the safe level of exposure to TCE is, and the same applies to PCE.

BY MR. TUBIN:

Q. Okay. And is it your opinion that any

exposure to any level of benzene is significant and increases the risk of leukemia?

A. No. So I think we live in an industrial society. So we are all exposed to certain levels of toxins. My opinion is that the above background level of exposure to benzene or TCE and PCE increases the risk of leukemia.

Q. How much benzene above background is needed to increase the risk?

MR. TELAN: Object to the form of the question.

A. Well, it is a linear relationship. So if they increase the levels of benzene, the risk increases. Based on the study that we just discussed has a ratio of 2 for every quartile of exposure above 0.18 part per billion for benzene.

(Whereupon, Madam Reporter asked for clarification from the witness.)

BY MR. TUBIN:

Q. Did I hear you say it's a linear relationship?

A. It is positive association.

Q. So just so I understand, does that mean --

MR. TELAN: I don't know if he was finished.

MR. TUBIN: Okay. Sorry.

MR. TELAN: Were you finished?

A. It is a positive association. Whether it's purely linear, I'm not entirely sure that's the case.

We did mention about benzene metabolism and how the response curve is steep as the below level and then flattens out at the higher level. So it is linear in some range of exposure, but it may not be the same slope or linear at different range of exposures.

BY MR. TUBIN:

Q. What I'm trying to get at is how far above background exposure do we need to get before you can say that's enough to cause leukemia?

MR. TELAN: Object to the form of the question.

A. I don't think we can precisely estimate how far above background. If it's -- if the exposure

is linear in a certain range, every level above the background increases the risk proportionally.

BY MR. TUBIN:

Q. So any amount above background increases risk?

A. If the background is associated with leukemia -- and we have a reason to believe it is -- the level above background will be associated with a higher risk of leukemia.

Q. Any level above that background?

A. Any level above background. The magnitude of risk will be different, obviously, but it will be higher than background.

Q. You cannot identify the minimum length of benzene exposure required to cause leukemia, correct?

MR. TELAN: Object to the form.

A. I don't think it's just purely length. I think it's a little bit more complicated than that. It's probably cumulative exposure.

Sometimes the lower level of exposure for a longer period of time may be more significant than the higher level of exposure for a shorter period of

time even though the cumulative exposure may be the same.

BY MR. TUBIN:

Q. Well, would you say one year of occupational benzene exposure has a very low possibility to cause cancer?

MR. TELAN: Object to the form.

A. Depending on the level of exposure, but I wouldn't say it has a very low risk. I think it's impossible to tell unless you put context to it on the level, the time of exposure, the route of exposure. Was it inhaled? Was it ingested? Was it dermal? I think I can answer this question based on that.

BY MR. TUBIN:

Q. All those considerations you just mentioned, did you address those in your report in this case?

A. No, I did not specifically.

Q. Can you on Exhibit 3, the Godfrey transcript, go to page 80?

MR. TELAN: Give me a second, Marcus,

because it's buried under here.

MR. TUBIN: Okay.

MR. TELAN: You did say page 80, correct?

MR. TUBIN: Yes, of the transcript pages.

A. Yes.

BY MR. TUBIN:

Q. So -- and you can feel free to look at page 79 preceding it. There is no dose or exposure level given on page 79 or page 80; is that correct?

A. That is correct.

Q. And you're asked -- look at page 79 on line 21:

"QUESTION: Okay. If he had a two-year history with the railroad reporting similar exposures, would that be enough for you to attribute at least in part his multiple myeloma to his railroad career and exposures he may have had therein?"

Your answer:

"ANSWER: So it is a very difficult question to answer, but I would say yes, obviously to a lesser degree than 30 years. But, in my opinion, that would also be a significant exposure."

"QUESTION: Move two years to one year, reduce it to one year. Same question."

And your response:

"ANSWER: I would say -- so let me put it this way: The risk decreases significantly as you know with the length of exposure, but what is the minimum length of exposure biologically that's consequential, we don't know. It is in (sic) the way kind of lottery in terms of cancer."

Further down on page 80:

"ANSWER: Is it possible that within the one year or six months or five days the benzene causes defect in the gene that will be responsible for someone's malignancy? Yes, it is possible. It's very low possibility, but it's possible."

And then further down -- well, let's stop there. Now, would you agree one year of occupational benzene exposure has a very low possibility to cause cancer?

MR. TELAN: Object to the form of the question. Mischaracterization.

A. Like I said, it depends on the level of

exposure. The one year meaning one year is .1 part per billion and one year at 20 part per million. I think it's -- again, it's very difficult to answer.

BY MR. TUBIN:

Q. Were you given the level in this line of questioning I just read from?

MR. TELAN: Object to the form.

A. I was not.

BY MR. TUBIN:

Q. Would you agree the risk of cancer decreases significantly for those with shorter exposures?

MR. TELAN: Object to the form of the question.

A. Yes, I would agree with that.

BY MR. TUBIN:

Q. Age is the most significant risk factor for most cancers, correct?

MR. TELAN: Object to the form.

A. For most cancers, that is correct.

BY MR. TUBIN:

Q. And similarly for occupational exposure,

you typically see the diagnosis at the end of a career, correct?

MR. TELAN: Object to the form.

A. I'm not sure. I don't think I can answer this question. I just don't know.

BY MR. TUBIN:

Q. All right. Well, let's turn to page 66 of that transcript. So if you look at lines -- you can read on page 66, lines 17 to 24, and let me know if you agree that occupational exposure --

A. That starts --

MR. TELAN: Let him finish the question and then you can answer it.

BY MR. TUBIN:

Q. Let's do it this way. Go ahead and read on page 66, 17 to 24.

A. The one that starts with, "I'm sorry"?

Q. Right.

A. "QUESTION: I'm sorry. I didn't mean to interrupt you. When you talk about occupational exposures to a given substance, which is generally a lifetime, or not a lifetime, a career or lengthy

period of time of exposure, that also would be one of the reasons you would see it later in life, right, because it requires a certain dose?"

Q. And then your response, can you please read that?

A. "ANSWER: Yes. They increase the number of these DNA errors."

Do you want me to read further?

Q. You can read to line 3 of page 67.

A. Right.

"ANSWER: But it can take time for them to accumulate to the level that it causes malignancy."

Q. Now, everyone is exposed to benzene at some level on a daily basis, correct?

MR. TELAN: Object to the form.

A. At some point in their life, I would assume so.

BY MR. TUBIN:

Q. Well, I'm asking about on a daily basis, not at some point.

MR. TELAN: Object to the form.

A. I don't know if that's true.

MR. TUBIN: Are we on 18?
MADAM REPORTER: Yes.
(Exhibit Number 18 was marked for identification and was attached to the deposition.)
BY MR. TUBIN:
Q. Can you turn to page 47, please?
A. I'm on it.
Q. Oh, you opened right to it?
A. Yes.
Q. Okay. So page 47, line 15 to 18 reads:
"QUESTION: Would you agree with me that virtually everyone is exposed to benzene at some level on a daily basis?"
And your answer is yes, correct?
A. Yes.
Q. Okay.
A. Can I clarify that?
Q. Sure.
A. So, in general, that's true on a daily basis. I think while if you live in the mountains on a high altitude in the hot for a day are you exposed to benzene on this day? I would say, well, probably

not.

But, in general, that's true because we're all exposed to benzene. Living in the city and an industrial society probably every day. But is everyone every single day exposed to a certain level of benzene, I don't know. Most of us are.

Q. So it varies by location, correct?

A. The level varies by location definitely.

Q. And, therefore, the amount that someone is exposed to varies by where they are?

A. That is correct.

Q. Someone in Camp Lejeune, North Carolina will be exposed to a different background level than someone in Los Angeles, California, correct?

A. That is correct.

Q. It may also vary by time of year, right?

A. Correct.

Q. It can vary by local circumstances, correct?

A. Yes.

Q. And it's your opinion that each and every benzene exposure above background significantly

1 increases the risk of malignancy, correct?
2 MR. TELAN: Object to the form.
3 A. My opinion is that above background level
4 of exposure increases the risk of hematological
5 malignancy, yes.
6 BY MR. TUBIN:
7 Q. Significantly increases it?
8 A. It depends on the above background level
9 of exposure. If you're talking about one molecule
10 above the level of exposure, probably not. If you're
11 talking about at ten times, five times, four times
12 decrease, yes. So I think we have to be reasonable
13 in terms of any level above background.
14 Q. Can you take a look at Exhibit 3 and go to
15 page 102?
16 A. Yes.
17 Q. Line 19 to 21 reads:
18 "ANSWER: Yes, it's helpful in a way that
19 if it was above background exposure, that's (sic)
20 significantly contributed to his malignancy."
21 MR. TELAN: I'm sorry, Marcus. Where are
22 you --

MR. TUBIN: Page 102, lines 19 to 21.
BY MR. TUBIN:
Q. Did I read that correctly, Dr. Gondek?
A. Yes.
Q. Now, do you have a similar conclusion for TCE and PCE?
MR. TELAN: Object to the form.
A. That the above background level of exposure increases the risk of cancer?
BY MR. TUBIN:
Q. Um-hum.
A. Yes.
Q. What is that based on?
A. Biological evidence, biology of -- hematological malignancy and what we know about mechanism of action of these agents and how they impact DNA.
Q. Those mechanistic studies and that biological evidence is all about how TCE or PCE damage DNA, correct?
A. It's about the metabolism, lipophilicity, meaning -- predeliction for lipids or fat tissue,

enzymes involving metabolism, toxic metabolites.

Q. So those studies, that literature, are there leukemia diagnoses or just DNA damage in those materials you're referring to?

A. DNA damage.

Q. So there's another step to get to cancer, correct?

A. Yes, but it's a necessary step in carcinogenesis.

Q. Right. So what are the studies or data that show any amounts above background for TCE or PCE increase the risk of leukemia?

A. Epidemiological studies.

Q. Which studies?

A. You're talking about epidemiological studies, not biological?

Q. Correct, not a biological study. One that shows there's damage to DNA and then that led to leukemia, not just there's damage to DNA.

MR. TELAN: Object to the form.

A. What are the studies to show that damage to DNA may lead to leukemia later?

BY MR. TUBIN:

Q. For TCE and PCE in any amount.

(Whereupon, Madam Reporter asked for clarification from Counsel.)

BY MR. TUBIN:

Q. So what are the epidemiological studies that show any TCE or PCE exposure above background increases the risk of leukemia?

A. There is (sic) no studies that look exactly above background level. There are studies looking at elevated levels of TCE and PCE at certain thresholds.

Q. What levels did they look at?

A. That was addressing the Cohn study. We can review that.

Q. Any other studies besides the Cohn study?

A. Hematological malignancies, I'm not aware.

Q. Would you agree there's no way to quantify how much toxic exposure increases cancer risk above background?

MR. TELAN: Object to the form.

A. Not for all agents. For some, yes.

BY MR. TUBIN:

Q. So I used the words "toxic exposure." And you're saying it's true for some toxic exposures but not all?

A. Toxic exposures, you mean exposure to toxins?

Q. Correct.

A. I don't think we have data on every single toxin and dose and response in humans. The data don't exist. At some point we have to extrapolate based on what's available to us and what kind of data are available to us.

Q. So you're saying there is a way to quantify how much a toxic exposure increases cancer risk above background?

A. Yes.

Q. If you look at Exhibit 3 again, Godfrey, on page 49 --

A. Yes. I'm on it.

Q. Okay. Now, if you could read lines 2 to 12, please.

A. "QUESTION: Okay. Increased risk,

significant contributor. Is there any way to quantify the increased risk?"

"ANSWER: Everything above the background risk that's seen in people who are not exposed to toxins."

"QUESTION: All right. I understand that, but are we talking about he's at twice the risk of the unexposed or the normally exposed, the background exposed, or is he at 20 times? Is there any way to quantify that?"

"ANSWER: No."

Q. So -- but now you're saying it is possible to quantify that?

MR. TELAN: I'll object as improper impeachment.

A. If we have data, yes. So is it possible to quantify it? It is possible. Do we have data to do it? No, we don't.

BY MR. TUBIN:

Q. Okay. You cannot tell whether a toxic exposure played a greater or lesser role in causing cancer than any other particular risk factor,

correct?

MR. TELAN: Object to the form.

A. I don't think we can precisely say that.

BY MR. TUBIN:

Q. Medical science doesn't allow to us do that, correct?

MR. TELAN: Object to the form.

A. Yeah, I don't think we have enough evidence or data to exactly assess the impact of each individual exposure to cancer risk in -- again, it's generalization, but on average that's true.

BY MR. TUBIN:

Q. But not -- I don't mean we can't tell a precise amount. You can't tell whether they play a greater role or a lesser role, correct?

MR. TELAN: I'll object to the form as overbroad.

A. We could address that looking at well-controlled studies. Let me give you an example. If you can -- if you have a study that you can control for all confounders, then you can -- you can definitely say what the impact would be.

And the one way to do it, which unfortunately we can't do, is a randomized control trial. And this is, obviously, unethical to do, and we're not going to get the answer to that.

But this you would precisely measure the effect of toxin to someone's risk of cancer if you can do it in a prospective fashion and control for all the possible confounders in the study.

BY MR. TUBIN:

Q. Well, you're saying it's possible to do?

A. Well, it's not going to be done because it's unethical.

Q. If you can turn to page 50 of the Godfrey transcript?

A. Page 50?

Q. Yes. And if you could read -- I'll read from line 13:

"QUESTION: Okay. Are you able to quantify the role of his race, his weight, and his gender from a causality standpoint?"

"ANSWER: Precisely?"

"QUESTION: Yes (sic)."

"ANSWER: No."

"QUESTION: They all played a role potentially?"

"ANSWER: Yes."

"QUESTION: Do they all play the same role as his exposure to benzene and diesel exhaust? Do they play a greater role, a lesser role, or do you have any such opinion?"

Your answer:

"ANSWER: I don't have such opinion. It would be impossible to determine."

"QUESTION: Medical science doesn't allow us that, right?"

"ANSWER: That is correct."

Did I read that accurately?

A. Yes, but I think you asked a different question.

Q. What's the distinction?

A. You asked the question if it would be possible to do, and the answer is, yes, it would be possible to do in randomized control trials.

Is it going to happen? No, it's never

1 going to happen. So can we quantify it based on the
2 data we have? No, we can't.
3 Q. Right. But the first question I asked
4 was: You cannot tell whether toxic exposure played a
5 greater or lesser role in causing cancer than any
6 other risk factor?
7 MR. TELAN: I'll object to the form of the
8 question. That wasn't a question. He was just
9 telling you what his last question was. So that
10 wasn't a question.
11 BY MR. TUBIN:
12 Q. Do you recall that as my last question?
13 A. Yes.
14 Q. Okay. Now, page 10 of your report, the
15 first complete paragraph, the third sentence:
16 "Several studies demonstrate an increased risk of AML
17 at near-ambient benzene concentrations."
18 And the first paper you cite is Rushton
19 2014, correct?
20 A. Correct.
21 MR. TUBIN: Exhibit No. 19.
22 (Exhibit Number 19 was marked for

identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recognize this as the Rushton study?

A. Yes.

Q. Would you consider this a reliable study?

A. Yes.

Q. So the conclusion on the first page, what does that first sentence state, if you could read it, please?

A. "Overall, this study does not persuasively demonstrate a risk between benzene and AML. A previously reported strong relationship between myelodysplastic syndrome (potentially previously reported as AML) at our study's low benzene levels suggests that MDS may be the more relevant health risk for lower exposure."

Q. Now, turn to page 784. Under Discussion, can you read those first two sentences?

A. The first sentences?

Q. First two sentences under discussion.

A. Okay. "Overall, this study did not show a

strong or consistent relationship between benzene exposure and AML or CLL. The study has high quality, precise benzene exposure estimates for individual participants, tailored to their job role (sic), site, and era and high quality diagnoses."

Q. Do you have any reason to disagree with those statements?

A. No.

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. We'll look at page 4 of your report -- your report in this case, not the --

A. Um-hum.

Q. So your search terms up at the top include AML, ALL, but not CLL, correct?

A. That is correct.

Q. You're not offering an opinion on CLL in this case, correct?

A. That is correct.

Q. Okay. Between AML and ALL -- well, strike that. I'm sorry.

The only chemical you searched for with

AML is benzene, correct?

A. I think so. Yes, I would say so.

Q. You did not search acute myeloid leukemia and TCE, correct?

A. I didn't list it here. That is correct.

Q. Well, I believe earlier you testified this contains all of your search terms; is that correct?

A. Yes.

Q. Okay. You did not search for acute myeloid leukemia and PCE, correct?

A. So let me clarify that. The reason why I included cancer in tetrachloroethylene and trichloroethylene is because there were very few hits in my search for acute myeloid leukemia and its agents.

So I may have done that at some point, and I think I decided to extend it to something just above AML just because I didn't get any hits in the database.

Q. So the results didn't come back with as many as you wanted?

A. Maybe not.

Q. So you broadened the search, correct?
A. Correct.
Q. Now, the only chemical you searched for with ALL is benzene, correct?
A. That is correct.
Q. You didn't search for ALL and TCE, correct?
A. Like I said, I may have. But it may have been very few study. I extended my search to beyond ALL and AML.
Q. I understand you're saying you may have. But earlier you testified under oath that this contained all of your search terms, correct?
A. It includes all of the search terms, yes.
Q. So you're now saying that you left out search terms that didn't contain enough results?
MR. TELAN: Object to the form.
A. I did not. So if your search term is very precise, extremely precise in terms of diagnosis and you don't get any hits, obviously, you extend your search term to include what you're looking for plus potentially all other malignancies.

I did it just to make sure I'm not missing any association or -- or studies that may have included hematological malignancies, but they were not in the keywords.

BY MR. TUBIN:

Q. So there were searches that you performed that you did not list here?

A. I -- perhaps -- I assume it's just because I don't remember, but every time I included something more broad than AML and ALL, that was probably the reason why, because there was either zero results or one result that was not relevant to our discussion.

Q. Would you agree that a study that groups leukemia subtypes together under the umbrella of leukemias can mask differences in risk or causation?

MR. TELAN: Object to the form.

A. To some extent, I do believe that, yes.

BY MR. TUBIN:

Q. On page 4, the second paragraph down under methodology, when you say: "I followed the weight of the evidence approach to investigating and analyzing the data," what are the steps you took in that

approach?

A. Well, I look at the type of studies. So if you use these broad search terms there's a lot of things that you're going to see. And some you can see case reports, you can see review manuscript, you can see case series, you can see opinion pieces, you can see editorials.

They're not considered helpful in making my opinion because they're not epidemiological studies. So that was Number 1 criterion. There's just a lot of hits that you just cannot use. Some, for whatever reason, discuss a very different topic but they came up as a search. So that was the first exclusion.

So I focused on the studies that were helpful, which was the epidemiological studies, ecological studies, clinical studies, meta-analysis, and so on.

Q. So the first step is to find studies or -- and filter by the type of study?

A. Correct.

Q. What's the next step?

A. So the next step you look at the type of studies, which whether it's the epidemiological studies, ecological studies, clinical observation, and so on.

Then you look at the population, whether they assessed the outcomes that you wanted to study; whether they assessed the exposure that you wanted to do that's relevant to my opinion, which, in this case is benzene, TCE, PCE, and leukemia.

That was the next thing. And then I reviewed the studies that I considered to be meaningful in my analysis that can provide evidence and support the opinion that I'm issuing.

Q. And what criteria did you use to determine when a study was meaningful for the evaluation?

A. The things that I just mentioned, mostly study design, exposure assessment, outcomes analysis.

Q. And the data you're analyzing in that last step is the data with both positive results, meaning increased risk, and data with negative results, meaning decreased risk; is that correct?

A. No, increased risk -- yes. I take both

1 into consideration.
2 Q. And do you weigh them against each other?
3 A. I did not weigh them against each other.
4 I think that that would be -- well, the one way to
5 weigh a study against each other is if you perform a
6 meta-analysis type of analysis when you compare
7 individual epidemiological studies, and you want to
8 make sure you're adjusting for all possible
9 differences.
10 And there is a very well-established way
11 to do it. I think the one example that I can give --
12 we talked about Bove's study from Camp Lejeune and
13 why I think the mortality data are probably less
14 relevant than incidence data. So if you talk about
15 weighing the study in this way, yes, I did that.
16 Q. Okay. Because I'm trying to understand.
17 You know, it says the weight of the evidence
18 approach, and I want to understand the method you
19 used to weigh the evidence. What --
20 A. Yes. That's what I just said.
21 Q. Okay. So, again, in you could lay it out
22 for me, what are the factors that made one study more

weightful than another?

A. Yeah, so multiple things. We can start with very simple case numbers. If you have a study that studies 300 people and three cases versus the study that studies 10,000 people and 300 cases, obviously, I think there's a different weight to it.

The study that has precise or relatively precise assessment of the exposure level to the study that relies on recollection or positions, obviously, is going to have a different weight, right?

The study that's well-balanced means controls, and cases that are well-balanced will have more weight in the study that (sic) they're (sic) not. So I look at all the studies and that's what I mean by weighing the evidence between different studies.

Q. Okay.

A. They're all peer-reviewed studies. But as you know, the studies are of different quality and you would pay attention more to the results of the study that was either better designed or the data were more granular or it was just more data that we

could come -- we support or disprove our association between different agents and outcomes.

Q. So the precision of the exposure is an important consideration for you in that method, correct?

A. It is important, yes.

Q. Does that mean a study that has individualized exposure data is more precise than one that has population exposure data?

MR. TELAN: Object to the form.

A. Not necessarily. I think it all depends. If you compare two studies, even though -- there may be a study that has an individual exposure level, but it's not a very precise measure, right?

They may ask you, What was your position? Were you working in an office or were you working outside? And they consider it individual exposure level data, which as you can tell they're not precise.

The more precise exposure level data is if someone wears a monitor to monitor their exposure to a toxin, which unfortunately is not available for

many studies, the vast majority of the studies.

On the other hand, if you have very well-designed ecological studies with very precise measures of the exposure in certain areas, in some -- and you have large population cohort and a lot of cases, that may be actually more informative in some cases than poorly designed individual level data study.

BY MR. TUBIN:

Q. What's an example of very precise exposure data in an ecological study?

A. Ecological study?

Q. Yes.

A. So I think the one example would be the last one we discussed, right? I think they tried to confirm the precision of their assessment of the level of exposure in certain areas and they did it by comparing their model to what was actually measured on the surface. And that was, like we discussed, within 25 percent difference.

So I think I would consider that well-designed ecological studies with relatively

precise measurements of exposure data.

Q. Plus or minus 25 percent is --

A. Of the level.

Q. -- is precise?

A. I would say for this type of the study it's relatively precise.

Q. So just so I have a conception of this, if you take one -- take 10 parts per billion, right, what's 25 percent above that?

A. 12.5.

Q. And below?

A. Well, it's minus 2.5.

Q. So 7.5?

A. Yes.

MR. TELAN: Hold on. We're getting an instruction.

(Whereupon, there was an interruption by the videographer.)

BY MR. TUBIN:

Q. So unlike for Bradford Hill you don't have a section in your report explaining all of this about the weight of evidence approach, correct?

A. In terms of Bradford Hill?

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. Well, there's a lengthy section on Bradford Hill right from pages 24 to 26 when you detail your conclusions for the various considerations, but there's no similar section for a weight of evidence approach, correct?

A. That is correct.

Q. So for benzene what were the studies that you evaluated and found to have the most weight in this weight of evidence approach?

A. I think there's studies that are mentioned in my analysis and those references.

Q. Right.

A. It didn't mention all the studies, obviously, that I analyzed. I mentioned the studies that I thought were well-designed and either show or did not show association.

Q. Right. So can you name one for me that you applied the weight of the evidence approach that was part of that analysis and you said, yes, this is

a good benzene study?

A. Certainly. We're talking about the epidemiological studies right now, correct?

Q. Correct.

A. So I included I think the Rushton and Romaniuk study, Schnatter, S-C-H-N-A-T-T-E-R. I included Hayes study, which was a cohort study, Stenehjem study. These are some of the studies that I included in my analysis that I thought were informative.

Q. The Rushton study you mentioned, is that the one we looked at a moment ago?

A. Yes.

Q. And that was the study that concluded overall this study does not persuasively demonstrate a risk between benzene and AML, correct?

MR. TELAN: Object to the form.

A. That was the one we just reviewed, yes.

MR. TUBIN: I think we're on 20, right?

(Exhibit Number 20 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. So you also just mentioned the Schnatter study, correct?

A. That is correct.

Q. Do you recognize that as the Schnatter study?

A. Yes.

Q. Now, under results, on the first page it states: "There was little evidence of dose-response relationships for AML, CLL, CML, or MPT (sic), correct?

A. MPD, yes.

Q. MPD, yeah. And that's talking about cumulative benzene exposures, correct?

A. That is correct.

Q. Now, can you read the conclusions into the record, please?

A. Okay. "Relatively low-level exposure to benzene experienced by petroleum distribution workers who (sic) was associated with increased risk of MDS but not AML, suggesting that MDS may be the more relevant health risk for lower exposures."

Q. Now, if you could turn to Table 2 --

MR. TELAN: Table 2, not figure 2?
MR. TUBIN: No, not figure 2, Table 2.
A. Yes.
BY MR. TUBIN:
Q. So you -- on page 15 of your report -- let's put those side-by-side.
MR. TELAN: What exhibit number is this again?
MR. TUBIN: This is 20.
BY MR. TUBIN:
Q. So the middle of that paragraph on petroleum distribution workers --
A. Yes.
Q. -- "The study found that even low-level cumulative benzene exposure was associated with risk of myeloid malignancies, particularly MDS," correct?
A. Correct.
Q. This is for MDS but not AML, correct?
A. Yes, that's correct.
Q. And those sections from the front page that we read regarding little evidence of dose response relationships and low-level exposure being

associated with MDS but not AML, you don't mention those conclusions in your report, correct?

A. Yes. That's correct.

Q. Now, the -- in the following sentence, "In the further analysis of the same data, the risk of AML in terminal workers employed for at least one year was statistically significant" -- and you list the odds ratio 2.02, the 95 percent confidence interval 1.08 to 3.78, correct?

A. Um-hum -- yes.

MR. TELAN: Marcus -- I'm sorry -- what page are you on in his report?

MR. TUBIN: Oh, page 15 in the middle of the petroleum distribution workers.

MR. TELAN: Thank you.

BY MR. TUBIN:

Q. So that figure comes from Table 2 of the Schnatter article, correct. It should be page 1733.

A. The Schnatter was the initial in 2012. The other data comes from Rushton, 2014.

Q. Right. But the 2.02, 1.08 to 3.78, that's from Table 2 of Schnatter, correct?

A. I think so, yes.
Q. Well, let's look. I think it's every tanker driver versus other workers, and then for AML it's that first result. Let me know when you're there.
A. I'm just looking at it, yes.
Q. Okay. So you did not cite the refinery operator figure, correct?
A. Correct.
Q. And the figure you cite was statistically significant, correct?
A. That is correct.
Q. The refinery figure you didn't cite wasn't statistically significant, correct?
A. That is correct.
Q. And if we go over to the other side of the table, the AML figure for terminal versus other facility on the same table showed a decrease in AML risk, correct, with 0.63?
A. Yes.
Q. And you didn't cite this figure, correct --

A. The numbers yes, that is correct.
Q. -- nor did you cite the refinery versus other facility number for AML, correct?
A. Correct.
Q. And that was 1.04 with a confidence interval of .24 and 4.56?
A. Yes.
MR. TUBIN: Okay. Exhibit No. 21.
(Exhibit Number 21 was marked for identification and was attached to the deposition.)
BY MR. TUBIN:
Q. Do you recognize this paper, Dr. Gondek?
A. Yes.
Q. This is the -- how do you say it?
A. I would say it's Stenehjem.
Q. Oh, I've heard it said a million ways. I still don't know the right way.
All right. If we look at table -- Table 4, which is on page 1608, there's no statistically significant excess of leukemia or AML at any exposure level for the cumulative parts per million data here, correct?

A. You're looking at the entire Table 4? I'm sorry.

Q. Correct, for leukemia or AML.

A. That is correct.

Q. All right. And now if you turn to page 1604, in that first full paragraph and do you see the sentence that says, "Because of the paucity of the measurement data" --

A. That's page 1605, which part of the page?

Q. Oh, 1604.

A. Oh, 1604.

Q. It's in the first full paragraph, the lower third.

A. What does it start with?

Q. It starts with "Because of the paucity of measurement data."

A. I'm sorry. Can you show it to me?

Q. It's 1604 and then it's that sentence right there.

A. Oh, I see it. Yes.

Q. Okay. It's been a long day. So can you read that sentence, please?

A. "Because of the paucity of measurement data, experts on industrial hygiene developed semi-quantitative exposure estimates based on information on duration and frequency of job tasks that involved benzene exposure."

Q. And can you read the next sentence, please?

A. "Benzene exposure in everyday life has been considered less important but may have a role when effects from low occupational exposures are addressed."

Q. Thank you. The Bradford Hill criteria are used in epidemiology to evaluate causation, correct?

MR. TELAN: Object to the form.

A. As one of the components, yes.

BY MR. TUBIN:

Q. As a physician you do not use Bradford Hill criteria, correct?

A. Not in my everyday practice. That is correct.

Q. Well, outside of your everyday practice, do you ever use the Bradford Hill criteria as a

physician?

A. I use a component of it, yes.

Q. What component?

A. Epidemiological evidence, biological evidence, mechanistic studies, animal studies, studies in human cell lines.

Q. Would you agree you've only applied the Bradford Hill criteria in expert litigation work?

A. I think it's most often applied in litigation work.

Q. But have you only applied it in litigation work?

MR. TELAN: Object to the form.

A. Well, like I mentioned, I do apply a component of the Bradford Hill viewpoint in my everyday practice. But as stated as nine components, I think it's mostly for litigation work.

BY MR. TUBIN:

Q. Okay. Right. Do you have the Bonar transcript? Did we enter that as an exhibit? If you could look at Exhibit 18, page 75.

A. Yes.

MR. TELAN: Hold on. Let me get there real quick.

MR. TUBIN: Yeah, yeah.

BY MR. TUBIN:

Q. If you could read predominantly from line 15 to 18?

A. Sure.

"QUESTION: Do you apply the Bradford Hill criteria outside of that context that is in your work as a physician/scientist?"

"ANSWER: No."

Q. Now, you learned how to apply it through legal work, correct?

MR. TELAN: Object to the form.

A. Oh, nine Bradford Hill criteria, yes, I use it mostly for legal work.

BY MR. TUBIN:

Q. But I'm talking about how you learned how to apply it.

A. No.

MR. TELAN: Object to the form.

A. I learned how to apply epidemiological

evidence and mechanistic evidence a long time ago.

BY MR. TUBIN:

Q. So page 77, lines 8 to 11 of the Bonar transcript?

A. Um-hum -- yes.

Q. "QUESTION: Where did you learn how to apply the Bradford Hill criteria?"

"ANSWER: Like most of it, through medical/legal work," correct?

MR. TELAN: Wait. Are you reading it or are you asking him --

MR. TUBIN: I'm asking if I read it correctly.

A. Yes, you read it correctly.

BY MR. TUBIN:

Q. So you say "medical/legal."

A. Yes.

Q. So you learned some of this in coursework; medical work; is that correct?

A. Yeah. We all evaluate scientific literature on causation. We don't necessarily use all nine Bradford Hill viewpoints, I would call them,

in our everyday clinical practice, but we do use components of it to make sense of the medical data that we need to evaluate.

MR. TELAN: Let me just object. To the extent that this is going to be used or attempted to be used at trial as impeachment, I would object as improper impeachment.

BY MR. TUBIN:

Q. Dr. Gondek, you're not an epidemiologist, correct?

A. No, I'm not.

Q. But you've taken a basic course in epidemiology and in statistics, correct?

A. Yes.

Q. And did you learn any aspect of the Bradford Hill there?

A. In my epidemiology, like the components of it?

Q. How to apply it.

A. Yes. Yes.

Q. You learned how to apply it in your epidemiology coursework?

A. In my review of the medical literature, yes.

Q. Okay. And is the Bradford Hill criteria generally accepted in your field as a physician?

MR. TELAN: Object to the form of the question.

A. As a Bradford Hill criteria -- we don't generally use that as a Bradford Hill criteria in the medical field. It is more relevant, I would assume, to epidemiology. In a clinical realm we rely more on clinical trials, prospective clinical trials and mechanistic studies.

BY MR. TUBIN:

Q. So on page 76, so I understand this correctly, the question on line 18:

"QUESTION: And is the criteria" -- it's referring to the Bradford Hill criteria -- "generally accepted in your field?"

"ANSWER: As physicians we almost never use Bradford Hill criteria."

That's correct, right?

A. That's what I said, yes.

Q. And you began working as an expert witness in about 2022, correct?

MR. TELAN: Object to the form.

A. I think that's about right.

BY MR. TUBIN:

Q. Okay. And how many cases have you performed a Bradford Hill analysis in?

A. Five or six, I would say.

Q. Okay. Do you know the names of those cases?

A. I know some, but not all. I think some we just discussed. You have the depositions from those cases.

Q. Bonar and Godfrey?

A. Yes.

Q. Any others you can remember?

A. There was one report I provided for specific causation for Camp Lejeune. I think it was the Amsler, A-M-S-L-E-R, case. And I provided reports on maybe one or two additional cases on benzene in association with leukemia.

Q. Doucette, would that be one?

A. Yes.
Q. Doucette is the name of the case, D-O-U-C-E-T-T-E, I believe?
A. Yes.
Q. So Bonar, Godfrey, Doucette, Amsler. Any others you can recall?
A. Jones. I don't think I submitted the final report on that.
Q. What kind of case was that?
A. It was a case of benzene exposure through diesel exhaust, and I believe it was non-Hodgkin's lymphoma.
Q. What was your conclusion on causation in that case?
A. That benzene exposure level above background is associated with increased risk of non-Hodgkin's lymphoma.
Q. Okay. Let's turn to page 24 of your report. The bottom third of that first paragraph under Bradford Hill Considerations, you state: "Thus, biological plausibility -- the understanding of mechanisms through which an exposure leads to

1 cancer -- is often more critical than epidemiological
2 associations," correct?
3 A. That is correct.
4 Q. Is that an approach you came up with?
5 A. No. I think that's well-established in
6 2025 in cancer biology.
7 Q. 20 -- oh, just established --
8 A. I think this concept is well-established
9 nowadays in the cancer biology field.
10 (Simultaneously speaking addressed by
11 Madam Reporter.)
12 BY MR. TUBIN:
13 Q. Can you identify for me anyone else in
14 your field who shares this approach?
15 A. Yeah, many reviewers of my papers.
16 Q. Can you identify one for me?
17 A. Well, they're anonymous reviews. So every
18 time the paper is reviewed, it's an anonymous review
19 so we don't know who they are, but I can explain what
20 I mean by that.
21 Q. Do you have -- sorry.
22 Do you have any colleagues that share this

approach?

A. Many of them.

Q. Can you name a colleague that does?

A. All my colleagues I work with in the oncology center. You want me to give the names of the people I work with?

Q. Yes. I would like a name.

A. Of course Rick Jones, Mark Levitz, Gabriel Gower (phonetic) -- and I can keep going.

Q. No. That's fine. Do you have a reference in which I could look and see that biological plausibility is often more critical than epidemiological associations in Bradford Hill?

A. Yes. It's through most papers that I submitted and the reviews I got from peer reviewers. If you want me to explain, I can explain what I mean by that.

Q. One second, because I was under the impression that you don't perform Bradford Hill reviews in your work as a physician and scientist.

A. So that's why I would like to clarify.

Q. Okay. Before you do that, I just want to

1 know if you can identify for me a reference where
2 biological plausibility is more critical than
3 epidemiological associations in Bradford Hill
4 criteria?
5 A. The reference right now?
6 Q. Yes, an epidemiological reference --
7 A. I can't -- I don't know on (sic) the top
8 of my head.
9 Q. So this is just based off of your
10 experience?
11 A. That's based on experience and state of
12 knowledge nowadays, yes.
13 MR. TELAN: Why don't we take a break?
14 We've been going about an hour and 15 minutes. Take
15 five.
16 MR. TUBIN: Yep, sounds good.
17 THE VIDEOGRAPHER: Off the record at 3:00.
18 (A break was taken.)
19 THE VIDEOGRAPHER: Back on the record at
20 3:12.
21 BY MR. TUBIN:
22 Q. Dr. Gondek, looking at your report, pages

24 to 26, it's the Bradford Hill analysis, correct?

A. That is correct.

Q. For the Hill considerations you analyzed here, did you weigh them against each other?

A. The different viewpoints?

Q. Different considerations. Like, for example, for benzene the considerations you listed and analyzed, did you weigh them relative to one another?

MR. TELAN: Object to the form.

A. You mean different agents or different criteria to each other?

BY MR. TUBIN:

Q. Different criteria to each other for each analysis.

A. I did not because as far as I know, sir, Bradford Hill did not provide any references into how we should weigh those criteria. I think the only one that was provided was temporality, which was absolutely necessary to even entertain association.

Q. Okay. Now, nowhere in your Bradford Hill analysis for benzene do you identify an exposure

level, correct?
A. Well, in my opinion or in --
Q. Here in section A under benzene when you're going through the criteria; for example, strength --
A. Biological gradient, that's what --
Q. Sure.
A. Yeah.
Q. But there are no levels of exposure identified here, correct?
A. That is correct.
Q. And the same is true for TCE and PCE, correct?
A. That is correct.
Q. On page 18 --
MR. TELAN: Of his report?
MR. TUBIN: Yeah, of his report -- sorry.
BY MR. TUBIN:
Q. The second sentence that starts, "In 2017 the Agency for Toxic Substances and Disease Registry performed a meticulous analysis of available high quality of epidemiological studies and concluded" --

and then there's a conclusion for TCE, correct?

A. Yes.

Q. So you still would characterize that 2017 assessment as a meticulous analysis, correct?

A. That is correct.

Q. Let's go to page 22. Above VC and Leukemia, this is the tail end of your section on PCE and leukemia. And you disagree here with ATSDR's conclusion of below equipoise for PCE and leukemia, correct?

A. Yes, in the sense that I would put it in the above equipoise -- or equipoise and above, rather than below.

Q. Okay. And they said equipoise and below, correct? So ATR set is equipoise and below, and you disagree and say the evidence for PCE and leukemia is equipoise and above, correct?

A. That is correct.

Q. Now, you'd consider IARC's monographs to be reliable authorities on carcinogens; is that correct?

MR. TELAN: Object to the form.

A. I don't think I would use the term "authority." I would use an expert opinion on carcinogens and cancer. The reason why I would say that is because they do change the more data becomes available and they do change over time.

BY MR. TUBIN:

Q. So how would you -- a reliable assessment? How would you characterize IARC?

A. A reliable assessment based on the evidence available at the time.

Q. And you relied on the IARC monographs in your report, correct?

A. I did take it into consideration to form my opinion.

MR. TUBIN: Okay. We're at 21?

THE REPORTER: 22.

MR. TUBIN: 22.

(Exhibit Number 22 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recognize this document, Dr. Gondek?

A. Yes.
Q. What is this document?
A. It's an IARC monograph on trichloroethylene and tetrachloroethylene and some other chlorinated agents.
If you could turn to page 329, this is IARC's evaluation for PCE, correct?
MR. TELAN: Object to the form.
A. That is correct.
BY MR. TUBIN:
Q. And does IARC identify anywhere here an association between PCE and leukemia?
A. No.
Q. Go look at page 21 of your report. On page 21 you state that the overlap between TCE and PCE complicates any PCE study findings, correct?
A. That is correct.
Q. And these are studies of dry cleaning workers, correct, these PCE studies?
A. Most of them, yes.
Q. Can you identify any that aren't?
A. No -- well, Cohn's study. It wasn't dry

cleaners.

Q. Okay. Now, you say the majority of the PCE exposures studied are airborne, correct?

A. That is correct.

Q. Can you identify any exposures that are not airborne?

A. Cohn studies.

Q. And other than the Cohn study you found no studies on PCE exposure through ingestion, correct?

A. That is correct.

Q. You state on the next page, the top of page 22: "It is estimated that 100 percent of PCE is absorbed after oral ingestion"?

A. That is correct.

Q. Who has estimated this?

A. It's based on data from the similar volatile organic compounds and the absorption of the molecules with the similar structure. They're 100 percent observable through oral road (sic).

Q. Can you identify any studies that include that data?

A. Particularly for PCE or specifically for

PCE?

Q. Yeah. I want to know a study that supports the estimation that 100 percent of PCE is absorbed after oral ingestion?

A. That's, again, extrapolation from the data of other VOC that are 100 percent absorbable for oral roads (sic).

Q. So nothing on PCE specifically?

A. I don't think there's anything on PCE specifically.

Q. Okay. So, Dr. Gondek, have you reviewed Dr. Goodman's leukemia report in this case?

A. No, I did not.

MR. TUBIN: Mark this as Exhibit 23.

(Exhibit Number 23 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. This is excerpts from Dr. Goodman's leukemia report, and I want to direct your attention to attachment F. You may have to take a second to scroll through. It's PCE epidemiology studies.

MR. TELAN: If we're going to ask him

1 questions about this since he's never reviewed it,
2 Marcus, I'd like to give him some time to look it
3 over just to get a feel because I just don't want him
4 to answer questions about a single page in a document
5 that has 60 pages where he hasn't reviewed a single
6 page of it.
7 MR. TUBIN: Understood. How long would
8 you like to take?
9 MR. TELAN: Well, are you just going to
10 ask him questions about a specific table?
11 MR. TUBIN: I'm not going to ask him
12 details about any specific study.
13 MR. TELAN: I would say ten minutes at
14 least just to kind of have him run through it just to
15 get a feel for it on leukemia. And I suppose if --
16 is there a particular -- I guess page 78 to 101 seems
17 to be -- or 78 to 117 --
18 MR. TUBIN: I'm looking for a page number.
19 No. It's not going to be in the list of page
20 numbers. These are -- I provided the Table of
21 Contents, but these are from the attachments,
22 attachment F to her report. So this --

MR. TELAN: Oh, I see. This isn't the full report?

MR. TUBIN: No. No. No.

MR. TELAN: This all seems like an awfully light --

MR. TUBIN: Yeah. No, it's not full report. It's the tables at the end of the report. I just want to know if he's reviewed certain studies that are listed here. I'm not going to ask about --

MR. TELAN: Yeah. Go ahead and ask a question, and if I've got a specific objection, I'll make it.

MR. TUBIN: Okay.

MR. TELAN: I apologize for throwing you off. Was there a table you directed him to?

MR. TUBIN: Yeah. Attachment F is the PCE epidemiology studies. It's table F.2, PCE and Leukemia Cohort Study Results.

BY MR. TUBIN:

Q. Are you there?

A. Yes.

MR. TUBIN: You guys are set?

MR. BELL: Yes.

BY MR. TUBIN:

Q. Dr. Gondek, do you see the third study down Travier, et al. (2002)?

A. Yes.

Q. Have you reviewed that study?

A. I don't recall reviewing that one.

Q. What about two down below that, the Pukkala, et al. (2009)?

A. I don't recall that either.

Q. And then on the next page the first study listed there, Lipworth, et al. (2011), do you recall reviewing that?

A. I -- probably not.

Q. I'm sorry?

A. I don't recall that.

Q. So if we could go to attachment D, which is TCE epidemiology studies, table D2 -- D.2 TCE and Leukemia Cohort Study Results.

And the first study listed there is Garabrant, et al. (1988).

A. I see that.

Q. You did not review that study, correct?
MR. TELAN: Object to the form.
A. I don't believe so.
BY MR. TUBIN:
Q. The study at the end, Chang, et al. (2003) at the bottom of the page, you've not reviewed that study, correct?
A. I don't recall that.
Q. Going to the next page, the second from the bottom, Sung, et al. (2007), do you recall reviewing that study?
A. I don't remember reviewing that one.
Q. Okay. Two pages later do you see the study at the top is B-A-H-R, Bahr?
A. I don't remember.
Q. You don't recall reviewing that --
A. I don't recall reviewing it.
Q. One more on the next page, did you review the study Saberi, (2013)?
A. I'm sorry. What -- oh, I see it.
I don't remember on (sic) the top of my head whether I reviewed that or not.

Q. Is it true that if a material is not listed in your materials considered list you didn't consider it for this case?

MR. TELAN: Object to the form.

A. I probably reviewed more manuscripts that are listed on my reference list. I just didn't include it on my reference list.

The reason why I focused on New Jersey study is because the level -- well, not the level -- the routes of exposure was very similar to Camp Lejeune.

And knowing that three inhalation -- most studies addressed the inhalation part of it, we know that 75 percent of PCE is exhaled in unchanged form and 15 percent is absorbed. I thought the route of ingestion was important, so I think the New Jersey study was probably more relevant to the Camp Lejeune case.

BY MR. TUBIN:

Q. I may have misheard, so I just want to be certain. If a material is not on your materials considered list you didn't consider it; is that

correct?

MR. TELAN: Object to the form. Asked and answered.

A. No. There are certain materials that I put it -- on my references. I looked at more papers than I put on my reference list. I just don't remember whether I reviewed those papers or not. I reviewed more papers that are on my reference list, let me put it this way.

BY MR. TUBIN:

Q. So everything you reviewed in forming your opinions in this case is either on your reference list or materials considered list; is that correct?

MR. TELAN: Object to the form.

A. No.

BY MR. TUBIN:

Q. So there are studies or materials that you reviewed in forming your opinions that I have no way to find based on your report?

MR. TELAN: Object to form.

A. Well, I reviewed more studies that I put on my reference list. Some studies I didn't put on

the reference list like we discussed because they were not relevant.

Some studies -- like, I don't recall reviewing those studies. I just don't remember whether I reviewed them or not if they are not on my reference list. I did not include them in my report.

BY MR. TUBIN:

Q. Do you recall reviewing ATSDR's toxicological profiles?

A. The ATSDR document, yes.

Q. Not the assessment of evidence. ATSDR's toxicological profile for benzene, for example.

A. I don't remember if I did. I definitely reviewed the ATSDR assessment.

Q. Right. This is a different document. I didn't see it listed in your materials --

A. I don't recall reviewing that.

Q. Okay. Is it fair to say you didn't review ATSDR's toxicological profile for TCE?

MR. TELAN: Object to the form.

A. That's probably true.

BY MR. TUBIN:

Q. Is the same true for ATSDR's toxicological profile for PCE?

A. That's correct.

Q. You did not discuss the pliofilm worker studies in your analysis of the epidemiology for benzene, correct?

A. That is correct.

Q. Would you agree the pliofilm worker studies provide the best set of published data on human cancer risk from benzene exposure?

MR. TELAN: Object to the form.

A. The best set?

BY MR. TUBIN:

Q. Um-hum.

A. I can't say that. I will have to look back and -- look back again on the study. I don't think I can say that right now.

MR. TUBIN: Exhibit No. 24.

(Exhibit Number 24 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Do you recognize this document, Dr.

Gondek? It's an excerpt, the first portion of -- Carcinogenic Effects of Benzene: An Update from EPA.

A. I'm not sure I recognize that. I may have looked at it.

Q. You're unsure if you've ever reviewed it or not?

A. Hold on a second. I guess I -- I may have reviewed it.

Q. This document is from April 1998, correct?

A. Yes.

Q. And are you aware of any other carcinogenic effects of benzene updates from the EPA?

A. No.

Q. Turn to page 8, which in Roman numerals is viii. And the sentence that starts, "The study of pliofilm rubber workers," can you read from page viii to page ix that sentence and the sentence after it?

A. I'm sorry. Which paragraph is that on page viii?

Q. Page viii, "The study of pliofilm rubber workers."

A. Oh, the one -- the last?

Q. Yeah, that last --

A. "The study of pliofilm rubber workers at three facilities in Ohio provides the best published set of data to date for evaluating human cancer risks from exposure to benzene."

Q. And then the next sentence.

A. "Compared to other published studies, this cohort has the fewest reported co-exposures in the workplace to other potentially carcinogenic substances that might confound risk analysis for benzene."

Q. Thank you, Doctor.

A. I just want to mention this was in 1998, right?

Q. Um-hum -- correct. Are you familiar with the National Academies of Sciences?

A. Yes.

Q. Have you ever cited the work of the National Academies in any of your publications?

A. I don't remember.

Q. Do you think it's likely that you have?

A. Probably.

Q. Would you agree their publications on a topic are authoritative?

MR. TELAN: Object to the form.

A. Again, I wouldn't say authoritative, but they are helpful.

BY MR. TUBIN:

Q. In 2009 the National Research Council of the National Academies published a report titled, "Contaminated Water Supplies at Camp Lejeune Assessing Potential Health Effects."

That document is not on your materials considered list, correct?

A. That is correct.

MR. TUBIN: Exhibit No. 25.

(Exhibit Number 25 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. If you look at page v, little Roman numeral v, Committee on Contaminated Drinking Water at Camp Lejeune, do you know any of the people listed under members or staff?

A. Personally, I don't.

Q. Professionally?
A. I'm aware of some of the publications of some of the members.
Q. Who?
A. I think I cited Dr. Savitz's publication. I'm wondering if anyone else is. Maybe Clement.
Q. Clement?
A. Maybe, yes. I definitely included some of Dr. Savitz's references.
Q. If you could turn to page 8 of the report, you'll see this is box 2 --
A. Page 8?
Q. Yeah, page regular 8. It should be the second to last page of this excerpt.
A. Yes, I see it.
Q. Box 2, Categorization of Health Outcomes Reviewed in Relation to TCE, PCE, or Solvent Mixtures?
A. Yes.
Q. Do you see the categories are: Sufficient Evidence of a Causal Relationship, Sufficient Evidence of an Association, Limited/Suggestive

Evidence of an Association, Inadequate/Insufficient Evidence to Determine Whether an Association Exists, and Limited/Suggestive Evidence of No Association?

A. Yes.

Q. You're familiar with this categorization scheme, correct?

A. I don't think I am. I don't exactly know what goes into these categories. I don't remember that on (sic) the top of my head.

Q. Have you seen these categories used before in IARC?

A. Yes. I think there are multiple components, but I will have to see exact list in the way of each component. I just don't remember all the classifications and what criteria goes to it.

Q. Do you see there are no outcomes listed for sufficient evidence of a causal relationship?

A. I see that.

Q. And you see there's no outcomes listed for sufficient evidence of an association, correct?

A. I see that.

Q. You cite several papers by Dr. Martyn

Smith; is that correct?

A. Yes.

Q. Okay. Dr. Smith is a well-respected authority on benzene toxicity, correct?

MR. TELAN: Object to the form.

A. I would say so.

BY MR. TUBIN:

Q. Have you ever worked with Dr. Smith?

A. No.

Q. Do you consider Dr. Smith to be an authoritative expert on the relationship between benzene and leukemia?

MR. TELAN: Object to the form.

A. I don't think I have an opinion on that.

MR. TUBIN: Exhibit 26.

(Exhibit Number 26 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. On page 10 of your report you cite his work towards the top in support of the statement that "Several studies demonstrate an increased risk of AML at near-ambient benzene concentrations," correct?

A. Yes. I see that.

Q. This is Exhibit 26. This is an affidavit dated December 24th, 1996 from Professor Martyn T. Smith.

If you look at the bottom of page 1 of the affidavit, Dr. Smith being duly sworn and deposed says: "I have published more than 125 articles in peer-reviewed scientific journals and books, many of which relate to benzene toxicity"; is that correct?

A. Yes.

MR. TELAN: Object to the form.

BY MR. TUBIN:

Q. "I am also a recipient of several large research grants from the National Institutes of Health and other organizations, one of which is entitled 'Biomarkers of benzene exposure and genotoxicity.'"

Did I read that correctly?

A. Yes.

Q. Okay. Turn to the next page, page 2. In paragraph 3, Dr. Smith says: "Intake of an additional 56 micrograms of benzene per day from

drinking 2 liters of water contaminated with 28 parts per billion benzene would make little impact on the average person's exposure to benzene, especially if that person was a smoker or lived with a smoker."

Do you have any reason to disagree with Dr. Smith's testimony here?

MR. TELAN: Object to the form of the question. And to the extent that this is sought to be introduced into evidence, I don't want my silence to this to be construed as somehow an admission to that.

So I would object as there's no predicate for this or foundation as Dr. Smith has not been deposed and has not been listed as a witness by the Department of Justice.

So I know this is a speaking objection, but I didn't want silence to be construed as an admission. There's my objection.

BY MR. TUBIN:

Q. Do you have any reason to disagree with Dr. Smith's testimony here?

A. I would agree with the estimated level of

benzene. I'm not entirely sure what the second part of his opinion is based on, that it made little impact on an average person's exposure to benzene especially if the person was a smoker or lived with a smoker.

I don't think I have enough information right now to either agree or disagree with this statement.

Q. Knowing there's 28 parts per billion benzene in 2 liters of water per day is not enough information for you to know whether you disagree --

A. Well --

MR. TELAN: Object to the form.

A. -- we reviewed the literature recently on a much lower level of benzene and how it affects the incidence. So I think Dr. Smith's statement was probably based on the literature available at the time of the statement.

So I don't know if it's still valid. As you know, the opinions change as we have more information, more data, so I can't say I agree with that.

BY MR. TUBIN:

Q. Understood. So the next paragraph: "Using highly conservative assumptions, the U.S. EPA has estimated" --

A. I'm sorry. Where are we right now? I'm lost.

Q. 4.

A. Yes, I see it.

Q. "Using highly conservative assumptions the U.S. EPA has estimated that an intake of 1.32 micrograms of benzene per day over a 70-year lifetime poses a 1 in 1 million excess cancer risk. An intake of 56 micrograms per day for 70 years would therefore pose about a 40 in a million excess risk. If intake were for less than 70 years, then this risk would be reduced substantially."

Do you agree with that?

MR. TELAN: Same objection and same previous objection.

A. I can't agree or disagree without reviewing the data that went into this statement.

BY MR. TUBIN:

Q. Okay.

A. I don't know what this data or statement is based on.

Q. Well, it's based on an intake of 1.32 micrograms --

A. No. This kind of data -- like, this is an assumption. The assumption is made on some sort of data and results. I just don't have access to them, so I can't really answer your question.

Q. But doesn't it say it's based on an estimation of an intake of 12.32 micrograms of benzene per day? Isn't that the data?

MR. TELAN: Same objection as before.

A. That's the data about the intake, but I'm not aware of any data showing that this intake would increase the risk from one in a million to 40 in a million. I don't know what this statement is based upon.

BY MR. TUBIN:

Q. Okay. So it continues: "If intake were for less than 70 years, then this risk would be reduced substantially. For example, if exposure were

for only 12 years, the excess risk would be reduced to 6.8 in a million, and if it were only for 14 months, it would be reduced 60-fold to only .66 in a million.

"This is an insignificant and inconsequential risk and is considered such by federal and state regulatory agencies."

Do you have any reason to disagree with Dr. Smith's testimony here?

MR. TELAN: Object to the form and same objection as previously raised as to the substance.

A. I think my answer is similar to your last question. I can't agree or disagree without reviewing the data.

BY MR. TUBIN:

Q. Okay. On page 4 of your report --

A. Yes.

Q. -- in this section you discuss the "at least as likely as not" language, correct?

A. That is correct.

Q. And that language comes from the Camp Lejeune Justice Act; is that correct?

A. That is correct.

Q. Did the plaintiffs' attorneys provide you with the burdens and standard of proof section of the Camp Lejeune Justice Act and tell you to rely on that in your analysis?

A. They provided me with the Camp Lejeune Justice Act and the standard that I should form my opinion based on, which has made my interpretation or different standard at what we used to use in legal cases.

Q. Did you review the complete language of the statute?

A. I did.

Q. And you're not an attorney, correct?

A. That is correct.

Q. You have no legal training, correct?

A. That is correct.

Q. You have not used the phrase "at least as likely as not" in any of your scientific papers, have you?

A. No. I think that's the legal statute. And if I may say, I think ATSDR also touched upon

that, what that means, and they actually gave an example of what it means to be equipoised and above versus more likely than not.

Q. Would you agree "at least as likely as not" is not scientific vernacular?

MR. TELAN: Object to the form.

A. I don't think I would agree that it's not scientific. I think it's probably synonymous to equipoise, and equipoise is a well-defined term in the clinical studies.

BY MR. TUBIN:

Q. What's the definition of equipoise in clinical studies?

A. So the definition -- well, equipoise is the basis of clinical studies and mostly ethical concerns in prospective clinical trial.

What it means is that ethically the treatment that we apply should be as likely as not or less toxic than the control arm or control treatment. So equipoise means that it's kind of even or similar and that we don't have a reason to believe it's different.

And every time we submit study for ethical review I think they look at the equipoise between treatment arms just to make sure that we're not harming people with the intervention that may be harmful.

Q. Have you used the phrase "equipoise" in a scientific paper you've published?

A. I don't remember if I used this exact term. I just don't remember now.

Q. Would you agree it's a term used in the legal and regulatory framework and not in causation assessments?

MR. TELAN: Object to the form.

A. Yeah. I think it -- it's an interpretation of the results.

It's just a different standard of interpretation of the results. It's not a term used for causation. I agree.

BY MR. TUBIN:

Q. Have you not seen it in an epidemiological study for it?

A. The equipoise --

Q. Yes.
A. -- itself? I don't remember seeing that.
Q. And equipoise and above comes from the classification scheme in the 2017 ATSDR assessment of evidence, correct?
A. That is correct.
Q. And this classification scheme was adopted from the VA's disability decision-making scheme, correct?
A. Correct.
Q. It's different from IARC's classification scheme, correct?
A. I believe so, yes.
Q. Would you agree the scheme is regulatory-based?
MR. TELAN: Object to the form.
A. I don't have an opinion on that.
BY MR. TUBIN:
Q. Agency-based?
MR. TELAN: Object to the form.
A. I don't have an opinion on that.
BY MR. TUBIN:

Q. Is Karen A. Robinson a colleague of yours at Johns Hopkins University?

A. No.

MR. TUBIN: Exhibit No. 27.

(Exhibit Number 27 was marked for identification and was attached to the deposition.)

BY MR. TUBIN:

Q. Have you ever seen this document before, Dr. Gondek?

A. No.

Q. It appears to be from the National Academies of Sciences, correct?

A. Yes.

Q. Review of the Department of Veterans Affairs Presumption Decision-Making Process?

A. Yes.

Q. So turn to page 103.

MR. TELAN: Marcus, if you're going to ask him questions about this since he's not seen this before, I'd ask that he be given some time to review it in its entirety just so that he gets a basis for it.

MR. TUBIN: I'm only going to ask him questions about pages 103 and 104, which are part of a section that begins on 102. So if he wants to review 102 to 104, that's fine, but I don't want to go through the whole document.

MR. TELAN: I just don't think it's fair to hand the witness a document that's got multiple pages and then restrict his ability to review it to two pages. Even though you say that you're only going to ask -- I would ask that he be given sufficient time just to review from pages 100 to --

MR. TUBIN: How much time?

MR. TELAN: -- to 105 -- whatever that -- 106.

BY MR. TUBIN:

Q. Dr. Gondek, how long do you think it will take you to review from 100 to 106?

A. Ten or 15 minutes.

MR. TUBIN: I'm not going to ask much about your interpretation of what's in this document. I'm more going to ask about the definitions in this document.

So we can move forward and object if you want, and if, you know, you feel he has to stop to read it to answer the question, then, you know, we can reconsider that. But I'm not going to ask anything detailed about his interpretation.

MR. TELAN: Not that I don't trust you, but I just think it's appropriate protocol that if you're going to hand somebody a document that they haven't reviewed and ask them questions about it that they be given the appropriate time to review the document. I don't think that ten minutes is too much to ask to review six pages.

MR. TUBIN: I mean, I'm fine with five minutes for 102 to 104. That's what I'm going to ask about. That's two to three pages.

MR. TELAN: Let's go off the record. Why don't we see if he can get there in five minutes.

MR. BELL: We'll go off the record.

MR. TELAN: I say we stay on.

MR. BELL: Okay. Keep going.

MR. TELAN: We might go off video record. That way you're not burning tape. Do you want to go

1 off video record, Marcus?
2 MR. TUBIN: Sure, please. Thank you.
3 THE VIDEOGRAPHER: Off the record at 3:55.
4 (Whereupon, we went off the video record.)
5 THE WITNESS: Are you going to ask me
6 about page 104?
7 MR. TUBIN: Primarily 104, yeah.
8 THE VIDEOGRAPHER: Back on the record at
9 4:01.
10 BY MR. TUBIN:
11 Q. So, Dr. Gondek, on page 103 of this
12 document, the last full paragraph, first sentence
13 says: "The VA presumption decision process uses
14 'equipoise' to denote threshold for a presumption in
15 which the probability of a positive association is
16 judged to be equally likely as not, equivalent to a
17 probability of exactly 50 percent," correct?
18 A. Yes.
19 Q. Is that your understanding of the term
20 "equipoise"?
21 A. Yes.
22 MR. TELAN: Object to the form.

A. Yes. So equipoise is similar or above equipoise would be at least as likely as not.

BY MR. TUBIN:

Q. Okay. So then on the next page the committee authoring this document states that, "The committee notes" -- first full paragraph at the beginning: "The committee notes that 'equipoise' is used in clinical research to refer to a situation where there is uncertainty or conflicting expert opinion about the relative merits of two or more interventions rather than a perfect balance of opinions."

Do you agree with that?

A. Yes.

Q. And then at the bottom, the italicized portion: "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."

Do you agree with that?

MR. TELAN: Object to the form.

A. I would add I do. I would add it's a genuine uncertainty among the medical community. People may have strong opinions one way or the other.

I think in my understanding of equipoise it denotes that based on available data there is a genuine uncertainty whether there is or there's no association, for example.

BY MR. TUBIN:

Q. In the Doucette report you provided in February 2024, that was an AML case, correct?

A. That is correct.

Q. You did not write a standard applied section there, correct?

A. I did not.

Q. Okay. But you did here, correct?

A. I did.

Q. Would you agree that the major limitation of most studies is that we cannot quantify the level of exposure?

MR. TELAN: Object to the form.

A. I think that's one of them and the other one is that we don't really have good biomarkers of

exposure because there is -- well, there is a step between the exposure and the level of toxic metabolites, which is particularly important for the agents that we are discussing because there is another layer of what causes the level of toxic metabolites which is metabolism in a different issue.

So the exposure to predrug or prodrug, if you want to call it, and the end-organ damage in the toxic metabolites may be different. There's a few steps. So I think the ideal design or the ideal study would be if we have a biomarker of exposure which is kind of like an end result of the toxin in the body.

BY MR. TUBIN:

Q. One more question, and then we'll take a short break.

To establish a substance causes a disease in humans, would you agree we need well-designed human studies on that substance?

MR. TELAN: Object to the form.

A. Yes, and biological and mechanistic data to support the causation claim.

MR. TUBIN: All right. Take a five-minute break.

MR. TELAN: Sure.

THE VIDEOGRAPHER: Off the record at 4:05.

(A break was taken.)

THE VIDEOGRAPHER: Back on the record at 4:15.

MR. TUBIN: I'm all done, gentlemen. So pass the witness.

MR. TELAN: I need a break just to organize some of my papers. So give me ten. I think we'll be ready in ten to start.

THE VIDEOGRAPHER: Okay. Off the record at 4:16.

(A break was taken.)

THE VIDEOGRAPHER: We are back on the record at 4:50.

EXAMINATION

BY MR. TELAN:

Q. Dr. Gondek, I'm going to start actually with your background.

You were asked a number of questions today

over the last five hours about epidemiological studies that you did include in your report and some that you didn't. I think you told us that you are not an epidemiologist; is that correct?

A. That's correct.

Q. Tell me what you do for a living, and I'll probably ask you to expound upon what you do in the laboratory.

A. So I'm a physician scientist. What this means is that I have both M.D. and Ph.D. So part of my time I'm involved with patient care. So I treat patients with acute myeloid leukemia, MDS, myeloid diseases, and some people with lymphoma and --

MR. TELAN: Oh, it's time to close the door.

A. When I'm not in clinic I have my own research lab, and we study the mechanism of carcinogenesis particularly in myeloid leukemias, to some extent in lymphomas, and what causes cancer.

BY MR. TELAN:

Q. And you said "we" study. Where is it that you do your work both as a clinician and as a cancer

1 scientist?
2 A. At Johns Hopkins.
3 Q. And we're here in Baltimore today for your
4 deposition, obviously?
5 A. Correct.
6 Q. How long have you been affiliated with
7 Johns Hopkins University?
8 A. I came to Hopkins in 2011. I did part of
9 my training at Hopkins, which was hematology
10 fellowship, and I finished in 2014 and I stayed on as
11 faculty since.
12 Q. In terms of your focus within the realm of
13 oncology, do you have a particular focus within the
14 study of cancer?
15 A. Hematologic malignancies.
16 Q. And how do you define hematologic
17 malignancies?
18 A. Well, they're blood cancers. So cancers
19 originate from blood cells.
20 Q. I think that you were asked earlier about
21 whether or not -- and I will misstate the way it was
22 asked, but essentially that we should subdivide out

the different types of blood cancers.

Do you recall being asked about that early on in the deposition?

A. Yes.

Q. Do both non-Hodgkin's lymphoma and leukemias originate in the bone marrow?

A. Yes. So I -- myeloid diseases are from what we call myeloid lineage which are white cells in the body, and they grow and mature in the bone marrow from stem cell.

The non-Hodgkin's lymphomas are what we call B-cell malignancies, and the B stands for bone marrow because they originate in the bone marrow, unlike T-cells that comes from the thymus, which is a different organ.

Q. In terms of your role in this case I think you told us earlier that you focused on the relationship between the contaminants in the water at Camp Lejeune and leukemias; is that correct?

A. That is correct.

Q. But you were asked to some degree about non-Hodgkin's lymphoma. And so in your course as a

clinician scientist, have you had the opportunity to evaluate the association or lack of association between benzene and non-Hodgkin's lymphoma?

A. I did. I think I provided opinions in some of the cases we discussed earlier on the association between benzene and some of the lymphoid malignancies like non-Hodgkin's lymphoma and multiple myeloma.

Q. And in those depositions that we referenced -- and I don't know which one or ones it was specifically -- did you reach the opinion that benzene was, in fact, a causative factor in a person developing non-Hodgkin's lymphoma?

A. Yes. That's correct.

Q. So whether we group hematopoietic cancers together under an umbrella or subdivide it, is your opinion the same; that is, benzene is causative of leukemia and non-Hodgkin's lymphoma?

A. That is correct, and biologically it makes sense given what we know about the cell of origin and bone marrow being a target organ for benzene.

Q. Okay. And I don't know that you were

asked specifically about that, so I'm going to ask you to explain how it makes sense that benzene would cause both cancer -- leukemia as well as non-Hodgkin's lymphoma?

A. So outside of the epi study that we discussed at length, the biological study established that benzene itself is nontoxic to the cell, but the metabolites are.

And there are two main target organs that metabolize benzene: One is the liver and the other one is bone marrow. So most of the enzymes that convert this non-genotoxic metabolites to toxic metabolites reside inside the bone marrow.

So we have a reason to believe that the level of these toxic metabolites is higher in the bone marrow than anywhere else in the body, and that's where stem cells or hematopoietic stem cells reside. So they're direct targets of benzene.

And we know that because people who are exposed to benzene show genetic defects in immune cells or white cells, so we know that's the target organ for the benzene.

Q. Within your field -- and I think you described it as cancer biology?

A. Yes.

Q. Is it a small group of people who like yourself dedicate their career to looking at the origins of blood cancer specifically?

A. I think in places like Hopkins it's not such a small group. There is a small group of people who do both lab-based research and clinical work, but there is a number of people -- and I think there's at least four or five in our division -- who study biology in the lab but are also physician scientists.

Q. Do you belong to any societies in -- professional societies that deal with blood cancer research?

A. Yes. So I'm a member of American Society of Hematology and American Society of Clinical Oncology.

Q. Okay. Are there any committees that you serve on or groups that you participate in within the -- within those particular societies?

A. Yes. So I was a peer reviewer for certain

grants for American Society of Hematology. I served as a reviewer for Blood Journal, which is the Journal of American Society of Hematology.

I'm also a reviewer for JCO, which is the Journal of Clinical Oncology. This is a journal of American Society of Clinical Oncology.

Q. And so as part of your role with those journals and societies do you review papers that are submitted for publication by those societies?

A. Frequently.

Q. Okay. Have you ever come across a paper submitted by Julie Goodman related to causation of blood cancer?

A. As a reviewer, no.

Q. What about Peter Shields?

A. No.

Q. Outside of this case have you ever heard of either of those two individuals in the -- in the, I would say, paternity -- not the correct word -- but in the group of scientists who devote their careers to studying the origin of blood cancer?

A. No.

Q. In terms of your work, you do work -- you're awarded grants by both private and public entities, correct?

A. That is correct.

Q. Have you ever received a grant from the Department of Defense?

A. I recently received a grant. I think it was awarded a month ago.

Q. And what was that to look at?

A. So that looks at the origin of myelodysplastic syndrome which we discussed earlier. It's also a blood cancer sometimes classified as bone marrow failure.

So we propose to study or elucidate the mechanism by which the MDS occurs and what are the premalignant conditions that kind of increase the risk of MDS.

Q. And I know that you had mentioned MDS before. And you've told us, I believe -- you correct me if I'm wrong -- that you considered a number of papers that didn't make their way to your materials considered list or reviewed materials; is that

correct?

A. That is correct.

Q. Tell us why it's important to evaluate when looking at how benzene causes leukemia, why it's also important to look at MDS in that analysis.

A. So several reasons. Number 1) based on the old classification, which is called FAB, French-American-British, there was a different (inaudible) for blast percentage to make a distinction between acute myeloid leukemia and MDS.

Q. When did that change, by the way?

A. So they changed in -- with WHO classification in early 2000. Previously, it was required to have at least 30 percent of blasts in the marrow to classify someone as AML, or acute myeloid leukemia, versus MDS, which was under 30 percent. That has moved to 20 percent.

So some cases historically that were classified as MDS would have been now classified as AML. So they had 25 percent of blasts. Based on previous classification, they wouldn't meet the criteria for AML. They would be MDS. Based on

available classification now these cases are AML.

Q. Okay.

A. So it is really a continuum in most cases between MDS and AML. As an example, if someone comes in with what we called cytopenias, meaning low platelet count, low hemoglobin, maybe low healthy white cell counts and we do a bone marrow biopsy and we see 19 percent or 18 percent of blasts, we would classify it as MDS.

If the same person comes a few months later and we do a biopsy from a different spot and we see 21, 22 percent blasts, they would be classified as AML, which biologically doesn't make sense if you think about it. But we kind of -- not totally arbitrary, but we set this threshold for 20 percent. But it's truly a continuum.

Q. So if I can wrap my head around it as a layperson, if I'm a person that has 20 percent blasts in 1999, I wouldn't be considered to have AML, but in 2001 I would meet the diagnostic criteria for AML?

A. I don't remember exactly the data, but sometime in early 2000 that has changed.

Q. Okay. So studies that were done before that change could have actually underestimated the level or incidence of AML based upon the criteria that was later enacted?

MR. TUBIN: Objection. Form.

A. Some of those participants now would be classified as AML, not MDS.

BY MR. TELAN:

Q. And in light of the objection let me ask it a different way. I don't think it was properly phrased.

Hypothetically, if the cut-off changed in the year 2000, would a 1999 study have held a higher level of blasts -- strike that. That's going to be a poor question as well. I think you know what I'm asking. I just can't ask it correctly.

So if a study was done looking at benzene causing AML prior to the change in the criteria for AML, would it be a fair statement that that study, looking at it today, would have underreported the incidence of AML if it were done prior to the change in criteria?

A. Yes.

MR. TUBIN: Objection to form.

A. More people would be classified as AML now than in the '90s.

BY MR. TELAN:

Q. And I think -- we'll get to that paper. But you were shown a document from Martyn Smith which you had never seen before.

Do you recall that today?

A. Yes, I do.

Q. I don't know that you saw it, but that was a 1996 document?

A. I didn't see the date of the document.

Q. Okay. But that document would likely have been generated under the old criteria of AML?

A. Likely.

Q. Okay. In terms of TCE and PCE, can you explain as a cancer biologist how those two chemicals cause cancer, specifically leukemia, if you would?

A. So if you look at metabolism, well, from the biological perspective, there are studies -- and I think I did cite them in my report -- that showed

chromosomal abnormalities in blood cells, in patients, or in people exposed to TCE and PCE, that suggests that both agents are able to cause DNA damage in blood cells.

If you look at the metabolism of those agents, they're somewhat similar to benzene metabolism. Very similar enzymes are involved in metabolism of these chemicals, and the same enzymes reside in the bone marrow and liver.

Obviously, there is more data on benzene in target organs. We know more about it. We know a little bit less about PCE and TCE, but if you look at the biology and metabolism you can surmise that the bone marrow is also a target of PCE and TCE. And we know that being the case because we see this genetic abnormality in blood cells.

Q. And if you spoke about it, did you speak about the --

MR. TELAN: I know you looked at him when he said it.

BY MR. TELAN:

Q. -- lipophil --

A. Lipophilic?

Q. Lipophilic, yeah. Are both TCE and PCE lipophilic?

A. Yes, they are.

Q. Which means in lay terms that they --

A. They likely have a higher concentration in tissue that is rich in fat cells or lipids. Bone marrow -- we assess what's called cellularity of the bone marrow.

What it means is that it decreases with age. So if we say the cellularity is 60 percent, it means that if you look at the bone marrow cavity the 60 percent of the space is occupied by hematopoietic cells or blood cells or progenitors, P-R-O-G-E-N-I-T-O-R-S, and 40 percent is occupied by fat. So the similarity is basically cellular component to the fat component ratio.

Q. So in terms of -- the mode of action by which benzene causes leukemia, that is different from the mode of action by which TCE and PCE cause leukemia?

A. They both cause DNA damage. I think we

1 understand the mechanism of action a little bit
2 better for benzene, exactly what it does, but they --
3 all three of them are capable of introducing
4 chromosomal abnormalities and DNA damage to blood
5 cells.
6 Q. We talked a little bit about the
7 genotoxicity of benzene as well as TCE and PCE, but I
8 want to talk to you a bit about immunotoxicity.
9 Is benzene considered to be an immunotoxic
10 substance?
11 A. Yes. It affects the immune system.
12 Q. And is TCE also considered to be an
13 immunotoxic substance?
14 A. Yes.
15 Q. Is the immune system implicated in the
16 causal chain of blood cancers?
17 A. It is.
18 Q. Most of the epidemiological studies that
19 you were shown today were single-chemical studies,
20 were they not?
21 A. They were.
22 Q. The Marines who drank the water or bathed

in the water or inhaled the vapors from the water that was distributed by the Hadnot Point Treatment Facility were exposed to a combination of benzene and TCE and PCE and vinyl chloride and other substances in varying degrees. You're aware of that?

A. Yes.

MR. TUBIN: Objection to the form.

MR. TELAN: What's wrong with the form of that?

MR. TUBIN: Vague.

BY MR. TELAN:

Q. You reviewed the ATSDR study, correct?

A. Yes.

Q. Did you note in the study that there were samples drawn in the early 1980s revealing the presence of benzene, TCE, vinyl chloride, PCE, and some other substances from the Hadnot Point water treatment system?

MR. TUBIN: Objection. Form.

A. Yes.

BY MR. TELAN:

Q. You were asked a little bit about the term

"synergy" today.

Do you recall that?

A. Yes.

Q. What is the significance of being exposed not only to a single genotoxic chemical like benzene which also has immunotoxic properties but also at the same time being exposed to a chemical like TCE?

A. So cancer is a multistep process and multistep failure if you think about that. So agents that affect different levels of protection against cancer may increase the risk of malignancy.

So the ones -- that is just direct DNA damage. So the agents that cause DNA damage can result in a transformation of healthy cells to a cancer cell. There is failure at multiple levels.

Number 1 is when there's DNA damage the cell is trying to fix the damage. If -- and sometimes it can and sometimes it can't. It depends on the level of damage. If you have more damage because you have more agents, it's unlikely to happen.

The second thing is, if it cannot be

fixed, then there is this mechanism that's supposed to eliminate the cell from the body.

Q. Is that the immune system or is that --

A. No, no, no. That's just something -- that's usually the gene P53 that's supposed to eliminate the cell.

If that fails, there is the immune system that's supposed to eliminate these abnormal cells from the body again to prevent cancer. So if you have a weak immune system you can assume that the cells that are supposed to be destroyed by the immune system is not.

And the reason why we know it works is because we kind of use the opposite to treat cancer. We try to stimulate the immune system to get rid of cancer, so there is the whole field of cancer immunology when we have agents that actually activate in the system in order to kill existing cancer.

So we know an ineffective immune system or a weakened immune system is a risk factor for malignancies, and particularly blood cancer and non-Hodgkin's lymphoma is one of them.

Q. Just so that I'm clear on this, if one is exposed to benzene and that benzene gets into the bone marrow, does that then cause damage to the bone marrow directly through DNA strand breaks or --

(Whereupon, Madam Reporter asked for clarification from Counsel due to simultaneously speaking.)

MR. TUBIN: Objection.

MR. TELAN: All right. I'll just start over again.

BY MR. TELAN:

Q. Given your testimony today, this question relates to exposures to benzene above the ambient level of benzene.

If one is exposed to benzene above the ambient level does that -- does the benzene that gets into the bone marrow create a risk of leukemia through direct DNA damage?

A. Yes, it does.

Q. Does it increase the risk of leukemia through formation of reactive oxygen species?

A. That's one of the mechanisms.

Q. Does it create a risk of cancer through adduct formation?

A. Yes, that's another mechanism --

Q. And on the immune system side?

A. -- and direct DNA breaks.

Q. Okay. And on the immune system side does it also cause the immune system dysregulation which makes it less likely that the errors we've been talking about will be corrected or eliminated?

MR. TUBIN: Objection to form.

A. That is correct. It negatively affects the immune system.

BY MR. TELAN:

Q. And does TCE also negatively affect the immune system?

A. Yes.

MR. TUBIN: Objection. Form.

BY MR. TELAN:

Q. Does PCE negatively affect the immune system?

MR. TUBIN: Objection to form.

A. Yes.

MR. TELAN: I think after six hours we're finally not talking over each other.

BY MR. TELAN:

Q. The Cohn study that you were asked questions about, that was a study involving exposure to residents of New Jersey who drank and inhaled vapors from water that was contaminated with TCE and PCE --

MR. TUBIN: Objection to form.

BY MR. TELAN:

Q. -- is that correct?

A. That is correct.

Q. We'll talk about that in a bit more detail in just a few minutes.

The Yu study that we talked about, Yu study, today was a study that spoke about low-level exposures to benzene and the risk of leukemia; is that correct?

A. The risk of cancers in general including leukemia.

Q. I think you might have mentioned that that was an ecologic study. Was that, in fact, a

retrospective cohort?

A. So they used data from the UK Biobank. The UK Biobank is a prospective cohort study, but the assessment of exposure was done in a way we normally see in an ecological study when it's populational level data rather than an individual level data.

Q. But the study design was a prospective cohort study?

A. A prospective cohort study, yes.

Q. The Camp Lejeune Justice Act that you were asked about, the terminology "at least as likely as not," I want to focus on that just for a minute. We'll talk about it in a bit when we get into the documents.

But I think you had mentioned that that terminology was synonymous with the ATSDR's use of the term "equipoise" and above; is that correct?

A. Yes. That is correct.

MR. TUBIN: Objection to form.

BY MR. TELAN:

Q. Let me ask it in light of the objection, what is your understanding of the use of the

terminology "at least as likely as not" as considered by the ATSDR in its 2017 assessment of Camp Lejeune?

A. So I think they gave an example what that means. It's a different standard than what's normally used, which is "more likely than not."

And I think in -- what I understand, I think what was provided in the equipoise terminology, is that if the consensus is not reached whether -- let's say the weight of evidence cannot determine whether there is association or there is not association, that's equipoise.

And everything at this level and above would meet the standard set by Camp Lejeune Act and ATSDR.

Q. And is that the standard that you considered in this case?

A. Yes.

MR. TUBIN: Objection to form.

BY MR. TELAN:

Q. The studies that we've spoken about in, let's say, IARC when they use -- or when they formulate a monograph, do you know if they refer to a

standard that is different than the "at least as likely as not" standard?

A. My understanding of IARC is that it's "more likely than not."

Q. Okay. Let's go through, if we can, a few of the documents that you were asked about.

Exhibit 3 was the Godfrey deposition. I think you should have that there, if you'd go to that, please. And just so that I'm clear, if you could, would you confirm that this was a 142-page deposition that you gave on July 29th, 2024?

A. Yes.

Q. And what was your role in this case, the Godfrey case?

A. So I was asked to provide my opinion about the association between benzene and hematological malignancy. I think in this case was -- I think it was non-Hodgkin's lymphoma, if I recall correctly.

Q. Take a look -- I think it was multiple myeloma.

A. Multiple myeloma, yes. I apologize. Yes.

Q. That's okay. Were you given the

opportunity to read any more than just the page or two that you were directed to by the Department of Justice?

A. I don't believe so. I don't believe I reviewed this deposition and made corrections.

Q. Okay. Did you write a report before you gave this deposition?

A. I did write a report.

Q. And was your opinion in that report that benzene was, in fact, causative of multiple myeloma in Mr. Godfrey's case?

A. Yes.

Q. And so the -- one snippet at page -- I think it was 67 -- 64 and 65.

Do you see you were asked to begin reading, I believe, at page 64, line 23 down to page 65, line 3?

A. Yes.

Q. Would that have been an incorrect -- or an error on the part of the court reporter in taking that down, if, in fact, it is -- what is on the page is accurate that benzene is -- there isn't enough

evidence to suggest that benzene is a causal agent for hematologic malignancies?

A. No. That was clearly an error.

Q. And, in fact, if you go to page 49, if you look down at the page at lines 13 through 22, do you see there that you testified that it was a causative agent?

A. Yes.

Q. Okay. I'd like to go to your report, Exhibit 4. Let me ask you, generally speaking, before we get into that, you reviewed the 2017 ATSDR evidence at Camp Lejeune, did you not?

A. Yes, I did.

Q. Do you remember in the -- I'm sorry -- the 2024 cancer incidence study involving Camp Lejeune, not the 2017, the 2024.

A. The Bove?

Q. Yes, the Bove study.

A. Yes.

Q. Do you recall in that study an evaluation of the association between exposure to the water at Camp Lejeune and all myeloid malignancies being

1 looked at?
2 A. Yes.
3 Q. Do you recall approximately what the level
4 of association was for that?
5 A. I want to say it was close to 1.68. I
6 hope I remember that correctly.
7 Q. When we talk about the term "all myeloid
8 malignancies," does that include acute myeloid
9 leukemia?
10 A. Yes.
11 Q. Does it include chronic myeloid leukemia?
12 A. Yes.
13 Q. Does it include MDS?
14 A. Yes.
15 Q. Are there any other myeloid neoplasms that
16 that would include?
17 A. So there are a group of diseases called
18 myeloproliferative neoplasms that include
19 polycythemia vera -- I think it was mentioned among
20 the studies -- essential thrombocythemia, and primary
21 myelofibrosis. They're the main three.
22 Q. So in terms of whether or not benzene

would be causative of CML, would the 2024 cancer incidence study support an association between benzene and CML given the level of association that was reported?

MR. TUBIN: Objection to form.

A. Yes. I think so.

BY MR. TELAN:

Q. You were asked a question about the level of benzene that was found in your report. I don't remember the page exactly, but I think it mentioned 10 micrograms per liter.

Do you recall that question generally?

A. Yes.

Q. Where would you have gotten that information from?

A. So I compared the levels that were reported at Camp Lejeune or the levels in the water that were reported in Camp Lejeune.

So that's where I took the number from trying to compare the level of exposure that the person at Camp Lejeune was exposed to to put in the context of Camp Lejeune exposure.

Q. Would that have come from a review of the water modeling data?

A. Yes.

MR. TUBIN: Objection to form.

(Whereupon, Madam Reporter asked for clarification from Counsel due to simultaneously speaking.)

MR. TELAN: The objection will stand and I'll just ask the full question so that we don't get interrupted.

BY MR. TELAN:

Q. Would you have obtained that data from the Morris Maslia water modeling report?

A. Yes.

MR. TUBIN: And the objection still stands, right?

MR. TELAN: Yes.

BY MR. TELAN:

Q. Exhibit 5 was the 2014 mortality among Navy Marines and Navy personnel. You were asked a few questions about that.

I'd like for you, if you could, to turn to

Table 5 -- oh, and by the way before you do that, you do understand that the ATSDR is a government agency, right?

A. Yes.

Q. And that when Dr. Bove did his study he was an employee of the government agency ATSDR, right?

MR. TUBIN: Objection to form.

A. Well, I didn't know that he was an employee.

BY MR. TELAN:

Q. Okay.

A. But I understand it's a governmental agency.

Q. Okay. And do you have an understanding that the purpose and mission of that agency is to protect the public health?

MR. TUBIN: Objection to form.

A. Yes, that's my understanding.

BY MR. TELAN:

Q. If you'd go to Table 5 on this study, if you'd go to hematopoietic cancers.

A. Yes.

Q. And this is a mortality study. So I think you talked to us about mortality studies being relevant but not necessarily as strong as a cancer incidence study when you have incidence numbers; is that correct?

A. That is correct.

Q. But even if you look at Table 5, is the hazard ratio above 1?

A. It is.

Q. And if you go down to leukemias, is it 1.11?

A. It is.

Q. I think you were asked whether -- to agree whether or not there was statistical significance in those numbers.

Did you talk to us a bit about how you as a cancer biologist look at studies that have positive associations but perhaps lack statistical significance?

MR. TUBIN: Objection. Form.

A. Yes. I think what I -- like I mentioned

earlier, we have to take into account the point estimate, which is the hazard ratio and confidence interval. And I provide an example comparing to studies with a lower number of cases and a high number of cases and how confidence interval changes.

So when we evaluate the study that we know may not have enough cases, we look at the consistency in terms of the hazard ratio: Are they all in the same direction, whether the confidence intervals include 1 or not?

And this is one of the reasons why we perform meta-analysis to increase number of cases and be more precise in terms of confidence interval.

So that's one thing that we -- we took into account, and then we have to evaluate the data in the context of what we know about the biology of the disease and the agents and mechanism of action.

So I don't think that one piece of data and one study can either prove or disprove the association. I think we have to analyze in the context with other studies and what we know about the biology of the disease and agents.

BY MR. TELAN:

Q. To the extent that the Bove studies at times compared cancer incidence or mortality from the Camp Lejeune cohort to the general population, would the -- explain, if you would, how the healthy veteran effect would bias those results towards the null.

A. So the one thing that we discussed earlier was that the SMR was lower in Camp Lejeune and Camp Pendleton, and SMR was calculated based on comparing personnel at camps to the general population.

So the one possible explanation why SMR was lower is because it looked at the healthy population, and my impression is -- I haven't been in the military, but you have to be pretty healthy to join the Marines.

So you're kind of selecting for a young, healthy population with likely normal blood counts and everything else.

So when you compare this selected population of healthy young people to general population, the risk of cancer is likely to be lower just because you're selected -- it's a selection bias

in a way.

Q. What about the age of the cohort? In other words, I -- and I might be off on this, but I thought that the ATSDR cohort age may have been either 55 to 58, somewhere in that range.

Is AML typically -- is the onset of AML in the 65 range?

A. For both AML and MDS, yes.

Q. So if the Camp Lejeune cohort is younger than the average age of onset of cancer, would that also bias the results towards the null?

MR. TUBIN: Objection. Form.

A. Yes.

BY MR. TELAN:

Q. I meant to ask you the ATSDR in the 2017 analysis, evaluation of the water, did you happen to when you were reviewing that report look at pages 6 through 9 that spoke about how one might prove causality under the at least as likely as not standard?

MR. TUBIN: Objection to form.

A. Yes. They did provide examples of what it

meant to meet certain standards, which was more likely than not or at least as likely.

BY MR. TELAN:

Q. Do you recall there being several options to meet the standard under what they called equipoise and above?

MR. TUBIN: Objection to form.

A. So there was several, and one of them was that if epidemiological studies in humans are inconclusive, then we should look at animal and/or mechanistic studies.

BY MR. TELAN:

Q. And I think you told us before that as a cancer biologist mechanistic studies and animal studies are, in fact, how cancer is now being studied; is that correct?

MR. TUBIN: Objection to form.

A. That is correct. I think the main limitation of epidemiological studies is studying rare diseases. So they may be more informative in frequent cancers like lung cancer or colon cancer when we have enough cases to reach what they call

statistical significance.

If you look at studies with a very low number of cases, one case just by chance can change your hazard ratio from 1 to 2.

And as an example, if you have two cohorts and you have one case -- and a similar amount of participants in both cohorts -- and you have one case in one cohort and one case in the other cohort, your hazard ratio is 1.

If you changed that to one case and by chance you have two cases, the other cohort your hazard ratio is 2. So a very small number of cases may shift your hazard ratio significantly. That's why the confidence intervals are so wide.

So the reason why an epidemiological study may not be entirely informative in cancer is just because of the number of cases, and that's been a major limitation of epi study.

BY MR. TELAN:

Q. So in your opinion is it a reasonable approach to rely on mechanistic and animal studies to demonstrate causation even when epidemiological

1 studies are not sufficient?
2 MR. TUBIN: Objection. Form.
3 A. Yes. So in my field epidemiological
4 studies are meant to give us an idea whether
5 association exists, but mechanical studies and
6 biological studies are supposed to address the
7 question of whether there's causality between
8 exposure and outcome.
9 BY MR. TELAN:
10 Q. And I don't know if it was suggested. Are
11 you the only person at Johns Hopkins that believes
12 that?
13 MR. TUBIN: Objection. Form.
14 A. No. I don't think so. I've published
15 many papers and some papers were on epidemiological
16 studies. And I actually published the papers on UK
17 Biobank datasets, the one we talked about earlier.
18 And through my career I haven't been asked about
19 Bradford Hill criteria.
20 BY MR. TELAN:
21 Q. I was going to ask you about that.
22 A. But I've been asked if I suggest that

there is a causal effect -- I was asked to provide biological evidence behind that.

Q. So when you submit for publication to the major journals in hematology and oncology, do they ever ask you, well, Dr. Gondek, does your paper consider Bradford Hill framework?

A. Never.

Q. Do they ask you about biological plausibility?

A. Almost always.

Q. Why is that?

A. Because that's what addresses the question whether there is a causal effect or not, and I think the view on that has changed over the last 60 years significantly.

Q. When Bradford Hill came out in 1965 or thereabouts, did we even know mechanistically how chemicals caused cancer?

MR. TUBIN: Objection. Form.

A. We didn't know how cancer was caused, period. We knew cancer was this abnormal growth of tissue, but the structural DNA was discovered just a

few years earlier. There was no studies at that time linking DNA damage to cancer.

So the only thing that was available in the '60s and '70s was an epidemiological study. And if this is the only tool you have, you try to make an assumption based on certain viewpoints or criteria.

Now, over the last 60 years since we learned so much of cancer and we know it's a genetic defect, this way of evidence shifted significantly to biology. And that's true for most clinical trials that we do. We rely less on epi studies, more on biology and explanation of the mechanism.

BY MR. TELAN:

Q. Let me move on, if we could, to Exhibit 7, which is a supplemental table, I think, to one of the Bove studies. And incidentally, I think you were asked about this.

Do you recall now having seen some of the supplemental tables in the Bove studies?

A. I think it was available for my review, the supplemental studies or supplemental materials to the manuscript.

Q. In other words, when you were sent materials were --

A. The supplemental materials were there, I think.

Q. Okay. Let me ask you to look at table --

A. I'm sorry. Can you tell me what --

Q. It -- here. It says additional file 2, table --

A. Yeah, yeah, yeah.

Q. If you could go to page 3, cumulative exposure to benzene.

Do you see that?

A. Yes.

Q. Do you see that all of the hazard ratios are well above 1?

A. Yes.

Q. And do you see that there's statistical significance in the low exposure category?

A. Yes, for leukemia.

Q. And how would you interpret the 2.54 -- well, withdraw that question.

What is the significance of this

1 particular line as you see it from a hazard ratio
2 standpoint?
3 MR. TUBIN: Objection to form.
4 A. Can you repeat the question, please?
5 BY MR. TELAN:
6 Q. Sure. What significance do you ascribe to
7 the hazard ratios of cumulative benzene exposure and
8 leukemia?
9 MR. TUBIN: Objection. Form.
10 A. The ratio of 2.54?
11 BY MR. TELAN:
12 Q. Yeah. Actually, you can take it
13 individually or cumulatively across the low, medium,
14 and high.
15 A. So if you look at the hazard ratio,
16 they're all above 1. So there's this same
17 directionality of effect, meaning that there's
18 increased risk.
19 If you look at confidence intervals in
20 some levels of exposure like medium and high, they
21 include 1, but they're also wide and some they don't.
22 But what is also important beyond

1 confidence interval is whether the directionality is
2 consistent, and in this study it is above 1.
3 Q. Okay. You had explained earlier to some
4 degree how low exposures can cause cancers, whereas
5 high exposures may not. And I wanted to ask you:
6 Does that have to do with the notion that low
7 exposures damage but do not kill stem cells?
8 MR. TUBIN: Objection. Form.
9 A. So -- well, I think there is a level to it
10 because if you reach the certain level of exposure
11 that is lethal to the humans, obviously, you're not
12 going to see any cancers because you're just going to
13 kill the person, right?
14 What we know from biology of cancer is
15 that the more DNA damage can trigger cell death
16 because there is a critical threshold for DNA damage
17 when the cells cannot be fixed. It just overruns the
18 system and there is a mechanism in place to trigger
19 cell death.
20 So if you kill the cell and it gets DNA
21 damage, obviously, you're not going to get cancer
22 from this cell, right? Then we use it often

clinically for chemotherapy treatment. We want to reach the threshold when the DNA damage cannot be fixed and the cell has to die. That's how we kill leukemia.

If you look at the lower level of exposure for a prolonged period of time, if they increase the amount of damage to the level that's not critical and it's not fully fixed but the cell survives, these mutations will accumulate over time and the chances of the cell that stays alive to progress to leukemia is much higher.

Sometimes the higher level of exposure may actually kill the cell that's supposed to give cancer so you don't get cancer.

BY MR. TELAN:

Q. If we go to Exhibit 8, if we could, the mortality study of civilian employees -- if you could go to Table 5, I think you were asked about the confidence intervals of one or more of those compounds.

Do you recall that?

A. Yes.

Q. What is the significance of the hazard ratios for the high exposures, if you would?

A. So I think if you can compare the medium to high, the hazard ratio, and it seems like it's consistently elevated across all the agents and leukemias -- so, yes, the confidence intervals are wide and that's because of the number of cases.

But there is a consistent elevation of hazard ratio to -- up to 1.82, for example, for PCE.

Q. So where you might answer a question about statistical significance as saying that a particular result in this category may not be statistically significant given the confidence interval, is it biologically relevant to you with the understanding of benzene and leukemia?

MR. TUBIN: Objection to form.

A. It may be biologically relevant if -- like I mentioned earlier, if we see the same results in multiple studies with the same direction of increase of hazard ratio, that tells you something about biology, even though these individual studies may not be statistically significant.

1 And there are numerous examples of that.
2 When we look at individual studies, we see increased
3 hazard ratio. The studies are not, quote/unquote,
4 statistically significant because the confidence
5 intervals are wide.
6 But when we increase the numbers by doing
7 meta-analysis, then we see the results become
8 statistically significance just because we narrow the
9 confidence intervals.
10 BY MR. TELAN:
11 Q. Okay. If we can go to Exhibit 9, Table 4.
12 If you look at leukemias, again, looking at the
13 hazard ratios, are the hazard ratios both the
14 unadjusted and adjusted above 1?
15 A. Yeah, 1 is 1.1 and the other is 1.12.
16 Q. Okay. And if you go to AML, is it 1.2 in
17 the unadjusted and 1.11 in the adjusted?
18 A. Yes.
19 Q. And if you look at MDS, is it 1.96 and
20 2.26?
21 A. That is correct.
22 Q. What's the significance of those results?

A. Again, they all are going to the same direction showing the point estimate is higher but, again, wide confidence interval. But the same as other studies, the same directionality of the effect.

Q. If we go to Exhibit 10, the supplemental file, Exhibit -- table S2, if you could look at leukemias. If you look at the far right-hand column, risk ratio CL versus CP, do you see 1.13?

A. Yes.

Q. Does that indicate a positive association between exposure at Camp Lejeune and the mortality due to leukemias?

A. That would suggest that.

Q. Exhibit 12 -- let's skip ahead to that -- I think -- but I'm not sure -- this might have been the preprint. But I'd like for you to look at -- it's Bates stamp page -- there's no table number. It's kind of towards the back. It's Bates stamp page -- 163 are the last three digits.

If you go down all the way to the bottom at acute myeloid leukemia, do you see that low duration, medium duration, high duration?

A. Yes.
Q. What's the significance of that -- those values there on page 163?
A. 163.
Q. It's a -- the bottom line there's a .55 low duration, 1.52, 1.53.
Do you see that?
A. Oh, I see, yes. So if you look at the hazard ratio, it increases with a different level of duration.
Q. To the point where the medium duration and high duration are -- show an increase of more than 50 percent above the null value?
A. Yes.
Q. Let's go to Exhibit 14. This is the Yu study. This study was published when, Dr. Gondek?
A. Just less than two weeks ago.
Q. And when did you send this to us?
A. Actually, I saw the study yesterday.
Q. Is that when you provided it to us?
A. Yes.
Q. Did this study low-level benzene exposures

and a variety of cancers?

A. Yes.

MR. TUBIN: Objection to form.

BY MR. TELAN:

Q. My eyes aren't very good these days. So could you tell us -- is there a better table to look at?

A. Yes. There's a Figure 1. I think it's the third page --

Q. Is it table or --

A. That's the figure. It's the forest plot.

Q. Can you show me what you're looking at?

A. It's the third page from the end.

Q. Okay. Tell us, if you would, what that study revealed relative to exposures to benzene and leukemias.

MR. TUBIN: Objection to form.

A. So the study looked at ambient benzene exposure as determined by the atmospheric analysis from satellite and modeling and verified by on-site measurements and it showed a significant association with various cancers and one of the highest

significance for hematopoietic cancers with the hazard ratio of above 2 with a very tight confidence interval.

That was true for leukemia with the hazard ratio of 2.11 with confidence interval 1.89 to 2.36. The same was true for non-Hodgkin's lymphoma.

BY MR. TELAN:

Q. And what was it for non-Hodgkin's lymphoma?

A. It was 2.11 with the confidence interval of 1.92 to 2.3.

Q. Are you able to evaluate dose response? Was there any type of graph (sic) or table?

(Simultaneously speaking.)

A. So if you look at the next figure, when you look at the hazard ratio on the Y axis and benzene level on the X axis, you can see that there's no increased risk up to the level of 0.18 part per billion, and it goes up to a significant level above that threshold.

BY MR. TELAN:

Q. Is that a super linear curve or how would

you define that?

MR. TUBIN: Objection to form.

A. I would say it's a logger rhythmic. The super linear curve was defined by the levels around 1 part per million through a different study suggesting that the best correlation between incidence of cancer and the level is with the range up until 1 part per million.

If you cross this threshold, the curve flattens, so with the increased level of benzene exposure there's not necessarily proportional increase incidence of cancer. And that's related to metabolism of benzene, so some studies suggest that the system gets saturated at 1 part per million.

BY MR. TELAN:

Q. How does this study, the Yu study that we're looking at, change the thinking on the dose response relative to benzene and leukemia?

MR. TUBIN: Objection to form.

A. There are two messages. One is that what we kind of expected based on prior studies but we didn't have exact approval is that level of benzene

exposure way under 1 part per million were associated with increased risks of cancer, but we didn't quite know what the minimum threshold level was.

I think based on this study we have an idea of what this minimal threshold level is. And then this -- with ambient exposure level, an ambient exposure to benzene is way under 1 part per million.

It suggests that there is a very strong and significant association between the incidence of leukemia and the level of benzene exposure.

BY MR. TELAN:

Q. You were asked about the pliofilm study earlier. Is that a study you're familiar with?

A. Yes, I am.

Q. How do you become familiar with pliofilm?

A. Well, I reviewed the literature on benzene exposure and hematologic malignancies, and I know that the study was done and it was, I think, workers in Ohio. So I'm aware of this study.

Q. In terms of study power, is there a study that compares on a power standpoint with the Yu study?

A. I don't think there's any study that compares in terms of power to the Yu study. Like if you look at the Yu study, it's one prospective cohort of over half a million people that were followed for a long period of time with over 60,000 cases. I don't recall any other study coming close to this one.

Q. And how were the confidence intervals in the Yu study?

A. It was very -- they were very, very tight.

MR. TELAN: Okay. Let's go off record. Apparently, there's a message about us having to leave.

THE VIDEOGRAPHER: Off the record at 5:54.

(A break was taken.)

THE VIDEOGRAPHER: Okay. We're back on the record at 5:55.

BY MR. TELAN:

Q. You were asked a few questions about a confounder of obesity. How strong is obesity as a confounder relative to leukemia?

A. Not very strong.

Q. Is there any mechanistic data to support motive action on how obesity may cause leukemia?

A. No. There are many hypotheses, but there's nothing concrete.

Q. And did you say that the Yu study took into consideration all of the confounders?

A. They did adjust for most of the known potential confounders that have been associated with increased risk of cancer.

Q. Okay. Let's go to Exhibit 16, if we could, Exhibit 16, which is the Linet study that you were asked about. And if you look down at the bottom, do you see it says: "This work is written by U.S. Government employees and is in the public domain in the U.S.," at the very bottom?

A. Yes.

Q. Would you read into the record the first sentence in the paragraph beginning with -- this is on page 1 -- page 465 -- since 1979?

A. "Since 1979, an international consensus has developed that benzene exposure is causally related to leukemia, particularly acute myeloid

leukemia (AML), which was recently affirmed."

Q. Okay. If you'd continue -- yeah, continue on to the next sentence, if you wouldn't mind.

A. "In Chinese benzene workers followed during 1972 to 1987, we found increasing AML risks with increasing benzene exposure and stronger trends for AML combined with myelodysplastic syndromes (MDS)."

Q. And I think you were asked to read a specific sentence out of the abstract results starting -- and I'll read it for you. You can confirm that this was the sentence you were asked to read.

"There was little evidence of exposure response after at least ten years regardless of age at first exposure."

Do you recall that?

A. Yes.

Q. If you would go to page 469, under cumulative exposure 0 to 5 less than 5 parts per million years -- do you see that?

A. Yes.

Q. What is the relative risk ratio in Table 3 for MDS and AML cases, all ages?

A. So for 0 to 5 parts per million, 1.48; then between 5 and 40, 0.74; between 40 and 100, 2.52; and over 100, 1.84.

Q. And if you cut across the column horizontally from the 1.48, do you see there's a risk ratio of 1.12 and a risk ratio of 1.02?

A. Yes.

Q. All of those in the low exposure category, cumulative exposure are above the null value; is that correct?

A. That is correct.

Q. And the highest level is for the all ages category?

A. Yes, and there is also significant p-value for trend.

Q. If you go to Exhibit 19, the title of the document is Acute Myeloid and Chronic Lymphoid Leukaemias and Exposure to Low-Level Benzene Among Petroleum Workers.

This is the Rushton study that you cited

in your report. This is Rushton and Schnatter, I believe, and Tang and Glass.

Do you see that?

A. Yes.

Q. I think you were asked to read the conclusion sentence, which states: "Overall, this study does not persuasively demonstrate a risk between benzene and AML."

Do you recall being asked to read that?

A. Yes.

Q. Were you asked to look at the actual numbers in the odds ratios?

A. No.

Q. If you go to the odds ratios for acute myeloid leukemia, that's Table 1.

Do you see that?

A. Yes.

Q. Do you see you have an odds ratio for cumulative exposure between .348 to 2.93 of a 1.04?

A. Yes.

Q. And then above 2.93 parts per million years the risk ratio drops to 1.39; is that correct?

A. Yes. I see that.
Q. What is the significance of that?
A. Well, the point estimate would suggest that there's increased risk of leukemia that actually increases with increased level of exposure, but the confidence intervals are wide.
Q. And, again, is that because of the numbers?
A. Likely, yes.
Q. And if you go over to the next column, chronic lymphoid leukemia, CLL, are both of those hazard ratios also above the null value?
A. For cumulative exposure, yes.
Q. And the lower level of exposure of .348 parts per million years to 2.93 parts per million years shows a hazard ratio -- I'm sorry -- an odds ratio of 49 percent increase in association with exposure to benzene, correct?
MR. TUBIN: Objection to form.
A. Correct.
BY MR. TELAN:
Q. Let's go to 17, if you would. This is the

Glass study.

A. Yes.

Q. I just want you -- if you could, in the middle -- sort of middle bottom of the abstract, it starts a sentence with "Leukemia risk."

Can you read that into the record, please?

A. "Leukemia risk, however, was significantly increased for the subjects with greater than 16 parts per million years cumulative exposure, odds ratio of 51.9 with confidence intervals 5.6 to 477 or with greater than 0.8 parts per million intensity of highest exposed job."

Q. Does that demonstrate that for workers who had cumulative exposures of greater than 16 parts per million years they had a more than 5,100 percent likelihood of getting leukemia than those who were not exposed to that level?

MR. TUBIN: Objection to form.

A. That's correct.

BY MR. TELAN:

Q. I'd like to go to Exhibit 20. This is the Schnatter study of 2012. Incidentally, if you would

go back to the -- hold on one second. If you go -- go to the funding page. It's 2000 -- it's 1736. Take a look at that, if you would.

Do you see that this was an industry-funded study in part by the American Petroleum Institute?

MR. TUBIN: Objection to form.

A. Yes, I see that.

BY MR. TELAN:

Q. Do you know if Dr. Schnatter is an employee of ExxonMobil?

MR. TUBIN: Objection to form.

A. I didn't know that --

BY MR. TELAN:

Q. Okay.

A. -- but I see -- I see it now in correspondence.

Q. And even he says in the first sentence that, "Benzene is a well-known hematotoxin and leukemogen at a relatively high level of exposure."

Do you see that?

A. I see that.

Q. And one of the conclusions was that the -- at relatively low-level exposures to benzene petroleum distribution workers were associated with an increased risk of MDS; is that right?

A. I see that, yes.

Q. And that was the disease that you were talking about that has a separation from AML of two lymphocytes?

A. 20 percent blasts.

Q. 20 percent blasts. And is there a -- I think you may have mentioned this before, but then a certain percentage of MDS patients go on to develop AML?

MR. TUBIN: Objection to form.

A. Yes. It's around a third of the patients with MDS will end up progressing to AML.

BY MR. TELAN:

Q. If you wouldn't mind going to page 1734 --

A. Of the same study?

Q. It's Table 3, yeah.

A. I'm sorry. What page number?

Q. It's 1734, Table 3. Are you there yet?

A. Yes.
Q. Do you see that the risk ratio for AML is increased across every sector of worker?
A. Yes, I see that.
Q. And the lowest level of association is 1.27 in the column of peak versus referent?
A. Yes, I see that.
Q. The highest level is in the high exposure certainty category of peak versus referent.
Do you see that?
A. I see that.
Q. Under CLL do you see the refinery workers versus referent had a more than double increase in CLL?
A. I see that, and --
Q. Were you going to say something?
A. No.
Q. Okay. Exhibit 22, I know you were shown this. This is the IARC monographs. First of all, that's -- if you go two pages in this is a 2012 document.
Do you see that?

A. Yes.

Q. I'd like for you, if you could, to go to page 328 under the Mechanistic and other relevant data section.

You weren't asked to look at this particular section, were you?

A. Today?

Q. Yeah.

A. No.

Q. Okay. If you would, I'd like to direct you to the paragraph on the far right beginning with tetrachlorine -- sorry -- "Tetrachloroethylene has been associated."

Do you see that?

A. Yes.

Q. Would you read that into the record, please?

A. "Tetrachloroethylene has been associated with adverse health outcomes in the kidney, liver, and the immune and central nervous systems."

Q. And then if you would read one sentence down from that, if you wouldn't mind?

A. "There is evidence from toxicological studies in experimental animals that the kidney is a target organ for tetrachloroethylene and that genotoxic metabolites are formed in that organ."

Q. Can you skip a sentence and go to the sentence that starts with, "There is also strong evidence"?

A. "There is also strong evidence from toxicological studies in experimental animals that the liver is a target organ for tetrachloroethylene; with respect to liver" -- do you want me to read further?

Q. Sure. Yes, "with respect to liver" --

A. -- "with respect to liver carcinogenesis, it is likely that multiple mechanisms are operational in view of the evidence supporting genotoxic and non-genotoxic mechanisms."

Q. So even as of 2012, which is now 13 years ago, IARC had concluded that both the kidney and liver were target organs for tetrachloroethylene?

MR. TUBIN: Objection to form.

A. That is correct.

BY MR. TELAN:

Q. And if you look one sentence down from the one you were reading from, "The information that pertains" -- can you read that?

A. Yes. "The information that pertains to a mechanism of carcinogenesis related to the immune system is limited to studies of immunological and haematological toxicity."

Q. And have we discussed and have you in your report discussed how PCE causes leukemia?

MR. TUBIN: Objection to form.

A. I have.

BY MR. TELAN:

Q. And have you already discussed here today how immune system dysregulation creates increased risk for leukemia?

MR. TUBIN: Objection to form.

A. I have.

BY MR. TELAN:

Q. If we could go to -- this is the Danish study -- Stenehjem.

MR. TELAN: Marcus?

MR. TUBIN: Stenehjem, S-T-E-N-E-H-J-E-M, I think.

MR. BELL: There you go, 21.

BY MR. TELAN:

Q. So this is a 2015 Norwegian study, I believe.

Do you have that?

A. Yes.

Q. If you would go to the far right paragraph starting with, "The International Agency for Research on Cancer" -- can you read that into the record, please?

A. "The International Agency for Research on Cancer (IARC) has classified benzene as carcinogenic to humans, giving rise to acute myeloid leukemia, and possibly increasing the risk of acute lymphoblastic leukemia, chronic lymphocytic leukemia, multiple myeloma, and non-Hodgkin's lymphoma."

Q. So as of ten years ago IARC had already concluded that benzene caused AML?

MR. TUBIN: Objection to form.

A. That is correct.

BY MR. TELAN:

Q. And increase -- potentially increased the risk for other subtypes of leukemia, including ALL?

MR. TUBIN: Objection to form.

A. That is correct.

BY MR. TELAN:

Q. CLL --

A. Yes.

Q. -- which is really an NHL, right?

MR. TUBIN: Objection to form.

A. Yes. CLL is classified in the same categories as non-Hodgkin's lymphoma.

BY MR. TELAN:

Q. Okay. And do they also conclude that it potentially increased the risk for multiple myeloma?

A. That is correct.

Q. And NHL?

A. Yes.

Q. And that was ten years ago?

A. More than ten years ago. 2012, I believe.

Q. This is 2015, I think, it says.

A. IARC.

Q. Okay. Yeah. This study is -- you're right. You're right. I stand corrected.

Okay. If you would go to Table 4, page 1608, if you would. If you look -- I'm looking at the AML category there on the far right. If you look at the cumulative parts per million years -- do you see that?

A. I'm sorry. I'm in the wrong table.

Q. Table 4 to the far right.

A. Yes, I see that.

Q. What is the significance of that?

MR. TUBIN: Objection to form.

A. You can see that the hazard ratio increases with the level of exposure with nearly significant p-value for trend of 0.052.

BY MR. TELAN:

Q. Such that those that were exposed to .124 to .948 parts per million of benzene years had a 485 percent increase in leukemia -- AML leukemia over those not exposed; is that correct?

A. That is correct.

Q. Incidentally, I don't think we mentioned

the EPA here today.

Are you aware the EPA has actually banned TCE and PCE?

MR. TUBIN: Objection to form.

MR. TELAN: What's wrong with the form?

MR. TUBIN: Leading.

A. Am I aware?

BY MR. TELAN:

Q. Yes. What is your understanding as to whether or not the Environmental Protection Agency has taken any action as to TCE as a chemical?

A. Well, my understanding is that they recognized the carcinogenicity, they decided to limit the exposure of humans to these agents.

Q. Are you aware how often the EPA has instituted a ban like that on a chemical?

MR. TUBIN: Objection to form.

A. No.

BY MR. TELAN:

Q. Is it your understanding that the EPA instituted that ban in part because of its carcinogenicity?

MR. TUBIN: Objection to form.

A. I would assume so.

BY MR. TELAN:

Q. Are you aware that the EPA has also instituted a ban as to PCE?

A. Yes.

MR. TUBIN: Objection to form.

BY MR. TELAN:

Q. I think you were shown Exhibit 24, the April 1998 position on benzene.

Do you remember that?

A. Yes.

Q. This was the EPA's position on benzene 27 years ago, right?

MR. TUBIN: Objection to form.

A. I didn't look at the date, but yes.

BY MR. TELAN:

Q. Here, look at it.

A. Yes.

Q. Has the EPA's position on benzene changed, to your knowledge?

A. I don't remember exactly.

Q. Well, they instituted a complete ban just this past year.

A. I'm not aware of --

MR. TUBIN: Objection to form.

A. -- the EPA.

BY MR. TELAN:

Q. This is the -- do you have that?

A. For 1998?

Q. Yeah. Do you have that?

A. Let me see.

MR. TELAN: You're right. I stand corrected. It's getting late and I'm mixing up my chemicals.

BY MR. TELAN:

Q. So if we go to Exhibit 24, page xii --

A. I'm trying to find it. Yes, I got it.

Q. So can you confirm that this was the position or that this paper was published in April of 1998?

A. Yes.

Q. And you don't know whether the EPA has changed in any way its position on benzene?

MR. TUBIN: Objection to form.

A. I'm not aware of benzene being banned by EPA.

BY MR. TELAN:

Q. If you look at page xii, which for mine is the last page, under the second paragraph -- the second complete paragraph starting with "Data insufficiencies," could you read that into the record, please?

A. "Data insufficiencies in several areas are noted and research in these areas ultimately should provide a better understanding of how benzene causes cancer, particularly the mechanism of benzene-induced leukemia."

Q. If you go down a few more sentences under the -- starting with the line, "Continued basic research."

A. "Continued basic research in hematopoiesis and leukemia is critical for identifying the mechanism of leukemogenesis."

Q. Since 1998 has cancer science come to a better understanding of how benzene causes cancer,

1 particularly benzene-induced leukemia?
2 MR. TUBIN: Objection to form.
3 A. Yes.
4 BY MR. TELAN:
5 Q. Is that reflected in part by IARC's
6 position on benzene, if you know?
7 MR. TUBIN: Objection to form.
8 A. I -- they did take biological evidence
9 into consideration. So I presume that's correct.
10 BY MR. TELAN:
11 Q. If we go to Exhibit 26, this is the Martyn
12 Smith affidavit. This is the first time you've ever
13 seen this exhibit, right?
14 A. That is correct.
15 Q. Do you know the context of this affidavit,
16 if at all?
17 A. I don't.
18 Q. Has science on benzene and leukemia
19 changed from 1996 to the present?
20 A. Significantly.
21 MR. TUBIN: Objection to form.
22 BY MR. TELAN:

Q. And, in fact, you cited more recent publications from Dr. Smith on benzene and carcinogenicity?

A. I did.

Q. Dr. Goodman's report was handed to you, and I believe that's Exhibit 23.

Do you see that? Have you in your scientific work studying cancer, specifically blood cancers, have you ever heard of a company called Gradient?

A. No.

Q. If you go to Exhibit 27, I think you were handed this document about the Review of the Department of Veterans Affairs Presumption Decision Process.

Do you see that?

A. Yes.

Q. Would you look at page Roman numeral v?

A. Yes.

Q. Do you see that there is Gradient who appears as an advising principal to the National Academy of Sciences on this particular paper?

A. Sorry. What page? Yes, okay. I have it.

Q. So it appears that maybe one of Dr. Goodman's partners was involved in assisting the National Academies on the Review of the Department of Veterans Affairs Presumption Decision Process?

MR. TUBIN: Objection to form.

A. Can you repeat the question again?

BY MR. TELAN:

Q. Sure. Does it appear that -- if you look at Exhibit 23, we see Gradient here, right?

A. Yes.

Q. If you look at Exhibit 27, you see Gradient as being on the advising principal for the National Academy of Science?

A. I see it, yes.

Q. Let's see. Exhibit 26 -- oh, Exhibit 26 (sic) was a National Academy of Science 2009 contaminated water supplies at Camp Lejeune study.

Do you see that?

MR. TUBIN: That was 26?

MR. TELAN: I may have --

MR. TUBIN: I thought 26 was --

MR. TELAN: I'm sorry. Yeah, 27.

MR. BELL: This is 25.

MR. TELAN: Oh, 25 -- okay. So I might need to -- did I have the last one correct? 27 was the veterans affair one, right?

MR. BELL: Correct. You got it.

MR. TELAN: 25 is the 2009 Savitz. Okay. Yes, I've got it.

BY MR. TELAN:

Q. So turn to Exhibit 25, if you have it.

A. I have it.

Q. And I think you were asked some questions about this in particular, one of the authors, David Savitz.

Do you recall being asked about this study generally --

A. Yes.

Q. -- 2009? Were you aware that there was a -- one of the investigators on the panel who resigned from this panel?

A. I was not aware of that.

Q. Were you aware that there were two who

dissented?

A. I was not aware of that.

Q. And were you aware that there was a review officer on this panel who was an employee of Honeywell?

A. I was not aware of that.

Q. And would you have an understanding that Honeywell at this time would have had active contracts with the Department of Defense?

MR. TUBIN: Objection to form.

A. I'm not familiar with Honeywell.

BY MR. TELAN:

Q. Okay. Has science evolved since 2009 as it pertains to benzene and leukemia?

MR. TUBIN: Objection to form.

A. There were several manuscripts published since that investigated the role of benzene and leukemia.

BY MR. TELAN:

Q. And I can't recall if you had seen Dr. Savitz's deposition or not?

A. I don't think I reviewed that, no.

MR. TELAN: Okay. Give me just a few minutes, and I may be wrapping up.

BY MR. TELAN:

Q. Oh, you were asked earlier today about some of the studies you relied upon or reviewed or listed in your report and were asked about some of the limitations.

Do you recall that, generally?

A. Yes.

Q. Do all epidemiological studies have limitations?

A. Yes.

Q. I think that you mentioned this, but is it ethical to intentionally expose a person to a suspected or known carcinogen?

A. No.

MR. TELAN: That's all the questions I have. Thanks for your time.

THE WITNESS: Okay.

EXAMINATION

BY MR. TUBIN:

Q. I have a couple more questions briefly.

Dr. Gondek, you're not an immunologist, correct?
A. Correct.
Q. You don't have a degree in immunology, correct?
A. I thought you were referring to Ph.D., degree of immunology?
Q. Any degree in immunology.
A. No, I don't. I did publish several papers on immunology, but I don't consider myself an immunologist.
Q. Did you review any studies in this case on the healthy veteran effect or healthy soldier effect?
A. I don't recall.
Q. You don't recall?
A. Can you please specify what you mean by that?
Q. Have you reviewed any materials, any literature on the healthy veteran effect?
A. No. I don't think so.
MR. TUBIN: All right. That's all the questions I have.
THE WITNESS: Okay.

MR. TELAN: We'll read.

THE WITNESS: Okay.

THE VIDEOGRAPHER: Thank you very much. We are off the record at 6:29.

MR. TELAN: And I'd like a rough. Can we get it by tomorrow?

THE REPORTER: Yes, sir.

MR. TELAN: Okay. And we'll read and sign.

(Whereupon, at 6:29 p.m., the videotaped deposition of LUKASZ GONDEK, M.D., Ph.D. was concluded.)

* * * * *

United States of America :
: SS
State of Maryland :

CERTIFICATE

I, SHERRY L. BROOKS, a Certified LiveNote Reporter and Notary Public do hereby certify that, pursuant to notice, the deposition of LUKASZ GONDEK, M.D., PH.D., was duly taken at 10:04 a.m. before me. The said LUKASZ GONDEK, M.D., PH.D., was first duly sworn by me, according to law, to tell the truth, the whole truth, and nothing but the truth, and thereupon did testify as set forth in the above transcript of testimony. The testimony was taken down stenographically by me.

I do further certify that the above deposition is full, complete and a true record of all the te[illegible]ess.

Sherry L. Brooks

_____

SHERRY L. BROOKS
Certified LiveNote Reporter
Notary Public
July 9, 2027

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My Commission Expires: July 9, 2027

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

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

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