# Exhibit 605

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

IN RE: )
CAMP LEJEUNE WATER LITIGATION )
_____)

VIDEOTAPED DEPOSITION OF DAVID A. D'ALESSIO, M.D.

_____

PURSUANT TO NOTICE, the above-entitled deposition was taken on behalf of the Plaintiffs at the law offices of James Scott Farrin, 555 S. Mangum Street, Suite 800, Durham, North Carolina 27701, on Wednesday, June 25, 2025, at 8:58 a.m., before Sophie Brock, Registered Diplomate Reporter, Certified Realtime Reporter, and Notary Public in and for the State of North Carolina.

Job No. MDLG7434034

A P P E A R A N C E S

ON BEHALF OF THE PLAINTIFFS:

MCGOWAN, HOOD & FELDER, LLC
1539 Health Care Drive
Rock Hill, South Carolina 29732
Telephone: (803) 327-7800
By: CHAD A. MCGOWAN, ESQ.
cmcgowan@mcgowanhood.com
(In Person)

WEITZ & LUXENBERG P.C.
Fisher Building
3011 West Grand Blvd., 24th Floor
Detroit, Michigan 48202
Telephone: (313) 800-4170
By: DIANA GJONAJ, ESQ.
dgjonaj@weitzlux.com
(Via Zoom)

KELLER POSTMAN, LLC
111 Congress Avenue, Suite 500
Austin, Texas 78701
Telephone: (512) 690-0990
By: ZINA BASH, ESQ.
zina.bash@kellerpostman.com
(Via Zoom)

ON BEHALF OF THE DEFENDANT UNITED STATES OF AMERICA:

U.S. DEPARTMENT OF JUSTICE
1100 L Street, NW
Room 4054
Washington, D.C. 20005
Telephone: (202) 307-1024
By: WILLIAM V. KLOTZBUCHER, ESQ.
william.v.klotzbucher@usdoj.gov
(In Person)
ALLISON O'LEARY, ESQ.
allison.oleary@usdoj.gov
(In Person)

VIDEOGRAPHER:

Corey Parker

INDEX OF EXAMINATIONS



REPORTER'S NOTE: There were no exhibits marked.

P R O C E E D I N G S

THE VIDEOGRAPHER: Good morning. We are now on the record. Today's date is Wednesday, June 25th, 2025. The time is 8:58 a.m.

This is the case of the Camp Lejeune Water Litigation v. The United States of America.

The deponent is Dr. David D'Alessio.

Counsel, please introduce yourselves, after which our court reporter will swear in our witness.

MR. MCGOWAN: Chad McGowan for the plaintiff.

MR. KLOTZBUCHER: For the defense, William Klotzbucher and Allison O'Leary.

Whereupon,

DAVID A. D'ALESSIO, M.D.,

having first been duly sworn/affirmed,

was examined and testified as follows:

EXAMINATION BY COUNSEL FOR THE PLAINTIFFS

BY MR. MCGOWAN:

Q. Good morning, Doctor. My name is Chad McGowan. And your name is D'Alessio?

A. D'Alessio, yes.

Q. All right. So if I don't do that right, I apologize in advance --

A. It's okay.

Q. -- but I'm going to do my best.
Have you ever been deposed before?
A. No.
Q. Okay. So this is a chance -- I'm going to ask questions. Your job is to listen to the question and answer the question the best you can.
If you have any question about my question, it's unclear, please let me know. Otherwise it may look like we're communicating when we're really not. All right?
A. Mm-hmm.
Q. In front of you, hopefully, is a copy of your report that you made in this case?
A. Yeah.
Q. All right. Wonderful.
If you need a break at any time, for any reason, that is not a problem. I do not think this is going to go that long.
A. Okay.
Q. So we'll just get into it.
Do you have any --
A. No.
Q. -- concerns, problems, questions right now?
A. No, not at all.
Q. All right. Good. Let's get rolling.

Tell me how it is that you got involved in this case.

A. I was contacted by email. I get contacted for a lot of cases, just because I'm an endocrinologist and my name is on the website at Duke.

Q. Okay. So you get contacted for a lot of cases, but you've never been deposed before?

A. Right. I don't take most cases.

Q. I gotcha --

A. I mean, I just -- yeah, I don't have the time.

Q. Understood. So what made you want to get involved in this case versus other cases?

A. Just it -- I was thinking about that. It seemed interesting. I've worked in North Carolina for ten years. I do half my time at the veterans' hospital across the street from Duke. People talk about Camp Lejeune all the time. It's been in the newspaper. It's been in the medical journals. It just seemed interesting.

Q. Okay. And did you first get contacted by someone from the DoJ or some kind of service?

A. Yeah, DoJ.

Q. Okay. And did they share with you how they got your name?

A. No.
Q. Probably the Duke website?
A. Probably, yeah.
Q. Okay. And the -- I've noticed some of your bills, it's, like -- what -- $600 an hour, $3,500 half a day kind of thing; is that right?
A. Yeah.
Q. Tell me what your total billing has been so far.
A. I think I -- one invoice for $3,000 and one for $12,000.
Q. And then you have time that has been accrued but unbilled?
A. Yes.
Q. And how much is that?
A. About six or seven hours.
Q. All right. And does that money go to you, or does that go to a practice or --
A. That money goes to me.
Q. Okay. And half of it goes back to the government; right -- in taxes?
A. Yeah.
Q. Understood. All right, let's talk about litigation history.
Have you ever acted as an expert -- have you

written expert reports before?

A. No.

Q. All right. So this is literally your first time being involved in all this?

A. Yeah. Being involved at this level. So, in the past, I have -- somebody's -- when I was young and trying to make extra money, I would look at cases as an expert. Somebody would send me a case and say, "Is there a claim here?" or "Is there an endocrine problem here?" And I would give them a quick opinion. But it was never to the point of writing formal reports.

Q. And never to the point of giving testimony by deposition or at trial?

A. Never.

Q. Have you ever been sued before?

A. No.

Q. Tell me what you consider yourself an expert in. What field?

A. Endocrinology.

Q. Tell me what endocrinology is from a layperson's perspective.

A. Yeah. So endocrinology is, broadly, the diseases related to hormones. So what has fallen under our purview are diseases of the pituitary gland, the thyroid gland, the adrenal glands, the gonads, and

then metabolic problems like diabetes or cholesterol.

Q. Your day-to-day patient work is with mostly diabetic patients?

A. Well, 50 percent of the people that come to an endocrine clinic have diabetes. It's by far the most prevalent condition.

In my practice, I see allcomers, so I see a little bit of everything, but I would say my expertise is in diabetes.

Q. What does a normal workweek, outside of this sort of situation, look like for you?

A. So on Monday, I'm in -- I have a research lab -- we have lab meeting, and I meet with the postdoctoral fellows and the research people that day.

Tuesday morning, I do administrative work. Tuesday afternoon, I go to clinic.

Wednesday is a day that, again, I do mostly administrative work. I'm the division director. That's -- that afternoon is when I do writing, data analysis, that kind of stuff.

Thursday, I'm in clinic.

Friday morning, I'm in clinic. Friday afternoon, we have conferences, faculty meetings, grand rounds.

Q. How much of your time is spent teaching?

A. So I don't teach in the classroom anymore. In my two previous jobs I did a lot of classroom teaching. Now I mostly teach in the clinic. So that is -- all three of the clinic sessions I have in the week, there will be an endocrine fellow -- so somebody who's finished their residency who's training in endocrinology -- and they'll work with me, and I'll teach them there. I get students and medicine residents that come work with me in the clinic as well.

So I would say, you know, if I do 30 percent of my week as clinical time, probably half of that involves some teaching.

Q. And then the other 70 percent of your time is going to be research and administrative stuff?

A. Yeah.

Q. And you find that the more senior you get, the more administrative stuff there usually is?

A. Yeah.

Q. Okay.

A. Yeah. So it should be 30/30/30, and it's 30/30/60 because the administrative stuff never stops.

Q. Yeah, I understand.

Is endocrinology a sub-board of internal medicine?

A. Yes, it is.
Q. Do you consider yourself a toxicologist?
MR. KLOTZBUCHER: Objection. Form.
THE WITNESS: I don't.
BY MR. MCGOWAN:
Q. Okay. A cardiologist, oncologist, or an expert in chemotherapy?
MR. KLOTZBUCHER: Objection. Form.
You may answer.
THE WITNESS: No.
BY MR. MCGOWAN:
Q. All right. We've been provided an extensive list of materials considered, because that's what the rules require. Was there anything else that should have been on that list that just wasn't?
A. No. We were pretty complete. And the stuff that's on that list, I looked at damn near every page, but there was nothing else.
Q. Okay. I appreciate you not just putting "PubMed" on there.
A. Yeah.
Q. Narrowed it down a little.
All right. Let's talk about the timeline for this particular patient.
A. Yes.

Q. Let's lay out sort of what we know, because would you agree with me that there are gaps in the medical evidence?

A. Yes. I think I saw that in the report, too.

Q. Yeah. So let's just lay out some dates that we know or are pretty confident that we can rely upon; okay?

A. Mm-hmm.

Q. So when was the first date that you believe that we can rely upon that this patient had a diagnosis of diabetes?

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: Again, that data is secondary. Somewhere around 2008 or '9.

BY MR. MCGOWAN:

Q. And when was the NHL diagnosis?

A. January 1998.

Q. And when was the NHL treatment concluded, let's say?

A. Active treatment, I think, was done by July '98.

Q. And what was the actual treatment for the NHL that was provided to this patient?

A. I mean, I'd have to go back through the

records, but it seemed like a combination of chemotherapy and radiation therapy.

Q. Okay. Did you put in your report what the actual treatment was?

A. My report says they -- treated by Petersdorf Lindsley. They recommended chemotherapy, systemic radiation therapy, and stem cell transplant.

Q. Okay. So were you -- are you aware of the actual -- the chemotherapy regimen that was used in this case?

A. I mean, I know he had some version of CHOP before he went to Washington, and I can't remember what the drugs are in CHOP. Again, chemotherapy is not something I do a lot of.

I know he had -- that he had preparatory systemic radiation, because I know that's what you do for a stem cell transplant. And then the other thing he had was a couple of chemotherapeutics after his radiation.

Again, the details were pretty clear in the University of Washington records.

Q. Okay. So are you aware of, or an expert in, the effect of that treatment on kidney function and/or cardiac function?

MR. KLOTZBUCHER: Objection. Form.

You may answer.

THE WITNESS: I know that -- so one of the drugs, Adriamycin, can cause cardiac toxicity, but I don't know a lot about the other chemotherapies and their effect on renal function -- kidney function.

BY MR. MCGOWAN:

Q. When was kidney impairment first evident for this patient?

A. That was another one -- I mean, when he was referred to Dr. Leach -- and I think that was May of 2013 -- he had kidney failure, or he had chronic -- what looked like chronic kidney disease, as marked by his eGFR, his creatinine.

In the hospital, he had three days where his creatinine was elevated during his chemotherapy, and I think he got infected, but that corrected before he was discharged.

And other than that, there's -- that's part of the big gap. I didn't see a lot of tracking of his renal function.

Q. Did he have a creatinine of 1.4 in the early 2000s?

I have a note, and I'll read it to you just to save a little time --

A. Oh, yeah here it is. (As read):

"During an outpatient visit in April 2000, Mr. Keller was noted to have a normal creatinine; in July 2003, his creatinine was noted to be 1.4."

Q. Is that slightly above normal?

A. It depends on -- in some places, that's -- it goes to 1.5. But 1.4 is upper limit of normal.

Q. Do you have any conclusion as to whether that would be, basically, on the upswing in a bad way latterly?

MR. KLOTZBUCHER: Objection.

You can answer.

Form.

THE WITNESS: I mean, with a single value sitting out there -- I mean, the most common thing -- when did I say that was? July 2023. The most common reason for your creatinine to be up a little bit in July is dehydration. I'm going to play golf tonight at 6:00. I think, when I get off the golf course, my creatinine will be a few points higher because of dehydration.

Could be that -- the other common thing we see, when people's creatinine goes up, is oftentimes that's a harbinger that their blood sugar has been

high, because when your blood sugar goes up, you tend to get dehydrated as well.

So I can't tell whether a single value like this is part of a trend or if it's, you know, one of the normal fluctuations we see in kidney function, of which there are lots of causes.

BY MR. MCGOWAN:

Q. And we do have -- and it's in your report -- that in 2012, that there was a Stage 3 chronic kidney disease diagnosis?

A. Yes.

Q. Okay. How does one progress to Stage 3? Is it you go through -- I assume there's a 1 and a 2?

A. Yeah. I mean, so chronic kidney disease is defined by the eGFR, the glomerular filtration rate. This is calculated from the creatinine, which is a common blood test. And those are roughly equivalent. The eGFR includes age and body size, so it corrects for two things that can make the creatinine go.

Stage 1 is GFR less than 90; Stage 2, less than 60; and then -- so you're right, it progresses gradually -- in some cases it progresses gradually from Stage 1-2 to 3 and 3A, 3B, and 4.

Q. So do you have any reason to believe that in this case Mr. Keller's kidney disease did not progress

gradually?

MR. KLOTZBUCHER: Objection. Form. You can answer.

THE WITNESS: Yeah, I mean, again, in the absence of any information, I -- you know, any clinical information, it's really hard to tell.

BY MR. MCGOWAN:

Q. Well, do most people --

A. Yes --

Q. -- progress gradually?

A. -- most people, it would be a gradual progression.

Q. And is that over years?

A. Yes.

Q. And is there any reason to suspect that Mr. Keller would not be in the majority group for that metric?

A. No.

Q. Okay. So is it fair to say that it's more likely than not that Mr. Keller had a gradual progression to Stage 3 chronic kidney disease that was noted as of 2012?

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: Okay.

Yes. I think it's likely that he had a gradual progression.

BY MR. MCGOWAN:

Q. Okay. Are you -- help explain this to me. If you have -- I mean, diabetes is known to cause kidney disease; is that right?

A. It's the most common cause of kidney failure in the United States. It would be on the top of anybody's list of the causes of chronic kidney disease.

Q. How does it do that?

A. A lot of it's related to high blood sugar, hyperglycemia; right? Which sort of -- I'll make -- it floods the kidneys with glucose, makes them work extra hard, starts to cause damage in what is a pretty delicate network of cells; and over time, these cells start to become damaged, and you have progression.

So the steady progression of CKD has been worked up for diabetes better than most conditions.

Q. So what I hear you saying is that, basically, the high blood sugar, and what it causes, causes a scarring of those little delicate cells and a deposition of scar tissue --

A. Yeah.

Q. -- which then makes the kidney less able to

do its job?

A. Yes.

Q. Okay.

A. Yes.

Q. Now, is this a -- does the kidney have a certain reserve, in that it can take some amount of damage before you start having creatinine rises?

A. There's some reserve, yeah. I mean, we know that because you can donate a kidney to somebody and one kidney left over is usually enough to compensate.

Q. So you can have progressive kidney damage, from whatever source, that takes a while for it to be evident in lab work?

A. Damage is a -- so when we measure creatinine clearance, or eGFR, and use that to tag the progression through chronic kidney disease, that's a functional measure; that's telling us how the kidney is functioning.

For -- in the field of nephrology, damage is something that you would see on a biopsy, on an image, or with -- attached to specific biomarkers for particular diseases. So damage and function are two different things.

We generally follow people with function because doing biopsies is invasive and painful. And

so, you know, we make a lot of assumptions. Now, the assumptions are based on old biopsy data. But to say -- you know, damage is different than chronic kidney disease and loss of function, I think. Just have to be clear that those are specific terms.

Q. Right. Well, the damage would presumably cause a function problem at some point?

A. Not necessarily. I mean, we see patients all the time that they get a -- well, again, we don't have biopsies to tell. But we see people in the hospital all the time get sick, have their blood pressure fall, get a noxious drug, et cetera, and have their function impaired; and then when they come back to the clinic, their creatinine is fine; the next year, their creatinine is fine, et cetera.

So one episode of a decreased function or one episode of a challenge to the function doesn't necessarily mean that you're going to be on the road to progressive kidney failure.

Q. I appreciate that, and I'm not doing a great job, so I apologize.

The question is, can you be sustaining kidney damage initially without it reflecting in the function measures?

Like, can your kidney absorb some amount of

injury before the functions start showing impaired through the normal lab work that you would make these diagnoses through?

A. Again, that's -- you know, that's a really difficult -- I mean, that's a question that's really hard to answer because I don't think people have looked -- I mean, the reason people look for kidney damage is they see abnormal kidney function. So there's just not a lot of data on saying, you know, we should biopsy a bunch of people that got this noxious drug -- even though they didn't get kidney functional abnormalities, let's biopsy them and see if there's any damage. So there's just not a deep dataset in that regard. Right?

The reason you look for damage and do biopsies is you see abnormality in function.

Q. I understand.

Tell me how it is that you ruled out any damage caused to this man's kidneys by the chemo or the treatment for the NHL. How did you rule that out, in this particular case, as contributing to the problem?

MR. KLOTZBUCHER: Objection. Form.

You may answer.

THE WITNESS: So "rule in," "rule out"

is a general term.

So this guy had a transient change in his kidney function, as many cancer patients do. He rebounded really quickly, and for three or four years after his treatment for stem cell transplantation, he had normal kidney function. You know, based on that, he did not -- in my view, he didn't have a lot of residual damage. Now, without a biopsy, I couldn't tell you for sure. But subsequent to that, he had lots and lots of common kidney challenges.

And so, you know, for me to speculate that there was a subclinical kidney insult during his stem cell therapy that didn't manifest past his discharge from hospital made it seem really unlikely in the face of all these other things that are listed in my report that commonly cause chronic kidney disease.

And so did I rule it out? Maybe not. Was it at the bottom of my differential? The absolute bottom in terms of likelihood? Yeah.

BY MR. MCGOWAN:

Q. Okay. So how did you evaluate its contributing factor? Because you can have both; right? You can certainly have damage to your kidney from stem cell treatments, chemo, whatever, and damage from high blood sugar and diabetes; right? Those two

can coexist?

A. Oh, they could coexist --

MR. KLOTZBUCHER: Objection. Form. Excuse me, Doctor.

THE WITNESS: Sorry.

MR. KLOTZBUCHER: You may answer.

THE WITNESS: Okay.

Yeah, I think they can coexist. But again, he had -- we have to -- I don't know when he developed diabetes, but he had normal kidney function for the first few years after his stem cell transplant.

BY MR. MCGOWAN:

Q. Well, he also had normal kidney function for the first few years after his diabetes?

A. We don't know that. We don't know when he got diabetes. And we don't know what his kidney function was after 2003 until 2012 -- or at least I didn't see any evidence to support that.

Q. Okay. So you did not rule out the treatment for NHL as a contributing -- potential contributing cause to that, in addition to the other things you identified; is that right?

A. I guess that's -- I mean, I just thought it was very unlikely.

Q. Well, I appreciate that, but it just seems to

me that it can be both. I mean, tell me how it -- tell me why it is you think it's not both.

A. I guess --

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: I guess it's -- if you have two things -- or four things, and three things are well documented and high likelihood to cause kidney failure, to throw in something that's really unlikely to add to them just doesn't -- you know, I mean, I don't know -- he could have had kidney damage anytime up to his transplant, and I didn't consider that in this at all.

40 percent of people that have type 2 diabetes are going to get some kind of kidney dysfunction from it.

People that take a lot of nonsteroidals for gout and other arthritis problems have an increased risk of heart -- of kidney failure as well.

People with gout and high uric acid levels have some risk for kidney failure.

So these are all common explanatory causes for a guy that develops Stage 3 kidney disease over time; and to sort of decide how many cells you can put on the top of a pin about his chemotherapy just, you

know, doesn't seem that relevant to me.
BY MR. MCGOWAN:
Q. Well, you said it was unlikely that his chemo and the treatment for the NHL -- and I mean this with all due respect -- how do you know how unlikely it is if you don't even know what the particular treatment was and the effect of that treatment on kidney cells is?
MR. KLOTZBUCHER: Objection. Form. You may answer.
THE WITNESS: Again, part of it is the -- again, he -- whether he had any damage to the kidney with his chemotherapy, it didn't manifest as a significant functional deficit for some time; so, you know, for the most part, that suggests that any damage he incurred was not very serious.
And whether I know the particulars of rates of kidney failure with whatever drugs he got sort of doesn't matter in that case. It wasn't enough to cause a measurable effect on his kidney function.
And so, therefore, to extend that to causation for his Stage 3 kidney disease is just a little bit fanciful, I think.
BY MR. MCGOWAN:
Q. Okay. Well, let's get to the bottom of that.

I'm talking about kidney damage, and you're talking about the measures -- the creatinine and the function -- the function of the kidney?

A. Yeah.

Q. And we have discussed that you can have damage to the kidney but preserved function?

A. Even that's -- even that's murky. I mean, damage is, again, defined by pathology on biopsy or an abnormality on a CAT scan or an ultrasound or a particular biomarker; but almost all of those things are tied to abnormal function. That is, we just don't look for damage in people with normal kidney function. And so it's tying -- tying "damage" to function is an area where there's just not a lot of information.

Q. Well, let's use the situation that you mentioned where you're a kidney donor, and you have two kidneys, and one of them is removed and surgically implanted in another patient.

A. Yeah.

Q. Frequently, the donor, the person who has the one kidney now, has normal kidney markers, normal function; right?

A. Yeah.

Q. So does that not tell us that you can at least lose a whole kidney without it affecting your

lab work, your function?

MR. KLOTZBUCHER: Objection. Form.

You may answer.

THE WITNESS: It tells you --

MR. MCGOWAN: Hang on, Doctor.

What's wrong with the form of that question? You've made these objections, I think, 12 or 15 times, and I'm -- what's wrong with the form of the question, please?

MR. KLOTZBUCHER: It's a form objection. That's all I'm allowed to offer.

MR. MCGOWAN: Well, the form objection is supposed to allow me to correct an improperly formed question so that it's admissible at trial down the road; and if you don't have an actual form objection, then I would ask you to not make that objection because it's just disruptive.

THE WITNESS: Can you repeat the question about the transplant?

BY MR. MCGOWAN:

Q. Yeah. So if you lose one kidney -- let's take that analogy to you can have normal kidney function and lose a whole kidney; right?

A. Yeah.

Q. All right. So does that still -- I mean, can

we extend that to say that you can have -- and we'll just for lay purposes call it "dead kidney cells" here -- and I know they're glomeruli and all that good stuff, but --

A. Yeah.

Q. -- that are not sufficient -- they're dead, they're gone, they're scarred up, nonfunctional -- but they do not rise to the level of causing kidney function impairment noticeable by lab at that time?

MR. KLOTZBUCHER: Objection. Form.

You may answer.

THE WITNESS: I think -- I mean, I think you're -- again, over the course -- I mean, I think you're conflating the -- well, let me just back up.

We probably have damage that goes on to our kidneys day in, day out, and these dead cells you referred to and scars and stuff happen. That's why our kidney function declines from about age 20 to the rest of our life. So that just happens.

Is that damage? Doesn't usually show you up as a functional abnormality.

Does it have consequences? Not for the vast majority of people.

So, you know, to say that -- to speculate

that there's some theoretical damage that happens at a particular point in time but is not enough to show any functional consequence, you know, I mean, just -- possible? Maybe. But, I mean, there's just not a lot of evidence that that -- that you could hang your hat on any one time point for that.

I mean, I'm sorry to be -- to not be more precise in the answer, but it's a really hypothetical question.

BY MR. MCGOWAN:

Q. Well, I'm trying to not make it that way, but that's fine.

Tell me what chemicals Mr. Keller was exposed to at Camp Lejeune.

MR. KLOTZBUCHER: Objection. Foundation.

You may answer.

THE WITNESS: I mean, I know -- I mean, I read the report once. So benzene. Maybe trichloroethylene. I mean, they're in there; we could find them.

BY MR. MCGOWAN:

Q. TCE, PCE, and benzene: does that ring a bell?

A. Yeah, those sound -- those ring a bell.

Q. Okay. Can you tell me what effect those

chemicals have on kidney function, or kidney cells?
A. Yeah, not my area of expertise, and I can't -- as I said, I'm not a toxicologist.
Q. Okay. So what did you do to rule out the contaminants in the water as affecting his kidneys?
MR. KLOTZBUCHER: Objection. Foundation.
You may answer.
THE WITNESS: The first records I had of his kidney function was that it was absolutely normal; so, therefore, I concluded that he hadn't had significant damage to interfere with his kidney function.
BY MR. MCGOWAN:
Q. All right. Do you know how those chemicals work over time and latency periods and all that kind of stuff?
A. I don't. I think it's known, but I -- that's not an area I'm familiar with.
Q. And you know his exposure levels?
A. I know that he had some. I've seen in the other expert reports that you can make estimates of those and calculations of exposure and stuff, but that's, again, not my area of expertise.
Q. Do you agree with me that those chemicals are

known to cause kidney damage?

MR. KLOTZBUCHER: Objection. Foundation.

THE WITNESS: I don't know.

BY MR. MCGOWAN:

Q. Okay. So if you don't know the effect of those chemicals and his dosing and latency periods and all of that, and if you don't know the chemotherapy regimen and the stem cell and the effect of the kidneys and all of that, how is it that you can exclude those two as contributors to the problem? Or can you not? You're just saying that diabetes was the most prominent cause?

MR. KLOTZBUCHER: Objection. Form.

BY MR. MCGOWAN:

Q. See the question I'm asking?

A. Yeah. I mean, what you're asking is, if you have theoretical risks to kidney function, but that they don't show up in clinical measurements, is it possible that they were still part of the problem? And I can't answer that.

Q. Just because --

A. Because we don't -- there's not enough information, both from this patient and from the literature, to support that. Anything I said would be

purely speculative.

Q. What exactly did the DoJ ask you to do in this case?

A. They asked me to review the files and give an opinion on this man's clinical course.

Q. Okay. So --

A. So my expertise in diabetes, so that's what I focused on.

Q. Okay. But the specific question that you set out to answer was what?

A. What was the likely cause of the kidney failure that manifests as dialysis in 2024?

Q. Okay. So literally the question that was posed to you was: What was the likely cause of the kidney failure in Mr. Keller?

A. It wasn't posed in a specific way, but the notion is that was what case was about, and so that's what I focused on.

Q. All right. I need to -- if we need to take a moment to think, that's fine -- look at something -- but I need to know the specific question that you are answering in your report.

A. The -- so I can tell you the specific question that I addressed in my report, but it wasn't given to me by the Department of Justice. It was my

interpretation of this is what this case is about, so that's what I focused on. And what I focused on is what's the likely cause of kidney failure -- of end-stage renal disease in Mr. Keller.

Q. You did not address, and do not now address, whether it is as likely as not that the water contamination was a contributing cause to his kidney failure; is that right?

A. That's right.

Q. And you did not address the question as likely as not whether the treatment for the NHL was a contributing cause of his kidney failure; right?

MR. KLOTZBUCHER: Objection. Foundation.

You may answer.

THE WITNESS: Yeah, I didn't think I could answer either of those, either based on my experience or my literature review.

BY MR. MCGOWAN:

Q. Was it ever shared to you the different standard of proof in this particular case?

A. I'm not sure I understand.

MR. KLOTZBUCHER: Objection. Foundation.

BY MR. MCGOWAN:

Q. Well, you used the term "likely cause." And "likely" means more than 50 percent; is that fair?

A. Yeah.

Q. Is that the way you used it in this case? Because I know medical folks, unless -- and I'm generalizing -- but in general, that if it's not 95 percent plus, a lot of medical folks are not willing to say something is a likely cause. Versus us legal people who "more likely than not" is 50 percent plus a little. Right?

And so when you're using the term "likely cause," what level are you at when you're using it in your report? The medical side, which is more, or the legal side, which is half plus a little?

A. The medical side.

Q. Okay. So how sure is the medical side of "likely cause"? And it doesn't have to be a specific number. Is it greater than 80 percent? 90 percent? Where would we need to fall?

A. Yeah, so this man had risk factors for chronic -- that is the -- all the necessary risk factors are there to explain his ultimate outcome based on, you know, what we have medical records for: so history of diabetes, his variable history of

high blood pressure, obesity, hypercholesterolemia, nonsteroidal painkillers, and hyperuricemia. Those are all well-established common causes of kidney disease.

And the time course is really important here because, you know, in general, people who have a noxious insult, whether it be from a hospitalization or other things, tend to manifest the insult right away. And this guy, it looks like he developed his kidney disease gradually, as we see with diabetes, hypertension, obesity, all of that stuff.

So that was -- I just -- I thought this looked like a fairly straightforward case of diabetic nephropathy in a man who had other risk factors. And I thought the -- rather than saying -- you know, moving from 50 percent to 95 percent, I thought, from 100 percent down, how much would I give a contribution to his treatment for chemotherapy, and it's just infinitesimal.

Q. And in that conclusion, was the infinitesimalness -- that's probably a new word --

A. Yeah, good one.

Q. -- is not based on knowing the precision of the chemotherapy and the stem cell and all of that, and its effect on kidney function and latency, nor the

chemicals to which he was exposed and the latency for that?

A. Yeah, I don't know the -- I don't know anything about the chemicals.

The latency for kidney failure after stem cell transplant or lots of aggressive cancer treatments is a year. You see the abnormality right away, like I said. That's a short latency. And that had a lot to do with my interpretation, the fact that this guy got out of the hospital, got away from his stem cell therapy, maintained a normal creatinine for several years.

So I think, in general, gradual kidney failure is what you see with the kinds of problems he had; whereas kidney failure related to, you know, intensive medical therapy, like you'd get with a stem cell transplant, tends to manifest right away.

So "latency" is a good term that -- your term -- the latency here matters.

Q. Okay. So how much experience do you have in evaluating the kidney function or kidney damage -- either one -- in patients that have undergone NHL treatment or who have been exposed to TCE, PCE, and benzene?

A. Very little. None.

Q. Okay. So I'm still struggling with the -- you can have -- and this is hypothetical, and this is part of the fun of being a trial lawyer, I've found, is that we get to learn a lot of things from educated people and then go about our business and forget about it later -- is that you can -- chemicals, or whatever it is, an insult, can damage the kidney, and you can have kidney-damaged dead cells and yet it will not be evident on lab work.

The penultimate example would be you have donated a kidney, you have one less kidney, and yet your lab work is completely normal. Right? You can have dead kidney cells that do not yet rise to the level -- or cause of functional impairment?

MR. KLOTZBUCHER: Objection. Form.

You may answer.

BY MR. MCGOWAN:

Q. That's true; right?

A. That's theoretical.

Q. Yeah, but there's nothing wrong with the -- I mean, that's a true statement; right?

A. Yeah, I mean, I agree about the kidney transplant. The -- you know, the damage -- the supposed damage that's not -- that's there but not able to be seen on function, I'd have to see some

evidence that showed that that was true. Yeah.

Q. Okay. So in your view, any damage to kidney cells should result in function impairment?

A. I just think we don't know a lot about damage in people with normal function because we don't study them very much. We don't do kidney biopsies on them and say, "There was damage, but there was normal function."

Q. Well, is it your view, then, that any damage to the kidney will cause functional impairment?

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: Again, that's the flip side of the same question we can't answer. That is, we don't have a lot of data on normal kidneys -- or damaged kidneys or normal kidneys and function because we don't biopsy those people; we don't look for abnormalities.

BY MR. MCGOWAN:

Q. All right. So do you hold the belief, one way or the other, as to whether you can have damaged kidney cells, glomeruli --

A. Yeah.

Q. -- that does not give rise, at that point, to functional impairment?

A. Let me see if I can say this a different way. I -- regardless of how many damaged cells this guy had in 2001, do I think that that damage contributed to his kidney disease? I do not.

Q. Tell me the precise medical and factual basis for that opinion, please.

A. Yeah. So that opinion is just that any -- he was able to sustain normal kidney function for three years after his stem cell transplant, and there's just not a lot of evidence that had he had nothing else -- that he not had gout, that he not had diabetes, that he not had hypertension -- that this would lead to eventual end-stage renal disease.

Q. Okay. Well, tell me -- 40 percent of type 2 diabetics have impaired renal function?

A. Yeah. Somewhere between 30 and 40 percent.

Q. So if you're counseling a patient with type 2 diabetes, you say -- legally -- "It is less likely than not that you're going to get kidney disease" --

MR. KLOTZBUCHER: Objection --

BY MR. MCGOWAN:

Q. -- because it's 30 to 40 percent; right?

MR. KLOTZBUCHER: Foundation. You may answer.

THE WITNESS: Yeah, so what I tell

people is -- when I see them for the first time -- is, "Our goal here is to try and prevent you from having kidney disease, and we don't know exactly what the various risks are, but we know that if we treat your blood pressure and we treat your glucose, it's more likely -- you'll have a better likelihood of not having kidney disease."

BY MR. MCGOWAN:

Q. Did Mr. Keller have uncontrolled hypertension?

A. He had several episodes of elevated blood sugar -- blood pressures, at least one up to 99 and -- but it wasn't a prominent part of his treatment history. At least Dr. Leach didn't talk about it very much.

Q. Okay. So did you, for the purposes of your work in this case, assume that he had uncontrolled hypertension?

A. No.

Q. Did you credit hypertension as being a contributing cause of the kidney failure?

A. I did not.

MR. KLOTZBUCHER: Objection.

BY MR. MCGOWAN:

Q. The way that diabetes -- uncontrolled

diabetes causes kidney failure more often than controlled diabetes?

A. Yeah, I mean, what I would say is that there are some factors that we can impact that will decrease your risk of kidney failure, and blood sugar is -- treating blood sugar is one of them.

If you have bad genetics -- and we know that susceptibility to diabetic complications, to a certain extent, is bred in the bone; I mean, some people are more susceptible than others -- that's something we can't affect. So we treat the things we can affect.

But uncontrolled blood sugar is definitely a risk for progression to kidney disease.

Q. Did Mr. Keller have genetic predisposition?

A. Don't know.

Q. Is it accepted that the lower the A1c, chronically over time, the less likely you are to get kidney disease? Do those track?

A. Yeah. No, those are -- that's been done in clinical trials, where one group gets usual care and one group gets more aggressive care, and the A1c in the usual care is 9 percent, and the A1c in the intensive group is 7 percent, and the group with 7 percent, over five years, has half the kidney disease as the other group.

Q. If a diabetic is controlled -- their A1c is controlled to normal or prediabetic levels, do they show a rise in kidney disease frequency?

A. That's a good question. We don't know. We haven't had drugs yet that would do that -- that would absolutely normalize blood sugar. So even in these clinical trials, the intensively treated group just got to an A1c of 7, where an A1c of less than 5.8 is considered normal.

So we don't have great information even that 6.5 is better than 7, or 6 is better than 6.5. But we know that 7 is better than 9, and it's been done a couple of times. And so we're pretty confident that high blood sugar is worse.

Q. All right. So 7 -- how much better is 7 than 9 on -- for preventing or stemming kidney disease?

A. 50 percent better. But again, it's a finite time period: it's five years. And that's actually short in the development -- you know, that's kind of what it takes to see diabetic kidney disease. But a lot of people play out over longer periods of time.

Q. So there's not science to support it yet, but the theory would be that lower -- normal -- if you get down to normal, hopefully it would be a normal risk of kidney disease, which --

(Over-speaking.)

A. Yeah. No, I think that's what every -- that's the assumption. And most of the data would suggest that diabetic -- that the common diabetic complications -- eye disease and kidney disease -- the lower, the better.

Now, what we really know is that if your A1c is 12, then your rates are really high, and that you get more bang for your buck going from 12 to 7 than from 7 down. So it's a kind of a -- it's not a linear curve.

Q. Okay. And it's the -- then it's the level of sugar in the blood that causes the inflammation, or whatever it is --

(Over-speaking.)

A. Well, I mean, that's the association. And, you know, we know that glucose is filtered by the glomeruli, and we know that it goes into the kidney tubules, and we know that over time the glomeruli and the kidney tubules are damaged. And in just a wide variety of models from cells to animals to humans, you can show that having hyperglycemia affects kidney function in whatever measurement you're making.

So hyperglycemia is an insult to kidney function.

Q. So high blood sugar is an insult to your kidney, and the higher, the more insulting it is?

A. Yes.

Q. All right. So list for me -- and use your report if you need to -- what his blood sugar levels were up until the time of his diagnosis of Stage 3, which I believe was 2012.

A. Yeah, so I -- I have -- so he had -- the only blood sugars I found were during his hospitalization for bronchitis in 1999, and he had mildly elevated blood sugars in the hospital there.

Q. Was he also on steroids at that time?

A. He was.

Q. Can prednisone cause a transient rise in blood sugar?

A. Oh, yeah.

Q. Do you attribute that to -- that rise at that time to that?

A. Well, what I always use it as is as a hint that somebody's prone to diabetes. That is, most people don't get hyperglycemic on steroids, but if you do get hyperglycemic on steroids, that's -- it's kind of a prediabetic challenge. And people who are prone to diabetes are more likely to get high.

So I -- when I see a patient like that in

the hospital, I always worry that -- I ask him "Has your family got diabetes?" Oftentimes it does. So --

Q. And you might mention it to him and say, "You want to be looking out for that in the future"?

A. Yes.

Q. Okay. So what was his blood sugar level at that time?

A. He got up to 186.

Q. Was that a fasting sugar?

A. I don't know. It was a random sugar.

Q. And so what's a high number for a random sugar?

A. Well, 186 -- if we do a glucose tolerance test on you -- that is, give you 75 grams of glucose to drink -- normal is considered less than 140.

So, you know, a random blood sugar over 200, on two occasions, is diabetes. But somewhere in between there, we would say you're glucose intolerant.

Q. Does that matter? Like, timing of -- between the time you ate lunch to --

(Over-speaking.)

A. Oh, yeah, I mean, there's -- yeah. Formal -- formally, you'd do it with a -- you'd do a two- -- you'd give the glucose and you measure two hours later, and that's how you define normal glucose

tolerance.

Q. Okay. I think pregnant women sometimes go through that?

A. More than anybody else right now.

Q. I gotcha. Okay.

So but 186 random: are you telling me that's an elevated sugar or that might be an elevated sugar?

A. It might be an elevated sugar. That would be one -- I mean, just as you said, this would be one where I would say to the patient, "That's not a diabetic blood sugar, but that's higher than most guys, and we should watch that going forward."

Q. Okay. Unless you discovered that the lab was drawn 20 minutes after he had lunch?

A. No.

Q. No? Doesn't matter?

A. No, not at all. I mean, most of us that don't have diabetes never get a blood sugar over 140.

Q. All right. So tell me -- that was in 1999 --

(Over-speaking.)

A. Yeah. And then, again, I was -- the next blood sugars I see -- (as read):

"Dr. Leach noted a diagnosis of type 2 diabetes dating from 2009, but there are no medical

records... There is a comment in Dr. Leach's notes from November 2012 that hemoglobin A1c was above goal and required attention."

Don't see a number for that.

Here -- no, that's uric acid.

"He was encouraged" --

May 9th, 2013, last A1c measure had been 8.6 percent. So that was the first A1c I saw in the records that I got.

Q. And that was after his diagnosis of kidney disease Stage 3, which was in 2012?

A. Yeah. Yeah.

Q. Okay. So do you have any blood sugar data prior to his diagnosis for Stage 3 kidney disease?

A. Just the -- just the remarks in the chart that he had a diagnosis of diabetes from before that. But I don't have any blood -- there was no blood sugar in the records that I got.

Q. And there were no -- there were no point -- what do you call those, like, when you have an actual blood sugar level versus an A1c? Because A1c is, like, for a period of time; right?

A. Yeah.

Q. It's a marker of, like, the last three months

or something?

A. Exactly.

Q. And then the other one -- like, if you do a finger stick, that's --

(Over-speaking.)

A. Or a venous blood draw. So a finger stick -- a point-of-care, they call them -- or just a venous blood draw for -- I didn't see any of those, or see any remarks about high blood sugars there, no.

Q. And in the medical records from the hospitals, is blood sugar a -- is that part of the normal Chem 7?

A. Yes, it is.

Q. Okay. So if they're doing a basic chemistry, it should show up?

A. Yeah.

Q. And then hemoglobin A1c, that is the longer-term measure, which can be diagnostic of diabetes as well, and is also a measure of control; is that right?

A. Yes.

Q. And the goal is to get people less than 7?

A. Yeah, I mean, the goal is to establish a target that's best for the patient. In general, we talk about 7 as being a goal for preventing

complications.

Q. And I think that maybe older patients, that (indiscernible) up a little bit --

(Over-speaking.)

A. Yeah, exactly.

Q. -- because the burden of treatment is more than --

A. Yes.

Q. -- not enough juice in the squeeze?

A. Yeah.

Q. Okay. So in terms of what his actual blood sugars were between 1998 and 2012, we have a single point of 186, point-of-care, while he was on steroids in the hospital --

A. Yeah.

Q. -- and that itself was not diagnostic?

A. Yes.

Q. And so we're assuming that he had elevated blood sugars up until his diagnosis of Stage 3 chronic kidney disease, but we don't know the numbers?

A. Yeah. That was my assumption based on the medical records.

Q. And we also are assuming that that level was high enough to cause kidney injury in whatever period of time it would -- how long would you normally expect

that to take?

A. For -- I mean, it's hard to say, but usually that takes years, three to five years, to start to manifest.

In type 1 diabetes, we always think of it as five years; and the reason is because Type 1 patients tend to be young -- juvenile onset is what they used to call it -- and only have diabetes as their -- you know, they're otherwise pretty fit.

In type 2 diabetes, it's hard to say because they're older patients, and they -- besides diabetes, they are almost always obese, they frequently have high blood pressure, they have a bunch of other meds. But I would say it plays out over three to five years to get started, and you can have, you know, Stage 3 kidney disease inside of eight years.

Q. So Stage 3 within eight years. Is there a normal -- because, I mean, assuming that you could track people's data -- I guess you can with Type 1s; right?

A. Mm-hmm.

Q. That you can -- most of the time, you start seeing abnormalities in the GFRs and the creatinines in normal everyday physical blood tests; right?

A. Yeah.

Q. Because that's normally what's also checked as part of a physical on a yearly basis?
A. It's part of your Chem 7.
Q. That's a pretty standard lab. There's probably 10,000 of them going on right this second?
A. Yeah.
Q. Okay. And what you see is a gradual rise in those numbers, usually?
A. Yeah -- well, in somebody who's developing CKD.
Q. Understood.
And that rise can be more accelerated in some patients and less in others?
A. Absolutely.
Q. And in some patients, it can be a low rise in your Stage 1, and it's medically treated with lifestyle modification, maybe some medicines -- blood pressure medicines, what have you, lisinoprils --
A. Yeah.
Q. -- and things that help the kidneys?
A. Yeah.
Q. And I guess there are some new-generation drugs for that now: There's Ozempic and all that kind of stuff --
A. Yep.

Q. -- that's showing some promise in that already also?

A. Yes.

Q. Okay. So the data -- the assumption that we have to make that -- let me make this clear. You are not here to say that diabetes was the sole cause of his kidney disease; right?

MR. KLOTZBUCHER: Objection.

You may answer.

THE WITNESS: I mean, I don't think I can say that.

BY MR. MCGOWAN:

Q. How does obesity cause kidney disease? Is it what obesity causes which then causes it? Or is there something about being overweight that actually affects the cells?

A. It's --

Q. Or is that a Nobel Prize that we'll win if we figure it out?

A. Yeah. If you want to take some time off, maybe this summer, we can work on that.

But no, it comes up all the time. So it's an association. But you're right, the company that obesity keeps -- hypertension, dyslipidemia, diabetes -- they all add.

I think most of the data suggests that being heavier, per se, adds to the risk for CKD. But the mechanisms are so hard to separate out because of, as you say, there's a lot of confounders. But, I mean, the -- for the last 15 years, that's been -- you know, that's the kind of stuff that gets into textbooks because enough people agree that that is a risk factor.

Now -- well, I won't --

Q. Yeah, but whether being overweight is the direct cause of kidney disease or not is --

A. I guess what I would say is how much it is a cause. What is the percentage contribution it makes to your development of CKD is unknown.

Q. I see.

Now, dyslipidemia: that's high cholesterol, basically?

A. Yeah.

Q. Okay. So how does that play into causing kidney disease?

A. Atherosclerosis and vascular damage.

Q. So just less blood vessel to and through the kidney, basically?

A. Yeah.

Q. Okay. Have you ever --

(Off-record comment.)
BY MR. MCGOWAN:
Q. Has anybody ever studied a patient like Mr. Keller who's had toxic exposure, he's had chemo stem cell, and he's got type 2 diabetes, and its relation to kidney disease?
MR. KLOTZBUCHER: Objection. Form. You may answer.
THE WITNESS: You know, has anybody, say, taken one of those people and done a detailed study of that particular person? Or has anybody looked at the question broadly?
BY MR. MCGOWAN:
Q. Yeah, so let's look at -- let's talk about three factors, and we can combine them however we want. But, you know, broadly speaking --
MR. KLOTZBUCHER: Counsel, I'm sorry to interrupt. We have been going about an hour. I don't know if anyone wants a break or if the doctor needs a break --
MR. MCGOWAN: Anytime is fine.
THE WITNESS: Yeah, it sounds like this is a watershed, but why don't I go shed some water.
MR. MCGOWAN: Sounds good.
THE VIDEOGRAPHER: Off the record at

9:58 a.m.

(Recess taken from 9:58 a.m. to 10:04 a.m.)

THE VIDEOGRAPHER: On the record at 10:04 a.m.

BY MR. MCGOWAN:

Q. All right, Doctor, we're back from a break. But I think the subject matter we were talking about was the combination of patients that had chemical exposures, chemo stem cell treatments, and diabetes. Okay? So --

A. Okay.

Q. -- has there been studies of patient populations that had kidney disease and two or more of that list of three?

A. Not that I'm aware of.

Q. Okay. So you're aware of no evidence that -- or there's no medical studies -- no medical evidence that's been compiled yet that knows what to do with a patient like Mr. Keller who had all three of those, and its contribution to kidney disease?

A. Yeah, I couldn't find anything.

Q. Did you search for that kind of stuff?

A. Oh, yeah.

Q. Let's go -- I'm going to go through your report, and we're not going to read the whole thing,

but there are just some questions I have.

A. Mm-hmm.

Q. Did you write this yourself, or did you have assistance with it?

A. No, I wrote it myself.

Q. Okay.

A. I write semi-professionally.

Q. I'd like to say I do, too, but mostly it's lawsuits and junk that they're not nearly as exciting.

Okay. And I'm on page 4 of 27. It's about his creatinine in July of 2023 noted to be 1.4.

A. Yeah.

Q. I looked it up -- because, you know, Dr. Google --

A. Yeah.

Q. -- and normal creatinine for a man is -- and how old he would have been at the time -- would have been 1.1 to 1.3? The high limit?

A. Yeah. Again, did you -- did Dr. Google mention what the normative range was in 2003? Because it's changed over time.

Q. No. That's why you get the medium-sized bucks, Doctor.

A. Yeah. You understand endocrinology, it sounds like.

No. But I think your point is well taken, that a 1.4 is either the upper limit -- in the upper limit of normal or just over the upper limit of normal.

Q. And we don't have other data until, basically, 2012?

A. Yeah. Well, 2012, I think, is -- yeah, that's when he's referred to Dr. Leach.

Q. And by then -- at or about that time, it was diagnosed as Stage 3?

A. Yeah.

Q. So if we track a normal course of Stage 3 kidney disease, six to eight years, that would track --

A. Yeah.

Q. -- as being the beginning?

A. If we -- oh, you're saying if we went backwards from 2012 to 2004?

Q. Well, we only have two data points, and the question is whether we can reasonably connect the dots, or if it's just so random that you just couldn't do that?

You know, could this be that that 1.4 was the beginning of that progression that was finally -- the other data point we got to in 2012 was Stage 3?

A. I thought that --

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: I thought that your -- what did you say? We could connect the dots or it was random?

BY MR. MCGOWAN:

Q. Yeah.

A. I thought it was likely to be random. But I think, you know, it's a -- either way, we would be making inferences based on really, really limited data.

Q. Okay. So -- well, there's a conclusion it's either random or could be connected. So why did you choose it was random versus not? Just --

(Over-speaking.)

A. Just because that would be -- a number that small would be -- well, I thought -- I think that's a kind of number that can be part of day-to-day variation.

If that number had been 1.8, 1.9, then I would have been much more suspicious that he had active disease going on then.

Q. Is it as likely as not that that 1.4 was the -- was connected to the Stage 3 CKD that was

diagnosed in 2012?

MR. KLOTZBUCHER: Objection.

You may answer.

BY MR. MCGOWAN:

Q. Even money, Doctor.

A. Yeah, I have no way to say. As likely as not? I would say less likely. That is, I think this is more likely to just be random variation because the number is so small. That would be -- that would be my intuition. That's the way I'd frame it. I couldn't show you a study that had 1,000 patients with that number.

Q. Do you have any viewpoint on when the chronic kidney disease was clinically evident to start?

A. No.

Q. Okay. So a normal course of Stage 3 CKD would be six to eight years, generally?

A. I would say, you know, could be three to eight. You know, sometimes it's faster than other times.

Q. Okay. Is there any way to accurately or reasonably backdate when Mr. Keller would have had the first clinical indication of kidney impairment based upon the sole piece of data 2012 Stage 3?

A. No. I don't have -- there was no data --

that was the first thing -- that was my first paragraph, was there was no data to say when he started having kidney disease -- when he had clinically apparent kidney disease and when he had diabetes. There's a huge gap there.

Because we know he had an A1c of 8.6 in 2013, and he was on -- when did we see that he was on glimepiride?

I mean, again, we are making inferences about both of those diagnoses, when they started, CKD and diabetes, and somewhere between, I would say, 2003 and 2012 for the kidney disease.

Q. Does it matter which occurred first --

A. Well, I mean --

Q. -- in your opinion?

A. Yeah. If he had diabetes, and that was the cause of his kidney disease, it does matter.

Q. And what if his kidney disease was before he had diabetes?

A. Then the diabetes would have contributed to its progression, but it wouldn't be the initial insult.

Q. And so there's no data to know whether we have chicken or the egg in this particular case; is that true?

A. Well, there's -- when you have gaps in the medical database like this, there's no way to know a lot of stuff.

What I said in my report, and I think is pretty well documented in the literature, is that at the time most people are diagnosed with type 2 diabetes, they've had subclinical disease for three or four years. And I gave the references; they're in there.

And so if in -- my calculation was if -- if he had a diagnosis of type 2 diabetes in 2008 or '9, which we infer from Leach's records and some of the records further on, then he probably was having hyperglycemia starting in 2003, '4, '5, because that would be the normal course for most people with diabetes.

Again, all projections. But in a -- you know, when there's a big gap in the records, what I -- what I was doing was projecting what I thought was most likely based on -- you know, most people with type 2 diabetes, hyperglycemic before it has a long subclinical period, so I thought that -- I thought that held up pretty well.

Q. I'm curious that -- isn't it normal for the care of a diabetic patient to do basic labs?

I mean, were you a little struck by, you know, "Where's the data?" Because that's about as basic -- I mean, that's blocking and tackling, it seems like.

A. I mean, I asked a lot of those questions myself. I mean, I've been to Walla Walla. It's not, you know, Afghanistan or something. They have doctors and clinics and stuff. I just -- I couldn't believe that there wasn't some more documentation. And this is a man that had had a serious illness. His oncologist said he was in remission, but then he seemed to get out of the medical system for a long period of time.

Q. And can you see signs and symptoms -- or signs of diabetes in something as basic as a urine dipstick?

A. Yeah.

Q. Sugar in the urine?

A. Yeah.

Q. That's the way it used to be diagnosed; right?

A. Yeah. Diabetes smell, it is sweet urine.

Q. Yeah. Doctors used to do that. That's the way they knew. It's true.

All right. Let's -- did you ever see the

original -- and now I'm on page 5. It's "Dr. Leach noted a diagnosis of type 2 diabetes dating from 2009."

Did you ever see the origin document of that?

A. No.

Q. And then we have Dr. Leach's note from November of 2012, which just says his A1c was above goal, but we don't know what -- how much it was?

A. No.

Q. What would be a goal for a patient like Mr. Keller? Is that keep it below 7? Below 8? What's -- and I guess it would be back then because that's now 13 years ago.

A. If I'd have seen him then, at his age, I would have had a goal of 7. And I think, you know -- I mean, you correctly alluded to the fact that older, sicker patients, you don't run them so tight. But I think most non-endocrinologists would say the A1c goal is 7. And that was what I assumed of Dr. Leach; I assumed it was over 7 then.

Q. And we don't know if it was 7.1 or 12.9?

A. We don't.

Q. Okay. What can you infer from the medicine that was given, the glimepiride? What is it?

That's not an insulin --

A. No.

Q. -- so we didn't need that at that point. And it's not Glucophage?

A. No.

Q. And -- tell me how that acts and its roll in treating.

A. So glimepiride is part of the sulfonylurea class of drugs that go back to the '50s. So those are really old diabetes drugs. Glimepiride is a new sulfonylurea that came out in the '80s --

Q. 45 years ago now.

A. Yeah. And it -- these drugs stimulate the pancreas to make more insulin than it would normally. And the notion is that people with type 2 diabetes still make insulin, they just don't make enough, so the sulfonylurea kind of gives them a little boost, a little kick. They're easy to prescribe, they're once a day, and they're almost free, they're so cheap. And so it's, you know, a common drug to start.

I mean, usually you would start metformin. That's the current recommendation. But not unusual for a primary care doctor, particularly one that was trained when I was trained, in the '80s, to use sulfonylurea first. So you just put people on it.

I mean, the reason we don't use them is that if you have borderline diabetes, you can actually get hypoglycemic with these drugs, unlike with, say, Glucophage metformin. But this is not a -- this is an old-timey drug, but it would be a common primary care prescription early on with somebody --

(Over-speaking.)

Q. -- it was more common 13 years ago than it is today, probably?

A. Yeah. Yeah.

Q. Does that have any impact on kidney function?

A. No. Not except -- except that in one of the studies that showed that lowering blood sugar decreased kidney function, a sulfonylurea was one of the intensive treatments.

So, again, it doesn't impair kidney function, but if it lowers blood sugar adequately and for enough time, it will decrease the risk of diabetic kidney disease.

Q. The next paragraph down here --

A. Is this on 4 still?

Q. Yes, sir -- actually, it's 5, and I'm in the middle paragraph --

A. "Mr. Keller was seen by Leach"?

Q. Yes, sir. And (as read):

"... had declined as indicated by a GFR of 32, and his uric acid level was elevated to greater than 10."

Was that indicative, in your view, of the kidney disease?

A. I -- it was hard to say. I mean, this is a guy that had clinical gout. Again, in notes that are ten years after this one, somebody refers to him having his gout diagnosed with an aspiration of a gouty elbow. So he had clinical gout.

High uric acid is a risk factor -- one of the primary risk factors for gout. But this could also be high because his GFR is 32. So no way to say for sure that it was -- you know, he may have had high uric acid as a precipitant of his gout. We don't have those numbers. But you're also correct in your inference that a GFR of 32 can cause you to not excrete as much uric acid. 10 is still a high number.

Q. And gout is, like, crystallized --

A. Uric acid --

(Over-speaking.)

Q. -- uric acid in your joints?

A. When they precipitate in the joint space, you get inflammation and arthritis.

Q. Okay. According to this, it says -- in that sentence I'm reading, it says, "At that time" -- which would be, I guess, around this 2023, May -- he was taking glimepiride and sitagliptin?

A. Sitagliptin.

Q. Sitagliptin. What kind of drug of is?

A. Sitagliptin is another -- it's a newer pill. It's not a sulfonylurea. It actually prevents -- so you'd already alluded to Ozempic, or semaglutide, which is based on a hormone that the gut makes called GLP-1. When we eat, GLP-1 goes up; it helps us secrete insulin. But GLP-1 isn't -- it's degraded quickly in the plasma. Sitagliptin prevents that. So, again, it's another way to boost insulin secretion. Very popular in primary care.

Q. It looks like 2012/2013 his -- he had an A1c of 10.7. That's really pretty high?

A. Yeah, 10.7 is pretty high.

Q. Does one -- if you're treating a patient who's presumably taking their medicine, and their A1c is continuing to rise, that's not uncommon?

A. No.

Q. And you have to change the medical management.

And, in fact, his was ultimately changed

again. I believe he was put on insulin at some point?
A. Yeah. Soon thereafter.
I mean, this is -- this is a common scenario for somebody -- for an endocrinologist, is that the primary care doctors are uncomfortable with insulin, and they oftentimes don't have the resources to get people started on insulin, so they'll use pills, and they'll use -- the patient will come in and say, "Well, I know my A1c is high, Doc, but I'll follow my diet. This is going to get better." And that will go on for a while, and the doc will keep giving them pills and they need to be on insulin, and then they come to us, and we put them on insulin.
So this is not an uncommon scenario, to have a patient whose diabetes is being managed by primary care on pills that aren't effective.
Q. And probably every patient says, "I promise, Doctor, I'm going to eat better and I'm going to get more exercise"?
A. I've been fooled many times.
Q. Easy to say and hard to do. I get it.
All right. So it looks like -- I'm in the next paragraph now, 2018.
A. Yeah.
Q. Macular degeneration: Is that a complication

of some other illness? Or is that sort of a primary --

A. As far as I know, it's a primary illness. So it's not diabetic retinopathy, if that's what you're asking.

Q. Which is basically where the vessels in your back of your eye, they leak, basically?

A. Yeah. Macular degeneration is a separate diagnosis. And I -- to my knowledge -- well, my knowledge is not worth recounting on that.

Q. All right. So in October of 2028, his GFR was mildly reduced to 32 with moderate proteinuria. And that is the same number, at least -- the GFR of 32 was from 2013. Does that mean that his kidney disease had plateaued, do you think?

A. Yeah. Again, when the kidney function gets to be that level, just small differences in, you know, hydration can make a big difference, and so the -- the variability is wide.

I -- you know, I've seen people on their way down to dialysis stabilize. I've seen them drop and come back. But it starts to be -- I mean, once it gets to 32, it's not -- you know, unless -- it's not likely to stay there for a long time, like years.

Q. So how do you -- how do we explain or talk

about that in 2013 his GFR was 32, and there doesn't appear to be -- and in August of 2013, it was 45. Is that a material difference? Or is that just kind of a measurement?

A. Yeah, it could be -- you can see that kind of variability, not day-to-day, but over time --

(Doctor's pager alerts.)

Q. If you need to take that, you're welcome, Doctor.

A. I don't. I don't. Sorry.

Yeah, I -- to my way of thinking, he had -- he still had borderline kidney function, and it hadn't -- it certainly hadn't gotten better from '13 to '18. And I think we see further on that his eGFR will go up and down several times over the course of the rest of the report.

So, yeah, could he have plateaued? Could have, but it turned out he didn't.

Q. Well, the more data that we have -- and this is on the next page, page 6. It says November of 2019, his GFR was 29?

A. Yeah. But again -- so, and then -- then it was 26. And then it was 30.

So, again, there's a -- as you start to get kidney function, there's a lot less reserve and

there's a lot more wobble, and small things like blood pressure, hydration, other medicines can have a -- can push that number around a little bit.

Q. It looks like in June of 2022, his GFR was 27?

A. Yeah.

Q. Which is bouncing around for the last -- looks like probably ten years, I guess?

A. Yeah. And that -- you know, that -- when it gets under 30 -- now, again, it shows you the arbitrariness of the stages of CKD -- but now when he's 27, he's Stage 4. That's under 30, but again, you know, in reality, we just know that that's bad, and it's -- the difference between a 27 and a 31, it's a continuous variable.

Q. You got to draw the line somewhere?

A. Yeah.

Q. I understand.

When does one go on dialysis?

A. Usually it's -- once the eGFR gets under 50 -- 15, you're starting to think about it. But you wouldn't put somebody on a dialysis just for that number. What you look for is are they having trouble excreting potassium. So if the potassium is really high, that can be dangerous. Are they having trouble

excreting acid; so their bicarbonate is low. And then are they having symptoms of uremia; are they confused, fatigued, do they have heart failure.

So, you know, it's a clinical decision that a nephrologist makes. And sometimes I've seen people with eGFRs of 8, you know, tank along for a while; and then I've seen people go on dialysis for eGFRs in the 20s, if they're having some acute problem related to kidney impairment.

Q. It appears, at least from your report, that he at one point got off all of the diabetic medication?

A. Yeah.

Q. Okay. How's that work?

A. The paradox of renal failure is -- one of my professors told me it's an irony that you see all the time -- somebody whose blood sugar has never been controlled until they have the ultimate complication.

So a couple of things have happened here. One, he's lost weight over time; and that -- I was attributing that to just progressive renal failure, where weight loss is a thing. So this is a guy that, you know, I think when we met him, his body weight was around 250, plus or minus 20 pounds, and at some point in the late 20-teens, he gets down to body weights of

170, 180. So just that amount of weight loss can improve your diabetes a lot.

The other thing is, as your GFR gets low, the amounts of insulin that you secrete aren't cleared anymore. About half the insulin that we make gets filtered through the kidneys and out. So all of a sudden, an insufficient amount of insulin starts to be more.

So this is really typical of a diabetic patient who's approaching end stage, is that you start throwing all the diabetes medicines off. It becomes a hypoglycemia risk for them.

Q. "Congratulations, you don't have diabetes anymore, but" --

A. Yeah.

Q. -- "but your kidneys are shot"?

A. That's the -- that's the irony.

Q. Yeah.

All right, let's talk about the opinion part, and then we'll --

A. Yeah.

Q. I was hoping it was going to be shorter, but we're going to be here for a minute. I'm on page 6.

A. Uh-huh.

Q. (As read):

"... chronic renal failure, with steady worsening from 2012 through the present ... clearly documented in the notes from [the nephrology clinic]."

I think we covered all of that. The first time that we would have seen that would have been in that 2012 note that said Stage 3 CKD?

A. And I would just add that, like the diabetes, we don't know what the number was that caused him to be referred.

Q. In the next bit, were he to have a biopsy, you could -- there's signature findings on kidney tissue that a pathologist could look for to identify the cause?

A. Yeah, if they'd have done it ten years before. Once the kidneys get -- that is -- there's a lot of reasons to get chronic kidney disease. As the GFR goes down, and you get more and more dead cells and scar and fibrosis and stuff, they all start to look alike.

Q. I see.

A. Similarly, you know, you do images and the kidneys just gradually sort of atrophy. And so, you know, when you look at biopsies and things, it's hard

to tell what the actual cause was.

Q. At this point?

A. At this point. This --

Q. Presumably --

A. At this point, you're talking about, you know, a scarred, fried, kidney.

Q. Okay. Tell me exactly what you understand Dr. Felsher's opinion about the contribution of the cancer treatment to the kidney disease?

MR. KLOTZBUCHER: Objection. Form. You may answer.

THE WITNESS: Yeah, I couldn't make much of it. He mentioned it in two or three places in his opinion but didn't discuss it very much.

There was a lot of interesting stuff in there about lymphoma and the genesis of lymphoma and cancer genetics and things like that, but I -- he just didn't discuss it enough. He just mentioned it twice -- that the kidney disease and the heart disease were related to the treatment of lymphoma.

BY MR. MCGOWAN:

Q. Do you believe that the heart disease in this case was related to the kidney disease?

A. I mean, it's -- again, it's like we were saying about obesity and kidney disease. So diabetes

is a big risk for heart disease, and kidney disease is a -- both of those things promote atherosclerosis. But he also had hypercholesterolemia that probably contributed. So multifactorial cause of heart disease.

But certainly 75 percent of people with type 2 diabetes die of heart disease, and it's 50 or 60 percent of people with CKD die of heart disease. So both of those things go together with -- and he had ischemic heart disease. Now, he may -- they called it ischemic heart disease.

Again, by the time he was getting all his cardiac workup, he could have had -- there's a diabetic cardiomyopathy that's non-ischemic. But at that point, we're just talking about a beat-up heart. He did have some vascular disease, so we could say he had atherosclerosis too.

Q. You wrote (as read):

"Moreover, there was no evidence from his basic lab tests that his kidney function had declined in the three years after his chemotherapy and SCT, which would be expected if these were the cause of eventual CKD."

A. Yes.
Q. Tell me your basis for having a three-year -- your basis for that statement.
A. My basis for that statement were, you know, some observational studies, both prospective and retrospective, that looked at kidney function in people after hematopoietic stem cells, and the people that got kidney disease got it in the first year, but I extended it to say, you know, it might go two or three years.
Q. Okay. So which study was that? Because --
A. This would be in the supplemental data that we sent to you. The -- what did we call that?
Q. Materials Considered list?
A. Yeah.
Q. We love to say that. It's fancy --
(Over-speaking) --
A. Yeah. So there's five papers listed there. One, I realize, I sent the wrong PMID number because it's totally irrelevant, it's about colon cancer. But there's some series, one from Fred Hutchinson in Seattle, that talk about this -- talk about kidney function and after stem cell therapy.
Q. So did you read those studies before you wrote the report? Or did you --

A. No, afterwards. I mean, I read -- no, I read them before the report.

Q. Okay. And they just -- I know you had a lot of references -- it just fell off the list, basically?

A. Yeah. I mean, again, it wasn't diabetes related, so...

We're on page 7?

Q. Yes, sir.

All right. I'm at, basically, the first paragraph, about NSAIDs. Tell me -- that's, like, ibuprofen? "Vitamin I," as many people call it?

A. Yeah.

Q. Apparently that's bad for your kidneys?

A. Yes.

Q. How so?

A. I mean, they -- part of their mechanism of action, I mean, inhibiting anti-inflammatory molecules, can be damaging to the kidneys. And they're concentrated in the kidneys because they're --

Q. Excreted through the kidneys?

A. Yeah.

Q. Okay.

A. And it turns out, you know, for people that take a lot of them, it takes a toll.

Q. So how much was he taking?

A. We don't know.
Q. Okay. So how do you know if it was enough to take a toll?
A. I didn't. I didn't say it took a toll. I just said it was something he did that was marked in his -- he had gout and he took NSAIDs, and it was remarked on in a couple of places in the chart.
So I don't know that -- I just know that he was taking NSAIDs, and that that is a risk factor for kidney disease.
Q. Well, you made a list (as read):
"Important among these" --
And you're talking about the risk factors for CKD?
A. Yeah.
Q. "Hyperuricemia" --
A. Yeah.
Q. (As read):
"... NSAIDs, dyslipidemia and atherosclerosis, obesity, and diabetes."
A. Yeah.
Q. Okay. It is your view that diabetes was the cause?
A. I mean, my whole point in listing all these

risk factors was I wasn't going to attribute them to one cause. They're all risk factors for CKD.

Q. You didn't include in your list exposure to chemicals nor the treatment for the cancer?

A. Those aren't -- I mean, I didn't include them because, one, those are outside my area of expertise, but two, you don't find those in common lists of risk factors for CKD.

Now, common lists in textbooks or review articles don't include every single thing that's ever been connected to CKD, but they include all the major ones, and so did I.

Q. Well, I appreciate that, but we're here about Mr. Keller as an individual person. You understand?

A. Yeah, I do understand. I -- but I -- what I -- what I didn't come across, and what I really am not, I think, qualified to comment on, is toxicology. I mean, I have no idea what benzene does to eGFR. Just don't. But I know about these, and so I put in what I know about.

And I've told you -- you know, we've talked some about the stem cell transplant, and I've told you why my -- why I didn't think that was a -- proximal to his kidney failure.

Q. So being fair about this, and acknowledging

your expertise -- and I don't mean this in a bad way -- and the lack of expertise -- can't know everything about it --

A. Yeah.

Q. -- despite what Dr. Google tells us -- is that you could not fairly evaluate the contribution of chemicals nor the chemo or the stem cell -- the treatment for the cancer because that is simply just not something of which you have adequate knowledge to make an evaluation; is that right?

A. Yeah, so I would --

MR. KLOTZBUCHER: Objection. Form.

THE WITNESS: I would say -- I would separate those two.

I can't comment on the chemical exposure, which is toxicology and well outside my expertise.

I am more confident about my opinion that his stem cell therapy treatment did not contribute meaningfully to his kidney disease. I can't say that's impossible, but I think the fact that he had relatively normal -- he had normal kidney function for three years after the treatment makes me think that that was a very low likelihood.

And then he has all these other risk factors on top, none of which I could attribute 10 percent,

15 percent, et cetera, but people that have those exposures have an increased risk of CKD.

BY MR. MCGOWAN:

Q. Did Mr. Keller have normal kidney function after the treatment? Or is there just no data to show it was abnormal?

A. He had normal creatinines, the same value he went into the hospital with before his stem cell treatment, on several occasions.

Q. I'm on page 8.

A. Okay. Me too.

Q. Top of it says (as read):

> "Susceptibility to end-organ complications varies among diabetic patients, with most of the variance determined by multiple genetic factors conferring different levels of susceptibility."

A. Yeah.

Q. Okay. Tell me what that has to do with Mr. Keller. Does he just have certain genetic makeup that makes him more likely to get CKD under the circumstances, whereas another patient might not?

A. Yeah, I mean, this was just trying to account

for the time course, the latency, and including all the things that contribute to his kidney disease. That, as we talked about, some people progress rapidly and other people slowly. And we assume -- we know that this genetic risk -- we haven't found the genes yet -- there's genes that associate with diabetic nephropathy -- and we know that some of the susceptibility is heritable: that is, you can find it in families. And so that's where -- and then when I talk about most of the variance being genetic factors, if you do studies where you treat the blood sugar, treat the blood pressure, there's still a big explanatory part that's left over, and we attribute that to genetics.

But all I was saying here is that rates of development of type 2 diabetes vary among patients, but Mr. Keller followed sort of an average course, based on my assumption of when he got diabetes and what the progress was once he saw Dr. Leach.

Q. Does diabetes -- or high blood sugar, I guess, cause kidney disease more frequently than uncontrolled hypertension?

A. I mean, uncontrolled hypertension is a continuous variable, too. I mean, people that have really high blood pressures can have acute damage that

is -- that progresses really quickly. But to say, you know, one is worse than the other is hard.

Q. I mean, hypertension can cause chronic kidney disease?

A. No question.

Q. I think Barry White died of that --

(Over-speaking.)

A. Yeah.

Q. -- a lot of people do, I guess?

A. Yeah.

Q. And diabetes can cause kidney disease. And frequently people that have one have the other?

A. All true.

Q. Okay. All right.

What's going to happen now is I'm going to take probably five minutes, go over my notes --

A. Okay.

Q. -- and then we'll go from there.

A. Yeah. Terrific.

MR. MCGOWAN: Thanks.

THE VIDEOGRAPHER: Off the record at 10:42 a.m.

(Recess taken from 10:42 a.m. to 10:57 a.m.)

THE VIDEOGRAPHER: On the record at 10:57 a.m.

BY MR. MCGOWAN:

Q. All right, Doctor. Is there any other answer that you want to explain further, that I may have cut you off on? Anything else that's germane to my questions that you want to say that I didn't give you the chance to?

A. No.

MR. MCGOWAN: All right. Well, that's all I have for you, Doctor. Thank you.

THE WITNESS: Okay.

CROSS-EXAMINATION BY COUNSEL FOR THE DEFENDANTS

BY MR. KLOTZBUCHER:

Q. I have just a few questions, Dr. D'Alessio.

In your practice, you've treated patients who have had NHL -- non-Hodgkin's lymphoma treatment?

A. I'd have trouble -- I've seen patients that have had lymphoma in the past and been treated for it. I've seen patients in the hospital who are undergoing stem cell treatments -- and I've done consults on them as well, yeah. But -- no, I think that's true. But it's -- the lymphoma part is always background to me. I'm always involved to focus on an endocrine problem on top of that.

Q. I understand. So it's -- you're part of a care team --

A. Yes.
Q. -- or it's part of the patient's medical history?
A. Yes.
Q. Okay. Did you review the drugs Mr. Keller took when you reviewed his records? The --
A. Yeah.
Q. I'll rephrase. The drugs that he took regarding his NHL treatment?
(Court reporter asks for clarification.)
A. So I looked through the -- you know, I looked through -- in Walla Walla, Dr. Iacoboni gave him what's called CHOP -- C-H-O-P -- or CHOP-R -- something like that -- and that's something I remember from my residency. They used to use CHOP. "CHOP till you drop," we used to say. And -- you know, so I know Cytoxan and Vincristine and those.
And then I think he got Adriamycin and etoposide after his radiation. So I've heard of those drugs too.
Q. Did you review whether those drugs were toxic to the kidneys?
A. Yeah.
Q. Are those drugs toxic to the kidneys?
A. He got -- and one of the other experts wrote

that he got vancomycin. That's an antibiotic that's a nephrotoxin.

And to -- on the search I did, which was just a quick internet search, I couldn't find nephrotoxicity to the other chemotherapies. But again, not my area of expertise.

MR. KLOTZBUCHER: Understood.

I have nothing further. Thank you for your time, Doctor. I appreciate it.

THE WITNESS: Yeah.

MR. MCGOWAN: Thanks, Doctor.

THE WITNESS: Yeah.

THE VIDEOGRAPHER: Off the record at 11:00 a.m. This concludes the deposition of David D'Alessio.

(Whereupon, at 11:00 a.m., the deposition ceased. Signature was reserved.)

ACKNOWLEDGMENT OF DEPONENT

I, DAVID A. D'ALESSIO, M.D., do hereby acknowledge that I have read and examined the foregoing testimony, and the same is a true, correct, and complete transcription of the testimony given by me, and any corrections appear on the attached errata sheet signed by me.

_____ _____

(DATE) (SIGNATURE)

E R R A T A

CASE NAME: IN RE: CAMP LEJEUNE WATER LITIGATION

WITNESS NAME: DAVID A. D'ALESSIO, M.D.

CASE NUMBER: 7:23-CV-897

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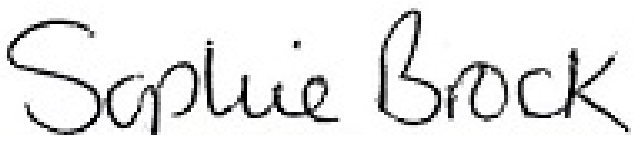
STATE OF NORTH CAROLINA )
) C E R T I F I C A T E
COUNTY OF ORANGE )

I, Sophie Brock, Registered Diplomate Reporter, Certified Realtime Reporter, and Notary Public of North Carolina, the officer before whom the foregoing proceeding was conducted, do hereby certify that the witness whose testimony appears in the foregoing proceeding was duly sworn by me; that the testimony of said witness was taken by me to the best of my ability and thereafter transcribed under my supervision; and that the foregoing pages, inclusive, constitute a true and accurate transcription of the testimony of the witness.

I do further certify that I am neither counsel for, related to, nor employed by any of the parties to this action, and further, that I am not a relative or employee of any attorney or counsel employed by the parties thereof, nor financially or otherwise interested in the outcome of said action.

This, the 7th day of July, 2025.



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Sophie Brock, RDR, CRR
Notary Number: 200834000001

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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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