

# 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 Diplomat Reporter, Certified Realtime Reporter, and Notary Public in and for the State of North Carolina.

Job No. MDLG7434034

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REPORTER'S NOTE: There were no exhibits marked.

1 P R O C E E D I N G S

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

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

7 The deponent is Dr. David D'Alessio.

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

10 MR. MCGOWAN: Chad McGowan for the  
11 plaintiff.

12 MR. KLOTZBUCHER: For the defense,  
13 William Klotzbucher and Allison O'Leary.  
14 Whereupon,

15 DAVID A. D'ALESSIO, M.D.,  
16 having first been duly sworn/affirmed,  
17 was examined and testified as follows:

18 EXAMINATION BY COUNSEL FOR THE PLAINTIFFS  
19 BY MR. MCGOWAN:

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

22 A. D'Alessio, yes.

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

25 A. It's okay.

1 Q. -- but I'm going to do my best.

2 Have you ever been deposed before?

3 A. No.

4 Q. Okay. So this is a chance -- I'm going to  
5 ask questions. Your job is to listen to the question  
6 and answer the question the best you can.

7 If you have any question about my question,  
8 it's unclear, please let me know. Otherwise it may  
9 look like we're communicating when we're really not.  
10 All right?

11 A. Mm-hmm.

12 Q. In front of you, hopefully, is a copy of your  
13 report that you made in this case?

14 A. Yeah.

15 Q. All right. Wonderful.

16 If you need a break at any time, for any  
17 reason, that is not a problem. I do not think this is  
18 going to go that long.

19 A. Okay.

20 Q. So we'll just get into it.

21 Do you have any --

22 A. No.

23 Q. -- concerns, problems, questions right now?

24 A. No, not at all.

25 Q. All right. Good. Let's get rolling.

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

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

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

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

9 Q. I gotcha --

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

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

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

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

23 A. Yeah, DoJ.

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

1 A. No.

2 Q. Probably the Duke website?

3 A. Probably, yeah.

4 Q. Okay. And the -- I've noticed some of your  
5 bills, it's, like -- what -- \$600 an hour, \$3,500 half  
6 a day kind of thing; is that right?

7 A. Yeah.

8 Q. Tell me what your total billing has been so  
9 far.

10 A. I think I -- one invoice for \$3,000 and one  
11 for \$12,000.

12 Q. And then you have time that has been accrued  
13 but unbilled?

14 A. Yes.

15 Q. And how much is that?

16 A. About six or seven hours.

17 Q. All right. And does that money go to you, or  
18 does that go to a practice or --

19 A. That money goes to me.

20 Q. Okay. And half of it goes back to the  
21 government; right -- in taxes?

22 A. Yeah.

23 Q. Understood. All right, let's talk about  
24 litigation history.

25 Have you ever acted as an expert -- have you



1 written expert reports before?

2 A. No.

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

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

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

14 A. Never.

15 Q. Have you ever been sued before?

16 A. No.

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

19 A. Endocrinology.

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

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

1 then metabolic problems like diabetes or cholesterol.

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

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

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

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

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

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

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

21 Thursday, I'm in clinic.

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

25 Q. How much of your time is spent teaching?

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

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

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

16           A. Yeah.

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

19           A. Yeah.

20           Q. Okay.

21           A. Yeah. So it should be 30/30/30, and it's  
22    30/30/60 because the administrative stuff never stops.

23           Q. Yeah, I understand.

24                 Is endocrinology a sub-board of internal  
25    medicine?

1 A. Yes, it is.

2 Q. Do you consider yourself a toxicologist?

3 MR. KLOTZBUCHER: Objection. Form.

4 THE WITNESS: I don't.

5 BY MR. MCGOWAN:

6 Q. Okay. A cardiologist, oncologist, or an  
7 expert in chemotherapy?

8 MR. KLOTZBUCHER: Objection. Form.

9 You may answer.

10 THE WITNESS: No.

11 BY MR. MCGOWAN:

12 Q. All right. We've been provided an extensive  
13 list of materials considered, because that's what the  
14 rules require. Was there anything else that should  
15 have been on that list that just wasn't?

16 A. No. We were pretty complete. And the stuff  
17 that's on that list, I looked at damn near every page,  
18 but there was nothing else.

19 Q. Okay. I appreciate you not just putting  
20 "PubMed" on there.

21 A. Yeah.

22 Q. Narrowed it down a little.

23 All right. Let's talk about the timeline  
24 for this particular patient.

25 A. Yes.

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

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

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

8 A. Mm-hmm.

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

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

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

16 BY MR. MCGOWAN:

17 Q. And when was the NHL diagnosis?

18 A. January 1998.

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

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

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

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

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

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

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

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

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

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

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

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

25 MR. KLOTZBUCHER: Objection. Form.

1                   You may answer.

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

6 BY MR. MCGOWAN:

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

9           A. That was another one -- I mean, when he was  
10 referred to Dr. Leach -- and I think that was May of  
11 2013 -- he had kidney failure, or he had chronic --  
12 what looked like chronic kidney disease, as marked by  
13 his eGFR, his creatinine.

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

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

21           Q. Did he have a creatinine of 1.4 in the early  
22 2000s?

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

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

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

6 Q. Is that slightly above normal?

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

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

12 MR. KLOTZBUCHER: Objection.  
13 You can answer.  
14 Form.

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

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



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

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

7 BY MR. MCGOWAN:

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

11 A. Yes.

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

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

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

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

1 gradually?

2 MR. KLOTZBUCHER: Objection. Form.

3 You can answer.

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

7 BY MR. MCGOWAN:

8 Q. Well, do most people --

9 A. Yes --

10 Q. -- progress gradually?

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

13 Q. And is that over years?

14 A. Yes.

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

18 A. No.

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

23 MR. KLOTZBUCHER: Objection. Form.

24 You may answer.

25 THE WITNESS: Okay.

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

3 BY MR. MCGOWAN:

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

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

11          Q. How does it do that?

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

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

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

24          A. Yeah.

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

1 do its job?

2 A. Yes.

3 Q. Okay.

4 A. Yes.

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

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

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

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

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

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

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

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

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

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

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

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

25 Like, can your kidney absorb some amount of

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

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

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

17 Q. I understand.

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

23 MR. KLOTZBUCHER: Objection. Form.

24 You may answer.

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

1 is a general term.

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

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

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

20 BY MR. MCGOWAN:

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

1 can coexist?

2 A. Oh, they could coexist --

3 MR. KLOTZBUCHER: Objection. Form.

4 Excuse me, Doctor.

5 THE WITNESS: Sorry.

6 MR. KLOTZBUCHER: You may answer.

7 THE WITNESS: Okay.

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

12 BY MR. MCGOWAN:

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

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

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

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

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



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

3 A. I guess --

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

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

14 40 percent of people that have type 2  
15 diabetes are going to get some kind of kidney  
16 dysfunction from it.

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

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

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

1 know, doesn't seem that relevant to me.

2 BY MR. MCGOWAN:

3 Q. Well, you said it was unlikely that his chemo  
4 and the treatment for the NHL -- and I mean this with  
5 all due respect -- how do you know how unlikely it is  
6 if you don't even know what the particular treatment  
7 was and the effect of that treatment on kidney cells  
8 is?

9 MR. KLOTZBUCHER: Objection. Form.

10 You may answer.

11 THE WITNESS: Again, part of it is  
12 the -- again, he -- whether he had any damage to the  
13 kidney with his chemotherapy, it didn't manifest as a  
14 significant functional deficit for some time; so, you  
15 know, for the most part, that suggests that any damage  
16 he incurred was not very serious.

17 And whether I know the particulars of rates  
18 of kidney failure with whatever drugs he got sort of  
19 doesn't matter in that case. It wasn't enough to  
20 cause a measurable effect on his kidney function.

21 And so, therefore, to extend that to  
22 causation for his Stage 3 kidney disease is just  
23 a little bit fanciful, I think.

24 BY MR. MCGOWAN:

25 Q. Okay. Well, let's get to the bottom of that.

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

4 A. Yeah.

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

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

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

19 A. Yeah.

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

23 A. Yeah.

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

1 lab work, your function?

2 MR. KLOTZBUCHER: Objection. Form.

3 You may answer.

4 THE WITNESS: It tells you --

5 MR. MCGOWAN: Hang on, Doctor.

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

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

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

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

20 BY MR. MCGOWAN:

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

24 A. Yeah.

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

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

5 A. Yeah.

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

10 MR. KLOTZBUCHER: Objection. Form.

11 You may answer.

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

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

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

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

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

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

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

10 BY MR. MCGOWAN:

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

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

15 MR. KLOTZBUCHER: Objection.  
16 Foundation.

17 You may answer.

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

22 BY MR. MCGOWAN:

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

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

25 Q. Okay. Can you tell me what effect those

1 chemicals have on kidney function, or kidney cells?

2 A. Yeah, not my area of expertise, and  
3 I can't -- as I said, I'm not a toxicologist.

4 Q. Okay. So what did you do to rule out the  
5 contaminants in the water as affecting his kidneys?

6 MR. KLOTZBUCHER: Objection.  
7 Foundation.

8 You may answer.

9 THE WITNESS: The first records I had  
10 of his kidney function was that it was absolutely  
11 normal; so, therefore, I concluded that he hadn't had  
12 significant damage to interfere with his kidney  
13 function.

14 BY MR. MCGOWAN:

15 Q. All right. Do you know how those chemicals  
16 work over time and latency periods and all that kind  
17 of stuff?

18 A. I don't. I think it's known, but I -- that's  
19 not an area I'm familiar with.

20 Q. And you know his exposure levels?

21 A. I know that he had some. I've seen in the  
22 other expert reports that you can make estimates of  
23 those and calculations of exposure and stuff, but  
24 that's, again, not my area of expertise.

25 Q. Do you agree with me that those chemicals are

1 known to cause kidney damage?

2 MR. KLOTZBUCHER: Objection.  
3 Foundation.

4 THE WITNESS: I don't know.

5 BY MR. MCGOWAN:

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

14 MR. KLOTZBUCHER: Objection. Form.

15 BY MR. MCGOWAN:

16 Q. See the question I'm asking?

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

22 Q. Just because --

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



1 purely speculative.

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

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

6 Q. Okay. So --

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

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

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

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

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

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

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

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

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

9 A. That's right.

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

13 MR. KLOTZBUCHER: Objection.  
14 Foundation.

15 You may answer.

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

19 BY MR. MCGOWAN:

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

22 A. I'm not sure I understand.

23 MR. KLOTZBUCHER: Objection.  
24 Foundation.

25

1 BY MR. MCGOWAN:

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

4 A. Yeah.

5 Q. Is that the way you used it in this case?  
6 Because I know medical folks, unless -- and I'm  
7 generalizing -- but in general, that if it's not  
8 95 percent plus, a lot of medical folks are not  
9 willing to say something is a likely cause. Versus us  
10 legal people who "more likely than not" is 50 percent  
11 plus a little. Right?

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

16 A. The medical side.

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

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

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

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

12 So that was -- I just -- I thought this  
13 looked like a fairly straightforward case of diabetic  
14 nephropathy in a man who had other risk factors. And  
15 I thought the -- rather than saying -- you know,  
16 moving from 50 percent to 95 percent, I thought, from  
17 100 percent down, how much would I give a contribution  
18 to his treatment for chemotherapy, and it's just  
19 infinitesimal.

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

22 A. Yeah, good one.

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

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

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

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

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

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

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

25 A. Very little. None.

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

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

15           MR. KLOTZBUCHER:   Objection.   Form.

16           You may answer.

17   BY MR. MCGOWAN:

18           Q.   That's true; right?

19           A.   That's theoretical.

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

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

1 evidence that showed that that was true. Yeah.

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

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

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

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

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

19 BY MR. MCGOWAN:

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

23 A. Yeah.

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

1 A. Let me see if I can say this a different way.

2 I -- regardless of how many damaged cells  
3 this guy had in 2001, do I think that that damage  
4 contributed to his kidney disease? I do not.

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

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

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

16 A. Yeah. Somewhere between 30 and 40 percent.

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

20 MR. KLOTZBUCHER: Objection --

21 BY MR. MCGOWAN:

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

23 MR. KLOTZBUCHER: Foundation.

24 You may answer.

25 THE WITNESS: Yeah, so what I tell



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

8 BY MR. MCGOWAN:

9 Q. Did Mr. Keller have uncontrolled  
10 hypertension?

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

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

19 A. No.

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

22 A. I did not.

23 MR. KLOTZBUCHER: Objection.

24 BY MR. MCGOWAN:

25 Q. The way that diabetes -- uncontrolled

1 diabetes causes kidney failure more often than  
2 controlled diabetes?

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

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

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

14 Q. Did Mr. Keller have genetic predisposition?

15 A. Don't know.

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

19 A. Yeah. No, those are -- that's been done in  
20 clinical trials, where one group gets usual care and  
21 one group gets more aggressive care, and the A1c in  
22 the usual care is 9 percent, and the A1c in the  
23 intensive group is 7 percent, and the group with  
24 7 percent, over five years, has half the kidney  
25 disease as the other group.

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

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

10 So we don't have great information even that  
11 6.5 is better than 7, or 6 is better than 6.5. But we  
12 know that 7 is better than 9, and it's been done a  
13 couple of times. And so we're pretty confident that  
14 high blood sugar is worse.

15 Q. All right. So 7 -- how much better is 7 than  
16 9 on -- for preventing or stemming kidney disease?

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

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

1 (Over-speaking.)

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

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

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

15 (Over-speaking.)

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

24 So hyperglycemia is an insult to kidney  
25 function.

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

3 A. Yes.

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

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

12 Q. Was he also on steroids at that time?

13 A. He was.

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

16 A. Oh, yeah.

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

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

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

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

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

5 A. Yes.

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

8 A. He got up to 186.

9 Q. Was that a fasting sugar?

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

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

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

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

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

21 (Over-speaking.)

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

1 tolerance.

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

4 A. More than anybody else right now.

5 Q. I gotcha. Okay.

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

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

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

15 A. No.

16 Q. No? Doesn't matter?

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

19 Q. All right. So tell me -- that was in 1999 --  
20 (Over-speaking.)

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

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

1 records... There is a comment in  
2 Dr. Leach's notes from November  
3 2012 that hemoglobin Alc was above  
4 goal and required attention."

5 Don't see a number for that.

6 Here -- no, that's uric acid.

7 "He was encouraged" --

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

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

13 A. Yeah. Yeah.

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

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

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

24 A. Yeah.

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



1 or something?

2 A. Exactly.

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

5 (Over-speaking.)

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

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

13 A. Yes, it is.

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

16 A. Yeah.

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

21 A. Yes.

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

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

1 complications.

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

4 (Over-speaking.)

5 A. Yeah, exactly.

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

8 A. Yes.

9 Q. -- not enough juice in the squeeze?

10 A. Yeah.

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

15 A. Yeah.

16 Q. -- and that itself was not diagnostic?

17 A. Yes.

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

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

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

1 that to take?

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

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

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

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

21 A. Mm-hmm.

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

25 A. Yeah.

1 Q. Because that's normally what's also checked  
2 as part of a physical on a yearly basis?

3 A. It's part of your Chem 7.

4 Q. That's a pretty standard lab. There's  
5 probably 10,000 of them going on right this second?

6 A. Yeah.

7 Q. Okay. And what you see is a gradual rise in  
8 those numbers, usually?

9 A. Yeah -- well, in somebody who's developing  
10 CKD.

11 Q. Understood.

12 And that rise can be more accelerated in  
13 some patients and less in others?

14 A. Absolutely.

15 Q. And in some patients, it can be a low rise in  
16 your Stage 1, and it's medically treated with  
17 lifestyle modification, maybe some medicines -- blood  
18 pressure medicines, what have you, lisinoprials --

19 A. Yeah.

20 Q. -- and things that help the kidneys?

21 A. Yeah.

22 Q. And I guess there are some new-generation  
23 drugs for that now: There's Ozempic and all that kind  
24 of stuff --

25 A. Yep.

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

3 A. Yes.

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

8 MR. KLOTZBUCHER: Objection.

9 You may answer.

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

12 BY MR. MCGOWAN:

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

17 A. It's --

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

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

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

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

9 Now -- well, I won't --

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

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

15 Q. I see.

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

18 A. Yeah.

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

21 A. Atherosclerosis and vascular damage.

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

24 A. Yeah.

25 Q. Okay. Have you ever --

1 (Off-record comment.)

2 BY MR. MCGOWAN:

3 Q. Has anybody ever studied a patient like  
4 Mr. Keller who's had toxic exposure, he's had chemo  
5 stem cell, and he's got type 2 diabetes, and its  
6 relation to kidney disease?

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

9 THE WITNESS: You know, has anybody,  
10 say, taken one of those people and done a detailed  
11 study of that particular person? Or has anybody  
12 looked at the question broadly?

13 BY MR. MCGOWAN:

14 Q. Yeah, so let's look at -- let's talk about  
15 three factors, and we can combine them however we  
16 want. But, you know, broadly speaking --

17 MR. KLOTZBUCHER: Counsel, I'm sorry to  
18 interrupt. We have been going about an hour. I don't  
19 know if anyone wants a break or if the doctor needs a  
20 break --

21 MR. MCGOWAN: Anytime is fine.

22 THE WITNESS: Yeah, it sounds like this  
23 is a watershed, but why don't I go shed some water.

24 MR. MCGOWAN: Sounds good.

25 THE VIDEOGRAPHER: Off the record at

1 9:58 a.m.

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

3 THE VIDEOGRAPHER: On the record at  
4 10:04 a.m.

5 BY MR. MCGOWAN:

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

11 A. Okay.

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

15 A. Not that I'm aware of.

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

21 A. Yeah, I couldn't find anything.

22 Q. Did you search for that kind of stuff?

23 A. Oh, yeah.

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



1 but there are just some questions I have.

2 A. Mm-hmm.

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

5 A. No, I wrote it myself.

6 Q. Okay.

7 A. I write semi-professionally.

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

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

12 A. Yeah.

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

15 A. Yeah.

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

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

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

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

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

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

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

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

11          A. Yeah.

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

15          A. Yeah.

16          Q. -- as being the beginning?

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

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

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

1 A. I thought that --

2 MR. KLOTZBUCHER: Objection. Form.

3 You may answer.

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

7 BY MR. MCGOWAN:

8 Q. Yeah.

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

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

16 (Over-speaking.)

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

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

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

1 diagnosed in 2012?

2 MR. KLOTZBUCHER: Objection.

3 You may answer.

4 BY MR. MCGOWAN:

5 Q. Even money, Doctor.

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

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

15 A. No.

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

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

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

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

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

6 Because we know he had an A1c of 8.6 in  
7 2013, and he was on -- when did we see that he was on  
8 glimepiride?

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

13 Q. Does it matter which occurred first --

14 A. Well, I mean --

15 Q. -- in your opinion?

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

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

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

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

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

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

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

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

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

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

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

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

17 A. Yeah.

18 Q. Sugar in the urine?

19 A. Yeah.

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

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

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

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

1 original -- and now I'm on page 5. It's "Dr. Leach  
2 noted a diagnosis of type 2 diabetes dating from  
3 2009."

4 Did you ever see the origin document of  
5 that?

6 A. No.

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

10 A. No.

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

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

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

23 A. We don't.

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



1                   That's not an insulin --

2           A.   No.

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

5           A.   No.

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

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

12          Q.   45 years ago now.

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

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

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

7 (Over-speaking.)

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

10 A. Yeah. Yeah.

11 Q. Does that have any impact on kidney function?

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

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

20 Q. The next paragraph down here --

21 A. Is this on 4 still?

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

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

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

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

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

5 A. Sitagliptin.

6 Q. Sitagliptin. What kind of drug of is?

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

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

18 A. Yeah, 10.7 is pretty high.

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

22 A. No.

23 Q. And you have to change the medical  
24 management.

25 And, in fact, his was ultimately changed

1 again. I believe he was put on insulin at some point?

2 A. Yeah. Soon thereafter.

3 I mean, this is -- this is a common scenario  
4 for somebody -- for an endocrinologist, is that the  
5 primary care doctors are uncomfortable with insulin,  
6 and they oftentimes don't have the resources to get  
7 people started on insulin, so they'll use pills, and  
8 they'll use -- the patient will come in and say,  
9 "Well, I know my A1c is high, Doc, but I'll follow my  
10 diet. This is going to get better." And that will go  
11 on for a while, and the doc will keep giving them  
12 pills and they need to be on insulin, and then they  
13 come to us, and we put them on insulin.

14 So this is not an uncommon scenario, to have  
15 a patient whose diabetes is being managed by primary  
16 care on pills that aren't effective.

17 Q. And probably every patient says, "I promise,  
18 Doctor, I'm going to eat better and I'm going to get  
19 more exercise"?

20 A. I've been fooled many times.

21 Q. Easy to say and hard to do. I get it.

22 All right. So it looks like -- I'm in the  
23 next paragraph now, 2018.

24 A. Yeah.

25 Q. Macular degeneration: Is that a complication

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

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

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

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

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

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

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

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

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

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

7 (Doctor's pager alerts.)

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

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

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

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

19 Q. Well, the more data that we have -- and this  
20 is on the next page, page 6. It says November of  
21 2019, his GFR was 29?

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

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

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

4             Q.   It looks like in June of 2022, his GFR was  
5     27?

6             A.   Yeah.

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

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

16            Q.   You got to draw the line somewhere?

17            A.   Yeah.

18            Q.   I understand.

19                    When does one go on dialysis?

20            A.   Usually it's -- once the eGFR gets under  
21    50 -- 15, you're starting to think about it.  But you  
22    wouldn't put somebody on a dialysis just for that  
23    number.  What you look for is are they having trouble  
24    excreting potassium.  So if the potassium is really  
25    high, that can be dangerous.  Are they having trouble



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

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

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

13 A. Yeah.

14 Q. Okay. How's that work?

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

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

1 170, 180. So just that amount of weight loss can  
2 improve your diabetes a lot.

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

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

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

15 A. Yeah.

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

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

18 Q. Yeah.

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

21 A. Yeah.

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

24 A. Uh-huh.

25 Q. (As read):

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

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

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

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

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

22 Q. I see.

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

1 to tell what the actual cause was.

2 Q. At this point?

3 A. At this point. This --

4 Q. Presumably --

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

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

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

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

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

21 BY MR. MCGOWAN:

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

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

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

6 But certainly 75 percent of people with  
7 type 2 diabetes die of heart disease, and it's 50 or  
8 60 percent of people with CKD die of heart disease.  
9 So both of those things go together with -- and he had  
10 ischemic heart disease. Now, he may -- they called it  
11 ischemic heart disease.

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

18 Q. You wrote (as read):

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

1 A. Yes.

2 Q. Tell me your basis for having a three-year --  
3 your basis for that statement.

4 A. My basis for that statement were, you know,  
5 some observational studies, both prospective and  
6 retrospective, that looked at kidney function in  
7 people after hematopoietic stem cells, and the people  
8 that got kidney disease got it in the first year, but  
9 I extended it to say, you know, it might go two or  
10 three years.

11 Q. Okay. So which study was that? Because --

12 A. This would be in the supplemental data that  
13 we sent to you. The -- what did we call that?

14 Q. Materials Considered list?

15 A. Yeah.

16 Q. We love to say that. It's fancy --

17 (Over-speaking) --

18 A. Yeah. So there's five papers listed there.  
19 One, I realize, I sent the wrong PMID number because  
20 it's totally irrelevant, it's about colon cancer. But  
21 there's some series, one from Fred Hutchinson in  
22 Seattle, that talk about this -- talk about kidney  
23 function and after stem cell therapy.

24 Q. So did you read those studies before you  
25 wrote the report? Or did you --

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

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

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

7                     We're on page 7?

8           Q. Yes, sir.

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

12          A. Yeah.

13          Q. Apparently that's bad for your kidneys?

14          A. Yes.

15          Q. How so?

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

20          Q. Excreted through the kidneys?

21          A. Yeah.

22          Q. Okay.

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

25          Q. So how much was he taking?

1 A. We don't know.

2 Q. Okay. So how do you know if it was enough to  
3 take a toll?

4 A. I didn't. I didn't say it took a toll.  
5 I just said it was something he did that was marked in  
6 his -- he had gout and he took NSAIDs, and it was  
7 remarked on in a couple of places in the chart.

8 So I don't know that -- I just know that he  
9 was taking NSAIDs, and that that is a risk factor for  
10 kidney disease.

11 Q. Well, you made a list (as read):

12 "Important among these" --

13 And you're talking about the risk factors  
14 for CKD?

15 A. Yeah.

16 Q. "Hyperuricemia" --

17 A. Yeah.

18 Q. (As read):

19 "... NSAIDs, dyslipidemia and  
20 atherosclerosis, obesity, and  
21 diabetes."

22 A. Yeah.

23 Q. Okay. It is your view that diabetes was the  
24 cause?

25 A. I mean, my whole point in listing all these



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

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

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

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

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

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

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

25 Q. So being fair about this, and acknowledging

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

4 A. Yeah.

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

11 A. Yeah, so I would --

12 MR. KLOTZBUCHER: Objection. Form.

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

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

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

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

1 15 percent, et cetera, but people that have those  
2 exposures have an increased risk of CKD.

3 BY MR. MCGOWAN:

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

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

10 Q. I'm on page 8.

11 A. Okay. Me too.

12 Q. Top of it says (as read):

13 "Susceptibility to end-organ  
14 complications varies among  
15 diabetic patients, with most of  
16 the variance determined by  
17 multiple genetic factors  
18 conferring different levels of  
19 susceptibility."

20 A. Yeah.

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

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

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

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

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

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

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

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

5 A. No question.

6 Q. I think Barry White died of that --  
7 (Over-speaking.)

8 A. Yeah.

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

10 A. Yeah.

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

13 A. All true.

14 Q. Okay. All right.

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

17 A. Okay.

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

19 A. Yeah. Terrific.

20 MR. MCGOWAN: Thanks.

21 THE VIDEOGRAPHER: Off the record at  
22 10:42 a.m.

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

24 THE VIDEOGRAPHER: On the record at  
25 10:57 a.m.

1 BY MR. MCGOWAN:

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

7 A. No.

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

10 THE WITNESS: Okay.

11 CROSS-EXAMINATION BY COUNSEL FOR THE DEFENDANTS

12 BY MR. KLOTZBUCHER:

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

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

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

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

1 A. Yes.

2 Q. -- or it's part of the patient's medical  
3 history?

4 A. Yes.

5 Q. Okay. Did you review the drugs Mr. Keller  
6 took when you reviewed his records? The --

7 A. Yeah.

8 Q. I'll rephrase. The drugs that he took  
9 regarding his NHL treatment?

10 (Court reporter asks for clarification.)

11 A. So I looked through the -- you know, I looked  
12 through -- in Walla Walla, Dr. Iacoboni gave him  
13 what's called CHOP -- C-H-O-P -- or CHOP-R --  
14 something like that -- and that's something I remember  
15 from my residency. They used to use CHOP. "CHOP till  
16 you drop," we used to say. And -- you know, so I know  
17 Cytosan and Vincristine and those.

18 And then I think he got Adriamycin and  
19 etoposide after his radiation. So I've heard of those  
20 drugs too.

21 Q. Did you review whether those drugs were toxic  
22 to the kidneys?

23 A. Yeah.

24 Q. Are those drugs toxic to the kidneys?

25 A. He got -- and one of the other experts wrote

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

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

7             MR. KLOTZBUCHER: Understood.

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

10            THE WITNESS: Yeah.

11            MR. MCGOWAN: Thanks, Doctor.

12            THE WITNESS: Yeah.

13            THE VIDEOGRAPHER: Off the record at  
14     11:00 a.m. This concludes the deposition of David  
15     D'Alessio.

16     (Whereupon, at 11:00 a.m., the deposition ceased.

17            Signature was reserved.)  
18  
19  
20  
21  
22  
23  
24  
25



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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Sophie Brock

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS

COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

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

Veritext Legal Solutions is committed to maintaining the confidentiality of client and witness information, in accordance with the regulations promulgated under the Health Insurance Portability and Accountability Act (HIPAA), as amended with respect to protected health information and the Gramm-Leach-Bliley Act, as amended, with respect to Personally Identifiable Information (PII). Physical transcripts and exhibits are managed under strict facility and personnel access controls. Electronic files of documents are stored in encrypted form and are transmitted in an encrypted

fashion to authenticated parties who are permitted to access the material. Our data is hosted in a Tier 4 SSAE 16 certified facility.

Veritext Legal Solutions complies with all federal and State regulations with respect to the provision of court reporting services, and maintains its neutrality and independence regardless of relationship or the financial outcome of any litigation. Veritext requires adherence to the foregoing professional and ethical standards from all of its subcontractors in their independent contractor agreements.

Inquiries about Veritext Legal Solutions' confidentiality and security policies and practices should be directed to Veritext's Client Services Associates indicated on the cover of this document or at [www.veritext.com](http://www.veritext.com).