Volume 2

Pages 226 - 370

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

Before The Honorable Vince Chhabria, Judge

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	)	**NO. 16-md-02741 VC**

San Francisco, California
Monday, February 4, 2019

TRANSCRIPT OF PROCEEDINGS

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I N D E X

Monday, February 4, 2019 - Volume 2

E X H I B I T S

Monday - February 4, 2019 .m.

P R O C E E D I N G S

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THE CLERK: Calling Case Number 16-MD-2741, In Re: Roundup Products Liability Litigation.

Counsel, please step forward and state your appearances for the record.

MS. WAGSTAFF: Good morning, Your Honor, Aimee Wagstaff. With me I have Jennifer Moore, Robin Greenwald, Brian Brake, Kathryn Forgie, Nadina Beach, and Tesfay Tsadik.

MR. STEKLOFF: Good morning, Your Honor. Brian Stekloff, and along with me are Rakesh Kilaru -- Ms. Tamarra Matthews Johnson is here today, but I don't see her -- and Michael Imbroscio.

THE COURT: Okay. Should we have a chat about Portier or should we do that after we are done with the witness today?

MS. WAGSTAFF: Whichever.

THE COURT: Why don't we have a chat about it now? So, you know, I don't remember the exact schedule that you all proposed for my residing over his trial testimony; and I don't know if Monsanto agrees to that procedure at all. I'm concerned that those times are not going to work for me, and so -- but let me ask first, is Monsanto amenable to that procedure?

MR. STEKLOFF: Yes, Your Honor, so with two caveats I

think in Plaintiffs' counsel's e-mail, they had asked for a 7-hour/5-hour split. We are hoping to agree on a 6-hour/6-hour split; and then with respect to the first day, which is the day of jury selection, we are amenable -- if jury selection is completed -- to starting the deposition that day. We just don't want the deposition of Dr. Portier in anyway to short-circuit the jury selection process.

THE COURT: You used a word there which was deposition, and I guess that -- I don't know if that's the right word to describe what the Plaintiffs are proposing. However, it does make me wonder if this should be treated as a deposition. So my first thought was could we use Portier's existing deposition testimony, and I suspect the answer to that is no because it was not given -- among other things, it was not given with the idea that there would be a phased trial; right?

MS. GREENWALD: Right. Not only that, but we don't ask the questions in the deposition. It is really Monsanto asking Dr. Portier the questions.

THE COURT: That often happens when a witness is unavailable, you use the deposition testimony even though it is primarily the Defendant who asks the question, right? So I think that begs the question. I assume the bigger deal is that it would be very hard to separate out specific -- sorry, causation -- well, his testimony is only is supposed to be

about causation; but my sense is that he brings into his opinion a lot of the stuff that I would not understand as strictly being about causation.

MS. GREENWALD: There are a lot of questions about European issues and about bands and lack of bands. That's right. There is a lot of that. And, also, it wasn't really a preservation deposition. I would say it would be unusual for that type of deposition to be trial testimony, and we didn't think of it as a deposition. We thought of it as trial testimony it is remote.

THE COURT: In any event, it struck me that it would be unlikely that --

MS. GREENWALD: Right.

THE COURT: -- that deposition could be used; but why not just do another deposition or quasi deposition where you are -- perhaps the Plaintiffs do get to start even though usually the -- you know, the opposite side starts in a deposition. Plaintiffs do get to start. The Defendants can cross-examine him. It's not with me presiding remotely. Much as I would love to go to Australia and in different -- if it were -- if my schedule were a little different, I would actually welcome the opportunity to go; but I can't do it right now.

Why not just have this be a deposition, and you -- you know, you lodge your objections as you would during the

deposition but you separate it out, you know, into the stuff that Portier would testify about in Phase 1 versus Phase 2 to the best of your ability, given the guidance that I have provided thus far? And then you can -- you can designate the portions of his deposition that you believe should be played at trial; and if there are any evidentiary issues I need to resolve either, you know, at the pretrial conference or after the pretrial conference, whatever, I can do that.

MS. GREENWALD: I mean, I would like to talk to my colleagues for a moment, Your Honor, maybe when we get a break. It sounds like that is something we can work out. Obviously, it is a lot to ask you to be present in short notice.

THE COURT: I would be happy to, but the schedule is also filled.

MS. GREENWALD: The time difference with Australia is such that we don't really have a lot of flexibility with time.

MR. STEKLOFF: I understand what you're saying. I think we would prefer for you to be involved because even though you have given us guidance -- on 13, for example -- I could see in the motion in limine context getting additional guidance on the phasing of causation versus liability issues. I think that those disputes will -- especially with Dr. Portier being the first witness -- come up frequently and often, and then I think could very much dictate strategy.

THE COURT: Maybe he shouldn't be the first witness.

You mean being the first witness in the sense that you are going to be taking that testimony before the rest of the trial begins or first witness in the sense that he is the first witness that the Plaintiffs want to put on?

MR. STEKLOFF: I think at least prior to Dr. Portier's health issue, my understanding was that he was going to be their first live witness. I don't know what their intention is now --

MS. GREENWALD: That's correct.

MR. STEKLOFF: -- with the videotape and when they want to play that.

THE COURT: That may need to change. This logistical issue may make it so that the Plaintiffs need to reorder their presentation because it -- you know, depending on -- I mean, if you are talking about, you know, having -- what was it, the third day of his testimony was going to be the day of jury selection?

MS. GREENWALD: No, the first day.

THE COURT: First day was going to be the day of jury selection. Then I think you will probably have to reorder your presentation because it may be that you are not able to sift through the testimony and figure out what you want to designate and get any evidentiary rulings from me on what is appropriately designated by the time you begin putting on your case. So you are probably going to need to change the order.

MS. GREENWALD: So one option -- maybe we can do this at a break, Your Honor -- we can talk with Monsanto's counsel -- is maybe move it up a little bit instead of starting -- if you are not going to preside anyway, we were thinking about your schedule. If we don't have to factor in your schedule, maybe we can move it earlier; and that would allow us the opportunity to do designations. In other words, if we moved it up a day or two -- maybe we can take a break and talk about it and address it again if that's okay.

THE COURT: Okay.

MR. STEKLOFF: You know, I think we should continue to talk on both sides, Your Honor. One alternative also might be -- and I don't know your schedule -- if you are not able to sit through and listen to 12 hours of testimony and preside on a live basis, there might be -- and hopefully the parties can minimize this -- the need to contact, Your Honor, to raise a particular issue. So that might be -- hearing that you are going to have schedule difficulties and understanding that -- that might be a alternative where at least if something that is so fundamental to whether it is Phase 1 or Phase 2, and it really is going to dictate the strategy of direct or cross for either party comes up, we can contact Your Honor to explain the issue to you and to get your guidance.

THE COURT: That sounds good.

MS. GREENWALD: We were planning on starting at

8:00 to accommodate the California difference. So 8:00 o'clock in the morning in Australia is 1:00 o'clock here. So that we would make sure we do it so it is normal hours and not the middle of the night for you.

THE COURT: That sounds good middle ground. Speaking of, you were talking about seven hours, five hours, six hours, six hours.

MS. GREENWALD: Right.

THE COURT: It seems like you -- this is a good time to talk about time limits for the trial because I'm not sure you are going to want to spend 12 hours on Dr. Portier's testimony. So what I'm looking at -- you know, I could potentially re-visit this at the pretrial conference after I have gotten into it more deeply, but I have now started to get into it and have started to read all the papers; and my tentative view is that the time limits should be 32 hours for each side; 32 hours of air time for each side. Usually we have about a 4 hours of air time each trial day. It is actually a little bit more than four hours, and we would be going four days per week. So on that, I will admit there is a little bit of reverse engineering here; but I also think that it -- you know, this is a reasonable amount of time for each side to put on its case; that 32 hours for each side will make for a four-week trial. And the idea would be that that would include opening statements and closing arguments, and it would be up to

each side to decide how much time to spend on opening statements and closing arguments.

As is always the case with civil trials, if I determine -- let's say a quarter of the way through the trial or something, if I determine that the parties are using their time efficiently; and I feel that I have squeezed them too much, I can give you extra time. I will say that -- of all my civil trials, that has happened once where I have given each side some extra time. But usually, I feel like my estimates are pretty right on; and the parties end up using less time than they have been given. So that's, you know -- like I said, we can have a further discussion about it at the pretrial conference, but the parties should operate under the assumption that they are getting 32 hours per side.

MS. WAGSTAFF: Are you considering that for both Phase 1 and Phase 2?

THE COURT: Yes.

MS. WAGSTAFF: Okay. And are you --

THE COURT: And I think it would be up to the parties -- I mean, we can talk more about this at the pretrial conference. If it makes sense to have separate time limits for Phase 1 and Phase 2, you know, we can talk about that; but I was kind of assuming that, again, it would be up to the parties how to decide their time between Phase 1 and Phase 2.

MS. WAGSTAFF: I will take your comments back to my

team, and we will be ready to talk about it on the 13th.

THE COURT: Okay.

MR. STEKLOFF: I think that was consistent with what we requested in the joint pretrial statement. So we are okay with the proposal that you just laid out.

THE COURT: Oh, my God. I hope I didn't give more time than what you requested. That is my honest assessment, at this stage anyway, of what I think would be appropriate.

MS. WAGSTAFF: One more question: Are you intending after we get a verdict on Phase 1, to start Phase 2 the next morning or do you anticipate a period of time between them or -- just in terms of preparation.

THE COURT: I don't anticipate a period of time between them. If there is a verdict on Phase 1 at 10:00 a.m., then I would expect, you know, you to begin your opening statements on Phase 2, you know, later that day.

MS. WAGSTAFF: Okay.

THE COURT: Okay. Shall we proceed?

MR. STEKLOFF: Yes, Your Honor. I think Dr. Nabhan is here, your Honor.

MS. WAGSTAFF: Yes.

THE COURT: Come on up, Dr. Nabhan.

CHADI NABHAN,

called as a witness for the Plaintiffs, having been duly sworn, testified as follows:

THE CLERK: For the record, please state your first and last name and spell both of them.

THE WITNESS: Chadi Nabhan, C-H-A-D-I, N-A-B-H-A-N.

THE CLERK: Thank you.

MR. STEKLOFF: May I proceed, your Honor?

THE COURT: You may.

CROSS-EXAMINATION

BY MR. STEKLOFF

Q. Good morning, Dr. Nabhan.

A. Good morning.

Q. Just as a housekeeping matter, I think you have your three reports in front of you; is that right?

A. Yes, I do.

Q. So you also have some binders in front of you, and binder volume 1 of 3 is labeled exhibits. So I'm going to just move into the record those three reports.

A. Yes.

THE COURT: Any objection?

MS. WAGSTAFF: No objection.

THE COURT: Admitted.

MR. STEKLOFF: So those are, for the record, the first tab is Exhibit 2000 is the Hardeman report; the second tab, 2001 is the Gebeyehou report; and the third tab, 2002 is the Stevick report.

(Defense Exhibits 2000, 2001, and 2002 received in

evidence)

BY MR. STEKLOFF

Q. Dr. Nabhan, you wrote all three of those reports yourself, correct?

A. I did.

Q. And you did those -- you previously have testified in a *Daubert* proceeding in this court, right?

A. I did.

Q. And you reviewed the Court's opinion at the -- that came out after that proceeding, correct?

A. I did.

Q. Including the opinion with respect to your -- the opinions you had offered at the general causation phase, right?

A. Yes.

Q. You had read that opinion prior to writing these three reports, correct?

A. Yes.

Q. Okay. So why don't we use, just as an example, the Hardeman report which is Exhibit 2000. You can use the version that you have in front of you.

A. Okay.

Q. And in that report starting on page 5, you include a discussion about exposure to pesticides at the bottom -- a paragraph at the bottom of that page, correct?

A. Yes, I do.

Q. And that discussion was also in your general causation report; do you recall that?

A. I don't recall it if it was exactly the same words, but I did obviously discuss that.

Q. You discussed that meta-analysis by Schinasi and Leon, correct?

A. It is amongst the other things I discussed. It was one of the things I discussed at the time.

Q. Correct. You are also aware that in that discussion there is a mistake, correct?

A. Yeah, the third line from the bottom it is the odds ratio for B-cell lymphoma and somehow it was DLBCL.

Q. So just to read that sentence, it says, this meta-analysis found in association between glyphosate and B-cell lymphoma with an odds ratio 2.0; and then puts the confidence interval, correct?

A. Yes.

Q. And that is in that paper, right?

A. It is in the meta-analysis.

Q. And then you go onto say, And this was the same odds ratio for DLBCL, correct?

A. Yes. In that meta-analysis they looked at the various types and the B-cell lymphoma amongst the non-Hodgkin's lymphoma has the same odds ratio. I wrote DLBCL but I actually meant B-cell lymphoma.

Q. In the prior section you said B-cell lymphoma, right?

A. Yes.

Q. So when you then said, And this was the same odds ratio for DLBCL, there was no odds ratio specific to DLBCL, correct?

A. Yes, this was inadvertent.

Q. Now if you turn to the next page of your report, page 6, you then go onto discuss the IARC classification, correct?

A. Yes, I do discuss it.

Q. That was also a part of your general causation opinions, right?

A. Again, amongst other things, yes.

Q. You go onto discuss the *McDuffie* paper. That was part of your general causation opinions, correct?

A. Yes.

Q. If you turn the page, you discuss *De Roos* 2003. That was also, among other things, part of your general causation discussion, correct?

A. Yes, some of the things I discussed then may be a little bit different than how I discussed it now. Yes, I discuss it back then and now.

Q. Same with *Eriksson*, correct?

A. Correct.

Q. And for *Eriksson* you cite the unadjusted odds ratio of 2.36, unadjusted for other pesticides, correct?

A. Correct.

Q. And that paper contains an odds ratio that is adjusted for other pesticides, correct?

A. Yes.

Q. And you did not include that in this report, right?

A. Right. This ***Eriksson*** paper is actually more important for the dose exposure and the response exposure. The ***Eriksson*** paper shows the more exposure you get, the more likely you are going to get non-Hodgkin's lymphoma. That is actually the critical part of the ***Eriksson*** paper. In addition to that, whenever you have a dose response, it actually overcomes the possibility of confounding factors causing a problem. In other words, if the confounders would actually reduce the odds ratio, then you wouldn't see the dose exposure or the response exposure.

Q. As I understand your explanation, Dr. Nabhan, that wasn't my question. My question was: You did not discuss the adjusted odds ratio in this report, right?

A. I didn't discuss it, no.

Q. We are going to talk a lot about the two days and the --

A. Sure.

Q. And here it is your testimony that you are -- despite what we just went over, you are not offering any general causation opinions, correct?

A. I'm not offering general causation, but obviously I have reviewed the literature extensively; and I have testified in

general causation. I'm very familiar with the literature, and it obviously factored in somehow in how I included Roundup as a possible risk in these patients.

Q. So your understanding of the general causation is relevant to your specific causation opinions?

A. It would have to be relevant because I'm trying to look at all the risk factors for each patient.

Q. Now, one thing I just want to put -- put up front is you have also specific causation opinions in other cases beyond the three that we are here today about, correct?

A. I have.

Q. In all of those cases you have used the same methodology, right?

A. I have used the same methodology which is a similar methodology which I have done in clinical practice as well as when I write peer-reviewed articles, and I have over 300 papers I have written solely pertaining critical methodology and how I analyze any case.

Q. You call that here in this context the differential diagnosis or differential etiology, correct?

A. Differential etiology, I think, is more appropriate because we are looking at the etiology and the causation, so differential etiology -- although I understand that sometimes from a legal term differential diagnosis is used well.

Q. I was going to ask that. Do you agree that differential

diagnosis is a legal term, correct?

A. Differential diagnosis from a clinician's standpoint, that's when you have somebody who presents to your clinic with symptoms; and you are trying to do the diagnosis, so you look at various possibilities that may present with similar symptomology. And differential etiology, you are trying to look at what the etiology of whatever that symptom may be, so you are looking at differential etiology. I understand that they maybe used synonymously here. I'm just trying to explain the differences from a clinician's standpoint. In these reports if you see differential diagnosis with reference to etiology, these are used synonymously from my standpoint.

Q. Just to be clear, differential diagnosis is a legal terminology from your standpoint, right?

A. That's not what I said. I said when you see them in these reports, it is used for legal terminology. But differential diagnosis is when any patient presents to a clinician with symptoms, through your mind you have to think of what might be causing these symptoms until you reach a diagnosis. So I think a differential etiology is probably more appropriate term when you look at causation and the etiology of these -- of what caused the lymphoma of these patients.

THE COURT: It sounds like the upshot is that the courts and the lawyers should not have started calling these differential diagnosis, and it was a mistake on the part of the

courts and lawyers; and we should stop doing this, and we should start calling it differential etiology because that's what it is.

THE WITNESS: I would never testify in this court what you should do. I wouldn't do that. I know better but maybe you should have consulted us.

THE COURT: That would make a good expert witness.

BY MR. STEKLOFF

Q. You talk about your clinical practice. Dr. Nabhan, it is true in your clinical practice you have never told a patient that his or her non-Hodgkin's lymphoma was caused by glyphosate or Roundup, correct?

A. This is true.

Q. Now, for all three of the Plaintiffs -- Mr. Hardeman, Ms. Stevick and Mr. Gebeyehou -- you have no tests to determine what level, if any, of glyphosate was in their system at the time of their cancer diagnoses, right?

A. I don't. None of these tests were performed on any of these Plaintiffs.

Q. And you are aware that for all three Plaintiffs, they stopped using Roundup -- they have testified that they have -- they have told you they stopped using Roundup years before their cancer diagnoses, correct?

A. Yes, several years, I mean, give and take. Each one was a little bit different.

Q. Okay. But all of them had clearly -- give or take years, they had stopped using Roundup; and then give or take years later, they had their NHL diagnoses, correct?

A. That's correct.

Q. And you would have serious doubts that the cause of an individual's lymphoma was glyphosate if there was not glyphosate in his or her system at the time of his cancer diagnosis, correct?

A. I don't know the answer to that. Again, I think when you look at the studies, many of these studies -- the epidemiologic literature that linked glyphosate to non-Hodgkin's lymphoma -- they actually did not test patients or look at serum levels or urine levels. You have to look at the evidence based on what is published in the epidemiologic literature. Much of this did not look at serum levels and actual concentration in patients that were found to have non-Hodgkin's lymphoma after being exposed.

Q. Okay. Dr. Nabhan, the big binder, the middle binder, is volume 2 of 3 and has some of your prior testimony.

A. Which one?

Q. The middle binder of the three. And if you go to the fourth tab, that's labeled 2035. Do you see that?

A. Yes.

Q. This is in a case *Adams versus Monsanto*, correct?

A. Yes.

Q. And this involved a Plaintiff named Ms. Gordon, correct?

A. Yes.

Q. And you were deposed in this case on November 15, 2018, right?

A. Correct.

Q. So I would like you to turn to page 188 of this deposition. If you want to look back at page 187 for the context, you are being asked in Ms. Gordon's case about the possibility of her glyphosate levels being zero at the time of her diagnosis, okay?

A. Which page? I'm sorry.

Q. Look at page 187 starting at line 9.

A. Okay. Yeah, I can see that.

Q. Okay. Now I want you to read page 188, lines 5 through 12. This was your testimony under oath, correct?

A. Yeah --

Q. You were asked: That is correct. If there is no presence of glyphosate if her body at the time she is diagnosed with lymphoma, you wouldn't be able to say that glyphosate was a cause of her lymphoma, correct?

A. Yeah, but that doesn't mean the serum. It could be exposing the skin. You can inhale it. We don't know -- all of the studies that look at how patients usually get Roundup glyphosate does not necessarily measure blood or serum. The level of exposure or how these patients are being exposed, it

varies. We don't know, sometimes it is aerial spray and sometimes people can inhale it or it can just be exposed on the skin. I mean, you asked me if it is in the blood or the serum. We don't know if these patients had any of this checked.

Q. I just want to read the question and answer and see if this was your answer. If there is no presence of glyphosate in her body, regardless of how she -- if whether it was through the skin or inhalation or otherwise -- if there is no presence of glyphosate in her body at the time she is diagnosed with lymphoma, you wouldn't be able to say that glyphosate was a cause of her lymphoma, correct? And your answer was: I would have serious doubts about it, yes. Right?

A. Right, but I need to explain. In her body at some point -- somebody will have the actual offending hazard in the body. It doesn't have to be necessarily -- at some point through a 20 year, you would have had glyphosate in the body. It may not necessarily happen the day when you are diagnosed. You could have had it ten years before. You could have had it five years before, and the damage has already been done. That's how I understood the question.

At some point through a 20 year of being exposed to a particular compound, you must have had the offending material in the body. It doesn't have to be the day you were diagnosed or the year before you were diagnosed. That, we don't know. At some point it was present in the body, and most of the

studies don't really look at that. Because you don't actually -- you can't look at thousands of patients and measure the serum for each particular person.

Q. You didn't give any of those explanations when you were asked about Ms. Gordon, right?

A. Nobody asked me to explain exactly what it is, but that's exactly what is implied.

Q. Okay. All right.

A. I mean, we give chemotherapy, the chemotherapy is in the blood, right? We don't always check the level of chemotherapy unless we need to, but it is present; and then it disappears from the body after you finish chemotherapy, but the possible toxicities that happened with chemotherapy years later occurred despite the fact that chemotherapy isn't in the blood. It is already gone. You were exposed to it.

At some point throughout your medical journey or whatever you were doing, you would be exposed to these compounds and material and if you test, you would find them. They just don't need to be tested the day of the diagnosis or the year before the diagnosis.

Q. Ms. Gordon also used Roundup for several years, right?

A. She did.

Q. Then you were asked if at the time of her cancer diagnosis glyphosate was not in her body, would you -- would you be able to say that glyphosate was a cause; and you said, I would have

serious doubts. That was your answer.

A. I'm explaining to you what I meant by the answer, and I'm explaining to you that at some point you would find the actual compound in the body if you look for it. Most people don't have a standardized test to look for it, and they have not and at some point throughout the journey you will see it. You just don't know when.

Q. Dr. Nabhan, let's talk about the two days and the ten days that you referenced earlier. For your methodology you are looking at two studies to determine whether there has been sufficient exposure to say that glyphosate is a risk factor for each of the three individuals, right?

A. That's not the only thing I looked for for my methodology. That is actually incorrect. Some of the studies I looked for for my methodology -- my methodology I looked at way more studies than just these two studies.

Q. Okay. You --

A. You ask me what I look for. This is just part of what I looked for.

Q. We can agree that part of what you looked for was how many days the three Plaintiffs had to be exposed to Roundup under the ***Eriksson*** study and the ***McDuffie*** study, correct?

A. So -- yes, I mean, this is part of what I looked for in my -- in determining my methodology, correct.

Q. One of those studies tells you two days per year, correct?

A. Yes.

Q. And one of those studies tells you ten lifetime days correct?

A. Correct. More, more than two days per year and more than ten days per lifetime.

Q. Okay. You would look to see for all three Plaintiffs whether his or her exposure fits within those criteria -- two days per year -- more than two days per year and/or more than ten lifetime days, correct?

A. So as part of the methodology when you are looking at glyphosate specifically, and you are ruling in or ruling out glyphosate, you look at the -- all epidemiologic literature that is part of it, not just these two studies. And then you look at the level of exposure as determined by previously published epidemiologic studies such as the ***Eriksson*** paper and the ***McDuffie*** paper. Both of them just give you an estimate. We don't know what the minimum exposure we need to have. We know if somebody is exposed more than two days per year or more than ten days per lifetime, their risk of developing non-Hodgkin's lymphoma doubles. So that is important.

Q. And so in that case you then ruled in glyphosate or Roundup as a potential contributing cause to each of the Plaintiffs' lymphomas, correct?

A. Part of the way to rule it in would have to be level of exposure that is compatible with what is published in the

epidemiologic literature. Yes, that was an important piece of the puzzle that I had to lock in. That's why I talked to them and read their depositions and looked at exposure history. That was not the only piece that I looked for. I want to make sure this is clear.

Q. I think you have said that several times now. I'm not saying it is the only piece. You looked for each of the three of them and then compared their exposure to whether it was more than two days year or more than ten lifetime days, correct?

A. Yes, and the three of them had substantially more than either cutoff of these two papers.

Q. Right. And then that would -- was what you needed to show that glyphosate as a risk factor was truly impacting the development of their diseases, correct?

A. Again, yes, part of what I needed. It is not the only thing that I needed. It was part of what I needed. You have to look at each case with other risk factors that they have and other things they have in order for me to determine the causation.

Q. Let's look at the same deposition we were just looking at, the *Adams* deposition, and turn to page 105, lines 8 through 22.

A. Which page?

Q. 105 of Exhibit 2035. Are you with me?

A. Yeah.

Q. You were asked: Okay. How -- what is the extent of the

exposure that someone has to have in order for you to determine that Roundup caused their cancer, not number of days but whatever you said, the magnitude of exposure or -- and your answer was: Yeah, to me, I have to see that the exposure in that particular patient that I'm assessing is in line with what is published in the epidemiologic literature such as the one that you showed me, so more than two days per year or more than ten days per lifetime. These are two particular aspects of the exposure that I will need to see in order for me to show that the risk factor is truly impacting the development of disease. That was your testimony, correct?

A. Yes, that's what I just said.

Q. Okay. And once you have made that determination -- if you don't believe there are other risk factors, then you can't dismiss Roundup as a risk factor or a substantial cause for a particular patient, right?

A. You can't dismiss it if somebody had a level of exposure, but you have to look at other risk factors for each particular patient because it may not be the only factor. There may be more than other factors. You have to look at every single contributing factor and determine which one probably the most substantial of each particular individual.

So in each particular case you look at all the risk factors that I have known over the years that they contribute to the development of non-Hodgkin's lymphoma -- through taking

cares of thousands of patients and through my clinical practice -- as well as -- including Roundup, and the reason Roundup is included in this is based on all of the epidemiologic literature we talked about and reviewed a couple years ago.

Q. Dr. Nabhan, if a patient fits within the epidemiological literature that you have said links Roundup to non-Hodgkin's lymphoma, and if there are not other risk factors, then you will not dismiss Roundup as a substantial contributing factor in a particular patient, right?

A. You can't dismiss it automatically. You have to look at each case individually, but yes, you cannot dismiss it automatically and ignore a risk factor. It is like, you know, if you have somebody with lung cancer and is a smoker. You can't dismiss smoking. You have to put it on the list because you know smoking causes lung cancer. You can't dismiss it, no.

Q. Okay. I want to be clear, then once it is in, right, it has been ruled in in your analysis, correct, if it fits within the epidemiological literature, correct?

A. Yes.

Q. If there are no other, what you would say, causative risk factors -- let's put aside age and gender.

A. I disagree that age causes cancer, but we can talk about that later.

Q. I know you disagree. That's why I'm putting that to the

side. Let's say there are no other, what you would call, causative risk factors, then you are going to say that Roundup was the substantial contributing factor in that individual's lymphoma?

A. I have to look at each individual case separately. I would rule it in, and I would have to look at each particular patient before I can rule it out, yes. I mean, it will be part of the -- you know, you are more inclusive. You have to be very inclusive in all of the cases. The hypothetical example that you provided would make me rule Roundup in, and then I will look at each particular patient to rule it out or keep it in.

THE COURT: Can I ask a follow-up question on that?

THE WITNESS: Sure.

THE COURT: Two things, first, try to slow down a little bit although I appreciate how fast you are going because it saves time; but it makes it harder for the court reporter. We are a team here. Try to slow down a little bit, but I think the question is: You have ruled Roundup in, okay, and then you are at the process of analyzing all the potential risk factors; and this person has exceeded your exposure threshold in terms of Roundup or glyphosate.

THE WITNESS: Okay.

THE COURT: And no other significant risk factors are present. The question is: At that point do you automatically

conclude that it is Roundup that is the substantial factor causing the NHL or is there some other analytical process you need to go through before reaching that conclusion, once you determine that that there is no other risk factor present?

THE WITNESS: Thanks, your Honor, for clarifying. As a clinician, I would never make any determination automatically. It is just not the way our clinician brain thinks. We have to be very analytical in each particular case. So that threshold that we talked about is important for me to rule it in, as you described. Then I go through the process of all other risk factors. If everything is ruled out completely, it will be very hard to ignore the possibility that it was a contributing factor to that particular patient.

It's, again, analogous to all other diseases that we are trained for, cardiac disease, other types of cancers. If a person develops a heart attack and you can't find any risk factor and there is hypertension, you may not be -- you know, you can't say that hypertension did not contribute to the development of heart disease; but I do think it is very critical to go through the analytical process to be convinced that not just only the particular compound met the minimum criteria that I believe as a clinician needs to be achieved; but it's very critical to rule all other factors; and I think a lot of time need to be spent making sure that none of these factors actually contributed to the development of

non-Hodgkin's lymphoma, and then I will have to use the additional literature, maybe the other experts that look at the toxicology data, at animal studies; and then I make my determination whether this is contributing or not.

THE COURT: I guess the question is: Once you have gone through your analysis and you have ruled out other risk factors, does that automatically mean -- I'm not talking about the case of smoking. I'm talking about in the case of Roundup -- once you have ruled out all the other risk factors, does that automatically mean in your opinion that Roundup will be deemed a substantial factor in causing the person's NHL?

THE WITNESS: I guess I'm just a little bit -- need clarification when you say automatically what it means. Again, as I think of things, I just don't necessarily just say, oh, for sure it is.

THE COURT: Right. What I mean is -- I don't mean automatic in the sense that you are not doing any thinking or analysis. I'm saying after you have done your thinking and analysis regarding the other risk factors and you have ruled out the other risk factors, is -- could there be a scenario where you have ruled out all the other risk factors but you haven't ruled out Roundup, but you would nonetheless conclude that you cannot conclude that Roundup is a substantial factor in causing the person's NHL? Does that make sense?

THE WITNESS: Yes. It makes sense, and it would be

very difficult to exclude Roundup at that point. I think it would be very difficult to be aware of a risk factor that I believe has a link to non-Hodgkin's lymphoma and somehow ignore that risk factor and say, Okay, I have ruled out all the risk factors; but I'm going to treat this particular risk factor differently. Despite my knowledge of the association, I'm just going to believe it's not related. So if I have done the analytical method appropriately and I ruled all of the other risk factors and this patient does have non-Hodgkin's lymphoma and there is evidence of the exposure and -- I would not rule it out. I would keep it in.

THE COURT: So stepping back from Roundup and NHL and just speaking generally, when you are conducting an analysis like this and you have got -- let's say you rule in five potential risk factors and then you rule out four of them.

THE WITNESS: Yes.

THE COURT: You are left with one potential risk factor. Does it matter how strong a risk factor that is before you conclude that that risk factor caused the disease?

THE WITNESS: Absolutely. I think they have to be evidence that I'm convinced with it to start with. In order for me to even put these five risk factors in, you know, you have to be convinced as a clinician that all of these factors have evidence in the literature that they should belong in that big basket that you are putting in.

THE COURT: But I -- and I understand that, but I would assume -- and maybe this is -- maybe I'm assuming this incorrectly -- I would assume that you put in risk factors --

THE WITNESS: Right.

THE COURT: -- at the outset and some -- some risk factors are stronger than others.

THE WITNESS: Absolutely.

THE COURT: For example, you get lung cancer from a variety of different things working in an asbestos mine, smoking and then some lesser causes; and you might rule all of those in at the front end when you are looking at a patient's history. Am I right about that?

THE WITNESS: Sure. You look at the weight of the evidence and rule things out.

THE COURT: So if you -- if you rule out a bunch of stuff and you leave one -- but there is one risk factor remaining, I assume that you have to ask before concluding that this is the factor that caused the person's cancer, you would have to ask how strong is this risk factor?

THE WITNESS: Yes, you have to look at --

THE COURT: How strong is the evidence that this -- that this exposure causes this particular cancer.

THE WITNESS: Absolutely. You have to look at the general evidence about this particular risk factor, and you have to be convinced that the evidence is strong enough for you

to keep it in despite the epidemiologic threshold that we talked about. I agree with that a hundred percent.

THE COURT: Does that mean in this context -- coming back to the Roundup/NHL context -- that whenever a patient has been exposed to Roundup above the threshold that you have identified and whenever you have ruled out all the other risk factors, that you would never conclude that we don't know what caused this person's NHL? You would always conclude that it was Roundup?

THE WITNESS: So it is my opinion that the evidence that links Roundup to non-Hodgkin's lymphoma is strong to start with, and I obviously recognize that there are other folks in this courtroom that disagree with me; and that's why we are here. But the premise -- and it is my opinion that the evidence that links Roundup glyphosate to non-Hodgkin's lymphoma is actually strong; and I -- as you know, your Honor, I have looked at the literature and I have reviewed the literature and I have testified to the literature including some papers that did control for other pesticide exposure, so *De Roos* 2003, and we have talked about this previously. So my opinion is that the evidence is actually strong between non-Hodgkin's lymphoma and Roundup.

THE COURT: And so therefore your answer is yes, that in that hypothetical scenario that I spun out, that you would always conclude that the Roundup caused the NHL?

THE WITNESS: If I ruled out all the risk factors and I believe -- as I told you in my opinion, that the evidence is strong -- in my opinion as a clinician it would be inappropriate to ignore a risk factor that I know is strong and I know the literature supports it in my opinion; and I'm more than happy to go through all the literature again; but, again, I believe the literature is strong with this. The link does exist, and the risk factor -- to ignore such a risk factor as a clinician would be inappropriate in my opinion from a clinical standpoint.

Again, not to keep comparing other diseases; but, you know, there are lung cancers that occur in non-smokers. We know that. Not every person who gets lung cancer is actually a smoker; but if you have somebody who smokes, it would be hard-pressed as a clinician to say, okay, I know you smoke; but I still think your lung cancer is idiopathic.

THE COURT: But the evidence between the link of smoking and lung cancer -- I assume you would agree -- is much stronger than the evidence of the link between Roundup and NHL.

THE WITNESS: I would think 30 years ago that evidence was not strong. In 2019 I think you are very correct; that the evidence is strong. If we go back to 30 years ago, there were a lot of people who would disagree with you; and doctors were actually smoking in the hospital in medical rounds. We have pictures of that. So that may be accurate today and -- but if

you had said that to somebody 30 years ago, they would have laughed at us. They would say there was no evidence of that.

You know, it is my opinion that the evidence actually is very strong between non-Hodgkin's lymphoma and Roundup; and as a clinician, I cannot ignore that. And, again, I recognize that we may disagree on some of these things. Some people may say there is evidence but the evidence is weak, and some people may say there is evidence and the evidence is strong. I belong in the camp that says the evidence is strong, and I promise you 30 years ago, many of us did not think that smoking was linked to any cancer; and it took a lot until now -- nobody even disputes that.

THE COURT: So as part of your -- I mean, this is not so much of a question to you as a comment that I will need to hash out with the lawyers later -- but I'm thinking about somebody who either has not offered a general causation opinion or somebody whose general causation opinion has been excluded, you know, there is a going to be a question about how that -- how the specific causation opinion is presented to the jury.

I assume what it will be for someone like Dr. Nabhan, assuming he testifies, is that he is adopting an assumption based on the testimony presented by the general causation experts that there is a link and that the link is strong, right?

And with the specific causation experts, you are not going

to be -- I think you are not going to be getting into -- I don't know. This is more of a question than a statement, but I think you are not going to be getting too much into how strong the link is. They are going to have to borrow that from the general causation experts. Does that sound about right?

MS. WAGSTAFF: Yeah. So our three specific causation experts that we have proffered fit into three different categories; right. We had Dr. Weisenburger who passed through general causation. We had Dr. Nabhan who actually reviewed all the literature and has the knowledge base but for X, Y or Z reasons wasn't able to give a general causation and Dr. Shustov, who didn't participate at all. Absolutely with respect to Dr. Shustov, what you just said, and also with respect to Dr. Nabhan to the extent they don't ask him about it. I mean, his knowledge base -- you can't erase his mind from what he knew and what -- you know, the studies and the testimony that he previously gave, but we will --

THE COURT: But the way they would ask it is: You are not here today to give an opinion on how strong the connection is between glyphosate and NHL. You are adopting an assumption based on the general causation testimony that there is a strong link between the two. That's how they would ask the question to avoid getting into it with that witness; right?

MS. WAGSTAFF: Yeah. I mean, if that's the way Monsanto presents the question, then, yeah, that's the way he

would probably respond according to your orders. I mean, the whole bifurcation system that we have set up has sort of created some wacky testimony issues that we will have to work through with you. He is a unique witness because he did participate in Phase 1. He does have all the knowledge base. He was, for example, just using the Andreotti 2018 paper, the AHS paper, which is going to be central to Monsanto's defense, I assume. He presented his only expert report on that. He was deposed on that individually. You know, your Honor, didn't necessarily say he was struck in total about that; but if that comes up in a specific causation testimony, how are we supposed to handle that? I mean, this is some stuff that we are hoping --

THE COURT: I assume that's how -- I don't know if this is the subject of a motion in limine or not. I assume that's how it would be handled with somebody who is not testifying on general causation; right. They would be adopting the assumption that there is a link. They would be adopting the assumption that it is strong, but it will be up to the general causation witnesses to testify that there is a link and explain why it is strong.

MS. WAGSTAFF: Yeah, sure, absolutely. However, you know, on cross-examination if he is asked a question and that is part of his answer; that he has reviewed that study and he has criticisms of that study or why did he use *McDuffie* and

Eriksson, which he used dose response and AHS also uses dose response, he needs to be able to explain his opinion of the AHS study and why he weighted certain things in different ways; and, of course, he has a superior knowledge base of that because he has been participating in Phase 1 and Phase 2.

THE COURT: That is not an issue we have to hash out now. It is certainly an issue that is hanging over all of the causation testimony that we will need to deal with. Okay. Sorry. You can continue.

MR. STEKLOFF: Thank you, your Honor.

BY MR. STEKLOFF

Q. Well, Dr. Nabhan, using the discussion you had with, his Honor, you have a hundred Plaintiffs who you rule in glyphosate because it meets the exposure threshold; and then you look through all their medical records and you find no other -- what you would call -- causative risk factors, the conclusion here is for all one hundred, you wouldn't be saying Well, some of them are idiopathic. You would say for all one hundred, it was a -- glyphosate or Roundup was a substantial contributing factor in all of their cases, correct?

A. Idiopathic by definition, it means you failed to find any cause. Idiopathic is not a cause. Idiopathic by definition, it is a term us physicians like to use just to sound smart. Whenever we say something is idiopathic, it means we have looked at every single cause under the sun; and somehow we

failed to find the cause. I understand you have to always rule out idiopathy. When we say we rule out idiopathy, that means we didn't find any cause. That is really what it means.

If you have an actual cause that you know is linked to a particular disease, to ignore that cause and say, I still believe that this particular disease is caused by something I don't know -- although I have something I know that causes it -- how could this be done from a clinical standpoint? As a clinician, when you are sitting in front of a patient, if you already know a particular compound causes a problem, can you actually look that patient in the eye and say, Despite my knowledge that this may have caused your cancer, I still think your cancer has no known cause.

THE COURT: So before he pushes back on that answer, let me make sure I understand the answer. So to use the terms that I was using earlier, you rule in five risk factors, okay; and then you go through the process of analyzing each risk factor and deciding which ones to rule out. Are you -- it sounds like what you are saying is you would never conclude that it is idiopathic unless you rule out all five of those risk factors? As long as one risk factor is present, you would always conclude that it is not idiopathic; is that correct?

A. Idiopathic by definition means that as a researcher or as a clinician, you were unable to find any cause for this particular disease. It is synonomous of someone age 35 having

a heart attack and there is absolutely no reason for it, and you looked hard and you can't find the reason. Sometimes we don't know why a particular disease occurs. And if that's the case, we call it idiopathic.

THE COURT: But in the heart attack example -- like somebody is 35 and has a heart attack -- might there be a scenario where you say, Well, you know, you were -- you know, you smoked half a pack a day for five years in your 20s; and, you know, you -- you have, you know, six drinks a week and, you know, you don't get quite as much exercise as you should; and you are 10 pounds overweight. So there are any number of things that might have caused your heart attack, but there is nothing that really -- and those are all risk factors for a heart attack, but there is nothing that really jumps out. So I have to tell you we just have no idea how this heart attack was caused.

THE WITNESS: That is a good question but that's not idiopathic. You have already identified various risk factors that this person has. He is an ex-smoker. He did not exercise. He has weight gain, and it is possible that maybe none of them by themself caused the heart attack but maybe combined they actually led to this person having a heart attack in their 30s. I have had my share of younger persons who have diseases. You have already identified all of these risk factors.

Idiopathic in that example would be somebody who is in their late 30s, who exercises, who is fit, who is not overweight at all, who has never smoked, who has no family history of heart attack, that is idiopathic. That is when you look and you find nothing; but in the example you provide, your Honor, it is not idiopathic. You have enough risk factors in there that you cannot ignore.

THE COURT: Forgetting for the moment about the word idiopathic and just using the example that I gave you, are -- in that example, might you say to a patient, look, there are a number of risk factors that could be at play here; but there is no one that jumps out in particular and so we cannot attribute your heart attack to any one of these risk factors?

THE WITNESS: Yeah.

THE COURT: We just don't know.

THE WITNESS: It is possible. You can say maybe none of them maybe jumps as the most offending agent or risk factor that causes your heart attack, but maybe all of them together they led to this. There is no question before that person in the recovery, you are going to tell them to exercise and stop smoking and lose weight because in your mind as a clinician you know this somehow led to this disease. You are correct. It is possible that none of them jump at you as substantially really causing it, but collectively all of this together was risk factors.

THE COURT: So would you always conclude that collectively those factors caused the disease? I mean, maybe the answer is that this is kind of an artificial discussion because when you are treating a patient, you are not trying to figure out what caused the disease. You are trying to figure out how to prevent --

THE WITNESS: Absolutely.

THE COURT: -- prevent a recurrence of it, right; but would you ever say, you know -- would you always say, Well, it must have been all of these things in combination that caused your heart attack? Would you say it that way or would you say we just don't know? Might have been these risk factors in combination that caused your heart attack. Might have been something else. We just don't know. All we know is these are risk factors, so you should change your behaviors with respect to these risk factors.

THE WITNESS: Right. We know the risk factors are there. We just don't know. That's what I was trying to say. We just don't know means that I have looked at everything, and I found nothing. That's when we say, We just don't know. I don't know what happened.

But the -- in this example that we are talking about, we know that each of these risk factors by themselves could cause the cardiac disease. We just don't believe you had any of them long enough or you have done any of them substantially for me

to attribute that this is only the reason, but it is possible that all of these risk factors collectively led to your heart attack. Again, I think maybe, you know, the terminology -- as a physician, when we talk about idiopathic, it means you have done everything possible in your power as a clinician and reviewed the literature and you can't find the reason.

It is when somebody goes to the doctor with a skin rash, you know, they are going to go through everything in their mind. Maybe you have an allergy. Maybe you have eczema, whatever it is. They go through everything possible to know what the skin rash is from. If they can't find the reason why you had the skin rash, we say, I really don't know why you have the skin rash. Let me just give you hydrocortisone cream to stop the itching, and the rash will go away. This just happened to my dad last week. The doctor had no idea why it happened. That is idiopathic. It is a rash that we don't know why it is. Yes, I do look at the possibility of idiopathic.

I think what probably is important as a clinician and researcher is is it possible that maybe other factors in the future that we will find out that we don't know about today in February 2019, that in five years from now, we may find that there are other things that may lead to the development of non-Hodgkin's lymphoma; and if we are having this conversation again, we would rule in other risk factors that today we are not aware of; but, I mean, as an example, there are many things

that we rule in now that we weren't ruling in ten years ago. Our knowledge is expanding. We know a little bit more today thankfully than we knew ten years ago or twenty years ago.

So maybe five years from now, you can't -- you can't rule it in or rule it out because now you have more stuff that you put in the basket because we knew more about what may cause a particular disease. Did I answer your question, your Honor?

THE COURT: Yes, thank you.

BY MR. STEKLOFF

Q. Dr. Nabhan, going back to my hypothetical, you have a hundred patients -- patients or Plaintiffs and glyphosate -- they exceed two days per year and ten lifetime days of Roundup use. So you have ruled in glyphosate. You find no other causative risk factors -- so no viruses or HIV or radiation, nothing that you would say is causative -- for all one hundred, you would say that Roundup was more likely than not a substantial contributing factor in the development of his or her lymphoma, correct?

A. Without looking at a particular case, I'm going to have to say correct. But, again, I just want to reserve the issue that you always have to look at each patient, each medical records; talk to each patient. It is just not -- you make it sound so simple, and it is just not a simple process as you describe.

Q. Okay. Now you agree that every methodology has an error rate, right?

A. Every methodology has an error rate, yeah, talking statistics. Not every methodology is a statistical test. Statistics have error rate, alpha error rate and meta error rate, yes. You have to explain to me what you mean by that because I was asked about this question in my deposition about error rate previously, and I can explain to you what I assume you mean.

Q. Well, your methodology has an error rate, right? Out of those hundred, you might be wrong about some of them, right?

A. Do you mind repeating the question, please?

Q. Sure. Your methodology has an error rate, correct?

A. The error rates exist in statistical tests. I mean, again, so -- you know, the methodology that I applied was not a statistical test. It is a simple thing that we actually teach residents and fellows and students and we are -- we learn about this in medical school. When you sit in front of a patient and you try to determine causation, you look at all risk factors that you can think about that you have learned about, that you read about; and then you do the process of elimination. You start looking at each particular individual risk factor. That is the methodology. It is not a statistical test that we are doing. It is a differential etiology. It is not statistics.

Q. Let me ask it a different way then. You agree out of those hundred patients that we just agreed upon, you might be wrong about some of them; that glyphosate wasn't the -- the

risk factor that caused their lymphomas, correct?

A. You can never be a hundred percent certain of anything, but you have to exercise your clinical judgment. At the same time you can't really ignore, you know, the -- again, it's -- it's -- I don't want to keep bringing up examples; but you could have a smoker who gets lung cancer not from smoking, right? You can make an argument that -- we just said -- not every lung cancer is caused by tobacco smoking. The reality is when you have a smoker, you are going to say this is probably smoking related because it is a risk factor. Is it possible that this lung cancer patient may have had, for whatever reason, lung cancer even if he wasn't smoking? No one really a hundred percent knows. You are asking for a hundred percent certainty. All I can tell is more likely than not. All I can tell you is with high degree of medical probability that I have always done. That is the best I can tell you.

Q. That smoking analogy that you just used, you are applying the same reasoning here? In other words, smoking is likely -- you have someone who smokes --

A. You have a hundred smokers --

Q. Yes.

A. A hundred of them get lung cancer. How many of these hundred are you going to tell them, I don't think it is smoking related? If you bring a hundred oncologists today, and you face them with a hundred patients -- all of them are smokers --

how many oncologists will say, I don't think smoking was causing the lung cancer? That is what you are asking for a hundred percent. What I can tell you is the majority of oncologists will say that it is smoking related. That is what we operate on; right.

Q. My question is: Using that smoking analogy, you are doing the exact same thing here with the glyphosate or Roundup and non-Hodgkin's lymphoma, correct?

A. So in that analogy, you still have to put the other risk factors for lung cancer. As your Honor just mentioned, asbestos and other things, for example. Again, you do the same thing. In the methodology we are talking about, you have to rule in all risk factors that you know has potential cause for this particular disease; and then you start the process of elimination by looking at each particular individual risk factor.

Some risk factors are very weak. Such as, for example, when somebody tells me age causes cancer, no, it just -- cancer is more prevalent in older people. It doesn't cause it necessarily. We just happen to have cancer in older patients. It doesn't mean age causes it directly and some of them are stronger. That is how we start the process of elimination until you are left with either nothing and then it is idiopathic, and you don't know the cause or you are left with one reason; and then it's the contributing factor or you may be

left with three reasons, and then you have to decide whether one of them was more contributing than the other two or the three of them were contributing at the same rate or whatever.

That's how -- that's the methodology we do of differential etiology. It is something we are taught in medical school, and we teach the new generation of how to do it if they are looking at causation or etiology. The problem is unfortunately a lot of people are more focused on treatment, which is appropriate.

A lot of times when we are dealing with cancer, and a patient comes in and really just want to get the treatment and have the plan proceed. There are times when physicians, clinicians are guilty as charged by not spending enough time trying to investigate causation or etiology for particular cancers.

Q. Right. You and I met before, and you told me you have never told a fellow oncologist or pathologist that he or she should -- that you believe glyphosate or Roundup is a cause a general cause of lymphoma, correct?

A. I have never said that, no.

Q. You have never said that to a medical student, correct?

A. I have never said that in public. I actually didn't know that I could say that because I thought in litigation practice I'm not allowed to say that. I guess that tells you how much I know about the law.

Q. You said that in open court, right?

A. I understand, but I just didn't know this is something I can go and tell other people so but I have not, correct.

Q. Okay. So let's talk about other risk factors in the process that you just described. Just because a patient has a separate risk factor from glyphosate or Roundup, that in your methodology doesn't eliminate glyphosate or Roundup, correct?

A. You put everything in, but some people might have other risk factors that are more pressing than Roundup.

Q. Let's just use active hepatitis C as an example, okay?

A. Define active hepatitis C so we make sure we are talking about the same thing.

Q. Sure. Hepatitis C that the virus is active in the bloodstream at the time of the cancer diagnosis.

A. If the virus is still present in the blood at the time of lymphoma diagnosis?

Q. Correct. It hasn't been treated yet. We are not talking about Mr. Hardeman who we will talk about later.

A. Sure.

Q. Even in that situation, you would say that you couldn't rule out glyphosate or Roundup because you could have multiple causes of lymphoma, correct?

A. You may not be able to rule it out, but you may be convinced that hepatitis C is more important factor than the Roundup in that situation. Again, that's where the issue is. If you have somebody with active hepatitis C and is using

Roundup, it is possible -- again, depending on each case -- it is possible in this particular scenario that the hepatitis C itself was more contributing or equally contributing or both of them contributing, and you really can't assign a weight to either one of them. It is possible you may not be able to tell which one is more.

Q. Right. You would, in this hypothetical, still say that Roundup was a substantial contributing factor to the development of the patient's lymphoma, correct?

A. You could still say it is probably a substantial factor; but, again, you can't get out -- you have hepatitis C now that is active of this particular patient, so hepatitis C is obviously another substantial contributing factor for this particular patient. It is active, and it is not treated and it is replicating in the bloodstream.

Q. You would say they were both substantial contributing factors, correct?

A. You could say both. It is possible that one of them be more pressing than the other depending on the exposure history, depending on how many years was exposed, how many days, how many hours in the day, and the hepatitis C situation, how long hepatitis C was in the body, the viral load, the RNA load, all of these things. Was there really any evidence of organ damage with the hepatitis C? Is the cirrhosis advanced? Is the cirrhosis not advanced?

You have to look -- I understand the hypothetical piece, but it is very difficult without knowing the details of the exposure in that hypothetical example and the details of the hepatitis C because not every hepatitis C is created equal and not every exposure is created equal. Again, both of them will be in the basket. They are ruled in. You have to decide as a clinician on each particular case, each particular risk factor, the hep C and the Roundup which one, if any, was more pressing than the other.

Q. But under your methodology using active hepatitis C or using HIV positivity, you could -- it is your methodology that we could have two or three causative factors of an individual patient's lymphoma including glyphosate; correct?

A. You could have more than one causative factor, correct. In a particular patient, you could have more than one cause.

Q. Okay. Now let's talk about the three individual plaintiffs.

First of all, for all three you agree that absent exposure to glyphosate, they could have developed the same non-Hodgkins lymphoma; correct?

A. Yes, they could. We -- anybody can develop non-Hodgkin lymphoma.

Q. And in -- now, specifically talking about the three plaintiffs, so we're not talking about hypotheticals, for each of the three plaintiffs, had they never been exposed to

Roundup, you would say that their non-Hodgkins lymphoma was idiopathic; is that correct?

A. You want to go with each one?

Q. Sure. Let's start with -- and maybe Mr. Hardeman is different so let's go backwards. Let's start with Mr. Gebeyehou.

If Mr. Gebeyehou had never been exposed to Roundup and you've now looked at all of his medical records and his risk factors, you would say that his non-Hodgkin's lymphoma was idiopathic; correct?

A. I would say we don't know the cause. I mean, again, he was diagnosed at an older age and it's common -- again, patients get diagnosed usually with this disease in a median age of 68 to 70. So I would say that if he was diagnosed with a non-Hodgkin lymphoma, that I don't know exactly what caused it.

Q. Okay.

A. Which most non-Hodgkin lymphoma, I said that many times before, the majority of non-Hodgkin lymphoma that are diagnosed are of unknown cause. We have 75,000 new non-Hodgkin lymphoma a year, the majority of which are of unidentifiable cause. Some of them we know the cause. In the majority we don't.

Q. But so the answer to my question was yes; right?

A. Yes.

Q. Okay. Now, let's use --

A. Sometimes I need to explain what I mean because, I mean, you've showed me a couple of things that were not clear what I meant.

Q. I think we can tell you like to explain what you mean.

Let's talk about Ms. Stevick. Ms. Stevick, same history but never exposed to Roundup, you would say that her lymphoma was idiopathic; correct?

A. Yes.

Q. Okay. Now, Mr. Hardeman, and you tell me, would you say that his lymphoma was idiopathic or would you say that his lymphoma was caused by his hepatitis C exposure?

A. I don't believe his lymphoma was caused by hepatitis C exposure. He did not have active hepatitis C. There was no virus in his blood whatsoever for eight, nine years prior to the diagnosis so it's different than the hypothetical example that you provided me.

So, again, it would be -- in my opinion, hepatitis C did not cause his non-Hodgkin lymphoma; and if it did and there is something that, again, it may have contributed, it would be a very minor contribution of that just because of his particular scenario.

Q. So in Mr. Hardeman's case, then, you would say the same thing as you said with Mr. Gebeyehou and Ms. Stevick; had he never been exposed to Roundup, the cause of his lymphoma was idiopathic?

A. I believe so, yes.

Q. Okay. So you have also offered opinions in a case involving a plaintiff called Jeff Hall; correct?

A. Yes.

Q. Okay. And so can you look at, we're shifting topics a little bit, the first binder with the exhibits?

THE COURT: Could I ask you a question?

MR. STEKLOFF: Oh.

THE COURT: You a question.

MR. STEKLOFF: Yes, of course.

THE COURT: Are you off of hep C as it relates to Mr. Hardeman?

MR. STEKLOFF: I was going to come back to that in a moment.

THE COURT: Okay.

BY MR. STEKLOFF:

Q. Okay. In --

A. Which volume?

Q. In Volume 1, Exhibit 2010. So you have to go in a little bit.

MS. WAGSTAFF: Did you say Volume 1?

MR. STEKLOFF: Yes.

THE WITNESS: Yes, 2010. I see that.

BY MR. STEKLOFF:

Q. Okay. And this was a plaintiffs' disclosure of expert

witnesses in that case. It involved at the time two plaintiffs, Ronald Peterson and Jeff Hall; correct?

A. Correct.

Q. And this wasn't a document that you drafted but you can --

MR. STEKLOFF: Well, first of all, Your Honor, I would move *Daubert* Exhibit 2010 into evidence.

THE COURT: Any objection?

MS. WAGSTAFF: Hang on. Let me -- just one second.

(Pause in proceedings.)

MS. WAGSTAFF: Your Honor, we would ask for a proffer of relevance as this is for a state court case.

THE COURT: Why don't you ask him about it --

MR. STEKLOFF: Sure.

THE COURT: -- and then you can move to admit it after you're done asking him about it.

MR. STEKLOFF: No problem.

Q. You've seen this before; correct, Dr. Nabhan?

A. It's been a while. I mean, I have seen it at some point, but I haven't seen it recently.

Q. Okay. And this disclosed on page 1 that you would be an expert in this case; correct?

A. Yes.

Q. And then if you turn to page 2 about six lines down, it summarized what your opinions in that case would be. It said (reading):

"He will explain his opinions regarding general causation that Roundup exposure can cause non-Hodgkin lymphoma and specific causation that Roundup use was a causal factor to Mr. Peterson's and Mr. Hall's lymphomas." Correct?

A. Correct.

Q. And with respect to Mr. Hall, that was true at the time that -- well, let's look at the last page.

The last page puts the date that this was disclosed of March 1st, 2018; correct?

A. (Witness examines document.)

Q. Under the Certificate of Service, do you see the date March 1st, 2018?

A. Yes, I do.

Q. Okay. And so as of that date, March 1st, 2018, it was true that you were going to say that Roundup use was a causal factor to Mr. Hall's lymphoma; correct?

A. Yes, and I recall this actually came up during my deposition. I've had -- I mean, prior to this, we -- I -- the medical record and the exposure history and all of the history pertaining to this particular patient were reviewed with counsel that retained me with this firm. So we've talked actually about all the things pertaining to this particular case prior to me meeting this patient, and so forth.

So I was aware of a lot of things pertaining to this

particular individual as I explained in my deposition in this case. Because a lot of times if I get asked to look into a case, I have a lot of questions that I ask even before anything is sent or reviewed in terms of, you know, how old is the patient, what type of lymphoma did they have, tell me about the exposure history, what are the comorbidities, what is the medical history, what type of lymphoma it is, what type of treatment they had, and so forth and so forth. I recall this came up and we clarified it during the deposition.

Q. All right. You're anticipating where I'm going so let's just break that down and walk through the history.

As of March 1st, 2018, you had not yet reviewed any of this plaintiff's medical records; correct?

A. I don't believe I had the actual medical -- physical medical record at the time, if my memory serves me right. I believe that we went through everything pertaining to the case that I needed to know that are very important and very relevant.

It's like when somebody calls you as a clinician for a second opinion, and I've had my share of second opinions when patients call me or when other physicians call me, they call me and they say, "I'm seeing this patient. This is the history. This is the treatment. This is X, Y, and Z."

And you go over all of these things and say, "Okay. You know, I would like to meet this patient. I would like to

review additional records. I would like to take a look at additional information pertaining to this patient."

Q. Okay.

A. So kind of analogous to that.

Q. Right. And just to be clear, when you say "we had," "we went through everything in the case," the "we" is you and counsel for Mr. Hall; correct?

A. Yeah. I was asked to take a look at this case, and I asked a lot of questions pertaining to this case because, again, there are situations where I was asked to look at patients and I said, "I don't believe that this particular individual has a lymphoma that is related to Roundup."

So there's really no point for me to review 4,000 pages of medical records if I already determine prior to the review that there is no causation between that Roundup and this particular lymphoma. So that's -- I mean, there are patients I've looked at that I determined their lymphoma is not related to Roundup.

Q. Just to be clear, Dr. Nabhan, you spoke to plaintiffs' counsel and then based on that, you were willing to opine that Roundup was a substantial contributing factor in Mr. Hall's lymphoma, and you made that determination prior to reviewing any medical records or any other court original documents in the case; correct?

A. I made that determination with the premise I'm going to still review the additional records. However, again, it's not

that I had a two-minute phone call conversation and this is how it happened. That is actually not -- the way you're portraying it is incorrect. I've had several phone call conversations pertaining to this particular case where I asked --

THE COURT: Dr. Nabhan, can I interrupt you for a second?

This line of questioning seems like a complete waste of time as far as I can tell. So can you try to -- whatever, like, small point you want to get out of it, go ahead and then move on?

MR. STEKLOFF: If he had said "yes" to that question, I was ready to move on. Can I -- nonetheless, I'll just for the record move Exhibit 2010 into the record.

THE COURT: That's fine. I kind of doubt it would be admissible at trial.

MR. STEKLOFF: Oh, I agree.

THE COURT: And I doubt this line of questioning would be appropriate at trial, but any objection?

MS. WAGSTAFF: No objection, Your Honor.

THE COURT: Admitted.

(Defense Exhibit 2010 received in evidence)

MR. STEKLOFF: No, Your Honor, I agree with you on trial. This, I think, goes to his methodology.

Q. So now let's talk about Mr. Hardeman's hepatitis C.

A. I'm done with this?

Q. Yes. You might need other exhibits in there so keep the binder, but we're not looking at that document.

Okay. And you have reviewed documents that show that Mr. Hardeman had what his doctors have called chronic hepatitis C; correct?

A. I have seen some -- some record that said chronic hepatitis C. I don't believe he actually had chronic hepatitis C the way we define chronic hepatitis C.

Chronic hepatitis C, it means that despite that there's still actual virus that you're able to detect. That's what's chronic.

He had cured hepatitis C. He had hepatitis C at some point prior to treatment, and then he received appropriate therapy that cured his disease.

So the proper way to label him would be history of hepatitis C currently cured, but I have seen some of these documents that you are alluding to.

Q. Well, let's break that down a little bit.

The first medical record that you -- you reviewed all of Mr. Hardeman's medical records; correct?

A. I have.

Q. And the first medical record that you have is dated in 2005; correct?

A. Yes, I believe so.

Q. So you have no medical records from the 1960s to 2005 for

Mr. Hardeman; correct?

A. I did not see that.

Q. Okay. And so you have no medical -- and you know that Mr. Hardeman developed cirrhosis of the liver; correct?

A. He had mild degree of cirrhosis at the time in 2005 when he presented. It was a mild degree, I believe.

Q. Okay. And you are aware that the latency period for the development of cirrhosis that is associated with hepatitis C is on average decades; correct?

A. Yes. I mean, some people would call it 15 to 20 years in terms of finding cirrhosis, but the degree of cirrhosis sometimes gives you an idea as to the potency of the hepatitis C in this particular individual. So you have to look at that as well; but you're correct, it takes decades to develop cirrhosis.

Q. And you also reviewed, and I don't need to go into detail about what they are, but the risk factors that Mr. Hardeman had in the 1960s for developing hepatitis C; correct?

A. I did ask him questions about some virus behavior pertaining to how he acquired hepatitis C.

Q. And you're aware that those risks -- you need to be exposed to something to contract hepatitis C; correct?

A. Yes. We don't acquire it from thin air. Yes, you have to be exposed to something.

Q. Right. And you are aware that in his history, the time

that he was most likely exposed was in the 1960s; correct?

A. Yeah. I mean, I believe that's where he probably contracted it. It's not really clear to me when he did actually have the hepatitis C. I think you and I know that we don't know. We can only assume that it's been decades because obviously it takes time until you have some degree of damage to the liver, such as early cirrhosis and so forth.

Q. Okay. And so going back to your definition of chronic hepatitis C, he could have had chronic hepatitis C from some point in the 1960s up through 2005; correct?

A. He would have had active hepatitis C. Again, chronic hepatitis C, there are some patients that you still -- you know, after they acquire hepatitis C, the hepatitis C they -- you know, it still lingers around and it's still present. So he could have had the hepatitis C for many years prior to being discovered in 2005.

I don't know when but he may have acquired it in the late '60s, and maybe at some point after that when he developed the actual infection.

Q. You just don't know?

A. We just don't know exactly when, but we only can have an educated guess as to when he may have acquired it, and I -- you know, I think it's possible some of the high-risk behavior in the late '60s when he may have had it.

Q. But you do agree that the cirrhosis of the liver in his

case relates to his active hepatitis C, whenever it existed?

A. Yeah, I believe so. Again, it was a I think they called it mild degree of cirrhosis when he presented in 2005.

Q. Okay. Now, let's look at your report because you talked about hepatitis C. That's the first exhibit, Number 2000, in Mr. Hardeman's report.

A. Okay.

Q. And on page 4, that's your discussion of hepatitis C; correct?

A. Yes. This is what I discussed in hepatitis C.

Q. Okay. And so you discussed -- first of all, you didn't have any -- well, you didn't make any assumptions, like we were talking about a moment ago, about the epidemiology generally of hepatitis C from other experts; correct?

A. I know hepatitis C is a known causative factor of non-Hodgkin lymphoma. I mean, this is well known. Any lymphoma specialist would tell you that hepatitis C is a known cause. I'm not sure what you mean by "epidemiology." I know it's one of the causes.

Q. Okay. And then you --

A. I didn't need to -- I mean, it's something that is well known for any lymphoma specialist that hepatitis C --

THE COURT: I'm sorry, if I could interrupt.

Can you please take that down off the screen?

MR. STEKLOFF: Oh, sorry. Yes.

THE COURT: Thank you.

When you put something up, it's showing to the audience, I believe.

MR. STEKLOFF: Got it.

THE COURT: And, you know, I will say that just the ground rules for sealing, I mean, as you are acknowledging in the way you're proceeding, you know, the fact that a particular plaintiff has a particular medical condition is not going to be kept confidential but it could be that the cause of the medical condition could be kept confidential, and I think that would probably be appropriate in this case. So you need to be careful in that regard.

MR. STEKLOFF: I apologize, Your Honor. And I verbally have been trying to be careful, and there is a *motion in limine* pending on that issue; and so I think the same as last Monday, I've been trying to be cautious about that.

THE COURT: So you might want to back up. I got distracted by that a couple questions ago --

MR. STEKLOFF: Yes.

THE COURT: -- so you might want to back up.

BY MR. STEKLOFF:

Q. First of all, I was asking, Dr. Nabhan, and I think you were testifying that you are very aware that active hepatitis C is a known causative risk factor for the development of non-Hodgkin's lymphoma; is that right?

A. Absolutely.

Q. Okay. And my question was a little bit different. You didn't have a separate epidemiological analysis about hepatitis C as a risk factor from any other general causation experts; correct?

A. From general causation experts?

Q. Yes. You didn't review any other expert in the case who gave you some sort of analysis of the epidemiology associated with hepatitis C; correct?

A. No. I mean, I reviewed some of the epidemiology literature myself, and I'm aware of a lot of the epidemiology literature with hepatitis C and non-Hodgkin lymphoma.

I did recently get some of Monsanto's experts' reports, as you know, for this case and I did a high-level review of some of the references that they cite in their expert reports, most of which I was already aware of, and I didn't necessarily cite all of the literature. There's hundreds of references on the topic that I think is not disputable in terms of the association between active hepatitis C and non-Hodgkin lymphoma.

Q. Okay. And so that's actually what I wanted to focus on without pulling it up on the screen.

In your report on page 4 you cite three studies that relate to hepatitis C and lymphoma; correct?

A. Yes, but these are not inclusive. I mean, I could list

hundreds of studies. Again, to me it wasn't an issue, that it was already known the risk and, in fact, the first paragraph says (reading):

"Hepatitis C is a known risk factor for developing NHL."

So, again, it wasn't something that I needed to bring 20 references to tell you that hepatitis C is a risk factor for NHL. I've already known that and I assumed everybody knew that.

I think there's a lot of literature out there that confirms if you treat the hepatitis C and if you eradicate the hepatitis C, the risk of developing NHL is either eliminated or substantially reduced and I just provided a couple of examples. But, again, this list is by no means inclusive of everything.

Q. Right. My question was very simple. You cited three papers; correct?

A. And I'm just trying to explain that these three papers does not mean that these are the only papers I relied on or I knew of. I just didn't want to list every single paper I know of because the list would be endless.

Q. Okay. Well, let's first start --

A. These are the papers I'm listing here.

Q. Okay. So let's first start in the second little bullet with the two papers you list, Pellicelli, which is P-E-L-L-I-C-E-L-L-I, and Tsutsumi, T-S-U-T-S-U-M-I. Those are

two of the three papers that you discussed; correct?

A. Yes.

Q. And both of those involved patients who received antiviral therapy to treat their hepatitis C after completing treatment for their non-Hodgkin's lymphoma; correct?

A. Yes.

Q. And so both of those papers analyzed whether or not patients who had non-Hodgkin's lymphoma would have a relapse of non-Hodgkin's lymphoma with or without treatment for their hepatitis C; correct?

A. Correct.

Q. And we can agree that Mr. Hardeman -- that that doesn't apply specifically to Mr. Hardeman in the sense that he received his treatment prior to his non-Hodgkin's lymphoma diagnosis; correct?

A. Yes. But the reason I put this in is just to illustrate that relationship between hep C and NHL and that relationship is close enough that if you treat the hep C, you actually eliminate or diminish substantially the risk of NHL.

In fact, there is a lot of literature that for some of the hep C-associated NHL, if you treat the hep C, the lymphoma even regresses and goes away. And this one, an example in this link and this association that sometimes if you continue therapy for the hep C, it reduces the relapse of the lymphoma if you have a lymphoma that is associated with hep C.

So the illustration of this example is more along the lines that that link is strong but the antiviral therapy, when you do antiviral therapy, you even prevent the relapse. So that's how important the antiviral therapy of the hepatitis C.

So if you treat the hepatitis C, you are preventing relapse in some patients with lymphoma that developed because of the hepatitis C. So it's just an illustration.

Q. Okay. So it was an illustration, but you agree that Mr. Hardeman doesn't fall into the subjects being studied in either of these two studies; correct?

A. He doesn't fit in this particular one, no.

Q. Okay. And in the Kawamura study, which is the first study you cite, you agree that it does not discuss anywhere how long the patients who had hepatitis C infection had that infection prior to being treated?

A. Most studies, by the way, don't have a particular --

THE COURT: Why don't you -- I think a good strategy for this is it's appropriate if you feel like his question and your yes-or-no answer alone might leave some --

THE WITNESS: Sure.

THE COURT: -- misimpression, that's fine for you to explain; but when possible, try to answer the question first and then caveat it how you think is appropriate.

THE WITNESS: Yes, Your Honor.

\\\

BY MR. STEKLOFF:

Q. Okay. So my question was: You agree that there were hepatitis C infected patients that were studied as part of the Kawamura study; correct?

A. Correct.

Q. And they were then treated with Interferon to try to eliminate or address the hepatitis C; correct?

A. Try to treat and eliminate hepatitis C, yes.

Q. And you do not know how long the patients in that study had active hepatitis C prior to that treatment; correct?

A. I don't know that.

Q. Okay. Now, there are other studies -- you said there are hundreds of studies on this topic; correct?

A. Yes.

Q. Okay. And you agree that -- well, first of all, let's talk about the latency period associated with hepatitis C and non-Hodgkin's lymphoma. Okay?

A. Okay.

Q. Do you agree that the hepatitis C -- the latency period associated with hepatitis C and the development of non-Hodgkin's lymphoma ranges between 5 and 35 years with an average around 15 years?

A. It's long. Yes, I agree with that.

Q. Okay. And so just to give us a timeline for Mr. Hardeman, his treatment of hepatitis C that you are focused on occurred

between 2005 and 2006; correct?

A. Correct.

Q. And then he developed his non-Hodgkin's lymphoma in 2015; correct?

A. Correct.

Q. Okay. And walking through -- you said you've now looked at some of the studies that were cited by Monsanto's experts?

A. Not all of them. Again, there were hundreds of references. I looked at some of them high level. I read some abstracts. But there were a lot of references, as I'm sure you're aware, so I did not look at every single one.

Q. Okay. Well, let's look at in -- now I think this is a new binder for you, Dr. Nabhan -- Volume 3 called "Scientific Literature," and let's look at 2063, which is a study called the Giordano study?

THE COURT: Before we get into that, can I ask a question about timing?

MR. STEKLOFF: Yes.

THE COURT: I was -- you know, it's been an hour and 45 minutes so I'm guessing it's probably potentially a good time for a break, but how much longer do you have do you think with the understanding that I keep interrupting you?

MR. STEKLOFF: I'd rather have you ask the questions that are on your mind. I really think I have 20 minutes or less.

THE COURT: Okay. I think what we should do, then, is why don't we take a 15-minute break now, then we'll come back, and then we'll go till about 12:30; and if there is -- if more time is needed after that, we'll break for lunch and come back.

MR. STEKLOFF: Okay.

THE COURT: Maybe there won't be more time needed after that.

MS. WAGSTAFF: Your Honor, could I ask that page 4 of Dr. Nabhan's expert report with respect to Mr. Hardeman is under seal?

THE COURT: Well, I will tell you that it's not going to be -- page 4 -- no. The top bullet point of page 4 can be under seal.

MS. WAGSTAFF: Okay.

THE COURT: I don't think anything else should be.

MS. WAGSTAFF: Sure. That's fine.

THE COURT: Yeah. And you guys are probably going to have to go back and redo all your sealing stuff because the materials you submitted were oversealed, overredacted.

MS. WAGSTAFF: I think it was just because we were -- the time constraints were tight.

THE COURT: I understand. Yeah.

Okay. So, Dr. Nabhan, you can step down, and we'll resume at 35 after the hour.

THE CLERK: Court is in recess.

MR. STEKLOFF: Your Honor, is it possible that Dr. Nabhan be instructed not to discuss the content of his testimony with counsel on the break?

THE COURT: Any objection?

MS. WAGSTAFF: No.

THE COURT: Okay. Don't discuss the contents of your testimony with counsel during the break.

THE WITNESS: Okay.

THE COURT: Thank you.

(Recess taken at 11:20 a.m.)

(Proceedings resumed at 11:38 a.m.)

THE COURT: Okay. You can resume.

MR. STEKLOFF: Thank you, Your Honor.

Q. Dr. Nabhan, first of all, you agree that IARC has categorized hepatitis C as a Category 1 carcinogen; correct?

A. Yes, I do.

Q. And if you have a person exposed to two IARC categorized carcinogens, one is Category 1 and the other is Category 2A, you would give the Category 1 carcinogen more weight than the Category 2A carcinogen; correct?

A. In each case is different. I mean, IARC is what IARC is in terms of classification. Group 1 usually is more in the IARC hierarchy than Group 2A. So obviously it's -- you give it a lot of weight, but then you look at the particular case and see if that weight applies to this individual particular

patient.

Q. Okay. So look at now -- sorry, you're going to have to open the testimony binder behind you to your right, back right shoulder, and turn to Tab 2035, please, and turn to page 262.

A. (Witness examines document.) Okay.

Q. And you were asked at line 15 (reading):

"So if you have a person exposed to two IARC-ranked carcinogens and one is Category 1 and the other is Category 2A, in that circumstance would you be able to determine" -- "would you be able to determine what caused the patient's cancer?"

And your answer was (reading):

"I would say Category 1 would have more weight than Category 2A."

Correct?

A. That's what I just said, but then for a particular patient might be different. So if you're talking in abstract, of course Category 1 is more than 2A and 2A is better than 2B and 2B is better than 3, but in a particular patient you have to apply that in a particular patient.

Q. So now let's turn to the literature --

A. Are we done with this?

Q. Yes.

We're looking at the literature binder, and I would like to go over three studies with you.

A. Am I still looking at 2063?

Q. Yes. Let's look at 2063, which is the Giordano study.

A. I had a chance to read a little bit through it on break.

Q. Okay. You didn't include this in your discussion in your report about Mr. Hardeman; correct?

A. As I told you, there are hundreds of studies I did not include.

Q. Now, if you --

MR. STEKLOFF: And, Your Honor, can I admit Exhibit 2063 for the hearing?

THE COURT: Any objection?

MS. WAGSTAFF: No objection, Your Honor.

THE COURT: Admitted.

(Defense Exhibit 2063 received in evidence)

BY MR. STEKLOFF:

Q. And if you turn to page 2 of this study, in the middle column do you see where it says the heading "Study Patients Infected and Uninfected With HCV"?

A. (Witness examines document.)

THE COURT: What page are you on?

MR. STEKLOFF: The back page, which is I guess page 2011 of the study, the second page, Your Honor.

THE COURT: Which exhibit? I think I may be on the wrong exhibit. Sorry.

MR. STEKLOFF: 2063, which is a study titled "Risk of

Non-Hodgkin Lymphoma and Lymphoproliferative Precursor Diseases in U.S. Veterans With Hepatitis C Virus."

THE COURT: Okay. I have that. And you said the last page of that?

MR. STEKLOFF: No. The second page, Your Honor.

THE COURT: Oh. I'm sorry.

THE WITNESS: Which? Second page, second column?

BY MR. STEKLOFF:

Q. Second page, middle column, and at the bottom there's a header that says "Study Patients Infected and Uninfected With HCV." It's also on the screen if it helps, Dr. Nabhan.

A. Oh, yeah, yeah. In bold, yes.

Q. Yeah. And so this explains the type of patients that they included in the study; correct?

A. Yes.

Q. And they identified patients with HCV and then they used their VA visits, but then they also used their ICD-9 diagnosis codes; correct?

A. Yes. So they didn't have the actual lab data to confirm whether the person has hepatitis C or not. They relied on the ICD-9 code and presumed that the ICD-9 code reflects that the person has the particular disease that it's billed for.

ICD-9 code is basically billing codes, as you know, so that's what they used because they didn't have any information on the actual -- actual labs for these patients that they

studied.

Q. Correct. And one of the codes, as you can see, is 070.54; correct?

A. Yes, I see that.

Q. And I'm happy to show you if it helps, but do you recall that that is also the ICD-9 code that Mr. Hardeman's treating physicians used when explaining his diagnosis of chronic hepatitis C?

A. I'm sure you have it. I don't recall it because we actually never rely on billing codes to confirm or exclude a diagnosis. These are often -- often wrong, to be honest.

I was a director of the Cancer Center at University of Chicago. It was a contentious thing that I tell people you have to bill for the right code and pretty much because most physicians don't know all of the codes.

So that's why -- that's one of the limitations anytime you rely on billing codes to determine a disease, and you really have -- you cannot say somebody has a disease because somebody billed for that code. This is a billing code and that's it.

Q. Okay. But my question is, and if you need me to show you a medical record, do you recall that this code is in Mr. Hardeman's records associated with his chronic hepatitis C?

A. I don't recall that because I don't look at the billing codes.

Q. Okay. Well, this is a case control study; correct?

A. This is -- yes, it's a retrospective analysis based on the ICD-9 codes.

Q. And we can agree all case control studies, including the glyphosate ones, have limitations; right?

A. All case control studies have their own limitations.

Q. Okay.

A. There's actually something important in this trial I'd like to explain as I was reading through it.

Q. Now, if you look at the "Conclusions" section on the first page.

A. That's the abstract?

Q. Yes.

A. Uh-huh.

Q. You see the conclusion that hepatitis C confers a 20 to 30 percent increased risk of non-Hodgkin's lymphoma overall, and then the authors discuss some other conditions and go on to say (reading):

> "These results support an etiological role for HCV in causing lymphoproliferation and causing non-Hodgkin lymphoma."

Correct?

A. That's active hepatitis C because, as you just said earlier on, this -- this was trying to compare HCV infected to HCV uninfected.

So, in fact, if you turn to Table 2, I mean, if you

don't -- I mean, Table 2 shows only the HCV infected they have the risk. When they compare HCV infected cohort to HCV uninfected cohort, it was statistical significance. If you're not HCV infected, you actually don't have the risk.
Mr. Hardeman was not HCV infected for eight years prior to the diagnosis.

Q. But they went back and looked at patients in past medical records; correct?

A. No. They used the ICD-9 codes to determine infected versus not infected. I think where they were able to, they would go back to the records. But this study looked at -- looked at thousands of patients and they used the ICD-9 codes in their methodology to determine.

So, for example, if you were billed at ICD-9 code of hepatitis C, they assumed you have it. And, in fact, you know, on page -- that paper, on page 2016, the last paragraph, here's what it says (reading):

> "Other limitations should be mentioned. First, we did not validate the cancer diagnoses through a separate chart review" -- contrary to what you just said, Counsel -- "and our reliance on diagnoses coded in the patient treatment file and outpatient clinic file could have introduced inaccuracies. Also, the ICD-9-CM codes for NHL did not allow us to distinguish the various pathologic subtypes, which should be the goal of future

research."

If you fast forward, the fourth line from below (reading):

"Third, we did not have data on some known or postulated risk factors for NHL, such as family history and pesticide exposure."

So I think the authors by themselves they acknowledge there are limitations. So, yes, it's hypothesis generating. All of this tells me if you're HCV infected, then you have high risk. If you're HCV uninfected, you don't. It acknowledges ICD-9 codes limitations. And the authors by themselves, they talk about pesticides, they couldn't account for them.

Q. Dr. Nabhan, I understand that you believe there are limitations to this study. My questions are different so will you please just try to answer my questions?

A. Well, but, listen, I'm a clinician and a researcher. When you show me a paper and you ask me to comment on two lines in the paper, I need to put things in context. I mean, it's not fair just show me conclusions and say "Hepatitis C virus confers 20 to 30 percent increased risk" not taking into context the methodology, the other limitations of the paper.

I mean, you can't just pick and choose the lines that you think they suit what you're trying to tell me and ask me to comment on them inappropriately. I mean, I have to acknowledge the limitations.

Q. Do you think you have to acknowledge the limitations in

the glyphosate papers?

A. Absolutely.

Q. Okay. Now, what the authors did here -- this study was published in 2007; correct?

A. Yep.

Q. They looked at ICD-9 codes in patient files between 1997 and 2004; correct?

A. Yes.

Q. And then they also looked to see if those patients developed non-Hodgkin's lymphoma; correct?

A. Again, based on the ICD-9 codes of non-Hodgkin lymphoma.

Q. Yes.

A. You just have to -- I've done the work and research on some of these things with ICD-9 codes. These are billing codes. So you presume based on what the doctor is billing that this patient has the disease. So you're assuming that the disease exists because of the billing code. You didn't really necessarily look at the viral load, at whether the person has it or not. If I billed for hepatitis C, then this person has hepatitis C. If I billed for non-Hodgkin lymphoma, they have it. That's how you make the assumptions.

They're important studies and it's published in *JAMA*, they have a lot of importance, but there are limitations so we have to take it in context and try to make sense of the information that's being provided to us.

Q. Correct. But the point is that using the ICD-9 codes with all of the limitations you just described, they also look to see if the patients developed a non-Hodgkin's lymphoma; correct?

A. Yes, comparing HCV infected to HCV uninfected.

Q. Correct. But there was no requirement that the HCV infection had to be in existence at the time of the development of non-Hodgkin's lymphoma; correct?

A. They assumed it's there based on the ICD-9 code.

Q. But the ICD-9 code could have been, let's say, 1997 and then they could have had a development of non-Hodgkin's lymphoma at a later date with a different ICD-9 code; correct?

A. No. Let me show you the page that you just told me. Page 2 under methodology, "Study Patients Infected and Uninfected With HCV" (reading):

> "We identified patients with HCV as individuals with two or more VA visits during the fiscal years 1997-2004."

So this is the assumption. They said, "If you have two visits, then we are going to include you," and then they used the ICD-9 code that this person may have had the hepatitis C. They don't know, based on the ICD-9 code, whether it was in '98, whether in 2000, whether in 2001.

Again, this is an assumption that the ICD-9 code reflects the disease. I hope everybody in this courtroom recognizes that this is not how we make a diagnosis. We make a diagnosis

based on labs, seeing patients, examining a patient. I don't rely on ICD-9 codes.

And, you know, I would --

THE COURT: If I could just interrupt for a moment. I don't think that's the question he's asking you.

The question he's asking you is: Understanding the limitations of using ICD-9 codes, it appears from this study that we don't know whether the people being studied -- assuming they had -- assuming they were HCV infected, we don't know whether they were HCV infected at the time of NHL -- that they were diagnosed with NHL or HCV infected previously.

I think that's the issue you're getting at; is that right?

MR. STEKLOFF: Yes, Your Honor.

THE COURT: So forget about -- we understand all of your caveats about the ICD-9 codes. You don't need to repeat those again. The issue that he's getting at is that it sounds like from what you were saying about this article, that you are assuming that when people are diagnosed with NHL, they are currently HCV infected or currently HCV positive.

And the question that he's asking you is getting at the possibility that that's not the case with this study, that some of the people in the study may just have easily been previously HCV infected.

THE WITNESS: Yes. I mean, so --

THE COURT: So that's what he's asking about, and so

if I could get you to focus on that concept in response to his questions as opposed to the ICD-9 codes. Okay?

THE WITNESS: Sure.

THE COURT: So why don't you go ahead and now -- hopefully we're focused on the issue that you're getting at. Why don't you go ahead and ask your question.

BY MR. STEKLOFF

Q. It is a simple question. Isn't it true that it was not a requirement that the patients in the study have active HCV at the time of their non-Hodgkin's lymphoma diagnosis?

A. Correct. They needed to have at some point an ICD-9 diagnosis of hepatitis C versus the other one, they could not have an ICD-9 diagnosis of hepatitis C.

Q. Let's move to another study which is in tab 2056.

THE WITNESS: Your Honor, may I comment one other thing about the ICD-9 codes?

THE COURT: Sure.

THE WITNESS: So, when it comes to non-Hodgkin's lymphoma, for example -- and this may be a nice exercise for counsel to go back and see -- there are many types of non-Hodgkin's lymphoma, and I can tell you I have been taking care of non-Hodgkin's lymphoma patients for years, but the most common used ICD-9 code of non-Hodgkin's lymphoma is 202.8, for example; but the proper diagnosis code for diffused large B-cell lymphoma is 200.78 or 200.79; and I cannot tell you how

many times I have looked at charts, as part of my administrative role as the director of the cancer center at University of Chicago, where I have seen all lymphoma patients being labeled 202.8, which is NHL otherwise unspecified; and I always made the argument, Let's try to make sure we diagnose and bill properly 200.78. I just want to make sure.

THE COURT: That point is very intuitive. I'm not sure it relates to the point we were just discussing about active versus inactive. The point that people use the wrong billing codes with regularity is intuitive.

THE WITNESS: It is the reason why hospitals actually have hired a lot of billing coders to improve accuracy as well as revenue.

THE COURT: I understand.

THE WITNESS: That's the entire thing I just want to make sure.

BY MR. STEKLOFF

Q. Dr. Nabhan, two more studies. Can we look first at 2056 --

A. 2056.

Q. -- which is a study by Dr. De San Jose.

THE WITNESS: One second.

(Whereupon, a brief pause was had.)

THE WITNESS: I have it.

\\\

BY MR. STEKLOFF

Q. You also didn't discuss this study in your report, correct?

A. I did not.

MR. STEKLOFF: Your Honor, I would move this exhibit 2056 into the record.

THE COURT: Any objection?

MS. WAGSTAFF: No objection.

THE COURT: Admitted.

(Defense Exhibit 2056 received in evidence)

BY MR. STEKLOFF

Q. First as background, so you can do a test that tells you on a patient whether -- who has hepatitis C whether there is active virus, correct?

A. You can.

Q. That is called RNA, correct?

A. Yes. You do the viral RNA, yes.

Q. You can treat a patient with hepatitis C with interferon; and there would be no active RNA, correct?

A. Yes, interferon or interferon -- it is to eradicate the virus.

Q. It is still possible, even if it is not active -- if the viral load isn't active to have hepatitis C deep in your bloodstream, correct?

A. It will always depend on how sensitive the method is. All

of the guidelines that I'm aware of -- even if you go to the CDC or any type of guidelines -- outside of clinical trials and outside of research, they recommend the ELISA method, which you are trying to detect the viral RNA dimension, which is exactly the method that Mr. Hardeman had. That is what the guideline says. I think there are lots of research out there that tries to say that maybe despite the eradication, an inability to detect the virus, maybe if we use ultra sensitive methods or whatever type of methods, maybe there are still something lingering out there; but we don't know is whether that is something that is lingering out there has any link to NHL because that has never been studied; and all of the studies, by the way, including the IARC and the group one that you just mentioned, relied on the ELISA and relied on what is the standard of practice. The standard of practice is, you do the ELISA. That is the one you check for viral load or not.

Q. You agree with me -- and it may not be standard of care -- but there is an additional test that is -- I think you used the word ultra sensitive, that can still identify hepatitis C in the blood even if the viral load isn't active, correct?

A. There is some research into ultra sensitive methods -- I try to tell you -- the clinical significance of the detection in ultra sensitive way has not been determined and there is no data I'm aware of that detecting something in an ultra sensitive manner that you failed to detect in the traditional

standard of care way has any link to the development of non-Hodgkin's lymphoma. In fact -- or liver cancer. In fact, if there was, it would be inappropriate then not to check the ultra sensitive methods. The guidelines don't tell you that. So are we giving our patients a disservice by not doing the ultra sensitive methods? No. The reality is they are there. They are being researched. We don't know their clinical significance. We are doing what the standard of practice is, the ELISA test.

Q. This is a very interesting discussion. I think it shows -- you haven't reviewed the D. San Jose study before, have you?

A. I have not.

Q. Let's look right on page 1 where it talks about methods. Do you see that?

A. I do.

Q. And, first of all, you can see above that that the authors pooled case controlled study data to provide robust estimates of the risk of non-Hodgkin's lymphoma subtypes after HCV infection. Do you see that?

A. Yep.

Q. And do you see that if you go down under methods, it then says: All studies used third generation enzyme-linked immunosorbent assays to test for antibodies against HCV in serum samples. Do you see that?

A. I do.

Q. Those are exactly the ultra sensitive tests we were just talking about, correct?

A. This is the ELISA test, yes.

Q. These are the ultra sensitive tests, right, third generation enzyme-linked immunosorbent assays?

A. I just don't think if third generation is the ultra sensitive one versus first generation. I thought there was a difference between the ones in clinical practice normal when you order the test and the test goes to the lab. We are not talking about research. They do the test. I don't know if it is third generation or not. If it is, then it should be mentioned somewhere in the methods. I don't have a reason to think it's not. I'm just seeing this paper for the first time.

Q. Just to be clear, this study pooled studies that all use this ultra sensitive method to see if there was hepatitis C still in the patient's blood serums?

A. Yes. I think what I'm trying to say -- if this is the ultra sensitive method they are talking about, is this the standard of care that we currently do or there is another type of ultra sensitive method you are going to ask me about.

Q. You can look through the study as you see fit, but this demonstrates that this was not pooling patients who were required to have active HCV at the time. This study was pooling patients who at least had antibodies against HCV in

their serum samples but not necessarily the active viral load. Do you understand that?

A. I'm reading here in the introduction. They were trying to look at the association between NHL and HCV and determined using third generation --

THE COURT: Slow down a little bit.

THE WITNESS: I'm sorry. I'm just trying to make sure, okay. They use third generation ELISA to measure the HCV antibodies. Yes, so that's -- I believe this is the one that we normally use to measure the antibodies of HCV.

BY MR. STEKLOFF:

Q. My question is different, Doctor. If you don't know, you don't know. These patients were not required to have active HCV to fall into the HCV category in this study, correct, active viral load, the RNA that we talked about before?

A. It seems to me they are measuring for the antibodies. Maybe I'm not answering your question. So they are measuring the antibody to HCV to check if the person had HCV.

Q. Right, exposure to HCV not but necessarily active RNA.

A. Now I understand your question. I don't believe -- I can see where they did the viral RNA analysis. Again, I'm sure you read it. I believe they haven't done it.

Q. So this could have been someone like Mr. Hardeman who at some point had viral RNA but then later was treated but still had the antibody for HCV using ultra sensitive tests, correct?

A. Yes. Once you have the antibody, you are always going to have the antibody.

Q. Thank you. Now, let's turn to page 5 of this study under discussion.

A. I need to see the results first. Okay. Go ahead.

Q. And the third line down, the author's summarize their results and say, Our results show increased risks of DLBCL and then two other types of lymphoma, correct?

A. Yes.

Q. Associated with HCV infection. These risk estimates were particularly robust for DLBCL with a twofold increased risk overall and a statistically significant increased risk observed in three of the seven studies. Do you see that?

A. I do.

Q. Okay. And this again was a paper that you did not consider as part of your analysis in Mr. Hardeman's case, correct?

A. I did not consider this paper. It is just in association which I know about.

Q. All right. Now, can we look at one last paper which is in tab 2070, which is the Mahale paper?

THE WITNESS: Can I just take one minute to read a couple of things in this paper, Your Honor, that I was just asked about?

THE COURT: Yeah, sure.

(Whereupon, a brief pause was had.)

MR. STEKLOFF: Just for timing, Your Honor, this is the last topic I have.

(Whereupon, a brief pause was had.)

BY MR. STEKLOFF

Q. Dr. Nabhan, please turn to tab 2070. This paper is titled, the effect of sustained virological response on the risk of extrahepatic manifestations of hepatitis C virus infection by Dr. Mahale and others. Do you see that?

A. Yes, I do.

Q. This is another paper you did not include in your discussion in Mr. Hardeman's case, correct?

A. I did not include this paper.

Q. Have you reviewed this paper before?

A. No. Maybe the abstract, I don't remember. I think this was cited in the -- in one of the expert's reports. I may have read the abstract. I don't recall. There were a lot of references that you provided.

MR. STEKLOFF: Your Honor, I would move this study into evidence.

MS. WAGSTAFF: No objection.

THE COURT: Admitted.

(Defense Exhibit 2070 received in evidence)

BY MR. STEKLOFF

Q. First of all, if you look at the abstract on page 1,

Dr. Nabhan, I want to just go over the methods with you.

A. Sure.

Q. It says that the authors conducted a retrospective cohort study using data of patients from the U.S. Veterans Affair -- Affairs HCV clinical case registry who had a positive HCV RNA test between October 1999 and August 2009. Patients receiving interferon based antiviral therapy were identified. Do you see that?

A. I do.

Q. And now if you could turn to page 6 -- let's be clear, Mr. Hardeman received interferon based antiviral therapy, correct?

A. Yes, he did.

Q. And that was the therapy that he received between 2005 and 2006, correct?

A. That's correct.

Q. And then the development of his non-Hodgkin's lymphoma, his lymphoma was diagnosed in 2015, correct?

A. Correct.

Q. And the basis for you to rule out hepatitis C is that for that approximately eight to nine year period prior to his diagnosis, he did not have active hepatitis C, the active viral load, correct?

A. That is one of the reasons. There are many other reasons actually. I'm more than happy to get into it, your Honor, if

you want me to. This is one of the reasons. There are other reasons that actually even solidifies my opinion that hepatitis C was not involved in this because if --

THE COURT: I will give you a chance. I was planning on asking you that myself anyway. Why don't you let him continue on this.

BY MR. STEKLOFF

Q. We looked before. In your report you cited two studies -- we won't bring it back up -- you cited two studies that talked about this interferon based antiviral therapy that occurred after patients had a diagnosis of lymphoma but then before -- hopefully that they didn't have a remission, correct?

A. So they -- they were treated with interferon and ribavirin versus the comparative group that was not treated, and the ones who were treated and responded to therapy. The likelihood of them having relapse disease was almost zero or negligible up to a follow-up of 15 years. That was the Kawamura study.

Q. Those two studies involved the treatment for hepatitis C after the development of non-Hodgkin's lymphoma, correct?

A. So it is -- it is the -- are we talking about the same one, 2007 paper?

Q. We are talking about the Pellicelli 2018 and Tsutsumi 2017.

A. This one. I thought you were referring to the one on top of that, the Kawamura study.

Q. No, I'm talking --

A. It's different.

Q. I'm talking about Pellicelli and Tsutsumi.

A. Yes, that's correct.

Q. Now, in this study if you turn to page 6, there is a discussion of risk of EHMs by treatment status. Do you see that?

A. I just want to know what EHM means, one second.

Q. No problem. I was going to ask to clarify that, so that's helpful.

A. What is EHM? Do you have that?

Q. Sure. If you look at the introduction on page 2 in the first paragraph, it says chronic HCV infection is also associated with several extrahepatic manifestations, EHMs, including -- and then it lists some various conditions, but it includes some subtypes of B-cell non-Hodgkin's lymphoma. Do you see that?

A. Yeah, I do.

Q. So do you see -- now turning back to page 6 -- it talks about risk by EHMs by treatment status.

A. Yes, I do.

Q. Do you see at the bottom paragraph of that section that starts, in comparing individuals? Five lines down, do you see where it starts, We observed gradual reductions?

A. I'm trying to read the paper from the beginning. The one

in comparing individuals, that is the paragraph you are asking me about?

Q. Yes. Feel free to read the whole paragraph. I want to focus your attention on where it starts We observed.

A. Okay. That's fine.

Q. The authors wrote: We observed gradual reductions in the magnitude of protective AHRs towards the null with increasing time to initiation of AVT for -- a condition that I will not pronounce correctly -- NHL and stroke, Figure 2. Do you see that?

A. Common sense, the faster you start therapy, the less risk it is.

Q. Then it says: The AHRs were significantly protective only when AVT -- so that is antiviral therapy, correct?

A. Yes.

Q. -- was initiated at one or two years after the HCV index date for the two conditions and one year for NHL, correct?

A. Correct.

Q. And so what that means is that the antiviral therapy was significantly protective only when initiated one year after the patients had active hepatitis C, correct?

A. That's okay. I mean, that's -- what this means is that the faster you start therapy, when you detect an infection -- because we can acknowledge in all of this -- on all of this paper that you are showing me, we don't know exactly when each

particular individual first was diagnosed.

From a clinician's standpoint, what this paper tells you is two things. Number one, when you detect an infection, the faster you start therapy, you need to actually start; and if you do so, you are going to reduce the risk of disease. The infection was detected in Mr. Hardeman's case in 2005. He was immediately started on therapy, so it has aligned the fact that the risk will go down. And if we take the assumption that, you know, if you don't start after one year, you are not going to reduce the risk, what you would be telling a patient who comes to see you -- and the infection was a year and a half ago -- you would say, yeah, well, I don't think if I treat you -- it's been a year and a half -- the risk is going to subside. It's, again, the reality is all this paper tells you when you get an infection, the sooner you start, the better the outcomes are. That's all it tells you.

Q. Right. And Mr. Hardeman, we discussed before it is -- his infection may have first occurred in the 1960s, correct?

A. We don't know when it was. It could have been in the early '70s; could be late '60s. I don't think we have the date; but, yes, it was probably was a couple decades before it was first diagnosed in 2005.

Q. It led to cirrhosis of his liver, correct?

A. Very mild cirrhosis. That was an important part for me to look at to get a little bit of the degree to see how much the

hepatitis C caused liver damage. The degree of the cirrhosis was actually mild from reading the notes. The liver function tests were actually excellent, and he tolerated chemotherapy very well without any issues with liver or abnormal liver function tests or even reactivation of the hepatitis C. You would think if it was a problem, when you give chemotherapy, which is pretty potent, the hepatitis C would surface and cause issue or the cirrhosis gets worse or something happens but none of that happened. Yes, there was mild degree of cirrhosis which led to the detection of the hepatitis C, but it was not severe by any means.

Q. It was certainly a load of cirrhosis, correct?

A. A what?

Q. Load of cirrhosis?

A. I have never used the term of load of cirrhosis. Maybe other physicians have. Load of cirrhosis, I don't know what that means. Maybe it is a GI terminology. There was cirrhosis. I just don't know what that means. To me when I asses cirrhosis as a clinician or as an oncologist, I look at the liver function tests. I look at the bilirubin, the ALT, the AST. That's how we look at it. I don't know look at whether somebody describes load of cirrhosis, that's -- load of cirrhosis to one physician may be different than to another physician.

Q. Let's look at your deposition. Now, you will need that

big binder behind you.

A. This one?

Q. Yes. And let's look at the first tab, tab 2032.

A. I'm here.

Q. And if you turn to page 42 of that -- this is the -- this is your deposition, just for the record, in the Hardeman case, if you look at the first page, correct?

A. Yes.

Q. And on page 42, let's look at lines 10 through 20, okay?

A. Okay.

Q. And I think I asked you: And is it your view to a reasonable degree of medical certainty that he had active hepatitis C for approximately 40 years? And your answer was: I don't think we know. I don't think we -- I can't find anything in the records as to when his hepatitis C was present. All I can tell you is that the hepatitis C was diagnosed for the first time in 2005 when he had an ultrasound, and we found to have a load of cirrhosis; and his primary physician sent him to a gastroenterologist and they found the cirrhosis.

A. I'm pretty sure this would be a typo error. I don't even know what load of cirrhosis is. He had a degree of cirrhosis that was present for sure, and that's what led to the GI referral. This is -- I mean, this may have either been a typo or miss -- I don't know what it means. One moment, Your Honor.

(Whereupon, a brief pause was had.)

MR. STEKLOFF: Your Honor, depending on any questions that you have, I might follow up. I have no further questions.

THE COURT: Okay. I want to -- I have a few follow-up questions just about the hep C issue.

THE WITNESS: Sure, Your Honor.

THE COURT: First, on this -- on this issue of latency in 5 to 35 years, where does the 5 to 35 years come from?

THE WITNESS: Originally actually from the -- from the history of cirrhosis and knowing that it takes -- in order for cirrhosis to develop, it just doesn't happen in a year or two years, that it takes longer time for patients to develop cirrhosis when they are exposed to hepatitis C.

THE COURT: What I was asking about is the relationship between hepatitis C and NHL.

THE WITNESS: Right.

THE COURT: So -- and you were talking about cirrhosis. So I wasn't sure if we were talking about something different, but I was asking -- I thought you testified that there is a 3 to 35 year latency period between hep C and NHL. Is that what you testified to?

THE WITNESS: Yeah. I mean, I think I have said several times before that the latency period for many of the offending agents that may cause non-Hodgkin's lymphoma varies. It is not really the same for agent A versus agent B. It is suggested that it takes longer time based on each agent that we

are talking about. So for the hepatitis C, for example, it takes time, I believe, to replicate; and the virus has to be active inside the B lymphocytes in order for the virus to cause damage to the liver and cause the non-Hodgkin's lymphoma outside of the liver.

THE COURT: So before we get into that detail, let me just ask one clarification question. Are you talking about -- so when we say 5 to 35 years, are we talking about 5 to 35 years from the time you contract hep C or is it from the time you are diagnosed with hep C? What is the -- where is the starting point for the 5 to 35 years?

THE WITNESS: Truly it is a very good question. It is very difficult to answer because the literature is not -- is not a hundred percent on that. A lot of the epidemiologic studies that looked at hepatitis C and the association with non-Hodgkin's lymphoma did not necessarily know when these patients contracted versus diagnosed because we know obviously they may contract year 1. Somehow the diagnosis could happen year 7, and then the disease could happen year 15 or year 20. But the studies that were out there did not have that granularity of information. So we are left out by using the information that is published and associate that and trying to compare with other type of diseases knowing, for example, how long does it take for hepatitis C in general to cause some degree of cirrhosis in the liver and then assume that it is

probably unlikely that the hepatitis C will cause NHL before it caused some cirrhosis because we don't have that granularity. So if we know --

THE COURT: Sorry, say that last point again. It is unlikely that it will cause NHL before it causes some sort of cirrhosis; is that what you said?

THE WITNESS: Yes. That is part of the assumption because we don't have the granularity of the information you are asking in the epidemiologic literature. Some of this will become educated guess and a little bit of an assumption, and one of the appropriate assumptions, I would think, that if there was no cirrhosis whatsoever, for example, it is probably unlikely to be -- that virus will be implicated in other types of diseases; but some of this is a little bit of an assumption based upon the lack of granularity of information.

So because we know it takes one to two decades for cirrhosis to develop from hepatitis C, then some of this become an educated guess; that we really think it might take the same amount of time for hepatitis C to cause NHL; but when you go back and look at the literature, you really don't have that detailed information because you would like to know, you know, I contracted it in 1970. Diagnosed in 1980 maybe, and then in 2000 something happened. That detail, that graph, doesn't exist with the accuracy that all of us would like to have. So that's how we end up trying to speculate, but the speculation I

think to a reasonable degree of accuracy is appropriate in this case.

THE COURT: But -- so Mr. Stekloff asked you, you know that NHL developed as a result of hep C infection has a 5 to 35 year latency period, and you said yes.

THE WITNESS: I can buy that, yes.

THE COURT: So you must in your mind have had an understanding when you answered that question of when the 5 to 35 year period starts. Are you talking about from the time -- from the estimated time that the disease was contracted or are you talking about the -- the time the disease was diagnosed?

THE WITNESS: No, contracted, contracted.

THE COURT: Contracted, okay.

THE WITNESS: For me, again --

THE COURT: I understand you can't --

THE WITNESS: Right.

THE COURT: -- measure that with precision because you are never going to know exactly when it is contracted. That is -- when I'm thinking about the 5 to 35 years, I should be thinking about the time from when it was contracted.

THE WITNESS: Sure. I presume somehow in the late 60s, early '70s when the disease was contracted -- that answers your question, your Honor -- it was not discovered until 2005. But to assume or hypothesize that this disease that was contracted -- let's say 1970 for rounding error -- in 1970 and

went on without causing any NHL until 2005, which is 45 years -- my math is right -- no, 2005, 35 years. So it went from --

THE COURT: 45 years to the NHL diagnosis.

THE WITNESS: Right. But the assumption here -- if we are going to implicate hep C in the NHL, the assumption would be that Mr. Hardeman contracted HCV in 1970. Went on for 35 years without developing NHL whatsoever. Was treated appropriately. The virus is gone, and somehow after ten years of the virus gone, we sustain viral response; then developed NHL that we are going to blame now the HCV from 45 years prior. So somehow the HCV did not cause any problems for 35 years and decided to cause trouble 45 years later, ten years after we treated it appropriately. That is a lot of speculation that I don't think it honestly stands the rigor of science in my opinion.

THE COURT: So when Mr. Stekloff asked you -- he said the reason that you excluded hep C is because it wasn't active for nine or ten years before he was diagnosed with NHL, you said that was one of the reasons. I have others. What you just gave me is another reason, I take it, why you rule out hep C?

THE WITNESS: Yes, and I have a third reason too that I strongly believe in; that we all know that by giving patients chemotherapy we suppress the immune system. They are exposed

to other infections and so forth. This is fertile ground for viruses and infections to play in and cause trouble. Not only that, the assumption like we said, 35 years cause no trouble; treated it; somehow it caused the problem 45 years later, after 45 years -- we even gave this patient aggressive chemotherapy including steroids -- which suppress the immune system, chemotherapy and so forth and so forth -- and that virus decided not to reactivate. That is even a third assumption that I also cannot buy into.

I think there are several reasons in this case where it is very appropriate to include hep C in the differential. It is very important. I have done that in my clinical practice. When you look at the details of this particular case, you have a situation of 35 years this hepatitis C caused no NHL; caused mild degree of cirrhosis. The liver function tests actually were pretty good, and then 45 years later -- and then was treated appropriately; adequately sustained viral response for ten years -- we couldn't detect it using standard of care methods that we teach our students and fellows to look at, which is the ELISA method -- and then the virus activated and caused the disease although it wasn't even there -- it wasn't present but decided to cause NHL without even advancing the cirrhosis; causing worse cirrhosis, without having any type of abnormal liver function tests -- I mean, the liver was still doing very well and had no issues for this patient. So it is

really -- it is an issue that is very -- I don't -- I don't believe hepatitis C in Mr. Hardeman's case is the cause of his NHL. That may be different in another case, but in this particular case when you look at the particular situation, it would be very difficult for me to even understand how anyone could blame hepatitis C on the cause of this NHL given the particular circumstances.

The data, by the way -- and we didn't go into this -- the data on how hepatitis C causes NHL, there is a lot of data which I didn't cite in my report. I didn't think it was the topic of conversation -- maybe I should have -- but a lot of the data that implicates, by the way, hepatitis C in the oncogenesis in the development of lymphoma includes having the actual virus present in the B lymphocytes and replicating in the B lymphocytes -- so the suggestion that the virus that didn't exist for ten years is the cause of a particular disease is a stretch in my opinion.

THE COURT: That -- you actually anticipated the next question I was going to ask you which is: What do we know about how hep C causes NHL? And to the extent you can cite literature, that will help me understand that.

THE WITNESS: So there is actually a lot of research on that in terms of the etiology of how hepatitis C causes NHL, which -- again, in that scenario you have reached a conclusion that the hep C is the cause; and you are trying to determine

how does it do it, right, how does it actually do it. And, you know, that's a little bit more of a topic that basic scientists are able to answer more intelligently than I can. I can tell you from a clinician perspective, the data I'm aware of it requires the actual virus to be able to enter the B lymphocytes and causes intracellular problems in some enzymes or pathways that we know are implicated in the pathogens of lymphoma. So if we know that a particular pathway inside the cell is important so you need a mutation in a particular oncogene or a mutation in a particular over expression or under expression of a particular protein, there is a lot of research into hepatitis C entering the B lymphocytes and affecting that pathway; and there are several pathways that I have come across in my clinical practice.

I don't know -- I don't believe that today we have a hundred percent understanding how it does -- how it causes the lymphoma, but we have a lot of theories into how it could possibly do it. All of these theories are contingent on the fact that the virus is somehow able to enter the B lymphocytes and cause problems inside the cell and B lymphocytes.

THE COURT: If you don't have it off the top of your head, that's fine. Do you have any studies you can point me to that establish the point that you were just making?

THE WITNESS: I actually do know of several studies. The authors' list escapes my mind. I'm more than happy, if

that's allowed, to provide some of these studies later onto the Court.

THE COURT: Okay. Do you -- for somebody who gets NHL from hep C as opposed to -- from some other cause, are the symptoms different or the collateral consequences different?

THE WITNESS: So the symptoms are not -- so lymphoma in general, it could be whatever causes the lymphoma. Most often these lymphomas are the same. In other words, the diffuse large B-cell lymphoma whether it is caused by Roundup, hep C, idiopathic, you don't know why, the actual DLBCL is the same. There are studies trying to look at the genetic signature of the actual lymphoma and trying to determine whether we can answer this question, which means that can we actually tell the actual signature of the lymphoma, whether this lymphoma -- if it is caused by agent A versus agent B -- there are difference. So these studies are ongoing. Today we actually don't know.

What we know, by the way, is that there are particular -- I would say phenotype of a patient that may fit the criteria, let's say of hepatitis C related NHL. One of these criteria that we know -- a lot of the patients who have hep C related NHL -- a lot doesn't mean a hundred percent -- it is kind of -- you see it more commonly in those patients is external disease. External disease means that you could have the presence of lymphoma outside of the lymphoid structure. So anything in the

body that is not lymphoid, you would see that more often than patients who don't have external disease. There are a couple of papers that have suggested higher risk NHL when it is related to HCV and the LDH, which stands for lactate dehydrogenase, it is a marker that there is more tumor turnover. So when you have high LDH in the blood, that means the tumor is replicating fast. So high LDH, we see that in HCV.

I think more -- there are some, a couple of studies, which I believe they are older, so I take those with a grain of salt, just shorter survival for HCV related. I think some of these studies were before we had more potent therapies for HCV. I think some of these studies we have to take with a grain of salt because I believe that we have improved on two things, the treatment of lymphoma and the treatment of HCV. I tend to -- I would hope that the outcomes is not that different although there are some studies to support the opposite.

So we don't know on a genetic level the differences between these lymphomas although it is a very active area of research. I think my clinical hat is a little bit skeptical about this because I don't believe studying the genetic signature of every lymphoma is scalable to every single hospital, every single physician. I think it is still more of an area of research investigation in highly trained academic centers where they are trying to look at that. Even if they

find something, it is always goes back, is it going to change my management? I'm going to still treat the same way I treat, and that's why it may be a good question to ask but because it doesn't affect management, that's why people aren't as interested as they should be in answering that question.

THE COURT: Can you think of any other potential signatures of hep C caused NHL?

THE WITNESS: Signatures?

THE COURT: Signatures of lymphoma caused by hep C was the phrase I think you used.

THE WITNESS: Yes. When I talk about genetic signature, I mean that you take the actual tumor and you sequence the tumor and do gene expression profiling. All I'm saying is there are some studies that try to do that and try to identify are there mutations.

THE COURT: What about other symptoms that somebody would be more likely to experience if their NHL was caused by hep C?

THE WITNESS: There could be nothing, nothing different at all unless the liver -- for example, if the liver is damaged significantly, you might see liver function tests abnormal. If there is -- if you have somebody that advanced liver disease, they could be jaundiced if the liver is really advanced. In terms of symptoms, it could be exactly like any other large cell lymphoma; and the only reason you would check

for the hep C is because either you identified high-risk behavior that might put the patient into HCV category or because this is some part of your routine work-up. You check the HCV on every lymphoma patient and make sure that the patient doesn't have HCV. If they have it, you would want to treat it with antiviral therapy.

THE COURT: Okay. You want to follow up on anything that I asked?

MR. STEKLOFF: Yes, very briefly.

BY MR. STEKLOFF

Q. You agree with Mr. Hardeman, he started using Roundup in the 1980s, correct?

A. Late '80s, I believe.

Q. He then used it until 2011 or 2012, correct?

A. My memory says 2012 -- towards the end of 2012 when he changed residences, but I may be off by a year.

Q. Exactly. He changed residences in that timeframe, 2012; and then at his -- I believe his current residence did not use Roundup.

A. That's my understanding, yes.

Q. So between 2012 and 2015 he was not using Roundup, right?

A. He was not.

Q. From the late '80s until 2012, let's say, so 25 years approximately he was using Roundup, right?

A. He was.

Q. And he was -- I mean, quickly in the '80s had exceeded that two day or ten lifetime day exposure, correct?

A. I believe so.

Q. Okay. And but it's clear that his non-Hodgkin's lymphoma was not diagnosed until 2015, correct?

A. Absolutely. February 2015, I believe.

Q. Just to follow up a little bit -- it sounded much more complicated -- but I think the hypothesis you were stating on the mechanism of action is that hepatitis C causes -- during the cell replication process can cause DNA damage in cells, correct?

A. When I said we don't know how precisely it causes lymphoma genesis. We just don't know. It is an active area of research. There is a lot of theories. For the virus to be able to cause NHL, it has to be present somehow to cause the problem which is very different than other types of offending agents that could cause problems to DNA and cause genotoxicity and you are not exposed to them. The damage is already done.

From a viral perspective -- again, when you are looking at viruses, you have to have that virus present to cause the problem. It's actually why we treat -- I mean, just think about, again, from -- I'm talking here as a clinician. If I have a patient who comes in and they have the virus, if I don't believe treating the virus is going to reduce the risk of NHL, liver cancer and cirrhosis, why do I even treat it? The reason

we treat -- we don't treat flu most of the time because we don't think it will cause trouble -- although it does -- the reason we treat hepatitis C is because we believe the continuous presence of the virus is what causes the problem and what causes the damage to the liver and cancers. That is the essence of why we treat these viruses. There are many viruses we get exposed to every day that we never treat because they have no damage to any end organ that we have.

MR. STEKLOFF: I have nothing further, your Honor.

THE COURT: You might after -- I have one follow-up -- maybe a couple follow-ups to see if I can better understand this. So my understanding -- thinking back to the general causation testimony of you and all the other witnesses -- is that the theory for how glyphosate from a mechanistic standpoint causes NHL is that probably it damages cells; right?

THE WITNESS: Damages cell, oxidative stress. There is some evidence of that, yes, it causes some genotoxicity and DNA damage.

THE COURT: Nonetheless there is a relatively long latency period in general for NHL. I gather what that means is that you have glyphosate or whatever causing cell damage or oxidative stress and that happens in the year 2000 and someone may not be diagnosed with NHL until the year 2010, right?

THE WITNESS: Right.

THE COURT: So I want to make sure I'm not missing

something here. It sounds like what you are saying is that the virus similarly does damage to the cells in a way that causes NHL; is that right?

THE WITNESS: Yeah. It does damage to the cell. We don't know what it does exactly. What we know if it is not present, it can no longer damage the cell.

THE COURT: It can no longer damage the cell; but in the case of glyphosate, according to the Plaintiffs' experts, the glyphosate might damage the cell, say, from 1990 to the year 2000; might do damage to the cells, but the person might not get NHL or might not get diagnosed with NHL until 2010.

THE WITNESS: Right. Even if they stop the use because the damage is already done with glyphosate and even if you stop the use for several years, the damage is already done to the cell which is very different than how viruses operate.

THE COURT: That is my question: How is it different with viruses? Why is it not also the case, you have active hep C from 1990 to 2000, and then the virus gets cleared. You are diagnosed with NHL in 2010. Why couldn't it be the damage that was done to the cells by the virus during the 1990 to 2000 period? Can you explain that a little bit more?

THE WITNESS: The way the viruses work, you have to have the continuous exposure to the virus in order for the damage to continue; and it is like really any type of virus we get exposed to.

THE COURT: How is that different from the glyphosate scenario?

THE WITNESS: Because viruses don't need to necessarily to cause genotoxicity, per say. They can actually enter the actual cell and cause -- they have to replicate within the cell, and then the effect -- the balance between cell growth and cell survival; and if that balance is tipped off between how cells -- we have this constant balance between cell survival and cell death, right? The cells die and survive. If that balance is tipped off differently, you can have tumors that are malignant or not. In order for viruses to do that, they need to be present and cause this type of replication within the B lymphocytes, within the cells in order to cause this particular lack of balance, which is very different -- so when we give chemotherapy to a patient, when we give chemotherapy to a patient -- say, when we give high-dose chemotherapy in stem cell transplant, these are high doses of chemotherapy that we give; and the patient undergoes stem cell transplant. They are no longer getting chemotherapy. They are done. The chemotherapy is out of their body. They are done with the chemotherapy. Yet, they could have leukemia or myelodysplasia several years later as a result of the chemotherapy that they were exposed to. Not all of them have it. Some patients may never have leukemia or myelodysplasia. Some of these patients do. Their DNA gets damaged to a degree

that eventually they could develop leukemia or myelodysplasia.

Viruses don't operate like this. Viruses in order for them to cause the imbalance between cell survival and cell death, they need to be present in the body. If you eradicate them, they are no longer present. The damage that they may have caused is negligible in my opinion because they are no longer there. They can't cause any of this problem which is very different than chemicals that we use. Did I answer your question, your Honor?

THE COURT: Well, except that I -- in Mr. Hardeman's case, the virus was apparently working on him for 35 years and so that -- based on the way you are describing it, it sounds like a lot of damage can be done; and I'm still not -- I'm still not understanding why it couldn't be the case -- given what we know about the latency of NHL generally -- why it couldn't the case that all this damage would be done over the course of 35 years; and then the virus stops doing its work in 2005, and Mr. Hardeman is diagnosed with NHL in 2015. Why, given what we know about the NHL, couldn't it be as a result of the damage that the virus did over that 35-year period?

THE WITNESS: Just because how viruses work. In order for the virus -- in order for the virus to have been causing the NHL, the NHL would have needed to be diagnosed in 2005 or earlier.

THE COURT: Why? I'm still having trouble

understanding that.

THE WITNESS: Because viruses work in a way that -- how chemicals work and how they damage cells. The way viruses work is by being present in the actual body or in the actual cell in order for them to cause any kind of disease or any kind of trouble. If you think of, again, all other --

THE COURT: Why is that not the case with Roundup? Then why is it not the case with Roundup that it needs to be present in the system to cause any kind of trouble?

THE WITNESS: It is not a virus. The way Roundup works is by --

THE COURT: What I need is a better explanation of why the two act on cells differently in a way that matters from the standpoint of NHL latency.

THE WITNESS: It is just how viruses work. It is something that we actually -- you can try to think of other viruses and think of them different than cancer or latency. I mean, there are other viruses that we get exposed to. In order for the virus to cause any damage -- what is GI distress, anything that is very simple, if the virus -- patients are not going to have traveler's diarrhea or a problem from a virus unless the virus is present. Once the virus is out, usually the symptoms subside; and the symptoms go away. I see what you are saying.

THE COURT: I understand once the virus is out, the

symptoms of hepatitis C go away. I thought the point was that the virus while present does damage to the cells in a way that causes NHL. So what is it about the way that the virus damages the cells that is different from the way that glyphosate damages the cells that means within the case of glyphosate, NHL can come up can be diagnosed ten years later; but in the case of the virus it can't?

THE WITNESS: Because the viruses -- the viruses have to have this persistent continuous damage to the cell in order to cause oncogenesis or lymphomagenesis. In order for viruses to be implicated, they still have to be present in order for them to cause the cancer.

Let's take an example for non --

THE COURT: The virus has been damaging Mr. Hardeman's cells for 35 years; right?

THE WITNESS: But it didn't cause anything for these 35 years. That's what I was trying to explain to you how I ruled out hepatitis C.

For 35 years, as the virus was actually active, we can agree it was active. It was present in the body. It was in the cells. It was causing some damage. Somehow nothing happened before 2005 despite what the virus was trying to do.

What now we are trying to say, that after 35 years of this virus failing to cause NHL in 2005, so we have a good latency period of 35 years, but we're still not convinced. We are

going to take 45 years and we're going to implicate this despite the fact that the virus is no longer present, it's eradicated, and we can't detect it.

THE COURT: What about a different scenario when, let's say, somebody was disinfected with hep C in 1980, they were treated in 1990, so they had active hep C for 10 years, and then in 1995 they're diagnosed with NHL? Would you say the same thing, that the NHL could not have been caused by the hep C?

THE WITNESS: Your Honor, I'll have to look at obviously every case individually and separately. From my understanding your question is the only difference in the example you gave me was the -- it's a 10-year period, 1980 to 1990, and it's shorter period between the eradication of the virus and the diagnosis.

I will have to look at every case differently and factor in other risk factors for this particular patient, but it is truly my belief and my opinion that if you treat the virus effectively and adequately and eradicate that virus, the risk is substantially lower, significantly lower. It's really why we treat -- it is the reason why we treat these viruses.

If there is -- if there is a belief -- and we all know how expensive hepatitis C therapy could be with -- you know, I mean, there are newer therapies for hepatitis C right now, and so forth, than the ones earlier on; but the point is if we

really believe that after a particular cutoff point, binary point, whatever that binary cutoff point is, our treatment is not going to reduce the risk of NHL or liver cancer or cirrhosis, then you should -- we should look ourselves in the mirror and ask "Why am I treating the hepatitis C if I don't really think it's going to treat this?"

The reason we do that is because we believe that eradicating that virus reduces these risks. How does it do that? Because you eliminate the offending agent from the cell and the continuous exposure to the cell, which is very different than chemicals, chemotherapy and occupational hazards and so forth.

It's just -- I mean, it's just the way how things work differently when you talk about the cell, but it's really why we treat these patients because we eliminate or diminish the risk significantly.

I mean, the reason we don't treat other viruses --

THE COURT: That doesn't -- I mean, the question I'm trying to get an answer to is how. How is the damage from the virus to the cells different than damage from glyphosate to the cells such that we can assume that NHL was caused by glyphosate use that stopped 10 years ago but we cannot assume that NHL was caused by a virus whose activity stopped 10 years ago? I need a better explanation of how -- why the -- you just keep saying because it operates differently.

THE WITNESS: Well, because --

THE COURT: I would like either an explanation of or a citation to literature which explains how it operates differently and why it matters from this standpoint.

THE WITNESS: Not all viruses cause genotoxicity or oxidated stress. What I said is that the way viruses operate differently is when they are virulent and they are present, they could cause cell replication in a way that overcomes the balance between cell survival and cell death. I mean, that's how -- this is how viruses work. This is -- I mean, they don't really elicit the problem on a cellular level if they are completely gone, if they are no longer present.

I mean, I know I'm probably not answering you, but it's just the mechanism of how viruses work on the cellular level are very different than the mechanisms that other compounds work on the cellular level.

The viruses have to be present, virulent, and because how they tip the balance between cell survival and cell death, and that imbalance is what leads to the potential development of cancer.

There are a lot of basic science involved in this but that basic science -- I mean, I'm not a basic scientist *per se*. I just know that there's a lot of research into the mechanisms by which these viruses cause the cancer. And most of these mechanisms usually take patients who are already still having

NHL and they still have HCV, and they try to make a cell line and study that cell line in the lab to try to understand how this is actually happening.

THE COURT: All right. Anything further?

MR. STEKLOFF: Nothing further, Your Honor.

THE COURT: Okay.

Oh, it's 10 to 1:00. So what do you want to do? Do you want to take a lunch break or how much time do you have?

MS. WAGSTAFF: I mean, I think I have just about five minutes or so.

THE COURT: Okay.

MS. WAGSTAFF: And I know he's got a plane to catch so I'd like to be able to talk to my co-counsel for a few minutes and then maybe spend five or ten minutes with him --

THE COURT: Is that okay with everybody?

MS. WAGSTAFF: -- instead of the lunch break.

MR. STEKLOFF: That's fine with us, Your Honor.

THE COURT: Okay. Thank you.

THE CLERK: Court is in recess.

(Recess taken at 12:51 p.m.)

(Proceedings resumed at 1:03 p.m.)

THE COURT: Okay. You can proceed.

REDIRECT EXAMINATION

BY MS. WAGSTAFF:

Q. All right. Dr. Nabhan, you would agree with me that a

genotoxic chemical causes DNA mutations; correct?

A. Yes.

Q. And that is damage at the DNA level; correct?

A. Correct.

Q. Which is different than damage at the cellular level; correct?

A. Yes.

Q. All right. And DNA mutations, meaning when the DNA is changed, those are permanent changes; correct?

A. Usually, yes.

Q. Okay. And it's your opinion that those DNA mutations don't go away when the genotoxic chemical is removed; correct?

A. Not usually because you've already had several hits if you continue to be exposed to that offending agent.

Q. Okay. And it's your opinion that glyphosate is genotoxic --

THE COURT: Why don't you ask him what his opinion is --

MS. WAGSTAFF: Okay.

Q. What is --

THE COURT: -- instead of telling him what his opinions are. I mean, this is pretty obnoxious.

BY MS. WAGSTAFF:

Q. All right. Is it your opinion that glyphosate is genotoxic?

A. Yes.

Q. All right. And you testified earlier when you were talking to the Court that not all viruses were genotoxic is what I wrote down.

A. It's my belief that viruses work differently than compounds that cause genotoxicity. The viruses have to replicate and have to be present on the intracellular level to cause the particular damage that they usually cause. If they are no longer present, that damage can -- you know, doesn't exist.

Q. Okay. So --

A. They just work differently.

Q. All right. So if you -- when there's damage at the cellular level, when you remove the offending agent, do the cells repair themselves?

MR. STEKLOFF: Your Honor, can we ask better questions?

THE COURT: What?

MR. STEKLOFF: Objection. Leading to all these questions.

THE COURT: Well, that question actually was not leading.

I mean, I think there's a real question here about the witness was not able to answer my questions, and now the lawyer is feeding the witness the answers to my questions, and I think

there's a possibility that those answers will simply need to be excluded.

And, actually, what I will say now is the answers to those leading questions are going to be excluded. So you might want to start over. All that testimony is stricken, and you might want to start over without --

MS. WAGSTAFF: Okay.

THE COURT: Normally I don't care that much when you're talking to an expert, but since you're feeding the expert answers that he wasn't able to give me in response to my questions, I think it's particularly inappropriate here. And so that --

MS. WAGSTAFF: Okay. I'll start over.

THE COURT: -- so his entire testimony on redirect is stricken, and you can start over.

BY MS. WAGSTAFF:

Q. Okay. What effects does -- can you tell the Court what "genotoxicity" means?

A. And I tried to explain it. Maybe I didn't really articulate that. But, again, there are differences in how viruses work, for example, and how compounds that cause genotoxicity usually work.

So the -- you know, whenever you have damage and chromosomal breakage and DNA damage to -- from exposure to a particular compound, that's how I view genotoxicity is. So

you're actually having damage on the chromosomal level.

Q. All right. And so are the -- is DNA damage permanent?

A. Sometimes it could be and sometimes it's not. I mean, there are situations where the cell is able to repair certain DNA damage, and so you could see that occasionally someone may be exposed to a particular compound or a toxin but the repair -- the mechanisms of how the cells repair themselves are still intact and they may actually work, and sometimes it's not. And usually it's not if you have the continued exposure to a particular offending agent that continued exposure lead to affecting the cellular mechanisms of how they repair themselves.

A lot of the cancers develop when the cellular mechanisms to repair the ability of imbalance between growth and cell death is no longer there, whether it's a mutation, whether it's a gene that is overexpressed, underexpressed; but ultimately something happens that leads to that balance between cell survival and cell death to be affected or impacted.

With continued exposure to particular toxins, to particular agents, that mechanism of cell repair is impaired, and that's why sometimes you see that in particular toxins.

In viruses, it's different, as I explained, but it just seems my explanation is not adequate enough. What I said in viruses, if the virus is no longer there, the ability of the virus to cause that damage is no longer present.

Now, the question becomes: Could the viruses cause damage that is already permanent, that it doesn't matter if they are there or not? And my opinion is not. They have to be present to continue to cause that damage, but that's what I said.

Q. And is that because the virus damages at the cellular level?

MR. STEKLOFF: Objection, Your Honor.

THE COURT: Sustained.

BY MS. WAGSTAFF:

Q. All right. Does the virus cause damage at a cellular level or at the DNA level?

MR. STEKLOFF: Objection, Your Honor.

THE COURT: Overruled.

THE WITNESS: Again, the most viruses when they cause damages, they cause damages on the cellular level. They're not really necessarily causing the chromosomal breakage and the chromosomal aberration and the genotoxicity, and that's why there's a critical difference in how viruses cause oncogenesis, which is development of cancer, versus other compounds that may be implicated in causing cancers.

BY MS. WAGSTAFF:

Q. So when damage occurs at the cellular level and the offending agent is removed, what happens to the cells?

A. Most cells are able to repair themselves. I mean, again, that's really where the issue is.

When you remove these offending agents, such as viruses, you might be able to repair. In fact, we went over several studies earlier and there are many others where if you treat -- again, if you treat sometimes the virus and you don't treat the cancer, so you just treat the virus, and there are examples for HCV as well, you might have regression of the actual lymphoma.

So there are studies that look at treating HCV or look at HCV-associated NHL, that are several studies that treated the HCV alone without treating the lymphoma and some of these lymphomas regressed and remission occurred because you're treating the underlying virus. Because the way the viruses work, they have not caused a permanent genotoxic damage that you cannot repair. It's just the way they work.

MS. WAGSTAFF: All right. No further questions.

THE COURT: Could I ask you? When looking at Monsanto's binder of studies --

THE WITNESS: Which one, Your Honor?

THE COURT: It should be Binder Number 3, I think.

THE WITNESS: Okay.

THE COURT: Can you pull up Exhibit Number 2052?

THE WITNESS: (Witness examines document.) Yes.

THE COURT: Have you reviewed this study?

THE WITNESS: I have not looked at this study before.

THE COURT: Okay. Well, I will ask you. You can take your time and look at it, but what I want to point you to is

page 98 of that study. And if you look at Figure 4 on page 98, it talks about alternative mechanisms of transformation.

And I was wondering if you could take a couple minutes to look at that chart, look at the paper to the extent you need to, and see if you can explain that to me.

THE WITNESS: Okay. (Witness examines document.)

THE COURT: And if you can't, that's fine. I'm just curious if --

THE WITNESS: Sure. I'm just reading the abstract first. Then I'll look at the figure if it's okay.

THE COURT: Take your time.

THE WITNESS: (Witness examines document.) I'm done with the abstract. I'm going to look at the figure right now.

THE COURT: Take your time.

THE WITNESS: (Witness examines document.) Okay, Your Honor. I can try my best to explain.

THE COURT: Okay.

THE WITNESS: So, you know, again in the abstract, the authors just acknowledge the fact that we're still not really sure what -- how it causes -- how -- what's the mechanism so they talk about, again, just to mention --

THE COURT: Mechanism by which hep C causes NHL?

THE WITNESS: Right. So they talk (reading):

"Pathophysiological processes at stake leading from HCV infection to overt lymphoma still need to be further

elucidated."

So at least they acknowledge. This is 2018 paper. So as of just a year ago, still that mechanism -- these mechanisms are under investigation.

They acknowledge three mechanisms essentially. One of them is the chronic antigenic stimulation that usually occur. And the chronic antigenic stimulation, it means that there is an actual virus present that causes this continued exposure to the actual cell.

So you have -- on the figure that you point out, Figure 4, on the left-hand side you have chronic infections or the infection is present. You have sustained B-cell activation so because the virus is present, it continues -- and that was one of the things I mentioned earlier -- you continue to have this B-cell activation. In essence, once you remove the infection, that activation is no longer present so -- you know, but that's one theory.

So you have the continued activation, chronic antigenic stimulation, and that leads to -- somehow to lymphomagenesis. And they had an arrow to NOTCH pathway mutations with a question mark because at some point some of these low-grade lymphomas that occur might have something that lead to transformation. We don't know actually what transforms them. The rates of transformation is about 5 to 10 percent per year; but, you know, they're suggesting maybe some cellular pathway

that gets mutated or affected that lead to the transformation.

THE COURT: So would that reflect the point at which you might still slightly later be diagnosed with NHL even after you've been treated for the virus?

THE WITNESS: No. This actually doesn't -- this suggests that you need to have the chronic stimulation. So you have to be able to have constant stimulation of these cells with the chronic hepatitis C infection in order for you to develop the marginal zone lymphoma.

And what it's saying, that if you develop low-grade lymphoma, something might occur later on to transform into DLBCL because we know that patients with indolent lymphomas, such as marginal zone, could transform to a more aggressive lymphoma such as DLBCL.

THE COURT: Right. But what I'm saying if you develop low-grade lymphoma, it might transform to DLBCL even if you've already been treated for your hep C.

THE WITNESS: Yes, you could. Yes.

THE COURT: All right.

THE WITNESS: Transformation does occur at the rate of about 5 to 10 percent per year.

THE COURT: Okay.

THE WITNESS: If you look at the right side, it's a little bit of a different hypothesis, and they acknowledge it's a little bit highly speculative on the page before, which is

"HCV-Positive FL: A third pathogenetic pathway?" And they say it's highly speculative but they propose it.

And what they essentially say is that because they found cells in patients who are HCV infected who have the BCL2 oncogene expression, which is usually in patients who have the 14, 18 chromosomal translocation, see, they speculate the hepatitis C infection through chronic inflammation would favor the GC re-entries.

So, in other words, there are patients who already have the -- what they're trying to look at, Your Honor, just to be clear, they're trying to look at the transformation. So that's why they have marginal zone on the left and they have follicular lymphoma on the right. So these are two indolent type of lymphomas, and they're trying to see how HCV might be implicated in the transformation process versus the one in the middle, which is *de novo* DLBCL.

I just want to make sure I clarify that. So on the left side it was chronic antigenic stimulation. It's there stimulating the B cells, mild- to low-grade marginal zone, and then we don't know why it could transform.

I've seen patients who transform 5 to 10 percent per year. That's what the literature supports, and they're saying maybe there's a pathway that gets mutated although it's not proven.

On the right-hand side they're looking at a different type of indolent lymphoma, which is follicular lymphoma. And just

to -- I will say follicular lymphoma patients usually have the 14, 18 chromosomal translocation. The 14, 18 chromosomal translocation leads to overexpression of BCL2, which you see in the right on top. BCL2 is a proto-oncogene. So when it is present because of the 14, 18 chromosomal translocation, it leads to overgrowth of cells.

So what they're saying now, we have these B cells, somebody already have these cells, and we see that they have presence of the BCL2 and then they have the chronic infection with HCV and they have GC re-entries.

So somehow there is an environment of inflammation because of the presence of the HCV and antigenic stimulation that allows these BCL2 cells, the 14, 18 cells, to keep re-entering and not leave. And by doing that, they lead to the development of follicular lymphoma and then something happens that might lead the transformation into DLBCL.

But I have to look at what this AID question mark is, the one on the right. They don't say what AID is. One second.

(Witness examines document.) Okay. But -- I'll look at the AID, but the point is that follicular lymphoma could transform also to DLBCL because this is maybe another pathway or something that just leads to the transformation.

THE COURT: Okay.

THE WITNESS: The theory in the middle, I believe, it looks at the possibility of looking at direct transformation

and involvement into patients with DLBCL, which is not the two theories they are proposing.

So the one on the left and the one on the right are the two novel mechanisms that they believe they may be causing -- these are the two theories that they are bringing in and they're saying maybe there is -- there are these two alternative theories that we are bringing in.

So if you look on page 96, they have HCV-positive marginal zone and DLBCL, two distinct models of HCV-related lymphomagenesis. And they talk in the second paragraph it's now established that chronic external stimulation leading to protracted stimulation of antigen-specific B cells clones is likely to constitute the main driving mechanism in marginal zone lymphoma.

So you just really need that chronic antigenic stimulation, which, when you treat it, you cannot take out. You don't have this chronic antigenic stimulation anymore after therapy.

So the third paragraph they talk alternative pathway of transformation based on direct HCV infection of the B cells. So if you don't have HCV infection, you can't really infect the B cells. So their theory is based on direct HCV infection of B cells especially in HCV-positive *de novo* DLBCL subgroup.

THE COURT: Does that paragraph reflect the middle chart in Figure 4?

THE WITNESS: Yes, I believe so.

THE COURT: Okay. So could you explain that to me?

THE WITNESS: So, they are -- so, again --

THE COURT: If you can.

THE WITNESS: I'm talking --

THE COURT: You're only just glancing at this paper now.

THE WITNESS: Well, it says (reading):

"Mixed cryoglobulinemia" -- and they say, "Mixed cryoglobulinemia, rheumatoid factor, and VH1 to 69 positive and VK3-20/15 restriction usage are indeed unusual features of *de novo* DLBCL."

You know, all I can say, Your Honor, is on page 97 the first -- the first paragraph and the last sentence before the HCV-positive FL, it says (reading):

"Finally the presence of viral proteins has been detected in tumor cells of HCV-positive DLBCL."

I'm not sure I can explain right now the middle figure that you showed me. I explained the right side and the left side, but I think the authors also acknowledge that the presence of these viral proteins has been detected in tumor cells of HCV-positive DLBCL, which once the HCV is treated, you really can't detect that virus.

It's really in line with my knowledge, as well as my training, into how viruses cause lymphomas or cancers in

general.

THE COURT: Okay. All right.

Does anybody else want to ask any follow-up questions in the wake of that before we wrap up?

MR. STEKLOFF: Can I just quickly follow-up if Ms. Wagstaff is done?

MS. WAGSTAFF: Yes.

RECROSS-EXAMINATION

BY MR. STEKLOFF:

Q. I just want to ask -- we've had a lot of talk about mechanisms of action. I want to ask a very simple question.

You agree that regardless of the exact mechanism, hepatitis C can cause genetic mutations that become cancerous; correct?

A. Yes, it can.

Q. And you agree that the longer an individual is exposed to hepatitis C, the more likely he or she is to have those genetic mutations occur; correct?

A. I believe it can, yeah.

Q. Okay. And you also agree that the exact mechanism by which glyphosate in your opinion contributes to the development of non-Hodgkin lymphoma is not entirely clear; right?

A. There are theories, but you're right. I mean, I don't think we know hundred percent the mechanisms of a lot of things, including how Roundup causes non-Hodgkin lymphoma.

MR. STEKLOFF: No further questions, Your Honor.

THE COURT: Okay. Thank you, Dr. Nabhan. You may step down. Hopefully you'll catch your flight.

THE WITNESS: Thank you, Your Honor.

(Witness excused.)

THE COURT: And is there anything else for us to discuss right now before we -- we meet again next Monday; is that right?

MS. MOORE: Your Honor, Jennifer Moore.

We just had a couple of housekeeping matters if it's okay with the Court.

One, we wanted to know if Your Honor had a chance to look at the jury questionnaires because we have to submit our *voir dire* questions on Wednesday, and it would be helpful to have that before we submit the *voir dire* questions.

THE COURT: I think I looked at a final -- did I sign off on a final version? No?

Oh, apparently not. So I'll get on that today.

MS. MOORE: Okay. Well, I mean, if -- as long as we have it before Wednesday, that's fine, Your Honor. Thank you.

THE COURT: Okay.

MS. MOORE: And as part of your order, we are supposed to submit jury instructions and verdict form on Wednesday. I know we've already done that for Phase I, and I assume we're going to be arguing that if there's any additional questions on

the 13th. Do you want us to submit Phase II instructions on Wednesday?

THE COURT: No. You don't have to do that. We'll deal with that later.

MS. MOORE: Okay. Thank you, Your Honor.

And then is Your Honor anticipating any time limits on Wednesday the 13th as far as the motion for summary judgment or *Daubert* arguments?

THE COURT: I'm not sure I'm in a position yet to give you guidance on that. What time are we supposed to meet on that day?

MS. MOORE: I believe 9:30 in the morning for summary judgment and *Daubert*, Your Honor.

THE COURT: And then are we meeting for summary judgment and *Daubert*, and then we were planning on the afternoon for the *motions in limine*?

MS. MOORE: That's correct, Your Honor.

THE COURT: Yeah. I mean, well, plan on coming at 9:30 and we'll address whatever we need to -- we'll begin addressing whatever needs to be addressed at 9:30.

I may decide -- I mean, there are certain issues -- certain summary judgment issues that I am not going to hear argument on. For example, I highly doubt that I will hear argument on the failure to warn motion. There may be others that I don't need to hear argument on.

I suspect I will want to hear argument on the specific causation motions. And I haven't really -- I've only started glancing at the motions to exclude the other experts so I'm not really sure about that yet.

MS. MOORE: Could we address that again on Monday, Your Honor, just to help assess with the preparation for Wednesday?

THE COURT: Yes. I can hopefully give you some further guidance on Monday.

MS. MOORE: That would be really helpful.

And then in the afternoon you mentioned *motions in limine*. We also -- I guess Brian and I talked -- Mr. Stekloff and I talked about this, but we have our joint exhibit list that we're submitting to the Court this Wednesday. It is roughly 11- or 1200 exhibits, and so we may meet --

THE COURT: Is that all?

MS. MOORE: Well, on the will-use list, Your Honor. On the may-use list, I can't even tell you the total number on that.

But one thing that we were talking about was to meet and confer to see if we could come up with categories, and that may be the best way to approach that if that would work with Your Honor.

THE COURT: Yes. That would be good.

MS. MOORE: Okay.

THE COURT: If you can identify a couple of important categories from that list where it would be particularly helpful to resolve it in advance and stuff that you actually know is going to come up.

MS. MOORE: Right.

THE COURT: 98 percent of what you've put in that exhibit list is not going to come in --

MS. MOORE: I understand, Your Honor.

THE COURT: -- by your own choice. So try to focus on the things that you know are really going to be an issue.

MS. MOORE: And then the last couple of housekeeping things, Your Honor, is on depo designations, we're submitting those on February the 18th to the Court; and if there are any unresolved objections, we'll try to work those out before. When would you anticipate us arguing that? Would that be during the trial? Just for planning purposes.

THE COURT: I don't really know. Let's see, you said you're submitting your depo designations by the 18th?

MS. MOORE: Yes.

THE COURT: The 18th is Presidents Day so you want to submit them the 19th or the 15th. That might be better for your staff.

MS. MOORE: They probably would prefer many things, Your Honor, but they're not getting that right now.

THE COURT: I mean, I don't know. That's sort of a

hard question to answer without knowing how much dispute there's going to be --

MS. MOORE: I know.

THE COURT: -- and the volume that we're talking about.

MS. MOORE: We're doing our first exchange today actually. So we may be able to be in a better position on the 13th to bring it up with Your Honor, but we just would -- that's something we may need to do piecemeal too. I just want you to be aware of it.

THE COURT: Yeah. But if we have time on the 13th to start getting into that stuff, we can do that.

MS. MOORE: Okay. That would be great, Your Honor.

And then the last thing that I have is for Dr. Portier. We did speak on the break. One thing we would be amenable to is to go ahead and do what really would be essentially a trial deposition of Dr. Portier that week before trial starts.

If Your Honor would be available, if there are -- I mean, I'm not talking about really minor things, but just objections. If we could have a way to contact you, it wouldn't be in the middle of the night, Your Honor, but if we could work something out like that, that would help us because then once we get the transcript, we wouldn't have to go through arguing about the objections. Because for our burden of proof we do plan to call him first so we're going to be ready to roll after opening

statements on the 25th.

THE COURT: Okay. Well, if you -- but you also need to be light on your feet and not call him first if, you know, circumstances dictate that, but I can make myself available. You can work with Kristen on sort of figuring out when I will be available.

MS. MOORE: Okay.

THE COURT: But I assume for the most part you're just going to be making your objections to preserve them and only if it's something big, you're going to be calling me.

MS. MOORE: That's what I would anticipate, Your Honor. We wouldn't take your time if it wasn't something on a larger scale.

And then we'll work out as far as when that -- when the actual deposition would occur that week, whether it's Tuesday, Wednesday, Thursday or Wednesday, Thursday, Friday, and we would let the Court know through Ms. Mellen.

THE COURT: Now, obviously on, you know, examining experts, you know, generally speaking I've tolerated more leading questions of experts. I shut this segment down this afternoon, but generally speaking I've been pretty tolerant of leading questions of experts during the *Daubert* hearings. I will obviously be a lot less tolerant of that at trial.

And, you know, I think that, particularly in situations where, you know, nobody is challenging the qualifications of

the experts, and I think that's largely the case here is that the qualifications of the respective experts are not being challenged --

MS. MOORE: Right.

THE COURT: -- I don't have a problem with asking leading questions to make it quicker to get those qualifications in and maybe some of the other general background stuff, like high-level general background stuff.

MS. MOORE: I understand.

THE COURT: But after that, you know, if you ask leading questions of Dr. Portier on the stuff that matters, I'm going to -- I'm not going to allow it in. Okay?

MS. MOORE: I understand, Your Honor. So when we get to the substance, I get it. But for time purposes, especially since we have time limitations, so we can help move the record along, that would be great.

THE COURT: Yes.

MS. MOORE: The only other thing, I did have one other thing -- and I apologize, Your Honor -- is that last Monday at the *Daubert* hearing the defense submitted a bench memo on -- or maybe it wasn't last Monday. I apologize if it wasn't last Monday -- on substantial factor, and we do want the opportunity to respond to that before you rule on that Phase I jury instruction. Could we have until Friday of this week to file a response?

THE COURT: Sure.

MS. MOORE: Okay.

THE COURT: I'm not sure it's necessary but, yes, you can file a response on Friday.

MS. MOORE: Okay. Great. Thank you, Your Honor.

MR. STEKLOFF: And from my perspective, Your Honor, the only question I have is it sounds like we're down this path, which I understand, that with Dr. Portier, while we would prefer to have you involved, given the timing, we should just proceed down this path.

THE COURT: That's right.

MR. STEKLOFF: Okay. If I didn't ask, people might wonder why I didn't ask; but as long as you are available, and I'm sure the parties will try to limit, maybe not even call you at all, but limit the amount of time they spend with you. That's a good compromise, I think, from our perspective.

THE COURT: Okay.

MS. MOORE: Thank you, Your Honor.

THE COURT: Thank you. We'll see you next week.

ALL: Thank you, Your Honor.

THE CLERK: Court is adjourned.

(Proceedings adjourned at 1:33 p.m.)

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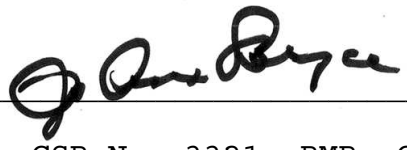
CERTIFICATE OF REPORTERS

I certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

DATE: Monday, February 4, 2019



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