Exhibit 4

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	MDL No. 2741 Case No. 16-md-02741-VC
This document relates to: ALL ACTIONS	

VIDEOTAPED DEPOSITION OF DR. CHADI NABHAN
Waukegan, Illinois
Wednesday, August 23, 2017

Reported by:
PAULA CAMPBELL, CSR, RDR, CRR, CRC
JOB NO. 127897

August 23, 2017
9:07 A.M.

Videotaped discovery deposition of DR. CHADI NABHAN, held at the offices of CARDINAL HEALTH, 3651 Birchwood Drive, Waukegan, Illinois, pursuant to notice before Paula Campbell, CSR, RDR, CRR, CRC.

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ALSO PRESENT:
Robert Zellner, Videographer

---------- I N D E X ----------

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NABHAN	PAGE	LINE
Exhibit 5 entitled "Evaluation of carcinogenic potential of the herbicide glyphosate drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies" by Greim, et al.	75	11
Exhibit 6 IARC Monograph on glyphosate	99	21
Exhibit 7 article entitled, "Key characteristics of carcinogens as a basis for organizing data on mechanisms of carcinogenesis, by Smith, et al.	119	3

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Exhibit 8 Toxicology and Applied Pharmacology article entitled, "Oxidative stress and oxidative damage in chemical carcinogenesis," by Klaunig, et al.	130	19
Exhibit 9 article entitled, "Baseline determinatino in social, health, and genetic areas in communities affected by glyphosate aerial spraying on the northearstern Ecuadorian border," by Paz-y-Mino, et al.	139	18

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Exhibit 10 article entitled, "Biomonitoring of genotoxic risk in agricultural workers from five Columbian regions: Association to Occupational exposure to glyphosate," by Bolognesi, et al.	142	15
Exhibit 11 article entitled, "Cancer epidemiology, biomarkers & prevention," by McDuffie, et al.	166	14
Exhibit 12 article entitled, "Exposure to pesticides as risk factor for non-Hodgkin's lymphoma and hairy cell leukemia: Pooled analysis of two Swedish case-control studies," by Hardell, et al.	197	10

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Exhibit 13 article entitled, "Integrative assessment of multiple pesticides as risk factors for non-Hodgkin's lymphoma among men," by DeRoos, et al.	205	7
Exhibit 14 article entitled, "Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among Men in Iowa and Minnesota," by Cantor, et al.	207	1
Exhibit 15 article entitled, "Non-Hodgkin's lymphoma among asthmatics exposed to pesticides," by Lee, et al.	213	4

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VIDEOGRAPHER: Good morning. This is the start of tape labeled No. 1 of the videotape deposition of Dr. Chadi Nabhan in the matter of In re: Roundup Products Liability Litigation in the United States District Court for the Northern District of California, bearing MDL No. 2741, Case No. 16-md-02741-VC.

This deposition is being held Cardinal Health at 3651 Birchwood Drive in Waukegan, Illinois 60085, on Wednesday, August 23rd, 2017, at approximately 9:07 A.M.

My name is Robert Solomon from TSG Reporting, Inc., and I'm the legal video specialist. And the court reporter is Paula Campbell, in association with TSG Reporting.

And will counsel now please introduce yourselves for the record.

MR. LITZENBURG: Timothy Litzenburg for the plaintiffs.

MS. TABATABAIE: Tara Tabatabaie for the plaintiffs.

MR. GRIFFIS: Kirby Griffis for -- with Hollingsworth, LLP, for Monsanto.

MS. SALEK: Stephanie Salek with Hollingsworth, LLP, for Monsanto.

VIDEOGRAPHER: Thank you. And will the court reporter --

MS. ROBERTSON: Pearl Robertson with Weitz & Luxenberg for the plaintiff.

REPORTER: I'm sorry. I didn't -- can you repeat?

MS. TABATABAIE: Can you repeat that?

MS. ROBERTSON: Yes. Pearl Robertson with Weitz & Luxenberg for plaintiff.

VIDEOGRAPHER: Thank you.

And will the court reporter please swear in the witness.

REPORTER: Would you please raise your right hand.

C H A D I N A B H A N,

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

VIDEOGRAPHER: I may be picking up a cell phone in your pocket. If you have one, if you wouldn't mind putting it off to the side, as far as you can do it. Thank you so much. Thank you.

EXAMINATION

BY MR. GRIFFIS:

Q. Good morning, sir.

A. Good morning.

Q. My name is Kirby Griffis, and we have just met; is that correct?

A. Correct.

Q. Would you please pronounce your name for the jury? I want to get it right today.

A. Chadi, C-h-a-d-i, is my first name. Nabhan, N-a-b-h-a-n, is my last name.

Q. Chadi Nabhan -- Nabhan?

A. Correct.

Q. Thank you.

(Nabhan Exhibit 1 marked for identification.)

Q. I've marked as Exhibit 1 and I'm handing you a copy of your current CV.

Have I correctly identified that document, sir?

A. Correct.

Q. Okay. The "Summary" section at the top of your CV in bold says that you are vice president and chief medical officer of an $11 billion division in a Fortune 15 company; is that right?

A. Correct.

Q. What is the division?

A. Specialty Solutions.

Q. And what is the Fortune 15 company?

A. Cardinal Health. That's my current position.

Q. What does -- okay. It's your current position.

Has your position changed?

A. No, no.

Q. What is a Fortune 15 company? What does that mean?

A. Fortune magazine, they have a list of the companies every year that they come up with, and they reflect 500 of the top companies in the U.S.

Q. And they're top companies in what way?

A. I think they have a variety of metrics. I'm not really sure what they are. I have not looked at the metrics per se that they use. But could be sales, revenue, culture, employee retention. I'm not really clear what they use.

Q. Okay. The next bullet says that you're a senior level executive and a member of the operating company, reporting directly to the president; correct?

A. Correct.

Q. Under "Professional Experience," you list the positions that you've held in the past and

currently.

A. Correct.

Q. This is also on the first page.

And the first one is your current position that you've just been describing, vice president and chief medical officer of Cardinal Health; is that right?

A. Correct.

Q. And underneath that, there are 16 bullets describing your various duties as someone who reports to the president of Cardinal Health Specialty Solutions; is that right?

A. Correct.

Q. How much time do you spend in your current position, sir, on this job on business and administrative tasks?

A. About 80 percent and 20 percent research.

Q. And how much time seeing cancer patients?

A. At this point, I'm not seeing patients by choice. It's been about 11 months since I've seen actual patients because of my travel schedule.

I have the flexibility of having a clinic or seeing patients if I choose to. It's been very challenging with my travel to make sure that I can have a dedicated day for clinic. I don't want to

shortchange my patients and cancel clinic because of short notice, so this is still in the works.

Q. So at the time -- at this time, it's been 11 months since you've seen a patient?

A. That is correct.

Q. And --

A. I continue, however, to, you know, lecture, publish, and work on the field; but I have not seen an actual patient in 11 months.

Q. Yes, sir.

You said 20 percent of your time is on research; right?

A. Correct.

Q. Cardinal Health Specialty Solutions, would you describe that as a service provider to hospitals and doctors' offices?

A. Hospitals, biopharma, and doctors, yes.

Q. And it provides help with all sorts of logistical things with supply chains, with billing, with administration, all sorts of --

A. Yeah, I mean --

Q. -- difficulties?

A. I think, you know, there are -- again, there are two major segments within Cardinal Health. One, the medical segment that works a lot with

supply chain hospitals, and so forth. And there's the biopharma segment to work with providers as well as with biopharma, providing a lot of logistical help as well as educational platforms, helping with billing, et cetera.

Q. You recently got an MBA; is that right?

A. It's been a year.

Q. Okay. Not recently?

A. That's recent. No, it's recent, 2016.

Q. And you got an MBA, I presume, in support of your current role as a business person; is that right?

A. I actually decided to go back on the -- to get my MBA when I was at the University of Chicago as the director of the cancer center -- the clinical cancer center and cancer clinics. And I wanted to better understand the economics, business, accountings, which will help in my role at the time.

So I got the MBA focusing on healthcare management. My goal was to help more patients at a larger scale. And, you know, this opportunity came along after the fact that I was already on the MBA. This was not -- my current role is not why I got the MBA. That is -- I got -- I went back to school in August 2014. I was still at the University of

Chicago.

My goal was just to better understand business of medicine. I think what's going on in medicine is very important for physicians to take lead into understanding business and the impact on patients.

Q. It reflected a shift in your interest from patient care to a more broad administration and business side and serving medicine through that means. Is that fair to say?

A. No, I don't think it's fair to say. I think -- I think delivering patient care is both sides, right. I mean, I think when you take care of patients in clinic, you still have to bill for services. You have to run a business.

So being able to deliver quality care to patients implies that you know how to run your business.

Q. Yes, sir.

And you're focused now --

A. So I think it's important to do both.

Q. You're focused now on the business side?

A. I am focused on the business side, but I don't think it's irrelevant to patient care.

Q. You, sir, are not an epidemiologist, and

you never were one; is that right?

A. Correct.

Q. You're not a toxicologist, and you never were one; right?

A. Correct.

Q. You don't call yourself an expert in the mechanisms of carcinogenesis; is that right?

A. I'm not an expert in the mechanism of carcinogenesis. I can understand the papers that discuss carcinogenesis, and I try my best to look into how this might imply clinical decisions in clinical care.

(Nabhan Exhibit 2 marked for identification.)

Q. I've marked as an -- Exhibit 2 a May 16th, 2017, letter from Weitz & Luxenberg to Heather Pigman at Hollingsworth, LLP, sir. And the -- I will read the letter. You will follow along with me and make sure I get it right.

"Dear, Heather: To follow up on our letter dated May 3rd, 2017, and to respond to your inquiry about our expert specialties, we provide the following information."

And then there is a list of six experts, including yourself, with a very brief description of

their specialties; is that right, sir?
A. Correct.
Q. For you, it says "oncology" and "NHL," non-Hodgkin's lymphoma; is that right?
A. Correct.
Q. Does that accurately reflect your understanding of your role in this litigation?
A. Yes.
Q. And you know that there are epidemiologists and toxicologists who have also been named as experts for the plaintiffs; is that right?
A. I do.
Q. And what do you -- what is your understanding of what you add to what the epidemiologists have to say and what the toxicologists have to say on the issue of whether glyphosate is capable of causing non-Hodgkin's lymphoma?
A. So I think -- I think, as somebody who took care of patients with lymphomas and a variety of lymphoid malignancies, it is very important to look at the overall body of literature and understand what might cause the disease that I'm treating.
A, it actually helps in a conversation with patients. B, it might allow the ability to be

proactive into preventing additional exposure if there's a particular pathogen that might actually -- causing an issue.
There's -- it's similar to when you take care of a patient who is a smoker and has a particular malignancy. If you reduce or stop tobacco use, you will actually prevent another malignancy that could occur. So actually understanding the epidemiologic evidence is very critical to clinical care.
Q. Yes, sir.
And you have explained, I believe, why it would be important to --
A. Right.
Q. -- a cancer doctor --
A. Correct.
Q. -- to look at some of the epidemiology and toxicology. My question a little bit different, though.
It is this: With regard to the scientific question of whether glyphosate causes non-Hodgkin's lymphoma or is capable of causing non-Hodgkin's lymphoma, once epidemiologists have spoken to that subject and toxicologists have spoken to that subject, what expertise do you bring, what

perspective do you bring to the scientific question does glyphosate cause non-Hodgkin's lymphoma?
A. Well, number one is I could interpret the evidence as well. I am very capable of looking at the literature and looking at the epidemiological literature. Just because I don't have an epidemiology degree and -- it does not mean that I cannot actually interpret the literature and look at the actual evidence.
So I -- I will -- I form my own independent review of the available literature, and I put that into clinical perspective. That's what I bring to the table.
Q. And what do you -- what can you say that an epidemiologist or toxicologist cannot say?
A. Well, I'm not a toxicologist, as we just established. I mean, a toxicologist is able to look at the -- at the evidence when the product or compound is going through the process of being approved through toxicology assays, through animal studies, et cetera.
I don't do that. I just look at the literature and review the literature.
Q. You never conducted an animal cancer bioassay; right?

A. I have not.
Q. You've never conducted an experimental genotoxicity study; right?
A. I have not.
Q. You've never conducted a study assessing the possibility that a particular chemical exposure or pharmaceutical exposure or other kind of exposure causes oxidative stress; is that right?
A. I worked -- when I was a fellow at Northwestern, I worked for three years doing bench work and lab work. And part of my work at the time was doing certain cytotoxicity assays of particular drugs to understand what they actually impact cells and on cell culture.
So we did a lot of apoptotic assays, and so forth, as part of my fellowship training. That's the extent of what I did in terms of lab work.
I'm not sure if that answers your question.
Q. Yes, sir.
Did any of those involve -- you were talking about cytotoxicity studies.
A. Cytotoxicity, apoptotic assays, and so forth.
Q. Were any of those looking at reactive oxidative species or other oxidative stress markers?

A. No, we did not -- I did not do these assays.
Q. You say in your expert report, sir, that you are a specialty in -- you have specialty in diagnosis and management.
A. Of?
Q. Patients, I presume.
A. Can you show me where that is?
Q. Certainly.
A. It seems like the sentence is truncated.
(Nabhan Exhibit 3 marked for identification.)
MR. GRIFFIS: Do you need a copy, Tim?
A. What page?
Q. One.
A. So it says, "Diagnosis and management of patients with all types of lymphoma, including non-Hodgkin's lymphoma."
Q. Yes, sir.
What do you mean by "diagnosis and management"?
A. It means I specialize in diagnosing patients who have lymphoid malignancies, because lymphomas are very heterogenous. There's not one type of lymphoma, so you really have to diagnose the

type of lymphoma the patient has because the proper diagnosis will lead to the proper management.
So once I diagnose a patient, then I will take care of designing a therapeutic regimen for that patient and implement that therapy.
Q. So your specialty, when you were seeing patients, was in diagnosing, which would include both determining that they had cancer at all and in determining which specific subtype of cancer, and here, non-Hodgkin's lymphoma that they had; is that correct?
A. I was a lymphoma specialist. So I did not see breast cancer. I did not see lung cancer. So the patients that I saw, they all had lymphoma. I had a small clinic of prostate cancer as well because I had a little bit of an interest in prostate cancer. But the bulk of the patients, I saw lymphomas.
So it's either a patient who has a known lymphoma that I will verify, confirm the diagnosis, and design a treatment plan or someone with a suspicion of lymphoma that the oncologist referring to me is not certain. And they will send to me, and I make the diagnosis.
So my area of expertise is lymphoma. I

didn't see other -- I didn't see general oncology.
Q. Yes, sir.
And you alluded to the heterogenous nature of the non-Hodgkin's lymphomas.
Would you explain that, please?
A. So, you know, every few years, there's a classification of lymphoid malignancies that changes based on, you know, better understanding of the science of lymphoma. So the last classification was actually published in the journal Blood in 2016 last year by the WHO, the World Health Organization, and pretty much divides lymphomas into almost 60, 6-0, subtypes. And it's very critical for oncologists as well as -- as well as patients to know which type of lymphoma the patient has to decide the therapy that the person needs.
So, in general, we divide lymphomas into Hodgkin and non-Hodgkin. Hodgkin lymphoma is divided, in my opinion, into two categories, classical Hodgkin lymphoma and nodular lymphocyte predominant Hodgkin lymphoma.
The non-Hodgkin lymphoma broadly is divided into B-cell lymphoma and T-cell lymphoma. And then within T-cell, you have about 20 to 25 types. Within the B-cell, you have about 40 types.

So you can see how complex it could be because each one has a different prognosis, treatment, management, et cetera.
I mean, I could group them for you, if you want, into broader categories. But for the most part, it's very important for us to know which type we're dealing with.
Q. There's also a great deal of etiologic heterogeneity in the non-Hodgkin's lymphomas; correct?
A. For some. I think, you know, there are some lymphomas, as an example, that are associated with -- that are associated with viruses, Epstein-Barr virus; CMV, cytomegalovirus; HIV; HHV; HTLV. All of these and -- you know, all of these -- HSV. All of these viruses could be associated with a particular type of lymphoma.
In general, however, when we look at epidemiology or we look at certain particular aspects, we can look at lymphomas as a collective one homogenous group despite the heterogeneity.
I mean, I can give you an analogous example. So smoking is associated with lung cancer, but there are about six types of lung cancer. But you can look at the association between tobacco and

lung cancer in general, and then you could look at other particular types.

Q. Sir, I've marked as Exhibit 4 a scientific article entitled "Comprehensive evaluation of medical conditions associated with risk of non-Hodgkin lymphoma using Medicare Claims ('MedWAS')," by Engels and others.

Are you familiar with this article from 2016?

A. I have never seen it.

Q. Take a look in the "Introduction" section, sir.

A. Sure.

Q. The second -- the third sentence reads, "Although considered a single entity for descriptive purposes, NHL comprises a group of heterogenous subtypes with distinct clinical presentations and, as is increasingly recognized, differing causal pathways, i.e., etiologic heterogeneity."

MR. LITZENBURG: I object to the questions --

Q. Do you agree with that?

MR. LITZENBURG: -- questions about something he's never seen before and did not rely on.

With that objection, you can answer if you like.

Q. Do you agree with that, sir?

A. I don't. I think -- I think there are two ways of looking at things. I think sometimes certain lymphomas could have one causal factor and some others don't. So I think it's making a blank statement that takes away, frankly, from -- from the actual clinical encounters that we see with patients.

Q. And this is based on clinical encounters rather than scientific literature, sir?

A. And scientific literature, of course.

Q. And what scientific literature says that?

A. So HIV, as an example, I'll bring that, it's a known viral infection. It could cause Hodgkin lymphoma, could cause Burkitt lymphoma, could cause diffused large B-cell lymphoma. But it's one factor.

So I think that you could look sometimes -- the variety of lymphomas, we want causality. So you can't really make a general statement that -- that every single one is different or together.

There are a variety of lymphomas, as we just talked about, the last WHO classification. And

some of them could be grouped and have one causal factor or two causal factors, and some don't. There are many lymphomas we don't even know why they happen. I mean, they just happen.

Q. Which lymphomas, that involve more than 1 percent of the total lymphomas, do we not know why they happen?

A. I don't understand the question.

MR. LITZENBURG: I object to form.

Q. Yes, sir.

What -- which specific subtypes involving -- and I don't want a microscopic subtype with -- that's only .1 percent of all the non-Hodgkin's lymphomas.

But, say, 1 percent or greater of the non-Hodgkin's lymphomas, which subtypes are unknown in their etiology?

MR. LITZENBURG: Same objection.

A. So it is my opinion that just because we have a patient in front of me that -- that has a lymphoma and I couldn't find an identifying factor that it occurred, it doesn't mean that there is no factor. It just means I'm not able to identify it at the time.

20, 30 years ago, we only thought lymphomas

were about four types. Hodgkin lymphoma was one disease. Now it's five diseases. Large-cell lymphoma was one entity. It is now about six entities.

So science does evolve and does change. So I don't know today, as I sit here, what type of lymphomas we -- you have to give me a clinical case, a particular patient situation where I'll look at all the factors and I say okay, well, with this patient, I'm not sure why this lymphoma occurred. In the other patient, I may find a reason.

There are about -- about close to 15,000 new patients with lung cancer in the United States that are never smokers. We -- when I was in training, we had no idea, actually, why would somebody with no smoking history get lung cancer.

Five, six years ago, there was a mutation that was identified that leads to a particular development of these cancers. So things evolve. I don't know -- I don't have any other answer to the question you posed.

Q. Did you tell me a few minutes ago, sir, that there are some types of non-Hodgkin's lymphoma for which the cause is unknown?

A. Yes, I did.

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Q. What are those types?
A. Again, any type of lymphoma, any type -- so you have 60 types of lymphoma. Any type of them, you may be able to identify why they occurred. Could be a chromosomal aberration, a genetic mutation, et cetera. And you may not be able to. Each case is different.
There is no particular type that you say, well, this one, I have no idea why it occurs; but, this one, I know why it occurred. In any type of lymphoma, you can't always find a predisposing factor; while in others you can. Each case is very different. I can't generalize.
Q. Lymphoma is very strongly associated with age; correct?
A. It does occur in patients who are older as opposed to younger, correct.
Q. Age is a major risk factor for all types of lymphoma; correct?
A. For all types of cancer.
Q. And that is because --
A. You don't see cancers in 30-year-olds, commonly. So I think, you know, what happens as we age is a lot of cellular disruption occurs, and you see the majority of cancers occur in patients over

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the age of 65. The majority of cancer-related deaths occur over 65, in Medicare population.
Q. And that is because of the ongoing process of cell division, cell replication, and endogenous errors creeping into that process as the years pass; correct?
A. That's only one factor. I think there are other factors that actually are involved. As we get older, whatever things that have occurred in the past start accumulate for us. So you could smoke in your 30s all you want; you probably won't get cancer until the mid 50s.
The point being is certain occupational hazards, certain factors that we've done in our youth may not actually pan out until later in age. You add this to the age and cellular division and other things, so together that's really why we see most cancers diagnosed in patients over 65 and most cancer-related deaths occur in patients over 65.
Q. What -- what is the latency period from an environmental insult -- you mentioned smoking just now, sir -- to the manifestation of a cancer?
A. It varies. It varies significantly. And I'm not sure, really, anyone could be certain or accurate in saying if it's 5, 10, 15, or 20 years.

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It's really impossible.
Because what happens is, in order for you to accurately determine a latency period, you are going to say that your exposure to whatever that is has to be constant and stable for all of these coming years. It has not to go up or down. What if you -- you know, you smoked one pack of cigarettes a day for 10 years and then you decide three packs of cigarettes a day for the next 10 years. Your latency changes. Your exposure changes.
So I don't think we can accurately predict a latency period for a malignancy that it is -- it's not a binary option. You know what I mean? It's not 5 years less or more, 10 years less or more, 15 years less or more.
Q. Yes, sir. But when you're doing something like epidemiology and relying on statistics, what latency period would you like to see in an epidemiology study before you would consider the results to be actually reflecting a possible result of the exposure that you're looking at?
A. I don't rely on the latency period per se to make a decision whether there is -- the exposure has any relation to that because every disease is different.

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So the latency period per se is not a factor, in my opinion, to make a determination in terms of exposure-related developed of disease.
Q. And it's not a factor in considering the adequacy of an epidemiology study, sir?
A. No, I didn't say that. I said it's not -- you can't take it as a binary option. You can't take it as a one factor. There are a variety of factors involved in making that determination, and the latency period, in my opinion, is not the most important factor in making that determination. That's what I said.
Q. Okay. I'm talking about epidemiology studies right now.
A. Sure.
Q. In an epidemiology study, sir, what period of time would you like to see between the exposure under consideration and the manifestation of the diseases being measured to consider that there may be a valid relationship between the exposure and the diseases?
A. So I will repeat my answer, because I already answered this. I don't believe -- I don't need a minimum or a maximum. The latency period -- there is no minimum or a maximum period that a

latency period has to have in order for you to believe that an exposure was related to a development of disease. And I will stop at that.

Q. Okay. Sir, do you know that the epidemiologists or plaintiffs in this case have criticized the agricultural health study in part for the short latency period, the -- what they call the short period of time between the exposures and the manifestation of cancer and say that's not long enough to detect cancer?

MR. LITZENBURG: I object to that characterization.

Go ahead.

A. If he did, that's his opinion.

Q. Okay. And you disagree?

A. I didn't say I disagree. Again --

Q. Do you agree?

A. Well, if you let me just finish, what I said is that latency period -- there is no minimum or a maximum latency period that is needed for me as a clinician, as a lymphoma researcher, to determine that the exposure was related to disease. That's what I said.

Q. Okay. And are you talking about in an individual patient?

A. No. In any patient there is no such a thing as you have to have a minimum exposure or a maximum exposure. I mean, a latency period -- you're trying to treat latency period as such a binary option that, you know, in order for you have -- you have to have a minimum latency period of 5 years or 10 years or 15 years to -- to have a valid study.

That's not how it works. There is no such a thing as an actual number that has to be fulfilled in order for us to buy into the results or the output of an epidemiologic study from a latency period perspective.

Q. So you do not consider a short latency period to be a valid criticism of an epidemiology study looking at cancer causation. Is what that you're trying to say?

A. If you are trying to equate latency period with a follow-up, you may want to clarify this because I would say follow-up, short follow-up, in any study is always something to be criticized, because you want to follow up patients longer to understand what actually happens.

So maybe you want to clarify for me. If you're thinking latency as a follow-up or you're

thinking -- because latency, to my understanding, is before you even started a study, before you even started the follow-up. Right? I mean, if you design a study today, in 2017, and you want to follow-up patients until 2020, the latency period would be probably since 1990, before 2017.

I think the follow-up, short follow-up, is always a major criticism in any study, frankly, whether it's interventional, observational, epidemiologic, any study. And I do quite -- my share of peer review -- I peer review papers for over ten journals. So short follow-up is always a red flag for us.

But if you want to clarify for me what you mean by "latency," because maybe we're mixing latency with follow-up.

Q. Sir, whether you call it follow-up or whether you call it latency --

A. They're different, sir. They're different. Latency is different than follow-up.

Q. They're different terms in terms of the design of the study; but in either case, they refer to a period of time between the exposure --

A. But that's not true.

Q. -- and the manifestation of the disease;

correct?

A. The follow-up starts from the day you started the study. I just gave you an example. If we design a study today, in 2017, my follow-up starts in 2017.

Q. And if you are looking --

A. And the latency period would be probably 10 years before the patients that were enrolled in 2017 in the study had been exposed to for the past 10 years. That's the latency.

Q. And if you're looking at historical exposures 20 years old, why would it matter if you did any follow-up? If you looked at --

A. Can you repeat the question?

Q. Yes, sir.

If you were looking at patients who were exposed 20 years ago --

A. Uh-hum.

Q. -- and then looking today whether they have cancer, why would it matter whether you added an additional follow-up period to that?

A. Well, because, you know, with more follow-up, additional information will be generated. I mean, it's just -- this is common sense for us who do clinical research. I mean, you need longer

follow-up to make sure that you separate the noise from the truth.

Q. One criticism that you had of the DeRoos 2005 study, the agricultural health study data, was relatively short follow-up; is that right?

A. Do you mind showing me that paper?

Q. Sure. You have your expert report there; right?

A. Sure. It's Exhibit 3.

Q. Yes.

A. I reviewed a lot of papers, so sometimes a refresher will help so I could provide you with the accurate answers.

Q. On page 18 of your expert report, you're talking about some findings of the EPA SAP Panel review; correct?

A. Yes. I see that.

Q. Yes.

And about halfway down, you say, "The EPA clearly criticized the EHA publication, DeRoos, et al. 2005, for its limited follow-up period."

Is that a criticism that you shared?

A. Yes, I do. Like, not just with -- any study with limited follow-up, in my opinion, is always -- could be always criticized.

Q. And the previous sentence says, "In fact, the panel recommended the EPA contact the HS investigators to determine whether updated data on incidents of NHL and other cancers are available."

Do you see that?

A. I see that.

Q. And do you share the view that the HS investigators should be contacted to determine whether updated data is available?

MR. LITZENBURG: Object to form.

THE WITNESS: Sorry?

MR. LITZENBURG: I just make objections from time to time.

If you can answer it, you are welcome to.

A. Okay. I apologize. Would you read the question.

Q. Yes, sir.

Do you share the view that you express here and attribute to the panel that the AHS investigators should be contacted to determine whether updated data on incidence of NHL and other cancers are available, i.e., updated data from the DeRoos 2005?

A. I do.

Q. And do you know whether such data exists?

A. I don't.

Q. Have you been told about a draft paper from Alavanja, et al., from 2013, sir, with updated data?

A. I have not seen that paper.

Q. How did you decide which epidemiology studies to look at, sir?

A. Through my research, through PubMed, Google Scholar, and the literature.

Q. Were you provided with epidemiology studies or other studies by plaintiffs' counsel?

A. I did my own independent research. And when I had some questions, I would contact the plaintiff counsel to -- if I need to.

Q. Were you given any guidance as to what additional information might exist relevant to the question that you were asked to look at, i.e., whether NHL can be caused by glyphosate?

A. No. I was provided with the -- I reviewed the deposition of the epidemiologist. I don't know -- I don't know how his last name is -- Neugut.

Q. You reviewed his deposition?

A. I did review it, yes.

Q. Okay. And did you look at any of the studies discussed therein that you had not previously looked at?

A. I don't honestly recall if I reviewed additional papers based on what he actually stated. I just -- I did not go back and look at more papers based on his deposition. I just reviewed his deposition.

Q. I'd like to go back to your CV for a moment, sir.

A. Sure.

Q. You list a number of publications there. Did any of them involved assessing whether a particular substance causes cancer?

A. A particular?

Q. Particular substance?

A. Causes cancer?

Q. Causes cancer, yes.

A. Not particularly, no.

Q. Did any of your publications involve you reviewing the science and epidemiology on whether a particular substance causes cancer, i.e., for example, a review article?

A. Not a particular substance. We did a lot of research through the CR database and other things to look at disparities and outcomes, and so forth, but we did not -- I did not personally review a particular substance per se.

Q. Other than the work that you have done for plaintiffs' counsel in this case, have you been called upon to conduct a scientific review in the past of whether a particular substance causes cancer?

A. As part of my peer review. Like I said, I review for a lot of journals and some of the manuscripts that get submitted, which I can't disclose because that's how we do peer review. So if I'm asked to review a paper, then I -- I do that.

Q. Other than peer -- I'm talking about your own work, though, sir. As part of your own work, have you conducted such a study or done such a review?

A. No. The only one that I just thought of -- it's been a while back -- was a 2004 paper that -- I'll let you know where it is -- we looked at a compound. It's a radioimmunotherapy for lymphoma, and it showed a secondary leukemia. But I'm going to tell you exactly where that is.

Okay. One second. So these are the abstracts or -- these are the abstracts. Okay. So make sure I show you the . . .

Well, I can't believe we didn't write this paper. This is a paper that I wrote in 2004 in

Leukemia & lymphoma on the association of Zevalin, which is a radioimmunotherapy that is used for lymphoma and secondary leukemia. And I just realized it's not even put in my -- maybe -- there's no way I should have put it in -- just do one last attempt at it, because maybe you -- oh, here it is, I think, on page 12, Reference No. 65. It's actually '02.

So this is a secondary acute myeloid leukemia with MLL gene rearrangement following radioimmunotherapy for non-Hodgkin's lymphoma. This is -- radioimmunotherapy is a form of treatment that we give for non-Hodgkin's lymphoma. It was associated with the secondary malignancy.

So I just recall that this is one of the things that you could consider looking at in association between a particular therapy and cancer.

Q. Was this a case report?

A. Yes. And it was one of the few case reports that looked at particular rearrangements that we discovered after radioimmunotherapy.

Q. Okay. So to sum up, then, the one publication in your CV in which you assess the issue of whether a particular substance caused a particular cancer was a case report; is that right?

A. Correct.

Q. And the one you just identified.

And for the jury's sake, a case report is an anecdotal report by a physician or by anyone of an observation that we expose someone to this particular substance and this outcome occurred?

A. Well, a case report, to be published in Leukemia & lymphoma, has to go to through a strict peer-review process, and you have to -- when you say that a particular compound causes ML gene rearrangement, I had to show that the actual genes were rearranged.

So it does -- while it is a case report, it does go through the same peer-review process and rigorous peer review to be published. You can't just publish any case report. I've had many case reports rejected, so it's okay.

Q. Yes, sir.

The new -- I mean, the new data in a case report is the observation, and it is surrounded by --

A. Sure.

Q. -- the scientific context, which involves research and additional --

A. Correct.

Q. -- writings. That's what you were just discussing; right?

A. Correct.

Q. Okay, sir.

Would you tell me, before we turn away from your CV, sir, where else in your past publications you have used the Bradford Hill criteria?

A. I have not, in my publications, used the Bradford Hill criteria.

Q. Where did you get the idea to use them in your work for plaintiffs' counsel, sir?

A. Repeat the question.

Q. Yes, sir.

Where did you get the idea to use those in your expert report in your work for plaintiffs' counsel?

A. Well, when you do -- I mean, I've been spending a lot of time looking at research and reviewing the literature, so it does pop up as some of the criteria that is -- that could be used to look at causality and look at the evidence.

I also forgot -- I forgot if -- I mean, I read this, I think, and, again, my memory -- I think I read that in the IARC monograph, that it was -- it was looked at, but I'll have to refresh my memory

if -- if they spelled out the Bradford Hill criteria.

Q. Okay. So you got the idea to use the Bradford Hill criteria as a methodology to assess causation from the articles that you found when you were looking at the issue of glyphosate and non-Hodgkin's lymphoma; is that fair?

A. Not as the only methodology. I mean, you'll have -- you'll have to remember, really, that the Bradford Hill criteria or any criteria, for that matter, in medical literature is just simple guidelines tool. You have to take it in context.

If you are going to just take any type of criteria and say "I'm going to follow this criteria," then a robot could do our job. It just doesn't work like this.

You take the criteria, you take the guidelines, and you try to put in context into the clinical evidence that you see and see if it makes sense or not. You could disagree with some of the criteria; you could agree with some of the criteria. But all of the criteria that we have in medicine, in general there's supposed to be some guidelines that you take in context and you still use your clinical judgment. It's not to replace clinical judgment.

Q. You choose -- you chose to organize your thoughts and your clinical judgment as expressed in your expert report in terms of the Bradford Hill criteria?

A. I use it as part of my expert's report, that's correct.

Q. And you got the idea to do that from the various -- from some of the various articles that you found in doing your research on the issue of glyphosate in non-Hodgkin's lymphoma?

A. I thought it was very reasonable to apply and just see if it fits or not.

Q. Okay. You had no opinion on glyphosate and non-Hodgkin's lymphoma before being retained by plaintiffs' counsel; correct?

A. That is correct.

Q. Turn to your expert report, please, sir. I'm on page 4. You said, "The opinions in this report are my own and are held to a reasonable degree of medical and scientific certainty."

Then you said, "These opinions were formed after comprehensive review of medical literature focusing on epidemiologic studies and analyses, as well as my background, education, and experience."

Would you explain, please, why your review

was focused on epidemiologic studies and analyses, why you went to that part of the science.

A. Well, because it's really -- it is -- in order for you to establish causality or to look at causality and between exposure to an occupational hazard or to anything that is -- you can't really have -- there would be never randomized study to say we can have a thousand patients and expose them to Compound A and a thousand patients, no exposure, and then we're going to see what happens. That clearly would be unethical and will never be done.

So you really -- that's really the only way that you can go back and try to investigate the literature when you're looking at something like this.

Q. And could you explain a little more why it is epidemiology that was your primary focus rather than toxicology or --

A. I just did. I just said you can't -- there's no prospective randomized trials that --

Q. In humans?

A. In humans, of course.

Q. Right.

A. I mean, in order for you to say that Compound A is associated with Disease B, you will

need to have a randomized trial where you have a thousand patients that expose to Compound A and a thousand patients that are not exposed to Compound A and you follow it them through and see if one of them develop Disease B or not. And that will never happen in humans. It's unethical. It just didn't work like this.

So in order for me to look at whether an exposure to glyphosate is -- causes non-Hodgkin's lymphoma, epidemiological studies are the ones that I have to use, just by default.

Q. And what do you need to see in epidemiologic studies to conclude that a particular substance causes non-Hodgkin's lymphoma?

A. I think you will need to see that the individuals, the people that were exposed to the compound in question have had increased risk of developing a particular malignancy. You want to see if there is a trend into developing this malignancy in these particular individuals, and then you look at the totality of evidence and try to form an opinion --

Q. When you say --

A. -- to the best of my ability.

Q. Yes, sir.

When you say that -- you would need to see that exposed people have increased risk, what constitutes increased risk in an epidemiology study?

A. Anything above and beyond the folks who were not exposed that is clinically and/or statistically significant.

Q. So you would need to see a statistically significant association in the studies?

A. I'd like to, but sometimes you may not be able to establish statistical significance if there's not enough cases or not enough patients. I mean, there is -- obviously, you know, if you have thousands and thousands of patients, you probably need to see a statistical significance.

Sometimes you can't because of the number of cases that you actually have, and then you look at trend. You look -- does it really make sense seeing the trend, and so forth.

You always want -- you prefer to see statistical significance if you can, but the lack of statistical significance in an epidemiologic study does not, frankly, preclude the possibility of causation between a compound and a disease.

Q. In -- is it your opinion, sir, having reviewed -- you said you made a comprehensive review

of medical literature focusing on epidemiologic studies.

Is it your opinion that the body of epidemiologic evidence is sufficient to detect with statistical significance an association between glyphosate and non-Hodgkin's lymphoma if one exists?

A. The short answer is yes, but I did not see statistical significance. I said that sometimes you may not see statistical significance and you still can establish the causation because it's a matter of number of cases and patients.

In my opinion and based on my review, there is sufficient evidence that glyphosate has a causation to non-Hodgkin's lymphoma. But the statistical significance part in your question may not be always established because, again, you look at powers of the study and the number of patients and you may not always see statistical significance.

Q. You --

A. Any clinician-researcher will tell you that. We've had situations in clinical trials where we -- the statistical significance was established, but that's because they -- they've had thousands and thousands of patients. And then you step back and say, well, is it really clinically meaningful and

vice versa.

Q. Yes, sir.

I want to understand the answer that you just gave. It was a little long.

A. Sorry.

Q. Did you say that, in your view, the body of epidemiologic evidence that exists on the subject of glyphosate and non-Hodgkin's lymphoma is adequate to establish a statistically significant association if one exists?

A. In my opinion, yes, but this does not mean that every study that I reviewed has statistical significance.

Q. Yes, sir.

And some of them do and some don't, in your opinion --

A. Correct.

Q. -- is that right?

A. Correct.

Q. Why is it that statistical significance is used in epidemiology, including in cancer epidemiology?

A. You'd like to see it because you are more certain. You would just solidify your clinical opinion, if possible. But you have to acknowledge

that you may not be always able to see it, in any study that you design, not just epidemiology.

I mean, you do an interventional study and randomized trial and whatever it is. As you design the study, you establish a priori. And you say, okay, I'm going to power this study to establish statistical significance; and, based on that, I need 500 patients randomized, 250 in one arm, 250 in the other arm. You establish that beforehand, and you proceed with the trial, and so forth. This is for interventional studies.

So because -- in order for you to be more certain, you would like to show the statistical significance. When you see it, I think it's very important. When you don't see it, you go back and say, well, why didn't I see it? Is there really a trend? Is there not a trend? Were the numbers too small? Were there some issues in the study?

So the lack of statistical significance, in my opinion, is not always a negative finding. It should be explained. People should look back and say, well, why was it not statistically significant? What was something special in this trial or in this study that was not in the other study?

Q. Please explain what "confounding" is.

A. Confounding --
Q. Yes.
A. -- factors, you mean?
Q. What is --
A. Confounding factors.
Q. What is the concept of confounding in epidemiology and cancer epidemiology?
A. That's -- the concept -- and, again, I'm not an epidemiologist, so I'll answer to the best of my ability.
Q. Yes, sir.
A. The confounding factors means that exposed individuals may be also exposed to additional elements or factors that may -- may impact the causation or the association of the disease in question.
Q. And when it is possible to statistically control for a confounding factor, then the adjusted data is more valuable than the unadjusted data; correct?
A. I think if you all -- if you can control for confounding factors, it's always -- it's always a good thing to do. You will have to control for both arms of each study -- of any study, and I -- what I've seen in many of the papers -- I'm not

talking about the review here but as a peer reviewer for many journals, sometimes the control doesn't happen in a balanced way between both arms.

But you're correct. If you can control for confounding factors, you should at least try. You should at least attempt to do it. You sometimes can't always do it. I mean, there are certain things you just can't control for. Especially you can't, you know -- especially in epidemiology. I mean, these are not patients that are coming and seeing you in the office every week where you're taking what medicine they're taking, et cetera.

But you are accurate. You are correct that you can try.

Q. Sir, can you name for me three substances that are generally accepted to oncologists to cause non-Hodgkin's lymphoma for which there are epidemiology studies and those studies are negative, i.e., do not show a statistically significant association between that substance and non-Hodgkin's lymphoma?

A. To my knowledge, there is no data to suggest that smoking is associated with non-Hodgkin lymphoma. And alcohol is not associated with non-Hodgkin lymphoma, to the best of my knowledge.

These are substances that are commonly used by folks. While there is good evidence that smoking is associated with certain malignancies, bladder cancer and lung cancer. Certain amounts of alcohol, especially with tobacco, is associated with esophageal cancer and other things.

That's the answer I have.

Q. Okay. So that I understand your answer, are you saying that those are generally accepted by oncologists to cause non-Hodgkin's lymphoma?

A. I said do not. I said, in my opinion -- you asked me the question to provide you -- maybe we go back and repeat the question so I answer it correctly.

Q. I must --

A. I thought you were asking me can you -- can I give you an example of things that do not cause non-Hodgkin lymphoma.

Q. Let me ask again. I must have --

A. Please do.

Q. -- done a bad job.

Can you give me an example of three substances that are generally accepted by oncologists to cause non-Hodgkin's lymphoma for which epidemiology exists and that epidemiology is

negative, i.e., does not show a statistical significance?

A. I do not understand the question.

MR. LITZENBURG: You can ask him to rephrase if you don't understand.

Are you talking about a single paper or an entire body of epidemiology?

Q. The question is -- I'm asking for three substances that oncologists generally accept to be a cause of non-Hodgkin's lymphoma in the face of negative epidemiology.

A. I don't think I'm qualified to answer this question. I have to do my research. I did not do research for that topic.

Q. Okay.

A. But more than happy to -- you intrigued me. I'll do some research on that.

Q. You can't tell me any today anyway?

A. I can't.

Q. Do you -- when you were discussing IARC in your expert report, you mentioned that they performed a hazard assessment; is that right, sir?

A. Is there a particular page you want me to look at?

Q. Never mind. It's not a question about your

expert --
A. I have it, page 16, yeah.
Q. Do you understand that the nature of the assessment that they did was a hazard assessment and not a risk assessment?
A. I do.
Q. Okay. And would you tell the jury what the difference is, please.
A. Well, when you do a hazard assessment, you look at the particular compound -- to my knowledge, again -- and please recognize I'm not an epidemiologist. But, to my understanding, that -- when you do a hazard assessment, you look at the particular hazard that you are actually investigating and you look at the, you know, animal studies and then you look at the epidemiologic evidence and try to come up with a conclusion based on the available evidence.
When you do a risk type of an assessment, you actually have more of a prospective evaluation to -- that you can identify the risk easily. That's my understanding.
Q. Okay. Have you -- have you heard it described this way, sir, that a hazard assessment is looking at the possibility for a substance to cause

cancer, whether it is possible for a substance to cause cancer at any exposure at any level; whereas, a risk assessment assesses whether there is a genuine risk to human health from that substance at the levels at which humans will be exposed?
A. I have not heard this definition. I apologize.
Q. Okay, sir.
Do you claim that glyphosate is a substance capable of causing cancer in humans?
A. I looked at the evidence of non-Hodgkin lymphoma. I think when you say "cancer," it's a very general broad term.
So are you asking the question cancer or non-Hodgkin lymphoma?
Q. Let's -- let's ask about non-Hodgkin's lymphoma first.
A. I do.
Q. Do you have the opinion that it causes any other kind of cancer in humans?
A. I did not research other kinds of cancer.
Q. Now, do you also hold the opinion that glyphosate actually causes cancer in humans at the levels at which humans are exposed to it?
A. I do. It's very difficult to -- to know

exactly the levels, and so forth. It's very, very difficult. It's not like a pill that you take 10 milligram here and 15 milligram here and you really know exactly the dose. So it's just -- by its nature, it's just very difficult to establish that.
But the body of evidence suggests that the current exposure, whatever that exposure may be, appears to be causative of the development of non-Hodgkin lymphoma.
Q. And you're talking about the epidemiology evidence?
A. Yes.
Q. Anything else?
A. We talked about the fact you cannot do really prospective randomization. You just can't do that.
Q. Yes, sir.
Of the other studies that you discussed in your expert report, are you relying on any other for the conclusion that glyphosate is capable of causing cancer in humans at the levels to which humans are exposed?
A. I rely heavily on IARC. I think IARC is a world authority in making a decision, whether

certain compounds and materials are associated or causative of developing cancer and malignancy or non-Hodgkin lymphoma.
So I think it's very important to rely on -- on authority in the field. I mean, that's what IARC is.
Q. Okay. Anything else?
A. I think you see that in my expert report into what else I reported -- I relied on. I relied on some meta-analysis that were published in some epidemiologic studies. You have that.
Q. Okay. So the meta-analyses, the epidemiologic studies, and IARC; is that right?
A. That is correct.
Q. Explain to me, sir, how it is that you relied on IARC, how that formed a -- something that you leaned on in your -- forming your opinion about this.
A. I read the Lancet paper that was published. I think it was Guyton, the first author. I think it's '015. I read the IARC Monograph and the information that they provided, and that's how I relied on them.
Q. And did you defer to the expertise of the epidemiologists and the toxicologists and the

mechanism experts and the other experts who were not cancer -- cancer doctors --

A. You mean the authors of the paper?

Q. Yes, the authors of the paper.

-- in their evaluations in forming your opinion?

A. Well, you have to remember that, for a paper to get submitted and accepted in a journal like Lancet, it has gone through the utmost rigor of peer-review process. So, you know, these folks who authored this paper, the output and whatever they actually wrote has been reviewed by experts in the field in order for this to be accepted.

And so I'll have to rely on this because it is not just an opinion piece that you actually write. You write the paper. You submit the evidence. And then it gets peer-reviewed by your own peers that they understand toxicology, they understand epidemiology, they understand all of these things. And then it's -- either get accepted or not accepted.

So, clearly, the body of evidence was robust enough that it was accepted in a major journal like Lancet.

Q. Okay. Sir, I'm talking about you and what

you relied on.

A. I --

Q. Did you -- did you -- do you defer to the epidemiologists and the toxicologists who are involved with IARC for the opinions that they formed about glyphosate in reaching your opinions?

MR. LITZENBURG: Objection. Asked and answered.

THE WITNESS: Do I answer?

MR. LITZENBURG: You can answer if you have anything additional.

A. As I said, I'm not going to re-peer review the actual paper. I take the paper. I take the output, and I form my opinion based on the evidence. But it is not my role to perform a peer-review process and ask for original material, and so forth. I have enough evidence based on that paper.

So do I defer to them and their opinions? I respect their opinions. I may agree; I may not agree with everything that a particular toxicologist or epidemiologist say, but I certainly weighed heavily on the IARC output and the IARC paper because of who IARC is and because where it was published.

This was not published in a throwaway

journal. This was in one of the major journals. The impact factor is top. So clearly I respect the output, and I relied very heavily on the information that were provided.

Q. In the absence of the IARC report, if that had not existed and the Lancet article had not existed, would you have reached the same conclusion?

A. I would have to reach my own conclusion based on the epidemiologic evidence. So it was very nice to see that my own opinion was solidified with a major organization like the IARC. So I think, you know, you have to take it both together.

I can't really answer what I would have concluded had the IARC not available. That's complete speculation for me. I don't know what I would have done. IARC was part of the literature that I reviewed. So if I take the IARC away, then I'll have to go back to a different mindset and re-review everything, and I can't answer that. It's not a fair question to me.

Q. So you might have come to a different conclusion?

A. I didn't say that. I said I can't answer that.

Q. You don't know --

A. I don't know what my conclusion is. I mean, you're just taking away a paper, and say what would your conclusion be if you take away this paper. How do -- how am I supposed to answer that? I don't know.

I know my opinion based on the current papers I reviewed. If you take piece of the papers I reviewed, then I'll have to re-review everything and decide whether I come to the same conclusion, different conclusion, the same conclusion.

But you -- you can't just take away part of the evidence I relied on and say, what would you have concluded, because that's then a completely different case.

Q. Yes. You'd have to go do the work over to know what you would come up with; right?

A. Exactly.

Q. The -- you know that the EPA has concluded on multiple occasions that glyphosate is not a carcinogen; correct?

A. I have seen some of these reports, yes.

Q. And you disagree with the EPA on that; correct?

A. I think the IARC report is more convincing than the EPA conclusion, and it's more substantial.

Q. Why?
A. Because of, you know, again, the EPA report is not something that is submitted to a peer-reviewed journal where it really gets the rigor of other peers looking at things and evaluating things. It's an -- it's almost an opinion piece where folks who -- that, you know, sit on the EPA, they come up with an opinion, and they publish that opinion. Nobody is really looking at and critiquing it.

At the same time, there are certain methodological things that I read into how the EPA came to some of their conclusions that did not really follow the guidelines that they should have followed. There are a lot of critique from my reading into the methodology that they actually used was not as clean as the IARC methodology.
Q. Critiques written by whom?
A. I mean, just go on the web and research EPA. It's like -- I mean, public information.
Q. These are critiques that were written by people like Chris Portier, who is one of the members of the IARC, generating criticisms of EPA and others after the fact in the press; right?
A. I have read some of his, and I -- I mean,

again, I think -- I think, if you have a solid -- in my opinion, if there are certain opinions and there are certain evidence-based facts, then submit them to the rigor of the peer-review process and let's see if they withstand the peer-review process where we can actually get them out in public.

So I'm a strong believer in evidence-based and a strong believer in the peer-review process and -- because it's very rigorous.
Q. You say in your expert report, sir, on page 17 -- this is a section where you are talking about IARC; correct?
A. I see that, yes. Excuse me.
Q. I'm on page 17, about two-thirds of the way down the page.
A. Okay.
Q. You said, IARC -- "IARC report was conceived in 2015 after an in-person meeting took place between 17 experts in the field from 11 countries." Correct?
A. Uh-hum.
Q. And you say this meeting took place after a three-months period of reviewing the literature and analyzing all available data and evidence; correct?
A. I do, yeah.

Q. Where did you get the information that there was a three-month period of reviewing the literature and that all available data and evidence was reviewed?
A. I forgot. I don't remember where I got this. It's probably from the paper by Guyton, et. al, in Lancet or by some of the editorials. I do know these are facts. I looked it up at the time.
Q. Do you know that Dr. Aaron Blair, who was the head of the group, was deposed -- had his deposition taken?
A. I do know he was deposed. I never read his deposition.
Q. Do you know that he testified that the IARC working group spent only one or two days total assessing whether glyphosate could cause cancer?
A. I did not know that.
Q. Do you know that he testified that they didn't really start work on any of the analysis until they arrived in Lyon, France?
MR. LITZENBURG: Object to the characterization.
A. I did not know that. Did not know.
Q. Okay. Does that alter your opinion about

the rigor of the IARC review?
A. No, it does not.
Q. Why not?
A. Because you could do a lot of the research before you come to Lyon, France. I mean, I have been there on committees where you -- you know, the actual two-day meeting is to discuss what you have been researching for the past few months as a committee. And then you come in and you debate what you actually did.

So the -- whether you spent two hours or two days during the actual meeting does not mean that you did not spend months or weeks before researching the subject. This is the -- you're not going to meet for three months over certain things, or four months. You do the research beforehand, and you say, okay, in March or April we're going to meet; and whatever you've researched, we're going to discuss and come up where a report. It happens all the time.
Q. Sir --
A. The WHO for lymphoma, for example, the publication that we just went over, the types of lymphoma. This was a one-day meeting, and it was published. It doesn't mean that the research was

just one day. It was a year in the making and debates, and so forth. And then you get together in a particular time and you come up and generate an output. So it doesn't alter my opinion.

Q. Do you know if there is testimony in this litigation, sir, that the IARC working group was provided with data from Greim, et al., the Greim paper involving 14 animal cancer bioassays on glyphosate that in the published literature and did not review it?

MR. LITZENBURG: Objection. He said he hadn't read the Blair testimony.

A. I did not know that. I did not see the testimony.

Q. You mentioned the Greim paper in your expert report; correct, sir? You mention it on page 16.

A. One second. Yep.

Q. And this is at the bottom of a paragraph that's discussing a meta-analysis by Chang and Delzell?

A. Uh-hum.

Q. And at the end of the discussion of the Chang and Delzell meta-analysis, you say, "Notably no increased risk for Hodgkin's lymphoma was found

in this study." And then you said, "To the contrary, Greim, et al., suggested lack of association, "Critical Reviews of Toxicology," 2015."

And you understood, sir, that the Greim paper was a review of animal cancer bioassays, right, not a meta-analysis?

A. I really have to relook at the paper. It's -- I mean, I'm more than happy to relook at it. I do remember looking at it at the time, but it's -- I want to make sure I provide you with the accurate answer.

Q. Yes, sir.

(Nabhan Exhibit 5 marked for identification.)

Q. So Exhibit 5 is a review article from the Critical Reviews in Toxicology by Greim, et al., entitled "Evaluation of carcinogenic potential of the herbicide glyphosate drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies."

Correct?

A. I see that, yes.

Q. And did you read this?

A. A while back. But I'm trying to refresh my

memory. If you are going to ask me questions in particular to this study, I'm -- I would like some time just to review it and make sure I provide you the accurate answers. If you're not going to ask me about it, then I don't have to waste time.

Q. Well, let's see.

A. Sure.

Q. First of all, I want to know if you reviewed it.

A. Yes, a while back I did.

Q. And did you consider the contents of this review article informing your conclusion about glyphosate?

A. Well, of course. I mean, I wouldn't really mention it -- I mean, you know, I wouldn't mention it in my report if it's not something that I did not consider it.

I did, obviously, pause, given the fact that one of the coauthors is employed by the company that makes the drug, the compound. So to me, as a researcher, I'll always have to pause about this and see how -- how fair and balanced and no bias was in a paper like this.

Q. What you wrote in your expert report is "To the contrary, Greim, et al., suggested lack of

association; however, one of the coauthors of this work was employed by Monsanto and provided ghostwriting, making my question the credibility of this work."

Correct?

A. Correct.

Q. That's the only thing you say about Greim in your whole expert report?

A. Correct.

Q. Right?

And nowhere in your expert report do you assess the actual content of this article; right?

MR. LITZENBURG: Object to form.

A. Well, I read the paper. And, again, I bring it up here because the conclusion or the output of that paper suggests lack of association. And I mentioned why the credibility of the paper was of low value to me as a clinician, as a researcher, because I'll have to wonder whether it was really fair and balanced.

It's a fair thing for me to question the evidence based on the authors. We do that all the time.

Q. How did you form the opinion that one of the authors was involved in ghostwriting?

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A. Well, two things. The -- if you look at David Saltmiras --
Q. Yes, sir.
A. -- affiliation is Monsanto and glyphosate task force. And I think I'm trying to remember where I read that it is possible that he had a lot of contribute -- I mean, he's a coauthor; so he, you know, again, as a coauthor of the -- whether you call this ghostwriting or not ghostwriting, I mean, but he's a coauthor that's employed by the company that makes glyphosate.
So I guess, you know, I mean, you'll have to wonder whether the opinions in the paper were fair and balanced and free of bias.
Q. Did you discount the opinions expressed in the paper on the grounds that one of the authors was employed by Monsanto?
MR. LITZENBURG: Objection. Asked and answered.
A. It made me question the conclusion. I think if you were me, you would probably have the same question.
Again, you know, how likely is an employee of the company that makes a compound is going to go on the record in a peer review and say "The compound

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of the company that employs me causes cancer"? I mean, it's probably almost going to be zero, the chances are, or be fired.
So I think for me, you know, great. It's good paper, I guess. But, I mean, I'll have to put my clinician-researcher critical hat and say, I'll take this with a grain of salt, whatever the output is. It's very difficult for me now to assess objectively the literature because I know what the conclusion will be. I mean, I know the conclusion will be that there is no association; otherwise, an employee of the company will not be a coauthor.
Q. Sir, this is not original research; right? It's a -- it's a --
A. Whatever research.
Q. -- summary --
A. Whatever it is.
Q. It's a summary of 14 animal studies; correct?
A. Yeah. But you asked me whether I discounted the output of the research based on the coauthor, and I said it made me look at with a high degree of skepticism. Because it's only fair, if I already looked at the authors before I even read anything, I knew what the conclusion will be. So

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then -- then if I already knew the conclusion, nothing was shocking there. You know, it's already clear where the paper will be heading.
Q. Sir?
A. Yes.
Q. I asked a simpler question than that.
This is not original research; it is a summary of 14 animal studies. Correct?
A. Yes.
Q. The data tables from those 14 animal that are summarized herein were available and remain available online for review; correct?
A. Which table are you looking at?
Q. All of the data tables from which the information in this -- come.
A. There's Table 1 and there's Table 2.
Q. I'm talking about an online annex.
A. Where is the online annex? I'm not sure.
Q. Online, sir.
A. Okay. Well, I'll have -- are you going to show me that?
Q. Do you see at the back of the paper, "Supplemental material available online, data supplementary study 1-14"? The data is all available online; right?

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A. Right. If you want me to comment on this data, I need to see it.
Q. Did you look?
A. On the -- on the supplementary data?
Q. Yes, sir.
A. I don't remember if I did.
Q. Do you know if any statement in the Greim article is false or misrepresents in any way the original data that is available online for you to review?
A. My opinion was formed based on the fact that one of the coauthors is employed by the company. So I question the evidence.
Q. You question the accuracy of the data that is published online?
A. I do.
Q. You question whether it's fraudulently misrepresented?
A. I didn't say that.
MR. LITZENBURG: Object to form.
A. I said -- I didn't say it's fraudulent. I'm not making any accusations. I said I have an author on a paper that's employed by a company that is making the compound in question. I think any fair clinician and researcher -- you can ask a

hundred of them -- will put the skepticism hat and say, Well, you know, I don't know. I need to -- you know, I'll have to take a look at this more carefully, and so forth.

Q. Did you?

A. I don't remember if I looked at the supplementary data.

Q. Okay.

A. Like I said, I read this paper, and the output of this paper became questionable to me because I knew what the conclusion will be even before I read the paper based on who the authors were.

Q. You just told us -- sir, you just told the jury that you would doubt the accuracy of the data, the original data --

A. The conclusion.

Q. -- from the studies.

A. The conclusion of the paper.

Q. Okay. Do you doubt the data?

A. I will need to relook at the data more critically and assure that there is transparency and everything is actually being provided and given. And I'm not the animal toxicologist to actually give that, so this data should be, you know, given to

whoever reviewed or whoever is involved.

I'm not an animal toxicologist to provide an opinion. But if that data is available, then should be critically assessed and evaluated by others who are experts in the field. I'm just giving you my opinion as a reviewer of something that I saw in the literature that is written, technically, by the company that makes the compound. I mean, wouldn't it be fair for me to question that?

Q. You gave it no weight because of the authorship. Is that fair to say?

MR. LITZENBURG: Object to form.

A. I actually said -- I said in my report that makes me question the credibility of this work.

Q. How much weight did you give it?

A. I --

MR. LITZENBURG: Object to form.

A. I looked at the entire -- at everything. I didn't weigh every study. It's not what a clinician does. It's not like I take one study and I give it zero weight and another study ten. It's not a point system. You review the literature, and then you come up with a conclusion based on everything. It's not a weight system for each study.

You know, then you have meta-analysis.

Then you go and rely on a couple of meta-analyses. And the meta-analyses, they go in and they try to take a look at all of the studies that were going on. So they've done some of the work for me. And there were two meta-analyses that showed -- showed -- again, and I referenced in my expert report, showed an odds ratio and risk ratio that is in terms of causation and association.

So I didn't do a point system for every single study. It's just not how I reviewed things.

Q. Yes, sir.

When you say that the meta-analysis did some of your work for you, what do you mean by that?

A. I said a lot of times, when you have so many studies going on, a meta-analysis is a way of trying to lump the evidence into, you know, comprehensively assess all of these studies and try to come up with a conclusion that is either a yea or a nay in terms of an association or a causation.

And the two meta-analyses that I saw were referenced on -- on page 15. One is by Schinasi and León and the other one by another authors. The other one is by Chang and Delzell.

Q. And in what way did that do some of your work for you?

A. What I mean by doing some work for me is the meta-analysis in general, there's a methodology for meta-analysis. To conduct a meta-analysis in a systemic review, there's an actual methodology where they look at all of the studies collectively. So whatever the authors are, they looked at the complete body of evidence and literature, and they came up with these conclusions.

So I didn't do my own meta-analysis. That's what I'm trying to say.

Q. Had the meta-analyses yielded a non-statistically significant result, how would that affect your opinion?

MR. LITZENBURG: Object to form.

A. Yeah, I mean, I think -- I think, again, it is -- I'll say this, and I think I said that a couple times before: It is really important to not rely on one study or another. It is impossible in epidemiology and occupational exposure literature. You'll have to rely on all of the evidence.

And, again, I think you've asked me the question if the IARC was not there, what was your conclusion -- what could have been your conclusion? Which is a complete speculation. The same answer is here.

I mean, I don't know, if the meta-analyses were negative, what type of report I would come up with or what type of conclusion I would come up with. It's just a completely different review. I don't know the answer to that.

Q. Well, you've said that -- we've established earlier that you primarily were focused on the epidemiology in conducting your analysis here, and you just said that a meta-analysis is a review of all the available epidemiology --

A. Is an attempt -- is an attempt to lump a lot of the studies that were peer-reviewed and published in the literature to come up with a conclusion.

Q. Yes, sir.

A. And I have done some meta-analysis while looked at a particular compound, you know. I mean, you just -- sometimes you just want to try to come in the totality of evidence.

Q. And when you look at the totality of the evidence through the tools of meta-analysis and those results turn out to be not statistically significant, what does that mean to your -- to you as someone who is trying to do a causality analysis?

MR. LITZENBURG: Object to form.

A. Yeah. I will have to understand the methodology of the meta-analysis, how was it done? Did they have individual data? Did they have patient-level data? What have they done to come up to this conclusion? I think it's very important to -- to look at what was done.

Q. You can't say what effect it would have on your opinion if the meta-analyses --

A. Of course not.

Q. -- not statistically significant?

A. I just don't know.

Q. Yes, sir.

On the subject of the Greim study being ghostwritten, as you say, it is transparent on the face of this published study that one of the coauthors is a Monsanto employee; correct?

A. Correct.

Q. You didn't need to hear from anybody else to know that; it says it right at the top?

A. No.

Q. And it says it right at the bottom, "To address for correspondence David Saltmiras, Monsanto Company." Right?

A. Good. Yeah.

Q. So that was very --

A. Which is a little bit unusual.

Q. -- clearly --

A. Which is a little bit unusual, because most often, if you look at most papers, the corresponding author is either the first or the last author. Always. And I've been last author and first author on over 200 papers. It's very unusual for the corresponding author to be the second author and the employee.

Q. Is that a sinister thing?

A. It is -- I would say this happens in less than 1 percent. So why?

Q. Is it a sinister thing?

A. It's unusual. Why?

Q. Is it sinister?

A. What do you mean by "sinister"? Define "sinister" to me. It's something that is not common --

Q. Why are you flagging this as an important thing?

A. Well, why -- why is it a deviation from what we've always written pages? If we've always had the corresponding author as the first and the last author, why all the sudden I have a second author who's an employee as the corresponding

author?

You have -- I think the burden of proof is on the authors to explain to me why. So I don't know why. I mean, I've never been a second author as a corresponding author.

The first and last author in medical literature are always the corresponding authors. In fact, there are fights. People fight who's going to the first and last author so they can get the corresponding author. Because it's an honorary thing to be a corresponding author.

So to me, again, you just have to -- you'll have to explain to me why is a second author, who is not the original researcher, who is an employee, is the corresponding author?

Q. What I want you to explain to me, sir, is why you say that Dr. Saltmiras ghostwrote a paper that he is a listed author on where his institutional affiliation is clearly disclosed?

A. Yeah. I think I may have, you know -- the term "ghostwriting" here was -- is not what I meant by ghostwriting that he is not there. I meant that he provided writing. You're right. Ghostwriting means that you don't even put your name, if that's -- you know, I didn't imply that ghostwriting

means that he was not a coauthor. I meant that he probably was the most responsible author of the entire manuscript. I mean, he is the employee; he's the correspondence author; he provided all the data.

Q. So ghostwriting --

A. A very good question.

Q. I'm sorry. Were you done?

A. What I meant by "ghostwriting" is that cowrote or was an author on that paper.

Q. "Ghostwriting" really isn't the right word for the situation presented by the Greim article; is that right?

A. You're correct.

MR. LITZENBURG: If we are done with Greim, we've been going about an hour and a half. Can we take a break?

MR. GRIFFIS: Sure.

VIDEOGRAPHER: Ending Disc No. 1 of the deposition of Dr. Chadi Nabhan. Off the record at 10:30 A.M.

(Recess taken from 10:30 A.M. to 10:44 A.M.)

VIDEOGRAPHER: And beginning Disc No. 2 of the deposition of Dr. Chadi Nabhan. We are back on the record at 10:44 A.M.

BY MR. GRIFFIS:

Q. Sir, do you understand what it is that is contained in the Greim article, what these animal studies are?

A. I did not look into each particular study by itself.

Q. And do you know that this is the main body of regulatory evidence that was reviewed by the EPA and by European and other regulators in approving glyphosate as safe and effective for sale in the United States?

MR. LITZENBURG: Object to form.

A. I did not know that this is the sole evidence or the most important evidence that was reviewed by the EPA.

Q. And, I'm sorry. With regard to the issue of carcinogenicity. Did you know that?

A. I did not know that.

Q. And do you know that in addition to these 14 rodent studies, studies done in mice and rats that formed the basis for the conclusions by EPA and by foreign regulators that glyphosate was not a threat for human cancer, there were additional animal studies done by other registrants, other people choosing to sell generic forms of glyphosate

that were also considered by EPA and by foreign regulators?

A. I did not know that.

MR. LITZENBURG: Object to form.

Q. And do you know that EPA and foreign regulators consider long-term cancer bioassays, like the 14 described herein, critical in assessing whether a substance that's been submitted for registration review is carcinogenic?

MR. LITZENBURG: Objection.

A. I have not been a part of the EPA review panel or decision maker for the EPA, so I don't know what their process is.

Q. And you know that IARC did not review this data in any form; correct?

A. I don't know if the IARC reviewed this particular data. What I know is that the IARC concluded that there's sufficient evidence based on animal studies that there is carcinogenicity.

Q. You know that IARC has a policy of not reviewing anything unpublished; correct?

A. I think it's fair to review only published data.

Q. And you know that none of this data was published except in the form of this article;

correct?

A. Again, I'll go back and say that my understanding from my review that the IARC saw sufficient evidence on animal studies that they reviewed that there's carcinogenicity. Whether they reviewed this particular paper or not, I don't know, but I know that their review collectively demonstrated that the animal studies that they looked at had sufficient evidence to establish carcinogenicity.

Q. And you know that it's IARC's policy not to review unpublished studies regardless of their quality; correct?

A. I think if there are studies of good quality, they should be published. So if they're not published, then why should they be reviewed?

Q. Do you know that registration studies -- and that's the term for studies that are performed by companies in order to secure registration -- are considered their intellectual property and that, if they were published in their entirety, then another registrant could just submit them to the EPA and get a generic form of glyphosate registered thereby?

MR. LITZENBURG: Object to form.

A. In my life and in -- as a

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clinician-researcher, pretty much almost all registration studies for cancer therapies have to be published in peer-reviewed journals. So I'm not sure if there's a different thing for compounds like this, but pretty much every drug that has been approved for the treatment of cancer through registration trial has been published in a peer-reviewed journal.

Q. Okay. And that's not the case for --

A. These are registration --

Q. -- for herbicides. Did you know that?

A. I did not -- like I said, I did not -- I don't know the actual process of the -- of herbicides with the EPA, and so forth.

But, in my opinion, if there is literature that is sufficient and compelling, then it should be subject to a peer-review process and the rigor of peer review and get published. There is no reason not to get published.

Q. Having gone through the rigor of peer review and publication and having been published, this should have been reviewed by IARC; right?

A. I think -- yeah, I mean, I think this is peer-reviewed paper. So it may have been, may have not been reviewed by IARC. I don't know.

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But what I'm saying is that the collective evidence from IARC or the output from IARC suggested that the animal studies that they looked at established carcinogenicity collectively. I don't know if this particular paper that you're referencing was reviewed by IARC.

Q. And this particular paper constituting a report on data from the 14 key registration studies considered to be pivotal and critical by the EPA is certainly the sort of thing that IARC should consider?

A. I can't speak for the --

Q. You would agree?

A. I cannot speak for the IARC. I mean, I think -- I don't represent the IARC.

Q. No, sir. You're someone who -- you're someone who has said you are relying --

A. I do rely on them.

Q. -- on the conclusions of IARC and rejecting the conclusions of the EPA. So what I'm exploring right now is the difference in what they considered --

A. Right.

Q. -- in reaching those conclusions.

A. I'm not in a position to evaluate their process or critique their process. I'm in the position to either believe or disbelieve the output. The actual process that they go by, that is something you have to take on with IARC.

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Q. And there is testimony in this litigation, sir -- are you aware of this -- that testimony, uncontradicted testimony -- that this study, the Greim study, was available to them in their hands and they did not consider it?

A. I have not --

MR. LITZENBURG: Objection.

A. -- seen this testimony but more than happy to look at it.

Q. Would that cause you any concern?

A. I'll need to see the testimony.

Q. Would it cause you any concern if this was in their hands and they chose not to review it?

A. I will need to under -- A, I need to look at the testimony; B, I need to know why they didn't look at it. They may have had a very good reason or a valid reason, and I don't know that.

But that is something to ask the IARC. I mean, if they -- if they had a paper and they chose not to review it, then the IARC must have a reason. And I don't know what that reason may be.

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I'm not aware of the testimony, but it's the IARC's decision to look at the literature. I can't really speak for them, but I think it's a valid question to ask them why was it -- why -- why was this not considered. And let's see what they say.

Q. You're relying on the animal study conclusions of IARC, a group that did not look at the key studies that all regulatory agencies consider in assessing the carcinogenicity of a substance, and you are rejecting the opinions of EPA and the British authorities and the Canadian authorities and the German authorities and the European Union authorities who did look at this very same data --

A. Well, the IARC --

Q. -- is that correct?

A. The IARC, in my opinion, is the most authoritative agency to look at causation between compounds and cancer. That is my opinion. And what they review and why they reviewed some things or not reviewed some thing, that is something that the IARC has to decide based on their processes and procedures and their SOPs. I don't know what studies they decide to look at versus not.

Q. Tell --
A. What I know is I look at the output of it and then review my own data and try to come up with a conclusion.
Q. Why do you consider IARC to be more authoritative than the EPA on the subject of the safety of an herbicide?
A. I think that's well known. The IARC is a subset of the -- I think the acronym is the International Agency for Research and -- on Cancer.
And I think, you know, pretty much this is -- in my mind, this is the authority that looks at these things. It's not me considering this. I think there are a lot of folks in the field that consider the IARC as the most authoritative agency.
Q. Do you know how --
A. This is not my own opinion.
Q. Do you know how substances IARC has looked at in the past as to whether they are carcinogenic --
A. I don't know that.
Q. -- and concluded that it is not?
A. Don't know.
Q. You don't know how many they've found not to be carcinogenic?

A. Why should I know? I don't know. It's not -- I mean, this is not something within scope of what I was asked to look at.
Q. You don't know that as an ep- -- as an oncologist?
A. I do not.
Q. Now, the IARC broke up into subgroups for its review. You understand that?
A. I do.
Q. And one of the subgroups looked at the epidemiologic evidence; correct?
A. Yes.
Q. And it found that evidence to be limited; right?
A. Do you have the Guyton paper with you? I mean, again, I want to --
Q. I have the Monograph.
A. Well, the major one, I think, that -- I know that they broke into groups and each group looked at the particular evidence and so forth. So I -- I want to make sure I answer accurately.
Or the Monograph, whatever it is.
Thank you.
(Nabhan Exhibit 6 marked for identification.)

Q. Exhibit 6, I've marked as the IARC Monograph, sir.
A. Okay. Go ahead.
Q. Okay. So the working group that focused on the epidemiologic evidence concluded that there was, quote, limited evidence --
A. Yes.
Q. -- that -- in humans -- limited evidence in humans, right, referring to the epidemiology evidence?
A. Do you mind showing me which -- which part of the --
Q. Yes, sir. Page 78.
A. Okay.
Q. The Evaluation section, 6.1, Cancer in Humans.
A. Okay. I see that.
Q. It says, "There is limited evidence in humans for the carcinogenicity of glyphosate." And then the specific cancer that they're talking about, they say, "A positive association has been observed for non-Hodgkin's lymphoma." Right?
A. I see that, yeah.
Q. Okay. Now, do you know the meaning of "limited evidence --"

A. That you cannot be --
Q. -- to IARC?
A. -- you cannot be 100 percent certain. The only way to be 100 percent certain, as we talked about that earlier, it's a randomized controlled study. That is literally the only absolute way to be 100 percent sure. It is unethical or impossible to do.
Q. And you know that IARC, when they say "limited evidence," they mean something much different than just not ruled out beyond any chance, like you were saying?
A. Well, "limited evidence" means that the evidence is not -- is not certain, is not 100 percent.
Q. They mean something much less than that, sir. They mean -- I'll quote, "A positive association has been observed between exposure and the outcome for which a causal interpretation is credible but chance, bias, or confounding could not be ruled out with reasonable confidence."
Do you understand that that's what IARC means when they say "limited evidence"?
A. Now I do.
Q. Do you agree with that?

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A. Yes.
Q. So you agree that the epidemiology evidence with regard to glyphosate and NHL is credible but chance, bias, or confounding cannot be ruled out without reasonable confidence; is that right?
A. If this is what the IARC said, then I do agree with that.
Q. And with regard to any cancer other than non-Hodgkin's lymphoma, they didn't even find limited evidence; right? They found no evidence?
MR. LITZENBURG: Objection. Beyond the scope.
A. Again, I -- I -- I did not really evaluate what evidence they looked at outside. I mean, I looked at the non-Hodgkin lymphoma.
Q. Okay. You're not giving the opinion that glyphosate is associated with any cancer other than non-Hodgkin's lymphoma; right?
A. I'm just talking about non-Hodgkin lymphoma, correct.
Q. And when you say that glyphosate is associated with non-Hodgkin's lymphoma, do you say that it is also associated with every single subtype of non-Hodgkin's lymphoma?
A. Yeah, I think it's -- it's -- it's very

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difficult to establish that because of how many types of lymphomas there are and also because the understanding of the current classification of lymphoma was not the same classification that we had in the mid or late '90s, et cetera. So what we knew back in the '90s about the types of lymphoma is not what we know today.
So I think you'll have to look at non-Hodgkin lymphoma as one entity when you look at this causation and association.
Q. And you -- you're saying that we're forced to look at non-Hodgkin's lymphoma as one entity because we don't have much data on how glyphosate might be associated or not associated with various sub types?
A. No, for various reasons, I think. A, the classification of lymphomas was different back then versus now. I mean, even -- just to give you an idea, the -- the 2016 classification, the earlier one was '014, then was '07, and there was 1999. So, again, it changes.
Number 2, once you actually start looking at every single subtypes, the numbers become too small to actually be able to detect statistical significance. So when we look at causation in

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association with occupational exposures, you'll have to look at the actual entity as a whole in order for you to establish this such association.
It's just by default. It's very difficult in lymphoma because you can't have a study for 60 types.
Q. Yes, sir.
You are saying that, because of the inadequacy of the scientific data and because of our inability to distinguish between subtypes, we can only form a conclusion about non-Hodgkin's lymphoma as a whole; is that fair?
MR. LITZENBURG: Objection. Mischaracterization.
A. Well, I didn't say inadequacy of the scientific data. What I said is that lymphoma classification has changed over the past 20 years.
So today I have 60 types. 20 years ago, I had probably 10 types or 20 types. So it's very difficult to look at each subtype because these types and subtypes have been refined and changed in classification. That's one reason.
Q. Yes, sir.
A. The number-two reason is, because of the number of subtypes for lymphomas, if you want to

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look at each one by itself, it becomes very difficult in terms of statistical or clinical significance.
Q. Yes, sir.
A. And the third reason is, because many of these studies that are case-controlled that you're asking the cases to recall what type of lymphomas they had, there are many patients or many folks, they don't really understand the granularity of the type of lymphomas.
So you ask -- you know, a patient of mine, they say, "I have lymphoma." They may not even know it's non-Hodgkin versus Hodgkin, if it's follicular or it's large cell. So I think it's just -- it's not something you can actually logistically do accurately.
Q. We don't have the information --
A. Or the ability.
Q. -- to distinguish between what association glyphosate may have or may not have with each subtype?
A. Correct.
Q. And so the only conclusion that we can reach is about non-Hodgkin's lymphoma as a whole and not about specific subtypes; is that fair?

A. Yes. But that doesn't take away that it -- it is associated with all other subtypes. What I'm trying to say is, just because I don't have information for each subtype, it doesn't mean that it cannot be associated with it. When you are --

Q. Yes, sir.

A. When you have -- when you have an association or a causation between a compound and a disease, you could be causing all of the subtypes of that disease as well. You don't really need to study each subtype.

We know the association of tobacco and lung cancer. We don't need to establish this for the six subtypes of lung cancer.

Q. It may be the case, sir, that glyphosate is causally associated with every subtype of non-Hodgkin's lymphoma, and it may be the case that it's only associated with some of the subtypes and we can't tell the difference?

MR. LITZENBURG: Objection.

A. We don't have the data today to show either/or.

Q. Yes, sir.

And is there any particular subtype that, in your opinion, we have the data to say

non-Hodgkin's lymphoma -- that specific subtype of non-Hodgkin's lymphoma is caused by glyphosate, or can you not say that for any subtype?

A. Yeah, I don't have an opinion today. What I have an opinion is that there is an association and a causation between glyphosate and non-Hodgkin lymphoma, meaning that it could actually impact all subtypes.

Q. Yes, sir.

You told us earlier that you read the deposition of plaintiffs' expert witness, epidemiologist Dr. Alfred Neugut; correct?

A. I did.

Q. And do you agree with him, sir, that the epidemiology alone is not sufficient to show a causal relationship between glyphosate and non-Hodgkin's lymphoma?

A. I -- it was a 400-page. I don't remember this is exactly what he said. Do you -- I mean --

Q. Do you agree with that statement?

A. Say the statement again.

Q. Yes, sir.

"The epidemiology alone is not sufficient to show a causal relationship between glyphosate and non-Hodgkin's lymphoma."

MR. LITZENBURG: Object to mischaracterization in the testimony.

A. If he meant by "not sufficient" as an absolute in terms of 100 percent, then I agree. You cannot -- you will never be able to say 100 percent because of the nature of what we are talking about. But you take the epidemiologic evidence in the context of the clinical scenarios and additional information and you try to form an opinion.

So I agree with the fact that you cannot take just one piece of data or one piece of information and rely solely on it. You have to rely on everything to form an educated and a comprehensive opinion.

Q. When you say that there is a causal relationship between glyphosate and non-Hodgkin's lymphoma, you don't mean 100 percent; right?

A. There is not 100 percent in life.

Q. Okay. Well, I'm going to ask you this question again, whether you agree or disagree with this statement. And when -- when you answer it, please answer by your own standards of establishing a causal relationship, not 100 percent, but your own standards of what is sufficient to establish a causal relationship.

Do you agree or disagree that the epidemiology alone is not sufficient to show a causal relationship between glyphosate and non-Hodgkin's lymphoma?

A. I disagree.

Q. You believe that the epidemiology alone is sufficient to show a causal relationship?

A. It can be if it's strong enough, absolutely.

Q. I don't mean epidemiology as an abstract. I mean the epidemiology that you looked at on this subject.

A. Okay. And what do you include in epidemiology? Are you including IARC? Is that part of epidemiology?

I just went to make sure -- I mean, is IARC considered epidemiology literature?

Q. I don't know. Do you consider it to be an epidemiology study?

A. I do. But if you don't, then I -- I want to make sure I answer the right question.

Q. Okay.

A. Is it the original epidemiology data? Or, I mean, the IARC, for example, took -- I mean, to me it's obviously part of epidemiology. It looked

concisely at all of the literature, and they generated a peer-reviewed manuscript that is easily researchable and publishable and I read it. And meta-analysis is part of epidemiology literature, but if you're just talking about the actual paper, it may be different.

So in my opinion, there are -- there are situations where the epidemiology literature is sufficient to establish causation. And based on my review of the epidemiology literature for glyphosate, I see sufficient evidence to demonstrate causation.

Q. Okay. And when you said that sentence, what did you mean by "epidemiology evidence"?

A. I meant the original papers of epidemiology that I reviewed, plus the IARC, plus the meta-analyses, plus, you know, some of the review articles that I looked at.

Q. And what review articles are you talking about?

A. They -- they -- I mean, they -- again, they were included in the -- I mean, maybe I -- meta-analysis, I consider them sometimes part of review because they're not original data. So the meta-analysis, they're really reviewing the actual

collective type of research. So I -- that's what I meant by review articles per se.

Q. Okay. And IARC is also a review article in that sentence because it didn't generate new data?

A. Yeah. No, IARC --

Q. It reviewed --

A. -- did not have original data. They looked at the available data, and they came up with a robust conclusion.

Q. So when you say that the epidemiology -- in your opinion, the epidemiology alone is sufficient to show a causal relationship between glyphosate and non-Hodgkin's lymphoma, you're including the original epidemiology studies and IARC in that; right?

A. I am, and the meta-analysis.

Q. And if you take IARC out, do you still feel it's sufficient?

MR. LITZENBURG: Objection. It's been asked and answered this morning.

A. You really can't keep asking me about taking evidence out. I mean, I'm sorry, but I cannot comment on -- on taking stuff out and what I conclude. This is the third time.

I can't -- I don't know what I would

conclude if I take some of the evidence that I reviewed out.

Q. Okay. Sir --

A. It's not fair.

Q. The thing about IARC is that it is not original data.

A. It's paper in Lancet.

Q. It is not original data.

A. But it's in Lancet.

Q. It's a review article; right?

A. It's not a review article. It's in Lancet. Have you tried publishing in Lancet? They reject the 95 percent of the papers. So I think this is not fair. Lancet will not accept papers unless they go through peer-review process and robust evidence.

So whether it's a review article, meta-analysis, collection of research, it's gone through the peer-review process. And there was sufficient information in there to generate a publication in the most prestigious, most competitive journal that we have.

Q. To what extent did you substitute the judgment of the authors of the Lancet article for your own, sir, in reviewing the original data?

MR. LITZENBURG: Object to form.

A. I don't re-perform a peer review. This is not my job. All published papers have gone through peer-review process before they get published. They sometimes have three peers, four peers, four -- five peers, whatever the journal policy is.

And, as you know, the process, the peers provide comments and they might go back and forth. And sometimes papers take six months until they get published.

So I did not re-conduct a formal peer-review process for this paper nor for all other papers, frankly. I -- I look at the paper, look at the evidence, and look at the totality of information that's available.

But this does not mean that I substituted my judgment. There's a difference between you being a peer reviewer for a particular paper or just basically reading the paper, taking the conclusion, and putting it in the context of other research that's available.

Q. Do you agree with Dr. Neugut that there is no epidemiology study that reports a statistically significant association between glyphosate and non-Hodgkin's lymphoma adjusted for other pesticide exposures?

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MR. LITZENBURG: Object to the mischaracterization.
A. I don't remember -- do I -- I don't remember that particular statement.
Q. Do you agree with the statement, whether he said it or not, then?
A. That there is no positive association between --
Q. There is no epidemiology study that reports a statistically significant association between glyphosate and non-Hodgkin's lymphoma once you control for other pesticide exposures.
MR. LITZENBURG: Same objection.
A. I -- I don't remember -- I know that not all studies were able to control for other exposures. That's for sure. It just was very difficult.
I don't recall -- you know, I have to -- I wrote the few studies here to remember. I don't recall if no study has controlled for everything. I know some studies try to control and some studies did not.
So I'll agree, but I will -- I have some reservation because I want to make sure I review all of these studies as well. If he said that, then,

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for the most part, it's going to be correct. But I know for a fact that some studies did actually attempt to control. I just don't know if these were statistically significant or not. I'll have to review that.
Q. Okay. We'll go over epidemiology later.
A. No problem.
Q. You don't know -- without going through each study, you don't know if there is a single statistically significant association in the epidemiology that can -- once you control for other pesticide exposures?
A. Yeah, I don't know that. I know that there were attempts to control.
Q. Why would it be important to control for other pesticides?
A. As we said earlier, I mean, I think you always want to try to control for other pesticide exposures to eliminate contamination if you can. I mean, obviously -- I mean, here's how you'd look at things. So if you have contamination where there's exposure to other pesticides, then it might cloud the picture. It might increase the risk of developing lymphoma or other cancers.
Having said that, in general, if you have

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the similar exposure rate between cases and controls, then they actually, you know, wash out, technically.
Q. That's why you do the controlling; right?
A. And you try to control --
Q. Why you do the statistical controls, to see if washes out; right?
A. Exactly. You want to try to always control for both to see if it's actual the same. But, you know, I acknowledge, and I think everybody that, you know, look at this or have done some of this research will always have to acknowledge, that it's not always possible in a case-control study to do these controlling for confounding factors.
It is very different when you're doing prospective randomized control study. You can actually control for certain things in the randomization process.
So I don't think it's really because of lack of attempt. I think it's the inherent limitation of these studies to be able to scientifically control for confounding factors between both cohorts in a robust manner.
Q. Do you have any opinion, sir, about how, in a human being, glyphosate would gain access to human

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lymph cells in a way that could cause them to become carcinogenic?
A. I have some opinion, but I want to maybe just mention a couple of things.
It is -- it's very difficult to sometimes know the exact mechanism of action of any carcinogen. I think, you know, we have a body of evidence in oncology. And, as a cancer specialist who've done this for over 17 years, there's a body of evidence to show that sometimes we don't really understand the mechanism by which, A, a drug causes cancer -- even drug works. You know, we have drugs that actually work in cancer that we don't know how they work.
I think there's good data on -- on how it causes chromosomal aberrations and causes chromosomal and DNA breakage which might predispose the cells to developing cancer or -- and/or non-Hodgkin's lymphoma. So there is some evidence of that. There is some evidence that I cite in my expert report on oxidative stress as well that might be a plausible mechanism of action.
But it's -- there is no one way that you can say, well, this is how this is actually caused. The same way I view as many drugs in treating

patients that I knew that they worked but we still didn't know exactly how they actually worked. And years later there was research into how this drug -- why this drug was effective and so forth.

So it's not unusual to see that in cancer, at least in my experience.

Q. Okay. First of all, sir, I'm going to circle back to what I originally asked you, but I want to ask you some other things first based on your answer.

Do you claim, to a reasonable degree of medical certainty, that oxidative stress is a mechanism by which glyphosate, in fact, causes non-Hodgkin's lymphoma in human beings?

A. It's probably one of the mechanisms. It is unlikely to be the sole mechanism. There is no such a thing as sole mechanism.

Q. Okay. Are you claiming that it is one of the mechanisms?

A. It is likely one of the mechanisms to a certain degree of medical probability.

Q. And do you claim that genotoxicity is one of the mechanisms by which glyphosate causes non-Hodgkin's lymphoma to a reasonable degree of medical certainty?

A. I do believe it's one of the other mechanisms, yes.

Q. Now, you know about the Smith and Guyton article on key characteristics of carcinogens, sir?

A. I -- can I see it? I don't know what --

Q. Yes, sir.

(Nabhan Exhibit 7 marked for identification.)

A. I have not reviewed that paper before.

Q. Well, turn, please, to page -- first of all, do you see that it's written by Kathryn Guyton, Christopher Portier, Ivan Rusyn, some of the other people who were involved in the IARC monograph, sir?

A. I do see that, yeah.

Q. And I will tell you for your information that this is a theory paper that was generated by the authors here, the authors listed here, listing characteristics that IARC would, in the future, look for in identifying carcinogens, sir.

A. Okay.

Q. And if you turn to page 715, do you see where those characteristics are listed?

A. I see that, yes.

Q. In the bold?

A. Uh-hum. So this is to demonstrate

characteristics for carcinogenesis. Okay.

Q. And it says here that "Not every carcinogen will have all these characteristics."

A. Right.

Q. "Having a characteristic doesn't necessarily mean that something is a carcinogen, but we will look for these characteristics in identifying carcinogens," in a nutshell?

A. Uh-hum.

Q. And you would agree with that approach. Is that fair to say? Or do you not know?

A. No, I actually like standardization. I think it's very good to have a mechanism by which you look at carcinogenicity. When you standardize the approach, this actually is a better way of looking at things. It doesn't -- but they acknowledge, obviously, that you can't meet all of the criteria and so forth. But it's good -- it's probably a good starting point to standardize things.

Q. And you know that the IARC found evidence for Characteristic 2, that glyphosate was genotoxic, and Characteristic 5, that glyphosate induces oxidative stress; correct?

A. Yes.

Q. And in reading the IARC monograph, did you see that they reached a conclusion as to every single one of the other characteristics and said that there is not -- no evidence or inadequate evidence for the other characteristics?

A. Well, I don't know if they looked at every single one, because this paper was published in June '16 and the IARC paper was in '15. So I doubt that they did because this appears to be characteristics that they actually brought up a year after the IARC was published. So I -- I doubt that they did, but I --

Q. Okay. They did, but let's see whether we need to go through and find them all.

A. I don't know. Yeah, no problem.

Q. Do you claim that glyphosate is electrophilic or can be metabolically activated to electrophiles, Characteristic 1?

A. I'm not qualified to answer this question.

Q. Do you claim that it alters DNA repair or causes genomic instability?

A. I believe there was some data that it causes DNA breakage and chromosomal aberrations. So I believe there is some data to that that I looked at.

Q. And do you disagree with IARC that the data on that was not conclusive?
A. I said there is data. It may have not been conclusive. But I think your question was -- I think if you want to repeat the question, you said, do you believe that it causes DNA repair, genomic instability? I said I saw some data to that effect.
Q. Okay. Do you claim, to a reasonable degree of medical certainty, that that is a mechanism by which glyphosate causes non-Hodgkin's lymphoma?
A. I don't know. I don't know if it is.
Q. Do you claim to a reasonable degree of medical certainty that inducing epigenetic operations, Characteristic 4, is a mechanism by which glyphosate causes non-Hodgkin's lymphoma?
A. I don't believe there's sufficient data to look at the epigenetic alterations of glyphosate.
Q. Let's look at --
A. Just as an FYI.
Q. Yes, sir?
A. The epigenetics is not something we knew about until less than ten years ago. I mean, it's not something that people even know what epigenetics meant.
So, again, this actually tells you, in '16,

now that we know a lot of things, let's apply our knowledge and standardize how we approach things. But if you ask somebody in '95 or in 2000 about epigenetics, they would just say, "What is that?"
Q. Characteristic 6, sir. Do you claim, to a reasonable degree of medical certainty, that glyphosate causes non-Hodgkin's lymphoma by inducing chronic inflammation?
A. I don't know.
Q. Do you claim, to a reasonable degree of medical certainty, that glyphosate causes non-Hodgkin's lymphoma by immunosuppression?
A. There's not enough data to show that.
Q. Or by immunomodulation, for that matter?
A. I have not seen sufficient data for that.
Q. Do you claim, sir, to a reasonable degree of medical certainty, that glyphosate causes non-Hodgkin's lymphoma by modulating receptor-mediated effects?
A. Again, I have not seen data to that -- to that characteristic.
Q. Do you claim, to a reasonable degree of medical certainty, that glyphosate causes non-Hodgkin's lymphoma by causing immortalization?
A. Can you define "immortalization" for me?

Because there is data on apoptosis and affecting the apoptotic pathways and that glyphosate could actually inhibit the ability for the cells to die. And it does affect the apoptosis. So if that's what they mean by immortalization, there's some data. I'm not sure, again, how robust that data is there, but it's there.
Q. Yes, sir. I mean, you'll be testifying as an expert, and I'm -- this is my chance to ask you questions --
A. But this is not my area of expertise.
Q. Okay. Do you claim, to a reasonable degree of medical certainty, that this is a mechanism by which glyphosate causes non-Hodgkin's lymphoma?
A. I don't know if it does.
Q. Do you claim, to a reasonable degree of medical certainty, that glyphosate alters cell proliferation, cell death, or nutrient supply and, by that mechanism, causes non-Hodgkin's lymphoma?
A. Again, I don't -- I don't know if this is the case.
Q. Do you claim, sir, to a reasonable degree of medical certainty, that glyphosate can initiate as opposed to promote cancer?
A. I don't think the -- I don't think that we

have evidence that it does one versus the other. And I think it's a very gray area between initiation or -- or promoting or -- or helping. It's very gray. So it's not clear how it does that.
Again, I mean, it's -- it's -- I'll say this: Not understanding the mechanism of action of a particular compound, whether it works against cancer or it causes cancer, is not something unusual for us who have dealt with cancer for 20 years.
This happens all the time. I have hundreds of examples I can provide in the lack of our understanding of causation or mechanistic, et cetera.
So -- so I don't know whether this is something that is one of the mechanisms of action, and nor is it also important to know the mechanism of action. A lot of times the studies of mechanism on action come after the fact, after you actually show that there's a problem. Like, okay, well, there's a problem. Let's try to figure out why, because then we can eliminate other compounds or other things that may have similar mechanisms of action.
So, you know, I mean, to me, I don't think we understand fully the mechanism of action, but we

have enough evidence in terms of affecting DNA, genotoxicity, oxidative stress.

So there's a plausible evidence out there that it does cause malignancy, but I don't think we have the full picture.

Q. Do you know of any evidence saying that glyphosate causes -- glyphosate promotes non-Hodgkin's lymphoma as opposed to initiating non-Hodgkin's lymphoma?

A. What do you mean by promote versus initiate? Just so I understand so I answer accurately. What's the difference in your mind?

Q. Tell me what the difference is.

A. Well, I told you I don't think there is -- I think it's very gray. That's what I was just trying to say. I said that I think to try -- and, again, we always try to go back to -- you know, I don't believe you can say it's promotes versus initiate versus -- I mean, this is -- these terminologies are very vague and they're very gray. That's why, if you want an answer, I need to understand your definition.

In my definition, I don't believe it matters. I don't believe there's -- I don't believe the discussion of whether it promotes or initiates

or something is very fruitful at all. It doesn't really matter. It doesn't take away or add anything. I don't look at -- I don't evaluate a substance from that angle.

Q. Okay, sir. You agree with me that the transformation of healthy cells into cancer cells is a multistage process?

A. Yes.

Q. And it involves many, many molecular transformations in the cell?

A. Sometimes, yes.

Q. Healthy cells are undergoing oxidative stress and DNA damage all the time without turning into cancer cells?

A. If you have the proper repair mechanism, you don't always turn into cancer, that's correct.

Q. Like thousands of DNA -- thousands of damages to --

A. Yes. If you go in the sun -- you go in the sun, you could have oxidative stress, but it doesn't mean you're going to melanoma right away. Sometimes you have the proper repair mechanism; sometimes you don't.

Q. The sun causes oxidative stress to your cells?

A. Radiation. I mean --

Q. Yes.

A. I brought this on. I mean, radiation causes DNA damages, for example, and you can repair the DNA if you have the proper repair mechanism.

Q. Another cause of oxidative stress is exercise?

A. Don't know.

Q. You don't know?

A. That's a problem, if exercise cause oxidative stress. I don't know if exercise causes oxidative stress. Don't know that.

Q. Every cell in your body is undergoing oxidative stress and dealing with that oxidative stress all the time; right?

A. Yeah. I just -- you asked me if exercise induces that, and I don't know if that's the case. You're right -- your first comment is accurate.

Q. Okay.

A. I think cells go through oxidative stress. And sometimes you are able to repair things; sometimes you can't repair things. But I'm not sure if exercises causes oxidative stress or not.

Q. Okay. So you don't know about the specific one.

A. But there are certainly compounds that could increase the oxidative stress beyond the body's ability to repair, and that is where problems happen. I mean, there's -- the body is in constant balance. There's a constant balance. I mean, what cancer is at the end is cell growth -- cells grow and cells die. So if the balance shifts towards cells growing and proliferating versus cells dying, that's where tumors form and cancer develop.

And so if you have oxidative stress in your body beyond your body's ability to repair things, then it could just shift that balance, and then tumors could develop.

Q. At a minimum for cancer to develop, there has to be damage to DNA that is not repaired and then that is copied successfully and that is of a sort that alters the genetic machinery of the cell towards growth and --

A. And proliferation.

Q. -- immortalization; correct?

A. Yeah. Basically, something happens where these cells continue to grow, and they grow in an exponential manner that cancer develops. Sometimes we know why they grew, because of genomic aberrations, molecular alterations, et cetera. And

sometimes we don't, and we try to study why these happen.

Q. And the first step in that process, the damage to the DNA, is something that happens naturally, endogenously, all the time in every cell in your body but it's repaired ordinarily; correct?

A. It doesn't happen all the time. I mean, are you having DNA damage now? I mean, it doesn't happen all the time. We are not in a constant DNA damage, in a constant -- our body is not in a constant battle between cells trying to die and cells to proliferate. That's not accurate.

I think, you know, there are certain environmental, certain pathogens, certain other factors that get in the body that induces oxidative stress and other mechanisms, and then the body reacts. Either you are able to repair or not. If you are able to repair, you overcome the problem. If you are not, then you shift towards cancer.

But I don't think it's fair to say that, as we are sitting here, the six of us, we have DNA damage happening around the clock.

(Nabhan Exhibit 8 marked for identification.)

Q. Exhibit 8, sir, is an article in the

journal Toxicology and Applied Pharmacology by James Klaunig, et al., entitled "Oxidative stress and oxidative damage in chemical carcinogenesis."

Do you see that?

A. I do see that.

Q. And do you see that in the introduction section they say that the steps of cancer induction have been identified as initiation, promotion, and progression?

A. That's this author's opinion, yes.

Q. And you disagree with him on that?

A. I don't disagree with him. But the accurate thing is that this is their opinion, not mine.

Q. And on page 89, sir.

A. Okay.

Q. Under the heading "Oxidative DNA damage."

A. Yes.

Q. Do you see in the third sentence in that section that the estimated frequency of oxidative DNA damage is at 10 to the power of 4 lesions per cell per day in humans?

A. I see that sentence, yes.

Q. 10 to the 4 would be 10,000 lesions per cell per day oxidative damage?

A. Uh-hum. I see that.

Q. So that would be endogenous DNA damage to human cells caused by oxidative; correct?

A. That's what they're saying.

Q. And 10 -- so that would be 10,000 points of DNA damage per cell in your body every day; right?

A. How are you making -- how are you doing this math, please?

Q. 10 to the 4th?

A. Okay. That's 10,000.

Q. Yes, sir.

A. Yeah, that's what they said. So 10,000 lesions per cell per day in humans.

Q. Right. So it is the case that, as we sit here, we're constantly undergoing oxidative DNA damage and that damage is, for the most part, being repaired; correct sir?

A. Again, I don't know the reference. I'm trying to look at the references. I mean, you just gave me this paper right now that I have never seen. And I think, you know, if we -- if the discussion is about oxidative stress, then I'm sure there are papers that would debate this or not debate this and so forth.

But the conclusion, what I was trying to

say, is that oxidative stress is something that the human body does encounter. I don't know the frequency or what factors induces oxidative stress per se. But what I know, when certain factors that increase oxidative stress, either the body responds by countering the oxidative stress or there's no mechanism to counter the oxidative stress.

That's really all I can say on the subject. I mean, I -- you know, I will have to look at these references and so forth. I don't even know who the authors are.

Q. Okay, sir. So this is something you just don't know about?

A. I know enough about, but you've just given me a paper that I have not seen and you're asking me to comment, and you're just giving me one sentence on page 3 that -- and you want me to comment on that. I mean, so I think -- I think there's a difference between not knowing the subject versus not knowing this paper.

Q. Okay, sir. Do you know or do you not know whether every cell in your body is undergoing thousands of point damage to DNA from oxidative stress every day?

MR. LITZENBURG: Objection. Asked and

answered.
A. I think I answered that. I already answered that.
Q. And you answered that it may be the case; you don't know?
A. I think the answer is maybe, but there are -- again, not going to repeat the same answer.
Q. Now, on the subject of glyphosate, sir, and carcinogenesis, for purposes of this question, I'm going to define "initiation" as what we've just been talking about: Damage to the DNA that then may or may not be repaired that may or may not cause problems later. I'm going to call that initiation.
So do you have the opinion that glyphosate causes non-Hodgkin's lymphoma other than by that, by initiation, by causing initial damage to the DNA, that may or may not be repaired later?
A. I think there are certain -- certainly, there are possibilities that it might. We just don't know yet. I -- you know, again -- and I am more than happy to give you lots of examples, but this is what we know today, and maybe in a couple years there will be additional research to suggest different mechanism of action by which glyphosate causes non-Hodgkin's lymphoma.

So I -- I don't believe -- I don't believe there's any sole mechanism. I believe that we are still exploring this information and there's not enough data to show that this is exactly just the mechanism of action by which a drug works or a compound causes an occupational hazard.
Q. In your expert report, sir.
A. Sure.
Q. I'm on page 10.
A. Okay.
Q. In the last paragraph on that page, third sentence, you say, "The U.S. EPA analyzed immunotoxicity studies in mice exposed to glyphosate and issued a report on February 2013, the results of which were essentially negative."
Correct?
A. I see that, yes.
Q. And what report were you referring to there, sir?
A. I don't remember it. I mean, I -- that's what -- I mean, I -- I recall reading the EPA report. I don't have it handy with me, but I'm sure I can find it.
Q. Okay. And then you said, "These observations were in contrast with other studies

where lethal toxicity was demonstrated in other organisms." Correct?
A. Yes.
Q. And what studies were you talking about and what other organisms were you talking about?
A. I think what I meant by "organisms" is just living cells. I mean, there are some -- some studies that looked at lymphocytes -- bovine lymphocytes, some studies that look at the actual center or level that demonstrated some genotoxicity in DNA breakage. And that's really what I was referring to.
Q. Okay. And when you're talking about bovine lymphocytes, that's a study on page 9 of your report --
A. That's one of the studies, yeah.
Q. -- Sivakova. And that's an in vitro study; right?
A. Right.
Q. Glyphosate was placed directly onto cow lymphocyte cells; right?
A. Right.
Q. And Peluso, which you mentioned next, was also an in vitro study. Glyphosate was placed directly in contact with cells?

A. Right.
Q. And then you say, "These findings are critical as they have been observed in humans," and you go on in the next paragraph to talk about a biomonitoring -- two biomonitoring studies, Paz-y-Mino and Bolognesi; right?
A. I do.
Q. And that's what you mean by they've been observed in humans? You mean those two studies, right, Paz-y-Mino and Bolognesi; right?
A. These are the ones that I found more substantial.
Q. Okay.
A. So I -- I think -- you know, it's always difficult to -- it's not an easy process to actually demonstrate, right. I mean, you have to have the occupation. Plus you have to analyze, collect blood, and do all of these things.
Logistically, it's not always the easiest thing to do. So I found these studies to be interesting and informative.
Q. And in what way were they important to your analysis?
A. Well, specifically the Bolognesi paper where you have -- again, you saw the -- I think --

I'm sure you're aware of the paper where they -- they looked at the micronuclei presence in patients -- not patients -- in individuals that were exposed, and the presence of these micronuclei is a sign of genotoxicity.

So they took blood samples before they spray with glyphosate, five days after, and again four months after spraying. And they discovered the micronuclei in the lymphocytes of individuals exposed to glyphosate.

So at least to me this is somewhat of an evidence that the exposure to glyphosate does cause damage by the presence of these micronuclei, which is a sign of genotoxicity.

Q. How reliable did you find that study to be?

A. I found it to be informative.

Q. What is the difference between informative and reliable, sir?

A. There's really no difference. I mean, just -- I'm not -- do you have a difference? I mean, is informative different than reliable?

Q. I don't -- is it to you?

A. No. I want to make sure I answer the question as you -- based on your question. So to me, they're about the same. Informative and

reliable is the same. But if you have a different definition, I'd like to make sure I -- I don't want to answer the wrong question.

Q. You found these studies to be persuasive that glyphosate could cause non-Hodgkin's lymphoma in humans?

A. I think to the -- well, no. These -- genotoxicity, this is --

Q. It was in support of your opinion that glyphosate --

A. Well, right. Right. But I think these studies are supportive of causing genotoxicity. These studies did not necessarily talk about non-Hodgkin lymphoma. They just talk about the fact that glyphosate exposure causes genotoxicity.

I found the evidence to be compelling given the difficulty in demonstrating something like this. It's not something easy to actually demonstrate. So I actually believe the authors try to do a good job in understanding whether there's any evidence of genotoxicity or not.

(Nabhan Exhibit 9 marked for identification.)

Q. Wait a second. Hand me that back, please. I need to mark the right one.

A. (Witness complies.)

Q. Okay. Sir, this is Paz-y-Mino study from 2011 that you cited in your expert report --

A. Yep.

Q. -- on page 9.

And this is a study in which various people who were undergoing aerial spraying with glyphosate near the border of Colombia were compared to some controls; is that right?

A. Uh-hum. Yes.

Q. And take a look at the abstract where it says towards the bottom, "In conclusion." The conclusion of the authors here was, "In conclusion the study population did not present significant chromosomal and DNA alterations."

Correct?

A. I see that.

Q. So this was a negative study; right?

A. Well, depends how you really interpret this. I mean, I think the reality is that there was -- there was evidence of chromosomal damage and DNA alterations. It did not reach statistical significance, and I think that's really different.

So -- so sometimes -- you know, the fact that there was -- and we talked about this earlier.

The fact that, in some of these studies, you don't have the statistical significance defined as a P value less than 0.05 could be related to the number of cases, the way the study was done. And it might be related -- it's just a number game -- the number of folks that were actually in the study.

So you look at the trend and you look at the entire evidence, taken this study plus other studies involved.

Q. Under "Chromosomal Analysis" on page 48, sir, it says, "After analyzing the meta-phases and karyotyping the 92 individuals who belonged to the different communities of the province of Sucumbios, located in Ecuador's northeastern border, we observed that all the analyzed women obtained a normal karyotype."

Right?

A. I see that, yes.

Q. And there is no statistical trend, much less statistically significant association, between glyphosate exposure and genetic damage in this study; right?

A. I don't see this in this paragraph. I'll have to read the whole paper, because I remember reading this paper. I'll have to reread it.

Q. At the bottom of page 50, sir, it says, "Regarding our study" -- it's the very last partial sentence at the bottom of page 50, the first column. "Regarding our study, we obtained results showing no chromosomal alterations in the analyzed individuals."

Correct?

A. (Speaking sotto voce.)

I see that, yes.

Q. Everything we've looked at is negative; correct?

A. In this study, it appears that the authors believe there is very little association with chromosomal aberration.

Q. They didn't even say very little; they said none. Right?

A. That's what they said, yes.

Q. Okay.

(Nabhan Exhibit 10 marked for identification.)

Q. I'm marking as Exhibit 10 the Bolognesi 2009, which is the other paper that you cited in your expert report on genotoxicity; correct?

A. Correct.

(Whereupon a discussion was had off the record.)

BY MR. GRIFFIS:

Q. All right. So this study involved -- was looking at micronucleus formation in subjects from five regions in Columbia, again where aerial -- in some of those areas, aerial spraying of glyphosate was being done; correct?

A. Yes.

Q. And the highest frequency that was found was in an area where no aerial spraying was being done; correct?

A. Which page is that?

Q. It's in the abstract, "The highest frequency --"

A. I see that.

Q. -- of BNMN was in Boyacá" -- or Boyacá -- "where no aerial eradication spraying of glyphosate was conducted."

A. I see that, yes.

Q. And then on the next column of the abstract, "The increase in frequency of BNMN" -- what's BNMN by the way?

A. The micronuclei.

Q. Okay. So that's --

A. MN is micronuclei.

Q. -- the indicator of genotoxicity that we're looking for in this study; right?

A. Yeah, it's one of the indicators that's used for genotoxicity.

Q. So the increase in frequency of BNMN observed immediately after the glyphosate spraying was not consistent with the rates of application used in the regions, and there was no association between self-reported direct contact with eradication sprays and frequency of BNMN; right?

A. Yes, I see that.

Q. And then the -- the end of the conclusion of the abstract is, "Evidence indicates that the genotoxic risk potentially associated with exposure to glyphosate in the areas where the herbicide is applied for coca and poppy eradication is low; right?

A. That's the conclusion of the authors.

Q. So they did not find any dose -- any relationship with dose in --

A. That's not unusual. I mean, not everything is dose-dependent, especially in cancer. I mean, there are many drugs that we use that are class effect. You give a drug that causes a side effect, whether it's 10 milligram or 100 milligram, because

it's a class effect.

Just because you give 100 milligram, it doesn't mean you're going to have more side effect all the time. There are many examples of this.

So, I mean, I think the dose relation, to me, is not -- what I take from this paper is the fact that there is evidence that there is genotoxicity that is associated with this compound. And it's very hard to control, when you're just spraying aerially in these regions, to be 100 percent certain how this is -- it's very difficult to -- to really control for.

But in my opinion, the dose is not always associated with the actual output, especially in cancer.

Q. For genotoxicity of the sort that's measured in this -- that they tried to measure in this study, to lead to cancer, it would need to cause persistent DNA breaks, not just temporary ones; correct?

A. I don't agree with that. I think, if you have a DNA break that is not repaired, you -- it could manifest, you know, typically later on and develop cancer. It's not -- if it's not repaired right now and it's still having a damage, it doesn't

mean you're going to have cancer tomorrow. I mean, that's well known.
Q. Right.
If there are DNA breaks --
A. And it's not --
Q. -- and they're repaired, they will not cause cancer; and if they aren't repaired, they could cause cancer. Is that fair?
A. If there is a DNA breakage and it's repaired and everything is back to normal, the cell then -- then other mechanisms could be contributing to the evolution or the development of cancer, not this particular mechanism.
If the DNA breakage is witnessed and it's not repaired, then -- then it might contribute developing cancer, but that could actually happen later on, not necessarily now.
Q. And in this study, they didn't come back and look to see whether any of these breaks were persistent; right?
A. I don't think it's logistically possible. But, to my knowledge, they have not. I mean, you have to follow up this population for a long, long time.
Q. DNA breaks is the same thing that we were

talking about earlier when we were looking at the Klaunig article about 10,000 lesions per cell per day in the human body; right? Those are DNA breaks?
A. I think that was oxidative stress --
Q. Yes, sir.
A. -- if that's what you just mentioned.
Q. DNA breaks due to oxidative stress.
A. I think the -- it says here, "Estimate frequency of oxidative DNA damage." I mean, there are DNA -- DNA could be damaged by mechanisms outside of oxidative stress.
Q. Oh, sure.
A. Right. I mean, so this -- the Klaunig paper, I think they're talking about the DNA damage specifically for oxidative stress. I just want to emphasize that this is not the sole mechanism by which DNA damage occurs in the cell.
Q. DNA damage such as is purported to be measured in the Bolognesi paper is happening all the time, and what's important is whether it gets repaired or not; fair?
MR. LITZENBURG: Objection to the characterization.
A. I don't agree with that. I mean, can -- I mean, to -- to agree with you, you will have to show

me that the folks who -- again, the DNA damage is going to happen regardless of anything whatsoever.
And, again, I don't -- I'm not qualified to answer this question. I really have to research it to better understand whether DNA damage occurs regardless of any etiologic factors.
I see the paper that you've provided, and I see the reference. And I think, to some extent, this is true. You see sometimes the DNA damage and repair that happens in the cells in the body. But, in my mind, there's always some additional factors that are involved. It could be diet, could be environment, could be drugs, could be anything.
Q. You testified earlier that you looked -- in addition to the scientific articles that you reviewed and talked about in your expert report, you also looked at a number of articles about scientific articles criticizing IARC or criticizing the EPA and so on; correct?
A. I said that?
Q. Yes, sir.
A. I said that I looked at scientific articles as well as the IARC and so forth. That's what I said.
Q. And you saw criticisms of EPA and their

methodologies?
A. I saw criticism of EPA methodology, correct.
Q. Things like letters to the editor and press reports; right?
A. But you said criticism to the IARC and --
Q. Did you not read any criticisms of IARC?
A. I personally have not seen the criticisms of the IARC but more than happy to look at it, if you have it.
Q. Okay.
A. I mean, I saw -- I told you I read the IARC Monograph, which you provided to me, as well as the actual paper.
Q. In doing your self-directed research, you found only criticisms by IARC participants of EPA and EFSA, the European Food Safety --
A. I think you can critique --
Q. -- Agency, and you did not find any criticisms of IARC; is that right?
A. You can critique every study under the sun. Every study, you can critique. There is no perfect study. And we just established, I hope, earlier that the only perfect study is to take 2,000 patients and randomize them to exposure versus not,

which, hopefully, everybody around the table agrees, is unethical to do.

So there is a criticism for every trial that we have, for every study that we have. And because of this, because there's no perfect study, I have to look into the -- all of the evidence together and try to come up with a conclusion.

IARC, in my opinion, is more authoritative in this particular type of studies and in this particular type of situations than any other agency.

And so I do rely heavily on what the IARC says, especially when it's published in a very prestigious peer-review journal.

Could you critique it? I'm sure you can, but it doesn't take away from the weight of the evidence.

Q. Let me ask my question again, sir.

A. Please.

Q. In your self-directed research, you came across multiple criticisms of EPA and EFSA and others generated by IARC authors, but you did not come across and read any criticisms of IARC; is that right?

A. I have not seen that, no.

Q. Okay. Do you know that Dr. Solomon, one of

the coauthors of the Bolognesi 2009 paper that you quoted in your expert report, was interviewed and said that IARC got this paper, the Bolognesi 2009 article, totally wrong if they thought that it was evidence of genotoxicity because it's not?

A. But the IARC looks at all of the evidence. They don't really look at one paper versus another. I don't think the IARC's goal -- the IARC has to look at the collective evidence. They did not take this paper -- I don't think the IARC -- I don't want to speak for the IARC. And you can obviously interview them and -- and they're available. But I don't think the IARC took this paper and say, okay, based on the paper by Bolognesi, et al., there is evidence of genotoxicity.

I believe that they have looked at a collection of evidence, at a lot of evidence. And they came up with the conclusion that there is enough evidence here -- there is plausible evidence here that genotoxicity exists.

It is not fair to say that they just reviewed this paper. And, frankly, Dr. Solomon, if he said that, for him to assume that they relied on his paper only is a little bit strange because he is ignoring the evidence of other folks.

But I think these questions should be directed to IARC. I don't represent IARC.

Q. You think I should go ask IARC and they should answer my questions?

A. I don't represent IARC. That's for sure. They can't pay me enough.

MR. GRIFFIS: Let's take a break. What time is it?

VIDEOGRAPHER: We are going off the record at 11:52 A.M.

(Lunch recess taken from 11:52 A.M. to 12:41 P.M.)

AFTERNOON SESSION

(Time noted: 12:41 P.M.)

VIDEOGRAPHER: And we are back on the record at 12:41 P.M.

THE WITNESS: Before we start, I want to just say something for the record, please.

So in no way any of my testimony is related to Cardinal Health or my employment. The opinions I provide today are my own individual opinion. I do not represent the opinion of Cardinal Health, my current or previous employers. So these are my own opinions. Thanks.

C H A D I N A B H A N,

resumed and testified as follows:

CONTINUED EXAMINATION

BY MR. GRIFFIS:

Q. Sir, have you read the expert report of plaintiffs' epidemiology expert, Dr. Ritz?

A. I have not.

Q. Okay. In a section of her expert report where she is discussing epidemiology studies on non-Hodgkin's lymphoma and offering some critiques on the length of time that passed between initial exposures and onset of disease, she makes this

comment: "Typically, we would generally expect a five- to ten-year minimum latency between exposure and disease onset for blood system-related cancers."

She also notes, sir, that in an individual case it may be a lot shorter; it may be a lot longer, but talking about the studies.

So the statement, "Typically, we would generally expect a five- to ten-year minimum latency between exposure and disease onset for blood system-related cancers," in your opinion, is that an accurate statement with regard to non-Hodgkin's lymphoma?

MR. LITZENBURG: Object to the paraphrasing. And he's also said he hasn't reviewed that document.

A. Yeah. I have not reviewed it, but I don't agree with it. I really do not believe that we have -- I'd be very curious to know how she formed this opinion. What level of evidence did she -- I presume it's a she? You said she?

Q. Yes, sir.

A. I presume there is some evidence that she used to form this opinion. I don't know what that is, because latency period, as we talked about, is a very gray area, and I -- as you just articulated

could be less, could be more.

So I don't know if there is really a median, and I don't know why would that be different for hematologic cancers versus solid tumors.

I would say the latency period is a -- is a very broad category that will really vary based on each individual case.

Q. How quickly could a toxic exposure produce a non-Hodgkin's lymphoma?

A. Yeah. I mean, so we did talk about there are some non-Hodgkin's lymphoma that you may not find a toxic exposure. You have a clinical case. You sit with the patient and you talk with the patient and you go through the entire history, and you may not find that particular red flag that tells you that there was something in that patient's history that led to the development of non-Hodgkin's lymphoma. And you may find it. I mean, depends on each history.

If you find that there is a red flag, the actual period becomes irrelevant because it doesn't really effect what you do as a clinician. It doesn't affect management. It doesn't really impact anything else you would do.

But it would depend, I presume, based on

the actual disease.

I mean, it's unlikely to be that you get exposed to something today and you get cancer tomorrow. I mean, we understand the -- you know, logically, you would have to have some period of time.

All I'm trying to say is I'm not sure that we know in oncology what is that minimum versus maximum in terms of -- because there are so many or factors. Every patient that smoked that I've taken care of has said, "Well, my uncle smoked for a hundred years, and he's never died of cancer." And it's true, because maybe there are other factors involved versus somebody who is less lucky.

So I truly don't have an adequate scientific opinion that I can tell you that there should be five to ten years. I think if somebody is claiming this, I would like that claim to be supported and substantiated by actual evidence. I'd like to say the reference that she used, because we can have my opinion that is completely contradicting to this opinion.

Q. You don't believe that every patient or even most patients with non-Hodgkin's lymphoma got it because of a toxic exposure in their past, do

you?

A. Not every patient gets non-Hodgkin's lymphoma because of toxic exposure, that's correct.

Q. In fact, the majority probably don't get it due to a toxic exposure; right?

A. It depends on the occupation. I mean, I think if I'm studying folks in a particular occupation or in particular area that they may have similar occupation, or specific county or state, or so forth, I probably will find that common denominator. But if you're talking about the population, I mean, there is about close to 73,000 new non-Hodgkin's lymphomas designated every year, at least in '17. The majority, I may not be able to find that toxic exposure.

Q. You said, "I may not be able to find that toxic exposure" as if there is one and you just haven't found it.

A. Well, they may be one not necessarily toxic exposure. I mean, I think that -- you know, you don't always find the etiology of a particular malignancy to diagnose with patients. I mean, you would love to. As a clinician and as a researcher, I would like to have the cause of every single cancer, because if you have the cause, you find the

treatment. And we've demonstrated this, once you know the actual cause.

What I'm saying is that not in every clinical case you are able to find that red flag that tells you aha -- that aha moment -- I think you developed non-Hodgkin's lymphoma because this is what you do for a living. I don't always have that in every single case.

Q. And in which occupations do you believe that a majority of the cases are caused by an occupational exposure?

MR. LITZENBURG: Object to form.

A. I think there's good evidence that farmers have that. I think there is some good evidence out there that farmers have higher risk of developing non-Hodgkin's lymphoma as opposed to folks who do not work in farming.

Q. Any other occupation?

A. I can't recall now, but it's an interesting question that I've been interested in. I can't recall right now.

Q. With regard to farmers, there was epidemiologic evidence suggesting an increased risk of non-Hodgkin's lymphoma before glyphosate was on the market; right?

A. Yes, there is evidence that farmers do have increased risk of non-Hodgkin's lymphoma.

Q. Separately from the existence of glyphosate; correct?

A. I'm just trying to recall, because you said before the market. I'm trying to recall when that -- that --

MR. LITZENBURG: Do you have epidemiology published before 1974 or data from that?

A. Yeah. I'm trying to remember when did it go to market. I'm not remembering that exact.

Q. In the middle of 1974.

A. Yeah. So I did not review epidemiologic literature before 1974. I think the first paper I looked at was -- it's somewhere here probably by Cantor and colleagues -- was '92 paper. But, again, like we talked about, sometimes you don't have that time frame.

I think there is good evidence that farmers have increased risk from an occupational perspective to developing non-Hodgkin's lymphoma. How that relates to when glyphosate was in the market and the market uptick of that compound -- because once you go 1974, maybe the market uptick is higher than '84 and '94, you know. So I can't relate those

together.

Q. Okay. So you're not familiar with the literature on farmers and elevated risk of non-Hodgkin's lymphoma predating the existence of glyphosate in the U.S.; correct?

A. I did not review epidemiologic data before 1974, and I said I don't -- I don't know how fast the market uptick for glyphosate. I'm sure it's available, but I don't know how fast it got the uptick.

Q. You said earlier, if I heard you correctly, that, if you find the cause of a particular case of non-Hodgkin's lymphoma, then you have the treatment?

A. No. You -- no. No.

Q. Maybe I heard --

A. No. I said you at least start thinking, how can I develop treatment that's directed to the cause? If you -- if you know that a protein is mutated -- a gene is mutated that's causing a particular cancer, then you can develop a particular therapy against that gene or, you know --

Q. I see.

A. -- et cetera.

Q. But if you know that it was DDT that caused the non-Hodgkin's lymphoma, that doesn't give you

any clues about how to treat it; right?

A. Well, I would eliminate the cause. Right? It's like smoking. If you know that smoking causes cancer, you just say stop smoking. So I would stop using the causative factor. That's the easiest thing of prevention.

Q. You're not going to cure them, though?

A. Some lymphomas are curable, not all lymphoma -- I mean, lymphoma, like we say, it's 60 types of lymphomas. In fact, one of the rewarding things in lymphoma, that we cure some of these lymphoma. We cure many lymphoma. Depend how you define "many," but we do cure some lymphoma.

Q. Do you believe that the majority of cases of non-Hodgkin's lymphoma would not have occurred but for an environmental exposure?

A. I don't believe that. I think that there are not -- not only environmental exposures cause non-Hodgkin's lymphoma. We talked about viral association. We talked about environmental factors. And we talked about the fact that we may not understand completely and fully all of the causation for non-Hodgkin's lymphoma.

Q. Do you have an opinion as to relative prevalences of -- of heredity -- i.e., genetic

facts -- versus environmental factors versus just replicative factors, the ongoing division of cells and errors that randomly creep into the ongoing division of cells in the causation of non-Hodgkin's lymphoma?

A. So I don't know what you mean by heredity, but what I can say is that there is familial predisposition. There is data in non-Hodgkin's lymphoma, like a lot of cancers, not -- not the most common. But if there is a family history of non-Hodgkin's lymphoma, the offspring are at higher risk of developing lymphoma, like breast cancer and so forth. So there is such a thing in terms of familial association.

Now, you have to be careful. Familial association does not imply or mean that there's a particular gene that is necessarily mutated or so forth. These are different things.

So, yes, there is -- you know, family history is a known risk factor. That's not modifiable, frankly, except just good history and physical and good -- good medical care.

The other two areas which were -- that you asked --

Q. Environmental.

A. Yes. I think there is --

Q. And --

A. Yeah.

Q. And the other is replicative, just the fact that all of your cells are replicating themselves all the time and random errors, by biological definition, creep into in a process and can ultimately lead to cancer.

A. Yeah, that happens with age. Yeah, I mean, with age, as we age, the ability of our cells to repair some of the damage, unfortunately, becomes less. So, yes. I mean, I think these are the cases where that's why nobody lives till 200 years. I mean, at some point something is going to go wrong. And as we age, these things do happen.

Q. So do you have an opinion with regard to non-Hodgkin's lymphoma as to the relative prevalences of those three factors: environmental, hereditary, and replicative --

A. Yeah. I don't think we know the data.

Q. -- in causation?

A. I don't think we know that data. But we know that non-Hodgkin's lymphoma is more of a disease of the elderly. Median age of diagnosis for most non-Hodgkin's lymphomas are above 60, 65 plus,

actually. There are obviously some subtypes that you see in the 30s to 40s, et cetera. But I don't think -- to my knowledge, we don't have a percentage of how often you see something based on environmental factors, because to have that data you would have to eliminate all other factors. And this is tough to actually know.

Q. It's logical and accurate to think about the replicative risk as a sort of statistical risk that's imposed upon you over time. I.e., all the cells in your body reproduce themselves. By biological necessity, there are random errors in their reproduction and some percentage of those random errors will ultimately lead to cancer.

So everyone is at risk all the time, at some low level of risk, for all types of cancer, including non-Hodgkin's lymphoma, because of that biological fact. And that risk increases as the replications increase and, thus, over time. Is that fair?

A. I think if you're asking if everybody in the population at risk for developing cancer at some point because of this, the answer is yes. I mean, in fact, the last statistic from the American Cancer Society is that the lifetime risk of a male in the

U.S. develop cancer is, unfortunately, about close to 42 percent in a lifetime. So that's huge. In a female, it's about 43 percent or so. So I think, if we live long enough, we're going to have a problem.

Q. What is the lifetime risk of non-Hodgkin's lymphoma?

A. I don't know that. I think it looks usually -- I think the data that I read from the ACS was mainly in developing malignancy in general. But what I can tell you there are -- the last statistics paper, the number of new cases of non-Hodgkin's lymphoma in the U.S. was between 72 and 73,000. It's published by Siegel and colleagues.

Q. On your expert report, page 11, I'd like to turn to the epidemiological studies. On page 11, you have a large category header titled "Assessment of carcinogenic risk in humans," and your first category is "Epidemiologic studies." Right?

A. Yes.

Q. You say, "Several epidemiological studies showed statistically significant increased risks among people exposed to glyphosate." And the first study that you talk about is by McDuffie, et al., from 2001; is that right?

A. Yes.

Q. Sir, did you put these studies in any particular order?
A. I don't remember. I may have tried to put them in the order of the years that were published. I think that's what I tried to do. I think McDuffie's '01. Then you have Hardell '02. DeRoos '03. I may have tried to do that. I like to do that chronologically. It's possible that's what I did.
Q. Okay.
A. But it doesn't mean -- I did not order them by importance, if that's the question.
Q. Okay.
(Nabhan Exhibit 11 marked for identification.)
Q. I've handed you a copy, sir, of the McDuffie 2001 paper.
A. Okay.
Q. Now, this was a study of herbicides and pesticides in general and their association non-Hodgkin's lymphoma; correct?
A. Correct.
Q. It was not focused specifically on glyphosate; right?
A. It was on -- it was in general, but I think

they had -- I'm trying to see if they subanalyze glyphosate. I think it was for general exposure, to my knowledge.
Q. When they are describing the questionnaires that they sent out on page 1156, second column --
A. Uh-hum.
Q. -- the specific exposures that they talk about were first major classes, herbicides. I'm at the end of that first paragraph.
A. Okay.
Q. Chemical groups and the example they give is phenoxy herbicides and finally to individual compounds, 2,4-D MCPA, and 2,4,5-T. In their description of the initial hypotheses, they didn't specifically mention glyphosate; right?
A. That's correct.
Q. I'm sorry?
A. That's correct, I said.
Q. Yeah, I thought Mr. Litzenburg said something.
And the authors noted that, because they were looking at results for multiple chemical agents and exposures that weren't specifically set out in the hypothesis, the statistical analyses should be considered exploratory; right? That's on page 1161,

second column.
A. Yeah. I just saw that they looked at the glyphosate on page 1158. It shows an odds ratio of 1.26.
Q. Yes, sir. It's mentioned.
A. Right, right. I just -- initially, I said I didn't -- I didn't know. So 1161?
Q. 1161, second column.
A. Okay.
Q. They say, "We reported results for a number of chemical agents and exposures, not all of which were specified in the hypothesis. Therefore, the statistical analyses related to these unspecified agents should be considered exploratory. As a consequence of conducting multiple comparisons, a small number of statistically significant results may be attributable to chance."
I read that correctly?
A. You did.
Q. Would you explain to the jury what concept they're talking about where, when you do statistical analyses on many different chemicals simultaneously, you will get potentially, apparently, significant results only due to chance?
A. I mean, first, I can't speak for the

authors. I only can speculate. I think it's really fair, if you really want to know what they actually meant, to direct that question to them.
But what I would say is oftentimes, if you have a study that is looking at multiple occupational hazards or occupational exposures, there are limitations to how much you can control for these additional occupational hazards in order for you to tease out the impact of one particular compound versus another.
So I think they're leaving just some open room, which is appropriate, to say, okay, well, you know, these results are important, but they have to be taken in context. Additional studies are needed, and there may be some we cannot be 100 percent conclusive that this is not related to chance. So that's why we can't really take one study alone and we have to look at all of these studies that were done.
Q. For example, sir, if you're using a 95 percent confidence interval and -- confidence level, rather, and you looked at 20 different compounds, you would expect to find at least one statistically significant association solely due to chance; right?

MR. LITZENBURG: Object to form.
A. I'm not sure. I mean, based on what?
Q. That's how the statistics work. 95 percent is 1 in 20.
A. But why one, not two, why not zero? Where do you get one from? I mean, I don't know.
Q. An average of one.
A. No, but my point is each study is different. I mean, I don't think we know. I think your point is well taken that there are other factors that contribute. So that's why I think the authors here, they say some element of this could be attributable to chance.
I just don't believe that we can generalize and say, if you take 20 compounds, one or two would be due to chance. I don't know that. You'd have to conduct the study and to see what methodology that you've actually done before you have a general statement. Otherwise, you can't even review any epidemiology literature, positive or negative.
Q. Well, sir, if you're doing -- if you're using a 95 percent confidence level --
A. Yes.
Q. -- what that means is that a purportedly statistically significant result is at least 1 in 20

likely due to chance; right?
A. So the P value for statistical significance is usually less than 5 percent -- less than 0.05, which means that, as long as you have enough evidence that 5 percent or less of whatever you are doing is due to chance, then that's really clinically important or statistically significant.
So if I have an experiment, 5 percent -- you know, and the P value of this experiment less than 0.05, then I am admitting that 5 percent could be due to chance. That's really all you could say.
Q. All right. And 5 percent is 1 in 20?
A. I -- I see what you are saying. Okay. I guess so.
Q. Okay.
A. Now I understand what you mean.
Q. You say in your expert report, sir, on page 11, referring to the McDuffie study, "Among major" -- I'm sorry. I'll wait for you to get there.
A. I'm good.
Q. "Among major chemical classes of herbicides, the risk of NHL was statistically significantly increased among glyphosate-exposed individuals with an odds ratio of 1.26, 95 percent

confidence interval 0.87 to 1.80, which changed slightly after adjustment for covariants to an odds ratio of 1.2, 95 percent confidence interval of 0.83 to 1.74."
Did I read that correctly?
A. You did.
Q. And neither one of those odds ratios is, in fact, statistically significant; right?
A. I don't know that. I think you just -- you have to take the odds ratios above 1.
Q. A statistically significant odds ratio is one where the 95 percent confidence interval does not cross 1; right?
A. No, no. I understand what you meant, but I'm just saying it doesn't take away that there was an increased risk, because we talked about this earlier that the statistical significance per se is dependent on the -- on the number of cases, the -- I mean, that's why certain studies may fail to have the statistical significance per se because you don't have enough numbers to show that, but you can't ignore increased odds ratio when you have an exposure like this.
A positive study will always -- if you have enough odds ratio that is above 1, it is something

important to look at. You can't ignore it. The lack of statistical significance is a completely different beast because then you look at the -- how many cases were looked at, how many controls were looked at, was the study powered enough to actually detect the statistical insignificance or not.
Q. Sir, you said it was statistically significantly increased in your expert report; right?
A. Yes. And what I meant by that was the odds ratio was above 1.
Q. By the definition of "statistical significance" used by the McDuffie authors, it wasn't statistically significant; right?
A. Where do you see that on the McDuffie paper?
Q. Well, I see it in the confidence interval that you put in your expert report. I also see it in Table 2.
A. But you said -- in the McDuffie paper, you said that they defined -- you have a different definition.
I mean, again, when I read this paper, I think the McDuffie paper, they say that we see increased risk and we really acknowledge that some

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of it could be related to chance. So additional studies are needed.

The conclusion of the authors is hypothesis generating that there's actually some risk here that cannot be ignored. And while this study may not be conclusive, additional studies are actually needed.

Q. Let's look --

A. So I don't see the interpretation that this was a negative study.

Q. Table 2.

A. Okay.

Q. Under Table 2, "Glyphosate" --

A. Uh-huh.

Q. -- they give two adjusted odds ratio, Odds Ratio A and Odds Ratio B.

A. Uh-hum.

Q. And they give a 95 percent confidence interval.

A. I see that.

Q. That's their definition of "statistical significance" selected in advance for purposes of this study. And by their definition of "statistical significance," a 95 percent confidence interval, neither of these results is statistically significant; right?

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A. Yeah, it may have not reached the P value of less than 0.05, but I personally would not ignore an odds ratio of 1.26 or 1.2.

Q. Okay. Let's just start with statistical significance.

A. If you're --

Q. Do you --

A. -- defining the statistical significance of less than 0.05, then this was not statistically significant.

Q. And when an author selects a confidence interval, that is their definition of statistical significance for purposes of their paper; right?

A. No, I mean, the -- when an author selects statistical significance of less than .05, then after that, they have to decide how many cases they need to get enough sample size to get to that threshold. So each case is different. That's why I was trying to read the methodology, to see how powered it was.

The 95 percent confidence interval is just the range that they actually get. So the narrower the range, the better it is if you can get that. But it's very difficult to demonstrate in epidemiologic studies just by the nature of how you

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do these studies.

Q. In science, when you're looking at a particular study, the definition of "statistical significance," for purposes of that study, is the confidence level that was selected in advance by the authors, here, 95 percent; right?

A. But let me just explain. I mean, statistical significance -- significance is a completely arbitrary chosen thing that's less than .005. So -- so if I have -- if -- I'm just saying, if I have -- if I have a P value of 0.06, I have to look at the trend, right. I mean, I have to look -- does it mean that only -- I will take only the 0.05 and ignore everything else? Because sometimes you have two patients -- just two patients that completely change the curve.

So as a clinician-researcher, you -- you look at this and you say, Okay, I mean, I get this. Let me look at additional data. Let me look at additional information to solidify the opinion.

At some point, statisticians and researchers have to agree on what is that point that we are allowing chance to play a factor, and they agreed on 5 percent. They could have done 4 percent. They could have done 6 percent. But

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that is why it's very -- it's a double-edged sword. We have to make sure that we put everything in context.

You can't -- you can't ignore a study that showed a P value of 0.06 and say it's not statistically significant, and you can't agree on every study that was significant. I mean, that's why, as clinicians, we have to interpret the evidence.

MR. LITZENBURG: Hang on for a second before we ask any more questions. It sounds like there's still hold music on the line for everybody dialing in. Can we figure that out?

MS. SALEK: Oh, really? Do you want to go off the record? I can dial in.

MR. GRIFFIS: Okay.

MR. LITZENBURG: Anybody on the line can hear us?

VIDEOGRAPHER: Going off the record at 1:11 P.M.

(Recess taken from 1:11 P.M. to 1:15 P.M.)

VIDEOGRAPHER: And beginning Disc No. 3 of the deposition of Dr. Chadi Nabhan. We're back on the record at 1:15 P.M.

BY MR. GRIFFIS:

Q. Okay. Dr. Nabhan, you were just giving us a critique of statistical significance as applied to causation.

What I'm focused on right now is your expert report and your claim in your expert report that the odds ratios reported in Table 2 of the McDuffie paper were statistically significant.

A. What I meant by this is that the odds ratio were more than 1. I did not imply that the P value was less than 0.05.

Q. So when you say "statistically significant," what you mean is an odds ratio of greater than 1?

A. Yes.

Q. Does anyone else mean that when they say "statistically significant"?

A. I can only speak for myself.

Q. You said that scientists, epidemiologists, I presume, oncologists, have settled on the convention of a P value of .05 for statistical significance.

Why have they done so?

A. They had to have a point to agree on. They accepted that 5 percent chance is okay. There are

many studies that were statistically significant that they had no clinically meaningful outcome in cancer therapies. Just you have to agree on something to standardize things.

Similar to the paper that you showed me into standardizing genotoxicity assays, at some point, the field has to agree that, if we're going to assess genotoxicity, these are the ten things we're going to do. So it's just standardized things so at least you compare apples to apples.

But you can't -- as a clinician, I can show you many papers that showed a P value of less than 0.05 that meant nothing, that showed an improvement in treatment of 1.5 weeks. Does this mean how clinically meaningful it was? It was great paper. It was New England Journal of Medicine paper, P value less than 0.05. It was in pancreas cancer, but the actual difference between the actual treatment and control was 1.5 weeks.

So we can argue as scientists all we want. We ultimately have to look at the totality of evidence. And a P value of less than 0.05 is very important, but it's not the only thing that we look at.

Q. And it's one of the things that the field

has settled on as part of the scientific discourse on causation; correct?

MR. LITZENBURG: Objection. Asked and answered.

A. Again -- I mean, again, it is one of the -- you can't -- you can't just be blindsided only and say I will only look at literature that has a P value of 0.05. I mean, it is -- you would -- you would be at fault to doing this.

I think that, if you design a study based on the goal of the trial or the study that you are trying to do, if your goal is to demonstrate statistical significance, then you want to power that study to have a P value of less than 0.05.

And I can assure you, by the way, if you have enough patients in any study, every study would be statistically significant. If you take 20,000 patients, eventually you would get a P value of less than 0.05, but it's not practical. So that's why you look at other things such as odds ratio, risk ratio, and so forth.

Q. Were the adjusted odds ratios in Table 2 adjusted for other pesticides?

A. So I think it has a footnote of D. It says, "Glyphosate is the only phosphonic acid

herbicide reported by more than 1 percent of responders. Roundup, Touchdown, Vector, Wrangler, Laredo do not include dicamba. And Rustler is mixture of dicamba and glyphosate."

I -- I presume B adjusted for statistically significant medical variables. So they adjusted for history of measles, mumps, cancer, allergy, desensitization shots, and a positive family history of cancer in first-degree relatives. These are the things that they adjusted for.

Q. They did not adjust for exposure to other pesticides?

A. No. It says -- they did not mention that here.

Q. Do you agree with me that negative data pretty much never makes it to the major journals?

A. No. I would say that people are always biased to publish positive data because they get that to more higher-impact journals and because it gets more press, but journals now are becoming increasingly interested in having negative data because they could be as important and as powerful as positive data.

But, in general, people are -- always like to report positive data, that it was a positive

trial, positive association, just an inherent bias.
Q. It's called publication bias; right?
A. Yeah, it is a publication bias.
Q. And publication --
A. Sometimes you can have a negative study that is sitting in your drawer that you decide never to publish it because you have more pressing needs. You publish your positive trial. You spend more time on it as opposed to publishing a negative study because you know, if you publish a positive study you are going to get a better journal, maybe get a grant, maybe get -- I mean, it's just the way it is.
Q. The -- because of publication bias, you're more likely to see, in the published literature, positive than negative results; right?
A. I think you have -- you'll see more positive literature published, but I think the main difference -- honestly, what I have seen lately is that the negative studies, they still get published, but they publish -- they are published in lower-impact journals. They still have a role. But, to your point, some negative studies will never be published because people will never get to them.
Q. And the positive ones -- the negative ones never make it to the major journals?

A. Not all of them. I mean, some of them will still make it. It's just not -- you know, not the same power. But as I said, a lot of trials -- negative trials now are making it to the -- to the -- to our major journals.
You know, a recent paper in the Journal of Clinical Oncology showed the lack of association of androgen-deprivation therapy and dementia in men. So that's -- it's a negative study. They didn't show positive association, et cetera.
So I think you are seeing this. But it's always the case, if you are the author and you have one negative trial and one positive trial, you're going to try to get the positive one out because it might allow you to advance academically more. It's just the world that we live in.
Q. I'm showing you a tweet you wrote.
A. Oh, I like that.
Q. "Negative data never make it to major journals" --
A. Are you following me on Twitter?
Q. -- "this would be big news."
A. Yes. Negative data never make it to major -- this is published still. So this is actually my point. This paper is published. It's a

lower-impact journal, "Clinical lymphoma, Myeloma & Leukemia" -- and Myeloma.
So if this was a positive study, I think this particular paper would have made it in a much higher-impact journal. It actually solidifies what I just said.
Q. The tendency of the published literature --
A. Is this -- do I leave this?
Q. -- to reflect positive results and to under-reflect negative results, that's called in science "publication bias." Right?
A. Yeah, I mean, I think I said that a few times. I'll say it one more time. Negative trials or negative data will still make it to journals, but it may not be the higher-impact journal.
And, in fact, this is, again, a lot of the things that we always debate. You know, this is an example of how negative data gets published, but the impact factor of the journal that it gets published in is very different.
You take the same exact data. And, if it's positive, all of the sudden, this would be in a major journal. It's just the way it is. This is how the academic world works.
Q. The Bradford Hill criteria that you

applied, sir, did you go back and read his original paper?
A. Not the actual paper, actually. I read all of the criteria online. It wasn't the original paper that he -- the 1965 paper, but it was referenced in a lot of other publications I was able to get to.
Q. And you know that, in the original paper, he said that, before you apply the criteria, you should have your observations reveal an association between two variables perfectly clear-cut and beyond what we would care to attribute to the play of chance?
MR. LITZENBURG: Objection to the characterization.
A. So I think -- I'm not aware of that. That's the short answer. But I think it's criteria, it's guidelines. We've talked about this before. You can't take it in absolute terms.
All of these guidelines that we establish and that we actually bring out outside, they are not meant to eliminate or exclude your clinical judgment. At least I'm hoping not to.
Q. Is it your opinion, sir, that you have observed, in the epidemiological data, an

association perfectly clear-cut and beyond what you would care to attribute to the play of chance?
A. There is sufficient evidence that I reviewed that demonstrates an association and causality that are both not related to chance.
Q. So is the answer yes, you believe that Sir Bradford Hill's criteria were met?
A. I believe that the Bradford Hill criteria were -- were met.
Q. And I mean the criteria for starting to use the procedure, i.e., I have observed an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance?
A. So what do you mean by "perfectly clear-cut"? Like, what is that? That's such a vague term.
Q. What is it to you?
A. It means that there's zero doubt. And there is no such a thing as zero doubt in science, in epidemiology. I mean, when you say "clear-cut," it means that you're leaving zero room for the possibility of chance, and I think we all agree that this thing doesn't exist in science.
It's just impossible to demonstrate unless

you do this prospective, randomized trial that we all agreed on that it's unethical to do. So you look at the criteria, and you try to apply the information that you reviewed in the criteria. And there's enough evidence out there to suggest that this is the case.
But "clear-cut" means that there's -- you've got zero doubt. And, I mean, I don't think anybody can say that.
Q. You think that's what Bradford Hill meant, before you apply my criteria --
A. Well, you said "clear-cut." I asked you what clear-cut is. You punted the question to me, and I told you clear-cut, to me, means zero doubt. That's what it means to me. So now it's your turn.
What does it mean to you?
Q. Well, Sir Bradford Hill was setting out criteria to apply to a possible statistical association between two variables to assess whether they're causal or not?
A. Okay.
Q. He said clear-cut --
A. Okay.
Q. -- and beyond the play of chance.
And do you think that he meant 100 percent

proven before you even start doing his causality analysis?
MR. LITZENBURG: Object to form.
A. I hope not. I don't believe that's what he meant, because I think he would probably know better that there is no such a thing as clear-cut. So I don't believe this is what he meant.
I think what he meant is that there is enough evidence out there to prove the association and causality between two variables. I mean, "clear-cut," again, it's a vague term. To some people, it means 100 percent certainty; others, 90 percent; and others, 50.1 percent. So I don't know what he meant by this.
Q. And do you know that most epidemiologists consider it to be a statistically significant association in a reliable study?
MR. LITZENBURG: I object to that characterization.
A. The Bradford Hill?
MR. LITZENBURG: Object to form.
Q. Yes.
A. I know that they used the Bradford Hill criteria to the extent possible, but I also know it is not used in absolute terms. I mean, you can't --

again, you try -- you have to have certain -- certain criteria or certain guidelines in order to compare apples to apples, but I don't believe any epidemiologist is going to tell you that we use this exclusively and with 100 percent certainty.
Q. The next statistic that you quote in your expert report from the McDuffie paper is an odds ratio, which you called statistically significant at 2.12, 1.2 to 3.73 confidence interval.
And that comes from Table 8 of the McDuffie paper, sir. Would you take a look at that.
A. I see Table 8.
Q. When we were looking at the not statistically significant association on Table 2, you looked for me and saw that the odds ratio that was reported there had been adjusted for various statistically significant medical variables and with the variables of age and province of residence; correct?
A. Yes.
Q. And here they did not adjust for even the medical variables; right?
A. I'm not sure that's accurate. If they have adjusted on the other one, they have adjusted for

this one.
Q. In Table 2?
A. Yeah. I see what Table 2 you said.
Q. In Table 2, Odds Ratio A was adjusted for age and province of residence, and B was also adjusted for statistically significant medical variables; right?
A. Right.
Q. That was the meaning of Table B?
A. In Table 8, they adjusted to the variables age and province of residence, that's correct. And in Table 2, they've adjusted for additional -- we talked about this, I think -- yeah, measles, mumps, cancer, et cetera.
Q. And in Table 8, they only give out the Ratio A; right?
A. That's what it says, yes.
Q. So they didn't give B, adjusting for the medical variables?
A. They didn't -- well, they did not address -- even in Table 2, they did not look at all medical variables. All that they looked at specifically are, to be clear, measles, mumps, cancer, allergy desensitization shots, and a positive family history of cancer in first-degree

relatives.
Q. So there were other --
A. This --
Q. Sorry.
A. Right. I mean, this is what they looked at. So they did not look at tobacco, alcohol, hypertension, diabetes.
There are other -- when you say "medical variables," there is a presumption or you're implying that they looked at all medical factors. And they did not. They actually say exactly what they looked at.
In Table 8, they specifically looked at age and province of residence.
Q. Okay. So they didn't make the same adjustment --
A. No.
Q. -- even that they made -- even the partial adjustment that you just described that they made in Table 2, and in neither table did they adjust for exposure to other pesticides; correct?
A. Correct.
Q. So in McDuffie we have no statistically significant association adjusted for other pesticides; right?

A. For other pesticides, I did not see that.
Q. Now, the definition of -- the frequency of exposure definition here was the number of days per year that glyphosate was used; correct?
A. Yes. I think it's more versus less than two days or something like that.
Q. So if somebody used glyphosate twice a year for ten years, they would be in the low exposure group?
A. Say again. I'm sorry.
Q. In someone used glyphosate twice a year for ten years on two different days over the course of a year for ten years, they'd be in the low exposure group, and someone who used it on fifth -- on three consecutive days or three different days in the same calendar year would be in the high group, even though their total exposures would be flipped; right?
A. I have to write down what you're saying.
Q. Yes, sir. Twice a year for 10 years; 20 exposures.
A. Okay.
Q. That would be in the low group.
A. And you say on the low group based on what?
Q. Based on the definition of the low group,

days per year.
A. Do you mind telling me where you read that in that paper?
Q. Greater than zero and less than or equal to 2. It's in the days per year column on Table 8, among other places.
A. Oh, Table 8. I see. I'm reading in the methods.
So they say here, "Each subject will report ten hour per year or more of exposure to pesticides as defined by the screening questions, and a 15 percent random sample of the remainder was mailed a list of pesticides in an information letter." And then they did a phone interview with them after that.
And then the pathology, I think, had -- was since re-reviewed, which is a very -- which is a very strong thing about -- when you're able to do a pathologic review.
Okay. So Table 8.
Q. Table 8?
A. Uh-hum.
Q. Two groups, the low exposed group, greater than zero or less than or equal to 2?
A. I see that.

Q. And greater than 2; right?
A. I see that.
Q. So if you had -- and that is days per year; right?
A. Two days per year. That is correct.
Q. So two days per year for 10 years, that's 20 exposures.
A. Uh-huh.
Q. And someone else who has three days in the same year and no other exposures whatsoever, three total exposures would be in the high exposure group; right?
A. But if you have a three days per year for one year, that's three.
Q. Yes.
A. Yeah. So it would be --
Q. It would be in the high exposure group?
A. That is correct.
Q. Despite having three lifetime exposures as compared to someone in the low exposure group with 20 lifetime exposures?
A. Yes.
Q. Much more exposure; right?
A. Yeah.
Q. So people's exposure could be reversed in

the study and the statistics could be reversed; right?
MR. LITZENBURG: Object to form.
A. I mean, the authors of this paper, this is how they defined exposure. And, again, I mean, when you write these papers you'll have to -- you'll have to decide how you define exposure in order for you to make any sense of the data you are accumulating.
So they have chosen to look at more -- you know, anything that's less than two days as low exposure versus unexposed, anything more than two days per year as high exposure. This is the definition that they used.
I -- you know, again, please recall that this paper was gone through peer-review process. It's published, so I think if the reviewers had any issues with the actual definition and if they found that the definition is inaccurate or inappropriate, it would have been -- there would have been issues to get published.
So this is the definition of the authors. So we'll have to take that based on what they say.
Q. You agree it's a limitation of the study, potentially?
A. I think there is a limitation for any

study. As I've told you, I can find limitation in every single study. There is no perfect study.
Q. And the failure to control for other pesticides is also a limitation in this study?
A. It's one of the limitations, yes. It is literally impossible to control in everything in epidemiology study because you don't have a controlled environment for these patients.
Q. Do you know if it would have been possible to apply a statistical test to control for exposure to other pesticides in this study?
A. I think you'd have to -- you'll have to rely on what the cases and controls are remembering in terms of what additional pesticides they were exposed to and so forth.
Q. So their pesticide exposures were collected. That information was collected; right?
A. Yeah. I mean, they did say that here in the methods that they asked questions about other pesticides and so forth, but they -- for some reason, they were unable to control for it. This is not unusual that you're not able to control for it. I don't know why exactly they weren't able to control for it.
Q. Do you know if they were unable or if they

just didn't?
A. I am certain that one of the peers that reviewed the paper must have raised this issue and they probably got a convincing answer. I don't know why. Nor do I believe it's my role to understand why they didn't do it. I have to take the evidence as is.
Q. The Hardell study is the next one that you talked about in your expert report, sir.
(Nabhan Exhibit 12 marked for identification.)
A. Okay.
MR. LITZENBURG: You didn't mark that tweet, or did you? Is this 12 or 13?
MR. GRIFFIS: I didn't mark it.
Q. And this is another study like McDuffie where data was gathered for a large group of herbicides and pesticides and other chemicals at once; right?
A. Yeah. Well, this one they actually went back and they looked at two older studies. One was published by Nordstrom, and the other was published by -- by Hardell. I think '98 and '99. And one of them was related to hairy cell leukemia, which is a low-grade type of non-Hodgkin's lymphoma, and the

other was non-Hodgkin's lymphoma.
So they tried -- basically, they pulled the data of both studies together. And they wanted to see if we pull the data altogether at the same time, would be able -- would we be able to find a more statistically meaningful information.
So they're trying to increase the power of their analysis by increasing the number of patients analyzed.
Q. There were only eight people with non-Hodgkin's lymphoma exposed to glyphosate out of 404 total cases in these two studies; right?
A. Which table is that?
Q. Table 1.
A. I'm trying to see where the eight is. So you have glyphosate, four cases and three control. Is that what you're looking at?
Q. I'm looking at Table 1.
A. I am looking at Table 1 too. Do you want to direct me what to look at in Table 1?
MR. LITZENBURG: Are you representing this to be Hardell 2002?
THE WITNESS: This is 1998.
MR. LITZENBURG: Yeah. I mean, we are looking at the different one.

THE WITNESS: This is not the paper I'm referencing.
MR. LITZENBURG: Did you mean to give it to us?
THE WITNESS: This is the older paper that we --
MR. GRIFFIS: Can I see?
THE WITNESS: This is -- what I said this is the older one that they pulled --
MR. GRIFFIS: Yeah, you are right. I've got -- I've got the right one here.
THE WITNESS: Thank you.
MR. LITZENBURG: Thank you.
Q. Okay.
A. Yeah. This is the one, the 2002.
Q. Yes, sir.
So Table 1 in this one, then.
A. Okay. So Table 1, you have number of cases -- just tell me what to look at. So I mean --
Q. There were only eight people with non-Hodgkin's lymphoma exposed to glyphosate out of 404 total cases; right?
A. Oh, I see. The eight. Yes, I see that now.
Q. Okay.

A. Okay.
Q. And that's certainly a low number of cases for an epidemiology study on cancer; right?
MR. LITZENBURG: Object to form.
A. It's not a high number, but it's not a number that we would ignore, because then you have to look at population basis.
Q. And Hardell did a multi-varied analysis to adjust for confounders; right?
A. Yes, he did. You adjusted for age, county, study site, and vital status.
Q. Do you know what vital status is?
A. Death versus alive, I presume.
Q. And Table 7 shows the odds ratio calculated with multi-varied analysis with the correction for those confounding factors; right?
A. Yes.
Q. And the result given there is not statistically significant; right?
A. I think because the lower portion of the 95 percent confidence interval is below 1, if that's what you mean.
Q. Yes.
A. Then it's not statistically significant. But, as we discussed earlier, the odds ratio is --

in my opinion, is very important. You can't ignore it.
Q. Yes, sir.
You believe that odds ratios above 1 are important regardless of whether --
A. I think it's important --
Q. -- they are measured to be statistically significant; is that fair?
A. I would -- I would say I would not dismiss an odds ratio that's above 1 without understanding why, and without looking at additional evidence to know where things are going.
Q. There was no adjustment made for exposure to other pesticides; right?
A. Based on my review, I don't think they adjusted for other pesticides. And I think that's always a limitation because it is difficult to adjust for.
Q. And they said that exposure to different types of pesticides did correlate in this study; right? So it would be a confounding factor. That's on 1047, first column, three paragraphs down.
A. "In the multi-varied analysis exposure to herbicides, fungicides increased the risk, although odds ratio was lower than in the uni-varied

analysis."
So your question is?
Q. The results in multi-varied analysis must be interpreted with caution since exposure to different types of pesticides correlate. They found there was correlation between --
A. Yeah.
Q. -- different types of pesticides?
A. Of course.
Q. And, therefore, there would be confounding; right?
A. I think it's -- like I said, you always to want try to control for confounding factors if you can. And there are a variety of reasons why they could or can't: number of cases, the belief in the recall, et cetera. So it's not really clear why sometimes they're not able to.
Q. In McDuffie and Hardell, you don't know if the odds ratios would even be above 1.0 if controlled for other pesticides; right?
MR. LITZENBURG: Object to form.
A. I don't know that. It was not -- it was not done.
But you have to remember that sometimes when you control for additional confounding factors,

the odds ratios actually go down. So the fact that you have odds ratios that's above 1 without controlling is very important. And that's why we can't ignore it, because once you control to -- to -- again, the fact that you see -- you see certain things with control, without control doesn't take away from the evidence, in my opinion.
Q. Did you just say, sir, that a failure to control for a factor known to be confounding does not take away from the quality of the evidence?
A. Yeah. If you're -- because you can't always control. That's really the major issue. I think, as we said earlier this morning, if you are able, when you design the study, to control for all variables to the extent possible, you will always try to do that. But there are a variety of reasons why you can't do it.
I think everybody acknowledges that you would like to do it if you can. I don't know why some studies can, some studies can't. I believe a lot is related to the numbers that they have, where they don't believe they have enough numbers to control for all the variables included.
Q. Low numbers yield less useful numbers across the board; right?

A. It makes it more difficult to control. If you have higher numbers, it's much easier to control for variables; but when you have low numbers, you have very little to work with to control for variables.
Q. And your statistics are less well controlled as well; right?
A. It becomes more difficult to show statistical significance.
Q. And it's also more likely that statistical findings that you think you have found don't hold up; correct?
A. Well, you can't tell that unless you do the actual control. I mean, I think it is possible that they won't hold up, but it's possible they would.
Q. All I'm asking in general, sir, if you do a small study in just a few people, you're more likely to get false negatives and false positives and falseness in every direction.
A. Of course.
Q. Correct?
A. Of course.
Q. And the more cases and controls that you can find, the more reliable your data gets in every way; right?

A. The more numbers you have, you will always have better more robust data.
Q. Okay. We have looked at McDuffie and Hardell, and now I'm turning to DeRoos 2003, the next epidemiology study discussed in your expert report, sir.
(Nabhan Exhibit 13 marked for identification.)
Q. And just like the last study, Hardell, that we looked at, is actually pooling two smaller earlier studies. This also pooled three small earlier studies; right?
A. This -- let me just make sure I know -- this is the '03 paper?
Q. Yes, sir.
A. Okay. I was -- so it says March '08 up there, so I was confused.
Sure. Go ahead.
Q. Okay. So this pooled three earlier small studies; right?
A. Yes. From Nebraska, Iowa, Minnesota and Kansas.
Q. It pooled the Cantor study from Iowa and Minnesota, the Zahm study from Nebraska, and the Hoar study from Kansas; correct?

A. Yes.
Q. And, again, these were studies that were looking at multiple pesticides and herbicides simultaneously; right?
A. It did.
Q. So like the others, you'd expect some false positives; right?
A. It's possible.
Q. The Cantor -- we just looked at a study -- the Hardell study with eight cases in it. The Cantor study had 26 cases, sir.
A. The Cantor study was mainly for farmers in farming population. I don't think this specifically looked at glyphosate.
Q. And -- right. And Dr. Neugut testified that it had low power, the Cantor study had low power, because there were only 26 cases of non-Hodgkin's lymphoma with exposure to glyphosate.
Do you agree that that many cases with exposure to -- with exposure is a low-powered study?
A. I think you have to look at the denominator, 26 out of how many, to accurately see how powerful the study was.
Q. Okay. Do you want to see the Cantor study?
A. Sure.

(Nabhan Exhibit 14 marked for identification.)
Q. Did you look at these individual substudies, sir?
A. Yes, I did. But there's a lot of information in each one. Difficult to remember everything.
This is a '92 paper. Yeah.
So they had 195 patients with follicular lymphoma, 198 with diffused, and 85 of small lymphocytic, and 144 of other. So this is the number of lymphoma cases that they had. They had 622 cases and 1,245 controls.
Q. And Table 6, sir, you can see how many were exposed to glyphosate, and the answer is 26; right?
A. Yes. But I think it's important -- that's what I meant by the denominator. I think it's a very respectable number, 622 cases and 1,245 controlled. That's the denominator, which is very important.
And then you look at Table 6, as you said. And in glyphosate, the number of cases were 26 versus 49. So I -- you know, I think that's -- you know, 26 out of 622. I mean, the total number of exposure is 26 plus 49.

Q. Well, do you agree or disagree with Dr. Neugut that the Cantor study has low power because of the numbers of people exposed to glyphosate?
A. I don't agree.
MR. LITZENBURG: Object to that characterization.
A. I don't think you can use the absolute numbers by themselves as the sole determination of a low versus high power. Many times you actually decide on the power of the study before you even embark on the study, not after the fact.
Q. In the DeRoos paper, sir, DeRoos 2003 --
A. Okay.
Q. -- he gives results for a logistic and a hierarchical regression analysis; right?
A. Which -- which table are you looking at?
Q. I'm actually looking at the statistical analyses section on page 2.
A. Okay. Sure.
Q. In that -- in the middle of the first paragraph under "Statistical analyses" on page 2 of the DeRoos 2003 paper, he said, "We employed two approaches to our analyses, standard logistical regression and hierarchical regression, calculating

odds ratios to estimate the relevant risk associated with each pesticide." Right?
A. I'm not familiar with all the statistical methodology. I'm not a statistician. You know, I think that is a very -- that's delving into the statistical detail, which I'm not really qualified to answer.
Q. Okay. Well, you see that that's --
A. I see what you're saying. I do see it.
Q. I'll try not to get too technical about it.
In the hierarchical regression of multiple pesticide exposures, the next paragraph, they say that in the hierarchical regression analysis, they regressed NHL disease status on the 46 pesticides exposure.
So they did some controlling for pesticide exposures in the hierarchical, not the logistic, regression analysis; right?
A. I really think you're delving into so much detail, that I'm struggling here to follow you. I -- whatever -- I mean, they've done a lot of statistical analysis, I guess. That's all I can say.
Q. Can you tell if they controlled for pesticide exposure in the logistic regression?

A. If they controlled for other pesticide exposures?
Q. Yes.
A. So I have in my notes here that they did control for confounders. So, obviously, I've looked and saw that they controlled for confounders. And I'll have to see if they -- I believe they actually tried to control for other pesticides.
Q. In the hierarchical section; right?
A. Because there were 47 pesticides out of -- that's what I wrote here in my notes, but I don't know the methodology of how they controlled. Maybe this is statistical way of controlling. My notes suggest that they have -- they did control for other pesticides.
Q. Yes, sir.
In the hierarchal regression they did; right?
A. Okay. I guess in the hierarchal regression.
Q. Okay. And the odds ratio that you reported in your expert report comes from the logistic regression on Table 3; true?
A. 2.1. Let me check. 2.1, that is from the logistic regression, that's correct.

Q. And the odds ratio reported from the hierarchical regression 1.6, confidence interval 0.9 to 2.8 is not statistically significant; correct?
A. The hierarchical regression is 1.6, and the other one is 2.1. That's correct.
Q. And the hierarchical regression is not statistically significant; correct?
A. Yes. I just don't know whether that is really -- again, you know, the controlling for pesticides, does it really matter if it's logistical regression versus hierarchical regression? I can't really answer that.
Q. Well, that's the one that is controlled for other pesticides --
A. Well, you control --
Q. -- isn't it?
A. You also -- when you do a logistic regression, you actually do control for other factors, including pesticides. So I'm not really sure whether they didn't -- you know, whether one negates the other. That's what I'm trying to say.
Q. Okay. Do you know of anywhere where they reported that, in the logistic regression, they controlled for other pesticides?
A. I'll have to read the whole paper again. I

don't think we have time for that. I'm trying to read the statistical --
Q. Go ahead and read it. We can take a -- we can --
A. Okay.
Q. We can pause while you do that.
(Pause.)
A. Okay. I read that.
Q. Okay. And did you find anywhere where they say that other pesticides were controlled in the logistic regression as opposed to the hierarchical?
A. I did not. But I saw just a couple things, and I'll just mention them. So they do mention here that they -- "We employed two approaches to our analysis, standard logistic regression -- maximum likelihood estimation and hierarchical regression -- calculating odds ratio to estimate the relative risk associated with each pesticide."
Then they go on to say, "All models included variables for age and indicator variables for study site, other factors known or suspected to be associated with NHL including first-degree relative with hematopoietic cancer. Education and smoking were evaluated and found not to be important confounders of the association between NHL and

pesticides," for whatever it's worth.
Q. The next study you mention is the Lee study, sir.
(Nabhan Exhibit 15 marked for identification.)
Q. This actually used data from Cantor, which we've already discussed, and one other U.S. case-control study; is that right?
A. Yes.
Q. And the odds ratios reported here were not adjusted for exposure to other pesticides; true?
A. Repeat again, please.
Q. The odds ratios reported were not adjusted for exposure to other pesticides; right?
A. No, it was not. It was adjusted for age, vital status, and state.
Q. The hypothesis under investigation was whether asthma modifies the risk of NHL associated with pesticide exposures; correct?
A. Correct.
Q. And in people exposed to glyphosate, there was no statistical significant association either in people with or without asthma; correct?
A. That is correct. The odds ratio was above 1 for both, but it was not -- it did cross the 1.

Q. It did cross the 1.

And do you know of any sort of analysis that has been done to compare the 1.2 to the 1.4 to see if there is a statistically significant difference between people with and without asthma?

A. I'm not aware of that. I don't know.

Q. Okay. You have no conclusion about whether asthma increases or decreases or has no effect on any risk that you believe exists of non-Hodgkin's lymphoma from glyphosate; is that fair?

A. Yeah, I -- I don't have any additional conclusions beyond what the authors have concluded. And the authors' conclusion suggests that -- and I quote -- "Our results suggest that the risk of NHL among asthmatics with pesticide exposure may be higher than among non-asthmatics with pesticide exposure."

I have no additional conclusions beyond what you just stated.

Q. And you don't know if that was specific to glyphosate; right?

A. They talked about pesticide exposure in general.

Q. Certainly, the point estimate for people with asthma was lower than the point estimate for

people without asthma for glyphosate-exposed people; right?

A. Correct.

MR. GRIFFIS: I need to tidy up my pile here. Let's take five minutes.

VIDEOGRAPHER: Going off the record at 2:00 P.M.

(Recess taken from 2:00 P.M. to 2:15 P.M.)

(Nabhan Exhibit 16 marked for identification.)

VIDEOGRAPHER: We are back on the record at 2:15 P.M.

BY MR. GRIFFIS:

Q. Exhibit 16 is the DeRoos 2005 article, which is the next one discussed in your expert report; correct?

A. Correct.

Q. Now, this is a prospective cohort study; correct?

A. Correct.

Q. All the other ones we've been looking at are case-control studies; right?

A. Yes.

Q. Would you explain the difference to the

jury.

A. So, I mean, case control is -- you know, in broad term is more of a retrospective study where you are looking at individuals who are diagnosed with the disease and those who are not diagnosed with the disease. And you retrospectively attempt to analyze exposure or contributing factors that might have led to the development of the particular disease.

The cohort study is more of a prospective evaluation of particular individuals, and you follow them prospectively. So you are presuming that, at the time of initiating the particular study, nobody has the particular disease per se. And you follow them for whatever period you decide to follow them, and you assess who developed the disease and why and what. And you make an analysis.

Q. So in the case-control studies that we've looked at so far -- like DeRoos 2003, Cantor, Hardell, McDuffie, et cetera -- the authors started out with a group of people with non-Hodgkin's lymphoma, and then they asked questions of those people and some others who they found without non-Hodgkin's lymphoma to be controls and compared what they said about their past

exposures to all sorts of different pesticides in all of those studies and then ran some statistics on them; is that fair?

A. Fair.

Q. Okay. And in this study and in a prospective cohort study, what they did instead was gather a bunch of people -- and these were what kind of people?

A. These were mainly folks that were licensed to apply restricted-use pesticides.

Q. So these were licensed pesticide applicators? People who would be exposed to pesticides; right?

A. But they're licensed, so they're -- usually, they -- they know what they're doing. They had to have, like, a particular exam criteria to enter the study and so forth. So they --

Q. Okay.

A. They had -- you know, they had more knowledge of what they are up against, if you will.

Q. And these are people who did not have non-Hodgkin's lymphoma. And they filled out questionnaires about their exposure to pesticides, which were renewed at various times.

And then the authors of this study followed

them going forward as the years moved on to see if they developed non-Hodgkin's lymphoma; correct?

A. Yes. To my recollection, the folks that were enrolled were from '93 to '97. And this particular paper reported on the outcome as of December 2001. So the follow-up was 6.7 years, median follow-up.

Q. And one of the things that a cohort study -- one of the advantages of a cohort study over a case-control study is that a cohort study avoids recall bias; is that correct?

A. It avoids the recall bias, but it has its other limitations.

Q. Recall bias is the bias that's caused by people who have come down with cancer being more likely to ruminate, to think about all of the exposures that they might have had and possibly even to exaggerate those exposures and to be a lot more likely to write down in a questionnaire, oh, yes, I was exposed to this and this and this, than someone who doesn't have cancer and is going about their regular life; correct?

A. I agree with everything you said except for the word "exaggerate." I think, in recall bias, it's I inherent that, you know, individuals who have

been diagnosed with a particular cancer, they -- they usually, you know, try to remember more. They try to look more into their past. They ask their friends. They ask their family and so forth. Because now you are diagnosed.

I don't know if they would exaggerate. I think they would probably just investigate more their history versus somebody who doesn't have cancer so they're less likely to do a robust or rigorous investigation.

Q. So in a case-control study, the people with cancer, the people in the case group, are more likely to report their past pesticide exposures than the people in the controls.

That's fair; right?

A. I think the recall bias is for both sides. I would agree with you that, in general, we do see that the recall bias could affect individuals who were diagnosed with cancer more. But, you know, you could make the same argument for recall bias for the controls as well, that they may actually forget the fact that they were exposed to something because they're not as diligent, because they were -- they were -- they don't -- they didn't get the diagnosis of cancer.

So I think the recall bias exists for both. But, you know, I tend to agree that it's probably going to be more in folks who are having cancer, just by human nature.

Q. Okay. So there's two kinds of recall bias. There is the recall bias of the people -- the cases, the people with cancer --

A. Right.

Q. -- who are reporting it more thoroughly than average?

And then there's the careless -- the relative carelessness of the controls who are just getting a questionnaire in the mail and don't have much of a personal stake in it who would be more likely to forget about things and miss and underreport their exposure?

A. I agree with that.

Q. Okay. And both of those would tend to bias the results towards an association, towards a finding --

A. Or the lack thereof.

Q. -- that a substance causes a particular outcome; right?

A. Or the lack thereof. I mean, I think it would bias the conclusion by -- by its inherent

limitations. I just don't know whether it would bias it to the positive association or a negative association.

Q. The AHS -- this is the Agricultural Health Study; right?

A. Yes.

Q. And the Agricultural Health Study is not a single study.

It's not the DeRoos 2005 paper; it's a larger research project. Correct?

A. Correct. But, to my knowledge, this is the only publication that came out of it, unless I missed something. But you are right; it is a continuous -- I mean, it's all on the website. You can -- you can -- it has its own website and own information, which I -- I gathered the data from. But, to my knowledge, this is the only paper that I found from the AHS.

Q. About glyphosate?

A. About glyphosate, yeah.

Q. There may be other papers from the AHS about other things; right?

A. Yeah, I didn't look at that.

Q. Okay. And it's funded by the U.S. government; right?

A. I honestly don't know who's funding it. It's probably the NIH, which is U.S. government but -- I believe it's the -- NIH, yeah.
Q. There were 92 individuals here with exposure to glyphosate who had non-Hodgkin's lymphoma; right?
A. 92, correct.
Q. And, again, these were people -- there was a very large body of people who were being tracked, and 92 of the ones who developed non-Hodgkin's lymphoma had an exposure to glyphosate; is that right?
A. Yeah. I mean, there were -- as you can see in Table 2, there are other cancers, but the NHL specifically was 92.
Q. They started out with 57 -- more than 57,000 private and commercial pesticide applicators; right?
A. Yes. There was 57,311.
Q. And they paid attention to all of the cancers that these people developed, although they especially looked at non-Hodgkin's lymphoma because there had been previous studies done like the ones we've been talking about; correct?
A. Yes.

Q. And they reported -- here, I'm on page 51, sir, the end of the long paragraph at the top of the third column.
They reported no association was observed between NHL and glyphosate exposure in any analysis, including an analysis comparing the highest with the lowest quintile of exposure, more than 108 versus 0 to 9 cumulative exposure days; correct?
A. That's what's written here.
Q. Okay. Now, when we were looking earlier at a -- at an exposure-days-per-year estimate, we were looking at 0 to 2 versus greater than 2; right?
A. Well, that was in one paper, though.
Q. Yes.
A. Yeah, I mean, that was one paper, I think McDuffie, that they looked at 0 to 2 versus over 2, yes.
Q. And this was looking at a much greater range of days; right?
A. I don't know if this one looks at days per se. I think they -- they had their different definition. If you -- when you read the -- page 50, the first column, they constructed three glyphosate exposure metrics, ever personally mixed or applied, cumulative lifetime, et cetera, et cetera.

So they have lowest exposed, higher exposed, never exposed. And so they just use a different way of deciding exposed versus nonexposed.
Q. And the one described here, the highest versus the lowest quintile of exposure, was more than 108 cumulative exposure days versus the lowest, 0 to 9, cumulative exposure days; right?
A. I see that, yes.
Q. And was that a relative risk point estimate of less than 1; right?
A. I see that, yes.
Q. In Table 2, the ever/never used, the relative risk point estimate adjusted for age, demographic, and lifestyle factors in other pesticides was 1.1 with a confidence interval of 0.7 to 1.9, which is not statistically significant; correct?
A. Correct.
Q. On Table 3, they looked at cumulative exposure days and intensity-weighted exposure days; correct?
A. That is correct.
Q. Now, cumulative exposure days is looking at how many days people were exposed for, and intensity-weighted exposure days is adjusting those

days further for how much exposure there was on the days of exposure; right?
A. Yes.
Q. So if you were just using it a little bit, that would be a lower intensity day; and if you were using it a lot, that would be a higher intensity day; right?
A. So the intensity-weighted cumulative exposure is a formula. It's years of use multiplied days per year multiplied by intensity level. And they're categorized in tertiles.
So I think that, if you use it for so many years, that will increase it. If you use it for so many days in a particular year, will increase it and the intensity will increase. So it's three factors that could actually bring the number up.
That's on page 50, the second paragraph.
Q. In both groups, the cumulative exposure days and the intensity-weighted exposure days, the point --
Well, the point estimate was set to 1.0 for the lowest exposure group, and then the next two levels of exposure were compared to that; correct?
A. I don't know if they were compared relatively or taken by themselves in absolute.

Q. Is there any --
A. In --
Q. In each case, the relative risk given for the lowest tertile was set to be 1.0.
You can see that all the way down the column; right?
A. I see that, 1.0.
Q. And then we can see whether there is any dose effect by seeing if that odds ratio goes up at the median and high tercile exposure levels; correct?
A. I see that, yes.
Q. For non-Hodgkin's lymphoma, the risk goes down at the median and high exposure group, both for cumulative exposure days and intensity-weighted exposures days compared to the lowest tercile; right?
A. That's what it says, but what does that mean?
Q. In these data, sir, when people were more exposed to glyphosate, their risk of non-Hodgkin's lymphoma went down below 1.0, although it was not statistically significant on any of these measures; correct?
A. Sorry. Are you suggesting glyphosate is a

preventive measure against non-Hodgkin lymphoma?
Q. You keep telling me that it's real important when it's above 1.
What does it mean to you when it's below 1?
A. I can be -- again, I said you can't take everything in just absolutes. So you can't have a -- you know, to suggest that, just because it's below 1, it's going to be a protective effect, then we should just all go outside and spray ourselves with glyphosate. Just -- just -- you can't -- I mean, it's not protective obviously.
Q. There's no way that you can use the figures in Table 3 to support a hypothesis that glyphosate causes non-Hodgkin's lymphoma; correct?
A. No, based on the data in Table 3, I cannot say that. You're correct.
Q. 92 individuals with exposure to glyphosate and non-Hodgkin's lymphoma, which is the number in the DeRoos 2005 study, is the most people with glyphosate exposure and non-Hodgkin's lymphoma of any published epidemiology study; correct?
A. It is the most number in the studies I reviewed. I don't know if that encompasses every single paper in literature.
Q. Okay. Of the ones that you know about,

sir --
A. Yes.
Q. -- this is the one with the highest power; right?
A. No. This is the highest number. That's different than power.
Q. Okay. Tell me --
A. Power, now, is statistics.
Q. Do you think that a different study that you reviewed has more power than this one?
A. I didn't look at the power of each study. I think you're correct by saying that this has the highest number of patients with non-Hodgkin's lymphoma, 92 cases. That's correct?
But when you say "highest power," then you'll have to compare the trials from a statistical standpoint, each one. And I did not perform that, nor am I qualified to compare statistical power between -- across studies, and I wouldn't recommend comparing different studies from a statistical standpoint. It's not a very good exercise to do from an academic standpoint.
Q. From an academic standpoint, it's not a good exercise to compare the power of different epidemiology studies?

A. To compare across studies, it's not something that we normally would like to do because each study has its own. So you're going to -- I mean, to compare across -- cross-trial comparisons are not something that we normally would like to do.
Q. And how does that comment apply to the field of meta-analysis, sir?
A. I'm not sure I understand the question.
Q. Well, a meta-analysis -- meta -- people who are performing a meta-analysis --
A. Well, there are methodologies for meta-analysis. If you're conducting a meta-analysis, you follow a methodology to make sure that you look at the trials. I mean, there is -- these are oftentimes scientists and statisticians that are equipped to perform a meta-analysis using robust criteria and looking at all of the data that's available.
You're not necessarily comparing the statistical power of each particular study against each other. You're trying to look at all of the studies combined and see, are you seeing any causation? Are you seeing any association when you look at the entire body of the literature?
Q. Do you know whether people who are

performing a meta-analysis, as part of the procedure that they follow, assess the power of each of the studies that they're looking at and weigh the meta-analysis in terms of the power of those studies?

A. I don't know if that's what they do.

Q. All right. Do you know of any other study besides the DeRoos 2005 study, of the ones that you looked at, sir, that measured the intensity of exposure?

A. And when you mean -- by "intensity," you mean the dose of the compound? Because the intensity could be just the years of exposure by the number of years. Or are you talking specifically in terms of the dose?

Q. I'm talking about intensity-weighted exposure, like in the second column of Table 3.

A. Yeah, I'm not aware of other studies that look -- that added the -- you know, again, you can see the -- you know, the actual number of years by the number of days per year.

But what they did here is they added another attempt by adding the actual intensity level, which is always commendable thing to do. It has its own limitations because always difficult to

be very accurate with it. But I'm not aware of other studies that did the same thing.

Q. Do you know of any other study with dose data like this?

A. Like exactly this one?

Q. Dose data at all, sir. It's important when you're looking at --

A. Early on, we -- you know, you showed me the -- and we talked about the Bolognesi paper, I mean, in terms of aerial spray and in some areas more, some areas less.

Is this a dose data? I don't know.

Q. I was talking about epidemiology, sir.

But in the Bolognesi paper that you mentioned, which was a genotoxicity study --

A. Right.

Q. -- not epidemiology --

A. I know.

Q. -- for non-Hodgkin's lymphoma, there was not a dose relationship; correct?

A. Well, I think we -- we are going to disagree on the interpretation the Bolognesi paper, but I just -- I'm trying to understand your question in terms of the dose per se. If you're asking about the intensity-weighted exposure days as it's defined

in this paper, I'm not aware of other papers that did the same formula. That's the short answer to your question based on the papers I reviewed.

Q. In the area of epidemiology --

A. Right.

Q. -- I'm talking about your section headed "Epidemiology" in your expert report --

A. Yes.

Q. -- is there any other paper where you purport to see or not -- or not see a dose response?

A. Let me check a couple of things --

Q. Yes, sir.

A. -- to be more accurate in answering that.

So I think there is a paper by Eriksson that I reviewed from 2008 that talked about more than ten days, less than ten days in terms of different odds ratio. So I don't know if you would consider that. Again, this is the number of days of exposure, and they used the cutoff of less than ten days or more than ten days.

But I think the DeRoos '05 paper, they specifically added the intensity multiplied by the number of years multiplied by the number of days per year. I have not seen that particular formula in other papers.

But the Eriksson paper, you know, you could consider this a form of dose response because they used ten days, less than ten days. The McDuffie paper that we actually reviewed more than ten days, less than -- more than two days, less than two days, you could easily say this was a dose response because, again, you have a couple of days less or more.

So each paper and each manuscript has its own definition of how they define dose intensity.

Q. Unlike the ones you mentioned, this one controlled for other pesticides; right?

A. I just want to make sure. I believe they tried to control for pesticides. You will have to remember that the actual controls here, that they're never exposed and so forth, were all licensed applicators that were using pesticides. So I think you are starting from such a high bar to be able to demonstrate statistical significance over the control group.

So, again, you look at the patient population or the individual population that's going in, and everybody in the AHS was actually a licensed applicator. So in order to demonstrate statistical significant above and beyond, it's way more

difficult than when we take controls that don't have any of these occupational exposures.

Q. I'm sorry. Were you done answering?

A. Yes, I was.

Q. Okay. The next paper that you mention is the Fritschi paper; right?

A. Yes, I mentioned that.

(Nabhan Exhibit 17 marked for identification.)

Q. This mentions as possible -- in the first paragraph, sir -- possible causes of increased risks non-Hodgkin's lymphoma among farmers, exposure to diesel exhaust and animal viruses.

Do you see that?

A. I see that, yes.

Q. Do you have an opinion as to whether those are risk factors for non-Hodgkin's lymphoma?

A. I don't have an opinion.

Q. Fritschi is an Australian study?

A. Yes.

Q. And the exposure was assessed by -- the exposure was established by an occupational hygienist who reviewed occupational histories and determined what they felt that the exposure of the individuals in the study would have been to various

herbicides and pesticides; right?

A. The hygienist and the interviewers were blinded to the case or control status of the subjects. So, yes, there were interviews, but there was blinding of the interviewers.

Q. And the interviews were about the occupations that people had worked in; correct?

A. Well, again, there are probably more than just the occupation. It looked at other factors as well.

Q. And specific tasks?

A. Right.

Q. But the exposure to particular substances was assigned by an occupational hygienist based on people's answers about their careers rather than people saying what substances they had been exposed to; right?

A. Well, the hygienist was blinded to all of these. I mean, the same expert occupational hygienist, again blind to the status, reviewed the occupational histories and the answers to the modular questions and determined exposure to various substance, including argon and phosphates, et cetera. So it wasn't a priority that the hygienist knew what was happening.

Q. Oh, sure.

A. They were blinded.

Q. But they didn't ask people, "Have you ever been exposed to glyphosate?" A hygienist assigned people based on what they said their occupational history was. They declared that somebody had or hadn't been exposed to glyphosate and at what level, and the same for all sorts of other pesticides and herbicides; right?

A. You know, I don't think they put the actual questionnaire. I'm trying to read here. It says here, "Case in controls and were mailed an introductory letter, an information leaflet, followed by self-administered questionnaire to each consenting subject. The questionnaire included a diary with a detailed lifetime history of each job the subject had held for one year or more. Information obtained on each job included job title, employer, industry, start and finish years, number of hours worked per day, and number of days worked per week."

And they looked at the cases in the controlled and -- and -- so I don't know if the assignment was before or after, if that's your question. Your question is was the assignment after

the answers were available? Is that your question?

Q. No, sir.

It is that when someone was said to be -- have been exposed to a particular substance in this study, that wasn't based on them saying that they had been exposed to that substance; it was based on an occupational hygienist proclaiming that they had been based on the jobs that they said that they'd performed in the past?

A. Well, based on their answers. Based on their answers.

Q. But not their answers about pesticide exposure.

A. I see. You're saying -- I see. Based on --

Q. Someone said --

A. -- the answers --

Q. Someone said, I worked -- I was an alfalfa farmer for two years, and then I herded kangaroos for six. And the occupational therapist, therefore, assigned --

A. You're correct.

Q. -- putative pesticide exposure?

A. Now I understand your question.

Q. Okay.

A. Yes, you're correct.
Q. And you don't know how reliable that methodology is; fair?
A. It's fair to say, yes.
Q. In your expert report, you gave an odds ratio of 3.29; correct?
A. Correct.
Q. And that was for, as you said, for all -- for other herbicides?
A. For all herbicides, collectively, yes.
Q. You don't know if glyphosate is included in the figure that you gave; correct?
A. It's not called out specifically in this trial. They don't recall call out glyphosate specifically. They include all herbicides.
Q. You don't know what glyphosate's contribution was, if anything; right?
A. I do not know that, yeah. But glyphosate is an herbicide, so I would presume it was part of the -- it was included.
Q. And you don't know if it pulled the odds ratio up or down or had no effect on it; right?
A. I can't tell.
Q. So what effect did this study have your opinion?

A. It just solidified that you see that with herbicides, and glyphosate is an herbicide. So, again, it's just another demonstration that herbicides, as a class, could have an increased risk of causing and contributing to non-Hodgkin's lymphoma.
Q. Is it your opinion, to a reasonable degree of medical certainty, that herbicides, as a class, cause or contribute to non-Hodgkin's lymphoma?
MR. LITZENBURG: Object to form.
A. It's -- it's tough to really tell. But you have data on farmers that use herbicides. You have a trial like this that has herbicides. So it's -- it's hard to actually lump all of them in just one basket. I believe that there is some increased risk with herbicides, and having glyphosate as one of them solidifies my opinion in terms of the causation between this compound and non-Hodgkin's lymphoma.
Q. Okay. So are you going to be testifying, do you have the opinion, that there is a class effect?
A. I would say there is enough evidence to suggest. Again, there's -- there would be no absolutes given what we've talked about several times. I mean, this -- this -- obviously, this

particular trial shows that herbicides have an increased odds ratio of developing non-Hodgkin's lymphoma. It didn't call out glyphosate. The Cantor study talks about farmers. And, again, farmers use herbicides.
So when you say a class effect, then your implying that every single herbicide will have this causation, and I don't think we can safely say that. I'm not prepared to say that because I can't say every single herbicide will have that.
Q. Do you have the opinion that there is any herbicide that does not cause or contribute to non-Hodgkin's lymphoma?
A. I don't have an opinion on that.
Q. Do you have an opinion that there is an herbicide that is safer with regard to non-Hodgkin's lymphoma than glyphosate?
A. I also don't have opinion on that. I did not review that.
(Nabhan Exhibit 18 marked for identification.)
Q. Marking as Exhibit 18 the Eriksson study, sir. And this is another exploratory study that wasn't designed to specifically test the hypothesis of an exposure between glyphosate and non-Hodgkin's

lymphoma, but to screen multiple herbicides and pesticides at the same time; right?
A. Correct. It did look at glyphosate, though, as you can see --
Q. Yes, sir.
A. Okay.
Q. They have results for multiple individual pesticides.
A. Sure. Okay.
Q. Now, the only adjusted odds ratio reported in this study that's controlled for confounding by other pesticides is in Table 7 on page 1661 of the study; right? The multi-varied analysis?
A. Table -- Table 7, they talked about adjustment for age, sex, and year of diagnosis or enrollment. I think on page -- on Table 2, they also have some adjustments was made for age, sex, and year of diagnosis -- yeah, these are the three things they adjusted for.
Q. They are not adjusted for medical conditions or family history of cancer or smoking or drinking or any of the lifestyle factors that you've discussed earlier; right?
A. No, they were not.
MR. GRIFFIS: Let's take five minutes.

VIDEOGRAPHER: Going off the record at 2:50 P.M.
(Recess taken from 2:50 P.M. to 3:01 P.M.)
VIDEOGRAPHER: And we are back on the record at 3:01 P.M.
BY MR. GRIFFIS:
Q. Okay. Sir, the Table 7, "Multi-varied analysis for glyphosate," the most adjusted odds ratio for confounders set forth in this study is not statistically significant; correct?
A. The odds ratio is 1.51.
Q. And it is not statistically significant; correct?
A. Correct.
Q. When you look at the unadjusted odds ratios for all the substances in this study, you see that virtually every single one of them is above 1.0; right?
A. Which table you are looking at? The same table?
Q. Let's look at Table 2 first, "Exposure to various herbicides."
A. Okay.
Q. Herbicides total. That's all greater than

1.
A. Right.
Q. Phenoxyacetic acids, all greater than 1. Subgroup of MCPA all greater than 1. 2,4,5-T and/or 2,4-D, all greater than 1. Other greater than 1. Herbicides except phenoxyacetic acids, all greater than 1. Glyphosate, greater than 1. Other herbicides, all greater than 1 except for when you get to greater than 32 days.
Do you see that?
A. I do.
Q. Okay. Now let's turn to Table 4, "Exposure to various other pesticides."
Without repeating every single thing in here, we have insecticides, DDT, mercurial seed dressing, pyrethrin, permethrin, other insecticides, fungicides, impregnating agents, chlorophenols, arsenic, creosote, tar, other impregnating agents and rodenticides.
And virtually every single one is greater than 1; correct?
A. Some of them are not.
Q. Yeah, some are not. The vast majority are greater; right?
A. Yes. As long as we acknowledge some are

not.
Q. One, two, three, four are not; right?
A. One, two, three, four. Yes.
Q. And that's out of 34 stated odds ratios; right?
A. Yes.
Q. Just in that table?
A. The -- that's actually not -- I mean, that's not -- 34 odds ratio, but not 34 compounds.
Q. Yes, sir.
A. Okay.
Q. 34 of the odds ratios.
A. Okay.
Q. For each of the ones that was not above 1, another one of the measurements for a different period of time for that same substance was greater than 1; right? So every substance was found to be greater than 1?
A. Do you mind repeating the question?
Q. Yes, sir.
Where there is an odds ratio in Table 4 below 1 --
A. Uh-hum.
Q. -- for a particular substance, if you look immediately above or below it, you will find that

same substance for a different time period with an odds ratio of above 1; right?
A. I see what you're saying. Yes, I do see that.
Q. So every substance in Table 2, various herbicides, and 4, pesticides, is greater than 1; right?
A. For the most part, yes.
Q. Now, when you have a study that is reporting unadjusted odds ratios for every substance that it looks at above 1, there is some suggestion that there is confounding in the results; right?
A. I'm trying to understand your question. So you're -- you're -- I mean --
Q. Do you believe that every one of these substances causes NHL?
A. I don't think we know the answer to that.
Q. Okay. Do you think this is evidence that every one of these substances causes NHL?
MR. LITZENBURG: Object to form.
A. I think the -- the -- you know, this is one paper, one suggestion that there are other substances that might be implicated in causation or predisposition to developing NHL. So I will need to do a formal investigation, let's say, on arsenic by

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itself and spend a lot of time to understand if there is any additional as to arsenic by itself, because I'm not going to take just one paper and make a bald conclusion that all of these substances have causation to NHL.

Q. Well, far as this paper goes, as far as the Eriksson paper goes, to the extent that you're using it --

A. It's hypothesis-generating.

Q. -- as evidence of glyphosate causing NHL, it's also just as good evidence for all these other substances causing NHL?

A. But this is not the only paper I use for glyphosate. It's not the only one I cited. So that's really not correct. I've actually cited a lot of other papers. So I use this paper in conjunction with other evidence.

Q. Let me ask the question again.

A. Please.

Q. With regard to this paper --

A. Yes.

Q. -- to the extent that you use this paper as evidence that glyphosate causes NHL, this paper is just as good evidence that each of these other substances causes NHL; right?

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MR. LITZENBURG: Object to form.

A. Again, I'm going to try to reemphasize that this is one paper, so I -- I will agree to one thing, that, if you take just this one paper, the evidence appears to be that for glyphosate. There are other substances as well. But you can't form a conclusion on causation between a compound and a disease based on one paper.

So, I mean, this is all I can say regarding this question.

Q. Yes, sir.

To the extent that this paper is a piece of the puzzle that you have put together to form an opinion that glyphosate causes NHL, it would be just as good a puzzle piece for every one of these other compounds?

A. If the other compounds have as much evidence regarding causation with NHL, as the evidence that I've seen for glyphosate, then the answer would be correct. But if I try to pick whichever compound and I find nothing after that, then it would be just simply hypothesis-generating, and it wouldn't mean much of it.

So, I mean, I think you're partly correct that there is something here, but needs to be

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investigated further.

Q. And when you do a study of multiple compounds and you're finding positive association after positive association -- and by positive, all I mean is greater than 1 because most of these are not statistically significant.

A. I understand.

Q. I'm using kind of your definition of --

A. Sure.

Q. -- positive from earlier today. It suggests that you need to control the exposures that you're most interested in for all of those other compounds so that you can find out whether you have a real effect or one that's confounded; correct?

A. See, it's not necessarily true, because the issue of controlling for these confounding factors is really for both cohorts, for the cases and the controls. I mean, you have two types of population. You have the population of those folks who developed non-Hodgkin's lymphoma and the population that did not develop non-Hodgkin's lymphoma.

So, you know, you're trying to apply the confounding factor in terms of controlling only to the one folks who have non-Hodgkin's lymphoma. The reality is you will control for these factors in

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both populations, in the cases and the controls. So you can make an argument, a valid argument, and it becomes a washout because you are going to control for these confounding factors for both of them.

Q. That's what you do when you control for confounding; you control for both. Right?

A. Right. That's what I'm saying.

Q. And what we've seen, in study after study today, is that when you control for other pesticide exposures, results that were statistically significant become not statistically significant for glyphosate; correct?

A. No. I think what we are -- what we -- what I can say is that there are studies that we reviewed together earlier today that did not control for other pesticide exposure. What I'm going to argue is I don't know whether controlling for these pesticide exposures would have changed the odds ratio positively or negatively. The test was not done to -- to -- so speculating that just by controlling, the odds ratio will disappear is a complete speculation. As a scientist, I can't agree to that.

Q. Tell me of a statistically significant association between glyphosate and non-Hodgkin's

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lymphoma in the epidemiology you looked at that is controlled for other pesticides.

A. But, again, I think we reviewed -- I mean, you have seen my -- my report. These are the studies I looked at, and they have to be solidified substantially with the IARC report as well as additional meta-analysis.

So, you know, the IARC report, which is the highest authority, in my opinion, in determining a causation between any type of a compound, occupational compound, and cancer, has looked at these data as well as other data and made a conclusion that it's probably carcinogenic to humans.

So I have to take that into consideration in addition to the data that I've actually looked at. The IARC folks obviously and clearly have looked at animal data and other data plus the epidemiologic literature that did not always control for confounding factors. And yet, after all of this, they did find evidence that it's probably carcinogenic to humans.

So, you know, I think there's enough literature out there between these studies that we just reviewed, the IARC, the meta-analysis to

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demonstrate, in my opinion, that there's a causation between this compound and the disease.

Q. Explain to the jury, please, why you believe IARC to be the highest authority on the subject of whether a substance causes cancer?

A. It is not just my own individual belief. I think it is the belief of everybody in the field that IARC is the -- this is what they do. This is what they are tasked with. And in addition to that, this is what they do and this is what they are tasked with, their data and their output was published in the most prestigious journal, more prestigious than all of these journals that we just reviewed today, in Lancet, where the acceptance rate is less -- is close to 5 percent. So they reject 95 percent of manuscripts that they get submitted.

So we can't ignore the most powerful evidence that is out there. So I have to -- I relied on it clearly, as well as additional studies that I cited.

Q. And do you believe that a Lancet peer review of an IARC Monograph means that the people who did the peer review believe that the conclusions of the authors were correct or just that they followed a particular methodology as stated and came

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to a particular conclusion?

MR. LITZENBURG: Object to form. Asked and answered.

A. So when you do a peer review -- and as you probably know from my CV, I do quite a few of peer review for very good and prestigious journals -- you actually have to look at the hypothesis, whether the methodology is sound, whether the authors were free of bias, and whether their conclusions actually were supported by the evidence that they provide. You can't conclude something and you have no evidence in the paper to it. I mean, you just can't. So that's what you look at.

Now, you may not always agree with the conclusion, but your job as a peer reviewer is to look at the evidence that they provide and to review that evidence and see if it correlates with the conclusion. And then you make a decision. Do you reject the paper because you don't believe -- not because you don't believe in the -- you don't believe it was the proper way to be done, or do you accept it but you request additional revisions and you have additional inquiries because you believe the author should really provide more details, et cetera?

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I'm sure you're familiar with the peer-review process. So I don't want to waste time by just talking about how it's done, but this is how we do it.

You don't have to always agree with what they come up with. But I can say that most peer reviews, if you believe the conclusions are so out there, you are going to reject the paper. You're going to say -- and you will e-mail the editor and say, This is making no sense to me.

Q. You know that, sir, in a hazard assessment, which is what IARC does -- hazard, not risk -- a hazard assessment is consistent with a conclusion that a chemical -- that humans are never exposed to a chemical at a level that can cause them to get cancer?

MR. LITZENBURG: Object to --

Q. Do you agree or disagree?

MR. LITZENBURG: -- form.

If you understand it . . .

A. I don't understand the question.

Q. Yes, sir.

A. I think we talked about it earlier this morning, but I think this -- you're asking the question differently, so I don't understand it.

Q. Do you believe that IARC's conclusion means that IARC thinks that human beings actually get cancer from glyphosate and are at risk of getting cancer from glyphosate at the levels at which humans are actually exposed to glyphosate?

A. I think their -- the IARC conclusion speaks for itself. They said it's probably carcinogenic to humans. That's what they said.

Q. Yes.

And do you believe that that means that IARC thinks that humans actually get cancer from humans [sic] and can actually get cancer at the levels at which humans are exposed?

A. I think they believe it's probably carcinogenic. That's what they believe. They obviously clearly did not say it's absolutely 100 percent positively. They said it's probably.

Q. Yes. And I'm asking if you understand what "carcinogenic" means in a hazard assessment.

Does it mean that it's actually out there causing human cancers or that there's a theoretical possibility of this substance causing cancer in human beings?

MR. LITZENBURG: You have asked and answered four times now. Just because you're

not getting the answer that you want --

A. I've answered the question.

MR. LITZENBURG: -- doesn't mean you get to keep asking it.

Q. Go ahead, sir.

A. I have answered this question before, and I believe that, yes, there is enough evidence to suggest that glyphosate probably causes human cancer.

Q. Do you understand that IARC -- according to IARC, the Monographs program may identify cancer hazards even when risks are very low with known patterns of use or exposure?

A. I did not know that.

Q. An agent is considered a cancer hazard if it is capable of causing cancer under some circumstances. That's hazard.

Risk measures the probability that cancer will occur, taking into account the level of exposure to the agent.

Do you understand that distinction, sir?

MR. LITZENBURG: Object to form. I'd like to know what you're reading from or have it marked.

Q. The IARC Monograph.

Go ahead, sir.

The IARC Monograph questions and answers.

Sir, my question to you is, do you understand that that's the difference between risk and hazard, according to IARC?

A. Okay. I would like to read what they -- what they wrote before I answer your question.

Q. It's not in Monograph 112.

A. Okay.

MR. LITZENBURG: I thought you just said it was.

Q. Well, here's my question.

A. Okay. I thought you just said this was in the Monograph.

Q. The Monograph Q&A. It's a different document --

A. Sure.

Q. -- than the Monograph 112.

Do you understand that the difference between hazard and risk, according to IARC, is that an agent is considered a cancer hazard if it is capable of causing cancer under some circumstances?

A. Okay.

Q. Whereas risk measures the probability that cancer will occur, taking into account the level of

exposure to the agent.

Do you understand that about IARC's --

A. I understand the difference. I did not know that this is how they define it. But as you frame it right now, I understand the differences, yes.

Q. Okay. And is your assessment of the hazard of glyphosate in non-Hodgkin's lymphoma the same as IARC's, i.e., that you have identified what you believe to be a cancer hazard, an agent capable of causing cancer under some circumstances, but you have not measured the probability that cancer will occur?

MR. LITZENBURG: Object. That's a mischaracterization.

You can answer.

THE WITNESS: I can answer?

MR. LITZENBURG: Yeah, I mean, if you . . .

A. I believe that there is a hazard with the exposure to glyphosate and development of non-Hodgkin lymphoma. I cannot quantify what that hazard is, so I cannot tell you that people who are exposed to glyphosate have 50 percent risk versus 2 percent risk.

If that is what you're asking, I don't

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believe I have sufficient information to quantify that risk, but I believe that risk exists.

And part of the reason we -- you know, in epidemiology study, occupational studies, it's really important to look at the hazard as opposed to actual absolute risk is because they're a very easy preventable thing. It's an easy -- you know, to prevent the development of a particular cancer, which all oncologists would love to see, if you just say, you know, whether the risk is 1 percent or a 10 percent or a 50 percent, you know what, it's great. We are going to eliminate this so you don't have that risk. Because that's an easy thing to do.

So the actual absolute risk, it's not really as important. I mean, we wear seat belts not because we're going to get into a car accident every day, because in case we get in a car accident, the risk of dying is significantly lower.

So I think, you know, that's why the absolute risk category is not as important in occupational hazards, in occupational studies, because we can eliminate that easily.

Q. You can't give an opinion that an individual exposed to glyphosate has their risk of NHL go up by 1 percent or 45 percent or 90 percent

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or any particular percent; right?

A. No. I can say it will increase, but I don't know by how much percentage. So it's not zero.

Q. But it could be 1?

A. It could be 1. It could be 15. It could be 90.

Q. Okay. And you have not made any attempt to quantify how much the risk increases for someone exposed to glyphosate, in your opinion; right?

A. I think it's difficult to quantify. I think it's -- you know, it's difficult for me, as a clinician, as a researcher, to actually quantify that risk. But I think the presence of the risk is sufficient because it's a preventable strategy to -- to reduce the risk.

Q. Do you know that IARC has stated that the term "probable" in "probable human carcinogen" -- in the phrase "probably human carcinogen" has no quantitative significance?

A. I'm not surprised.

Q. Okay. And does it have no quantitative significance in your evaluation as well?

A. Yeah, I just said it's hard -- it's impossible to really quantify that risk. Again,

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your questions are only answered by a prospective randomized trial where you expose folks to glyphosate and don't expose others and you follow them for whatever years you decide and see what is the risk difference. That's something that will never happen.

To accurately assess the risk and to quantify the risk, that cannot happen. That is impossible to -- all that we can say is that there is evidence that the risk exists, but it could be 1 percent to 99 percent. It's not 100 percent, and it's not zero.

Q. Do you have an opinion based on everything you've reviewed and everything you know that there is any way to tell in a particular person whether an exposure to glyphosate or something else caused their non-Hodgkin's lymphoma?

A. I think sometimes, if you have certain individuals that have been exposed more and -- you know, it's possible that this might actually -- I mean, I think we've reviewed a couple of studies where you have more than ten days, less than ten days, more than two days, less than two days.

So there is a possibility, although I acknowledge that it's not always that -- you know,

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the dose response is very vague in -- in these type of studies. But there is a possibility that sometimes, if you are more exposed for a longer period of time, you could logically have more risk.

I mean, if you are not wearing protective clothes or things like that, I mean, I -- it's -- you know, you have some skin abrasions or skin damage. I mean, so there are certain things that might lend me to believe that this particular individual has a higher risk than another individual. Each case is very different, obviously, but that's why you can't really quantify the risk, because it's just one element. It's one factor.

Q. Based on everything that you reviewed and everything that you know, is there any way to tell that someone opposed to glyphosate and to other substances capable of causing non-Hodgkin's lymphoma developed non-Hodgkin's lymphoma because of glyphosate rather than those other substances?

A. I think you'll have to look at each individual case. It's -- you know, it's hard to -- it's hard to speculate. You'll have to look at what other -- what are these substances, how often he or she was exposed to these substances, how were they applied, top -- I mean, it's just -- each substance

is different. So it's hard to really tell.

I think a lot of times we can try to exercise clinical judgment and scientific evidence to try to tease out which -- which is probably the more offending factor to the extent possible. Sometimes we're successful; sometimes we're not.

Q. So it would be a matter of weighing the different exposures, how much they were exposed, how toxic we believe the substances to be?

A. Which are the substances, do these substances really cause -- cause lymphoma or not, how often were they -- he or she were exposed to, et cetera. You know what I mean. I mean, you just have to look at the type of substances, the amount of exposure, how they were applied. And then you have to look at these substances where there's really data that truly are associated with the disease.

I mean, if the person is smoking heavily and drinking heavily and they're doing glyphosate, I don't have any evidence that smoking and alcohol necessarily cause lymphoma. So just because they're smoking and drinking, it doesn't mean that they're confounding factors. So I think you'd have to look at each case individually.

Q. Do you have the opinion, to a reasonable degree of medical certainty, that any substance other than glyphosate has a synergistic effect or an additive effect with glyphosate to increase the risk of non-Hodgkin's lymphoma?

A. I don't think this has been adequately studied in the literature.

Q. So you don't have that opinion at this time?

A. At this time, I don't have this opinion.

Q. The next epidemiology study that you mentioned, sir, is the Orsi study?

A. Do you have that?

Q. I'll get it out if you need it.

Do you remember that it has an odds ratio of exactly 1?

A. I'll look at what I wrote here. I don't write in my note here the odds ratio. I think what I wrote, a French study that spanned 2000 to 2004 by Orsi, L. suggested increased risk of developing Hodgkin lymphoma and myeloma in patients exposed to pesticides. This study was conducted amongst six centers looking at incident cases with lymphoid neoplasm diagnosis in patients aged 18 to 75 years. Control cases were patients with rheumatology and

orthopedic problems within the same participating institutions.

And I did acknowledge here in this study little evidence was shown in this study as to the relationship between NHL and exposure, but there was evidence with HL and myeloma. And myeloma is obviously a B-cell type of cancer that --

Q. Do you have an opinion, to a reasonable degree of medical certainty, that glyphosate causes multiple myeloma?

A. I did not investigate multiple myeloma to the extent it allows me to give you an opinion that I'm comfortable with at this point.

Q. Okay. Do you have the opinion that glyphosate causes any malignancy other than non-Hodgkin's lymphoma?

A. I did not look at other malignancies. So it -- it may cause other malignancies. It may not. But I only looked at non-Hodgkin lymphoma.

Q. And do you recall that the point estimate for non-Hodgkin's lymphoma with glyphosate in the Orsi study was 1.0?

A. If you can show me this, I can look at it.

Q. Sure.

A. In my report, I did not put the estimate.

Q. Okay.

A. But I did acknowledge that it was little evidence. And I wrote here, "This might be a sample effect as only 244 cases of NHL were evaluated in this study." So my opinion was that it wasn't enough sample size.

Q. When you say "sample effect," you mean the study was too small?

A. That's what I -- that's what I believe I concluded when I reviewed the study.

Q. Okay. I mean, that's what you mean by "sample effect"; right?

A. Yes.

(Nabhan Exhibit 19 marked for identification.)

Q. So I've marked as Exhibit 19 the Orsi study. If you'll look at Table 3, you'll see the non-Hodgkin's lymphoma results.

A. Yeah, 244 cases, 436 control. So I thought the number of cases were -- was pretty small.

Q. And you saw that the odds ratio for non-Hodgkin's lymphoma associated with glyphosate was 1.0?

A. One -- one second. 1.0, yes.

Q. Which, by any definition, is no effect?

A. Correct.
Q. Okay. The Cocco study -- I'm just trying to finish up your studies, sir -- from 2013 had only four individuals with exposure to glyphosate and non-Hodgkin's lymphoma?
A. If you don't mind, I'd like to look at it again.
Q. Yes, sir.
(Nabhan Exhibit 20 marked for identification.)
A. Thank you. Okay. I think this was a larger sample size study in terms of the number of lymphoma and the number of cases and was in several European countries.
Q. And there were four exposed cases, Table 4?
A. Which table that is?
Q. 4?
A. Table 4. Yeah, four cases, I see that.
Q. Two controls --
A. Right.
Q. -- and a nonsignificant and wide-range confidence interval; right?
A. I just show 3.1, but crosses the 1.
Q. And did you believe this study to have power and significance to you with four cases and

two controls?
A. I mean, yeah -- no. I mean, I think the -- here's what you would take from this study: So when you look at the Orsi study, the one before, we had only 244 cases of lymphoma and we saw an odds ratio of 1.0.
When you increase the sample size and you looked at 2,348 non-Hodgkin's lymphoma cases, 2,462 control cases, the odds ratio went up to 3.1.
Yes, it crosses the 1. Yes, the 95 percent confidence interval is 0.6 to 17.1, but it just goes to show that you that the Orsi study that we just reviewed that was odds ratio of 1.0 was so because of the number -- the number is too small, the number of cases.
So I think all what you can conclude from this trial, the Cocco trial, the number obviously four cases and two control, these numbers are small. But it just goes to show you that, sometimes when you have higher number in the denominators, you could have a significant odds ratio or at least above 1 odds ratio despite the fact it crosses the 1.
Q. When the sample size, meaning the number of cases --

A. The number of all cases, right.
Q. -- exposed cases to the -- to the substance under investigation goes down dramatically, then you have a bigger sample size problem; correct?
A. What I'm saying is when you have enough numbers, denominator-wise, of patients that you are looking at with the disease or without the disease, the likelihood of detecting something with the positive -- with the positive exposure, and that is -- with a higher odds ratio, becomes more likely.
So I only use this in contrast of the Orsi paper. The Orsi paper has an odds ratio of 1.0, but only 244 cases that were looked at with NHL. The Cocco paper looked at over 2,000 cases of NHL. And, yes, they found small number of exposure to glyphosate versus non, but the odds ratio went up.
So it's only used in my expert report in way to contrast the numbers. When you have higher numbers of patients that you are looking at, it is possible that the odds ratio will change.
Q. What it means is that you had a population that was much less exposed to glyphosate in that time period; right?
A. That's one way of looking at it, but that's not the only way.

Q. It's the right way, isn't it? Because the relevant factor that you look at when you're looking at the power and predictable value of a study is the number of exposed cases for the relevant exposure?
A. But that's one study. I mean, there are other studies that we looked at that had different numbers and different -- again, I mean, if we are looking at this particular study, I told you how I used it personally. I didn't use it to look necessarily at the percentage of glyphosate exposed individuals and how often did they develop -- non-Hodgkin's lymphoma. I looked at in contrast to a previous study, because I was trying to understand the one that had significantly low number.
I think we reviewed over 10 or 12 studies earlier that have different numbers. So I don't want to take this out of context as the only -- again, this is part of what I looked at, not the only thing I looked at.
Q. Certainly. I'm just trying to understand what you're telling me, sir --
A. Sure.
Q. -- that the number of people in the study who didn't have an exposure to glyphosate and non-Hodgkin's lymphoma is relevant to the validity

of the point estimate that you get in the study, independently of the number of exposed cases?

A. I think you increase -- I think you're more interested in the cases that were diagnosed and exposed versus the cases that were not diagnosed and exposed. I mean case control, when you are looking at things.

Q. Cases that were diagnosed and exposed in this is four?

A. Four, yeah.

Q. Very low number; right?

A. It's small. Very low number. I acknowledge that. And I -- you know, nobody can argue that four is a large number, but I was trying to explain that I used this type of paper just for simple fact it's a lot of numbers, it's over 2,000 patients -- 2,000 patients and 2,000 control. So obviously it's a large number.

Yes, the exposure rate was not as high. But, again, that's when you have higher number and so forth, you can have higher odds ratio, just similar to -- you know, in contrast with the previous paper. That's really the -- how I use this paper.

VIDEOGRAPHER: Can I take a moment to

change discs?

MR. GRIFFIS: Yeah.

VIDEOGRAPHER: Ending Disc No. 3 of the deposition of Dr. Chadi Nabhan. Off the record at 3:34 P.M.

(Recess taken from 3:34 P.M. to 3:37 P.M.)

VIDEOGRAPHER: And beginning Disc No. 4 of the deposition of Dr. Chadi Nabhan. We are back on the record at 3:37 P.M.

BY MR. GRIFFIS:

Q. Sir, the last epidemiology study that you mentioned is the Kato study, and let me know if we need to get it out, but that involves a point estimate for all pesticides put together; correct?

A. Yeah, I'd like you to give me a sample, please.

Q. Sure.

(Nabhan Exhibit 21 marked for identification.)

Q. Are you ready, sir?

A. I'm ready.

Q. Okay. So the Kato study involved women who worked at farms where pesticides were used; correct?

A. Yes.

Q. And you reported an odds ratio of 2.12, which was statistically significant at a 95 percent confidence interval; correct?

A. That is correct.

Q. And glyphosate was not broken down in this study; right?

A. I don't believe they looked at each particular one. This was more in general with pesticides.

Q. And what did you rely on this study for informing your opinion, sir?

A. Again, I think similar to other studies that did not look at particular compounds but they looked at pesticides as a whole, given the fact that the glyphosate in its nature is a -- is, again, could be considered part of the category. So that just solidifies the opinion.

Q. And do you have the opinion that glyphosate forms any part of the risk that is purported to be measured by this study?

A. As I said, they did not tease out glyphosate by itself.

Q. You can't say that it increased or decreased or had no effect on the risk measured in the study; correct?

A. Based on this study, I cannot say that.

MR. GRIFFIS: I know you just changed the tape, but I need to get organized for the next phase, so let's take a break.

VIDEOGRAPHER: Going off the record at 3:40 P.M.

(Recess taken from 3:40 P.M. to 3:50 P.M.)

VIDEOGRAPHER: And we are back on the record at 3:50 P.M.

BY MR. GRIFFIS:

Q. Okay. Sir, we've just gone through the entirety of your -- the section of your expert report entitled "Epidemiological studies," which went from page 11 to page 15. And now I'd like to look at the next section entitled "Meta-analyses."

And you talked about two meta-analyses, one by Schinasi and León and one by Chang and Delzell from 2016; correct?

A. Correct.

Q. You said that the meta-analysis by Schinasi and León found an association between glyphosate and B-cell lymphoma with an odds ratio of 2.0, and this was the same odds ratio for diffuse large B-cell lymphoma; correct?

A. Correct.
Q. And why did you give that particular point estimate and not the other point estimates? Why did you select that one from the Schinasi and León meta-analysis, sir?
A. Do you have a copy of this? I want to make sure I -- a copy of the meta-analysis, please.
Q. Maybe.
(Nabhan Exhibit 22 marked for identification.)
A. Thank you.
Q. Marked as Exhibit 22, sir.
A. Sure. So I think this meta-analysis, they started with 858 articles. 44 were included in the qualitative analysis, and of these 20 papers, provided estimates of association with herbicide chemical groups or active ingredients. And I think you -- I go on to mention, of the included papers, several had data on glyphosate specifically, and I cite the papers that the meta-analysis used for glyphosate in my expert report. And these studies were performed in the U.S., Canadian, Europe, Australia, and New Zealand.
Q. And you say you cited the ones that they relied on for the meta-analysis in your expert

report, you mean the ones listed on pages 15 to 16?
A. Yeah. I mean, I cite several of the included papers, several had data on glyphosate.
Q. So Cantor, Cocco, DeRoos 2003, DeRoos 2005, Eriksson, Hardell, and Orsi; correct?
A. Yes.
Q. And we've discussed all of those today; right?
A. We have.
Q. What -- and you say, after you discuss the findings of the meta-analysis briefly, that this represented "a summary of the data published in the preceding 25 years and solidifies a plausible association between glyphosate and NHL evolution and development." Correct?
A. Yes.
Q. I'd like to understand some of your terms, first of all, sir. What do you mean by a "plausible association"?
A. Plausible association means that there is a causation between the compound that we are looking at and the disease under investigation.
Q. Plausible association means the same as causation to you?
A. So you mean between -- the difference

between association and causation?
Q. I'm talking about the terms that you chose, "plausible association," and I asked you to explain what you meant by it.
A. To a reasonable degree of certainty, it does mean causation to me.
Q. Okay. So when you used it here, you meant causation. And you said between glyphosate and NHL evolution and development.
Did you mean by "NHL evolution and development" something other than glyphosate causes NHL?
A. Well, causing is development; evolution is the disease that progresses or changes course after it's being developed. So, I mean, I think there is -- it's -- you know, when you look at disease like diffused large B-cell lymphoma, sometimes it starts as a diffused large B-cell lymphoma, occasionally it transforms from a low-grade lymphoma to a diffuse large B-cell lymphoma.
So it's not clear, you know, how much of this disease is evolved from a different entity within lymphoma to diffused large B-cell lymphoma versus just start de novo as the large cell lymphoma.

So I think the meta-analysis suggests that there is this association between the compound and the disease.
Q. Are there any studies or any scientific articles, sir, on the subject of NHL evolution with glyphosate as you have just described it?
A. No. I'm -- I'm not aware. It's very difficult to look at that, because the evolution requires continued investigation pathologically. So when lymphoma changes from one entity to a different type of lymphoma, you have to confirm this with repeated prospective biopsies, and this can't be done. So you just speculate. Again, this is just a speculation that --
Q. Yes, sir.
A. We have diffused large B-cell lymphoma, but is it really the de novo large cell lymphoma versus something that's evolved from something else. That's really all that I meant.
Q. Okay. So you are not claiming, to a reasonable degree of medical certainty, that you have evidence that glyphosate causes evolution or development of NHL from one phase to another phase as opposed to just being associated with NHL in general; right?

A. Let me rephrase --
Q. Yes, sir.
A. -- to be very accurate. Diffused large B-cell lymphoma sometimes starts as diffused large B-cell lymphoma and sometimes it becomes diffused large B-cell lymphoma from a different type of lymphoma. So it could be some indolent lymphomas. And I wrote this in my background on NHL in my expert report. Some indolent lymphomas transform into the large cell lymphoma at a rate of about 5 to 10 percent per year after initial diagnosis.
Q. Right. And this isn't the other example. There are other kinds of non-Hodgkin's lymphoma that transform into different types as well?
A. That is correct. So there is this transformation thing.
So, really, all that I meant by this is that this -- the fact that I talked about diffused large B-cell lymphoma, it's fair to acknowledge that this could have evolved from something else I just don't know about.
Q. Okay.
A. I am not aware of a particular study that looked specifically at the evolution per se, because to do that you will need to have a cohort of

patients who have indolent lymphoma, all of them, and you expose all of them to glyphosate. And then you follow them prospectively and see what's the percentage of these folks that transform into large cell lymphoma. I think we both can agree that this can't happen.
Q. Right.
A. So, you know, that's really all that I meant by this statement.
Q. And all day, sir, you and I have been talking about your expert opinion and details of your expert opinion that glyphosate is causally associated with non-Hodgkin's lymphoma in general. And all I'm asking right now is whether you intend to testify, to a reasonable degree of medical certainty, that you have evidence that glyphosate causes NHL transformation or development in addition.
A. It can. Every case is different, as I said, you cannot rule it out; you cannot rule it in. So if you have somebody who has indolent non-Hodgkin's lymphoma, whatever that disease is, and you just spread them with glyphosate for the next five days and then the disease transforms to something else, can you rule out glyphosate could

have may caused this? You can't.
So I think every case is different. So, you know, your first question was is there evidence that specifically looked at this? I am not aware of that, but that doesn't take away that it could actually happen in a particular case. I'll have to look at the particular clinical scenario, the particular patient, the particular situation where I can really provide you with an accurate medical opinion.
Q. And you know of no evidence -- no scientific evidence that that happens as a general proposition; correct?
A. For the general population, no. But it could happen in some patients. So every case is different. I'm going to say that again for the third time. Every case is different.
So, yes, glyphosate, if it's exposed to somebody who has indolent disease, could transform into an aggressive disease. You can't say no. But I don't have a population base study to say that, you know, the risk of transforming from an indolent to an aggressive lymphoma is 15 percent with glyphosate. I don't have that.
Q. Do you know the difference between the

terms "general causation" and "specific causation"?
MR. LITZENBURG: Object to form.
A. Would you explain to me, please.
Q. Sure. When I use the term, sir, what I mean by "general causation" is evidence that a particular substance can cause or does cause a particular outcome in general.
A. Okay.
Q. So all day today we've been talking about your general causation opinion that glyphosate --
A. Correct.
Q. -- can cause non-Hodgkin's lymphoma.
Specific causation means an opinion by someone that this particular substance caused this patient's non-Hodgkin's lymphoma or caused some event like the transformation of a particular patient's non-Hodgkin's lymphoma. Okay?
A. Okay.
Q. Okay. So -- and I'm trying to understand what you just told me. Is it -- is what you're telling me that you can have -- without evidence of -- without general causation evidence that glyphosate can cause a transformation of non-Hodgkin's lymphoma from one type to another or an evolution or development of stages, that you

could nevertheless testify in a particular case as to specific causation, that although -- although I don't have scientific evidence that glyphosate causes transformation, I can testify that it's my opinion, to a reasonable degree of medical certainty, that it did in this particular patient?

A. Yes, I can. I might.

Q. Have you ever formed such an opinion for anyone?

A. Formed an opinion of what?

Q. That glyphosate caused a transformation?

A. I reviewed a case that -- right --

MR. LITZENBURG: That was just prognostics.

A. Prognostics. Just provided an opinion in terms of the prognosis of the actual --

MR. LITZENBURG: Yeah, they have your declaration.

A. Right.

Q. So you are talking about the D. Johnson case?

A. Yeah. That's what I just provided --

Q. The Dewayne Johnson case?

A. Yes. That's really the only thing that I looked at from a prognostication standpoint.

Q. I'm not just talking about in connection

with this litigation.

A. No, I am not.

Q. I'm talking your clinical practice as well, sir.

Have you ever formed the opinion that glyphosate caused a -- caused a patient with non-Hodgkin's lymphoma of a particular type to transform to another type --

A. In my practice I have -- in my practice, I have not had that. In my personal practice, I have not seen -- I have not had a patient that had indolent lymphoma that had glyphosate and then subsequently changed. I have not had that sequence of events.

Q. Okay. Outside of your practice, have you done that?

A. How would I do that outside my practice?

Q. Mr. Litzenburg just said that you did a prognostic thing with Dewayne Johnson --

A. Yeah. I looked at the case from a prognostic standpoint. I was asked to take a look at the case and provide an opinion in terms of the life expectancy.

Q. And I know about that.

Did you form an opinion about

transformation?

A. I did not.

Q. Okay. The meta-analysis by -- oh, I'm sorry. We didn't quite finish with the --

A. Sure.

Q. -- Schinasi and León meta-analysis here. I now understand the terms that you used in that sentence, that it solidifies a plausible association between glyphosate and NHL evolution and development.

What did the Schinasi and León meta-analysis add to the evidence that you were weighing in reaching the conclusions that you did, sir?

A. I mean, I think the -- we both have acknowledged, in all of the studies that we reviewed, that there are limitations to any one individual study. I mean, I think, you know, we can all pick each study apart and realize the limitations. And what the meta-analysis attempts to do is to overcome some of these limitations by looking at the aggregate evidence, by looking at all of these studies together.

So I think the -- you know, when you look at this particular meta-analysis, it showed an odd

ratio of 2.0 with a confidence interval between 1.1 and 3.6. So, to me, it really showed that when you look at the data in combination of everything together, you might be able to demonstrate statistical significance because you're able to look at everything combined. You try to overcome the limitations of each individual study. So you have larger numbers. You have larger denominators, larger cases, larger controls, et cetera.

So really that's -- that's what this meta-analysis gave me, that when you looked at the combined evidence, the odds ratio was significant and there's an association and causality between glyphosate and non-Hodgkin's lymphoma.

Q. And what does it mean to you to have a meta-analysis that, in your opinion, yields a statistically significant result?

A. It means that there is an association and causality between what the authors were looking at and the disease they were looking at.

Q. Okay. And did you not have that conclusion before you saw the meta-analysis, that there was an association and causality?

A. Well, I did, but you need to have -- you need to solidify your opinion. Again, you know, I

didn't look at one paper -- if I looked at one paper in isolation, I may not have had that conclusion. And so you look at all of the papers, the meta-analysis, the additional information, to form the opinion. So I -- you know, my opinion is not formed based on each isolated paper. It's really based on the aggregate collection of evidence.

Q. Okay. Now, since we talked about each one of these individual papers --

A. Right.

Q. -- what did the meta-analysis tell you that you had not learned by looking at each of the individual papers?

A. It showed a statistical significance that some of these papers did not have that. As you articulated earlier, some of them did cross the 1. So at least this -- this odds ratio of 2.0 not crossing the 1 is significant, and it overcomes some of the limitations that the previous individual studies suffered from.

Q. Now I want to ask you about the Chang and Delzell meta-analysis and what the Chang and Delzell meta-analysis added to your assessment of the evidence. And maybe it's the same as what Schinasi and León did.

A. If you are going to ask me about the actual data inside --

Q. Okay.

A. If it's just about my report, it's fine.

Q. Well, let's try from your report.

A. Sure. So, again, it has a risk ratio of 1.3 and original studies, which report PubMed, Google Scholar, with additional references that were found in the bibliography of review articles. Collectively, 19 articles were included, as well as one abstract and one letter to the editor. When analyzing NHL by subtype, the risk ratio for B-cell was 2.0, for CLL 1.3, follicular 1.7, and no increase over HL, Hodgkin's lymphoma.

Q. You said "Notably, no increased risk for Hodgkin's lymphoma was done." Why was that notably?

A. I need to quit the "notably." That's one of my writing skills I need to work on. I use a lot --

Q. You shouldn't say "notably" so much?

A. I've been told I say notably, surprisingly. I'm -- you know -- I'm working on that.

(Laughter.)

Q. All right. I'll leave you alone about it now.

A. When I get reviewers usually asking me, why is this notably?

Q. The overall risk of non-Hodgkin's lymphoma was 1.3 with a confidence interval at -- the lower bound was at 1.0, which would not be statistically significant; correct?

A. It's borderline, I would say.

Q. And then you broke it up by subtype.

A. Well, they broke it up.

Q. B-cell -- well, you --

A. Right.

Q. -- listed their breakdown.

A. I just listed their breakout, and they have -- yeah.

Q. B-cell 2.0, CLL 1.3, and follicular lymphoma 1.7. And two of those were not statistically significant and one was, sir.

Do you have the opinion that the epidemiological evidence shows a difference in the manifestation of non-Hodgkin's lymphoma across subtypes based on exposure to glyphosate?

A. Yeah. I don't think there's sufficient evidence, frankly, to have robust conclusions based on 60 subtypes, and I don't think it's honestly doable. It's impossible. And I think we alluded to

this earlier this morning.

Q. And everyone who's tried to stratisfy [sic], of course, has not gone to 60; they've gone to 3 or 4 like this B-cell.

A. Yeah. It's very difficult. Right? I mean, follicular lymphoma is a form of B-cell, for example. But they try to look at alone. CLL is, quote/unquote, a form of B-cell lymphoma, although it's leukemia.

So I think it's very difficult to look at the subtypes because there are so many right now. And because of the heterogeneity but also because of the fact that, in order for you to be convinced of the subtypes, you need to have pathologic confirmation of each particular patient, each particular subtype. And many patients don't even know what type of lymphoma they have. They just say, I have non-Hodgkin's lymphoma. So it's just a very difficult exercise to do.

Q. On page 18 of your expert report, you talked about -- when you were talking about the EPA SAP panel review, you mentioned that the panel recommended that the EPA talk to the AHS, the Agricultural Health Study investigators, to determine whether updated data on incidence of NHL

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and other cancers are available.
And why did you put that in your expert report?
A. I think, you know, it's -- it's -- one of the things that we have to acknowledge about the limitation of the Agricultural Health Study. It is the only prospective study in my review that I was able to find, but it does have limitation with the short follow-up time of 6.7 years. And many of the patients in the Agricultural Health Study were younger versus patients who are diagnosed usually with non-Hodgkin's lymphoma. 70 percent were younger than the age of 70, 46 percent were younger than the age of 50.
So I think it was important to highlight this limitation and the fact that the EPA wanted to get a follow-up, if available, and published.
(Nabhan Exhibit 23 marked for identification.)
Q. Sir, do you know who Aaron Blair is?
A. Aaron Blair -- is he IARC? No, he is -- the names are starting to blur a little bit.
Q. Aaron Blair was the chief investigator for the Monograph 112.
A. Okay. Yes. IARC.

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Q. He headed it up.
A. I was right for a change.
Q. And he is on the Agricultural Health Study as well.
A. Yes, correct.
Q. You see his name on the front of this draft, March 15, 2013, draft on lymphoma risk and pesticide use in the Agricultural Health Study; correct?
A. He's one of the coauthors, yes.
Q. Have you seen this document before?
A. I have never seen this document before.
Q. Okay. Let's take a look at it. This was a document, sir, that I'll represent was produced and marked as an exhibit at the deposition of Aaron Blair --
A. Okay.
Q. -- where he produced it as additional follow-up data that was available on glyphosate and other exposures from the Agricultural Health Study?
A. So this is an actual paper that was published somewhere?
Q. It was not published, sir. It was a draft that was never published. And we'll talk about that too.

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A. Okay. I'm just -- I thought I would have seen it if it was published. Okay.
Q. Yes. If it had been published, you would have seen it.
So the 213 draft manuscript was published with additional data -- five additional years of data, as you can see from page 3; correct?
A. Yes, I see five years later, '98 to '04. Okay.
Q. You see a discussion on page 9 of follow-up questionnaires being given, additional data was collected? Page 9, sir. I'm looking at the middle of the --
A. Okay.
Q. -- page numbers at the bottom of the middle of the page.
A. Okay.
Q. "So follow-up questionnaires were given and a data-driven multiple-imputation procedure was used, where there were -- where there were not responses."
Do you see that?
A. The middle paragraph, "A follow-up questionnaire" --
Q. Yes, sir.

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A. -- "which ascertained pesticide usage enrollment was administered about five years after enrollment and completed by 63 percent" -- so not everybody -- "of the original participants."
Q. Right.
A. Okay. "And for participants who did not complete a Phase 2 questionnaire, a data-driven multiple-imputation procedure" -- what does that mean in English, "a data-driven multiple-imputation procedure"?
Q. Well, it's a statistical method to figure out what the results would have been for the procedure -- for the questionnaires that were not returned based on the data that was provided.
A. Well, you could critique this right away. Okay. That's fine. I mean, ultimately --
Q. Take a look at the results on page 12, sir.
A. Only 63 percent answered. Okay. Page 12. Okay.
Q. Do you see that it says "The risk of non-Hodgkin's lymphoma increased significantly, and in near-monotonic fashion with age in the age S cohort."
It's the very first sentence, under "Results."

A. Oh.
So what do they mean by "monotonic fashion"? This is the first time I hear this --
Q. Oh, monotonic, sir, in statistics means that as age increases --
A. Oh, I see. Linear type of thing?
Q. As a stepwise linear progression, yes.
A. Okay. I mean, I'm not really sure that the linear thing, that we are 100 percent certain with occupational hazards, but that's fine. That's what they're saying. Okay.
Q. Okay. And, I mean, that's about what you would expect. You would expect that as age increases, the incidence of non-Hodgkin's lymphoma would also increase; right?
A. Agree. I just -- I don't know if it's linear. That's what I'm saying. Okay.
Q. And I don't mean it's in a straight line --
A. That's why --
Q. I mean every age cohort, as it goes up, you have more non-Hodgkin's lymphoma. You would agree with that?
A. I agree with that.
Q. Okay. "And the number of livestock on the farm and whether cohort members" -- I'm looking at

the end that paragraph -- "whether cohort members drove farm equipment with diesel engines significantly increased risk of non-Hodgkin's lymphoma."
Does that make sense given what you know about the causes of non-Hodgkin's lymphoma on the farm, sir?
A. Well, I mean, this one is looking at the number of livestock. I mean, so it's not just farming. They're trying to correlate the number of livestock with the increased risk. So I -- obviously, I know that there is data on farmers and increased risk. I wasn't aware that the number of livestock correlates with increase of NHL.
Q. It would increase exposure to a number of things, including animal viruses; right?
A. I get that, but I didn't realize -- I mean, again, I'm not aware of data that the number -- so if you have five cattles versus ten, I didn't know that the ten necessarily increased risk by -- versus five. Just the fact that you have more number, it doesn't necessarily mean you're going to have more exposure to particular pathogens.
It may appear this way, but unless you have actual -- what they're saying here is it does. So,

I mean, again, that's what they're saying.
Q. Okay. Yeah. Right. They said that it does.
A. But all of this is new to me, so . . .
Q. Yes, sir.
Go to page 17, the middle paragraph.
A. Okay.
Q. And I'm looking at the last sentence in that paragraph. "In our study, we could not evaluate MCPA, but found no excess risk of NHL or its subtypes with the use of glyphosate" --
A. I'm sorry. Where are you looking? Page 17?
Q. The last sentence --
A. Last sentence.
Q. -- in the middle paragraph on page 17, starting "In our study."
Do you see that?
A. This is page 17. I don't see "In our study." Where is it? Oh, here it is. That second paragraph.
Okay. I see it.
Q. "In our study, we could not evaluate MCPA, but found no excess risk of NHL or its subtypes with the use of glyphosate, 2,4-D, or 2,4,5-T."

Do you see that, sir?
A. I see that.
Q. Okay. I know that, as you said, all this is new to you.
Now let's turn to the data tables.
A. I mean, you do recognize there are so many comments on the side that I have not seen; right?
Q. Oh, yes. I know, sir.
A. Okay. Which -- where do you want me to go now?
Q. Let's go to page 31 first.
A. Okay.
Q. Table 2.
A. Yes.
Q. And this is "Pesticide exposure, lifetime days and intensity-weighted lifetime days, and the age-adjusted risk of NHL incidence, 1993 to 2008. So what this is is -- it's the same as the table we looked at in DeRoos 2005, the published AHS data with five less -- five fewer years that looked at lifetime days and intensity-weighted lifetime days with the new data in; right?
A. Okay.
Q. If you go to page 34, you see the data for glyphosate.

A. (Speaking sotto voce.)
Okay. I just want to pull the DeRoos paper. Which one is -- okay.
Q. So we looked earlier at the DeRoos 2005 paper, which you've expressed a criticism of, that there wasn't enough follow-up in terms of years of follow-up, sir.
And we looked at the table in which lifetime days and intensity-weighted lifetime days were assessed and saw that, in that table, there was no association between glyphosate and non-Hodgkin's lymphoma. I believe you testified that you could not use that table to support a hypothesis that glyphosate causes non-Hodgkin's lymphoma; correct?
A. You're talking the DeRoos '05; right?
Q. Yeah.
A. I said that, yes.
Q. Okay. So this is the corresponding table, sir, in the Alavanja 2013, the 2013 AHS data.
MR. LITZENBURG: I object to the representation.
Q. On page 34, do you see that they show glyphosate at no exposure, low exposure, medium exposure and high exposure levels for lifetime days and intensity-weighted lifetime days?

A. Yeah, but I -- I'm struggling in understanding how it is related to the Table 3 of DeRoos '05 in terms of the tertile. So in the DeRoos '05, Table 3, the first tertile is 1 to 20, the second tertile, 21 to 56. The other one is 57 to 2678.
I don't know how they're representing this here. They have none; low, 20; medium, 67.5; and high, 173.25. I don't know what these numbers mean.
Q. Well, breaking it into three even tertiles would depend on what kind of underlying data you have.
A. Well --
Q. How the tertiles break out.
A. -- I understand that, but the tertiles were years of use multiplied by the -- right? In the DeRoos '05, they had the cumulative lifetime days of use or cumulative exposure days, years of use multiplied by days per year categorized in tertile -- in tertiles among users, from 1 to 20, et cetera.
So did they use the same thing here? Because the numbers are different than the numbers here.
Q. Yes, sir. They broke it into three even

tertiles. And what constitutes an even tertile depends on the actual exposures of each individual in the tertile.
A. My question is, these numbers between parentheses, if -- Table 34, low, medium, and high, what do these numbers represent? You have 20, 65.75, 173.25. What are these numbers?
Q. Those are a measure of days of exposure and intensity-weighted days of exposure.
But my question is about the point estimate, sir, in the second and third columns.
A. I just wanted to make sure we're comparing apples to apples. That's all. Okay.
Q. In this chart in exhibit -- what exhibit is it? -- 23. Table 2, "Pesticide exposure, lifetime days and intensity-weighted lifetime days," there is no association between glyphosate and intensity-weighted or non-intensity-weighted lifetime days of exposure; correct?
A. This table shows no association.
Q. Okay.
A. I would like to review this paper in more detail. But it's not a paper; it's not published.
Q. On page 36, you see Table 3, "Pesticide exposure, lifetime days, and the age-adjusted risk

of NHL by cell type."
A. I see that, yes.
Q. And then we have a breakdown of four different groupings of NHL types; correct?
A. Uh-hum.
Q. And then on page 39, you see the data for glyphosate. And for all the subtypes, there was no association in the data in this study; correct?
A. Yeah, it does not seem that there is an association here based on this data.
Q. Page 53, there is a supplemental Table 2 entitled -- I'll wait until you're there.
A. I'm there.
Q. Entitled "Pesticide exposures, total days and intensity-weight total days, fully adjusted risks of NHL incidence, 1993 to 2008."
On page 59, we see the data. And, again, there is no association in these data, correct, between glyphosate and non-Hodgkin's lymphoma?
A. In this data as presented, you're correct. But this paper requires way too much review than this, and I -- it's, like, 70-page paper.
Q. I wish you'd reviewed, sir.
A. Well, I -- it's not in the published literature.

Q. On page 66, Supplemental Table 3, "Herbicide exposures, lifetime days, and age-adjusted NHL risk by cell type, 1993 through 2008."
A. Which page, sir? Which page are you on?
Q. Page 66.
A. Okay. Yep.
Q. The data is on page 69 for glyphosate. And, again, there is no association between glyphosate and non-Hodgkin's lymphoma in this data as reported; correct?
A. I don't see an association here based on the data that is represented.
Q. Supplemental Table 7 on page 84, sir, "Pesticide exposures, total days, and intensity-weighted total days, age-adjusted risks of NHL incidences, 1993 through 2008." On page 91 is the glyphosate data. And, again, there is no association between glyphosate and NHL in the data as presented here; right?
A. Page 91?
Q. Yes, sir.
A. Yes.
Q. So I'm correct that there was no association?

A. As depicted in this table, you're correct.
Q. Let's go back to page 34 to get some sense of how much larger this cohort is, sir.
A. Okay.
Q. So page 34, again, is the glyphosate data for lifetime days and intensity-weighted lifetime days. And the first column, after "none, low, medium, and high" gives the N, the number in each of those categories; right?
A. Uh-hum.
Q. So there were 70 people with no exposure to glyphosate who had non-Hodgkin's lymphoma, 89 with low exposure, 78 with medium, and 83 with high; correct?
A. This is correct.
Q. So 250 exposed cases compared to 94 from DeRoos 2005; right?
A. In DeRoos '95 -- 2005 at 29, 1 to 20; 15, that's 44; and 17, that's 61, according to this table.
Q. This is a much larger cohort?
A. Yes.
Q. Okay.
A. I still would like to understand the methodology, how they actually grouped these

patients. So if you can just show me how they grouped them, because they are being grouped differently between DeRoos '05 and this paper. But there's so much corrections on it, that it's very difficult to even tease out.
Q. Yes, sir.
There is a draft paper with comments on it.
A. Well, there are obviously a lot of comments that requires revision. And, clearly, there is so much corrections that are needed.
Q. We've discussed earlier that Aaron Blair, who is the head of IARC and on the Agricultural Health Study, had his deposition taken in this case and that you haven't read that deposition; right?
A. I have not.
Q. Okay.
And Dr. Blair at his deposition, when he was asked what the ever/never statistics would be from this study, the Alavanja 2013, admitted that it would be less than 1, it would be that 0.9 point estimate.
Did you know that, sir?
MR. LITZENBURG: I'm going to object to that characterization. You don't need to listen to any representation he makes about a

deposition transcript he hasn't seen -- or he hasn't shown you.
A. I don't know.
MR. LITZENBURG: You can answer the question if you want to --
A. I did not know that.
MR. LITZENBURG: -- with that caveat on the record.
Q. And, sir, did you know that Aaron Blair admitted that the meta-relative risk for NHL that was calculated by IARC, where he was presiding, would probably not have been statistically significant if IARC had had this data?
MR. LITZENBURG: Same objection.
A. I know nothing of what Aaron Blair has ever said.
Q. If that were true, sir, if IARC had had the 2013 data and calculated a meta-analysis that was not statistically significant, that was, in fact, near 1, how would that affect your opinion that glyphosate --
MR. LITZENBURG: Same objection.
Q. -- can cause non-Hodgkin's lymphoma?
A. I think we both know that we don't know the answer to that. I think it would be critical, if

this type of literature is sound and good, it would be submitted for rigorous peer-review process to a respectable journal for peers to look at. If it's written in 2013, and it's -- four years later, it has not been published, then there are clearly some issues in it that, to this date, has not been published.

Having said that, until it's published, peer-reviewed, and go through the process, all of the information here in my -- has nothing to do with my opinion or testimony.

Q. Is that because you have a policy of not reviewing unpublished literature?

A. Well, how am I supposed to find this? If it's not reviewed, I mean, how am I supposed to find this type of literature?

Q. It's in your hands now, sir.

A. You want me to review a 75-page document in five minutes?

Q. Is this something that you're going to weigh in forming your opinions about non-Hodgkin's lymphoma and glyphosate now that you have it?

MR. LITZENBURG: Object to form.

A. If it is not in the peer-reviewed literature that is published and been subjected to a

rigorous peer-review process, I will not rely on it.

Q. Why?

A. I think it's self-explanatory. I mean, I'm not going to rely --

Q. Go ahead.

A. -- on an opinion -- if a scientist has an opinion that is valid, they usually submit that opinion to a journal, to a peer review, so it could actually be looked at and evaluated.

Q. Do you know that Mr. Blair and his colleagues -- Dr. Blair and his colleagues discussed publishing this before IARC so that IARC would be able to consider it and chose not to do so and has testified to that effect?

MR. LITZENBURG: I'm going to object again about representations about Aaron Blair's testimony and . . .

A. I know nothing of what Aaron Blair did, said, or -- I have not looked at what he actually said, and I don't know what his opinion is in this matter.

Q. If this data were valid, if it -- if this could be written up and published and present these same data that we just looked at showing no association, how would that, as a published paper,

affect your opinion on glyphosate and non-Hodgkin's lymphoma?

A. Well, I think --

MR. LITZENBURG: Object to form, and asked and answered.

Go ahead.

A. The fair thing is really for the IARC to relook at things. And now there is additional evidence, and they probably have to relook at things and see whether this solidifies the evidence further, not solidify the evidence further. It's hard to tell, because, again, you have to remember that the evidence is not just based on one or two papers; it's based on the totality of evidence.

There is a lot of epidemiologic literature. There is some meta-analysis. There is some genotoxicity studies. We talked about some animal studies, et cetera.

So it's really not one thing that's going to sway the pendulum one way or the other. And I think you've asked me several times, if this study was reviewed or not reviewed, how would your opinion change. And it's impossible to answer this, because I'll have to put my mindset into a situation that I don't have evidence I already looked at. And it's

hard to do that because I already saw that evidence and I looked at and I critiqued it.

So I think if this paper ever makes it to light and gets published and peer-reviewed in a journal, then it should be looked at like all other journals that we looked at. I think the importance of a peer review is that -- so this paper, you know, would be sent to -- to folks who understand this type of literature. It would be subjected to a statistic -- the rigor of statistics. A statistician would review the methodology, a toxicologist, et cetera, an epidemiologist. And they would provide comments and do the things.

I mean, you could tell, frankly, just from the draft that you gave me -- I mean, it's kind of funny, frankly. Let's see how many comments there are already that's outstanding. I mean, you know, there's over 50 to 60 comments that, you know, from -- no, I take it back. 77. Look how many comments there are of certain outstanding things that are still not resolved in the author's opinion. That tells me they are way far from even getting close to agreeing on what this paper means.

77 comments on page 83. That's the last thing. Many of the comments are Aaron Blair

himself.
Q. If they chose not to publish this because they didn't want IARC to come to a conclusion other than what IARC came to, would you think that was scientifically proper?
MR. LITZENBURG: Object to form.
A. If they -- if they chose not to publish it intentionally, you mean?
Q. If they chose not to publish this because they didn't want IARC to have this data because it might influence IARC to find that glyphosate was not associated with non-Hodgkin's lymphoma, do you think that's scientifically proper?
MR. LITZENBURG: Same objection.
A. Yeah, I wouldn't agree to not publishing this for the sole purpose of affecting a committee review. If it were me, I would not withhold information for that sole purpose.
I can't speak as to why it is not published. I mean, what you're telling me is Dr. Blair has testified to the content of the data. So, clearly, he is willing to share that data with -- in the public domain. So I believe that the reason a paper like this is not published is the fact that it has a lot of methodological issues that

they're trying to go through.
There's -- again, this is -- the draft that you gave me is 12/5/16, almost a year old. And it's one of those studies that has a lot of issues that they're trying to address. And I think they're struggling in addressing them. That is my honest opinion when I look at a draft like this that's been sitting on the shelf for a year with 77 comments on it.
But, if it were me, I would not withhold information just so I would affect a committee decision. But that's me.
Q. Were you provided with any -- you have looked at several depositions in this case; right?
A. I have.
Q. Which ones?
A. I looked at Dr. Neugut, Dr. Saltmiras. I think I read his deposition.
MR. LITZENBURG: I think there's a list at the end of your --
Q. Well, you list -- in your expert report, you listed Donna Farmer, David Saltmiras, and William Heydens.
A. And Neugut as well.
Q. Okay. And Neugut. And that's it?

A. Yeah.
Q. How did you even know about those depositions?
A. The -- Tim forwarded them to me.
Q. Okay. You said earlier that you found all of the scientific literature that you relied on for your expert report and listed under documents reviewed by Dr. Nabhan yourself; is that right?
A. I have researched that myself, yes.
Q. Okay. Were any of those sent to you by counsel for plaintiffs?
A. When I struggled in finding particular information, I reached out. And they were able to help me if I struggled in finding some of those --
Q. Okay. So if you asked for a particular article, they sent it to you?
A. Yes.
Q. Otherwise, they didn't send you anything in particular?
A. Correct.
Q. And the depositions and exhibits they chose to send you, the four that you have listed?
A. It's the second -- it's the other way around. I -- even before I accepted this, I did a lot of literature myself to decide whether I can do

this or not. And, again, I did my literature search. But if I struggled sometimes in finding some of the information, they -- I reached out, and I was provided some help.
Q. Yes, I'm asking about something different now. I've moved on from the scientific literature.
A. Oh.
Q. I'm talking about the depositions now.
A. Oh, the deposition --
Q. For those, they chose what to send you; is that right?
A. Yes. They were -- I didn't have a choice.
Q. Yes, sir.
And anything that was unpublished that plaintiffs' counsel may have or know about, you didn't get that from them? You didn't hear about it from them or get it from them, like this Alavanja --
A. I reviewed whatever they sent me, plus I reviewed a lot of things on Google and CNN. It's very easy to search and see what's going on.
Q. This MON-GLY production, MON-GLY 01314233, et seq -- et seq means "and so on." Right?
A. I don't know. Which one is this?
Q. Do you see what I'm talking about?
A. I see what you're saying, yeah.

Q. These are -- these would be Monsanto documents?
A. Yes, I have reviewed -- I don't know which one is this, but I have reviewed some of the documents that were not necessarily papers that were sent to me by the plaintiff.
Q. Okay. And was it -- how large of a volume was it?
A. I think this may -- I don't remember which one is this.
MR. LITZENBURG: I don't know what it is.
A. I'll have to get back to you on that. I really don't know. I don't know.
Q. Did you review a whole box of documents --
A. No, no, no, no.
Q. -- or a little stack or what?
A. It's probably 10 pages or 12 pages.
Q. Okay. So this was one particular Monsanto document, and you --
A. Probably a couple of documents. Probably a couple of documents.
Q. And then IARC Monograph 112, how did you find that one?
A. I think I -- this is one of the things that I asked for help to get the actual monograph, and I

reviewed. But I reviewed the paper myself, the Guyton paper.
Q. And then the EPA SAP panel, final minutes and report, how did you have the idea to get that and read it?
A. I've asked the plaintiff for that.
Q. Did you know about it from your own research and ask them to get it for you?
A. Yeah, yeah. I've asked to see the last one as possible.
Q. And are there any other documents that you considered important in forming your opinions that aren't listed on these two pages, Attachment B to your expert report, sir?
A. Not for this particular report, no.
Q. Other than the --
A. But you have to add the Neugut deposition, just to be accurate.
Q. Yes, we discussed it. So it's -- it's on the record, sir.
Other than the declaration that you did in the Dewayne Johnston [sic] case, have you done other expert reports on the subject of glyphosate in any way?
A. I have not.

MR. GRIFFIS: Let's take a five-minute break.
VIDEOGRAPHER: Going off the record at 4:40 P.M.
(Recess taken from 4:40 P.M. to 4:52 P.M.)
VIDEOGRAPHER: And we are back on the record at 4:52 P.M.
MR. GRIFFIS: I'm going to stop my questioning now and reserve the rest of my time for redirect. And Mr. Litzenburg is going to ask some questions.
MR. LITZENBURG: Thank you. I do have a few questions in follow-up. I'm going to work backwards, so it will be a little awkward, and I apologize for that up front.
EXAMINATION
BY MR. LITZENBURG:
Q. Let me start out by asking the opinions you've given today, do they have anything to do with Cardinal Health or with your employment with Cardinal Health?
A. No, they're not. They're my individual opinion. My employer bears no opinion on this case whatsoever.

Q. And we are compensating you, but that's on an individual basis, has nothing to do with your company; is that right?
A. Correct.
Q. Okay. We discussed some depositions today. Well, let me back up.
You have stated today over and over again, and on many levels, your opinion as to general causation. You understand that term now as we are using it, general causation?
A. Yes, I do.
Q. Okay. Does -- that opinion of general causation, does that depend on any of these depositions as a basis for that -- for forming that opinion?
That's a terrible question. Let me ask it again.
Did you have to rely on any of these deposition transcripts in order to form that opinion?
A. No. I formed this opinion based on my research of the available evidence, the published literature, as well as my own expertise and treating patients with lymphoma for over 17 years.
Q. Okay. So your opinion didn't change with

what Dr. Neugut said at his deposition or what was written in the transcript of Dr. Neugut's deposition; is that fair?

A. No, it did not.

Q. Okay. I'm going to briefly -- again, we will work in reverse -- ask you a couple questions about this Exhibit 23.

First of all -- well, we spent a lot of time on the record. But, generally, do you have any idea what this is?

A. Well, I saw it for the first time today.

Q. Uh-hum.

A. I'm being told that this is an unpublished data on the Agricultural Health Study.

Q. Did you review -- what other sets of unpublished data did you review for your opinion today?

A. I just reviewed, you know, the EPA as well as some of the depositions, but everything else I reviewed and I relied upon was published in peer-reviewed journals.

Q. Okay. And so you drew a line at peer-reviewed published data in order to review for basing your opinion on; is that fair?

A. I think it's very important for anything

that is looking at situations like this to be reviewed by experts in the field and in the literature, because if it withstands the rigor of the peer-review process, then it just holds -- it holds more scrutiny that I would look at more critically and I will take more seriously.

Q. Did I ask you or any of my colleagues ask you to review any other draft papers?

A. No, you have not.

Q. Did you ask me to provide you with any draft papers to form your opinion?

A. No. The only thing I asked you about was the EPA report.

Q. Does this even look like a final draft to you?

A. It looks like an awful draft, in my humble opinion.

Q. But, I mean, does it look like it's final form, ready for submission to any peer --

A. Not even close.

Q. Okay. And I think you noted that these comments were authored comments, in other words, coauthors speaking to each other; is that correct?

A. Coauthors speaking to each other. It's very difficult to know what each author is saying to

each person. There's a lot of information that needs to be looked at, lots of -- this is just, you know, a very, very preliminary draft that is also one year old. The last it was looked at was December 2016, and the data was almost four years old.

And I don't think that it would withstand, frankly, the scrutiny of peer-review process when you lose 40 percent follow-up. I think that's really why the authors can't even submit it anywhere.

Q. Well, let's take that one step at a time.

Tonight is, again, the first time you've ever seen this document; is that fair?

A. Correct.

Q. And I don't want to get down in the weeds and examine all of the analyses and statistical power, et cetera, of this draft paper, but you had -- you said at the beginning that you had -- yeah, that you can critique this right away. And then you mentioned something about follow-up.

Would you very briefly and concisely let us know what you're talking about there?

A. Well, when you look at the -- at page 9, it says, "A follow-up questionnaire which ascertained

pesticide usage enrollment was administered about five years after enrollment, completed by 63 percent."

So you have almost 40 percent loss of follow-up with the second phase. And this application of impute likely -- you know, stratified sampling was employed to impute likely use of specific pesticide seems to me like an exercise to overcome a challenge. And that exercise will never stand the test of rigorous peer-review process.

Q. Fair to say that statistics we're dealing with today and you deal with in your job have to do with public health, cancer?

A. Yes.

Q. Okay. Does -- and you take -- is it fair to say that you try to take a more conservative approach with statistics when you're looking at matters of public health or life and death?

A. Of course.

MR. GRIFFIS: Objection to form.

Q. Okay. Does imputing 40 percent of data, is that an appropriate or conservative approach when looking at human cancer?

MR. GRIFFIS: Objection to form. Foundation.

A. No. The answer is you cannot really forego 40 percent of lack of follow-up.
Q. We can set that aside for now, that draft paper, if you would, Doctor.
Now, setting that aside and getting back to all the published literature that we spoke about for the rest of the day, are any of those studies perfect that we talked about today?
A. I don't believe any study is 100 percent perfect. I don't believe such a thing actually exists in epidemiology literature.
Q. So while you have reached a conclusion regarding general causation, there is not a single paper that you would hold out and say, "This is the perfect paper and by itself provides this evidence 100 percent"?
A. I don't --
MR. GRIFFIS: Objection to form. Leading.
A. I don't believe that any paper is perfect that I reviewed.
Q. Okay. There was -- give me a minute.
(Pause.)
Q. There were some quotes read to you from a IARC monograph question-and-answer. And that was not given -- provided to you, and I don't have a

paper copy either. But I'm going to read you another quote from that same document.
"Group 2A means that the agent is probably carcinogenic to humans. For agents in this category, there is usually convincing evidence that the agent causes cancer in laboratory animals and some evidence that it could cause cancer in humans, but the evidence is humans is not conclusive."
Do you agree with that statement -- do you agree with IARC's classification of glyphosate as a 2A agent?
A. I do.
MR. GRIFFIS: Objection to form.
Q. There's some discussion of hazard versus risk, hazard being absolute and risk being a measurement. Is that the way that you understood the discussion?
A. That's the way that it was phrased to me.
Q. Okay. And so we have seen IARC's decision as to whether or not this was a carcinogen. That's been discussed at length today; right?
A. Yes.
Q. And we've also discussed, probably more so of the day, a quantification of excess risk that's been made by various models; is that fair?

A. Yes.
MR. GRIFFIS: Objection to form.
Q. And that would be the risk portion of the hazard and risk delineation; right?
A. Yes.
MR. GRIFFIS: Objection to form. Leading.
Q. There were some questions about, you know, what -- trying to pin you down, I think, about, you know, what is the true increase in risk.
Epidemiology is the study of increase in risk across populations; is that fair?
A. That is epidemiology.
Q. Okay. It's not -- there is no one number for how much a person's risk is increased by exposure to something, these are all population subsets we're talking about today; is that fair?
A. That is fair.
MR. GRIFFIS: Just continued objection to form. Leading.
Q. There was one question -- well, I won't try to quote it verbatim. There was a question asking you to point to a single statistically significant positive result controlling for other pesticides.
We did look at some other issues above 1 that controlled for other pesticides today; is that

correct?
A. Yes, we did.
MR. GRIFFIS: Objection to form. Leading.
Q. And then when the meta-analyses -- the two meta-analyses that you reviewed that were published took into account the various odds ratios and powers, did they reach statistical significance?
A. Yes, they did.
Q. And those -- okay.
Let's look at Eriksson, 2008. Maybe 18?
A. Which one is it?
Q. Eriksson 2008.
A. Is it 19, you said?
Q. I'm sorry. It's 18.
A. Okay.
Q. If I wrote it down right.
A. Okay.
Q. There was criticism of numbers -- well, results are reached in these papers where the confidence interval crossed 1 by counsel today.
Do you remember questions like that?
A. I do.
Q. Okay. If you look at Table 4, we spent some time looking at that. And these are all different herbicides or classes of herbicides; is

that correct?
A. Correct.
Q. And defense counsel asked you to count up all the ones with an odds ratio above 1.
Do you remember that question?
A. I do.
Q. Nearly all of them, however, have a confidence interval which crosses 1; is that correct?
A. Correct.
Q. Okay. We can set that aside for now. Well, I'll just ask you a follow-up.
Have you done -- have you done any studies on whether any other herbicide causes non-Hodgkin lymphoma today?
A. I did not.
Q. And so you don't know if controlling for a specific herbicide or a group of herbicides in a given city would cause an odds ratio to go up, down, or what it would do to the statistical significance; is that fair?
A. That's fair.
Q. Let's look at -- there was a question about a failure of these papers to find a dose response. Let's look at McDuffie, if we can. And we'll just

have a race to see who figures out which exhibit it was first.
I think it was the first published paper that was marked.
A. I think McDuffie is Exhibit 11. It's Exhibit 11.
Q. Yeah. If you would look at page 1160 with me.
A. Okay.
Q. And in the second paragraph on that page, starting with Table 8, the authors in this published McDuffie paper concluded that they demonstrated a dose-response relationship for glyphosate.
Do you see that in the final sentence of that paragraph?
MR. GRIFFIS: Objection to form. Leading.
A. I see that.
Q. I'll ask it a different way.
Did the authors of McDuffie put in this published paper whether or not they've -- they demonstrated a dose-response relationship with glyphosate and NHL?
A. They did.
Q. Okay. And was that -- did they find such a response relationship or not?

A. They did.
Q. What is -- I think this was hinted at today, but I want to give you a chance to explain it. Is there a problem with using farmers as the control groups in these epidemiological studies?
A. I mean, I wouldn't say it's a problem, but I think, given the fact that farmers have an inherent increased risk of developing non-Hodgkin's lymphoma, your control group already has a high bar. So demonstrating a statistical significant above and beyond that in a case -- in the actual individuals affected becomes more harder.
And that's why you would still take an odds ratio as a hypothesis generating that you would look at despite the fact that it crosses the 1, because your control group already is establishing a much higher bar that you have to overcome.
Q. Okay. This is going to make my head hurt, but there was -- there was a comparison of logistic and hierarchical regression or something like that.
Do you remember that today? Do you have any reason --
A. Vividly.
Q. Do you have any reason to believe that the logistic regression method is inferior to

hierarchical?
A. I honestly -- as I said, this require a statistician to answer the question. I don't think I'm qualified to even know the difference between logistic regression and hierarchical regression. I've always, I believe -- and that's really the value of peer review, that you have to -- statisticians would look at that.
As a clinician, I don't understand quite the nuances in terms of differences between both methodologies.
Q. Okay. But do you -- well, we'll leave it at that.
A. I don't believe it would alter my opinion per se.
Q. Let's look at -- we talked a lot about odds ratios and a fair amount about confidence intervals, but let's go to Exhibit 12, if you would.
A. Okay. It's the Hardell paper.
Q. Yes. And we had two marked, so make sure it's Hardell 2002.
A. Yes.
Q. Okay. And I want to look at Table 1.
A. Okay.
Q. And so, for example, glyphosate has an odds

ratio of 3.04. In lay terms -- and I think we've all dealt with epidemiology a lot and there's a potential for a lay person to see this -- what does an odds ratio mean exactly?

A. Sorry?

Q. Leaving alone the numbers. I'm sorry. What exactly is an odds ratio? How would you explain it to a lay person?

A. I mean, I would say an odds ratio is basically the fact that the exposure to a particular offending agent increases the risk above and beyond other factors, above and beyond the control.

Q. Okay. And so just taking this as an example, in Table 1 of Hardell, there is an odds ratio -- and it is statistically significant -- for glyphosate; is that correct?

A. Yes. It's 3.04, and it's -- the confidence interval 1.08 to 8.52.

Q. Okay. Now, just using it as an example, to a lay person, does that mean, then, it raises the risk by 3 percent? What does that mean?

A. It means it raises the risk only by 30 percent.

Q. Is it a tripling?

A. No, it's not by 30 -- it's threefold.

That's what it means.

Q. So a tripling of the risk?

A. So -- yes. I mean, exposure to glyphosate will triple the risk compared to somebody who is not exposed. So you're increasing the risk by threefold, by that number, by 3.04.

Q. Okay. And the confidence interval, you talked about some of the arbitrariness of the P values, but we've essentially selected confidence intervals to mean that we could be confident to a 95 percent degree that the true value is within this range. Is that a fair representation?

A. Yes. So 95 percent of the values fall between those two numbers.

Q. Okay. So what we can tell here when we look at the confidence intervals for glyphosate is we could be 95 percent that it goes from 1.08 to as high as 8.52; is that fair?

A. That is absolutely correct.

Q. So just in this table alone, it's possible that the real result is underreported and could be 800 percent; is that right?

A. Could be 8 -- I mean, in some -- in some folks, it could be eightfold increased risk. And the lowest it could be is 1.8 -- 1.08-fold.

Q. Okay. And I won't go through all the papers. There are many of those that we've looked at today, but those are what those bounds of the confidence interval mean when we look at individual results; is that right?

A. Yes.

Q. Okay. You talked about -- you talked about age, the increased risk of cancers with increasing age. Do you remember that discussion earlier today?

A. Yes, I do.

Q. And I think you said something along the lines of -- that one reason for that is it could be a proxy for larger cumulative exposures; is that fair?

A. Yeah.

Q. Could you explain that a little more.

A. Well, I mean, I think the -- the -- you know, as we go through life, our bodies are exposed to a variety of environmental, dietary factors, some of them that we know they are carcinogen, some of them we don't. And then, as the body ages, there are lots of cellular disruptions that occur. And when you add insult to injury, older folks become at higher risk of developing certain cancers. So, I mean, cancer ultimately is a disease of older

patients.

Q. Okay. But being 60 doesn't cause cancer. Is that a fair way to say it?

A. No, just because you're -- I think you just -- you have a higher risk just by virtue of the fact that, as you age as a person, the cellular mechanisms just are altered. So, I mean, age -- you could -- you could make a blank statement and say age is a risk factor for every single cancer under the sun, and you would be correct.

Q. But a person that's 80 has had more insults to their cells and their DNA than somebody who is 10; is that fair?

A. Right. Exactly.

Q. Okay. If age is controlled for in some of these -- we talked a lot about controls. And, again, I think we all know what we're talking about in here, but in case a lay person sees this at any point, if a epidemiological study controls for age and, say, comes up with an odds ratio of 2, that means that if you take a group of people that are the same age, they may be elderly and at increased risk, and you take a group of the same people and expose them to the agent, that they still have a doubling of the risk; is that correct?

A. Yes. If you control for the age, that's correct.

MR. GRIFFIS: Excuse me. Objection to form. Leading.

Q. Well, let me ask again a different way.

What does it mean to control for age in an epidemiological study?

A. Well, you do your best to take out age as a contributing factor for both the cases and the controls. So you want to try to eliminate age as a confounding factor so you can go back. Well, the only reason that these folks have non-Hodgkin's lymphoma is simply because they're older.

So you control for this factor so you eliminate that as a possible contribution.

Q. And is it -- controlling is -- well, let me withdraw that.

A. You do this statistically through regression modeling, where you just control for some of these factors that you can control for. I mean, age in general is easy to control for because you have it available. But there are lots of factors that you would like to control for that you can't.

Q. You talked about modifiable risks and modifiable etiologies. And, again, I want to make

sure that anybody can understand this today.

Tell me what significance a modifiable risk or etiology has to you as a clinician.

A. Well, you know, the -- you could make an argument -- and it would be a valid argument -- that the best way to actually -- that the best drug that we have ever had for cancer is smoking cessation as an example. It has had the absolute highest risk reduction possible. It is not an innovative therapy. It's very inexpensive, it's cheap, and et cetera, et cetera.

So identifying risk factors that are easy to eliminate from the environment to affected individuals is very valuable. And it's very important for us as clinicians, because you can take one factor out, and then you would reduce the risk.

And I bring tobacco as an example because it's easy to understand for a lot of people. Even in somebody who has a diagnosis of a particular cancer, and they say, "Well, I have cancer now; I can smoke all I want," the fact is if you stop smoking, you reduce the risk of a secondary cancer because now your body was more predisposed to the first one individual cancer.

So, you know, environmental factors are

important for us to identify because it helps patients at the end.

Q. And in your clinic you can recommend to patients, based on your understanding of these factors, to avoid certain ones to try to avoid recurrence or progression of the disease?

A. Absolutely.

MR. GRIFFIS: Objection. Leading.

Q. There was some brief discussion about latency. And I appreciate that -- well, it was my understanding that you said basically it depends on a lot of different factors. Is that -- is that -- let me withdraw that question.

What was your overall answer to the questions about what's a latency period for non-Hodgkin's lymphoma today?

A. As I said, I think that it is very difficult -- it's a very gray area. It is very difficult to have a binary decision on a latency period and say you have to have 10 years of exposure or 5 years of exposure or 15 years of exposure before you develop cancer. It's just not the way real life works.

So I think that latency period does exist. I think it varies between individual patients and

other contributing factors, how often they get exposed to an offending agent, et cetera.

So I don't believe short latency period or long latency period should -- should be a factor. It was not a factor in me deciding that there's a causality between glyphosate and non-Hodgkin's lymphoma.

Q. Okay. So you could find causality with a latency period of significantly shorter than ten years as well as significantly longer that ten years? Is that --

A. Absolutely.

MR. GRIFFIS: Objection. Leading.

A. And I said that previously for sure.

Q. Let's take modifiable risk factors as an example. Do you need to know the mechanism of action of those risk factors in order to apply them to your clinic?

A. No. And, in fact, there are many things that we -- you know, we told people not to smoke before we even know how in the world nicotine or tobacco cause cancer, and we probably still don't know exactly how it happens.

So I think knowing the mechanism of action is good, is nice. I think it would be nice to have

a plausible mechanism of action to better understand as a scientist. I think it's always nice. But it's not an absolute. It's not really necessary to know that.

And, similar to this, we know many drugs that work against cancer. And we don't always understand the exact mechanisms by which these drugs work against cancer. But we know from clinical trials that they do. And there is usually some basic science studies to suggest that they could work.

So I think it's nice to know some mechanism of action to have this plausibility between -- and association, but it's not mandatory to fully understand.

Q. I was going to say, do you feel comfortable prescribing drugs to cancer patients where you're not sure of the exact mechanism of action?

A. We do it all the time, as long as it's supported by clinical trials that show the activity and they're FDA approved.

Q. Let's look at -- yeah, let's look at Exhibit 6, if we can.

A. 6?

Q. Yeah. That's not right.

A. That's the monograph?

Q. No, that's not right. Let me see if I can find it.

There was --

A. Which paper?

Q. Let me see if I can find it. What do you have for 5? I don't have a 5.

A. 5 is the Greim paper, 4 is the Engels paper, and 6 is the IARC monograph.

Q. Let me see if I turned the number upside down, 7.

A. The Smith paper?

Q. Yes. And that's something you --

A. Sure.

Q. Is this something you saw for the first time today?

A. Yes.

Q. Okay. And we looked at -- it doesn't say anything about glyphosate; correct?

A. No.

Q. We looked at a bunch of different characteristics, key characteristics of carcinogens. Do you remember that and see that in that paper?

A. It's proposed characteristics.

Q. Okay.

A. These authors are trying to propose characteristics that need to be satisfied to establish carcinogenicity.

Q. Okay. And there's ten. In order for something to cause cancer, does it have to have all ten of these characteristics?

A. Nope.

Q. Okay. Are there known carcinogens that lack some of these characteristics?

A. Yes, but I can't name anything right now.

Q. Let's -- let's confine the discussion of glyphosate to . . .

Just to make a clear record, I gave you the rough draft of Dr. Neugut's deposition transcript; is that correct?

A. Yes, it was un -- it had a lot of typos.

Q. Okay. And I haven't given you the final?

A. No.

Q. You haven't reviewed that yet?

I'm sorry. I think we've harped on this a couple times today, but explain to me why we can't draw different -- why we can't draw firm conclusions about the etiologies of the various subtypes of non-Hodgkin's lymphoma from the literature that's been produced to date.

A. So it's actually very difficult because you have so many types of lymphomas. I mean, there are probably 60 types of non-Hodgkin's lymphoma that we currently are aware of. So you will have to design study of thousands of patients that -- to have sufficient numbers of every single histology to be able to demonstrate the association, causality, and statistical significance. That's one reason.

The second reason is that the types of non-Hodgkin's lymphoma have changed over the years. So the way we know -- we classify lymphoma today is very different than the way we classified lymphoma in 1995. So it depends where the study was done, it becomes very difficult to know this.

And, lastly, in a case-control study, you are relying on the answers of individuals that oftentimes they really don't know the subtypes. I mean, I have cared for thousands of patients. And they know they have lymphoma. Sometimes they don't know if it's Hodgkin, non-Hodgkin.

So there are many situations where a patient may not know that what he or she has is large-cell lymphoma, follicular lymphoma, mycosis fungoides, et cetera. So I think it becomes very difficult to establish that.

Q. And, in fact, some of the papers -- one of the papers we looked at today had only, like, eight exposed cases that were -- that were -- we were drawing conclusions off of. Do you -- is that correct?

A. I recall that. I think maybe the Eriksson paper. I don't remember which one.

Q. Okay. Certainly, in a cohort of eight, we're not going to have every subtype represented; right?

A. That's correct.

Q. Even in a cohort of 50, we're not going to be able to draw statistical conclusions about the etiologies of subtypes and how they differ; is that right?

A. Impossible. You have 60 subtypes. I mean, it's just -- it's just not -- it's not possible.

Q. Let's find the Greim paper. It's an early -- I think you found it when I gave you the wrong number a minute ago.

A. Yes, it is -- it's Exhibit 5.

Q. And there was some discussion of -- well, number one, one of these authors in the corresponding authors, you pointed out, is a vice president at Monsanto; correct?

A. I don't know his title, but he is a Monsanto employee.

Q. Okay. And you see Christian Strupp there is a member of -- do you see the 6 number by his name?

A. Yes. He is part of the glyphosate task force.

Q. Okay. And I'll represent to you that he also works for a company that makes glyphosate.

Does that -- does that further give you a grain of salt with respect to this paper?

A. Yes.

Q. I am going to represent to you that I've found the date of publication in this journal to be March 16th of 2015 in Critical Reviews of Toxicology. Assuming that this was published on March 16th, 2015, and the IARC meeting was March 3 to March 10, 2015, can you see a reason why the IARC panel did not look at this paper?

A. I can.

Q. Okay. And what would that be?

A. It wasn't available in the peer-reviewed literature at the time of the IARC meeting.

Q. Okay. Now, some things are published in advance online. There's sometimes differing

publication dates for --

A. Sometimes, yeah. It depends on the journal and the article.

Q. Okay. Some of these papers that we went through today and we looked at in your -- that were mentioned in your -- well, let me back up. The majority of the papers that you looked at were ones that you found in your own literature research; right? Not sent by me --

A. Yes.

Q. -- or my colleagues? Okay.

And you looked at a number of papers and you mentioned a number of papers that did -- were not what we would call positive papers for associations of glyphosate --

A. Correct.

Q. -- and NHL; correct.

Is that because you -- let me see. So --

A. I think it's fair to be -- to represent the evidence in its totality. I mean, I think, you know, my -- my goal, when I looked at this evidence, was not only to cite papers that were positive. I don't think it would be fair, and I wouldn't do that. I wanted to present as balanced of a review as possible and as balanced of a testimony as

possible. So I looked at all of the evidence, and I did not shy away from explicitly citing evidence that was not significant. I think it's fair.

Q. That was my question, is you didn't go out just looking for positive papers in order to form your opinion; right?

A. No. I looked at all of the papers.

Q. And I think there was some question of why did you mention some papers in your report that didn't reach positive association. Is that just in fairness or --

A. I think that is the appropriate way of reviewing the literature and looking at the literature.

MR. GRIFFIS: Objection to form. Leading.

I'm having to object after you answer, because you are answering just a little fast, sir.

Q. There was, again, a delineation made between hazard and risk and IARC and its conclusions versus what was called real-world human exposure a couple of different times.

While IARC makes its classification of whether something is of a possible or probable human carcinogen -- you're familiar with that process and

those classifications; right?
A. Yes.
Q. And they draw a line between probable and possibly; correct?
A. Yes.
Q. And they put glyphosate in that -- in that former group of probable. Okay. And in order to look -- we've called that the hazard assessment.
You're familiar with that discussion today?
A. Yeah. We -- we've had an exhaustive discussion on that.
Q. The Q&A from IARC and all that.
Now, the literature review -- there was a question about real-world human exposures and risk assessments.
Does the epidemiological literature give a feel for real-world human exposures?
A. To the extent possible, it does.
Q. I mean, that's not measuring enormous doses of some chemical in a lab; right?
A. No. They're just looking at what really happens in real life. I mean, they take cases and controls and so forth. It's not -- they're not necessarily trying to -- you can't because it's really retrospective. So you're just looking at

what's happening in the real world to the extent possible. And despite the limitations, it's a good representation in general.
Q. Okay. I think we used the terms "clinical significance" or "clinically significant" and "statistical significance" a few different points today.
Can you explain, to the best you can, what the differences between those two is for you?
A. So statistical significance is a pure number. Right? It's, you know, a P value of less than 0.05, it says that the findings are -- could be related to chance in 5 percent of the cases, but we are 95 percent certain that they are not related to chance. However, these findings may not really impact your practice. You may not find them clinically significant.
And I think for those of us who have done this for a long time are always -- can cite so many papers that show the P value of less than 0.05 that meant nothing.
A pure example was published in the New England Journal of Medicine, the most prestigious journal in the world, in a study, randomized trial, prospective trial in patients with metastatic

pancreatic cancer. They were -- they had two arms. They were compared prospectively. It was probably -- it's probably included close to 7, 800 patients. The conclusion of that paper, the experimental arm that had the novel agent improved overall survival with a P value of less than 0.0 -- less than 0.05 -- it was actually probably 0.01 -- by 1.5 weeks.
So how often do you believe this novel agent was used in real life? Not often. And I think these are examples where you can see certain things that, based on numbers -- you have enough numbers, you will see a P value less than 0.05 but may not be clinically significant.
At the same time, there are situations that you may not see that P value, you may not see the 0.05, but you see a trend, and you kind of know, if you had enough numbers, you were going to see something significant.
So if you take just a small study, 50 versus 50, and you see a trend, you will know that, if you just had hundred versus hundred, you were going to reach that P value.
So I think it's very important for us, as clinicians and researchers, not to take -- not to

just, you know, hold everything on a P value that is just a simple number, you know. And I can assure you that statisticians will have a lot of creative ways to make the P value significant. I call it funny accounting.
Q. And so -- and I think you've just answered this, but there are things that are statistically significant that are not clinically significant to you?
A. And vice versa.
Q. And vice versa. Okay.
Now, you've said either today or you've said to me at some point recently that you find positive studies to be more important than negative studies. Is that fair?
A. I think it's fair, especially in situations like this. I mean, you know, if you see -- you start -- your baseline start is a negative association. Right? So if you say that this compound is not associated with this cancer, that's really the null hypothesis, if you will. That's really where you're starting from. So if you really confirm your null hypothesis, okay, that's great.
But if you see a positive association, no matter how small it is, it is very important to

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report for two reasons. Number one, you did not know about that association before; but, number two, you have to look at the impact on a population basis.

You know -- you know, yes, your trial may have included a couple hundred patients who have had eight or ten cases. Let's multiply that now by thousands, thousands, hundreds of thousands in the U.S., outside the U.S., in Europe, in Asia, in Australia. All of a sudden you see an epidemic that is very important for us to identify.

So all of what these small studies are trying to tell us is there's something there. You better act on it before it's too late and we see more patients with this disease.

Q. I'm going to ask you a hypothetical sort of about all those numbers that we've looked at today. Imagine if we had taken away the discussion of statistical significance, those confidence intervals, and all the things that we've taken today. The vast majority of results in all of these papers today, nearly all of them were above 1; is that correct?

MR. GRIFFIS: Objection. Leading.

A. That's correct.

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Q. Okay. And so if something truly doesn't -- you are familiar with what a forest plot is?

A. I am familiar with forest plot.

Q. Okay. So in a forest plot, if you were plotting all of those results and you had a center line of 0, those will all be on the right or positive side of that line; right?

A. Yes.

MR. GRIFFIS: Objection. Leading.

Q. And if you -- if something truly didn't cause cancer at all, you'd expect to see results of -- suggesting it was cancer protective, an amount of -- first of all, it's on the left side of that line too; is that fair?

MR. GRIFFIS: Objection. Leading and foundation.

A. I'm not sure about cancer protective. I would say it would be negative association with cancer. It would be on the left side or crossing the middle -- mid line, but I wouldn't go as cancer protective.

Q. But here what we're seeing is a lot of results on the right side of the line, just not all of them reach the P value that statisticians have decided is the confidence interval that we use as a

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standard?

A. Correct.

MR. GRIFFIS: Objection. Counsel is testifying, not the witness.

Q. You said earlier today -- we were talking about different -- differing between the subtypes. You said that some types have -- their causes remain unknown or they're unknown.

Are you talking about abstract subtypes of non-Hodgkin's lymphoma, or do you mean particular clinical presentations of particular patients?

A. No. We know enough about how patients present and how to treat them and the prognosis, I think we've done a great job in understanding subtype, subtypes of lymphoma, as well as prognostication.

We do a good job in treating the disease; we can always do better. But I think I meant by saying is that we -- it's very difficult to subclassify in these studies every particular trial to go look at the subtypes. And I already, I think, outlined why that is the case.

Q. And that was -- so you said you disagreed with a quote that was read about etiological heterogeneity among NHL subtypes.

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Is that that topic that we've just discussed?

A. We discussed that. I think it's very -- you cannot just have one etiologic factor that affects one type of non-Hodgkin's lymphoma, not the other. I don't think we are able to say that at this point. There are some types -- some subtypes of non-Hodgkin's lymphoma that we can actually tell what's causing them, but we don't have that for every single subtype.

And I think, if my memory serves me right, I provided an example as HIV that causes several types of lymphoma. And there are many other examples I could give, but at the same time, we don't have causation for every type.

Q. Okay. You figured out the particular histological subtype of a person's lymphoma. Does that give you more information about its etiology as a clinician, or does that give you more information about the treatment and prognosis of that disease?

A. More, really, treatment than prognosis. Like I said, for some subtypes you can talk about the etiology and you -- you always try. You always ask questions about occupational exposure, family history, all of these things, viral association,

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et cetera. But the reality, as a clinician, this really aids more in prognosticating as well as recommending treatment option.

Q. There was a question toward the beginning -- you see I'm getting almost to the very beginning -- about what you could add to a toxicologist or an epidemiologist in terms of expertise on this issue.

You are and have been, for most of your career, a clinician; correct?

A. Yes.

Q. Okay. And what disease do you specialize in?

A. Lymphoid malignancies and a little bit of prostate cancer.

Q. Do you consider yourself an non-Hodgkin's lymphoma specialist?

A. I would say lymphoma specialist, because I do take care of Hodgkin as well. When I was at the University of Chicago, I would see close to 50 lymphoma patients a week, at least five to six new patients a week. So, I mean . . .

Q. Doctor, do you -- do you rely on epidemiology in your interpretation of it in both your clinical and your academic realms?

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A. To the extent possible. I mean, I look at the literature. I understand the literature. I'm not an epidemiologist, but I can understand epidemiology papers with -- with their limitations and their strengths. So I think I -- I rely on them somewhat. I wouldn't say they are the sole thing I rely on.

Q. Okay. At the very beginning, there was a question about when you last treated a cancer patient. I think you said it was around 11 months ago; is that correct?

A. That is correct.

Q. To be very clear, you're not offering any opinions on the standard of care of a medical oncologist; is that correct?

A. No, but I can.

Q. Let's not.

A. I -- I continue to write in the area and lecture in the area. And I -- in fact, I'm giving a big seminar on non-Hodgkin's lymphoma at the American Society of Hematology in December. It will get at least 3 to 400 people in attendance. So I continue to work in the field.

Q. Completely aside from your business practice?

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A. Yes. And a lot of the papers that I have and continue to submit are in non-Hodgkin's lymphoma and chronic lymphocytic leukemia, so . . .

Q. Doctor, we've been here now for eight hours or more, and defense counsel has given you a lot of new things and a lot of arguments.

Has anything today knocked you off your opinion that exposure to glyphosate can cause non-Hodgkin's lymphoma?

A. No.

Q. Do all the opinion -- opinions that you've put in your report here stand at the end of this deposition?

A. They do stand.

MR. LITZENBURG: Okay. I have nothing further at this time. I may have some in follow-up.

EXAMINATION

BY MR. GRIFFIS:

Q. Doctor, what will your seminar at the American Hematological Society be about?

A. Updates on lymphoma and CLL.

Q. What about? Will you be updating people on all of the important literature since --

A. I'm chairing a panel. I'm chairing a panel

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with three other people. So each one of us will actually give a talk, and I'm moderating the panel.

Q. Will you be updating the audience in the important developments in the literature over the past year, for example?

A. Yeah. Usually I try to look at the submitted abstracts to the American Society of Hematology and what's new and choose which are really more relevant factors, both in the clinical connection in what we have known and where we are going. I did that last year, and they have asked me to do it again.

Q. Okay. So it's not a seminar in any particular topic?

A. No.

Q. At this point it's more of a -- an entree and an overview to the --

A. Yeah. I'm focusing in my talk on large-cell lymphoma, but I'm also chairing and moderating the seminar with two other speakers that one of them will talk on follicular lymphoma and the other person on chronic lymphocytic leukemia.

Q. Who are the other two speakers?

A. I'll have to actually check whether I can give you the information, because the program is not

out yet.
Q. Okay.
A. So if you don't mind, I'll check with them. I don't want to -- it may not be up.
MR. LITZENBURG: Don't disclose anything that you don't know that you are able to --
Q. When is the seminar?
A. December. December 8. You're welcome to attend.
Q. A 95 percent confidence interval, sir, only means that the real value is 95 percent likely to be within that range if the data is accurate and the data is not confounded and the data is not otherwise statistically biased; correct?
A. Yes.
Q. The Greim paper that we talked about earlier, do you know, sir, that there is sworn testimony in this case that IARC is able to review unpublished articles that have been accepted for publication once they have been accepted for publication?
A. Don't know that.
Q. And the Greim had been accepted for publication for a full three months before IARC met?
A. Did not have this information.

Q. I want to mark your billing record, sir, that you were kind enough to submit to us.
(Nabhan Exhibit 24 marked for identification.)
Q. Since there are two pages, I'm using two exhibit stickers, Exhibit 24 and 25.
(Nabhan Exhibit 25 marked for identification.)
Q. And these are labeled as for the first and second quarter of 2017, sir?
A. It looks like it, yes.
Q. Had you been working on this project of assessing for plaintiffs' counsel the literature on the association or lack of association between non-Hodgkin's lymphoma and glyphosate before the first quarter of 2017?
A. I did do a little bit of work when I was first approached last summer where I did my own research to make a decision whether I would be an expert or not. I forgot. It was probably 7 to 10 hours type thing. I think it was last year in May, looks like that. But that's it.
Q. And you billed for that and were paid for that?
A. I'm pretty sure I did.

Q. You've been billing at the rate of $550 an hour, sir?
A. It's a bargain. Yes.
Q. And Innovative Oncology Consulting, which you asked Mr. Litzenburg to make the check payable to, what is that?
A. That is my -- how do I call it? I formed an LLC, but I'm the sole owner of it.
Q. That's an entity that you use to get paid through; is that right?
A. Right. I had aspirations to be a consultant and didn't -- I stuck to my decision.
Q. You're being one right now, aren't you?
A. Yes.
MR. GRIFFIS: That's all I have, thank you.
MR. LITZENBURG: This is an "I'm probably done" break, but let's have a quick break.
VIDEOGRAPHER: Going off the record at 5:44 P.M.
(Recess taken from 5:44 P.M. to 5:44 P.M.)
VIDEOGRAPHER: And we are back on the record at 5:44 P.M.
MR. LITZENBURG: And we can go off. We are finished for today. Thank you, Doc.

THE WITNESS: You're welcome.
MR. GRIFFIS: Thank you, sir.
THE WITNESS: You're welcome.
VIDEOGRAPHER: This concludes the deposition today of Dr. Chadi Nabhan. We are off the record at 5:44 P.M.
(Time noted: 5:44 P.M.)

_____
CHADI NABHAN

SUBSCRIBED TO AND SWORN BEFORE ME
THIS _____ DAY OF _____, 20__.
_____
(Notary Public) MY COMMISSION EXPIRES:_____

C E R T I F I C A T E

I, Paula Campbell, CSR, RDR, CRR, CRC, do hereby certify that on Wednesday, August 23, 2017 appeared before me, CHADI NABHAN.

I further certify that the said witness was first duly sworn to testify to the truth in the cause aforesaid.

I further certify that the signature of the witness to the foregoing deposition was not specified by counsel.

I further certify that I am not counsel for nor in any way related to any of the parties to this suit, nor financially interested in the action.

IN TESTIMONY WHEREOF, I have hereunto set my hand on this 23rd day of August, 2017.

_____

Paula Campbell, CSR, RDR, CRR, CRC
Certified Shorthand Reporter
Registered Diplomate Reporter
Certified Realtime Reporter
Certified Realtime Captioner

ERRATA SHEET FOR THE TRANSCRIPT OF:
CASE NAME: In re: Roundup Products Liability
DEPOSITION DATE: August 23, 2017
WITNESS NAME: Chadi Nabhan
Reason codes:
1. To clarify the record.
2. To conform to the facts.
3. To correct transcription errors.

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CHADI NABHAN
SUBSCRIBED TO AND SWORN BEFORE ME
THIS _____ DAY OF _____, 20__.

_____
(Notary Public) MY COMMISSION EXPIRES:_____

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