

Exhibit 4

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UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

)
IN RE: ROUNDUP PRODUCTS) MDL No. 2741
LIABILITY LITIGATION) Case No. 16-md-02741-VC

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)
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

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6
7 August 23, 2017
8 9:07 A.M.
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10
11 Videotaped discovery deposition of
12 DR. CHADI NABHAN, held at the offices of
13 CARDINAL HEALTH, 3651 Birchwood Drive,
14 Waukegan, Illinois, pursuant to notice before
15 Paula Campbell, CSR, RDR, CRR, CRC.
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1 APPEARANCES:
2 THE MILLER FIRM
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18 BY: PEARL ROBERTSON, ESQ. (telephonically)
19 ///
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24 ///
25 ///

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1 APPEARANCES:
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3 Attorneys for the Defendant Monsanto Company
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5 Washington, D.C. 20005
6 BY: KIRBY T. GRIFFIS, ESQ.
7 STEPHANIE SALEK, ESQ.
8
9
10 ALSO PRESENT:
11 Robert Zellner, Videographer
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1 ----- I N D E X -----
2
3 WITNESS EXAMINATION BY PAGE
4 DR. CHADI NABHAN MR. GRIFFIS 14, 356
5 MR. LITZENBURG 316
6
7 -----EXHIBITS-----
8 NABHAN PAGE LINE
9 Exhibit 1 Chadi Nabhan, MD, MBA, 15 12
10 FACP curriculum vitae
11 Exhibit 2 5/16/17 letter from Robin 21 13
12 Greenwald to Heather
13 Pigman
14 Exhibit 3 Expert Report of Dr. 26 11
15 Nabhan in Support of
16 General Causation on
17 Behalf of Plaintiffs
18 Exhibit 4 article entitled 30 2
19 "Comprehensive evaluation
20 of medical conditions
21 associated with risk of
22 non-Hodgkin lymphoma using
23 Medicare Claims
24 ('MedWAS')," by Engels and
25 others

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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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NABHAN	PAGE	LINE
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 norhearstern Ecuadorian border," by Paz-y-Mino, et al.	139	18
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NABHAN	PAGE	LINE
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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NABHAN	PAGE	LINE
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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-----EXHIBITS-----

	NABHAN	PAGE	LINE
1	Exhibit 16 article entitled, "Cancer	215	10
2	incidence among		
3	glyphosate-exposed		
4	pesticide applicators in		
5	the agricultural health		
6	study," by DeRoos, et al.		
7	Exhibit 17 Article entitled,	234	8
8	"Occupational exposure to		
9	pesticides and risk of		
10	non-Hodkin's lymphoma," by		
11	Fritschi, et al.		
12	Exhibit 18 Article entitled,	240	20
13	"Pesticide exposure as		
14	risk factor for		
15	non-Hodgkin's lymphoma		
16	including		
17	histopathological subgroup		
18	analysis," by Eriksson, et		
19	al.		
20	///		
21	///		
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24	///		
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-----EXHIBITS-----

	NABHAN	PAGE	LINE
1	Exhibit 19 article entitled,	265	14
2	"Occupational exposure to		
3	pesticides and lymphoid		
4	neoplasms among men:		
5	Results of a French		
6	case-control study," by		
7	Orsi, et al.		
8	Exhibit 20 article entitled,	266	9
9	"Lymphoma risk and		
10	occupational exposure to		
11	pesticides: Results of the		
12	Epilymph study," by Cocco,		
13	et al.		
14	Exhibit 21 article entitled,	271	19
15	"Pesticide product use and		
16	risk of non-Hodgkin		
17	lymphoma in women," by		
18	Kato, et al.		
19	///		
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	NABHAN	PAGE	LINE
1	Exhibit 22 article entitled,	274	9
2	"Non-Hodgkin lymphoma and		
3	occupational exposure to		
4	agricultural pesticide		
5	chemical group and active		
6	ingredients: A systematic		
7	review and meta-analysis		
8	Exhibit 23 draft paper entitled,	290	18
9	"Lymphoma risk and		
10	pesticide use in the		
11	Agricultural Health		
12	Study," by Alvanja, et al.		
13	Exhibit 24 Monsanto billing :Q1-2017	359	3
14	Exhibit 25 e-mail from Chadi Nabhan	359	7
15	to Timothy Litzenburg		
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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.

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1 VIDEOGRAPHER: Thank you. And will the
 2 court reporter --
 3 MS. ROBERTSON: Pearl Robertson with
 4 Weitz & Luxenberg for the plaintiff.
 5 REPORTER: I'm sorry. I didn't -- can you
 6 repeat?
 7 MS. TABATABAIE: Can you repeat that?
 8 MS. ROBERTSON: Yes. Pearl Robertson with
 9 Weitz & Luxenberg for plaintiff.
 10 VIDEOGRAPHER: Thank you.
 11 And will the court reporter please swear in
 12 the witness.
 13 REPORTER: Would you please raise your
 14 right hand.
 15 CHADI NABHAN,
 16 called as a witness, having been duly sworn,
 17 was examined and testified as follows:
 18 VIDEOGRAPHER: I may be picking up a cell
 19 phone in your pocket. If you have one, if you
 20 wouldn't mind putting it off to the side, as
 21 far as you can do it. Thank you so much.
 22 Thank you.
 23 EXAMINATION
 24 BY MR. GRIFFIS:
 25 Q. Good morning, sir.

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1 A. Good morning.
 2 Q. My name is Kirby Griffis, and we have just
 3 met; is that correct?
 4 A. Correct.
 5 Q. Would you please pronounce your name for
 6 the jury? I want to get it right today.
 7 A. Chadi, C-h-a-d-i, is my first name.
 8 Nabhan, N-a-b-h-a-n, is my last name.
 9 Q. Chadi Nabhan -- Nabhan?
 10 A. Correct.
 11 Q. Thank you.
 12 (Nabhan Exhibit 1 marked for
 13 identification.)
 14 Q. I've marked as Exhibit 1 and I'm handing
 15 you a copy of your current CV.
 16 Have I correctly identified that document,
 17 sir?
 18 A. Correct.
 19 Q. Okay. The "Summary" section at the top of
 20 your CV in bold says that you are vice president and
 21 chief medical officer of an \$11 billion division in
 22 a Fortune 15 company; is that right?
 23 A. Correct.
 24 Q. What is the division?
 25 A. Specialty Solutions.

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1 Q. And what is the Fortune 15 company?
 2 A. Cardinal Health. That's my current
 3 position.
 4 Q. What does -- okay. It's your current
 5 position.
 6 Has your position changed?
 7 A. No, no.
 8 Q. What is a Fortune 15 company? What does
 9 that mean?
 10 A. Fortune magazine, they have a list of the
 11 companies every year that they come up with, and
 12 they reflect 500 of the top companies in the U.S.
 13 Q. And they're top companies in what way?
 14 A. I think they have a variety of metrics.
 15 I'm not really sure what they are. I have not
 16 looked at the metrics per se that they use. But
 17 could be sales, revenue, culture, employee
 18 retention. I'm not really clear what they use.
 19 Q. Okay. The next bullet says that you're a
 20 senior level executive and a member of the operating
 21 company, reporting directly to the president;
 22 correct?
 23 A. Correct.
 24 Q. Under "Professional Experience," you list
 25 the positions that you've held in the past and

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1 currently.
 2 A. Correct.
 3 Q. This is also on the first page.
 4 And the first one is your current position
 5 that you've just been describing, vice president and
 6 chief medical officer of Cardinal Health; is that
 7 right?
 8 A. Correct.
 9 Q. And underneath that, there are 16 bullets
 10 describing your various duties as someone who
 11 reports to the president of Cardinal Health
 12 Specialty Solutions; is that right?
 13 A. Correct.
 14 Q. How much time do you spend in your current
 15 position, sir, on this job on business and
 16 administrative tasks?
 17 A. About 80 percent and 20 percent research.
 18 Q. And how much time seeing cancer patients?
 19 A. At this point, I'm not seeing patients by
 20 choice. It's been about 11 months since I've seen
 21 actual patients because of my travel schedule.
 22 I have the flexibility of having a clinic
 23 or seeing patients if I choose to. It's been very
 24 challenging with my travel to make sure that I can
 25 have a dedicated day for clinic. I don't want to

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1 shortchange my patients and cancel clinic because of
 2 short notice, so this is still in the works.
 3 Q. So at the time -- at this time, it's been
 4 11 months since you've seen a patient?
 5 A. That is correct.
 6 Q. And --
 7 A. I continue, however, to, you know, lecture,
 8 publish, and work on the field; but I have not seen
 9 an actual patient in 11 months.
 10 Q. Yes, sir.
 11 You said 20 percent of your time is on
 12 research; right?
 13 A. Correct.
 14 Q. Cardinal Health Specialty Solutions, would
 15 you describe that as a service provider to hospitals
 16 and doctors' offices?
 17 A. Hospitals, biopharma, and doctors, yes.
 18 Q. And it provides help with all sorts of
 19 logistical things with supply chains, with billing,
 20 with administration, all sorts of --
 21 A. Yeah, I mean --
 22 Q. -- difficulties?
 23 A. I think, you know, there are -- again,
 24 there are two major segments within Cardinal Health.
 25 One, the medical segment that works a lot with

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1 supply chain hospitals, and so forth. And there's
 2 the biopharma segment to work with providers as well
 3 as with biopharma, providing a lot of logistical
 4 help as well as educational platforms, helping with
 5 billing, et cetera.
 6 Q. You recently got an MBA; is that right?
 7 A. It's been a year.
 8 Q. Okay. Not recently?
 9 A. That's recent. No, it's recent, 2016.
 10 Q. And you got an MBA, I presume, in support
 11 of your current role as a business person; is that
 12 right?
 13 A. I actually decided to go back on the -- to
 14 get my MBA when I was at the University of Chicago
 15 as the director of the cancer center -- the clinical
 16 cancer center and cancer clinics. And I wanted to
 17 better understand the economics, business,
 18 accountings, which will help in my role at the time.
 19 So I got the MBA focusing on healthcare
 20 management. My goal was to help more patients at a
 21 larger scale. And, you know, this opportunity came
 22 along after the fact that I was already on the MBA.
 23 This was not -- my current role is not why I got the
 24 MBA. That is -- I got -- I went back to school in
 25 August 2014. I was still at the University of

Page 20

1 Chicago.
 2 My goal was just to better understand
 3 business of medicine. I think what's going on in
 4 medicine is very important for physicians to take
 5 lead into understanding business and the impact on
 6 patients.
 7 Q. It reflected a shift in your interest from
 8 patient care to a more broad administration and
 9 business side and serving medicine through that
 10 means. Is that fair to say?
 11 A. No, I don't think it's fair to say. I
 12 think -- I think delivering patient care is both
 13 sides, right. I mean, I think when you take care of
 14 patients in clinic, you still have to bill for
 15 services. You have to run a business.
 16 So being able to deliver quality care to
 17 patients implies that you know how to run your
 18 business.
 19 Q. Yes, sir.
 20 And you're focused now --
 21 A. So I think it's important to do both.
 22 Q. You're focused now on the business side?
 23 A. I am focused on the business side, but I
 24 don't think it's irrelevant to patient care.
 25 Q. You, sir, are not an epidemiologist, and

Page 21

1 you never were one; is that right?
 2 A. Correct.
 3 Q. You're not a toxicologist, and you never
 4 were one; right?
 5 A. Correct.
 6 Q. You don't call yourself an expert in the
 7 mechanisms of carcinogenesis; is that right?
 8 A. I'm not an expert in the mechanism of
 9 carcinogenesis. I can understand the papers that
 10 discuss carcinogenesis, and I try my best to look
 11 into how this might imply clinical decisions in
 12 clinical care.
 13 (Nabhan Exhibit 2 marked for
 14 identification.)
 15 Q. I've marked as an -- Exhibit 2 a May 16th,
 16 2017, letter from Weitz & Luxenberg to Heather
 17 Pigman at Hollingsworth, LLP, sir. And the -- I
 18 will read the letter. You will follow along with me
 19 and make sure I get it right.
 20 "Dear, Heather: To follow up on our letter
 21 dated May 3rd, 2017, and to respond to your inquiry
 22 about our expert specialties, we provide the
 23 following information."
 24 And then there is a list of six experts,
 25 including yourself, with a very brief description of

1 their specialties; is that right, sir?

2 A. Correct.

3 Q. For you, it says "oncology" and "NHL,"
4 non-Hodgkin's lymphoma; is that right?

5 A. Correct.

6 Q. Does that accurately reflect your
7 understanding of your role in this litigation?

8 A. Yes.

9 Q. And you know that there are epidemiologists
10 and toxicologists who have also been named as
11 experts for the plaintiffs; is that right?

12 A. I do.

13 Q. And what do you -- what is your
14 understanding of what you add to what the
15 epidemiologists have to say and what the
16 toxicologists have to say on the issue of whether
17 glyphosate is capable of causing non-Hodgkin's
18 lymphoma?

19 A. So I think -- I think, as somebody who took
20 care of patients with lymphomas and a variety of
21 lymphoid malignancies, it is very important to look
22 at the overall body of literature and understand
23 what might cause the disease that I'm treating.

24 A. It actually helps in a conversation with
25 patients. B. It might allow the ability to be

1 proactive into preventing additional exposure if
2 there's a particular pathogen that might actually --
3 causing an issue.

4 There's -- it's similar to when you take
5 care of a patient who is a smoker and has a
6 particular malignancy. If you reduce or stop
7 tobacco use, you will actually prevent another
8 malignancy that could occur. So actually
9 understanding the epidemiologic evidence is very
10 critical to clinical care.

11 Q. Yes, sir.

12 And you have explained, I believe, why it
13 would be important to --

14 A. Right.

15 Q. -- a cancer doctor --

16 A. Correct.

17 Q. -- to look at some of the epidemiology and
18 toxicology. My question a little bit different,
19 though.

20 It is this: With regard to the scientific
21 question of whether glyphosate causes non-Hodgkin's
22 lymphoma or is capable of causing non-Hodgkin's
23 lymphoma, once epidemiologists have spoken to that
24 subject and toxicologists have spoken to that
25 subject, what expertise do you bring, what

1 perspective do you bring to the scientific question
2 does glyphosate cause non-Hodgkin's lymphoma?

3 A. Well, number one is I could interpret the
4 evidence as well. I am very capable of looking at
5 the literature and looking at the epidemiological
6 literature. Just because I don't have an
7 epidemiology degree and -- it does not mean that I
8 cannot actually interpret the literature and look at
9 the actual evidence.

10 So I -- I will -- I form my own independent
11 review of the available literature, and I put that
12 into clinical perspective. That's what I bring to
13 the table.

14 Q. And what do you -- what can you say that an
15 epidemiologist or toxicologist cannot say?

16 A. Well, I'm not a toxicologist, as we just
17 established. I mean, a toxicologist is able to look
18 at the -- at the evidence when the product or
19 compound is going through the process of being
20 approved through toxicology assays, through animal
21 studies, et cetera.

22 I don't do that. I just look at the
23 literature and review the literature.

24 Q. You never conducted an animal cancer
25 bioassay; right?

1 A. I have not.

2 Q. You've never conducted an experimental
3 genotoxicity study; right?

4 A. I have not.

5 Q. You've never conducted a study assessing
6 the possibility that a particular chemical exposure
7 or pharmaceutical exposure or other kind of exposure
8 causes oxidative stress; is that right?

9 A. I worked -- when I was a fellow at
10 Northwestern, I worked for three years doing bench
11 work and lab work. And part of my work at the time
12 was doing certain cytotoxicity assays of particular
13 drugs to understand what they actually impact cells
14 and on cell culture.

15 So we did a lot of apoptotic assays, and so
16 forth, as part of my fellowship training. That's
17 the extent of what I did in terms of lab work.

18 I'm not sure if that answers your question.

19 Q. Yes, sir.

20 Did any of those involve -- you were
21 talking about cytotoxicity studies.

22 A. Cytotoxicity, apoptotic assays, and so
23 forth.

24 Q. Were any of those looking at reactive
25 oxidative species or other oxidative stress markers?

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

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

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

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

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1 lung cancer in general, and then you could look at
 2 other particular types.
 3 Q. Sir, I've marked as Exhibit 4 a scientific
 4 article entitled "Comprehensive evaluation of
 5 medical conditions associated with risk of
 6 non-Hodgkin lymphoma using Medicare Claims
 7 ('MedWAS')," by Engels and others.
 8 Are you familiar with this article from
 9 2016?
 10 A. I have never seen it.
 11 Q. Take a look in the "Introduction" section,
 12 sir.
 13 A. Sure.
 14 Q. The second -- the third sentence reads,
 15 "Although considered a single entity for descriptive
 16 purposes, NHL comprises a group of heterogenous
 17 subtypes with distinct clinical presentations and,
 18 as is increasingly recognized, differing causal
 19 pathways, i.e., etiologic heterogeneity."
 20 MR. LITZENBURG: I object to the
 21 questions --
 22 Q. Do you agree with that?
 23 MR. LITZENBURG: -- questions about
 24 something he's never seen before and did not
 25 rely on.

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1 With that objection, you can answer if you
 2 like.
 3 Q. Do you agree with that, sir?
 4 A. I don't. I think -- I think there are two
 5 ways of looking at things. I think sometimes
 6 certain lymphomas could have one causal factor and
 7 some others don't. So I think it's making a blank
 8 statement that takes away, frankly, from -- from the
 9 actual clinical encounters that we see with
 10 patients.
 11 Q. And this is based on clinical encounters
 12 rather than scientific literature, sir?
 13 A. And scientific literature, of course.
 14 Q. And what scientific literature says that?
 15 A. So HIV, as an example, I'll bring that,
 16 it's a known viral infection. It could cause
 17 Hodgkin lymphoma, could cause Burkitt lymphoma,
 18 could cause diffused large B-cell lymphoma. But
 19 it's one factor.
 20 So I think that you could look sometimes --
 21 the variety of lymphomas, we want causality. So you
 22 can't really make a general statement that -- that
 23 every single one is different or together.
 24 There are a variety of lymphomas, as we
 25 just talked about, the last WHO classification. And

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1 some of them could be grouped and have one causal
 2 factor or two causal factors, and some don't. There
 3 are many lymphomas we don't even know why they
 4 happen. I mean, they just happen.
 5 Q. Which lymphomas, that involve more than
 6 1 percent of the total lymphomas, do we not know why
 7 they happen?
 8 A. I don't understand the question.
 9 MR. LITZENBURG: I object to form.
 10 Q. Yes, sir.
 11 What -- which specific subtypes
 12 involving -- and I don't want a microscopic subtype
 13 with -- that's only .1 percent of all the
 14 non-Hodgkin's lymphomas.
 15 But, say, 1 percent or greater of the
 16 non-Hodgkin's lymphomas, which subtypes are unknown
 17 in their etiology?
 18 MR. LITZENBURG: Same objection.
 19 A. So it is my opinion that just because we
 20 have a patient in front of me that -- that has a
 21 lymphoma and I couldn't find an identifying factor
 22 that it occurred, it doesn't mean that there is no
 23 factor. It just means I'm not able to identify it
 24 at the time.
 25 20, 30 years ago, we only thought lymphomas

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1 were about four types. Hodgkin lymphoma was one
 2 disease. Now it's five diseases. Large-cell
 3 lymphoma was one entity. It is now about six
 4 entities.
 5 So science does evolve and does change. So
 6 I don't know today, as I sit here, what type of
 7 lymphomas we -- you have to give me a clinical case,
 8 a particular patient situation where I'll look at
 9 all the factors and I say okay, well, with this
 10 patient, I'm not sure why this lymphoma occurred.
 11 In the other patient, I may find a reason.
 12 There are about -- about close to 15,000
 13 new patients with lung cancer in the United States
 14 that are never smokers. We -- when I was in
 15 training, we had no idea, actually, why would
 16 somebody with no smoking history get lung cancer.
 17 Five, six years ago, there was a mutation
 18 that was identified that leads to a particular
 19 development of these cancers. So things evolve. I
 20 don't know -- I don't have any other answer to the
 21 question you posed.
 22 Q. Did you tell me a few minutes ago, sir,
 23 that there are some types of non-Hodgkin's lymphoma
 24 for which the cause is unknown?
 25 A. Yes, I did.

1 Q. What are those types?
 2 A. Again, any type of lymphoma, any type -- so
 3 you have 60 types of lymphoma. Any type of them,
 4 you may be able to identify why they occurred.
 5 Could be a chromosomal aberration, a genetic
 6 mutation, et cetera. And you may not be able to.
 7 Each case is different.

8 There is no particular type that you say,
 9 well, this one, I have no idea why it occurs; but,
 10 this one, I know why it occurred. In any type of
 11 lymphoma, you can't always find a predisposing
 12 factor; while in others you can. Each case is very
 13 different. I can't generalize.

14 Q. Lymphoma is very strongly associated with
 15 age; correct?

16 A. It does occur in patients who are older as
 17 opposed to younger, correct.

18 Q. Age is a major risk factor for all types of
 19 lymphoma; correct?

20 A. For all types of cancer.

21 Q. And that is because --

22 A. You don't see cancers in 30-year-olds,
 23 commonly. So I think, you know, what happens as we
 24 age is a lot of cellular disruption occurs, and you
 25 see the majority of cancers occur in patients over

1 the age of 65. The majority of cancer-related
 2 deaths occur over 65, in Medicare population.

3 Q. And that is because of the ongoing process
 4 of cell division, cell replication, and endogenous
 5 errors creeping into that process as the years pass;
 6 correct?

7 A. That's only one factor. I think there are
 8 other factors that actually are involved. As we get
 9 older, whatever things that have occurred in the
 10 past start accumulate for us. So you could smoke in
 11 your 30s all you want; you probably won't get cancer
 12 until the mid 50s.

13 The point being is certain occupational
 14 hazards, certain factors that we've done in our
 15 youth may not actually pan out until later in age.
 16 You add this to the age and cellular division and
 17 other things, so together that's really why we see
 18 most cancers diagnosed in patients over 65 and most
 19 cancer-related deaths occur in patients over 65.

20 Q. What -- what is the latency period from an
 21 environmental insult -- you mentioned smoking just
 22 now, sir -- to the manifestation of a cancer?

23 A. It varies. It varies significantly. And
 24 I'm not sure, really, anyone could be certain or
 25 accurate in saying if it's 5, 10, 15, or 20 years.

1 It's really impossible.

2 Because what happens is, in order for you
 3 to accurately determine a latency period, you are
 4 going to say that your exposure to whatever that is
 5 has to be constant and stable for all of these
 6 coming years. It has not to go up or down. What if
 7 you -- you know, you smoked one pack of cigarettes a
 8 day for 10 years and then you decide three packs of
 9 cigarettes a day for the next 10 years. Your
 10 latency changes. Your exposure changes.

11 So I don't think we can accurately predict
 12 a latency period for a malignancy that it is -- it's
 13 not a binary option. You know what I mean? It's
 14 not 5 years less or more, 10 years less or more, 15
 15 years less or more.

16 Q. Yes, sir. But when you're doing something
 17 like epidemiology and relying on statistics, what
 18 latency period would you like to see in an
 19 epidemiology study before you would consider the
 20 results to be actually reflecting a possible result
 21 of the exposure that you're looking at?

22 A. I don't rely on the latency period per se
 23 to make a decision whether there is -- the exposure
 24 has any relation to that because every disease is
 25 different.

1 So the latency period per se is not a
 2 factor, in my opinion, to make a determination in
 3 terms of exposure-related developed of disease.

4 Q. And it's not a factor in considering the
 5 adequacy of an epidemiology study, sir?

6 A. No, I didn't say that. I said it's not --
 7 you can't take it as a binary option. You can't
 8 take it as a one factor. There are a variety of
 9 factors involved in making that determination, and
 10 the latency period, in my opinion, is not the most
 11 important factor in making that determination.
 12 That's what I said.

13 Q. Okay. I'm talking about epidemiology
 14 studies right now.

15 A. Sure.

16 Q. In an epidemiology study, sir, what period
 17 of time would you like to see between the exposure
 18 under consideration and the manifestation of the
 19 diseases being measured to consider that there may
 20 be a valid relationship between the exposure and the
 21 diseases?

22 A. So I will repeat my answer, because I
 23 already answered this. I don't believe -- I don't
 24 need a minimum or a maximum. The latency period --
 25 there is no minimum or a maximum period that a

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1 latency period has to have in order for you to
 2 believe that an exposure was related to a
 3 development of disease. And I will stop at that.
 4 Q. Okay. Sir, do you know that the
 5 epidemiologists or plaintiffs in this case have
 6 criticized the agricultural health study in part for
 7 the short latency period, the -- what they call the
 8 short period of time between the exposures and the
 9 manifestation of cancer and say that's not long
 10 enough to detect cancer?
 11 MR. LITZENBURG: I object to that
 12 characterization.
 13 Go ahead.
 14 A. If he did, that's his opinion.
 15 Q. Okay. And you disagree?
 16 A. I didn't say I disagree. Again --
 17 Q. Do you agree?
 18 A. Well, if you let me just finish, what I
 19 said is that latency period -- there is no minimum
 20 or a maximum latency period that is needed for me as
 21 a clinician, as a lymphoma researcher, to determine
 22 that the exposure was related to disease. That's
 23 what I said.
 24 Q. Okay. And are you talking about in an
 25 individual patient?

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1 A. No. In any patient there is no such a
 2 thing as you have to have a minimum exposure or a
 3 maximum exposure. I mean, a latency period --
 4 you're trying to treat latency period as such a
 5 binary option that, you know, in order for you
 6 have -- you have to have a minimum latency period of
 7 5 years or 10 years or 15 years to -- to have a
 8 valid study.
 9 That's not how it works. There is no such
 10 a thing as an actual number that has to be fulfilled
 11 in order for us to buy into the results or the
 12 output of an epidemiologic study from a latency
 13 period perspective.
 14 Q. So you do not consider a short latency
 15 period to be a valid criticism of an epidemiology
 16 study looking at cancer causation. Is what that
 17 you're trying to say?
 18 A. If you are trying to equate latency period
 19 with a follow-up, you may want to clarify this
 20 because I would say follow-up, short follow-up, in
 21 any study is always something to be criticized,
 22 because you want to follow up patients longer to
 23 understand what actually happens.
 24 So maybe you want to clarify for me. If
 25 you're thinking latency as a follow-up or you're

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1 thinking -- because latency, to my understanding, is
 2 before you even started a study, before you even
 3 started the follow-up. Right? I mean, if you
 4 design a study today, in 2017, and you want to
 5 follow-up patients until 2020, the latency period
 6 would be probably since 1990, before 2017.
 7 I think the follow-up, short follow-up, is
 8 always a major criticism in any study, frankly,
 9 whether it's interventional, observational,
 10 epidemiologic, any study. And I do quite -- my
 11 share of peer review -- I peer review papers for
 12 over ten journals. So short follow-up is always a
 13 red flag for us.
 14 But if you want to clarify for me what you
 15 mean by "latency," because maybe we're mixing
 16 latency with follow-up.
 17 Q. Sir, whether you call it follow-up or
 18 whether you call it latency --
 19 A. They're different, sir. They're different.
 20 Latency is different than follow-up.
 21 Q. They're different terms in terms of the
 22 design of the study; but in either case, they refer
 23 to a period of time between the exposure --
 24 A. But that's not true.
 25 Q. -- and the manifestation of the disease;

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1 correct?
 2 A. The follow-up starts from the day you
 3 started the study. I just gave you an example. If
 4 we design a study today, in 2017, my follow-up
 5 starts in 2017.
 6 Q. And if you are looking --
 7 A. And the latency period would be probably
 8 10 years before the patients that were enrolled in
 9 2017 in the study had been exposed to for the past
 10 10 years. That's the latency.
 11 Q. And if you're looking at historical
 12 exposures 20 years old, why would it matter if you
 13 did any follow-up? If you looked at --
 14 A. Can you repeat the question?
 15 Q. Yes, sir.
 16 If you were looking at patients who were
 17 exposed 20 years ago --
 18 A. Uh-hum.
 19 Q. -- and then looking today whether they have
 20 cancer, why would it matter whether you added an
 21 additional follow-up period to that?
 22 A. Well, because, you know, with more
 23 follow-up, additional information will be generated.
 24 I mean, it's just -- this is common sense for us who
 25 do clinical research. I mean, you need longer

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1 follow-up to make sure that you separate the noise
 2 from the truth.
 3 Q. One criticism that you had of the DeRoos
 4 2005 study, the agricultural health study data, was
 5 relatively short follow-up; is that right?
 6 A. Do you mind showing me that paper?
 7 Q. Sure. You have your expert report there;
 8 right?
 9 A. Sure. It's Exhibit 3.
 10 Q. Yes.
 11 A. I reviewed a lot of papers, so sometimes a
 12 refresher will help so I could provide you with the
 13 accurate answers.
 14 Q. On page 18 of your expert report, you're
 15 talking about some findings of the EPA SAP Panel
 16 review; correct?
 17 A. Yes. I see that.
 18 Q. Yes.
 19 And about halfway down, you say, "The EPA
 20 clearly criticized the EHA publication, DeRoos,
 21 et al. 2005, for its limited follow-up period."
 22 Is that a criticism that you shared?
 23 A. Yes, I do. Like, not just with -- any
 24 study with limited follow-up, in my opinion, is
 25 always -- could be always criticized.

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1 Q. And the previous sentence says, "In fact,
 2 the panel recommended the EPA contact the HS
 3 investigators to determine whether updated data on
 4 incidents of NHL and other cancers are available."
 5 Do you see that?
 6 A. I see that.
 7 Q. And do you share the view that the HS
 8 investigators should be contacted to determine
 9 whether updated data is available?
 10 MR. LITZENBURG: Object to form.
 11 THE WITNESS: Sorry?
 12 MR. LITZENBURG: I just make objections
 13 from time to time.
 14 If you can answer it, you are welcome to.
 15 A. Okay. I apologize. Would you read the
 16 question.
 17 Q. Yes, sir.
 18 Do you share the view that you express here
 19 and attribute to the panel that the AHS
 20 investigators should be contacted to determine
 21 whether updated data on incidence of NHL and other
 22 cancers are available, i.e., updated data from the
 23 DeRoos 2005?
 24 A. I do.
 25 Q. And do you know whether such data exists?

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1 A. I don't.
 2 Q. Have you been told about a draft paper from
 3 Alavanja, et al., from 2013, sir, with updated data?
 4 A. I have not seen that paper.
 5 Q. How did you decide which epidemiology
 6 studies to look at, sir?
 7 A. Through my research, through PubMed, Google
 8 Scholar, and the literature.
 9 Q. Were you provided with epidemiology studies
 10 or other studies by plaintiffs' counsel?
 11 A. I did my own independent research. And
 12 when I had some questions, I would contact the
 13 plaintiff counsel to -- if I need to.
 14 Q. Were you given any guidance as to what
 15 additional information might exist relevant to the
 16 question that you were asked to look at, i.e.,
 17 whether NHL can be caused by glyphosate?
 18 A. No. I was provided with the -- I reviewed
 19 the deposition of the epidemiologist. I don't
 20 know -- I don't know how his last name is -- Neugut.
 21 Q. You reviewed his deposition?
 22 A. I did review it, yes.
 23 Q. Okay. And did you look at any of the
 24 studies discussed therein that you had not
 25 previously looked at?

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1 A. I don't honestly recall if I reviewed
 2 additional papers based on what he actually stated.
 3 I just -- I did not go back and look at more papers
 4 based on his deposition. I just reviewed his
 5 deposition.
 6 Q. I'd like to go back to your CV for a
 7 moment, sir.
 8 A. Sure.
 9 Q. You list a number of publications there.
 10 Did any of them involved assessing whether a
 11 particular substance causes cancer?
 12 A. A particular?
 13 Q. Particular substance?
 14 A. Causes cancer?
 15 Q. Causes cancer, yes.
 16 A. Not particularly, no.
 17 Q. Did any of your publications involve you
 18 reviewing the science and epidemiology on whether a
 19 particular substance causes cancer, i.e., for
 20 example, a review article?
 21 A. Not a particular substance. We did a lot
 22 of research through the CR database and other things
 23 to look at disparities and outcomes, and so forth,
 24 but we did not -- I did not personally review a
 25 particular substance per se.

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1 Q. Other than the work that you have done for
 2 plaintiffs' counsel in this case, have you been
 3 called upon to conduct a scientific review in the
 4 past of whether a particular substance causes
 5 cancer?
 6 A. As part of my peer review. Like I said, I
 7 review for a lot of journals and some of the
 8 manuscripts that get submitted, which I can't
 9 disclose because that's how we do peer review. So
 10 if I'm asked to review a paper, then I -- I do that.
 11 Q. Other than peer -- I'm talking about your
 12 own work, though, sir. As part of your own work,
 13 have you conducted such a study or done such a
 14 review?
 15 A. No. The only one that I just thought of --
 16 it's been a while back -- was a 2004 paper that --
 17 I'll let you know where it is -- we looked at a
 18 compound. It's a radioimmunotherapy for lymphoma,
 19 and it showed a secondary leukemia. But I'm going
 20 to tell you exactly where that is.
 21 Okay. One second. So these are the
 22 abstracts or -- these are the abstracts. Okay. So
 23 make sure I show you the . . .
 24 Well, I can't believe we didn't write this
 25 paper. This is a paper that I wrote in 2004 in

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1 Leukemia & lymphoma on the association of Zevalin,
 2 which is a radioimmunotherapy that is used for
 3 lymphoma and secondary leukemia. And I just
 4 realized it's not even put in my -- maybe -- there's
 5 no way I should have put it in -- just do one last
 6 attempt at it, because maybe you -- oh, here it is,
 7 I think, on page 12, Reference No. 65. It's
 8 actually '02.
 9 So this is a secondary acute myeloid
 10 leukemia with MLL gene rearrangement following
 11 radioimmunotherapy for non-Hodgkin's lymphoma. This
 12 is -- radioimmunotherapy is a form of treatment that
 13 we give for non-Hodgkin's lymphoma. It was
 14 associated with the secondary malignancy.
 15 So I just recall that this is one of the
 16 things that you could consider looking at in
 17 association between a particular therapy and cancer.
 18 Q. Was this a case report?
 19 A. Yes. And it was one of the few case
 20 reports that looked at particular rearrangements
 21 that we discovered after radioimmunotherapy.
 22 Q. Okay. So to sum up, then, the one
 23 publication in your CV in which you assess the issue
 24 of whether a particular substance caused a
 25 particular cancer was a case report; is that right?

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1 A. Correct.
 2 Q. And the one you just identified.
 3 And for the jury's sake, a case report is
 4 an anecdotal report by a physician or by anyone of
 5 an observation that we expose someone to this
 6 particular substance and this outcome occurred?
 7 A. Well, a case report, to be published in
 8 Leukemia & lymphoma, has to go through a strict
 9 peer-review process, and you have to -- when you say
 10 that a particular compound causes ML gene
 11 rearrangement, I had to show that the actual genes
 12 were rearranged.
 13 So it does -- while it is a case report, it
 14 does go through the same peer-review process and
 15 rigorous peer review to be published. You can't
 16 just publish any case report. I've had many case
 17 reports rejected, so it's okay.
 18 Q. Yes, sir.
 19 The new -- I mean, the new data in a case
 20 report is the observation, and it is surrounded
 21 by --
 22 A. Sure.
 23 Q. -- the scientific context, which involves
 24 research and additional --
 25 A. Correct.

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1 Q. -- writings. That's what you were just
 2 discussing; right?
 3 A. Correct.
 4 Q. Okay, sir.
 5 Would you tell me, before we turn away from
 6 your CV, sir, where else in your past publications
 7 you have used the Bradford Hill criteria?
 8 A. I have not, in my publications, used the
 9 Bradford Hill criteria.
 10 Q. Where did you get the idea to use them in
 11 your work for plaintiffs' counsel, sir?
 12 A. Repeat the question.
 13 Q. Yes, sir.
 14 Where did you get the idea to use those in
 15 your expert report in your work for plaintiffs'
 16 counsel?
 17 A. Well, when you do -- I mean, I've been
 18 spending a lot of time looking at research and
 19 reviewing the literature, so it does pop up as some
 20 of the criteria that is -- that could be used to
 21 look at causality and look at the evidence.
 22 I also forgot -- I forgot if -- I mean, I
 23 read this, I think, and, again, my memory -- I think
 24 I read that in the IARC monograph, that it was -- it
 25 was looked at, but I'll have to refresh my memory

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1 if -- if they spelled out the Bradford Hill
 2 criteria.
 3 Q. Okay. So you got the idea to use the
 4 Bradford Hill criteria as a methodology to assess
 5 causation from the articles that you found when you
 6 were looking at the issue of glyphosate and
 7 non-Hodgkin's lymphoma; is that fair?
 8 A. Not as the only methodology. I mean,
 9 you'll have -- you'll have to remember, really, that
 10 the Bradford Hill criteria or any criteria, for that
 11 matter, in medical literature is just simple
 12 guidelines tool. You have to take it in context.
 13 If you are going to just take any type of
 14 criteria and say "I'm going to follow this
 15 criteria," then a robot could do our job. It just
 16 doesn't work like this.
 17 You take the criteria, you take the
 18 guidelines, and you try to put in context into the
 19 clinical evidence that you see and see if it makes
 20 sense or not. You could disagree with some of the
 21 criteria; you could agree with some of the criteria.
 22 But all of the criteria that we have in medicine, in
 23 general there's supposed to be some guidelines that
 24 you take in context and you still use your clinical
 25 judgment. It's not to replace clinical judgment.

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1 Q. You choose -- you chose to organize your
 2 thoughts and your clinical judgment as expressed in
 3 your expert report in terms of the Bradford Hill
 4 criteria?
 5 A. I use it as part of my expert's report,
 6 that's correct.
 7 Q. And you got the idea to do that from the
 8 various -- from some of the various articles that
 9 you found in doing your research on the issue of
 10 glyphosate in non-Hodgkin's lymphoma?
 11 A. I thought it was very reasonable to apply
 12 and just see if it fits or not.
 13 Q. Okay. You had no opinion on glyphosate and
 14 non-Hodgkin's lymphoma before being retained by
 15 plaintiffs' counsel; correct?
 16 A. That is correct.
 17 Q. Turn to your expert report, please, sir.
 18 I'm on page 4. You said, "The opinions in this
 19 report are my own and are held to a reasonable
 20 degree of medical and scientific certainty."
 21 Then you said, "These opinions were formed
 22 after comprehensive review of medical literature
 23 focusing on epidemiologic studies and analyses, as
 24 well as my background, education, and experience."
 25 Would you explain, please, why your review

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1 was focused on epidemiologic studies and analyses,
 2 why you went to that part of the science.
 3 A. Well, because it's really -- it is -- in
 4 order for you to establish causality or to look at
 5 causality and between exposure to an occupational
 6 hazard or to anything that is -- you can't really
 7 have -- there would be never randomized study to say
 8 we can have a thousand patients and expose them to
 9 Compound A and a thousand patients, no exposure, and
 10 then we're going to see what happens. That clearly
 11 would be unethical and will never be done.
 12 So you really -- that's really the only way
 13 that you can go back and try to investigate the
 14 literature when you're looking at something like
 15 this.
 16 Q. And could you explain a little more why it
 17 is epidemiology that was your primary focus rather
 18 than toxicology or --
 19 A. I just did. I just said you can't --
 20 there's no prospective randomized trials that --
 21 Q. In humans?
 22 A. In humans, of course.
 23 Q. Right.
 24 A. I mean, in order for you to say that
 25 Compound A is associated with Disease B, you will

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1 need to have a randomized trial where you have a
 2 thousand patients that expose to Compound A and a
 3 thousand patients that are not exposed to Compound A
 4 and you follow it them through and see if one of
 5 them develop Disease B or not. And that will never
 6 happen in humans. It's unethical. It just didn't
 7 work like this.
 8 So in order for me to look at whether an
 9 exposure to glyphosate is -- causes non-Hodgkin's
 10 lymphoma, epidemiological studies are the ones that
 11 I have to use, just by default.
 12 Q. And what do you need to see in
 13 epidemiologic studies to conclude that a particular
 14 substance causes non-Hodgkin's lymphoma?
 15 A. I think you will need to see that the
 16 individuals, the people that were exposed to the
 17 compound in question have had increased risk of
 18 developing a particular malignancy. You want to see
 19 if there is a trend into developing this malignancy
 20 in these particular individuals, and then you look
 21 at the totality of evidence and try to form an
 22 opinion --
 23 Q. When you say --
 24 A. -- to the best of my ability.
 25 Q. Yes, sir.

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

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

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

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

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

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

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

1 of medical literature focusing on epidemiologic
 2 studies.

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

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

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

19 Q. You --

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

1 vice versa.

2 Q. Yes, sir.

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

5 A. Sorry.

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

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

14 Q. Yes, sir.

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

17 A. Correct.

18 Q. -- is that right?

19 A. Correct.

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

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

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

3 I mean, you do an interventional study and
 4 randomized trial and whatever it is. As you design
 5 the study, you establish a priori. And you say,
 6 okay, I'm going to power this study to establish
 7 statistical significance; and, based on that, I need
 8 500 patients randomized, 250 in one arm, 250 in the
 9 other arm. You establish that beforehand, and you
 10 proceed with the trial, and so forth. This is for
 11 interventional studies.

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

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

25 Q. Please explain what "confounding" is.

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1 A. Confounding --
 2 Q. Yes.
 3 A. -- factors, you mean?
 4 Q. What is --
 5 A. Confounding factors.
 6 Q. What is the concept of confounding in
 7 epidemiology and cancer epidemiology?
 8 A. That's -- the concept -- and, again, I'm
 9 not an epidemiologist, so I'll answer to the best of
 10 my ability.
 11 Q. Yes, sir.
 12 A. The confounding factors means that exposed
 13 individuals may be also exposed to additional
 14 elements or factors that may -- may impact the
 15 causation or the association of the disease in
 16 question.
 17 Q. And when it is possible to statistically
 18 control for a confounding factor, then the adjusted
 19 data is more valuable than the unadjusted data;
 20 correct?
 21 A. I think if you all -- if you can control
 22 for confounding factors, it's always -- it's always
 23 a good thing to do. You will have to control for
 24 both arms of each study -- of any study, and I --
 25 what I've seen in many of the papers -- I'm not

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1 talking about the review here but as a peer reviewer
 2 for many journals, sometimes the control doesn't
 3 happen in a balanced way between both arms.
 4 But you're correct. If you can control for
 5 confounding factors, you should at least try. You
 6 should at least attempt to do it. You sometimes
 7 can't always do it. I mean, there are certain
 8 things you just can't control for. Especially you
 9 can't, you know -- especially in epidemiology. I
 10 mean, these are not patients that are coming and
 11 seeing you in the office every week where you're
 12 taking what medicine they're taking, et cetera.
 13 But you are accurate. You are correct that
 14 you can try.
 15 Q. Sir, can you name for me three substances
 16 that are generally accepted to oncologists to cause
 17 non-Hodgkin's lymphoma for which there are
 18 epidemiology studies and those studies are negative,
 19 i.e., do not show a statistically significant
 20 association between that substance and non-Hodgkin's
 21 lymphoma?
 22 A. To my knowledge, there is no data to
 23 suggest that smoking is associated with non-Hodgkin
 24 lymphoma. And alcohol is not associated with
 25 non-Hodgkin lymphoma, to the best of my knowledge.

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1 These are substances that are commonly used
 2 by folks. While there is good evidence that smoking
 3 is associated with certain malignancies, bladder
 4 cancer and lung cancer. Certain amounts of alcohol,
 5 especially with tobacco, is associated with
 6 esophageal cancer and other things.
 7 That's the answer I have.
 8 Q. Okay. So that I understand your answer,
 9 are you saying that those are generally accepted by
 10 oncologists to cause non-Hodgkin's lymphoma?
 11 A. I said do not. I said, in my opinion --
 12 you asked me the question to provide you -- maybe we
 13 go back and repeat the question so I answer it
 14 correctly.
 15 Q. I must --
 16 A. I thought you were asking me can you -- can
 17 I give you an example of things that do not cause
 18 non-Hodgkin lymphoma.
 19 Q. Let me ask again. I must have --
 20 A. Please do.
 21 Q. -- done a bad job.
 22 Can you give me an example of three
 23 substances that are generally accepted by
 24 oncologists to cause non-Hodgkin's lymphoma for
 25 which epidemiology exists and that epidemiology is

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1 negative, i.e., does not show a statistical
 2 significance?
 3 A. I do not understand the question.
 4 MR. LITZENBURG: You can ask him to
 5 rephrase if you don't understand.
 6 Are you talking about a single paper or an
 7 entire body of epidemiology?
 8 Q. The question is -- I'm asking for three
 9 substances that oncologists generally accept to be a
 10 cause of non-Hodgkin's lymphoma in the face of
 11 negative epidemiology.
 12 A. I don't think I'm qualified to answer this
 13 question. I have to do my research. I did not do
 14 research for that topic.
 15 Q. Okay.
 16 A. But more than happy to -- you intrigued me.
 17 I'll do some research on that.
 18 Q. You can't tell me any today anyway?
 19 A. I can't.
 20 Q. Do you -- when you were discussing IARC in
 21 your expert report, you mentioned that they
 22 performed a hazard assessment; is that right, sir?
 23 A. Is there a particular page you want me to
 24 look at?
 25 Q. Never mind. It's not a question about your

1 expert --

2 A. I have it, page 16, yeah.

3 Q. Do you understand that the nature of the
4 assessment that they did was a hazard assessment and
5 not a risk assessment?

6 A. I do.

7 Q. Okay. And would you tell the jury what the
8 difference is, please.

9 A. Well, when you do a hazard assessment, you
10 look at the particular compound -- to my knowledge,
11 again -- and please recognize I'm not an
12 epidemiologist. But, to my understanding, that --
13 when you do a hazard assessment, you look at the
14 particular hazard that you are actually
15 investigating and you look at the, you know, animal
16 studies and then you look at the epidemiologic
17 evidence and try to come up with a conclusion based
18 on the available evidence.

19 When you do a risk type of an assessment,
20 you actually have more of a prospective evaluation
21 to -- that you can identify the risk easily. That's
22 my understanding.

23 Q. Okay. Have you -- have you heard it
24 described this way, sir, that a hazard assessment is
25 looking at the possibility for a substance to cause

1 exactly the levels, and so forth. It's very, very
2 difficult. It's not like a pill that you take
3 10 milligram here and 15 milligram here and you
4 really know exactly the dose. So it's just -- by
5 its nature, it's just very difficult to establish
6 that.

7 But the body of evidence suggests that the
8 current exposure, whatever that exposure may be,
9 appears to be causative of the development of
10 non-Hodgkin lymphoma.

11 Q. And you're talking about the epidemiology
12 evidence?

13 A. Yes.

14 Q. Anything else?

15 A. We talked about the fact you cannot do
16 really prospective randomization. You just can't do
17 that.

18 Q. Yes, sir.

19 Of the other studies that you discussed in
20 your expert report, are you relying on any other for
21 the conclusion that glyphosate is capable of causing
22 cancer in humans at the levels to which humans are
23 exposed?

24 A. I rely heavily on IARC. I think IARC is a
25 world authority in making a decision, whether

1 cancer, whether it is possible for a substance to
2 cause cancer at any exposure at any level; whereas,
3 a risk assessment assesses whether there is a
4 genuine risk to human health from that substance at
5 the levels at which humans will be exposed?

6 A. I have not heard this definition. I
7 apologize.

8 Q. Okay, sir.

9 Do you claim that glyphosate is a substance
10 capable of causing cancer in humans?

11 A. I looked at the evidence of non-Hodgkin
12 lymphoma. I think when you say "cancer," it's a
13 very general broad term.

14 So are you asking the question cancer or
15 non-Hodgkin lymphoma?

16 Q. Let's -- let's ask about non-Hodgkin's
17 lymphoma first.

18 A. I do.

19 Q. Do you have the opinion that it causes any
20 other kind of cancer in humans?

21 A. I did not research other kinds of cancer.

22 Q. Now, do you also hold the opinion that
23 glyphosate actually causes cancer in humans at the
24 levels at which humans are exposed to it?

25 A. I do. It's very difficult to -- to know

1 certain compounds and materials are associated or
2 causative of developing cancer and malignancy or
3 non-Hodgkin lymphoma.

4 So I think it's very important to rely
5 on -- on authority in the field. I mean, that's
6 what IARC is.

7 Q. Okay. Anything else?

8 A. I think you see that in my expert report
9 into what else I reported -- I relied on. I relied
10 on some meta-analysis that were published in some
11 epidemiologic studies. You have that.

12 Q. Okay. So the meta-analyses, the
13 epidemiologic studies, and IARC; is that right?

14 A. That is correct.

15 Q. Explain to me, sir, how it is that you
16 relied on IARC, how that formed a -- something that
17 you leaned on in your -- forming your opinion about
18 this.

19 A. I read the Lancet paper that was published.
20 I think it was Guyton, the first author. I think
21 it's '015. I read the IARC Monograph and the
22 information that they provided, and that's how I
23 relied on them.

24 Q. And did you defer to the expertise of the
25 epidemiologists and the toxicologists and the

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1 mechanism experts and the other experts who were not
 2 cancer -- cancer doctors --
 3 A. You mean the authors of the paper?
 4 Q. Yes, the authors of the paper.
 5 -- in their evaluations in forming your
 6 opinion?
 7 A. Well, you have to remember that, for a
 8 paper to get submitted and accepted in a journal
 9 like Lancet, it has gone through the utmost rigor of
 10 peer-review process. So, you know, these folks who
 11 authored this paper, the output and whatever they
 12 actually wrote has been reviewed by experts in the
 13 field in order for this to be accepted.
 14 And so I'll have to rely on this because it
 15 is not just an opinion piece that you actually
 16 write. You write the paper. You submit the
 17 evidence. And then it gets peer-reviewed by your
 18 own peers that they understand toxicology, they
 19 understand epidemiology, they understand all of
 20 these things. And then it's -- either get accepted
 21 or not accepted.
 22 So, clearly, the body of evidence was
 23 robust enough that it was accepted in a major
 24 journal like Lancet.
 25 Q. Okay. Sir, I'm talking about you and what

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1 you relied on.
 2 A. I --
 3 Q. Did you -- did you -- do you defer to the
 4 epidemiologists and the toxicologists who are
 5 involved with IARC for the opinions that they formed
 6 about glyphosate in reaching your opinions?
 7 MR. LITZENBURG: Objection. Asked and
 8 answered.
 9 THE WITNESS: Do I answer?
 10 MR. LITZENBURG: You can answer if you have
 11 anything additional.
 12 A. As I said, I'm not going to re-peer review
 13 the actual paper. I take the paper. I take the
 14 output, and I form my opinion based on the evidence.
 15 But it is not my role to perform a peer-review
 16 process and ask for original material, and so forth.
 17 I have enough evidence based on that paper.
 18 So do I defer to them and their opinions?
 19 I respect their opinions. I may agree; I may not
 20 agree with everything that a particular toxicologist
 21 or epidemiologist say, but I certainly weighed
 22 heavily on the IARC output and the IARC paper
 23 because of who IARC is and because where it was
 24 published.
 25 This was not published in a throwaway

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1 journal. This was in one of the major journals.
 2 The impact factor is top. So clearly I respect the
 3 output, and I relied very heavily on the information
 4 that were provided.
 5 Q. In the absence of the IARC report, if that
 6 had not existed and the Lancet article had not
 7 existed, would you have reached the same conclusion?
 8 A. I would have to reach my own conclusion
 9 based on the epidemiologic evidence. So it was very
 10 nice to see that my own opinion was solidified with
 11 a major organization like the IARC. So I think, you
 12 know, you have to take it both together.
 13 I can't really answer what I would have
 14 concluded had the IARC not available. That's
 15 complete speculation for me. I don't know what I
 16 would have done. IARC was part of the literature
 17 that I reviewed. So if I take the IARC away, then
 18 I'll have to go back to a different mindset and
 19 re-review everything, and I can't answer that. It's
 20 not a fair question to me.
 21 Q. So you might have come to a different
 22 conclusion?
 23 A. I didn't say that. I said I can't answer
 24 that.
 25 Q. You don't know --

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1 A. I don't know what my conclusion is. I
 2 mean, you're just taking away a paper, and say what
 3 would your conclusion be if you take away this
 4 paper. How do -- how am I supposed to answer that?
 5 I don't know.
 6 I know my opinion based on the current
 7 papers I reviewed. If you take piece of the papers
 8 I reviewed, then I'll have to re-review everything
 9 and decide whether I come to the same conclusion,
 10 different conclusion, the same conclusion.
 11 But you -- you can't just take away part of
 12 the evidence I relied on and say, what would you
 13 have concluded, because that's then a completely
 14 different case.
 15 Q. Yes. You'd have to go do the work over to
 16 know what you would come up with; right?
 17 A. Exactly.
 18 Q. The -- you know that the EPA has concluded
 19 on multiple occasions that glyphosate is not a
 20 carcinogen; correct?
 21 A. I have seen some of these reports, yes.
 22 Q. And you disagree with the EPA on that;
 23 correct?
 24 A. I think the IARC report is more convincing
 25 than the EPA conclusion, and it's more substantial.

1 Q. Why?

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

11 At the same time, there are certain
12 methodological things that I read into how the EPA
13 came to some of their conclusions that did not
14 really follow the guidelines that they should have
15 followed. There are a lot of critique from my
16 reading into the methodology that they actually used
17 was not as clean as the IARC methodology.

18 Q. Critiques written by whom?

19 A. I mean, just go on the web and research
20 EPA. It's like -- I mean, public information.

21 Q. These are critiques that were written by
22 people like Chris Portier, who is one of the members
23 of the IARC, generating criticisms of EPA and others
24 after the fact in the press; right?

25 A. I have read some of his, and I -- I mean,

1 Q. Where did you get the information that
2 there was a three-month period of reviewing the
3 literature and that all available data and evidence
4 was reviewed?

5 A. I forgot. I don't remember where I got
6 this. It's probably from the paper by Guyton,
7 et. al, in Lancet or by some of the editorials. I
8 do know these are facts. I looked it up at the
9 time.

10 Q. Do you know that Dr. Aaron Blair, who was
11 the head of the group, was deposed -- had his
12 deposition taken?

13 A. I do know he was deposed. I never read his
14 deposition.

15 Q. Do you know that he testified that the IARC
16 working group spent only one or two days total
17 assessing whether glyphosate could cause cancer?

18 A. I did not know that.

19 Q. Do you know that he testified that they
20 didn't really start work on any of the analysis
21 until they arrived in Lyon, France?

22 MR. LITZENBURG: Object to the
23 characterization.

24 A. I did not know that. Did not know.

25 Q. Okay. Does that alter your opinion about

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

7 So I'm a strong believer in evidence-based
8 and a strong believer in the peer-review process
9 and -- because it's very rigorous.

10 Q. You say in your expert report, sir, on
11 page 17 -- this is a section where you are talking
12 about IARC; correct?

13 A. I see that, yes. Excuse me.

14 Q. I'm on page 17, about two-thirds of the way
15 down the page.

16 A. Okay.

17 Q. You said, IARC -- "IARC report was
18 conceived in 2015 after an in-person meeting took
19 place between 17 experts in the field from 11
20 countries." Correct?

21 A. Uh-hum.

22 Q. And you say this meeting took place after a
23 three-months period of reviewing the literature and
24 analyzing all available data and evidence; correct?

25 A. I do, yeah.

1 the rigor of the IARC review?

2 A. No, it does not.

3 Q. Why not?

4 A. Because you could do a lot of the research
5 before you come to Lyon, France. I mean, I have
6 been there on committees where you -- you know, the
7 actual two-day meeting is to discuss what you have
8 been researching for the past few months as a
9 committee. And then you come in and you debate what
10 you actually did.

11 So the -- whether you spent two hours or
12 two days during the actual meeting does not mean
13 that you did not spend months or weeks before
14 researching the subject. This is the -- you're not
15 going to meet for three months over certain things,
16 or four months. You do the research beforehand, and
17 you say, okay, in March or April we're going to
18 meet; and whatever you've researched, we're going to
19 discuss and come up where a report. It happens all
20 the time.

21 Q. Sir --

22 A. The WHO for lymphoma, for example, the
23 publication that we just went over, the types of
24 lymphoma. This was a one-day meeting, and it was
25 published. It doesn't mean that the research was

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

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

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

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

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

18 A. One second. Yep.

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

22 A. Uh-hum.

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

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

6 Q. Well, let's see.

7 A. Sure.

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

10 A. Yes, a while back I did.

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

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

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

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

1 in this study." And then you said, "To the
2 contrary, Greim, et al., suggested lack of
3 association, "Critical Reviews of Toxicology,"
4 2015."

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

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

13 Q. Yes, sir.

14 (Nabhan Exhibit 5 marked for
15 identification.)

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

22 Correct?

23 A. I see that, yes.

24 Q. And did you read this?

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

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

5 Correct?

6 A. Correct.

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

9 A. Correct.

10 Q. Right?

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

13 MR. LITZENBURG: Object to form.

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

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

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

1 A. Well, two things. The -- if you look at
2 David Saltmiras --

3 Q. Yes, sir.

4 A. -- affiliation is Monsanto and glyphosate
5 task force. And I think I'm trying to remember
6 where I read that it is possible that he had a lot
7 of contribute -- I mean, he's a coauthor; so he, you
8 know, again, as a coauthor of the -- whether you
9 call this ghostwriting or not ghostwriting, I mean,
10 but he's a coauthor that's employed by the company
11 that makes glyphosate.

12 So I guess, you know, I mean, you'll have
13 to wonder whether the opinions in the paper were
14 fair and balanced and free of bias.

15 Q. Did you discount the opinions expressed in
16 the paper on the grounds that one of the authors was
17 employed by Monsanto?

18 MR. LITZENBURG: Objection. Asked and
19 answered.

20 A. It made me question the conclusion. I
21 think if you were me, you would probably have the
22 same question.

23 Again, you know, how likely is an employee
24 of the company that makes a compound is going to go
25 on the record in a peer review and say "The compound

1 then -- then if I already knew the conclusion,
2 nothing was shocking there. You know, it's already
3 clear where the paper will be heading.

4 Q. Sir?

5 A. Yes.

6 Q. I asked a simpler question than that.

7 This is not original research; it is a
8 summary of 14 animal studies. Correct?

9 A. Yes.

10 Q. The data tables from those 14 animal that
11 are summarized herein were available and remain
12 available online for review; correct?

13 A. Which table are you looking at?

14 Q. All of the data tables from which the
15 information in this -- come.

16 A. There's Table 1 and there's Table 2.

17 Q. I'm talking about an online annex.

18 A. Where is the online annex? I'm not sure.

19 Q. Online, sir.

20 A. Okay. Well, I'll have -- are you going to
21 show me that?

22 Q. Do you see at the back of the paper,
23 "Supplemental material available online, data
24 supplementary study 1-14"? The data is all
25 available online; right?

1 of the company that employs me causes cancer"? I
2 mean, it's probably almost going to be zero, the
3 chances are, or be fired.

4 So I think for me, you know, great. It's
5 good paper, I guess. But, I mean, I'll have to put
6 my clinician-researcher critical hat and say, I'll
7 take this with a grain of salt, whatever the output
8 is. It's very difficult for me now to assess
9 objectively the literature because I know what the
10 conclusion will be. I mean, I know the conclusion
11 will be that there is no association; otherwise, an
12 employee of the company will not be a coauthor.

13 Q. Sir, this is not original research; right?

14 It's a -- it's a --

15 A. Whatever research.

16 Q. -- summary --

17 A. Whatever it is.

18 Q. It's a summary of 14 animal studies;
19 correct?

20 A. Yeah. But you asked me whether I
21 discounted the output of the research based on the
22 coauthor, and I said it made me look at with a high
23 degree of skepticism. Because it's only fair, if I
24 already looked at the authors before I even read
25 anything, I knew what the conclusion will be. So

1 A. Right. If you want me to comment on this
2 data, I need to see it.

3 Q. Did you look?

4 A. On the -- on the supplementary data?

5 Q. Yes, sir.

6 A. I don't remember if I did.

7 Q. Do you know if any statement in the Greim
8 article is false or misrepresents in any way the
9 original data that is available online for you to
10 review?

11 A. My opinion was formed based on the fact
12 that one of the coauthors is employed by the
13 company. So I question the evidence.

14 Q. You question the accuracy of the data that
15 is published online?

16 A. I do.

17 Q. You question whether it's fraudulently
18 misrepresented?

19 A. I didn't say that.

20 MR. LITZENBURG: Object to form.

21 A. I said -- I didn't say it's fraudulent.
22 I'm not making any accusations. I said I have an
23 author on a paper that's employed by a company that
24 is making the compound in question. I think any
25 fair clinician and researcher -- you can ask a

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

5 Q. Did you?

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

8 Q. Okay.

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

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

17 A. The conclusion.

18 Q. -- from the studies.

19 A. The conclusion of the paper.

20 Q. Okay. Do you doubt the data?

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

1 whoever reviewed or whoever is involved.

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

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

12 MR. LITZENBURG: Object to form.

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

15 Q. How much weight did you give it?

16 A. I --

17 MR. LITZENBURG: Object to form.

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

25 You know, then you have meta-analysis.

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

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

11 Q. Yes, sir.

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

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

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

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

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

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

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

14 MR. LITZENBURG: Object to form.

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

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

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

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

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

15 Q. Yes, sir.

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

20 Q. And when you look at the totality of the
21 evidence through the tools of meta-analysis and
22 those results turn out to be not statistically
23 significant, what does that mean to your -- to you
24 as someone who is trying to do a causality analysis?
25

MR. LITZENBURG: Object to form.

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

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

9 A. Of course not.

10 Q. -- not statistically significant?

11 A. I just don't know.

12 Q. Yes, sir.

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

17 A. Correct.

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

20 A. No.

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

24 A. Good. Yeah.

25 Q. So that was very --

1 A. Which is a little bit unusual.

2 Q. -- clearly --

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

10 Q. Is that a sinister thing?

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

13 Q. Is it a sinister thing?

14 A. It's unusual. Why?

15 Q. Is it sinister?

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

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

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

1 author?

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

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

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

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

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

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1 means that he was not a coauthor. I meant that he
 2 probably was the most responsible author of the
 3 entire manuscript. I mean, he is the employee; he's
 4 the correspondence author; he provided all the data.
 5 Q. So ghostwriting --
 6 A. A very good question.
 7 Q. I'm sorry. Were you done?
 8 A. What I meant by "ghostwriting" is that
 9 cowrote or was an author on that paper.
 10 Q. "Ghostwriting" really isn't the right word
 11 for the situation presented by the Greim article; is
 12 that right?
 13 A. You're correct.
 14 MR. LITZENBURG: If we are done with Greim,
 15 we've been going about an hour and a half. Can
 16 we take a break?
 17 MR. GRIFFIS: Sure.
 18 VIDEOGRAPHER: Ending Disc No. 1 of the
 19 deposition of Dr. Chadi Nabhan. Off the record
 20 at 10:30 A.M.
 21 (Recess taken from 10:30 A.M. to
 22 10:44 A.M.)
 23 VIDEOGRAPHER: And beginning Disc No. 2 of
 24 the deposition of Dr. Chadi Nabhan. We are
 25 back on the record at 10:44 A.M.

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1 BY MR. GRIFFIS:
 2 Q. Sir, do you understand what it is that is
 3 contained in the Greim article, what these animal
 4 studies are?
 5 A. I did not look into each particular study
 6 by itself.
 7 Q. And do you know that this is the main body
 8 of regulatory evidence that was reviewed by the EPA
 9 and by European and other regulators in approving
 10 glyphosate as safe and effective for sale in the
 11 United States?
 12 MR. LITZENBURG: Object to form.
 13 A. I did not know that this is the sole
 14 evidence or the most important evidence that was
 15 reviewed by the EPA.
 16 Q. And, I'm sorry. With regard to the issue
 17 of carcinogenicity. Did you know that?
 18 A. I did not know that.
 19 Q. And do you know that in addition to these
 20 14 rodent studies, studies done in mice and rats
 21 that formed the basis for the conclusions by EPA and
 22 by foreign regulators that glyphosate was not a
 23 threat for human cancer, there were additional
 24 animal studies done by other registrants, other
 25 people choosing to sell generic forms of glyphosate

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1 that were also considered by EPA and by foreign
 2 regulators?
 3 A. I did not know that.
 4 MR. LITZENBURG: Object to form.
 5 Q. And do you know that EPA and foreign
 6 regulators consider long-term cancer bioassays, like
 7 the 14 described herein, critical in assessing
 8 whether a substance that's been submitted for
 9 registration review is carcinogenic?
 10 MR. LITZENBURG: Objection.
 11 A. I have not been a part of the EPA review
 12 panel or decision maker for the EPA, so I don't know
 13 what their process is.
 14 Q. And you know that IARC did not review this
 15 data in any form; correct?
 16 A. I don't know if the IARC reviewed this
 17 particular data. What I know is that the IARC
 18 concluded that there's sufficient evidence based on
 19 animal studies that there is carcinogenicity.
 20 Q. You know that IARC has a policy of not
 21 reviewing anything unpublished; correct?
 22 A. I think it's fair to review only published
 23 data.
 24 Q. And you know that none of this data was
 25 published except in the form of this article;

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1 correct?
 2 A. Again, I'll go back and say that my
 3 understanding from my review that the IARC saw
 4 sufficient evidence on animal studies that they
 5 reviewed that there's carcinogenicity. Whether they
 6 reviewed this particular paper or not, I don't know,
 7 but I know that their review collectively
 8 demonstrated that the animal studies that they
 9 looked at had sufficient evidence to establish
 10 carcinogenicity.
 11 Q. And you know that it's IARC's policy not to
 12 review unpublished studies regardless of their
 13 quality; correct?
 14 A. I think if there are studies of good
 15 quality, they should be published. So if they're
 16 not published, then why should they be reviewed?
 17 Q. Do you know that registration studies --
 18 and that's the term for studies that are performed
 19 by companies in order to secure registration -- are
 20 considered their intellectual property and that, if
 21 they were published in their entirety, then another
 22 registrant could just submit them to the EPA and get
 23 a generic form of glyphosate registered thereby?
 24 MR. LITZENBURG: Object to form.
 25 A. In my life and in -- as a

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

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

10 A. These are registration --

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

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

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

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

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

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

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

12 A. I can't speak for the --

13 Q. You would agree?

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

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

18 A. I do rely on them.

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

23 A. Right.

24 Q. -- in reaching those conclusions.

25 A. I'm not in a position to evaluate their

1 process or critique their process. I'm in the
2 position to either believe or disbelieve the output.
3 The actual process that they go by, that is
4 something you have to take on with IARC.

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

10 A. I have not --

11 MR. LITZENBURG: Objection.

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

14 Q. Would that cause you any concern?

15 A. I'll need to see the testimony.

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

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

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

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

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

16 A. Well, the IARC --

17 Q. -- is that correct?

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

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

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1 A. Why should I know? I don't know. It's
 2 not -- I mean, this is not something within scope of
 3 what I was asked to look at.
 4 Q. You don't know that as an ep- -- as an
 5 oncologist?
 6 A. I do not.
 7 Q. Now, the IARC broke up into subgroups for
 8 its review. You understand that?
 9 A. I do.
 10 Q. And one of the subgroups looked at the
 11 epidemiologic evidence; correct?
 12 A. Yes.
 13 Q. And it found that evidence to be limited;
 14 right?
 15 A. Do you have the Guyton paper with you? I
 16 mean, again, I want to --
 17 Q. I have the Monograph.
 18 A. Well, the major one, I think, that -- I
 19 know that they broke into groups and each group
 20 looked at the particular evidence and so forth. So
 21 I -- I want to make sure I answer accurately.
 22 Or the Monograph, whatever it is.
 23 Thank you.
 24 (Nabhan Exhibit 6 marked for
 25 identification.)

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1 Q. Exhibit 6, I've marked as the IARC
 2 Monograph, sir.
 3 A. Okay. Go ahead.
 4 Q. Okay. So the working group that focused on
 5 the epidemiologic evidence concluded that there was,
 6 quote, limited evidence --
 7 A. Yes.
 8 Q. -- that -- in humans -- limited evidence in
 9 humans, right, referring to the epidemiology
 10 evidence?
 11 A. Do you mind showing me which -- which part
 12 of the --
 13 Q. Yes, sir. Page 78.
 14 A. Okay.
 15 Q. The Evaluation section, 6.1, Cancer in
 16 Humans.
 17 A. Okay. I see that.
 18 Q. It says, "There is limited evidence in
 19 humans for the carcinogenicity of glyphosate." And
 20 then the specific cancer that they're talking about,
 21 they say, "A positive association has been observed
 22 for non-Hodgkin's lymphoma." Right?
 23 A. I see that, yeah.
 24 Q. Okay. Now, do you know the meaning of
 25 "limited evidence --"

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1 A. That you cannot be --
 2 Q. -- to IARC?
 3 A. -- you cannot be 100 percent certain. The
 4 only way to be 100 percent certain, as we talked
 5 about that earlier, it's a randomized controlled
 6 study. That is literally the only absolute way to
 7 be 100 percent sure. It is unethical or impossible
 8 to do.
 9 Q. And you know that IARC, when they say
 10 "limited evidence," they mean something much
 11 different than just not ruled out beyond any chance,
 12 like you were saying?
 13 A. Well, "limited evidence" means that the
 14 evidence is not -- is not certain, is not
 15 100 percent.
 16 Q. They mean something much less than that,
 17 sir. They mean -- I'll quote, "A positive
 18 association has been observed between exposure and
 19 the outcome for which a causal interpretation is
 20 credible but chance, bias, or confounding could not
 21 be ruled out with reasonable confidence."
 22 Do you understand that that's what IARC
 23 means when they say "limited evidence"?
 24 A. Now I do.
 25 Q. Do you agree with that?

1 A. Yes.

2 Q. So you agree that the epidemiology evidence
3 with regard to glyphosate and NHL is credible but
4 chance, bias, or confounding cannot be ruled out
5 without reasonable confidence; is that right?

6 A. If this is what the IARC said, then I do
7 agree with that.

8 Q. And with regard to any cancer other than
9 non-Hodgkin's lymphoma, they didn't even find
10 limited evidence; right? They found no evidence?

11 MR. LITZENBURG: Objection. Beyond the
12 scope.

13 A. Again, I -- I -- I did not really evaluate
14 what evidence they looked at outside. I mean, I
15 looked at the non-Hodgkin lymphoma.

16 Q. Okay. You're not giving the opinion that
17 glyphosate is associated with any cancer other than
18 non-Hodgkin's lymphoma; right?

19 A. I'm just talking about non-Hodgkin
20 lymphoma, correct.

21 Q. And when you say that glyphosate is
22 associated with non-Hodgkin's lymphoma, do you say
23 that it is also associated with every single subtype
24 of non-Hodgkin's lymphoma?

25 A. Yeah, I think it's -- it's -- it's very

1 difficult to establish that because of how many
2 types of lymphomas there are and also because the
3 understanding of the current classification of
4 lymphoma was not the same classification that we had
5 in the mid or late '90s, et cetera. So what we knew
6 back in the '90s about the types of lymphoma is not
7 what we know today.

8 So I think you'll have to look at
9 non-Hodgkin lymphoma as one entity when you look at
10 this causation and association.

11 Q. And you -- you're saying that we're forced
12 to look at non-Hodgkin's lymphoma as one entity
13 because we don't have much data on how glyphosate
14 might be associated or not associated with various
15 sub types?

16 A. No, for various reasons, I think. A, the
17 classification of lymphomas was different back then
18 versus now. I mean, even -- just to give you an
19 idea, the -- the 2016 classification, the earlier
20 one was '014, then was '07, and there was 1999. So,
21 again, it changes.

22 Number 2, once you actually start looking
23 at every single subtypes, the numbers become too
24 small to actually be able to detect statistical
25 significance. So when we look at causation in

1 association with occupational exposures, you'll have
2 to look at the actual entity as a whole in order for
3 you to establish this such association.

4 It's just by default. It's very difficult
5 in lymphoma because you can't have a study for 60
6 types.

7 Q. Yes, sir.

8 You are saying that, because of the
9 inadequacy of the scientific data and because of our
10 inability to distinguish between subtypes, we can
11 only form a conclusion about non-Hodgkin's lymphoma
12 as a whole; is that fair?

13 MR. LITZENBURG: Objection.

14 Mischaracterization.

15 A. Well, I didn't say inadequacy of the
16 scientific data. What I said is that lymphoma
17 classification has changed over the past 20 years.

18 So today I have 60 types. 20 years ago, I
19 had probably 10 types or 20 types. So it's very
20 difficult to look at each subtype because these
21 types and subtypes have been refined and changed in
22 classification. That's one reason.

23 Q. Yes, sir.

24 A. The number-two reason is, because of the
25 number of subtypes for lymphomas, if you want to

1 look at each one by itself, it becomes very
2 difficult in terms of statistical or clinical
3 significance.

4 Q. Yes, sir.

5 A. And the third reason is, because many of
6 these studies that are case-controlled that you're
7 asking the cases to recall what type of lymphomas
8 they had, there are many patients or many folks,
9 they don't really understand the granularity of the
10 type of lymphomas.

11 So you ask -- you know, a patient of mine,
12 they say, "I have lymphoma." They may not even know
13 it's non-Hodgkin versus Hodgkin, if it's follicular
14 or it's large cell. So I think it's just -- it's
15 not something you can actually logistically do
16 accurately.

17 Q. We don't have the information --

18 A. Or the ability.

19 Q. -- to distinguish between what association
20 glyphosate may have or may not have with each
21 subtype?

22 A. Correct.

23 Q. And so the only conclusion that we can
24 reach is about non-Hodgkin's lymphoma as a whole and
25 not about specific subtypes; is that fair?

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1 A. Yes. But that doesn't take away that it --
 2 it is associated with all other subtypes. What I'm
 3 trying to say is, just because I don't have
 4 information for each subtype, it doesn't mean that
 5 it cannot be associated with it. When you are --
 6 Q. Yes, sir.
 7 A. When you have -- when you have an
 8 association or a causation between a compound and a
 9 disease, you could be causing all of the subtypes of
 10 that disease as well. You don't really need to
 11 study each subtype.
 12 We know the association of tobacco and lung
 13 cancer. We don't need to establish this for the six
 14 subtypes of lung cancer.
 15 Q. It may be the case, sir, that glyphosate is
 16 causally associated with every subtype of
 17 non-Hodgkin's lymphoma, and it may be the case that
 18 it's only associated with some of the subtypes and
 19 we can't tell the difference?
 20 MR. LITZENBURG: Objection.
 21 A. We don't have the data today to show
 22 either/or.
 23 Q. Yes, sir.
 24 And is there any particular subtype that,
 25 in your opinion, we have the data to say

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1 non-Hodgkin's lymphoma -- that specific subtype of
 2 non-Hodgkin's lymphoma is caused by glyphosate, or
 3 can you not say that for any subtype?
 4 A. Yeah, I don't have an opinion today. What
 5 I have an opinion is that there is an association
 6 and a causation between glyphosate and non-Hodgkin
 7 lymphoma, meaning that it could actually impact all
 8 subtypes.
 9 Q. Yes, sir.
 10 You told us earlier that you read the
 11 deposition of plaintiffs' expert witness,
 12 epidemiologist Dr. Alfred Neugut; correct?
 13 A. I did.
 14 Q. And do you agree with him, sir, that the
 15 epidemiology alone is not sufficient to show a
 16 causal relationship between glyphosate and
 17 non-Hodgkin's lymphoma?
 18 A. I -- it was a 400-page. I don't remember
 19 this is exactly what he said. Do you -- I mean --
 20 Q. Do you agree with that statement?
 21 A. Say the statement again.
 22 Q. Yes, sir.
 23 "The epidemiology alone is not sufficient
 24 to show a causal relationship between glyphosate and
 25 non-Hodgkin's lymphoma."

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1 MR. LITZENBURG: Object to
 2 mischaracterization in the testimony.
 3 A. If he meant by "not sufficient" as an
 4 absolute in terms of 100 percent, then I agree. You
 5 cannot -- you will never be able to say 100 percent
 6 because of the nature of what we are talking about.
 7 But you take the epidemiologic evidence in the
 8 context of the clinical scenarios and additional
 9 information and you try to form an opinion.
 10 So I agree with the fact that you cannot
 11 take just one piece of data or one piece of
 12 information and rely solely on it. You have to rely
 13 on everything to form an educated and a
 14 comprehensive opinion.
 15 Q. When you say that there is a causal
 16 relationship between glyphosate and non-Hodgkin's
 17 lymphoma, you don't mean 100 percent; right?
 18 A. There is not 100 percent in life.
 19 Q. Okay. Well, I'm going to ask you this
 20 question again, whether you agree or disagree with
 21 this statement. And when -- when you answer it,
 22 please answer by your own standards of establishing
 23 a causal relationship, not 100 percent, but your own
 24 standards of what is sufficient to establish a
 25 causal relationship.

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1 Do you agree or disagree that the
 2 epidemiology alone is not sufficient to show a
 3 causal relationship between glyphosate and
 4 non-Hodgkin's lymphoma?
 5 A. I disagree.
 6 Q. You believe that the epidemiology alone is
 7 sufficient to show a causal relationship?
 8 A. It can be if it's strong enough,
 9 absolutely.
 10 Q. I don't mean epidemiology as an abstract.
 11 I mean the epidemiology that you looked at on this
 12 subject.
 13 A. Okay. And what do you include in
 14 epidemiology? Are you including IARC? Is that part
 15 of epidemiology?
 16 I just went to make sure -- I mean, is IARC
 17 considered epidemiology literature?
 18 Q. I don't know. Do you consider it to be an
 19 epidemiology study?
 20 A. I do. But if you don't, then I -- I want
 21 to make sure I answer the right question.
 22 Q. Okay.
 23 A. Is it the original epidemiology data? Or,
 24 I mean, the IARC, for example, took -- I mean, to me
 25 it's obviously part of epidemiology. It looked

<p style="text-align: right;">Page 110</p> <p>1 concisely at all of the literature, and they 2 generated a peer-reviewed manuscript that is easily 3 researchable and publishable and I read it. And 4 meta-analysis is part of epidemiology literature, 5 but if you're just talking about the actual paper, 6 it may be different.</p> <p>7 So in my opinion, there are -- there are 8 situations where the epidemiology literature is 9 sufficient to establish causation. And based on my 10 review of the epidemiology literature for 11 glyphosate, I see sufficient evidence to demonstrate 12 causation.</p> <p>13 Q. Okay. And when you said that sentence, 14 what did you mean by "epidemiology evidence"?</p> <p>15 A. I meant the original papers of epidemiology 16 that I reviewed, plus the IARC, plus the 17 meta-analyses, plus, you know, some of the review 18 articles that I looked at.</p> <p>19 Q. And what review articles are you talking 20 about?</p> <p>21 A. They -- they -- I mean, they -- again, they 22 were included in the -- I mean, maybe I -- 23 meta-analysis, I consider them sometimes part of 24 review because they're not original data. So the 25 meta-analysis, they're really reviewing the actual</p>	<p style="text-align: right;">Page 112</p> <p>1 conclude if I take some of the evidence that I 2 reviewed out.</p> <p>3 Q. Okay. Sir --</p> <p>4 A. It's not fair.</p> <p>5 Q. The thing about IARC is that it is not 6 original data.</p> <p>7 A. It's paper in Lancet.</p> <p>8 Q. It is not original data.</p> <p>9 A. But it's in Lancet.</p> <p>10 Q. It's a review article; right?</p> <p>11 A. It's not a review article. It's in Lancet.</p> <p>12 Have you tried publishing in Lancet? They reject 13 the 95 percent of the papers. So I think this is 14 not fair. Lancet will not accept papers unless they 15 go through peer-review process and robust evidence.</p> <p>16 So whether it's a review article, 17 meta-analysis, collection of research, it's gone 18 through the peer-review process. And there was 19 sufficient information in there to generate a 20 publication in the most prestigious, most 21 competitive journal that we have.</p> <p>22 Q. To what extent did you substitute the 23 judgment of the authors of the Lancet article for 24 your own, sir, in reviewing the original data? 25 MR. LITZENBURG: Object to form.</p>
<p style="text-align: right;">Page 111</p> <p>1 collective type of research. So I -- that's what I 2 meant by review articles per se.</p> <p>3 Q. Okay. And IARC is also a review article in 4 that sentence because it didn't generate new data?</p> <p>5 A. Yeah. No, IARC --</p> <p>6 Q. It reviewed --</p> <p>7 A. -- did not have original data. They looked 8 at the available data, and they came up with a 9 robust conclusion.</p> <p>10 Q. So when you say that the epidemiology -- in 11 your opinion, the epidemiology alone is sufficient 12 to show a causal relationship between glyphosate and 13 non-Hodgkin's lymphoma, you're including the 14 original epidemiology studies and IARC in that; 15 right?</p> <p>16 A. I am, and the meta-analysis.</p> <p>17 Q. And if you take IARC out, do you still feel 18 it's sufficient?</p> <p>19 MR. LITZENBURG: Objection. It's been 20 asked and answered this morning.</p> <p>21 A. You really can't keep asking me about 22 taking evidence out. I mean, I'm sorry, but I 23 cannot comment on -- on taking stuff out and what I 24 conclude. This is the third time. 25 I can't -- I don't know what I would</p>	<p style="text-align: right;">Page 113</p> <p>1 A. I don't re-perform a peer review. This is 2 not my job. All published papers have gone through 3 peer-review process before they get published. They 4 sometimes have three peers, four peers, four -- five 5 peers, whatever the journal policy is.</p> <p>6 And, as you know, the process, the peers 7 provide comments and they might go back and forth. 8 And sometimes papers take six months until they get 9 published.</p> <p>10 So I did not re-conduct a formal 11 peer-review process for this paper nor for all other 12 papers, frankly. I -- I look at the paper, look at 13 the evidence, and look at the totality of 14 information that's available.</p> <p>15 But this does not mean that I substituted 16 my judgment. There's a difference between you being 17 a peer reviewer for a particular paper or just 18 basically reading the paper, taking the conclusion, 19 and putting it in the context of other research 20 that's available.</p> <p>21 Q. Do you agree with Dr. Neugut that there is 22 no epidemiology study that reports a statistically 23 significant association between glyphosate and 24 non-Hodgkin's lymphoma adjusted for other pesticide 25 exposures?</p>

1 MR. LITZENBURG: Object to the
 2 mischaracterization.
 3 A. I don't remember -- do I -- I don't
 4 remember that particular statement.
 5 Q. Do you agree with the statement, whether he
 6 said it or not, then?
 7 A. That there is no positive association
 8 between --
 9 Q. There is no epidemiology study that reports
 10 a statistically significant association between
 11 glyphosate and non-Hodgkin's lymphoma once you
 12 control for other pesticide exposures.
 13 MR. LITZENBURG: Same objection.
 14 A. I -- I don't remember -- I know that not
 15 all studies were able to control for other
 16 exposures. That's for sure. It just was very
 17 difficult.
 18 I don't recall -- you know, I have to -- I
 19 wrote the few studies here to remember. I don't
 20 recall if no study has controlled for everything. I
 21 know some studies try to control and some studies
 22 did not.
 23 So I'll agree, but I will -- I have some
 24 reservation because I want to make sure I review all
 25 of these studies as well. If he said that, then,

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

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

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

<p style="text-align: right;">Page 118</p> <p>1 patients that I knew that they worked but we still 2 didn't know exactly how they actually worked. And 3 years later there was research into how this drug -- 4 why this drug was effective and so forth. 5 So it's not unusual to see that in cancer, 6 at least in my experience. 7 Q. Okay. First of all, sir, I'm going to 8 circle back to what I originally asked you, but I 9 want to ask you some other things first based on 10 your answer. 11 Do you claim, to a reasonable degree of 12 medical certainty, that oxidative stress is a 13 mechanism by which glyphosate, in fact, causes 14 non-Hodgkin's lymphoma in human beings? 15 A. It's probably one of the mechanisms. It is 16 unlikely to be the sole mechanism. There is no such 17 a thing as sole mechanism. 18 Q. Okay. Are you claiming that it is one of 19 the mechanisms? 20 A. It is likely one of the mechanisms to a 21 certain degree of medical probability. 22 Q. And do you claim that genotoxicity is one 23 of the mechanisms by which glyphosate causes 24 non-Hodgkin's lymphoma to a reasonable degree of 25 medical certainty?</p>	<p style="text-align: right;">Page 120</p> <p>1 characteristics for carcinogenesis. Okay. 2 Q. And it says here that "Not every carcinogen 3 will have all these characteristics." 4 A. Right. 5 Q. "Having a characteristic doesn't 6 necessarily mean that something is a carcinogen, but 7 we will look for these characteristics in 8 identifying carcinogens," in a nutshell? 9 A. Uh-hum. 10 Q. And you would agree with that approach. Is 11 that fair to say? Or do you not know? 12 A. No, I actually like standardization. I 13 think it's very good to have a mechanism by which 14 you look at carcinogenicity. When you standardize 15 the approach, this actually is a better way of 16 looking at things. It doesn't -- but they 17 acknowledge, obviously, that you can't meet all of 18 the criteria and so forth. But it's good -- it's 19 probably a good starting point to standardize 20 things. 21 Q. And you know that the IARC found evidence 22 for Characteristic 2, that glyphosate was genotoxic, 23 and Characteristic 5, that glyphosate induces 24 oxidative stress; correct? 25 A. Yes.</p>
<p style="text-align: right;">Page 119</p> <p>1 A. I do believe it's one of the other 2 mechanisms, yes. 3 Q. Now, you know about the Smith and Guyton 4 article on key characteristics of carcinogens, sir? 5 A. I -- can I see it? I don't know what -- 6 Q. Yes, sir. 7 (Nabhan Exhibit 7 marked for 8 identification.) 9 A. I have not reviewed that paper before. 10 Q. Well, turn, please, to page -- first of 11 all, do you see that it's written by Kathryn Guyton, 12 Christopher Portier, Ivan Rusyn, some of the other 13 people who were involved in the IARC monograph, sir? 14 A. I do see that, yeah. 15 Q. And I will tell you for your information 16 that this is a theory paper that was generated by 17 the authors here, the authors listed here, listing 18 characteristics that IARC would, in the future, look 19 for in identifying carcinogens, sir. 20 A. Okay. 21 Q. And if you turn to page 715, do you see 22 where those characteristics are listed? 23 A. I see that, yes. 24 Q. In the bold? 25 A. Uh-hum. So this is to demonstrate</p>	<p style="text-align: right;">Page 121</p> <p>1 Q. And in reading the IARC monograph, did you 2 see that they reached a conclusion as to every 3 single one of the other characteristics and said 4 that there is not -- no evidence or inadequate 5 evidence for the other characteristics? 6 A. Well, I don't know if they looked at every 7 single one, because this paper was published in 8 June '16 and the IARC paper was in '15. So I doubt 9 that they did because this appears to be 10 characteristics that they actually brought up a year 11 after the IARC was published. So I -- I doubt that 12 they did, but I -- 13 Q. Okay. They did, but let's see whether we 14 need to go through and find them all. 15 A. I don't know. Yeah, no problem. 16 Q. Do you claim that glyphosate is 17 electrophilic or can be metabolically activated to 18 electrophiles, Characteristic 1? 19 A. I'm not qualified to answer this question. 20 Q. Do you claim that it alters DNA repair or 21 causes genomic instability? 22 A. I believe there was some data that it 23 causes DNA breakage and chromosomal aberrations. So 24 I believe there is some data to that that I looked 25 at.</p>

1 Q. And do you disagree with IARC that the data
2 on that was not conclusive?

3 A. I said there is data. It may have not been
4 conclusive. But I think your question was -- I
5 think if you want to repeat the question, you said,
6 do you believe that it causes DNA repair, genomic
7 instability? I said I saw some data to that effect.

8 Q. Okay. Do you claim, to a reasonable degree
9 of medical certainty, that that is a mechanism by
10 which glyphosate causes non-Hodgkin's lymphoma?

11 A. I don't know. I don't know if it is.

12 Q. Do you claim to a reasonable degree of
13 medical certainty that inducing epigenetic
14 operations, Characteristic 4, is a mechanism by
15 which glyphosate causes non-Hodgkin's lymphoma?

16 A. I don't believe there's sufficient data to
17 look at the epigenetic alterations of glyphosate.

18 Q. Let's look at --

19 A. Just as an FYI.

20 Q. Yes, sir?

21 A. The epigenetics is not something we knew
22 about until less than ten years ago. I mean, it's
23 not something that people even know what epigenetics
24 meant.

25 So, again, this actually tells you, in '16,

1 now that we know a lot of things, let's apply our
2 knowledge and standardize how we approach things.
3 But if you ask somebody in '95 or in 2000 about
4 epigenetics, they would just say, "What is that?"

5 Q. Characteristic 6, sir. Do you claim, to a
6 reasonable degree of medical certainty, that
7 glyphosate causes non-Hodgkin's lymphoma by inducing
8 chronic inflammation?

9 A. I don't know.

10 Q. Do you claim, to a reasonable degree of
11 medical certainty, that glyphosate causes
12 non-Hodgkin's lymphoma by immunosuppression?

13 A. There's not enough data to show that.

14 Q. Or by immunomodulation, for that matter?

15 A. I have not seen sufficient data for that.

16 Q. Do you claim, sir, to a reasonable degree
17 of medical certainty, that glyphosate causes
18 non-Hodgkin's lymphoma by modulating
19 receptor-mediated effects?

20 A. Again, I have not seen data to that -- to
21 that characteristic.

22 Q. Do you claim, to a reasonable degree of
23 medical certainty, that glyphosate causes
24 non-Hodgkin's lymphoma by causing immortalization?

25 A. Can you define "immortalization" for me?

1 Because there is data on apoptosis and affecting the
2 apoptotic pathways and that glyphosate could
3 actually inhibit the ability for the cells to die.
4 And it does affect the apoptosis. So if that's what
5 they mean by immortalization, there's some data.
6 I'm not sure, again, how robust that data is there,
7 but it's there.

8 Q. Yes, sir. I mean, you'll be testifying as
9 an expert, and I'm -- this is my chance to ask you
10 questions --

11 A. But this is not my area of expertise.

12 Q. Okay. Do you claim, to a reasonable degree
13 of medical certainty, that this is a mechanism by
14 which glyphosate causes non-Hodgkin's lymphoma?

15 A. I don't know if it does.

16 Q. Do you claim, to a reasonable degree of
17 medical certainty, that glyphosate alters cell
18 proliferation, cell death, or nutrient supply and,
19 by that mechanism, causes non-Hodgkin's lymphoma?

20 A. Again, I don't -- I don't know if this is
21 the case.

22 Q. Do you claim, sir, to a reasonable degree
23 of medical certainty, that glyphosate can initiate
24 as opposed to promote cancer?

25 A. I don't think the -- I don't think that we

1 have evidence that it does one versus the other.
2 And I think it's a very gray area between initiation
3 or -- or promoting or -- or helping. It's very
4 gray. So it's not clear how it does that.

5 Again, I mean, it's -- it's -- I'll say
6 this: Not understanding the mechanism of action of
7 a particular compound, whether it works against
8 cancer or it causes cancer, is not something unusual
9 for us who have dealt with cancer for 20 years.

10 This happens all the time. I have hundreds
11 of examples I can provide in the lack of our
12 understanding of causation or mechanistic,
13 et cetera.

14 So -- so I don't know whether this is
15 something that is one of the mechanisms of action,
16 and nor is it also important to know the mechanism
17 of action. A lot of times the studies of mechanism
18 on action come after the fact, after you actually
19 show that there's a problem. Like, okay, well,
20 there's a problem. Let's try to figure out why,
21 because then we can eliminate other compounds or
22 other things that may have similar mechanisms of
23 action.

24 So, you know, I mean, to me, I don't think
25 we understand fully the mechanism of action, but we

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1 have enough evidence in terms of affecting DNA,
 2 genotoxicity, oxidative stress.
 3 So there's a plausible evidence out there
 4 that it does cause malignancy, but I don't think we
 5 have the full picture.
 6 Q. Do you know of any evidence saying that
 7 glyphosate causes -- glyphosate promotes
 8 non-Hodgkin's lymphoma as opposed to initiating
 9 non-Hodgkin's lymphoma?
 10 A. What do you mean by promote versus
 11 initiate? Just so I understand so I answer
 12 accurately. What's the difference in your mind?
 13 Q. Tell me what the difference is.
 14 A. Well, I told you I don't think there is --
 15 I think it's very gray. That's what I was just
 16 trying to say. I said that I think to try -- and,
 17 again, we always try to go back to -- you know, I
 18 don't believe you can say it's promotes versus
 19 initiate versus -- I mean, this is -- these
 20 terminologies are very vague and they're very gray.
 21 That's why, if you want an answer, I need to
 22 understand your definition.
 23 In my definition, I don't believe it
 24 matters. I don't believe there's -- I don't believe
 25 the discussion of whether it promotes or initiates

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1 or something is very fruitful at all. It doesn't
 2 really matter. It doesn't take away or add
 3 anything. I don't look at -- I don't evaluate a
 4 substance from that angle.
 5 Q. Okay, sir. You agree with me that the
 6 transformation of healthy cells into cancer cells is
 7 a multistage process?
 8 A. Yes.
 9 Q. And it involves many, many molecular
 10 transformations in the cell?
 11 A. Sometimes, yes.
 12 Q. Healthy cells are undergoing oxidative
 13 stress and DNA damage all the time without turning
 14 into cancer cells?
 15 A. If you have the proper repair mechanism,
 16 you don't always turn into cancer, that's correct.
 17 Q. Like thousands of DNA -- thousands of
 18 damages to --
 19 A. Yes. If you go in the sun -- you go in the
 20 sun, you could have oxidative stress, but it doesn't
 21 mean you're going to melanoma right away. Sometimes
 22 you have the proper repair mechanism; sometimes you
 23 don't.
 24 Q. The sun causes oxidative stress to your
 25 cells?

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1 A. Radiation. I mean --
 2 Q. Yes.
 3 A. I brought this on. I mean, radiation
 4 causes DNA damages, for example, and you can repair
 5 the DNA if you have the proper repair mechanism.
 6 Q. Another cause of oxidative stress is
 7 exercise?
 8 A. Don't know.
 9 Q. You don't know?
 10 A. That's a problem, if exercise cause
 11 oxidative stress. I don't know if exercise causes
 12 oxidative stress. Don't know that.
 13 Q. Every cell in your body is undergoing
 14 oxidative stress and dealing with that oxidative
 15 stress all the time; right?
 16 A. Yeah. I just -- you asked me if exercise
 17 induces that, and I don't know if that's the case.
 18 You're right -- your first comment is accurate.
 19 Q. Okay.
 20 A. I think cells go through oxidative stress.
 21 And sometimes you are able to repair things;
 22 sometimes you can't repair things. But I'm not sure
 23 if exercises causes oxidative stress or not.
 24 Q. Okay. So you don't know about the specific
 25 one.

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1 A. But there are certainly compounds that
 2 could increase the oxidative stress beyond the
 3 body's ability to repair, and that is where problems
 4 happen. I mean, there's -- the body is in constant
 5 balance. There's a constant balance. I mean, what
 6 cancer is at the end is cell growth -- cells grow
 7 and cells die. So if the balance shifts towards
 8 cells growing and proliferating versus cells dying,
 9 that's where tumors form and cancer develop.
 10 And so if you have oxidative stress in your
 11 body beyond your body's ability to repair things,
 12 then it could just shift that balance, and then
 13 tumors could develop.
 14 Q. At a minimum for cancer to develop, there
 15 has to be damage to DNA that is not repaired and
 16 then that is copied successfully and that is of a
 17 sort that alters the genetic machinery of the cell
 18 towards growth and --
 19 A. And proliferation.
 20 Q. -- immortalization; correct?
 21 A. Yeah. Basically, something happens where
 22 these cells continue to grow, and they grow in an
 23 exponential manner that cancer develops. Sometimes
 24 we know why they grew, because of genomic
 25 aberrations, molecular alterations, et cetera. And

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1 sometimes we don't, and we try to study why these
 2 happen.
 3 Q. And the first step in that process, the
 4 damage to the DNA, is something that happens
 5 naturally, endogenously, all the time in every cell
 6 in your body but it's repaired ordinarily; correct?
 7 A. It doesn't happen all the time. I mean,
 8 are you having DNA damage now? I mean, it doesn't
 9 happen all the time. We are not in a constant DNA
 10 damage, in a constant -- our body is not in a
 11 constant battle between cells trying to die and
 12 cells to proliferate. That's not accurate.
 13 I think, you know, there are certain
 14 environmental, certain pathogens, certain other
 15 factors that get in the body that induces oxidative
 16 stress and other mechanisms, and then the body
 17 reacts. Either you are able to repair or not. If
 18 you are able to repair, you overcome the problem.
 19 If you are not, then you shift towards cancer.
 20 But I don't think it's fair to say that, as
 21 we are sitting here, the six of us, we have DNA
 22 damage happening around the clock.
 23 (Nabhan Exhibit 8 marked for
 24 identification.)
 25 Q. Exhibit 8, sir, is an article in the

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1 journal Toxicology and Applied Pharmacology by James
 2 Klaunig, et al., entitled "Oxidative stress and
 3 oxidative damage in chemical carcinogenesis."
 4 Do you see that?
 5 A. I do see that.
 6 Q. And do you see that in the introduction
 7 section they say that the steps of cancer induction
 8 have been identified as initiation, promotion, and
 9 progression?
 10 A. That's this author's opinion, yes.
 11 Q. And you disagree with him on that?
 12 A. I don't disagree with him. But the
 13 accurate thing is that this is their opinion, not
 14 mine.
 15 Q. And on page 89, sir.
 16 A. Okay.
 17 Q. Under the heading "Oxidative DNA damage."
 18 A. Yes.
 19 Q. Do you see in the third sentence in that
 20 section that the estimated frequency of oxidative
 21 DNA damage is at 10 to the power of 4 lesions per
 22 cell per day in humans?
 23 A. I see that sentence, yes.
 24 Q. 10 to the 4 would be 10,000 lesions per
 25 cell per day oxidative damage?

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1 A. Uh-hum. I see that.
 2 Q. So that would be endogenous DNA damage to
 3 human cells caused by oxidative; correct?
 4 A. That's what they're saying.
 5 Q. And 10 -- so that would be 10,000 points of
 6 DNA damage per cell in your body every day; right?
 7 A. How are you making -- how are you doing
 8 this math, please?
 9 Q. 10 to the 4th?
 10 A. Okay. That's 10,000.
 11 Q. Yes, sir.
 12 A. Yeah, that's what they said. So 10,000
 13 lesions per cell per day in humans.
 14 Q. Right. So it is the case that, as we sit
 15 here, we're constantly undergoing oxidative DNA
 16 damage and that damage is, for the most part, being
 17 repaired; correct sir?
 18 A. Again, I don't know the reference. I'm
 19 trying to look at the references. I mean, you just
 20 gave me this paper right now that I have never seen.
 21 And I think, you know, if we -- if the discussion is
 22 about oxidative stress, then I'm sure there are
 23 papers that would debate this or not debate this and
 24 so forth.
 25 But the conclusion, what I was trying to

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1 say, is that oxidative stress is something that the
 2 human body does encounter. I don't know the
 3 frequency or what factors induces oxidative stress
 4 per se. But what I know, when certain factors that
 5 increase oxidative stress, either the body responds
 6 by countering the oxidative stress or there's no
 7 mechanism to counter the oxidative stress.
 8 That's really all I can say on the subject.
 9 I mean, I -- you know, I will have to look at these
 10 references and so forth. I don't even know who the
 11 authors are.
 12 Q. Okay, sir. So this is something you just
 13 don't know about?
 14 A. I know enough about, but you've just given
 15 me a paper that I have not seen and you're asking me
 16 to comment, and you're just giving me one sentence
 17 on page 3 that -- and you want me to comment on
 18 that. I mean, so I think -- I think there's a
 19 difference between not knowing the subject versus
 20 not knowing this paper.
 21 Q. Okay, sir. Do you know or do you not know
 22 whether every cell in your body is undergoing
 23 thousands of point damage to DNA from oxidative
 24 stress every day?
 25 MR. LITZENBURG: Objection. Asked and

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1 answered.

2 A. I think I answered that. I already

3 answered that.

4 Q. And you answered that it may be the case;

5 you don't know?

6 A. I think the answer is maybe, but there

7 are -- again, not going to repeat the same answer.

8 Q. Now, on the subject of glyphosate, sir, and

9 carcinogenesis, for purposes of this question, I'm

10 going to define "initiation" as what we've just been

11 talking about: Damage to the DNA that then may or

12 may not be repaired that may or may not cause

13 problems later. I'm going to call that initiation.

14 So do you have the opinion that glyphosate

15 causes non-Hodgkin's lymphoma other than by that, by

16 initiation, by causing initial damage to the DNA,

17 that may or may not be repaired later?

18 A. I think there are certain -- certainly,

19 there are possibilities that it might. We just

20 don't know yet. I -- you know, again -- and I am

21 more than happy to give you lots of examples, but

22 this is what we know today, and maybe in a couple

23 years there will be additional research to suggest

24 different mechanism of action by which glyphosate

25 causes non-Hodgkin's lymphoma.

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1 So I -- I don't believe -- I don't believe

2 there's any sole mechanism. I believe that we are

3 still exploring this information and there's not

4 enough data to show that this is exactly just the

5 mechanism of action by which a drug works or a

6 compound causes an occupational hazard.

7 Q. In your expert report, sir.

8 A. Sure.

9 Q. I'm on page 10.

10 A. Okay.

11 Q. In the last paragraph on that page, third

12 sentence, you say, "The U.S. EPA analyzed

13 immunotoxicity studies in mice exposed to glyphosate

14 and issued a report on February 2013, the results of

15 which were essentially negative."

16 Correct?

17 A. I see that, yes.

18 Q. And what report were you referring to

19 there, sir?

20 A. I don't remember it. I mean, I -- that's

21 what -- I mean, I -- I recall reading the EPA

22 report. I don't have it handy with me, but I'm sure

23 I can find it.

24 Q. Okay. And then you said, "These

25 observations were in contrast with other studies

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1 where lethal toxicity was demonstrated in other

2 organisms." Correct?

3 A. Yes.

4 Q. And what studies were you talking about and

5 what other organisms were you talking about?

6 A. I think what I meant by "organisms" is just

7 living cells. I mean, there are some -- some

8 studies that looked at lymphocytes -- bovine

9 lymphocytes, some studies that look at the actual

10 center or level that demonstrated some genotoxicity

11 in DNA breakage. And that's really what I was

12 referring to.

13 Q. Okay. And when you're talking about bovine

14 lymphocytes, that's a study on page 9 of your

15 report --

16 A. That's one of the studies, yeah.

17 Q. -- Sivakova. And that's an in vitro study;

18 right?

19 A. Right.

20 Q. Glyphosate was placed directly onto cow

21 lymphocyte cells; right?

22 A. Right.

23 Q. And Peluso, which you mentioned next, was

24 also an in vitro study. Glyphosate was placed

25 directly in contact with cells?

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1 A. Right.

2 Q. And then you say, "These findings are

3 critical as they have been observed in humans," and

4 you go on in the next paragraph to talk about a

5 biomonitoring -- two biomonitoring studies,

6 Paz-y-Mino and Bolognesi; right?

7 A. I do.

8 Q. And that's what you mean by they've been

9 observed in humans? You mean those two studies,

10 right, Paz-y-Mino and Bolognesi; right?

11 A. These are the ones that I found more

12 substantial.

13 Q. Okay.

14 A. So I -- I think -- you know, it's always

15 difficult to -- it's not an easy process to actually

16 demonstrate, right. I mean, you have to have the

17 occupation. Plus you have to analyze, collect

18 blood, and do all of these things.

19 Logistically, it's not always the easiest

20 thing to do. So I found these studies to be

21 interesting and informative.

22 Q. And in what way were they important to your

23 analysis?

24 A. Well, specifically the Bolognesi paper

25 where you have -- again, you saw the -- I think --

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1 I'm sure you're aware of the paper where they --
 2 they looked at the micronuclei presence in
 3 patients -- not patients -- in individuals that were
 4 exposed, and the presence of these micronuclei is a
 5 sign of genotoxicity.
 6 So they took blood samples before they
 7 spray with glyphosate, five days after, and again
 8 four months after spraying. And they discovered the
 9 micronuclei in the lymphocytes of individuals
 10 exposed to glyphosate.
 11 So at least to me this is somewhat of an
 12 evidence that the exposure to glyphosate does cause
 13 damage by the presence of these micronuclei, which
 14 is a sign of genotoxicity.
 15 Q. How reliable did you find that study to be?
 16 A. I found it to be informative.
 17 Q. What is the difference between informative
 18 and reliable, sir?
 19 A. There's really no difference. I mean,
 20 just -- I'm not -- do you have a difference? I
 21 mean, is informative different than reliable?
 22 Q. I don't -- is it to you?
 23 A. No. I want to make sure I answer the
 24 question as you -- based on your question. So to
 25 me, they're about the same. Informative and

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1 reliable is the same. But if you have a different
 2 definition, I'd like to make sure I -- I don't want
 3 to answer the wrong question.
 4 Q. You found these studies to be persuasive
 5 that glyphosate could cause non-Hodgkin's lymphoma
 6 in humans?
 7 A. I think to the -- well, no. These --
 8 genotoxicity, this is --
 9 Q. It was in support of your opinion that
 10 glyphosate --
 11 A. Well, right. Right. But I think these
 12 studies are supportive of causing genotoxicity.
 13 These studies did not necessarily talk about
 14 non-Hodgkin lymphoma. They just talk about the fact
 15 that glyphosate exposure causes genotoxicity.
 16 I found the evidence to be compelling given
 17 the difficulty in demonstrating something like this.
 18 It's not something easy to actually demonstrate. So
 19 I actually believe the authors try to do a good job
 20 in understanding whether there's any evidence of
 21 genotoxicity or not.
 22 (Nabhan Exhibit 9 marked for
 23 identification.)
 24 Q. Wait a second. Hand me that back, please.
 25 I need to mark the right one.

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1 A. (Witness complies.)
 2 Q. Okay. Sir, this is Paz-y-Mino study from
 3 2011 that you cited in your expert report --
 4 A. Yep.
 5 Q. -- on page 9.
 6 And this is a study in which various people
 7 who were undergoing aerial spraying with glyphosate
 8 near the border of Colombia were compared to some
 9 controls; is that right?
 10 A. Uh-hum. Yes.
 11 Q. And take a look at the abstract where it
 12 says towards the bottom, "In conclusion." The
 13 conclusion of the authors here was, "In conclusion
 14 the study population did not present significant
 15 chromosomal and DNA alterations."
 16 Correct?
 17 A. I see that.
 18 Q. So this was a negative study; right?
 19 A. Well, depends how you really interpret
 20 this. I mean, I think the reality is that there
 21 was -- there was evidence of chromosomal damage and
 22 DNA alterations. It did not reach statistical
 23 significance, and I think that's really different.
 24 So -- so sometimes -- you know, the fact
 25 that there was -- and we talked about this earlier.

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1 The fact that, in some of these studies, you don't
 2 have the statistical significance defined as a
 3 P value less than 0.05 could be related to the
 4 number of cases, the way the study was done. And it
 5 might be related -- it's just a number game -- the
 6 number of folks that were actually in the study.
 7 So you look at the trend and you look at
 8 the entire evidence, taken this study plus other
 9 studies involved.
 10 Q. Under "Chromosomal Analysis" on page 48,
 11 sir, it says, "After analyzing the meta-phases and
 12 karyotyping the 92 individuals who belonged to the
 13 different communities of the province of Sucumbios,
 14 located in Ecuador's northeastern border, we
 15 observed that all the analyzed women obtained a
 16 normal karyotype."
 17 Right?
 18 A. I see that, yes.
 19 Q. And there is no statistical trend, much
 20 less statistically significant association, between
 21 glyphosate exposure and genetic damage in this
 22 study; right?
 23 A. I don't see this in this paragraph. I'll
 24 have to read the whole paper, because I remember
 25 reading this paper. I'll have to reread it.

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

7 Correct?

8 A. (Speaking sotto voce.)

9 I see that, yes.

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

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

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

17 A. That's what they said, yes.

18 Q. Okay.

19 (Nabhan Exhibit 10 marked for
20 identification.)

21 Q. I'm marking as Exhibit 10 the Bolognesi
22 2009, which is the other paper that you cited in
23 your expert report on genotoxicity; correct?

24 A. Correct.

25 (Whereupon a discussion was had off the

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

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

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

11 A. Yes, I see that.

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

18 A. That's the conclusion of the authors.

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

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

1 record.)

2 BY MR. GRIFFIS:

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

8 A. Yes.

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

12 A. Which page is that?

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

15 A. I see that.

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

19 A. I see that, yes.

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

23 A. The micronuclei.

24 Q. Okay. So that's --

25 A. MN is micronuclei.

1 it's a class effect.

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

5 So, I mean, I think the dose relation, to
6 me, is not -- what I take from this paper is the
7 fact that there is evidence that there is
8 genotoxicity that is associated with this compound.
9 And it's very hard to control, when you're just
10 spraying aerially in these regions, to be
11 100 percent certain how this is -- it's very
12 difficult to -- to really control for.

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

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

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

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

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

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

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

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

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

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

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

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

17 Q. Let me ask my question again, sir.

18 A. Please.

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

24 A. I have not seen that, no.

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

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

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

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

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

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

11 (Lunch recess taken from 11:52 A.M.
12 to 12:41 P.M.)
13
14
15
16

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

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

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

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

1 AFTERNOON SESSION

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

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

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

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

14 C H A D I N A B H A N,
15 resumed and testified as follows:
16 CONTINUED EXAMINATION
17 BY MR. GRIFFIS:

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

20 A. I have not.

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

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

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

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

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

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

21 Q. Yes, sir.

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

1 the actual disease.

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

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

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

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

1 could be less, could be more.

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

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

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

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

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

25 But it would depend, I presume, based on

1 you?

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

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

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

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

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

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1 treatment. And we've demonstrated this, once you
 2 know the actual cause.
 3 What I'm saying is that not in every
 4 clinical case you are able to find that red flag
 5 that tells you aha -- that aha moment -- I think you
 6 developed non-Hodgkin's lymphoma because this is
 7 what you do for a living. I don't always have that
 8 in every single case.
 9 Q. And in which occupations do you believe
 10 that a majority of the cases are caused by an
 11 occupational exposure?
 12 MR. LITZENBURG: Object to form.
 13 A. I think there's good evidence that farmers
 14 have that. I think there is some good evidence out
 15 there that farmers have higher risk of developing
 16 non-Hodgkin's lymphoma as opposed to folks who do
 17 not work in farming.
 18 Q. Any other occupation?
 19 A. I can't recall now, but it's an interesting
 20 question that I've been interested in. I can't
 21 recall right now.
 22 Q. With regard to farmers, there was
 23 epidemiologic evidence suggesting an increased risk
 24 of non-Hodgkin's lymphoma before glyphosate was on
 25 the market; right?

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1 A. Yes, there is evidence that farmers do have
 2 increased risk of non-Hodgkin's lymphoma.
 3 Q. Separately from the existence of
 4 glyphosate; correct?
 5 A. I'm just trying to recall, because you said
 6 before the market. I'm trying to recall when
 7 that -- that --
 8 MR. LITZENBURG: Do you have epidemiology
 9 published before 1974 or data from that?
 10 A. Yeah. I'm trying to remember when did it
 11 go to market. I'm not remembering that exact.
 12 Q. In the middle of 1974.
 13 A. Yeah. So I did not review epidemiologic
 14 literature before 1974. I think the first paper I
 15 looked at was -- it's somewhere here probably by
 16 Cantor and colleagues -- was '92 paper. But, again,
 17 like we talked about, sometimes you don't have that
 18 time frame.
 19 I think there is good evidence that farmers
 20 have increased risk from an occupational perspective
 21 to developing non-Hodgkin's lymphoma. How that
 22 relates to when glyphosate was in the market and the
 23 market uptick of that compound -- because once you
 24 go 1974, maybe the market uptick is higher than '84
 25 and '94, you know. So I can't relate those

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1 together.
 2 Q. Okay. So you're not familiar with the
 3 literature on farmers and elevated risk of
 4 non-Hodgkin's lymphoma predating the existence of
 5 glyphosate in the U.S.; correct?
 6 A. I did not review epidemiologic data before
 7 1974, and I said I don't -- I don't know how fast
 8 the market uptick for glyphosate. I'm sure it's
 9 available, but I don't know how fast it got the
 10 uptick.
 11 Q. You said earlier, if I heard you correctly,
 12 that, if you find the cause of a particular case of
 13 non-Hodgkin's lymphoma, then you have the treatment?
 14 A. No. You -- no. No.
 15 Q. Maybe I heard --
 16 A. No. I said you at least start thinking,
 17 how can I develop treatment that's directed to the
 18 cause? If you -- if you know that a protein is
 19 mutated -- a gene is mutated that's causing a
 20 particular cancer, then you can develop a particular
 21 therapy against that gene or, you know --
 22 Q. I see.
 23 A. -- et cetera.
 24 Q. But if you know that it was DDT that caused
 25 the non-Hodgkin's lymphoma, that doesn't give you

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1 any clues about how to treat it; right?
 2 A. Well, I would eliminate the cause. Right?
 3 It's like smoking. If you know that smoking causes
 4 cancer, you just say stop smoking. So I would stop
 5 using the causative factor. That's the easiest
 6 thing of prevention.
 7 Q. You're not going to cure them, though?
 8 A. Some lymphomas are curable, not all
 9 lymphoma -- I mean, lymphoma, like we say, it's 60
 10 types of lymphomas. In fact, one of the rewarding
 11 things in lymphoma, that we cure some of these
 12 lymphoma. We cure many lymphoma. Depend how you
 13 define "many," but we do cure some lymphoma.
 14 Q. Do you believe that the majority of cases
 15 of non-Hodgkin's lymphoma would not have occurred
 16 but for an environmental exposure?
 17 A. I don't believe that. I think that there
 18 are not -- not only environmental exposures cause
 19 non-Hodgkin's lymphoma. We talked about viral
 20 association. We talked about environmental factors.
 21 And we talked about the fact that we may not
 22 understand completely and fully all of the causation
 23 for non-Hodgkin's lymphoma.
 24 Q. Do you have an opinion as to relative
 25 prevalences of -- of heredity -- i.e., genetic

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

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

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

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

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

25 Q. Environmental.

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

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

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

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

1 A. Yes. I think there is --

2 Q. And --

3 A. Yeah.

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

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

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

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

21 Q. -- in causation?

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

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

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

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

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

19 A. Yes.

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

25 A. Yes.

<p style="text-align: right;">Page 166</p> <p>1 Q. Sir, did you put these studies in any 2 particular order? 3 A. I don't remember. I may have tried to put 4 them in the order of the years that were published. 5 I think that's what I tried to do. I think 6 McDuffie's '01. Then you have Hardell '02. DeRoos 7 '03. I may have tried to do that. I like to do 8 that chronologically. It's possible that's what I 9 did. 10 Q. Okay. 11 A. But it doesn't mean -- I did not order them 12 by importance, if that's the question. 13 Q. Okay. 14 (Nabhan Exhibit 11 marked for 15 identification.) 16 Q. I've handed you a copy, sir, of the 17 McDuffie 2001 paper. 18 A. Okay. 19 Q. Now, this was a study of herbicides and 20 pesticides in general and their association 21 non-Hodgkin's lymphoma; correct? 22 A. Correct. 23 Q. It was not focused specifically on 24 glyphosate; right? 25 A. It was on -- it was in general, but I think</p>	<p style="text-align: right;">Page 168</p> <p>1 second column. 2 A. Yeah. I just saw that they looked at the 3 glyphosate on page 1158. It shows an odds ratio of 4 1.26. 5 Q. Yes, sir. It's mentioned. 6 A. Right, right. I just -- initially, I said 7 I didn't -- I didn't know. So 1161? 8 Q. 1161, second column. 9 A. Okay. 10 Q. They say, "We reported results for a number 11 of chemical agents and exposures, not all of which 12 were specified in the hypothesis. Therefore, the 13 statistical analyses related to these unspecified 14 agents should be considered exploratory. As a 15 consequence of conducting multiple comparisons, a 16 small number of statistically significant results 17 may be attributable to chance." 18 I read that correctly? 19 A. You did. 20 Q. Would you explain to the jury what concept 21 they're talking about where, when you do statistical 22 analyses on many different chemicals simultaneously, 23 you will get potentially, apparently, significant 24 results only due to chance? 25 A. I mean, first, I can't speak for the</p>
<p style="text-align: right;">Page 167</p> <p>1 they had -- I'm trying to see if they subanalyze 2 glyphosate. I think it was for general exposure, to 3 my knowledge. 4 Q. When they are describing the questionnaires 5 that they sent out on page 1156, second column -- 6 A. Uh-hum. 7 Q. -- the specific exposures that they talk 8 about were first major classes, herbicides. I'm at 9 the end of that first paragraph. 10 A. Okay. 11 Q. Chemical groups and the example they give 12 is phenoxy herbicides and finally to individual 13 compounds, 2,4-D MCPA, and 2,4,5-T. In their 14 description of the initial hypotheses, they didn't 15 specifically mention glyphosate; right? 16 A. That's correct. 17 Q. I'm sorry? 18 A. That's correct, I said. 19 Q. Yeah, I thought Mr. Litzenburg said 20 something. 21 And the authors noted that, because they 22 were looking at results for multiple chemical agents 23 and exposures that weren't specifically set out in 24 the hypothesis, the statistical analyses should be 25 considered exploratory; right? That's on page 1161,</p>	<p style="text-align: right;">Page 169</p> <p>1 authors. I only can speculate. I think it's really 2 fair, if you really want to know what they actually 3 meant, to direct that question to them. 4 But what I would say is oftentimes, if you 5 have a study that is looking at multiple 6 occupational hazards or occupational exposures, 7 there are limitations to how much you can control 8 for these additional occupational hazards in order 9 for you to tease out the impact of one particular 10 compound versus another. 11 So I think they're leaving just some open 12 room, which is appropriate, to say, okay, well, you 13 know, these results are important, but they have to 14 be taken in context. Additional studies are needed, 15 and there may be some we cannot be 100 percent 16 conclusive that this is not related to chance. So 17 that's why we can't really take one study alone and 18 we have to look at all of these studies that were 19 done. 20 Q. For example, sir, if you're using a 21 95 percent confidence interval and -- confidence 22 level, rather, and you looked at 20 different 23 compounds, you would expect to find at least one 24 statistically significant association solely due to 25 chance; right?</p>

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

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

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

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

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

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

7 Q. Let's look --

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

10 Q. Table 2.

11 A. Okay.

12 Q. Under Table 2, "Glyphosate" --

13 A. Uh-huh.

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

16 A. Uh-hum.

17 Q. And they give a 95 percent confidence
18 interval.

19 A. I see that.

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

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

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

6 A. If you're --

7 Q. Do you --

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

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

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

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

1 do these studies.

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

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

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

21 At some point, statisticians and
22 researchers have to agree on what is that point that
23 we are allowing chance to play a factor, and they
24 agreed on 5 percent. They could have done
25 4 percent. They could have done 6 percent. But

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

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

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

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

16 MR. GRIFFIS: Okay.

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

19 VIDEOGRAPHER: Going off the record at
20 1:11 P.M.

21 (Recess taken from 1:11 P.M. to
22 1:15 P.M.)

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

1 BY MR. GRIFFIS:

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

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

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

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

15 A. Yes.

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

18 A. I can only speak for myself.

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

23 Why have they done so?

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

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

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

11 But you can't -- as a clinician, I can show
12 you many papers that showed a P value of less than
13 0.05 that meant nothing, that showed an improvement
14 in treatment of 1.5 weeks. Does this mean how
15 clinically meaningful it was? It was great paper.
16 It was New England Journal of Medicine paper,
17 P value less than 0.05. It was in pancreas cancer,
18 but the actual difference between the actual
19 treatment and control was 1.5 weeks.

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

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

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

3 MR. LITZENBURG: Objection. Asked and
4 answered.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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1 proven before you even start doing his causality
 2 analysis?
 3 MR. LITZENBURG: Object to form.
 4 A. I hope not. I don't believe that's what he
 5 meant, because I think he would probably know better
 6 that there is no such a thing as clear-cut. So I
 7 don't believe this is what he meant.
 8 I think what he meant is that there is
 9 enough evidence out there to prove the association
 10 and causality between two variables. I mean,
 11 "clear-cut," again, it's a vague term. To some
 12 people, it means 100 percent certainty; others,
 13 90 percent; and others, 50.1 percent. So I don't
 14 know what he meant by this.
 15 Q. And do you know that most epidemiologists
 16 consider it to be a statistically significant
 17 association in a reliable study?
 18 MR. LITZENBURG: I object to that
 19 characterization.
 20 A. The Bradford Hill?
 21 MR. LITZENBURG: Object to form.
 22 Q. Yes.
 23 A. I know that they used the Bradford Hill
 24 criteria to the extent possible, but I also know it
 25 is not used in absolute terms. I mean, you can't --

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1 again, you try -- you have to have certain --
 2 certain criteria or certain guidelines in order to
 3 compare apples to apples, but I don't believe any
 4 epidemiologist is going to tell you that we use this
 5 exclusively and with 100 percent certainty.
 6 Q. The next statistic that you quote in your
 7 expert report from the McDuffie paper is an odds
 8 ratio, which you called statistically significant at
 9 2.12, 1.2 to 3.73 confidence interval.
 10 And that comes from Table 8 of the
 11 McDuffie paper, sir. Would you take a look at
 12 that.
 13 A. I see Table 8.
 14 Q. When we were looking at the not
 15 statistically significant association on Table 2,
 16 you looked for me and saw that the odds ratio that
 17 was reported there had been adjusted for various
 18 statistically significant medical variables and with
 19 the variables of age and province of residence;
 20 correct?
 21 A. Yes.
 22 Q. And here they did not adjust for even the
 23 medical variables; right?
 24 A. I'm not sure that's accurate. If they have
 25 adjusted on the other one, they have adjusted for

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

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

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

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1 days per year.
 2 A. Do you mind telling me where you read that
 3 in that paper?
 4 Q. Greater than zero and less than or equal to
 5 2. It's in the days per year column on Table 8,
 6 among other places.
 7 A. Oh, Table 8. I see. I'm reading in the
 8 methods.
 9 So they say here, "Each subject will report
 10 ten hour per year or more of exposure to pesticides
 11 as defined by the screening questions, and a
 12 15 percent random sample of the remainder was mailed
 13 a list of pesticides in an information letter." And
 14 then they did a phone interview with them after
 15 that.
 16 And then the pathology, I think, had -- was
 17 since re-reviewed, which is a very -- which is a
 18 very strong thing about -- when you're able to do a
 19 pathologic review.
 20 Okay. So Table 8.
 21 Q. Table 8?
 22 A. Uh-hum.
 23 Q. Two groups, the low exposed group, greater
 24 than zero or less than or equal to 2?
 25 A. I see that.

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1 Q. And greater than 2; right?
 2 A. I see that.
 3 Q. So if you had -- and that is days per year;
 4 right?
 5 A. Two days per year. That is correct.
 6 Q. So two days per year for 10 years, that's
 7 20 exposures.
 8 A. Uh-huh.
 9 Q. And someone else who has three days in the
 10 same year and no other exposures whatsoever, three
 11 total exposures would be in the high exposure group;
 12 right?
 13 A. But if you have a three days per year for
 14 one year, that's three.
 15 Q. Yes.
 16 A. Yeah. So it would be --
 17 Q. It would be in the high exposure group?
 18 A. That is correct.
 19 Q. Despite having three lifetime exposures as
 20 compared to someone in the low exposure group with
 21 20 lifetime exposures?
 22 A. Yes.
 23 Q. Much more exposure; right?
 24 A. Yeah.
 25 Q. So people's exposure could be reversed in

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

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

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

1 other was non-Hodgkin's lymphoma.

2 So they tried -- basically, they pulled the
3 data of both studies together. And they wanted to
4 see if we pull the data altogether at the same time,
5 would be able -- would we be able to find a more
6 statistically meaningful information.

7 So they're trying to increase the power of
8 their analysis by increasing the number of patients
9 analyzed.

10 Q. There were only eight people with
11 non-Hodgkin's lymphoma exposed to glyphosate out of
12 404 total cases in these two studies; right?

13 A. Which table is that?

14 Q. Table 1.

15 A. I'm trying to see where the eight is. So
16 you have glyphosate, four cases and three control.
17 Is that what you're looking at?

18 Q. I'm looking at Table 1.

19 A. I am looking at Table 1 too. Do you want
20 to direct me what to look at in Table 1?

21 MR. LITZENBURG: Are you representing this
22 to be Hardell 2002?

23 THE WITNESS: This is 1998.

24 MR. LITZENBURG: Yeah. I mean, we are
25 looking at the different one.

1 A. Okay.

2 Q. And that's certainly a low number of cases
3 for an epidemiology study on cancer; right?

4 MR. LITZENBURG: Object to form.

5 A. It's not a high number, but it's not a
6 number that we would ignore, because then you have
7 to look at population basis.

8 Q. And Hardell did a multi-varied analysis to
9 adjust for confounders; right?

10 A. Yes, he did. You adjusted for age, county,
11 study site, and vital status.

12 Q. Do you know what vital status is?

13 A. Death versus alive, I presume.

14 Q. And Table 7 shows the odds ratio calculated
15 with multi-varied analysis with the correction for
16 those confounding factors; right?

17 A. Yes.

18 Q. And the result given there is not
19 statistically significant; right?

20 A. I think because the lower portion of the
21 95 percent confidence interval is below 1, if that's
22 what you mean.

23 Q. Yes.

24 A. Then it's not statistically significant.

25 But, as we discussed earlier, the odds ratio is --

1 THE WITNESS: This is not the paper I'm
2 referencing.

3 MR. LITZENBURG: Did you mean to give it to
4 us?

5 THE WITNESS: This is the older paper that
6 we --

7 MR. GRIFFIS: Can I see?

8 THE WITNESS: This is -- what I said this
9 is the older one that they pulled --

10 MR. GRIFFIS: Yeah, you are right. I've
11 got -- I've got the right one here.

12 THE WITNESS: Thank you.

13 MR. LITZENBURG: Thank you.

14 Q. Okay.

15 A. Yeah. This is the one, the 2002.

16 Q. Yes, sir.

17 So Table 1 in this one, then.

18 A. Okay. So Table 1, you have number of
19 cases -- just tell me what to look at. So I mean --

20 Q. There were only eight people with
21 non-Hodgkin's lymphoma exposed to glyphosate out of
22 404 total cases; right?

23 A. Oh, I see. The eight. Yes, I see that
24 now.

25 Q. Okay.

1 in my opinion, is very important. You can't ignore
2 it.

3 Q. Yes, sir.

4 You believe that odds ratios above 1 are
5 important regardless of whether --

6 A. I think it's important --

7 Q. -- they are measured to be statistically
8 significant; is that fair?

9 A. I would -- I would say I would not dismiss
10 an odds ratio that's above 1 without understanding
11 why, and without looking at additional evidence to
12 know where things are going.

13 Q. There was no adjustment made for exposure
14 to other pesticides; right?

15 A. Based on my review, I don't think they
16 adjusted for other pesticides. And I think that's
17 always a limitation because it is difficult to
18 adjust for.

19 Q. And they said that exposure to different
20 types of pesticides did correlate in this study;
21 right? So it would be a confounding factor. That's
22 on 1047, first column, three paragraphs down.

23 A. "In the multi-varied analysis exposure to
24 herbicides, fungicides increased the risk, although
25 odds ratio was lower than in the uni-varied

1 analysis."

2 So your question is?

3 Q. The results in multi-varied analysis must
4 be interpreted with caution since exposure to
5 different types of pesticides correlate. They found
6 there was correlation between --

7 A. Yeah.

8 Q. -- different types of pesticides?

9 A. Of course.

10 Q. And, therefore, there would be confounding;
11 right?

12 A. I think it's -- like I said, you always to
13 want try to control for confounding factors if you
14 can. And there are a variety of reasons why they
15 could or can't: number of cases, the belief in
16 the recall, et cetera. So it's not really clear why
17 sometimes they're not able to.

18 Q. In McDuffie and Hardell, you don't know
19 if the odds ratios would even be above 1.0 if
20 controlled for other pesticides; right?

21 MR. LITZENBURG: Object to form.

22 A. I don't know that. It was not -- it was
23 not done.

24 But you have to remember that sometimes
25 when you control for additional confounding factors,

1 A. It makes it more difficult to control. If
2 you have higher numbers, it's much easier to control
3 for variables; but when you have low numbers, you
4 have very little to work with to control for
5 variables.

6 Q. And your statistics are less well
7 controlled as well; right?

8 A. It becomes more difficult to show
9 statistical significance.

10 Q. And it's also more likely that statistical
11 findings that you think you have found don't hold
12 up; correct?

13 A. Well, you can't tell that unless you do the
14 actual control. I mean, I think it is possible that
15 they won't hold up, but it's possible they would.

16 Q. All I'm asking in general, sir, if you do a
17 small study in just a few people, you're more likely
18 to get false negatives and false positives and
19 falseness in every direction.

20 A. Of course.

21 Q. Correct?

22 A. Of course.

23 Q. And the more cases and controls that you
24 can find, the more reliable your data gets in every
25 way; right?

1 the odds ratios actually go down. So the fact that
2 you have odds ratios that's above 1 without
3 controlling is very important. And that's why we
4 can't ignore it, because once you control to --
5 to -- again, the fact that you see -- you see
6 certain things with control, without control doesn't
7 take away from the evidence, in my opinion.

8 Q. Did you just say, sir, that a failure to
9 control for a factor known to be confounding does
10 not take away from the quality of the evidence?

11 A. Yeah. If you're -- because you can't
12 always control. That's really the major issue. I
13 think, as we said earlier this morning, if you are
14 able, when you design the study, to control for all
15 variables to the extent possible, you will always
16 try to do that. But there are a variety of reasons
17 why you can't do it.

18 I think everybody acknowledges that you
19 would like to do it if you can. I don't know why
20 some studies can, some studies can't. I believe a
21 lot is related to the numbers that they have, where
22 they don't believe they have enough numbers to
23 control for all the variables included.

24 Q. Low numbers yield less useful numbers
25 across the board; right?

1 A. The more numbers you have, you will always
2 have better more robust data.

3 Q. Okay. We have looked at McDuffie and
4 Hardell, and now I'm turning to DeRoos 2003, the
5 next epidemiology study discussed in your expert
6 report, sir.

7 (Nabhan Exhibit 13 marked for
8 identification.)

9 Q. And just like the last study, Hardell, that
10 we looked at, is actually pooling two smaller
11 earlier studies. This also pooled three small
12 earlier studies; right?

13 A. This -- let me just make sure I know --
14 this is the '03 paper?

15 Q. Yes, sir.

16 A. Okay. I was -- so it says March '08 up
17 there, so I was confused.

18 Sure. Go ahead.

19 Q. Okay. So this pooled three earlier small
20 studies; right?

21 A. Yes. From Nebraska, Iowa, Minnesota and
22 Kansas.

23 Q. It pooled the Cantor study from Iowa and
24 Minnesota, the Zahm study from Nebraska, and the
25 Hoar study from Kansas; correct?

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1 A. Yes.
 2 Q. And, again, these were studies that were
 3 looking at multiple pesticides and herbicides
 4 simultaneously; right?
 5 A. It did.
 6 Q. So like the others, you'd expect some false
 7 positives; right?
 8 A. It's possible.
 9 Q. The Cantor -- we just looked at a study --
 10 the Hardell study with eight cases in it. The
 11 Cantor study had 26 cases, sir.
 12 A. The Cantor study was mainly for farmers in
 13 farming population. I don't think this specifically
 14 looked at glyphosate.
 15 Q. And -- right. And Dr. Neugut testified
 16 that it had low power, the Cantor study had low
 17 power, because there were only 26 cases of
 18 non-Hodgkin's lymphoma with exposure to glyphosate.
 19 Do you agree that that many cases with
 20 exposure to -- with exposure is a low-powered study?
 21 A. I think you have to look at the
 22 denominator, 26 out of how many, to accurately see
 23 how powerful the study was.
 24 Q. Okay. Do you want to see the Cantor study?
 25 A. Sure.

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1 (Nabhan Exhibit 14 marked for
 2 identification.)
 3 Q. Did you look at these individual
 4 substudies, sir?
 5 A. Yes, I did. But there's a lot of
 6 information in each one. Difficult to remember
 7 everything.
 8 This is a '92 paper. Yeah.
 9 So they had 195 patients with follicular
 10 lymphoma, 198 with diffused, and 85 of small
 11 lymphocytic, and 144 of other. So this is the
 12 number of lymphoma cases that they had. They had
 13 622 cases and 1,245 controls.
 14 Q. And Table 6, sir, you can see how many were
 15 exposed to glyphosate, and the answer is 26; right?
 16 A. Yes. But I think it's important -- that's
 17 what I meant by the denominator. I think it's a
 18 very respectable number, 622 cases and 1,245
 19 controlled. That's the denominator, which is very
 20 important.
 21 And then you look at Table 6, as you said.
 22 And in glyphosate, the number of cases were 26
 23 versus 49. So I -- you know, I think that's -- you
 24 know, 26 out of 622. I mean, the total number of
 25 exposure is 26 plus 49.

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

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

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1 A. If they controlled for other pesticide
 2 exposures?
 3 Q. Yes.
 4 A. So I have in my notes here that they did
 5 control for confounders. So, obviously, I've looked
 6 and saw that they controlled for confounders. And
 7 I'll have to see if they -- I believe they actually
 8 tried to control for other pesticides.
 9 Q. In the hierarchical section; right?
 10 A. Because there were 47 pesticides out of --
 11 that's what I wrote here in my notes, but I don't
 12 know the methodology of how they controlled. Maybe
 13 this is statistical way of controlling. My notes
 14 suggest that they have -- they did control for other
 15 pesticides.
 16 Q. Yes, sir.
 17 In the hierarchal regression they did;
 18 right?
 19 A. Okay. I guess in the hierarchal
 20 regression.
 21 Q. Okay. And the odds ratio that you reported
 22 in your expert report comes from the logistic
 23 regression on Table 3; true?
 24 A. 2.1. Let me check. 2.1, that is from the
 25 logistic regression, that's correct.

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1 Q. And the odds ratio reported from the
 2 hierarchical regression 1.6, confidence interval 0.9
 3 to 2.8 is not statistically significant; correct?
 4 A. The hierarchical regression is 1.6, and the
 5 other one is 2.1. That's correct.
 6 Q. And the hierarchical regression is not
 7 statistically significant; correct?
 8 A. Yes. I just don't know whether that is
 9 really -- again, you know, the controlling for
 10 pesticides, does it really matter if it's logistical
 11 regression versus hierarchical regression? I can't
 12 really answer that.
 13 Q. Well, that's the one that is controlled for
 14 other pesticides --
 15 A. Well, you control --
 16 Q. -- isn't it?
 17 A. You also -- when you do a logistic
 18 regression, you actually do control for other
 19 factors, including pesticides. So I'm not really
 20 sure whether they didn't -- you know, whether one
 21 negates the other. That's what I'm trying to say.
 22 Q. Okay. Do you know of anywhere where they
 23 reported that, in the logistic regression, they
 24 controlled for other pesticides?
 25 A. I'll have to read the whole paper again. I

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

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1 pesticides," for whatever it's worth.
 2 Q. The next study you mention is the Lee
 3 study, sir.
 4 (Nabhan Exhibit 15 marked for
 5 identification.)
 6 Q. This actually used data from Cantor, which
 7 we've already discussed, and one other U.S.
 8 case-control study; is that right?
 9 A. Yes.
 10 Q. And the odds ratios reported here were not
 11 adjusted for exposure to other pesticides; true?
 12 A. Repeat again, please.
 13 Q. The odds ratios reported were not adjusted
 14 for exposure to other pesticides; right?
 15 A. No, it was not. It was adjusted for age,
 16 vital status, and state.
 17 Q. The hypothesis under investigation was
 18 whether asthma modifies the risk of NHL associated
 19 with pesticide exposures; correct?
 20 A. Correct.
 21 Q. And in people exposed to glyphosate, there
 22 was no statistical significant association either in
 23 people with or without asthma; correct?
 24 A. That is correct. The odds ratio was above
 25 1 for both, but it was not -- it did cross the 1.

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1 Q. It did cross the 1.
 2 And do you know of any sort of analysis
 3 that has been done to compare the 1.2 to the 1.4 to
 4 see if there is a statistically significant
 5 difference between people with and without asthma?
 6 A. I'm not aware of that. I don't know.
 7 Q. Okay. You have no conclusion about whether
 8 asthma increases or decreases or has no effect on
 9 any risk that you believe exists of non-Hodgkin's
 10 lymphoma from glyphosate; is that fair?
 11 A. Yeah, I -- I don't have any additional
 12 conclusions beyond what the authors have concluded.
 13 And the authors' conclusion suggests that -- and I
 14 quote -- "Our results suggest that the risk of NHL
 15 among asthmatics with pesticide exposure may be
 16 higher than among non-asthmatics with pesticide
 17 exposure."
 18 I have no additional conclusions beyond
 19 what you just stated.
 20 Q. And you don't know if that was specific to
 21 glyphosate; right?
 22 A. They talked about pesticide exposure in
 23 general.
 24 Q. Certainly, the point estimate for people
 25 with asthma was lower than the point estimate for

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1 people without asthma for glyphosate-exposed people;
 2 right?
 3 A. Correct.
 4 MR. GRIFFIS: I need to tidy up my pile
 5 here. Let's take five minutes.
 6 VIDEOGRAPHER: Going off the record at
 7 2:00 P.M.
 8 (Recess taken from 2:00 P.M. to
 9 2:15 P.M.)
 10 (Nabhan Exhibit 16 marked for
 11 identification.)
 12 VIDEOGRAPHER: We are back on the record at
 13 2:15 P.M.
 14 BY MR. GRIFFIS:
 15 Q. Exhibit 16 is the DeRoos 2005 article,
 16 which is the next one discussed in your expert
 17 report; correct?
 18 A. Correct.
 19 Q. Now, this is a prospective cohort study;
 20 correct?
 21 A. Correct.
 22 Q. All the other ones we've been looking at
 23 are case-control studies; right?
 24 A. Yes.
 25 Q. Would you explain the difference to the

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1 jury.
 2 A. So, I mean, case control is -- you know, in
 3 broad term is more of a retrospective study where
 4 you are looking at individuals who are diagnosed
 5 with the disease and those who are not diagnosed
 6 with the disease. And you retrospectively attempt
 7 to analyze exposure or contributing factors that
 8 might have led to the development of the particular
 9 disease.
 10 The cohort study is more of a prospective
 11 evaluation of particular individuals, and you follow
 12 them prospectively. So you are presuming that, at
 13 the time of initiating the particular study, nobody
 14 has the particular disease per se. And you follow
 15 them for whatever period you decide to follow them,
 16 and you assess who developed the disease and why and
 17 what. And you make an analysis.
 18 Q. So in the case-control studies that we've
 19 looked at so far -- like DeRoos 2003, Cantor,
 20 Hardell, McDuffie, et cetera -- the authors
 21 started out with a group of people with
 22 non-Hodgkin's lymphoma, and then they asked
 23 questions of those people and some others who they
 24 found without non-Hodgkin's lymphoma to be controls
 25 and compared what they said about their past

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1 exposures to all sorts of different pesticides in
 2 all of those studies and then ran some statistics on
 3 them; is that fair?
 4 A. Fair.
 5 Q. Okay. And in this study and in a
 6 prospective cohort study, what they did instead was
 7 gather a bunch of people -- and these were what kind
 8 of people?
 9 A. These were mainly folks that were licensed
 10 to apply restricted-use pesticides.
 11 Q. So these were licensed pesticide
 12 applicators? People who would be exposed to
 13 pesticides; right?
 14 A. But they're licensed, so they're --
 15 usually, they -- they know what they're doing. They
 16 had to have, like, a particular exam criteria to
 17 enter the study and so forth. So they --
 18 Q. Okay.
 19 A. They had -- you know, they had more
 20 knowledge of what they are up against, if you will.
 21 Q. And these are people who did not have
 22 non-Hodgkin's lymphoma. And they filled out
 23 questionnaires about their exposure to pesticides,
 24 which were renewed at various times.
 25 And then the authors of this study followed

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

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

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

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

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

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

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

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

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

15 That's fair; right?

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

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

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

8 A. Right.

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

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

17 A. I agree with that.

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

21 A. Or the lack thereof.

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

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

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

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

6 A. Yes.

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

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

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

19 Q. About glyphosate?

20 A. About glyphosate, yeah.

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

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

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

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1 A. I honestly don't know who's funding it.
 2 It's probably the NIH, which is U.S. government
 3 but -- I believe it's the -- NIH, yeah.
 4 Q. There were 92 individuals here with
 5 exposure to glyphosate who had non-Hodgkin's
 6 lymphoma; right?
 7 A. 92, correct.
 8 Q. And, again, these were people -- there was
 9 a very large body of people who were being tracked,
 10 and 92 of the ones who developed non-Hodgkin's
 11 lymphoma had an exposure to glyphosate; is that
 12 right?
 13 A. Yeah. I mean, there were -- as you can see
 14 in Table 2, there are other cancers, but the NHL
 15 specifically was 92.
 16 Q. They started out with 57 -- more than
 17 57,000 private and commercial pesticide applicators;
 18 right?
 19 A. Yes. There was 57,311.
 20 Q. And they paid attention to all of the
 21 cancers that these people developed, although they
 22 especially looked at non-Hodgkin's lymphoma because
 23 there had been previous studies done like the ones
 24 we've been talking about; correct?
 25 A. Yes.

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1 Q. And they reported -- here, I'm on page 51,
 2 sir, the end of the long paragraph at the top of the
 3 third column.
 4 They reported no association was observed
 5 between NHL and glyphosate exposure in any analysis,
 6 including an analysis comparing the highest with the
 7 lowest quintile of exposure, more than 108 versus 0
 8 to 9 cumulative exposure days; correct?
 9 A. That's what's written here.
 10 Q. Okay. Now, when we were looking earlier at
 11 a -- at an exposure-days-per-year estimate, we were
 12 looking at 0 to 2 versus greater than 2; right?
 13 A. Well, that was in one paper, though.
 14 Q. Yes.
 15 A. Yeah, I mean, that was one paper, I think
 16 McDuffie, that they looked at 0 to 2 versus over
 17 2, yes.
 18 Q. And this was looking at a much greater
 19 range of days; right?
 20 A. I don't know if this one looks at days
 21 per se. I think they -- they had their different
 22 definition. If you -- when you read the -- page 50,
 23 the first column, they constructed three glyphosate
 24 exposure metrics, ever personally mixed or applied,
 25 cumulative lifetime, et cetera, et cetera.

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1 So they have lowest exposed, higher
 2 exposed, never exposed. And so they just use a
 3 different way of deciding exposed versus nonexposed.
 4 Q. And the one described here, the highest
 5 versus the lowest quintile of exposure, was more
 6 than 108 cumulative exposure days versus the lowest,
 7 0 to 9, cumulative exposure days; right?
 8 A. I see that, yes.
 9 Q. And was that a relative risk point estimate
 10 of less than 1; right?
 11 A. I see that, yes.
 12 Q. In Table 2, the ever/never used, the
 13 relative risk point estimate adjusted for age,
 14 demographic, and lifestyle factors in other
 15 pesticides was 1.1 with a confidence interval of 0.7
 16 to 1.9, which is not statistically significant;
 17 correct?
 18 A. Correct.
 19 Q. On Table 3, they looked at cumulative
 20 exposure days and intensity-weighted exposure days;
 21 correct?
 22 A. That is correct.
 23 Q. Now, cumulative exposure days is looking at
 24 how many days people were exposed for, and
 25 intensity-weighted exposure days is adjusting those

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

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

1 sir --
 2 A. Yes.
 3 Q. -- this is the one with the highest power;
 4 right?
 5 A. No. This is the highest number. That's
 6 different than power.
 7 Q. Okay. Tell me --
 8 A. Power, now, is statistics.
 9 Q. Do you think that a different study that
 10 you reviewed has more power than this one?
 11 A. I didn't look at the power of each study.
 12 I think you're correct by saying that this has the
 13 highest number of patients with non-Hodgkin's
 14 lymphoma, 92 cases. That's correct?
 15 But when you say "highest power," then
 16 you'll have to compare the trials from a statistical
 17 standpoint, each one. And I did not perform that,
 18 nor am I qualified to compare statistical power
 19 between -- across studies, and I wouldn't recommend
 20 comparing different studies from a statistical
 21 standpoint. It's not a very good exercise to do
 22 from an academic standpoint.
 23 Q. From an academic standpoint, it's not a
 24 good exercise to compare the power of different
 25 epidemiology studies?

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

1 A. To compare across studies, it's not
 2 something that we normally would like to do because
 3 each study has its own. So you're going to -- I
 4 mean, to compare across -- cross-trial comparisons
 5 are not something that we normally would like to do.
 6 Q. And how does that comment apply to the
 7 field of meta-analysis, sir?
 8 A. I'm not sure I understand the question.
 9 Q. Well, a meta-analysis -- meta -- people who
 10 are performing a meta-analysis --
 11 A. Well, there are methodologies for
 12 meta-analysis. If you're conducting a
 13 meta-analysis, you follow a methodology to make sure
 14 that you look at the trials. I mean, there is --
 15 these are oftentimes scientists and statisticians
 16 that are equipped to perform a meta-analysis using
 17 robust criteria and looking at all of the data
 18 that's available.
 19 You're not necessarily comparing the
 20 statistical power of each particular study against
 21 each other. You're trying to look at all of the
 22 studies combined and see, are you seeing any
 23 causation? Are you seeing any association when you
 24 look at the entire body of the literature?
 25 Q. Do you know whether people who are

1 performing a meta-analysis, as part of the procedure
2 that they follow, assess the power of each of the
3 studies that they're looking at and weigh the
4 meta-analysis in terms of the power of those
5 studies?

6 A. I don't know if that's what they do.

7 Q. All right. Do you know of any other study
8 besides the DeRoos 2005 study, of the ones that you
9 looked at, sir, that measured the intensity of
10 exposure?

11 A. And when you mean -- by "intensity," you
12 mean the dose of the compound? Because the
13 intensity could be just the years of exposure by the
14 number of years. Or are you talking specifically in
15 terms of the dose?

16 Q. I'm talking about intensity-weighted
17 exposure, like in the second column of Table 3.

18 A. Yeah, I'm not aware of other studies that
19 look -- that added the -- you know, again, you can
20 see the -- you know, the actual number of years by
21 the number of days per year.

22 But what they did here is they added
23 another attempt by adding the actual intensity
24 level, which is always commendable thing to do. It
25 has its own limitations because always difficult to

1 in this paper, I'm not aware of other papers that
2 did the same formula. That's the short answer to
3 your question based on the papers I reviewed.

4 Q. In the area of epidemiology --

5 A. Right.

6 Q. -- I'm talking about your section headed
7 "Epidemiology" in your expert report --

8 A. Yes.

9 Q. -- is there any other paper where you
10 purport to see or not -- or not see a dose response?

11 A. Let me check a couple of things --

12 Q. Yes, sir.

13 A. -- to be more accurate in answering that.

14 So I think there is a paper by Eriksson
15 that I reviewed from 2008 that talked about more
16 than ten days, less than ten days in terms of
17 different odds ratio. So I don't know if you would
18 consider that. Again, this is the number of days of
19 exposure, and they used the cutoff of less than ten
20 days or more than ten days.

21 But I think the DeRoos '05 paper, they
22 specifically added the intensity multiplied by the
23 number of years multiplied by the number of days per
24 year. I have not seen that particular formula in
25 other papers.

1 be very accurate with it. But I'm not aware of
2 other studies that did the same thing.

3 Q. Do you know of any other study with dose
4 data like this?

5 A. Like exactly this one?

6 Q. Dose data at all, sir. It's important when
7 you're looking at --

8 A. Early on, we -- you know, you showed me
9 the -- and we talked about the Bolognesi paper, I
10 mean, in terms of aerial spray and in some areas
11 more, some areas less.

12 Is this a dose data? I don't know.

13 Q. I was talking about epidemiology, sir.

14 But in the Bolognesi paper that you
15 mentioned, which was a genotoxicity study --

16 A. Right.

17 Q. -- not epidemiology --

18 A. I know.

19 Q. -- for non-Hodgkin's lymphoma, there was
20 not a dose relationship; correct?

21 A. Well, I think we -- we are going to
22 disagree on the interpretation the Bolognesi paper,
23 but I just -- I'm trying to understand your question
24 in terms of the dose per se. If you're asking about
25 the intensity-weighted exposure days as it's defined

1 But the Eriksson paper, you know, you could
2 consider this a form of dose response because they
3 used ten days, less than ten days. The McDuffie
4 paper that we actually reviewed more than ten days,
5 less than -- more than two days, less than two days,
6 you could easily say this was a dose response
7 because, again, you have a couple of days less or
8 more.

9 So each paper and each manuscript has its
10 own definition of how they define dose intensity.

11 Q. Unlike the ones you mentioned, this one
12 controlled for other pesticides; right?

13 A. I just want to make sure. I believe they
14 tried to control for pesticides. You will have to
15 remember that the actual controls here, that they're
16 never exposed and so forth, were all licensed
17 applicators that were using pesticides. So I think
18 you are starting from such a high bar to be able to
19 demonstrate statistical significance over the
20 control group.

21 So, again, you look at the patient
22 population or the individual population that's going
23 in, and everybody in the AHS was actually a licensed
24 applicator. So in order to demonstrate statistical
25 significant above and beyond, it's way more

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1 difficult than when we take controls that don't have
 2 any of these occupational exposures.
 3 Q. I'm sorry. Were you done answering?
 4 A. Yes, I was.
 5 Q. Okay. The next paper that you mention is
 6 the Fritschi paper; right?
 7 A. Yes, I mentioned that.
 8 (Nabhan Exhibit 17 marked for
 9 identification.)
 10 Q. This mentions as possible -- in the first
 11 paragraph, sir -- possible causes of increased risks
 12 non-Hodgkin's lymphoma among farmers, exposure to
 13 diesel exhaust and animal viruses.
 14 Do you see that?
 15 A. I see that, yes.
 16 Q. Do you have an opinion as to whether those
 17 are risk factors for non-Hodgkin's lymphoma?
 18 A. I don't have an opinion.
 19 Q. Fritschi is an Australian study?
 20 A. Yes.
 21 Q. And the exposure was assessed by -- the
 22 exposure was established by an occupational
 23 hygienist who reviewed occupational histories and
 24 determined what they felt that the exposure of the
 25 individuals in the study would have been to various

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1 herbicides and pesticides; right?
 2 A. The hygienist and the interviewers were
 3 blinded to the case or control status of the
 4 subjects. So, yes, there were interviews, but there
 5 was blinding of the interviewers.
 6 Q. And the interviews were about the
 7 occupations that people had worked in; correct?
 8 A. Well, again, there are probably more than
 9 just the occupation. It looked at other factors as
 10 well.
 11 Q. And specific tasks?
 12 A. Right.
 13 Q. But the exposure to particular substances
 14 was assigned by an occupational hygienist based on
 15 people's answers about their careers rather than
 16 people saying what substances they had been exposed
 17 to; right?
 18 A. Well, the hygienist was blinded to all of
 19 these. I mean, the same expert occupational
 20 hygienist, again blind to the status, reviewed the
 21 occupational histories and the answers to the
 22 modular questions and determined exposure to various
 23 substance, including argon and phosphates,
 24 et cetera. So it wasn't a priority that the
 25 hygienist knew what was happening.

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1 Q. Oh, sure.
 2 A. They were blinded.
 3 Q. But they didn't ask people, "Have you ever
 4 been exposed to glyphosate?" A hygienist assigned
 5 people based on what they said their occupational
 6 history was. They declared that somebody had or
 7 hadn't been exposed to glyphosate and at what level,
 8 and the same for all sorts of other pesticides and
 9 herbicides; right?
 10 A. You know, I don't think they put the actual
 11 questionnaire. I'm trying to read here. It says
 12 here, "Case in controls and were mailed an
 13 introductory letter, an information leaflet,
 14 followed by self-administered questionnaire to each
 15 consenting subject. The questionnaire included a
 16 diary with a detailed lifetime history of each job
 17 the subject had held for one year or more.
 18 Information obtained on each job included job title,
 19 employer, industry, start and finish years, number
 20 of hours worked per day, and number of days worked
 21 per week."
 22 And they looked at the cases in the
 23 controlled and -- and -- so I don't know if the
 24 assignment was before or after, if that's your
 25 question. Your question is was the assignment after

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1 the answers were available? Is that your question?
 2 Q. No, sir.
 3 It is that when someone was said to be --
 4 have been exposed to a particular substance in this
 5 study, that wasn't based on them saying that they
 6 had been exposed to that substance; it was based on
 7 an occupational hygienist proclaiming that they had
 8 been based on the jobs that they said that they'd
 9 performed in the past?
 10 A. Well, based on their answers. Based on
 11 their answers.
 12 Q. But not their answers about pesticide
 13 exposure.
 14 A. I see. You're saying -- I see. Based
 15 on --
 16 Q. Someone said --
 17 A. -- the answers --
 18 Q. Someone said, I worked -- I was an alfalfa
 19 farmer for two years, and then I herded kangaroos
 20 for six. And the occupational therapist, therefore,
 21 assigned --
 22 A. You're correct.
 23 Q. -- putative pesticide exposure?
 24 A. Now I understand your question.
 25 Q. Okay.

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1 A. Yes, you're correct.
 2 Q. And you don't know how reliable that
 3 methodology is; fair?
 4 A. It's fair to say, yes.
 5 Q. In your expert report, you gave an odds
 6 ratio of 3.29; correct?
 7 A. Correct.
 8 Q. And that was for, as you said, for all --
 9 for other herbicides?
 10 A. For all herbicides, collectively, yes.
 11 Q. You don't know if glyphosate is included in
 12 the figure that you gave; correct?
 13 A. It's not called out specifically in this
 14 trial. They don't recall call out glyphosate
 15 specifically. They include all herbicides.
 16 Q. You don't know what glyphosate's
 17 contribution was, if anything; right?
 18 A. I do not know that, yeah. But glyphosate
 19 is an herbicide, so I would presume it was part of
 20 the -- it was included.
 21 Q. And you don't know if it pulled the odds
 22 ratio up or down or had no effect on it; right?
 23 A. I can't tell.
 24 Q. So what effect did this study have your
 25 opinion?

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1 A. It just solidified that you see that with
 2 herbicides, and glyphosate is an herbicide. So,
 3 again, it's just another demonstration that
 4 herbicides, as a class, could have an increased risk
 5 of causing and contributing to non-Hodgkin's
 6 lymphoma.
 7 Q. Is it your opinion, to a reasonable degree
 8 of medical certainty, that herbicides, as a class,
 9 cause or contribute to non-Hodgkin's lymphoma?
 10 MR. LITZENBURG: Object to form.
 11 A. It's -- it's tough to really tell. But you
 12 have data on farmers that use herbicides. You have
 13 a trial like this that has herbicides. So it's --
 14 it's hard to actually lump all of them in just one
 15 basket. I believe that there is some increased risk
 16 with herbicides, and having glyphosate as one of
 17 them solidifies my opinion in terms of the causation
 18 between this compound and non-Hodgkin's lymphoma.
 19 Q. Okay. So are you going to be testifying,
 20 do you have the opinion, that there is a class
 21 effect?
 22 A. I would say there is enough evidence to
 23 suggest. Again, there's -- there would be no
 24 absolutes given what we've talked about several
 25 times. I mean, this -- this -- obviously, this

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1 particular trial shows that herbicides have an
 2 increased odds ratio of developing non-Hodgkin's
 3 lymphoma. It didn't call out glyphosate. The
 4 Cantor study talks about farmers. And, again,
 5 farmers use herbicides.
 6 So when you say a class effect, then your
 7 implying that every single herbicide will have this
 8 causation, and I don't think we can safely say that.
 9 I'm not prepared to say that because I can't say
 10 every single herbicide will have that.
 11 Q. Do you have the opinion that there is any
 12 herbicide that does not cause or contribute to
 13 non-Hodgkin's lymphoma?
 14 A. I don't have an opinion on that.
 15 Q. Do you have an opinion that there is an
 16 herbicide that is safer with regard to non-Hodgkin's
 17 lymphoma than glyphosate?
 18 A. I also don't have opinion on that. I did
 19 not review that.
 20 (Nabhan Exhibit 18 marked for
 21 identification.)
 22 Q. Marking as Exhibit 18 the Eriksson study,
 23 sir. And this is another exploratory study that
 24 wasn't designed to specifically test the hypothesis
 25 of an exposure between glyphosate and non-Hodgkin's

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1 lymphoma, but to screen multiple herbicides and
 2 pesticides at the same time; right?
 3 A. Correct. It did look at glyphosate,
 4 though, as you can see --
 5 Q. Yes, sir.
 6 A. Okay.
 7 Q. They have results for multiple individual
 8 pesticides.
 9 A. Sure. Okay.
 10 Q. Now, the only adjusted odds ratio reported
 11 in this study that's controlled for confounding by
 12 other pesticides is in Table 7 on page 1661 of the
 13 study; right? The multi-varied analysis?
 14 A. Table -- Table 7, they talked about
 15 adjustment for age, sex, and year of diagnosis or
 16 enrollment. I think on page -- on Table 2, they
 17 also have some adjustments was made for age, sex,
 18 and year of diagnosis -- yeah, these are the three
 19 things they adjusted for.
 20 Q. They are not adjusted for medical
 21 conditions or family history of cancer or smoking or
 22 drinking or any of the lifestyle factors that you've
 23 discussed earlier; right?
 24 A. No, they were not.
 25 MR. GRIFFIS: Let's take five minutes.

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1 VIDEOGRAPHER: Going off the record at
 2 2:50 P.M.
 3 (Recess taken from 2:50 P.M. to
 4 3:01 P.M.)
 5 VIDEOGRAPHER: And we are back on the
 6 record at 3:01 P.M.
 7 BY MR. GRIFFIS:
 8 Q. Okay. Sir, the Table 7, "Multi-varied
 9 analysis for glyphosate," the most adjusted odds
 10 ratio for confounders set forth in this study is not
 11 statistically significant; correct?
 12 A. The odds ratio is 1.51.
 13 Q. And it is not statistically significant;
 14 correct?
 15 A. Correct.
 16 Q. When you look at the unadjusted odds ratios
 17 for all the substances in this study, you see that
 18 virtually every single one of them is above 1.0;
 19 right?
 20 A. Which table you are looking at? The same
 21 table?
 22 Q. Let's look at Table 2 first, "Exposure to
 23 various herbicides."
 24 A. Okay.
 25 Q. Herbicides total. That's all greater than

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1 1.
 2 A. Right.
 3 Q. Phenoxyacetic acids, all greater than 1.
 4 Subgroup of MCPA all greater than 1. 2,4,5-T and/or
 5 2,4-D, all greater than 1. Other greater than 1.
 6 Herbicides except phenoxyacetic acids, all greater
 7 than 1. Glyphosate, greater than 1. Other
 8 herbicides, all greater than 1 except for when you
 9 get to greater than 32 days.
 10 Do you see that?
 11 A. I do.
 12 Q. Okay. Now let's turn to Table 4, "Exposure
 13 to various other pesticides."
 14 Without repeating every single thing in
 15 here, we have insecticides, DDT, mercurial seed
 16 dressing, pyrethrin, permethrin, other insecticides,
 17 fungicides, impregnating agents, chlorophenols,
 18 arsenic, creosote, tar, other impregnating agents
 19 and rodenticides.
 20 And virtually every single one is greater
 21 than 1; correct?
 22 A. Some of them are not.
 23 Q. Yeah, some are not. The vast majority are
 24 greater; right?
 25 A. Yes. As long as we acknowledge some are

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1 not.
 2 Q. One, two, three, four are not; right?
 3 A. One, two, three, four. Yes.
 4 Q. And that's out of 34 stated odds ratios;
 5 right?
 6 A. Yes.
 7 Q. Just in that table?
 8 A. The -- that's actually not -- I mean,
 9 that's not -- 34 odds ratio, but not 34 compounds.
 10 Q. Yes, sir.
 11 A. Okay.
 12 Q. 34 of the odds ratios.
 13 A. Okay.
 14 Q. For each of the ones that was not above 1,
 15 another one of the measurements for a different
 16 period of time for that same substance was greater
 17 than 1; right? So every substance was found to be
 18 greater than 1?
 19 A. Do you mind repeating the question?
 20 Q. Yes, sir.
 21 Where there is an odds ratio in Table 4
 22 below 1 --
 23 A. Uh-hum.
 24 Q. -- for a particular substance, if you look
 25 immediately above or below it, you will find that

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1 same substance for a different time period with an
 2 odds ratio of above 1; right?
 3 A. I see what you're saying. Yes, I do see
 4 that.
 5 Q. So every substance in Table 2, various
 6 herbicides, and 4, pesticides, is greater than 1;
 7 right?
 8 A. For the most part, yes.
 9 Q. Now, when you have a study that is
 10 reporting unadjusted odds ratios for every substance
 11 that it looks at above 1, there is some suggestion
 12 that there is confounding in the results; right?
 13 A. I'm trying to understand your question. So
 14 you're -- you're -- I mean --
 15 Q. Do you believe that every one of these
 16 substances causes NHL?
 17 A. I don't think we know the answer to that.
 18 Q. Okay. Do you think this is evidence that
 19 every one of these substances causes NHL?
 20 MR. LITZENBURG: Object to form.
 21 A. I think the -- the -- you know, this is one
 22 paper, one suggestion that there are other
 23 substances that might be implicated in causation or
 24 predisposition to developing NHL. So I will need to
 25 do a formal investigation, let's say, on arsenic by

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1 itself and spend a lot of time to understand if
 2 there is any additional as to arsenic by itself,
 3 because I'm not going to take just one paper and
 4 make a bald conclusion that all of these substances
 5 have causation to NHL.
 6 Q. Well, far as this paper goes, as far as the
 7 Eriksson paper goes, to the extent that you're using
 8 it --
 9 A. It's hypothesis-generating.
 10 Q. -- as evidence of glyphosate causing NHL,
 11 it's also just as good evidence for all these other
 12 substances causing NHL?
 13 A. But this is not the only paper I use for
 14 glyphosate. It's not the only one I cited. So
 15 that's really not correct. I've actually cited a
 16 lot of other papers. So I use this paper in
 17 conjunction with other evidence.
 18 Q. Let me ask the question again.
 19 A. Please.
 20 Q. With regard to this paper --
 21 A. Yes.
 22 Q. -- to the extent that you use this paper as
 23 evidence that glyphosate causes NHL, this paper is
 24 just as good evidence that each of these other
 25 substances causes NHL; right?

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1 MR. LITZENBURG: Object to form.
 2 A. Again, I'm going to try to reemphasize that
 3 this is one paper, so I -- I will agree to one
 4 thing, that, if you take just this one paper, the
 5 evidence appears to be that for glyphosate. There
 6 are other substances as well. But you can't form a
 7 conclusion on causation between a compound and a
 8 disease based on one paper.
 9 So, I mean, this is all I can say regarding
 10 this question.
 11 Q. Yes, sir.
 12 To the extent that this paper is a piece of
 13 the puzzle that you have put together to form an
 14 opinion that glyphosate causes NHL, it would be just
 15 as good a puzzle piece for every one of these other
 16 compounds?
 17 A. If the other compounds have as much
 18 evidence regarding causation with NHL, as the
 19 evidence that I've seen for glyphosate, then the
 20 answer would be correct. But if I try to pick
 21 whichever compound and I find nothing after that,
 22 then it would be just simply hypothesis-generating,
 23 and it wouldn't mean much of it.
 24 So, I mean, I think you're partly correct
 25 that there is something here, but needs to be

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1 investigated further.
 2 Q. And when you do a study of multiple
 3 compounds and you're finding positive association
 4 after positive association -- and by positive, all I
 5 mean is greater than 1 because most of these are not
 6 statistically significant.
 7 A. I understand.
 8 Q. I'm using kind of your definition of --
 9 A. Sure.
 10 Q. -- positive from earlier today. It
 11 suggests that you need to control the exposures that
 12 you're most interested in for all of those other
 13 compounds so that you can find out whether you have
 14 a real effect or one that's confounded; correct?
 15 A. See, it's not necessarily true, because the
 16 issue of controlling for these confounding factors
 17 is really for both cohorts, for the cases and the
 18 controls. I mean, you have two types of population.
 19 You have the population of those folks who developed
 20 non-Hodgkin's lymphoma and the population that did
 21 not develop non-Hodgkin's lymphoma.
 22 So, you know, you're trying to apply the
 23 confounding factor in terms of controlling only to
 24 the one folks who have non-Hodgkin's lymphoma. The
 25 reality is you will control for these factors in

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1 both populations, in the cases and the controls. So
 2 you can make an argument, a valid argument, and it
 3 becomes a washout because you are going to control
 4 for these confounding factors for both of them.
 5 Q. That's what you do when you control for
 6 confounding; you control for both. Right?
 7 A. Right. That's what I'm saying.
 8 Q. And what we've seen, in study after study
 9 today, is that when you control for other pesticide
 10 exposures, results that were statistically
 11 significant become not statistically significant for
 12 glyphosate; correct?
 13 A. No. I think what we are -- what we -- what
 14 I can say is that there are studies that we reviewed
 15 together earlier today that did not control for
 16 other pesticide exposure. What I'm going to argue
 17 is I don't know whether controlling for these
 18 pesticide exposures would have changed the odds
 19 ratio positively or negatively. The test was not
 20 done to -- to -- so speculating that just by
 21 controlling, the odds ratio will disappear is a
 22 complete speculation. As a scientist, I can't agree
 23 to that.
 24 Q. Tell me of a statistically significant
 25 association between glyphosate and non-Hodgkin's

1 lymphoma in the epidemiology you looked at that is
2 controlled for other pesticides.

3 A. But, again, I think we reviewed -- I mean,
4 you have seen my -- my report. These are the
5 studies I looked at, and they have to be solidified
6 substantially with the IARC report as well as
7 additional meta-analysis.

8 So, you know, the IARC report, which is the
9 highest authority, in my opinion, in determining a
10 causation between any type of a compound,
11 occupational compound, and cancer, has looked at
12 these data as well as other data and made a
13 conclusion that it's probably carcinogenic to
14 humans.

15 So I have to take that into consideration
16 in addition to the data that I've actually looked
17 at. The IARC folks obviously and clearly have
18 looked at animal data and other data plus the
19 epidemiologic literature that did not always control
20 for confounding factors. And yet, after all of
21 this, they did find evidence that it's probably
22 carcinogenic to humans.

23 So, you know, I think there's enough
24 literature out there between these studies that we
25 just reviewed, the IARC, the meta-analysis to

1 demonstrate, in my opinion, that there's a causation
2 between this compound and the disease.

3 Q. Explain to the jury, please, why you
4 believe IARC to be the highest authority on the
5 subject of whether a substance causes cancer?

6 A. It is not just my own individual belief. I
7 think it is the belief of everybody in the field
8 that IARC is the -- this is what they do. This is
9 what they are tasked with. And in addition to that,
10 this is what they do and this is what they are
11 tasked with, their data and their output was
12 published in the most prestigious journal, more
13 prestigious than all of these journals that we just
14 reviewed today, in Lancet, where the acceptance rate
15 is less -- is close to 5 percent. So they reject
16 95 percent of manuscripts that they get submitted.

17 So we can't ignore the most powerful
18 evidence that is out there. So I have to -- I
19 relied on it clearly, as well as additional studies
20 that I cited.

21 Q. And do you believe that a Lancet peer
22 review of an IARC Monograph means that the people
23 who did the peer review believe that the conclusions
24 of the authors were correct or just that they
25 followed a particular methodology as stated and came

1 to a particular conclusion?

2 MR. LITZENBURG: Object to form. Asked and
3 answered.

4 A. So when you do a peer review -- and as you
5 probably know from my CV, I do quite a few of peer
6 review for very good and prestigious journals -- you
7 actually have to look at the hypothesis, whether the
8 methodology is sound, whether the authors were free
9 of bias, and whether their conclusions actually were
10 supported by the evidence that they provide. You
11 can't conclude something and you have no evidence in
12 the paper to it. I mean, you just can't. So that's
13 what you look at.

14 Now, you may not always agree with the
15 conclusion, but your job as a peer reviewer is to
16 look at the evidence that they provide and to review
17 that evidence and see if it correlates with the
18 conclusion. And then you make a decision. Do you
19 reject the paper because you don't believe -- not
20 because you don't believe in the -- you don't
21 believe it was the proper way to be done, or do you
22 accept it but you request additional revisions and
23 you have additional inquiries because you believe
24 the author should really provide more details,
25 et cetera?

1 I'm sure you're familiar with the
2 peer-review process. So I don't want to waste time
3 by just talking about how it's done, but this is how
4 we do it.

5 You don't have to always agree with what
6 they come up with. But I can say that most peer
7 reviews, if you believe the conclusions are so out
8 there, you are going to reject the paper. You're
9 going to say -- and you will e-mail the editor and
10 say, This is making no sense to me.

11 Q. You know that, sir, in a hazard assessment,
12 which is what IARC does -- hazard, not risk -- a
13 hazard assessment is consistent with a conclusion
14 that a chemical -- that humans are never exposed to
15 a chemical at a level that can cause them to get
16 cancer?

17 MR. LITZENBURG: Object to --

18 Q. Do you agree or disagree?

19 MR. LITZENBURG: -- form.

20 If you understand it . . .

21 A. I don't understand the question.

22 Q. Yes, sir.

23 A. I think we talked about it earlier this
24 morning, but I think this -- you're asking the
25 question differently, so I don't understand it.

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1 Q. Do you believe that IARC's conclusion means
 2 that IARC thinks that human beings actually get
 3 cancer from glyphosate and are at risk of getting
 4 cancer from glyphosate at the levels at which humans
 5 are actually exposed to glyphosate?
 6 A. I think their -- the IARC conclusion speaks
 7 for itself. They said it's probably carcinogenic to
 8 humans. That's what they said.
 9 Q. Yes.
 10 And do you believe that that means that
 11 IARC thinks that humans actually get cancer from
 12 humans [sic] and can actually get cancer at the
 13 levels at which humans are exposed?
 14 A. I think they believe it's probably
 15 carcinogenic. That's what they believe. They
 16 obviously clearly did not say it's absolutely
 17 100 percent positively. They said it's probably.
 18 Q. Yes. And I'm asking if you understand what
 19 "carcinogenic" means in a hazard assessment.
 20 Does it mean that it's actually out there
 21 causing human cancers or that there's a theoretical
 22 possibility of this substance causing cancer in
 23 human beings?
 24 MR. LITZENBURG: You have asked and
 25 answered four times now. Just because you're

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1 not getting the answer that you want --
 2 A. I've answered the question.
 3 MR. LITZENBURG: -- doesn't mean you get to
 4 keep asking it.
 5 Q. Go ahead, sir.
 6 A. I have answered this question before, and I
 7 believe that, yes, there is enough evidence to
 8 suggest that glyphosate probably causes human
 9 cancer.
 10 Q. Do you understand that IARC -- according to
 11 IARC, the Monographs program may identify cancer
 12 hazards even when risks are very low with known
 13 patterns of use or exposure?
 14 A. I did not know that.
 15 Q. An agent is considered a cancer hazard if
 16 it is capable of causing cancer under some
 17 circumstances. That's hazard.
 18 Risk measures the probability that cancer
 19 will occur, taking into account the level of
 20 exposure to the agent.
 21 Do you understand that distinction, sir?
 22 MR. LITZENBURG: Object to form. I'd like
 23 to know what you're reading from or have it
 24 marked.
 25 Q. The IARC Monograph.

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1 Go ahead, sir.
 2 The IARC Monograph questions and answers.
 3 Sir, my question to you is, do you
 4 understand that that's the difference between risk
 5 and hazard, according to IARC?
 6 A. Okay. I would like to read what they --
 7 what they wrote before I answer your question.
 8 Q. It's not in Monograph 112.
 9 A. Okay.
 10 MR. LITZENBURG: I thought you just said it
 11 was.
 12 Q. Well, here's my question.
 13 A. Okay. I thought you just said this was in
 14 the Monograph.
 15 Q. The Monograph Q&A. It's a different
 16 document --
 17 A. Sure.
 18 Q. -- than the Monograph 112.
 19 Do you understand that the difference
 20 between hazard and risk, according to IARC, is that
 21 an agent is considered a cancer hazard if it is
 22 capable of causing cancer under some circumstances?
 23 A. Okay.
 24 Q. Whereas risk measures the probability that
 25 cancer will occur, taking into account the level of

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1 exposure to the agent.
 2 Do you understand that about IARC's --
 3 A. I understand the difference. I did not
 4 know that this is how they define it. But as you
 5 frame it right now, I understand the differences,
 6 yes.
 7 Q. Okay. And is your assessment of the hazard
 8 of glyphosate in non-Hodgkin's lymphoma the same as
 9 IARC's, i.e., that you have identified what you
 10 believe to be a cancer hazard, an agent capable of
 11 causing cancer under some circumstances, but you
 12 have not measured the probability that cancer will
 13 occur?
 14 MR. LITZENBURG: Object. That's a
 15 mischaracterization.
 16 You can answer.
 17 THE WITNESS: I can answer?
 18 MR. LITZENBURG: Yeah, I mean, if you . . .
 19 A. I believe that there is a hazard with the
 20 exposure to glyphosate and development of
 21 non-Hodgkin lymphoma. I cannot quantify what that
 22 hazard is, so I cannot tell you that people who are
 23 exposed to glyphosate have 50 percent risk versus
 24 2 percent risk.
 25 If that is what you're asking, I don't

1 believe I have sufficient information to quantify
 2 that risk, but I believe that risk exists.
 3 And part of the reason we -- you know, in
 4 epidemiology study, occupational studies, it's
 5 really important to look at the hazard as opposed to
 6 actual absolute risk is because they're a very easy
 7 preventable thing. It's an easy -- you know, to
 8 prevent the development of a particular cancer,
 9 which all oncologists would love to see, if you just
 10 say, you know, whether the risk is 1 percent or a
 11 10 percent or a 50 percent, you know what, it's
 12 great. We are going to eliminate this so you don't
 13 have that risk. Because that's an easy thing to do.
 14 So the actual absolute risk, it's not
 15 really as important. I mean, we wear seat belts not
 16 because we're going to get into a car accident every
 17 day, because in case we get in a car accident, the
 18 risk of dying is significantly lower.
 19 So I think, you know, that's why the
 20 absolute risk category is not as important in
 21 occupational hazards, in occupational studies,
 22 because we can eliminate that easily.
 23 Q. You can't give an opinion that an
 24 individual exposed to glyphosate has their risk of
 25 NHL go up by 1 percent or 45 percent or 90 percent

1 or any particular percent; right?
 2 A. No. I can say it will increase, but I
 3 don't know by how much percentage. So it's not
 4 zero.
 5 Q. But it could be 1?
 6 A. It could be 1. It could be 15. It could
 7 be 90.
 8 Q. Okay. And you have not made any attempt to
 9 quantify how much the risk increases for someone
 10 exposed to glyphosate, in your opinion; right?
 11 A. I think it's difficult to quantify. I
 12 think it's -- you know, it's difficult for me, as a
 13 clinician, as a researcher, to actually quantify
 14 that risk. But I think the presence of the risk is
 15 sufficient because it's a preventable strategy to --
 16 to reduce the risk.
 17 Q. Do you know that IARC has stated that the
 18 term "probable" in "probable human carcinogen" -- in
 19 the phrase "probably human carcinogen" has no
 20 quantitative significance?
 21 A. I'm not surprised.
 22 Q. Okay. And does it have no quantitative
 23 significance in your evaluation as well?
 24 A. Yeah, I just said it's hard -- it's
 25 impossible to really quantify that risk. Again,

1 your questions are only answered by a prospective
 2 randomized trial where you expose folks to
 3 glyphosate and don't expose others and you follow
 4 them for whatever years you decide and see what is
 5 the risk difference. That's something that will
 6 never happen.
 7 To accurately assess the risk and to
 8 quantify the risk, that cannot happen. That is
 9 impossible to -- all that we can say is that there
 10 is evidence that the risk exists, but it could be
 11 1 percent to 99 percent. It's not 100 percent, and
 12 it's not zero.
 13 Q. Do you have an opinion based on everything
 14 you've reviewed and everything you know that there
 15 is any way to tell in a particular person whether an
 16 exposure to glyphosate or something else caused
 17 their non-Hodgkin's lymphoma?
 18 A. I think sometimes, if you have certain
 19 individuals that have been exposed more and -- you
 20 know, it's possible that this might actually -- I
 21 mean, I think we've reviewed a couple of studies
 22 where you have more than ten days, less than ten
 23 days, more than two days, less than two days.
 24 So there is a possibility, although I
 25 acknowledge that it's not always that -- you know,

1 the dose response is very vague in -- in these type
 2 of studies. But there is a possibility that
 3 sometimes, if you are more exposed for a longer
 4 period of time, you could logically have more risk.
 5 I mean, if you are not wearing protective
 6 clothes or things like that, I mean, I -- it's --
 7 you know, you have some skin abrasions or skin
 8 damage. I mean, so there are certain things that
 9 might lend me to believe that this particular
 10 individual has a higher risk than another
 11 individual. Each case is very different, obviously,
 12 but that's why you can't really quantify the risk,
 13 because it's just one element. It's one factor.
 14 Q. Based on everything that you reviewed and
 15 everything that you know, is there any way to tell
 16 that someone opposed to glyphosate and to other
 17 substances capable of causing non-Hodgkin's lymphoma
 18 developed non-Hodgkin's lymphoma because of
 19 glyphosate rather than those other substances?
 20 A. I think you'll have to look at each
 21 individual case. It's -- you know, it's hard to --
 22 it's hard to speculate. You'll have to look at what
 23 other -- what are these substances, how often he or
 24 she was exposed to these substances, how were they
 25 applied, top -- I mean, it's just -- each substance

1 is different. So it's hard to really tell.

2 I think a lot of times we can try to
3 exercise clinical judgment and scientific evidence
4 to try to tease out which -- which is probably the
5 more offending factor to the extent possible.
6 Sometimes we're successful; sometimes we're not.

7 Q. So it would be a matter of weighing the
8 different exposures, how much they were exposed, how
9 toxic we believe the substances to be?

10 A. Which are the substances, do these
11 substances really cause -- cause lymphoma or not,
12 how often were they -- he or she were exposed to,
13 et cetera. You know what I mean. I mean, you just
14 have to look at the type of substances, the amount
15 of exposure, how they were applied. And then you
16 have to look at these substances where there's
17 really data that truly are associated with the
18 disease.

19 I mean, if the person is smoking heavily
20 and drinking heavily and they're doing glyphosate, I
21 don't have any evidence that smoking and alcohol
22 necessarily cause lymphoma. So just because they're
23 smoking and drinking, it doesn't mean that they're
24 confounding factors. So I think you'd have to look
25 at each case individually.

1 Q. Do you have the opinion, to a reasonable
2 degree of medical certainty, that any substance
3 other than glyphosate has a synergistic effect or an
4 additive effect with glyphosate to increase the risk
5 of non-Hodgkin's lymphoma?

6 A. I don't think this has been adequately
7 studied in the literature.

8 Q. So you don't have that opinion at this
9 time?

10 A. At this time, I don't have this opinion.

11 Q. The next epidemiology study that you
12 mentioned, sir, is the Orsi study?

13 A. Do you have that?

14 Q. I'll get it out if you need it.

15 Do you remember that it has an odds ratio
16 of exactly 1?

17 A. I'll look at what I wrote here. I don't
18 write in my note here the odds ratio. I think what
19 I wrote, a French study that spanned 2000 to 2004 by
20 Orsi, L. suggested increased risk of developing
21 Hodgkin lymphoma and myeloma in patients exposed to
22 pesticides. This study was conducted amongst six
23 centers looking at incident cases with lymphoid
24 neoplasm diagnosis in patients aged 18 to 75 years.
25 Control cases were patients with rheumatology and

1 orthopedic problems within the same participating
2 institutions.

3 And I did acknowledge here in this study
4 little evidence was shown in this study as to the
5 relationship between NHL and exposure, but there was
6 evidence with HL and myeloma. And myeloma is
7 obviously a B-cell type of cancer that --

8 Q. Do you have an opinion, to a reasonable
9 degree of medical certainty, that glyphosate causes
10 multiple myeloma?

11 A. I did not investigate multiple myeloma to
12 the extent it allows me to give you an opinion that
13 I'm comfortable with at this point.

14 Q. Okay. Do you have the opinion that
15 glyphosate causes any malignancy other than
16 non-Hodgkin's lymphoma?

17 A. I did not look at other malignancies. So
18 it -- it may cause other malignancies. It may not.
19 But I only looked at non-Hodgkin lymphoma.

20 Q. And do you recall that the point estimate
21 for non-Hodgkin's lymphoma with glyphosate in the
22 Orsi study was 1.0?

23 A. If you can show me this, I can look at it.

24 Q. Sure.

25 A. In my report, I did not put the estimate.

1 Q. Okay.

2 A. But I did acknowledge that it was little
3 evidence. And I wrote here, "This might be a sample
4 effect as only 244 cases of NHL were evaluated in
5 this study." So my opinion was that it wasn't
6 enough sample size.

7 Q. When you say "sample effect," you mean the
8 study was too small?

9 A. That's what I -- that's what I believe I
10 concluded when I reviewed the study.

11 Q. Okay. I mean, that's what you mean by
12 "sample effect"; right?

13 A. Yes.

14 (Nabhan Exhibit 19 marked for
15 identification.)

16 Q. So I've marked as Exhibit 19 the Orsi
17 study. If you'll look at Table 3, you'll see the
18 non-Hodgkin's lymphoma results.

19 A. Yeah, 244 cases, 436 control. So I thought
20 the number of cases were -- was pretty small.

21 Q. And you saw that the odds ratio for
22 non-Hodgkin's lymphoma associated with glyphosate
23 was 1.0?

24 A. One -- one second. 1.0, yes.

25 Q. Which, by any definition, is no effect?

1 A. Correct.
 2 Q. Okay. The Cocco study -- I'm just trying
 3 to finish up your studies, sir -- from 2013 had only
 4 four individuals with exposure to glyphosate and
 5 non-Hodgkin's lymphoma?
 6 A. If you don't mind, I'd like to look at it
 7 again.
 8 Q. Yes, sir.
 9 (Nabhan Exhibit 20 marked for
 10 identification.)
 11 A. Thank you. Okay. I think this was a
 12 larger sample size study in terms of the number of
 13 lymphoma and the number of cases and was in several
 14 European countries.
 15 Q. And there were four exposed cases, Table 4?
 16 A. Which table that is?
 17 Q. 4?
 18 A. Table 4. Yeah, four cases, I see that.
 19 Q. Two controls --
 20 A. Right.
 21 Q. -- and a nonsignificant and wide-range
 22 confidence interval; right?
 23 A. I just show 3.1, but crosses the 1.
 24 Q. And did you believe this study to have
 25 power and significance to you with four cases and

1 A. The number of all cases, right.
 2 Q. -- exposed cases to the -- to the substance
 3 under investigation goes down dramatically, then you
 4 have a bigger sample size problem; correct?
 5 A. What I'm saying is when you have enough
 6 numbers, denominator-wise, of patients that you are
 7 looking at with the disease or without the disease,
 8 the likelihood of detecting something with the
 9 positive -- with the positive exposure, and that
 10 is -- with a higher odds ratio, becomes more likely.
 11 So I only use this in contrast of the Orsi
 12 paper. The Orsi paper has an odds ratio of 1.0, but
 13 only 244 cases that were looked at with NHL. The
 14 Cocco paper looked at over 2,000 cases of NHL. And,
 15 yes, they found small number of exposure to
 16 glyphosate versus non, but the odds ratio went up.
 17 So it's only used in my expert report in
 18 way to contrast the numbers. When you have higher
 19 numbers of patients that you are looking at, it is
 20 possible that the odds ratio will change.
 21 Q. What it means is that you had a population
 22 that was much less exposed to glyphosate in that
 23 time period; right?
 24 A. That's one way of looking at it, but that's
 25 not the only way.

1 two controls?
 2 A. I mean, yeah -- no. I mean, I think the --
 3 here's what you would take from this study: So when
 4 you look at the Orsi study, the one before, we had
 5 only 244 cases of lymphoma and we saw an odds ratio
 6 of 1.0.
 7 When you increase the sample size and you
 8 looked at 2,348 non-Hodgkin's lymphoma cases, 2,462
 9 control cases, the odds ratio went up to 3.1.
 10 Yes, it crosses the 1. Yes, the 95 percent
 11 confidence interval is 0.6 to 17.1, but it just goes
 12 to show that you that the Orsi study that we just
 13 reviewed that was odds ratio of 1.0 was so because
 14 of the number -- the number is too small, the number
 15 of cases.
 16 So I think all what you can conclude from
 17 this trial, the Cocco trial, the number obviously
 18 four cases and two control, these numbers are small.
 19 But it just goes to show you that, sometimes when
 20 you have higher number in the denominators, you
 21 could have a significant odds ratio or at least
 22 above 1 odds ratio despite the fact it crosses the
 23 1.
 24 Q. When the sample size, meaning the number of
 25 cases --

1 Q. It's the right way, isn't it? Because the
 2 relevant factor that you look at when you're looking
 3 at the power and predictable value of a study is the
 4 number of exposed cases for the relevant exposure?
 5 A. But that's one study. I mean, there are
 6 other studies that we looked at that had different
 7 numbers and different -- again, I mean, if we are
 8 looking at this particular study, I told you how I
 9 used it personally. I didn't use it to look
 10 necessarily at the percentage of glyphosate exposed
 11 individuals and how often did they develop --
 12 non-Hodgkin's lymphoma. I looked at in contrast to
 13 a previous study, because I was trying to understand
 14 the one that had significantly low number.
 15 I think we reviewed over 10 or 12 studies
 16 earlier that have different numbers. So I don't
 17 want to take this out of context as the only --
 18 again, this is part of what I looked at, not the
 19 only thing I looked at.
 20 Q. Certainly. I'm just trying to understand
 21 what you're telling me, sir --
 22 A. Sure.
 23 Q. -- that the number of people in the study
 24 who didn't have an exposure to glyphosate and
 25 non-Hodgkin's lymphoma is relevant to the validity

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1 of the point estimate that you get in the study,
 2 independently of the number of exposed cases?
 3 A. I think you increase -- I think you're more
 4 interested in the cases that were diagnosed and
 5 exposed versus the cases that were not diagnosed and
 6 exposed. I mean case control, when you are looking
 7 at things.
 8 Q. Cases that were diagnosed and exposed in
 9 this is four?
 10 A. Four, yeah.
 11 Q. Very low number; right?
 12 A. It's small. Very low number. I
 13 acknowledge that. And I -- you know, nobody can
 14 argue that four is a large number, but I was trying
 15 to explain that I used this type of paper just for
 16 simple fact it's a lot of numbers, it's over 2,000
 17 patients -- 2,000 patients and 2,000 control. So
 18 obviously it's a large number.
 19 Yes, the exposure rate was not as high.
 20 But, again, that's when you have higher number and
 21 so forth, you can have higher odds ratio, just
 22 similar to -- you know, in contrast with the
 23 previous paper. That's really the -- how I use this
 24 paper.
 25 VIDEOGRAPHER: Can I take a moment to

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1 change discs?
 2 MR. GRIFFIS: Yeah.
 3 VIDEOGRAPHER: Ending Disc No. 3 of the
 4 deposition of Dr. Chadi Nabhan. Off the record
 5 at 3:34 P.M.
 6 (Recess taken from 3:34 P.M. to
 7 3:37 P.M.)
 8 VIDEOGRAPHER: And beginning Disc No. 4 of
 9 the deposition of Dr. Chadi Nabhan. We are
 10 back on the record at 3:37 P.M.
 11 BY MR. GRIFFIS:
 12 Q. Sir, the last epidemiology study that you
 13 mentioned is the Kato study, and let me know if we
 14 need to get it out, but that involves a point
 15 estimate for all pesticides put together; correct?
 16 A. Yeah, I'd like you to give me a sample,
 17 please.
 18 Q. Sure.
 19 (Nabhan Exhibit 21 marked for
 20 identification.)
 21 Q. Are you ready, sir?
 22 A. I'm ready.
 23 Q. Okay. So the Kato study involved women who
 24 worked at farms where pesticides were used; correct?
 25 A. Yes.

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1 Q. And you reported an odds ratio of 2.12,
 2 which was statistically significant at a 95 percent
 3 confidence interval; correct?
 4 A. That is correct.
 5 Q. And glyphosate was not broken down in this
 6 study; right?
 7 A. I don't believe they looked at each
 8 particular one. This was more in general with
 9 pesticides.
 10 Q. And what did you rely on this study for
 11 informing your opinion, sir?
 12 A. Again, I think similar to other studies
 13 that did not look at particular compounds but they
 14 looked at pesticides as a whole, given the fact that
 15 the glyphosate in its nature is a -- is, again,
 16 could be considered part of the category. So that
 17 just solidifies the opinion.
 18 Q. And do you have the opinion that glyphosate
 19 forms any part of the risk that is purported to be
 20 measured by this study?
 21 A. As I said, they did not tease out
 22 glyphosate by itself.
 23 Q. You can't say that it increased or
 24 decreased or had no effect on the risk measured in
 25 the study; correct?

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1 A. Based on this study, I cannot say that.
 2 MR. GRIFFIS: I know you just changed the
 3 tape, but I need to get organized for the next
 4 phase, so let's take a break.
 5 VIDEOGRAPHER: Going off the record at
 6 3:40 P.M.
 7 (Recess taken from 3:40 P.M. to
 8 3:50 P.M.)
 9 VIDEOGRAPHER: And we are back on the
 10 record at 3:50 P.M.
 11 BY MR. GRIFFIS:
 12 Q. Okay. Sir, we've just gone through the
 13 entirety of your -- the section of your expert
 14 report entitled "Epidemiological studies," which
 15 went from page 11 to page 15. And now I'd like to
 16 look at the next section entitled "Meta-analyses."
 17 And you talked about two meta-analyses, one
 18 by Schinasi and León and one by Chang and Delzell
 19 from 2016; correct?
 20 A. Correct.
 21 Q. You said that the meta-analysis by Schinasi
 22 and León found an association between glyphosate and
 23 B-cell lymphoma with an odds ratio of 2.0, and this
 24 was the same odds ratio for diffuse large B-cell
 25 lymphoma; correct?

1 A. Correct.
 2 Q. And why did you give that particular point
 3 estimate and not the other point estimates? Why did
 4 you select that one from the Schinasi and León
 5 meta-analysis, sir?
 6 A. Do you have a copy of this? I want to make
 7 sure I -- a copy of the meta-analysis, please.
 8 Q. Maybe.
 9 (Nabhan Exhibit 22 marked for
 10 identification.)
 11 A. Thank you.
 12 Q. Marked as Exhibit 22, sir.
 13 A. Sure. So I think this meta-analysis, they
 14 started with 858 articles. 44 were included in the
 15 qualitative analysis, and of these 20 papers,
 16 provided estimates of association with herbicide
 17 chemical groups or active ingredients. And I think
 18 you -- I go on to mention, of the included papers,
 19 several had data on glyphosate specifically, and I
 20 cite the papers that the meta-analysis used for
 21 glyphosate in my expert report. And these studies
 22 were performed in the U.S., Canadian, Europe,
 23 Australia, and New Zealand.
 24 Q. And you say you cited the ones that they
 25 relied on for the meta-analysis in your expert

1 between association and causation?
 2 Q. I'm talking about the terms that you chose,
 3 "plausible association," and I asked you to explain
 4 what you meant by it.
 5 A. To a reasonable degree of certainty, it
 6 does mean causation to me.
 7 Q. Okay. So when you used it here, you meant
 8 causation. And you said between glyphosate and NHL
 9 evolution and development.
 10 Did you mean by "NHL evolution and
 11 development" something other than glyphosate causes
 12 NHL?
 13 A. Well, causing is development; evolution is
 14 the disease that progresses or changes course after
 15 it's being developed. So, I mean, I think there
 16 is -- it's -- you know, when you look at disease
 17 like diffused large B-cell lymphoma, sometimes it
 18 starts as a diffused large B-cell lymphoma,
 19 occasionally it transforms from a low-grade lymphoma
 20 to a diffuse large B-cell lymphoma.
 21 So it's not clear, you know, how much of
 22 this disease is evolved from a different entity
 23 within lymphoma to diffused large B-cell lymphoma
 24 versus just start de novo as the large cell
 25 lymphoma.

1 report, you mean the ones listed on pages 15 to 16?
 2 A. Yeah. I mean, I cite several of the
 3 included papers, several had data on glyphosate.
 4 Q. So Cantor, Cocco, DeRoos 2003, DeRoos 2005,
 5 Eriksson, Hardell, and Orsi; correct?
 6 A. Yes.
 7 Q. And we've discussed all of those today;
 8 right?
 9 A. We have.
 10 Q. What -- and you say, after you discuss the
 11 findings of the meta-analysis briefly, that this
 12 represented "a summary of the data published in the
 13 preceding 25 years and solidifies a plausible
 14 association between glyphosate and NHL evolution and
 15 development." Correct?
 16 A. Yes.
 17 Q. I'd like to understand some of your terms,
 18 first of all, sir. What do you mean by a "plausible
 19 association"?
 20 A. Plausible association means that there is a
 21 causation between the compound that we are looking
 22 at and the disease under investigation.
 23 Q. Plausible association means the same as
 24 causation to you?
 25 A. So you mean between -- the difference

1 So I think the meta-analysis suggests that
 2 there is this association between the compound and
 3 the disease.
 4 Q. Are there any studies or any scientific
 5 articles, sir, on the subject of NHL evolution with
 6 glyphosate as you have just described it?
 7 A. No. I'm -- I'm not aware. It's very
 8 difficult to look at that, because the evolution
 9 requires continued investigation pathologically. So
 10 when lymphoma changes from one entity to a different
 11 type of lymphoma, you have to confirm this with
 12 repeated prospective biopsies, and this can't be
 13 done. So you just speculate. Again, this is just a
 14 speculation that --
 15 Q. Yes, sir.
 16 A. We have diffused large B-cell lymphoma, but
 17 is it really the de novo large cell lymphoma versus
 18 something that's evolved from something else.
 19 That's really all that I meant.
 20 Q. Okay. So you are not claiming, to a
 21 reasonable degree of medical certainty, that you
 22 have evidence that glyphosate causes evolution or
 23 development of NHL from one phase to another phase
 24 as opposed to just being associated with NHL in
 25 general; right?

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1 A. Let me rephrase --
 2 Q. Yes, sir.
 3 A. -- to be very accurate. Diffused large
 4 B-cell lymphoma sometimes starts as diffused large
 5 B-cell lymphoma and sometimes it becomes diffused
 6 large B-cell lymphoma from a different type of
 7 lymphoma. So it could be some indolent lymphomas.
 8 And I wrote this in my background on NHL in my
 9 expert report. Some indolent lymphomas transform
 10 into the large cell lymphoma at a rate of about 5 to
 11 10 percent per year after initial diagnosis.
 12 Q. Right. And this isn't the other example.
 13 There are other kinds of non-Hodgkin's lymphoma that
 14 transform into different types as well?
 15 A. That is correct. So there is this
 16 transformation thing.
 17 So, really, all that I meant by this is
 18 that this -- the fact that I talked about diffused
 19 large B-cell lymphoma, it's fair to acknowledge that
 20 this could have evolved from something else I just
 21 don't know about.
 22 Q. Okay.
 23 A. I am not aware of a particular study that
 24 looked specifically at the evolution per se, because
 25 to do that you will need to have a cohort of

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1 patients who have indolent lymphoma, all of them,
 2 and you expose all of them to glyphosate. And then
 3 you follow them prospectively and see what's the
 4 percentage of these folks that transform into large
 5 cell lymphoma. I think we both can agree that this
 6 can't happen.
 7 Q. Right.
 8 A. So, you know, that's really all that I
 9 meant by this statement.
 10 Q. And all day, sir, you and I have been
 11 talking about your expert opinion and details of
 12 your expert opinion that glyphosate is causally
 13 associated with non-Hodgkin's lymphoma in general.
 14 And all I'm asking right now is whether you intend
 15 to testify, to a reasonable degree of medical
 16 certainty, that you have evidence that glyphosate
 17 causes NHL transformation or development in
 18 addition.
 19 A. It can. Every case is different, as I
 20 said, you cannot rule it out; you cannot rule it in.
 21 So if you have somebody who has indolent
 22 non-Hodgkin's lymphoma, whatever that disease is,
 23 and you just spread them with glyphosate for the
 24 next five days and then the disease transforms to
 25 something else, can you rule out glyphosate could

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1 have may caused this? You can't.
 2 So I think every case is different. So,
 3 you know, your first question was is there evidence
 4 that specifically looked at this? I am not aware of
 5 that, but that doesn't take away that it could
 6 actually happen in a particular case. I'll have to
 7 look at the particular clinical scenario, the
 8 particular patient, the particular situation where I
 9 can really provide you with an accurate medical
 10 opinion.
 11 Q. And you know of no evidence -- no
 12 scientific evidence that that happens as a general
 13 proposition; correct?
 14 A. For the general population, no. But it
 15 could happen in some patients. So every case is
 16 different. I'm going to say that again for the
 17 third time. Every case is different.
 18 So, yes, glyphosate, if it's exposed to
 19 somebody who has indolent disease, could transform
 20 into an aggressive disease. You can't say no. But
 21 I don't have a population base study to say that,
 22 you know, the risk of transforming from an indolent
 23 to an aggressive lymphoma is 15 percent with
 24 glyphosate. I don't have that.
 25 Q. Do you know the difference between the

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1 terms "general causation" and "specific causation"?
 2 MR. LITZENBURG: Object to form.
 3 A. Would you explain to me, please.
 4 Q. Sure. When I use the term, sir, what I
 5 mean by "general causation" is evidence that a
 6 particular substance can cause or does cause a
 7 particular outcome in general.
 8 A. Okay.
 9 Q. So all day today we've been talking about
 10 your general causation opinion that glyphosate --
 11 A. Correct.
 12 Q. -- can cause non-Hodgkin's lymphoma.
 13 Specific causation means an opinion by
 14 someone that this particular substance caused this
 15 patient's non-Hodgkin's lymphoma or caused some
 16 event like the transformation of a particular
 17 patient's non-Hodgkin's lymphoma. Okay?
 18 A. Okay.
 19 Q. Okay. So -- and I'm trying to understand
 20 what you just told me. Is it -- is what you're
 21 telling me that you can have -- without evidence
 22 of -- without general causation evidence that
 23 glyphosate can cause a transformation of
 24 non-Hodgkin's lymphoma from one type to another or
 25 an evolution or development of stages, that you

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1 could nevertheless testify in a particular case as
 2 to specific causation, that although -- although I
 3 don't have scientific evidence that glyphosate
 4 causes transformation, I can testify that it's my
 5 opinion, to a reasonable degree of medical
 6 certainty, that it did in this particular patient?
 7 A. Yes, I can. I might.
 8 Q. Have you ever formed such an opinion for
 9 anyone?
 10 A. Formed an opinion of what?
 11 Q. That glyphosate caused a transformation?
 12 A. I reviewed a case that -- right --
 13 MR. LITZENBURG: That was just prognostics.
 14 A. Prognostics. Just provided an opinion in
 15 terms of the prognosis of the actual --
 16 MR. LITZENBURG: Yeah, they have your
 17 declaration.
 18 A. Right.
 19 Q. So you are talking about the D. Johnson
 20 case?
 21 A. Yeah. That's what I just provided --
 22 Q. The Dewayne Johnson case?
 23 A. Yes. That's really the only thing that I
 24 looked at from a prognostication standpoint.
 25 Q. I'm not just talking about in connection

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1 with this litigation.
 2 A. No, I am not.
 3 Q. I'm talking your clinical practice as well,
 4 sir.
 5 Have you ever formed the opinion that
 6 glyphosate caused a -- caused a patient with
 7 non-Hodgkin's lymphoma of a particular type to
 8 transform to another type --
 9 A. In my practice I have -- in my practice, I
 10 have not had that. In my personal practice, I have
 11 not seen -- I have not had a patient that had
 12 indolent lymphoma that had glyphosate and then
 13 subsequently changed. I have not had that sequence
 14 of events.
 15 Q. Okay. Outside of your practice, have you
 16 done that?
 17 A. How would I do that outside my practice?
 18 Q. Mr. Litzenburg just said that you did a
 19 prognostic thing with Dewayne Johnson --
 20 A. Yeah. I looked at the case from a
 21 prognostic standpoint. I was asked to take a look
 22 at the case and provide an opinion in terms of the
 23 life expectancy.
 24 Q. And I know about that.
 25 Did you form an opinion about

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1 transformation?
 2 A. I did not.
 3 Q. Okay. The meta-analysis by -- oh, I'm
 4 sorry. We didn't quite finish with the --
 5 A. Sure.
 6 Q. -- Schinasi and León meta-analysis here. I
 7 now understand the terms that you used in that
 8 sentence, that it solidifies a plausible association
 9 between glyphosate and NHL evolution and
 10 development.
 11 What did the Schinasi and León
 12 meta-analysis add to the evidence that you were
 13 weighing in reaching the conclusions that you did,
 14 sir?
 15 A. I mean, I think the -- we both have
 16 acknowledged, in all of the studies that we
 17 reviewed, that there are limitations to any one
 18 individual study. I mean, I think, you know, we can
 19 all pick each study apart and realize the
 20 limitations. And what the meta-analysis attempts to
 21 do is to overcome some of these limitations by
 22 looking at the aggregate evidence, by looking at all
 23 of these studies together.
 24 So I think the -- you know, when you look
 25 at this particular meta-analysis, it showed an odd

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1 ratio of 2.0 with a confidence interval between 1.1
 2 and 3.6. So, to me, it really showed that when you
 3 look at the data in combination of everything
 4 together, you might be able to demonstrate
 5 statistical significance because you're able to look
 6 at everything combined. You try to overcome the
 7 limitations of each individual study. So you have
 8 larger numbers. You have larger denominators,
 9 larger cases, larger controls, et cetera.
 10 So really that's -- that's what this
 11 meta-analysis gave me, that when you looked at the
 12 combined evidence, the odds ratio was significant
 13 and there's an association and causality between
 14 glyphosate and non-Hodgkin's lymphoma.
 15 Q. And what does it mean to you to have a
 16 meta-analysis that, in your opinion, yields a
 17 statistically significant result?
 18 A. It means that there is an association and
 19 causality between what the authors were looking at
 20 and the disease they were looking at.
 21 Q. Okay. And did you not have that conclusion
 22 before you saw the meta-analysis, that there was an
 23 association and causality?
 24 A. Well, I did, but you need to have -- you
 25 need to solidify your opinion. Again, you know, I

1 didn't look at one paper -- if I looked at one paper
2 in isolation, I may not have had that conclusion.

3 And so you look at all of the papers, the
4 meta-analysis, the additional information, to form
5 the opinion. So I -- you know, my opinion is not
6 formed based on each isolated paper. It's really
7 based on the aggregate collection of evidence.

8 Q. Okay. Now, since we talked about each one
9 of these individual papers --

10 A. Right.

11 Q. -- what did the meta-analysis tell you that
12 you had not learned by looking at each of the
13 individual papers?

14 A. It showed a statistical significance that
15 some of these papers did not have that. As you
16 articulated earlier, some of them did cross the 1.
17 So at least this -- this odds ratio of 2.0 not
18 crossing the 1 is significant, and it overcomes some
19 of the limitations that the previous individual
20 studies suffered from.

21 Q. Now I want to ask you about the Chang and
22 Delzell meta-analysis and what the Chang and Delzell
23 meta-analysis added to your assessment of the
24 evidence. And maybe it's the same as what Schinasi
25 and León did.

1 A. When I get reviewers usually asking me, why
2 is this notably?

3 Q. The overall risk of non-Hodgkin's lymphoma
4 was 1.3 with a confidence interval at -- the lower
5 bound was at 1.0, which would not be statistically
6 significant; correct?

7 A. It's borderline, I would say.

8 Q. And then you broke it up by subtype.

9 A. Well, they broke it up.

10 Q. B-cell -- well, you --

11 A. Right.

12 Q. -- listed their breakdown.

13 A. I just listed their breakout, and they
14 have -- yeah.

15 Q. B-cell 2.0, CLL 1.3, and follicular
16 lymphoma 1.7. And two of those were not
17 statistically significant and one was, sir.

18 Do you have the opinion that the
19 epidemiological evidence shows a difference in the
20 manifestation of non-Hodgkin's lymphoma across
21 subtypes based on exposure to glyphosate?

22 A. Yeah. I don't think there's sufficient
23 evidence, frankly, to have robust conclusions based
24 on 60 subtypes, and I don't think it's honestly
25 doable. It's impossible. And I think we alluded to

1 A. If you are going to ask me about the actual
2 data inside --

3 Q. Okay.

4 A. If it's just about my report, it's fine.

5 Q. Well, let's try from your report.

6 A. Sure. So, again, it has a risk ratio of
7 1.3 and original studies, which report PubMed,
8 Google Scholar, with additional references that were
9 found in the bibliography of review articles.
10 Collectively, 19 articles were included, as well as
11 one abstract and one letter to the editor. When
12 analyzing NHL by subtype, the risk ratio for B-cell
13 was 2.0, for CLL 1.3, follicular 1.7, and no
14 increase over HL, Hodgkin's lymphoma.

15 Q. You said "Notably, no increased risk for
16 Hodgkin's lymphoma was done." Why was that notably?

17 A. I need to quit the "notably." That's one
18 of my writing skills I need to work on. I use a
19 lot --

20 Q. You shouldn't say "notably" so much?

21 A. I've been told I say notably, surprisingly.
22 I'm -- you know -- I'm working on that.

23 (Laughter.)

24 Q. All right. I'll leave you alone about it
25 now.

1 this earlier this morning.

2 Q. And everyone who's tried to stratisfy
3 [sic], of course, has not gone to 60; they've gone
4 to 3 or 4 like this B-cell.

5 A. Yeah. It's very difficult. Right? I
6 mean, follicular lymphoma is a form of B-cell, for
7 example. But they try to look at alone. CLL is,
8 quote/unquote, a form of B-cell lymphoma, although
9 it's leukemia.

10 So I think it's very difficult to look at
11 the subtypes because there are so many right now.
12 And because of the heterogeneity but also because of
13 the fact that, in order for you to be convinced of
14 the subtypes, you need to have pathologic
15 confirmation of each particular patient, each
16 particular subtype. And many patients don't even
17 know what type of lymphoma they have. They just
18 say, I have non-Hodgkin's lymphoma. So it's just a
19 very difficult exercise to do.

20 Q. On page 18 of your expert report, you
21 talked about -- when you were talking about the EPA
22 SAP panel review, you mentioned that the panel
23 recommended that the EPA talk to the AHS, the
24 Agricultural Health Study investigators, to
25 determine whether updated data on incidence of NHL

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1 and other cancers are available.
 2 And why did you put that in your expert
 3 report?
 4 A. I think, you know, it's -- it's -- one of
 5 the things that we have to acknowledge about the
 6 limitation of the Agricultural Health Study. It is
 7 the only prospective study in my review that I was
 8 able to find, but it does have limitation with the
 9 short follow-up time of 6.7 years. And many of the
 10 patients in the Agricultural Health Study were
 11 younger versus patients who are diagnosed usually
 12 with non-Hodgkin's lymphoma. 70 percent were
 13 younger than the age of 70, 46 percent were younger
 14 than the age of 50.
 15 So I think it was important to highlight
 16 this limitation and the fact that the EPA wanted to
 17 get a follow-up, if available, and published.
 18 (Nabhan Exhibit 23 marked for
 19 identification.)
 20 Q. Sir, do you know who Aaron Blair is?
 21 A. Aaron Blair -- is he IARC? No, he is --
 22 the names are starting to blur a little bit.
 23 Q. Aaron Blair was the chief investigator for
 24 the Monograph 112.
 25 A. Okay. Yes. IARC.

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1 Q. He headed it up.
 2 A. I was right for a change.
 3 Q. And he is on the Agricultural Health Study
 4 as well.
 5 A. Yes, correct.
 6 Q. You see his name on the front of this
 7 draft, March 15, 2013, draft on lymphoma risk and
 8 pesticide use in the Agricultural Health Study;
 9 correct?
 10 A. He's one of the coauthors, yes.
 11 Q. Have you seen this document before?
 12 A. I have never seen this document before.
 13 Q. Okay. Let's take a look at it. This was a
 14 document, sir, that I'll represent was produced and
 15 marked as an exhibit at the deposition of Aaron
 16 Blair --
 17 A. Okay.
 18 Q. -- where he produced it as additional
 19 follow-up data that was available on glyphosate and
 20 other exposures from the Agricultural Health Study?
 21 A. So this is an actual paper that was
 22 published somewhere?
 23 Q. It was not published, sir. It was a draft
 24 that was never published. And we'll talk about that
 25 too.

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1 A. Okay. I'm just -- I thought I would have
 2 seen it if it was published. Okay.
 3 Q. Yes. If it had been published, you would
 4 have seen it.
 5 So the 213 draft manuscript was published
 6 with additional data -- five additional years of
 7 data, as you can see from page 3; correct?
 8 A. Yes, I see five years later, '98 to '04.
 9 Okay.
 10 Q. You see a discussion on page 9 of follow-up
 11 questionnaires being given, additional data was
 12 collected? Page 9, sir. I'm looking at the middle
 13 of the --
 14 A. Okay.
 15 Q. -- page numbers at the bottom of the middle
 16 of the page.
 17 A. Okay.
 18 Q. "So follow-up questionnaires were given and
 19 a data-driven multiple-imputation procedure was
 20 used, where there were -- where there were not
 21 responses."
 22 Do you see that?
 23 A. The middle paragraph, "A follow-up
 24 questionnaire" --
 25 Q. Yes, sir.

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1 A. -- "which ascertained pesticide usage
 2 enrollment was administered about five years after
 3 enrollment and completed by 63 percent" -- so not
 4 everybody -- "of the original participants."
 5 Q. Right.
 6 A. Okay. "And for participants who did not
 7 complete a Phase 2 questionnaire, a data-driven
 8 multiple-imputation procedure" -- what does that
 9 mean in English, "a data-driven multiple-imputation
 10 procedure"?
 11 Q. Well, it's a statistical method to figure
 12 out what the results would have been for the
 13 procedure -- for the questionnaires that were not
 14 returned based on the data that was provided.
 15 A. Well, you could critique this right away.
 16 Okay. That's fine. I mean, ultimately --
 17 Q. Take a look at the results on page 12, sir.
 18 A. Only 63 percent answered. Okay. Page 12.
 19 Okay.
 20 Q. Do you see that it says "The risk of
 21 non-Hodgkin's lymphoma increased significantly, and
 22 in near-monotonic fashion with age in the age S
 23 cohort."
 24 It's the very first sentence, under
 25 "Results."

1 A. Oh.
 2 So what do they mean by "monotonic
 3 fashion"? This is the first time I hear this --
 4 Q. Oh, monotonic, sir, in statistics means
 5 that as age increases --
 6 A. Oh, I see. Linear type of thing?
 7 Q. As a stepwise linear progression, yes.
 8 A. Okay. I mean, I'm not really sure that the
 9 linear thing, that we are 100 percent certain with
 10 occupational hazards, but that's fine. That's what
 11 they're saying. Okay.
 12 Q. Okay. And, I mean, that's about what you
 13 would expect. You would expect that as age
 14 increases, the incidence of non-Hodgkin's lymphoma
 15 would also increase; right?
 16 A. Agree. I just -- I don't know if it's
 17 linear. That's what I'm saying. Okay.
 18 Q. And I don't mean it's in a straight line --
 19 A. That's why --
 20 Q. I mean every age cohort, as it goes up, you
 21 have more non-Hodgkin's lymphoma. You would agree
 22 with that?
 23 A. I agree with that.
 24 Q. Okay. "And the number of livestock on the
 25 farm and whether cohort members" -- I'm looking at

1 I mean, again, that's what they're saying.
 2 Q. Okay. Yeah. Right. They said that it
 3 does.
 4 A. But all of this is new to me, so . . .
 5 Q. Yes, sir.
 6 Go to page 17, the middle paragraph.
 7 A. Okay.
 8 Q. And I'm looking at the last sentence in
 9 that paragraph. "In our study, we could not
 10 evaluate MCPA, but found no excess risk of NHL or
 11 its subtypes with the use of glyphosate" --
 12 A. I'm sorry. Where are you looking? Page
 13 17?
 14 Q. The last sentence --
 15 A. Last sentence.
 16 Q. -- in the middle paragraph on page 17,
 17 starting "In our study."
 18 Do you see that?
 19 A. This is page 17. I don't see "In our
 20 study." Where is it? Oh, here it is. That second
 21 paragraph.
 22 Okay. I see it.
 23 Q. "In our study, we could not evaluate MCPA,
 24 but found no excess risk of NHL or its subtypes with
 25 the use of glyphosate, 2,4-D, or 2,4,5-T."

1 the end that paragraph -- "whether cohort members
 2 drove farm equipment with diesel engines
 3 significantly increased risk of non-Hodgkin's
 4 lymphoma."
 5 Does that make sense given what you know
 6 about the causes of non-Hodgkin's lymphoma on the
 7 farm, sir?
 8 A. Well, I mean, this one is looking at the
 9 number of livestock. I mean, so it's not just
 10 farming. They're trying to correlate the number of
 11 livestock with the increased risk. So I --
 12 obviously, I know that there is data on farmers and
 13 increased risk. I wasn't aware that the number of
 14 livestock correlates with increase of NHL.
 15 Q. It would increase exposure to a number of
 16 things, including animal viruses; right?
 17 A. I get that, but I didn't realize -- I mean,
 18 again, I'm not aware of data that the number -- so
 19 if you have five cattles versus ten, I didn't know
 20 that the ten necessarily increased risk by -- versus
 21 five. Just the fact that you have more number, it
 22 doesn't necessarily mean you're going to have more
 23 exposure to particular pathogens.
 24 It may appear this way, but unless you have
 25 actual -- what they're saying here is it does. So,

1 Do you see that, sir?
 2 A. I see that.
 3 Q. Okay. I know that, as you said, all this
 4 is new to you.
 5 Now let's turn to the data tables.
 6 A. I mean, you do recognize there are so many
 7 comments on the side that I have not seen; right?
 8 Q. Oh, yes. I know, sir.
 9 A. Okay. Which -- where do you want me to go
 10 now?
 11 Q. Let's go to page 31 first.
 12 A. Okay.
 13 Q. Table 2.
 14 A. Yes.
 15 Q. And this is "Pesticide exposure, lifetime
 16 days and intensity-weighted lifetime days, and the
 17 age-adjusted risk of NHL incidence, 1993 to 2008.
 18 So what this is is -- it's the same as the table we
 19 looked at in DeRoos 2005, the published AHS data
 20 with five less -- five fewer years that looked at
 21 lifetime days and intensity-weighted lifetime days
 22 with the new data in; right?
 23 A. Okay.
 24 Q. If you go to page 34, you see the data for
 25 glyphosate.

1 A. (Speaking sotto voce.)
 2 Okay. I just want to pull the DeRoos
 3 paper. Which one is -- okay.
 4 Q. So we looked earlier at the DeRoos 2005
 5 paper, which you've expressed a criticism of, that
 6 there wasn't enough follow-up in terms of years of
 7 follow-up, sir.
 8 And we looked at the table in which
 9 lifetime days and intensity-weighted lifetime days
 10 were assessed and saw that, in that table, there was
 11 no association between glyphosate and non-Hodgkin's
 12 lymphoma. I believe you testified that you could
 13 not use that table to support a hypothesis that
 14 glyphosate causes non-Hodgkin's lymphoma; correct?
 15 A. You're talking the DeRoos '05; right?
 16 Q. Yeah.
 17 A. I said that, yes.
 18 Q. Okay. So this is the corresponding table,
 19 sir, in the Alavanja 2013, the 2013 AHS data.
 20 MR. LITZENBURG: I object to the
 21 representation.
 22 Q. On page 34, do you see that they show
 23 glyphosate at no exposure, low exposure, medium
 24 exposure and high exposure levels for lifetime days
 25 and intensity-weighted lifetime days?

1 tertiles. And what constitutes an even tertile
 2 depends on the actual exposures of each individual
 3 in the tertile.
 4 A. My question is, these numbers between
 5 parentheses, if -- Table 34, low, medium, and high,
 6 what do these numbers represent? You have 20,
 7 65.75, 173.25. What are these numbers?
 8 Q. Those are a measure of days of exposure and
 9 intensity-weighted days of exposure.
 10 But my question is about the point
 11 estimate, sir, in the second and third columns.
 12 A. I just wanted to make sure we're comparing
 13 apples to apples. That's all. Okay.
 14 Q. In this chart in exhibit -- what exhibit is
 15 it? -- 23. Table 2, "Pesticide exposure, lifetime
 16 days and intensity-weighted lifetime days," there is
 17 no association between glyphosate and
 18 intensity-weighted or non-intensity-weighted
 19 lifetime days of exposure; correct?
 20 A. This table shows no association.
 21 Q. Okay.
 22 A. I would like to review this paper in more
 23 detail. But it's not a paper; it's not published.
 24 Q. On page 36, you see Table 3, "Pesticide
 25 exposure, lifetime days, and the age-adjusted risk

1 A. Yeah, but I -- I'm struggling in
 2 understanding how it is related to the Table 3 of
 3 DeRoos '05 in terms of the tertile. So in the
 4 DeRoos '05, Table 3, the first tertile is 1 to 20,
 5 the second tertile, 21 to 56. The other one is 57
 6 to 2678.
 7 I don't know how they're representing this
 8 here. They have none; low, 20; medium, 67.5; and
 9 high, 173.25. I don't know what these numbers mean.
 10 Q. Well, breaking it into three even tertiles
 11 would depend on what kind of underlying data you
 12 have.
 13 A. Well --
 14 Q. How the tertiles break out.
 15 A. -- I understand that, but the tertiles were
 16 years of use multiplied by the -- right? In the
 17 DeRoos '05, they had the cumulative lifetime days of
 18 use or cumulative exposure days, years of use
 19 multiplied by days per year categorized in
 20 tertile -- in tertiles among users, from 1 to 20,
 21 et cetera.
 22 So did they use the same thing here?
 23 Because the numbers are different than the numbers
 24 here.
 25 Q. Yes, sir. They broke it into three even

1 of NHL by cell type."
 2 A. I see that, yes.
 3 Q. And then we have a breakdown of four
 4 different groupings of NHL types; correct?
 5 A. Uh-hum.
 6 Q. And then on page 39, you see the data for
 7 glyphosate. And for all the subtypes, there was no
 8 association in the data in this study; correct?
 9 A. Yeah, it does not seem that there is an
 10 association here based on this data.
 11 Q. Page 53, there is a supplemental Table 2
 12 entitled -- I'll wait until you're there.
 13 A. I'm there.
 14 Q. Entitled "Pesticide exposures, total days
 15 and intensity-weight total days, fully adjusted
 16 risks of NHL incidence, 1993 to 2008."
 17 On page 59, we see the data. And, again,
 18 there is no association in these data, correct,
 19 between glyphosate and non-Hodgkin's lymphoma?
 20 A. In this data as presented, you're correct.
 21 But this paper requires way too much review than
 22 this, and I -- it's, like, 70-page paper.
 23 Q. I wish you'd reviewed, sir.
 24 A. Well, I -- it's not in the published
 25 literature.

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1 Q. On page 66, Supplemental Table 3,
 2 "Herbicide exposures, lifetime days, and
 3 age-adjusted NHL risk by cell type, 1993 through
 4 2008."
 5 A. Which page, sir? Which page are you on?
 6 Q. Page 66.
 7 A. Okay. Yep.
 8 Q. The data is on page 69 for glyphosate.
 9 And, again, there is no association between
 10 glyphosate and non-Hodgkin's lymphoma in this data
 11 as reported; correct?
 12 A. I don't see an association here based on
 13 the data that is represented.
 14 Q. Supplemental Table 7 on page 84, sir,
 15 "Pesticide exposures, total days, and
 16 intensity-weighted total days, age-adjusted risks of
 17 NHL incidences, 1993 through 2008." On page 91 is
 18 the glyphosate data. And, again, there is no
 19 association between glyphosate and NHL in the data
 20 as presented here; right?
 21 A. Page 91?
 22 Q. Yes, sir.
 23 A. Yes.
 24 Q. So I'm correct that there was no
 25 association?

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1 A. As depicted in this table, you're correct.
 2 Q. Let's go back to page 34 to get some sense
 3 of how much larger this cohort is, sir.
 4 A. Okay.
 5 Q. So page 34, again, is the glyphosate data
 6 for lifetime days and intensity-weighted lifetime
 7 days. And the first column, after "none, low,
 8 medium, and high" gives the N, the number in each of
 9 those categories; right?
 10 A. Uh-hum.
 11 Q. So there were 70 people with no exposure to
 12 glyphosate who had non-Hodgkin's lymphoma, 89 with
 13 low exposure, 78 with medium, and 83 with high;
 14 correct?
 15 A. This is correct.
 16 Q. So 250 exposed cases compared to 94 from
 17 DeRoos 2005; right?
 18 A. In DeRoos '95 -- 2005 at 29, 1 to 20; 15,
 19 that's 44; and 17, that's 61, according to this
 20 table.
 21 Q. This is a much larger cohort?
 22 A. Yes.
 23 Q. Okay.
 24 A. I still would like to understand the
 25 methodology, how they actually grouped these

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1 patients. So if you can just show me how they
 2 grouped them, because they are being grouped
 3 differently between DeRoos '05 and this paper. But
 4 there's so much corrections on it, that it's very
 5 difficult to even tease out.
 6 Q. Yes, sir.
 7 There is a draft paper with comments on it.
 8 A. Well, there are obviously a lot of comments
 9 that requires revision. And, clearly, there is so
 10 much corrections that are needed.
 11 Q. We've discussed earlier that Aaron Blair,
 12 who is the head of IARC and on the Agricultural
 13 Health Study, had his deposition taken in this case
 14 and that you haven't read that deposition; right?
 15 A. I have not.
 16 Q. Okay.
 17 And Dr. Blair at his deposition, when he
 18 was asked what the ever/never statistics would be
 19 from this study, the Alavanja 2013, admitted that it
 20 would be less than 1, it would be that 0.9 point
 21 estimate.
 22 Did you know that, sir?
 23 MR. LITZENBURG: I'm going to object to
 24 that characterization. You don't need to
 25 listen to any representation he makes about a

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1 deposition transcript he hasn't seen -- or he
 2 hasn't shown you.
 3 A. I don't know.
 4 MR. LITZENBURG: You can answer the
 5 question if you want to --
 6 A. I did not know that.
 7 MR. LITZENBURG: -- with that caveat on the
 8 record.
 9 Q. And, sir, did you know that Aaron Blair
 10 admitted that the meta-relative risk for NHL that
 11 was calculated by IARC, where he was presiding,
 12 would probably not have been statistically
 13 significant if IARC had had this data?
 14 MR. LITZENBURG: Same objection.
 15 A. I know nothing of what Aaron Blair has ever
 16 said.
 17 Q. If that were true, sir, if IARC had had the
 18 2013 data and calculated a meta-analysis that was
 19 not statistically significant, that was, in fact,
 20 near 1, how would that affect your opinion that
 21 glyphosate --
 22 MR. LITZENBURG: Same objection.
 23 Q. -- can cause non-Hodgkin's lymphoma?
 24 A. I think we both know that we don't know the
 25 answer to that. I think it would be critical, if

1 this type of literature is sound and good, it would
2 be submitted for rigorous peer-review process to a
3 respectable journal for peers to look at. If it's
4 written in 2013, and it's -- four years later, it
5 has not been published, then there are clearly some
6 issues in it that, to this date, has not been
7 published.

8 Having said that, until it's published,
9 peer-reviewed, and go through the process, all of
10 the information here in my -- has nothing to do with
11 my opinion or testimony.

12 Q. Is that because you have a policy of not
13 reviewing unpublished literature?

14 A. Well, how am I supposed to find this? If
15 it's not reviewed, I mean, how am I supposed to find
16 this type of literature?

17 Q. It's in your hands now, sir.

18 A. You want me to review a 75-page document in
19 five minutes?

20 Q. Is this something that you're going to
21 weigh in forming your opinions about non-Hodgkin's
22 lymphoma and glyphosate now that you have it?

23 MR. LITZENBURG: Object to form.

24 A. If it is not in the peer-reviewed
25 literature that is published and been subjected to a

1 affect your opinion on glyphosate and non-Hodgkin's
2 lymphoma?

3 A. Well, I think --

4 MR. LITZENBURG: Object to form, and asked
5 and answered.

6 Go ahead.

7 A. The fair thing is really for the IARC to
8 relook at things. And now there is additional
9 evidence, and they probably have to relook at things
10 and see whether this solidifies the evidence
11 further, not solidify the evidence further. It's
12 hard to tell, because, again, you have to remember
13 that the evidence is not just based on one or two
14 papers; it's based on the totality of evidence.

15 There is a lot of epidemiologic literature.
16 There is some meta-analysis. There is some
17 genotoxicity studies. We talked about some animal
18 studies, et cetera.

19 So it's really not one thing that's going
20 to sway the pendulum one way or the other. And I
21 think you've asked me several times, if this study
22 was reviewed or not reviewed, how would your opinion
23 change. And it's impossible to answer this, because
24 I'll have to put my mindset into a situation that I
25 don't have evidence I already looked at. And it's

1 rigorous peer-review process, I will not rely on it.

2 Q. Why?

3 A. I think it's self-explanatory. I mean, I'm
4 not going to rely --

5 Q. Go ahead.

6 A. -- on an opinion -- if a scientist has an
7 opinion that is valid, they usually submit that
8 opinion to a journal, to a peer review, so it could
9 actually be looked at and evaluated.

10 Q. Do you know that Mr. Blair and his
11 colleagues -- Dr. Blair and his colleagues discussed
12 publishing this before IARC so that IARC would be
13 able to consider it and chose not to do so and has
14 testified to that effect?

15 MR. LITZENBURG: I'm going to object again
16 about representations about Aaron Blair's
17 testimony and . . .

18 A. I know nothing of what Aaron Blair did,
19 said, or -- I have not looked at what he actually
20 said, and I don't know what his opinion is in this
21 matter.

22 Q. If this data were valid, if it -- if this
23 could be written up and published and present these
24 same data that we just looked at showing no
25 association, how would that, as a published paper,

1 hard to do that because I already saw that evidence
2 and I looked at and I critiqued it.

3 So I think if this paper ever makes it to
4 light and gets published and peer-reviewed in a
5 journal, then it should be looked at like all other
6 journals that we looked at. I think the importance
7 of a peer review is that -- so this paper, you know,
8 would be sent to -- to folks who understand this
9 type of literature. It would be subjected to a
10 statistic -- the rigor of statistics. A
11 statistician would review the methodology, a
12 toxicologist, et cetera, an epidemiologist. And
13 they would provide comments and do the things.

14 I mean, you could tell, frankly, just from
15 the draft that you gave me -- I mean, it's kind of
16 funny, frankly. Let's see how many comments there
17 are already that's outstanding. I mean, you know,
18 there's over 50 to 60 comments that, you know,
19 from -- no, I take it back. 77. Look how many
20 comments there are of certain outstanding things
21 that are still not resolved in the author's opinion.
22 That tells me they are way far from even getting
23 close to agreeing on what this paper means.

24 77 comments on page 83. That's the last
25 thing. Many of the comments are Aaron Blair

1 himself.

2 Q. If they chose not to publish this because
3 they didn't want IARC to come to a conclusion other
4 than what IARC came to, would you think that was
5 scientifically proper?

6 MR. LITZENBURG: Object to form.

7 A. If they -- if they chose not to publish it
8 intentionally, you mean?

9 Q. If they chose not to publish this because
10 they didn't want IARC to have this data because it
11 might influence IARC to find that glyphosate was not
12 associated with non-Hodgkin's lymphoma, do you think
13 that's scientifically proper?

14 MR. LITZENBURG: Same objection.

15 A. Yeah, I wouldn't agree to not publishing
16 this for the sole purpose of affecting a committee
17 review. If it were me, I would not withhold
18 information for that sole purpose.

19 I can't speak as to why it is not
20 published. I mean, what you're telling me is
21 Dr. Blair has testified to the content of the data.
22 So, clearly, he is willing to share that data
23 with -- in the public domain. So I believe that the
24 reason a paper like this is not published is the
25 fact that it has a lot of methodological issues that

1 they're trying to go through.

2 There's -- again, this is -- the draft that
3 you gave me is 12/5/16, almost a year old. And it's
4 one of those studies that has a lot of issues that
5 they're trying to address. And I think they're
6 struggling in addressing them. That is my honest
7 opinion when I look at a draft like this that's been
8 sitting on the shelf for a year with 77 comments on
9 it.

10 But, if it were me, I would not withhold
11 information just so I would affect a committee
12 decision. But that's me.

13 Q. Were you provided with any -- you have
14 looked at several depositions in this case; right?

15 A. I have.

16 Q. Which ones?

17 A. I looked at Dr. Neugut, Dr. Saltmiras. I
18 think I read his deposition.

19 MR. LITZENBURG: I think there's a list at
20 the end of your --

21 Q. Well, you list -- in your expert report,
22 you listed Donna Farmer, David Saltmiras, and
23 William Heydens.

24 A. And Neugut as well.

25 Q. Okay. And Neugut. And that's it?

1 A. Yeah.

2 Q. How did you even know about those
3 depositions?

4 A. The -- Tim forwarded them to me.

5 Q. Okay. You said earlier that you found all
6 of the scientific literature that you relied on for
7 your expert report and listed under documents
8 reviewed by Dr. Nabhan yourself; is that right?

9 A. I have researched that myself, yes.

10 Q. Okay. Were any of those sent to you by
11 counsel for plaintiffs?

12 A. When I struggled in finding particular
13 information, I reached out. And they were able to
14 help me if I struggled in finding some of those --

15 Q. Okay. So if you asked for a particular
16 article, they sent it to you?

17 A. Yes.

18 Q. Otherwise, they didn't send you anything in
19 particular?

20 A. Correct.

21 Q. And the depositions and exhibits they chose
22 to send you, the four that you have listed?

23 A. It's the second -- it's the other way
24 around. I -- even before I accepted this, I did a
25 lot of literature myself to decide whether I can do

1 this or not. And, again, I did my literature
2 search. But if I struggled sometimes in finding
3 some of the information, they -- I reached out, and
4 I was provided some help.

5 Q. Yes, I'm asking about something different
6 now. I've moved on from the scientific literature.

7 A. Oh.

8 Q. I'm talking about the depositions now.

9 A. Oh, the deposition --

10 Q. For those, they chose what to send you; is
11 that right?

12 A. Yes. They were -- I didn't have a choice.

13 Q. Yes, sir.

14 And anything that was unpublished that
15 plaintiffs' counsel may have or know about, you
16 didn't get that from them? You didn't hear about it
17 from them or get it from them, like this Alavanja --

18 A. I reviewed whatever they sent me, plus I
19 reviewed a lot of things on Google and CNN. It's
20 very easy to search and see what's going on.

21 Q. This MON-GLY production, MON-GLY 01314233,
22 et seq -- et seq means "and so on." Right?

23 A. I don't know. Which one is this?

24 Q. Do you see what I'm talking about?

25 A. I see what you're saying, yeah.

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1 Q. These are -- these would be Monsanto
 2 documents?
 3 A. Yes, I have reviewed -- I don't know which
 4 one is this, but I have reviewed some of the
 5 documents that were not necessarily papers that were
 6 sent to me by the plaintiff.
 7 Q. Okay. And was it -- how large of a volume
 8 was it?
 9 A. I think this may -- I don't remember which
 10 one is this.
 11 MR. LITZENBURG: I don't know what it is.
 12 A. I'll have to get back to you on that. I
 13 really don't know. I don't know.
 14 Q. Did you review a whole box of documents --
 15 A. No, no, no, no.
 16 Q. -- or a little stack or what?
 17 A. It's probably 10 pages or 12 pages.
 18 Q. Okay. So this was one particular Monsanto
 19 document, and you --
 20 A. Probably a couple of documents. Probably a
 21 couple of documents.
 22 Q. And then IARC Monograph 112, how did you
 23 find that one?
 24 A. I think I -- this is one of the things that
 25 I asked for help to get the actual monograph, and I

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1 reviewed. But I reviewed the paper myself, the
 2 Guyton paper.
 3 Q. And then the EPA SAP panel, final minutes
 4 and report, how did you have the idea to get that
 5 and read it?
 6 A. I've asked the plaintiff for that.
 7 Q. Did you know about it from your own
 8 research and ask them to get it for you?
 9 A. Yeah, yeah. I've asked to see the last one
 10 as possible.
 11 Q. And are there any other documents that you
 12 considered important in forming your opinions that
 13 aren't listed on these two pages, Attachment B to
 14 your expert report, sir?
 15 A. Not for this particular report, no.
 16 Q. Other than the --
 17 A. But you have to add the Neugut deposition,
 18 just to be accurate.
 19 Q. Yes, we discussed it. So it's -- it's on
 20 the record, sir.
 21 Other than the declaration that you did in
 22 the Dwayne Johnston [sic] case, have you done other
 23 expert reports on the subject of glyphosate in any
 24 way?
 25 A. I have not.

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1 MR. GRIFFIS: Let's take a five-minute
 2 break.
 3 VIDEOGRAPHER: Going off the record at
 4 4:40 P.M.
 5 (Recess taken from 4:40 P.M. to
 6 4:52 P.M.)
 7 VIDEOGRAPHER: And we are back on the
 8 record at 4:52 P.M.
 9 MR. GRIFFIS: I'm going to stop my
 10 questioning now and reserve the rest of my time
 11 for redirect. And Mr. Litzenburg is going to
 12 ask some questions.
 13 MR. LITZENBURG: Thank you. I do have a
 14 few questions in follow-up. I'm going to work
 15 backwards, so it will be a little awkward, and
 16 I apologize for that up front.
 17 EXAMINATION
 18 BY MR. LITZENBURG:
 19 Q. Let me start out by asking the opinions
 20 you've given today, do they have anything to do with
 21 Cardinal Health or with your employment with
 22 Cardinal Health?
 23 A. No, they're not. They're my individual
 24 opinion. My employer bears no opinion on this case
 25 whatsoever.

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1 Q. And we are compensating you, but that's on
 2 an individual basis, has nothing to do with your
 3 company; is that right?
 4 A. Correct.
 5 Q. Okay. We discussed some depositions today.
 6 Well, let me back up.
 7 You have stated today over and over again,
 8 and on many levels, your opinion as to general
 9 causation. You understand that term now as we are
 10 using it, general causation?
 11 A. Yes, I do.
 12 Q. Okay. Does -- that opinion of general
 13 causation, does that depend on any of these
 14 depositions as a basis for that -- for forming that
 15 opinion?
 16 That's a terrible question. Let me ask it
 17 again.
 18 Did you have to rely on any of these
 19 deposition transcripts in order to form that
 20 opinion?
 21 A. No. I formed this opinion based on my
 22 research of the available evidence, the published
 23 literature, as well as my own expertise and treating
 24 patients with lymphoma for over 17 years.
 25 Q. Okay. So your opinion didn't change with

1 what Dr. Neugut said at his deposition or what was
2 written in the transcript of Dr. Neugut's
3 deposition; is that fair?

4 A. No, it did not.

5 Q. Okay. I'm going to briefly -- again, we
6 will work in reverse -- ask you a couple questions
7 about this Exhibit 23.

8 First of all -- well, we spent a lot of
9 time on the record. But, generally, do you have any
10 idea what this is?

11 A. Well, I saw it for the first time today.

12 Q. Uh-hum.

13 A. I'm being told that this is an unpublished
14 data on the Agricultural Health Study.

15 Q. Did you review -- what other sets of
16 unpublished data did you review for your opinion
17 today?

18 A. I just reviewed, you know, the EPA as well
19 as some of the depositions, but everything else I
20 reviewed and I relied upon was published in
21 peer-reviewed journals.

22 Q. Okay. And so you drew a line at
23 peer-reviewed published data in order to review for
24 basing your opinion on; is that fair?

25 A. I think it's very important for anything

1 each person. There's a lot of information that
2 needs to be looked at, lots of -- this is just, you
3 know, a very, very preliminary draft that is also
4 one year old. The last it was looked at was
5 December 2016, and the data was almost four years
6 old.

7 And I don't think that it would withstand,
8 frankly, the scrutiny of peer-review process when
9 you lose 40 percent follow-up. I think that's
10 really why the authors can't even submit it
11 anywhere.

12 Q. Well, let's take that one step at a time.

13 Tonight is, again, the first time you've
14 ever seen this document; is that fair?

15 A. Correct.

16 Q. And I don't want to get down in the weeds
17 and examine all of the analyses and statistical
18 power, et cetera, of this draft paper, but you
19 had -- you said at the beginning that you had --
20 yeah, that you can critique this right away. And
21 then you mentioned something about follow-up.

22 Would you very briefly and concisely let us
23 know what you're talking about there?

24 A. Well, when you look at the -- at page 9, it
25 says, "A follow-up questionnaire which ascertained

1 that is looking at situations like this to be
2 reviewed by experts in the field and in the
3 literature, because if it withstands the rigor of
4 the peer-review process, then it just holds -- it
5 holds more scrutiny that I would look at more
6 critically and I will take more seriously.

7 Q. Did I ask you or any of my colleagues ask
8 you to review any other draft papers?

9 A. No, you have not.

10 Q. Did you ask me to provide you with any
11 draft papers to form your opinion?

12 A. No. The only thing I asked you about was
13 the EPA report.

14 Q. Does this even look like a final draft to
15 you?

16 A. It looks like an awful draft, in my humble
17 opinion.

18 Q. But, I mean, does it look like it's final
19 form, ready for submission to any peer --

20 A. Not even close.

21 Q. Okay. And I think you noted that these
22 comments were authored comments, in other words,
23 coauthors speaking to each other; is that correct?

24 A. Coauthors speaking to each other. It's
25 very difficult to know what each author is saying to

1 pesticide usage enrollment was administered about
2 five years after enrollment, completed by
3 63 percent."

4 So you have almost 40 percent loss of
5 follow-up with the second phase. And this
6 application of impute likely -- you know, stratified
7 sampling was employed to impute likely use of
8 specific pesticide seems to me like an exercise to
9 overcome a challenge. And that exercise will never
10 stand the test of rigorous peer-review process.

11 Q. Fair to say that statistics we're dealing
12 with today and you deal with in your job have to do
13 with public health, cancer?

14 A. Yes.

15 Q. Okay. Does -- and you take -- is it fair
16 to say that you try to take a more conservative
17 approach with statistics when you're looking at
18 matters of public health or life and death?

19 A. Of course.

20 MR. GRIFFIS: Objection to form.

21 Q. Okay. Does imputing 40 percent of data, is
22 that an appropriate or conservative approach when
23 looking at human cancer?

24 MR. GRIFFIS: Objection to form.
25 Foundation.

1 A. No. The answer is you cannot really forego
2 40 percent of lack of follow-up.

3 Q. We can set that aside for now, that draft
4 paper, if you would, Doctor.

5 Now, setting that aside and getting back to
6 all the published literature that we spoke about for
7 the rest of the day, are any of those studies
8 perfect that we talked about today?

9 A. I don't believe any study is 100 percent
10 perfect. I don't believe such a thing actually
11 exists in epidemiology literature.

12 Q. So while you have reached a conclusion
13 regarding general causation, there is not a single
14 paper that you would hold out and say, "This is the
15 perfect paper and by itself provides this evidence
16 100 percent"?

17 A. I don't --

18 MR. GRIFFIS: Objection to form. Leading.

19 A. I don't believe that any paper is perfect
20 that I reviewed.

21 Q. Okay. There was -- give me a minute.
22 (Pause.)

23 Q. There were some quotes read to you from a
24 IARC monograph question-and-answer. And that was
25 not given -- provided to you, and I don't have a

1 paper copy either. But I'm going to read you
2 another quote from that same document.

3 "Group 2A means that the agent is probably
4 carcinogenic to humans. For agents in this
5 category, there is usually convincing evidence that
6 the agent causes cancer in laboratory animals and
7 some evidence that it could cause cancer in humans,
8 but the evidence is humans is not conclusive."

9 Do you agree with that statement -- do you
10 agree with IARC's classification of glyphosate as a
11 2A agent?

12 A. I do.

13 MR. GRIFFIS: Objection to form.

14 Q. There's some discussion of hazard versus
15 risk, hazard being absolute and risk being a
16 measurement. Is that the way that you understood
17 the discussion?

18 A. That's the way that it was phrased to me.

19 Q. Okay. And so we have seen IARC's decision
20 as to whether or not this was a carcinogen. That's
21 been discussed at length today; right?

22 A. Yes.

23 Q. And we've also discussed, probably more so
24 of the day, a quantification of excess risk that's
25 been made by various models; is that fair?

1 A. Yes.

2 MR. GRIFFIS: Objection to form.

3 Q. And that would be the risk portion of the
4 hazard and risk delineation; right?

5 A. Yes.

6 MR. GRIFFIS: Objection to form. Leading.

7 Q. There were some questions about, you know,
8 what -- trying to pin you down, I think, about, you
9 know, what is the true increase in risk.

10 Epidemiology is the study of increase in
11 risk across populations; is that fair?

12 A. That is epidemiology.

13 Q. Okay. It's not -- there is no one number
14 for how much a person's risk is increased by
15 exposure to something, these are all population
16 subsets we're talking about today; is that fair?

17 A. That is fair.

18 MR. GRIFFIS: Just continued objection to
19 form. Leading.

20 Q. There was one question -- well, I won't try
21 to quote it verbatim. There was a question asking
22 you to point to a single statistically significant
23 positive result controlling for other pesticides.

24 We did look at some other issues above 1
25 that controlled for other pesticides today; is that

1 correct?

2 A. Yes, we did.

3 MR. GRIFFIS: Objection to form. Leading.

4 Q. And then when the meta-analyses -- the two
5 meta-analyses that you reviewed that were published
6 took into account the various odds ratios and
7 powers, did they reach statistical significance?

8 A. Yes, they did.

9 Q. And those -- okay.

10 Let's look at Eriksson, 2008. Maybe 18?

11 A. Which one is it?

12 Q. Eriksson 2008.

13 A. Is it 19, you said?

14 Q. I'm sorry. It's 18.

15 A. Okay.

16 Q. If I wrote it down right.

17 A. Okay.

18 Q. There was criticism of numbers -- well,
19 results are reached in these papers where the
20 confidence interval crossed 1 by counsel today.

21 Do you remember questions like that?

22 A. I do.

23 Q. Okay. If you look at Table 4, we spent
24 some time looking at that. And these are all
25 different herbicides or classes of herbicides; is

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1 that correct?

2 A. Correct.

3 Q. And defense counsel asked you to count up

4 all the ones with an odds ratio above 1.

5 Do you remember that question?

6 A. I do.

7 Q. Nearly all of them, however, have a

8 confidence interval which crosses 1; is that

9 correct?

10 A. Correct.

11 Q. Okay. We can set that aside for now.

12 Well, I'll just ask you a follow-up.

13 Have you done -- have you done any studies

14 on whether any other herbicide causes non-Hodgkin

15 lymphoma today?

16 A. I did not.

17 Q. And so you don't know if controlling for a

18 specific herbicide or a group of herbicides in a

19 given city would cause an odds ratio to go up, down,

20 or what it would do to the statistical significance;

21 is that fair?

22 A. That's fair.

23 Q. Let's look at -- there was a question about

24 a failure of these papers to find a dose response.

25 Let's look at McDuffie, if we can. And we'll just

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1 have a race to see who figures out which exhibit it

2 was first.

3 I think it was the first published paper

4 that was marked.

5 A. I think McDuffie is Exhibit 11. It's

6 Exhibit 11.

7 Q. Yeah. If you would look at page 1160 with

8 me.

9 A. Okay.

10 Q. And in the second paragraph on that page,

11 starting with Table 8, the authors in this published

12 McDuffie paper concluded that they demonstrated a

13 dose-response relationship for glyphosate.

14 Do you see that in the final sentence of

15 that paragraph?

16 MR. GRIFFIS: Objection to form. Leading.

17 A. I see that.

18 Q. I'll ask it a different way.

19 Did the authors of McDuffie put in this

20 published paper whether or not they've -- they

21 demonstrated a dose-response relationship with

22 glyphosate and NHL?

23 A. They did.

24 Q. Okay. And was that -- did they find such a

25 response relationship or not?

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1 A. They did.

2 Q. What is -- I think this was hinted at

3 today, but I want to give you a chance to explain

4 it. Is there a problem with using farmers as the

5 control groups in these epidemiological studies?

6 A. I mean, I wouldn't say it's a problem, but

7 I think, given the fact that farmers have an

8 inherent increased risk of developing non-Hodgkin's

9 lymphoma, your control group already has a high bar.

10 So demonstrating a statistical significant above and

11 beyond that in a case -- in the actual individuals

12 affected becomes more harder.

13 And that's why you would still take an odds

14 ratio as a hypothesis generating that you would look

15 at despite the fact that it crosses the 1, because

16 your control group already is establishing a much

17 higher bar that you have to overcome.

18 Q. Okay. This is going to make my head hurt,

19 but there was -- there was a comparison of logistic

20 and hierarchical regression or something like that.

21 Do you remember that today? Do you have

22 any reason --

23 A. Vividly.

24 Q. Do you have any reason to believe that the

25 logistic regression method is inferior to

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1 hierarchical?

2 A. I honestly -- as I said, this require a

3 statistician to answer the question. I don't think

4 I'm qualified to even know the difference between

5 logistic regression and hierarchical regression.

6 I've always, I believe -- and that's really the

7 value of peer review, that you have to --

8 statisticians would look at that.

9 As a clinician, I don't understand quite

10 the nuances in terms of differences between both

11 methodologies.

12 Q. Okay. But do you -- well, we'll leave it

13 at that.

14 A. I don't believe it would alter my opinion

15 per se.

16 Q. Let's look at -- we talked a lot about odds

17 ratios and a fair amount about confidence intervals,

18 but let's go to Exhibit 12, if you would.

19 A. Okay. It's the Hardell paper.

20 Q. Yes. And we had two marked, so make sure

21 it's Hardell 2002.

22 A. Yes.

23 Q. Okay. And I want to look at Table 1.

24 A. Okay.

25 Q. And so, for example, glyphosate has an odds

1 ratio of 3.04. In lay terms -- and I think we've
2 all dealt with epidemiology a lot and there's a
3 potential for a lay person to see this -- what does
4 an odds ratio mean exactly?

5 A. Sorry?

6 Q. Leaving alone the numbers. I'm sorry.

7 What exactly is an odds ratio? How would
8 you explain it to a lay person?

9 A. I mean, I would say an odds ratio is
10 basically the fact that the exposure to a particular
11 offending agent increases the risk above and beyond
12 other factors, above and beyond the control.

13 Q. Okay. And so just taking this as an
14 example, in Table 1 of Hardell, there is an odds
15 ratio -- and it is statistically significant -- for
16 glyphosate; is that correct?

17 A. Yes. It's 3.04, and it's -- the confidence
18 interval 1.08 to 8.52.

19 Q. Okay. Now, just using it as an example, to
20 a lay person, does that mean, then, it raises the
21 risk by 3 percent? What does that mean?

22 A. It means it raises the risk only by
23 30 percent.

24 Q. Is it a tripling?

25 A. No, it's not by 30 -- it's threefold.

1 That's what it means.

2 Q. So a tripling of the risk?

3 A. So -- yes. I mean, exposure to glyphosate
4 will triple the risk compared to somebody who is not
5 exposed. So you're increasing the risk by
6 threefold, by that number, by 3.04.

7 Q. Okay. And the confidence interval, you
8 talked about some of the arbitrariness of the P
9 values, but we've essentially selected confidence
10 intervals to mean that we could be confident to a
11 95 percent degree that the true value is within this
12 range. Is that a fair representation?

13 A. Yes. So 95 percent of the values fall
14 between those two numbers.

15 Q. Okay. So what we can tell here when we
16 look at the confidence intervals for glyphosate is
17 we could be 95 percent that it goes from 1.08 to as
18 high as 8.52; is that fair?

19 A. That is absolutely correct.

20 Q. So just in this table alone, it's possible
21 that the real result is underreported and could be
22 800 percent; is that right?

23 A. Could be 8 -- I mean, in some -- in some
24 folks, it could be eightfold increased risk. And
25 the lowest it could be is 1.8 -- 1.08-fold.

1 Q. Okay. And I won't go through all the
2 papers. There are many of those that we've looked
3 at today, but those are what those bounds of the
4 confidence interval mean when we look at individual
5 results; is that right?

6 A. Yes.

7 Q. Okay. You talked about -- you talked about
8 age, the increased risk of cancers with increasing
9 age. Do you remember that discussion earlier today?

10 A. Yes, I do.

11 Q. And I think you said something along the
12 lines of -- that one reason for that is it could be
13 a proxy for larger cumulative exposures; is that
14 fair?

15 A. Yeah.

16 Q. Could you explain that a little more.

17 A. Well, I mean, I think the -- the -- you
18 know, as we go through life, our bodies are exposed
19 to a variety of environmental, dietary factors, some
20 of them that we know they are carcinogen, some of
21 them we don't. And then, as the body ages, there
22 are lots of cellular disruptions that occur. And
23 when you add insult to injury, older folks become at
24 higher risk of developing certain cancers. So, I
25 mean, cancer ultimately is a disease of older

1 patients.

2 Q. Okay. But being 60 doesn't cause cancer.
3 Is that a fair way to say it?

4 A. No, just because you're -- I think you
5 just -- you have a higher risk just by virtue of the
6 fact that, as you age as a person, the cellular
7 mechanisms just are altered. So, I mean, age -- you
8 could -- you could make a blank statement and say
9 age is a risk factor for every single cancer under
10 the sun, and you would be correct.

11 Q. But a person that's 80 has had more insults
12 to their cells and their DNA than somebody who is
13 10; is that fair?

14 A. Right. Exactly.

15 Q. Okay. If age is controlled for in some of
16 these -- we talked a lot about controls. And,
17 again, I think we all know what we're talking about
18 in here, but in case a lay person sees this at any
19 point, if a epidemiological study controls for age
20 and, say, comes up with an odds ratio of 2, that
21 means that if you take a group of people that are
22 the same age, they may be elderly and at increased
23 risk, and you take a group of the same people and
24 expose them to the agent, that they still have a
25 doubling of the risk; is that correct?

1 A. Yes. If you control for the age, that's
2 correct.

3 MR. GRIFFIS: Excuse me. Objection to
4 form. Leading.

5 Q. Well, let me ask again a different way.

6 What does it mean to control for age in an
7 epidemiological study?

8 A. Well, you do your best to take out age as a
9 contributing factor for both the cases and the
10 controls. So you want to try to eliminate age as a
11 confounding factor so you can go back. Well, the
12 only reason that these folks have non-Hodgkin's
13 lymphoma is simply because they're older.

14 So you control for this factor so you
15 eliminate that as a possible contribution.

16 Q. And is it -- controlling is -- well, let me
17 withdraw that.

18 A. You do this statistically through
19 regression modeling, where you just control for some
20 of these factors that you can control for. I mean,
21 age in general is easy to control for because you
22 have it available. But there are lots of factors
23 that you would like to control for that you can't.

24 Q. You talked about modifiable risks and
25 modifiable etiologies. And, again, I want to make

1 sure that anybody can understand this today.

2 Tell me what significance a modifiable risk
3 or etiology has to you as a clinician.

4 A. Well, you know, the -- you could make an
5 argument -- and it would be a valid argument -- that
6 the best way to actually -- that the best drug that
7 we have ever had for cancer is smoking cessation as
8 an example. It has had the absolute highest risk
9 reduction possible. It is not an innovative
10 therapy. It's very inexpensive, it's cheap, and et
11 cetera, et cetera.

12 So identifying risk factors that are easy
13 to eliminate from the environment to affected
14 individuals is very valuable. And it's very
15 important for us as clinicians, because you can take
16 one factor out, and then you would reduce the risk.

17 And I bring tobacco as an example because
18 it's easy to understand for a lot of people. Even
19 in somebody who has a diagnosis of a particular
20 cancer, and they say, "Well, I have cancer now; I
21 can smoke all I want," the fact is if you stop
22 smoking, you reduce the risk of a secondary cancer
23 because now your body was more predisposed to the
24 first one individual cancer.

25 So, you know, environmental factors are

1 important for us to identify because it helps
2 patients at the end.

3 Q. And in your clinic you can recommend to
4 patients, based on your understanding of these
5 factors, to avoid certain ones to try to avoid
6 recurrence or progression of the disease?

7 A. Absolutely.

8 MR. GRIFFIS: Objection. Leading.

9 Q. There was some brief discussion about
10 latency. And I appreciate that -- well, it was my
11 understanding that you said basically it depends on
12 a lot of different factors. Is that -- is that --
13 let me withdraw that question.

14 What was your overall answer to the
15 questions about what's a latency period for
16 non-Hodgkin's lymphoma today?

17 A. As I said, I think that it is very
18 difficult -- it's a very gray area. It is very
19 difficult to have a binary decision on a latency
20 period and say you have to have 10 years of exposure
21 or 5 years of exposure or 15 years of exposure
22 before you develop cancer. It's just not the way
23 real life works.

24 So I think that latency period does exist.
25 I think it varies between individual patients and

1 other contributing factors, how often they get
2 exposed to an offending agent, et cetera.

3 So I don't believe short latency period or
4 long latency period should -- should be a factor.
5 It was not a factor in me deciding that there's a
6 causality between glyphosate and non-Hodgkin's
7 lymphoma.

8 Q. Okay. So you could find causality with a
9 latency period of significantly shorter than ten
10 years as well as significantly longer than ten
11 years? Is that --

12 A. Absolutely.

13 MR. GRIFFIS: Objection. Leading.

14 A. And I said that previously for sure.

15 Q. Let's take modifiable risk factors as an
16 example. Do you need to know the mechanism of
17 action of those risk factors in order to apply them
18 to your clinic?

19 A. No. And, in fact, there are many things
20 that we -- you know, we told people not to smoke
21 before we even know how in the world nicotine or
22 tobacco cause cancer, and we probably still don't
23 know exactly how it happens.

24 So I think knowing the mechanism of action
25 is good, is nice. I think it would be nice to have

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1 a plausible mechanism of action to better understand
 2 as a scientist. I think it's always nice. But it's
 3 not an absolute. It's not really necessary to know
 4 that.
 5 And, similar to this, we know many drugs
 6 that work against cancer. And we don't always
 7 understand the exact mechanisms by which these drugs
 8 work against cancer. But we know from clinical
 9 trials that they do. And there is usually some
 10 basic science studies to suggest that they could
 11 work.
 12 So I think it's nice to know some mechanism
 13 of action to have this plausibility between -- and
 14 association, but it's not mandatory to fully
 15 understand.
 16 Q. I was going to say, do you feel comfortable
 17 prescribing drugs to cancer patients where you're
 18 not sure of the exact mechanism of action?
 19 A. We do it all the time, as long as it's
 20 supported by clinical trials that show the activity
 21 and they're FDA approved.
 22 Q. Let's look at -- yeah, let's look at
 23 Exhibit 6, if we can.
 24 A. 6?
 25 Q. Yeah. That's not right.

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1 A. That's the monograph?
 2 Q. No, that's not right. Let me see if I can
 3 find it.
 4 There was --
 5 A. Which paper?
 6 Q. Let me see if I can find it. What do you
 7 have for 5? I don't have a 5.
 8 A. 5 is the Greim paper, 4 is the Engels
 9 paper, and 6 is the IARC monograph.
 10 Q. Let me see if I turned the number upside
 11 down, 7.
 12 A. The Smith paper?
 13 Q. Yes. And that's something you --
 14 A. Sure.
 15 Q. Is this something you saw for the first
 16 time today?
 17 A. Yes.
 18 Q. Okay. And we looked at -- it doesn't say
 19 anything about glyphosate; correct?
 20 A. No.
 21 Q. We looked at a bunch of different
 22 characteristics, key characteristics of carcinogens.
 23 Do you remember that and see that in that paper?
 24 A. It's proposed characteristics.
 25 Q. Okay.

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1 A. These authors are trying to propose
 2 characteristics that need to be satisfied to
 3 establish carcinogenicity.
 4 Q. Okay. And there's ten. In order for
 5 something to cause cancer, does it have to have all
 6 ten of these characteristics?
 7 A. Nope.
 8 Q. Okay. Are there known carcinogens that
 9 lack some of these characteristics?
 10 A. Yes, but I can't name anything right now.
 11 Q. Let's -- let's confine the discussion of
 12 glyphosate to . . .
 13 Just to make a clear record, I gave you the
 14 rough draft of Dr. Neugut's deposition transcript;
 15 is that correct?
 16 A. Yes, it was un -- it had a lot of typos.
 17 Q. Okay. And I haven't given you the final?
 18 A. No.
 19 Q. You haven't reviewed that yet?
 20 I'm sorry. I think we've harped on this a
 21 couple times today, but explain to me why we can't
 22 draw different -- why we can't draw firm conclusions
 23 about the etiologies of the various subtypes of
 24 non-Hodgkin's lymphoma from the literature that's
 25 been produced to date.

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1 A. So it's actually very difficult because you
 2 have so many types of lymphomas. I mean, there are
 3 probably 60 types of non-Hodgkin's lymphoma that we
 4 currently are aware of. So you will have to design
 5 study of thousands of patients that -- to have
 6 sufficient numbers of every single histology to be
 7 able to demonstrate the association, causality, and
 8 statistical significance. That's one reason.
 9 The second reason is that the types of
 10 non-Hodgkin's lymphoma have changed over the years.
 11 So the way we know -- we classify lymphoma today is
 12 very different than the way we classified lymphoma
 13 in 1995. So it depends where the study was done, it
 14 becomes very difficult to know this.
 15 And, lastly, in a case-control study, you
 16 are relying on the answers of individuals that
 17 oftentimes they really don't know the subtypes. I
 18 mean, I have cared for thousands of patients. And
 19 they know they have lymphoma. Sometimes they don't
 20 know if it's Hodgkin, non-Hodgkin.
 21 So there are many situations where a
 22 patient may not know that what he or she has is
 23 large-cell lymphoma, follicular lymphoma, mycosis
 24 fungoides, et cetera. So I think it becomes very
 25 difficult to establish that.

<p style="text-align: right;">Page 342</p> <p>1 Q. And, in fact, some of the papers -- one of 2 the papers we looked at today had only, like, eight 3 exposed cases that were -- that were -- we were 4 drawing conclusions off of. Do you -- is that 5 correct?</p> <p>6 A. I recall that. I think maybe the Eriksson 7 paper. I don't remember which one.</p> <p>8 Q. Okay. Certainly, in a cohort of eight, 9 we're not going to have every subtype represented; 10 right?</p> <p>11 A. That's correct.</p> <p>12 Q. Even in a cohort of 50, we're not going to 13 be able to draw statistical conclusions about the 14 etiologies of subtypes and how they differ; is that 15 right?</p> <p>16 A. Impossible. You have 60 subtypes. I mean, 17 it's just -- it's just not -- it's not possible.</p> <p>18 Q. Let's find the Greim paper. It's an 19 early -- I think you found it when I gave you the 20 wrong number a minute ago.</p> <p>21 A. Yes, it is -- it's Exhibit 5.</p> <p>22 Q. And there was some discussion of -- well, 23 number one, one of these authors in the 24 corresponding authors, you pointed out, is a vice 25 president at Monsanto; correct?</p>	<p style="text-align: right;">Page 344</p> <p>1 publication dates for --</p> <p>2 A. Sometimes, yeah. It depends on the journal 3 and the article.</p> <p>4 Q. Okay. Some of these papers that we went 5 through today and we looked at in your -- that were 6 mentioned in your -- well, let me back up. The 7 majority of the papers that you looked at were ones 8 that you found in your own literature research; 9 right? Not sent by me --</p> <p>10 A. Yes.</p> <p>11 Q. -- or my colleagues? Okay.</p> <p>12 And you looked at a number of papers and 13 you mentioned a number of papers that did -- were 14 not what we would call positive papers for 15 associations of glyphosate --</p> <p>16 A. Correct.</p> <p>17 Q. -- and NHL; correct.</p> <p>18 Is that because you -- let me see. So --</p> <p>19 A. I think it's fair to be -- to represent the 20 evidence in its totality. I mean, I think, you 21 know, my -- my goal, when I looked at this evidence, 22 was not only to cite papers that were positive. I 23 don't think it would be fair, and I wouldn't do 24 that. I wanted to present as balanced of a review 25 as possible and as balanced of a testimony as</p>
<p style="text-align: right;">Page 343</p> <p>1 A. I don't know his title, but he is a 2 Monsanto employee.</p> <p>3 Q. Okay. And you see Christian Strupp there 4 is a member of -- do you see the 6 number by his 5 name?</p> <p>6 A. Yes. He is part of the glyphosate task 7 force.</p> <p>8 Q. Okay. And I'll represent to you that he 9 also works for a company that makes glyphosate. 10 Does that -- does that further give you a 11 grain of salt with respect to this paper?</p> <p>12 A. Yes.</p> <p>13 Q. I am going to represent to you that I've 14 found the date of publication in this journal to be 15 March 16th of 2015 in Critical Reviews of 16 Toxicology. Assuming that this was published on 17 March 16th, 2015, and the IARC meeting was March 3 18 to March 10, 2015, can you see a reason why the IARC 19 panel did not look at this paper?</p> <p>20 A. I can.</p> <p>21 Q. Okay. And what would that be?</p> <p>22 A. It wasn't available in the peer-reviewed 23 literature at the time of the IARC meeting.</p> <p>24 Q. Okay. Now, some things are published in 25 advance online. There's sometimes differing</p>	<p style="text-align: right;">Page 345</p> <p>1 possible. So I looked at all of the evidence, and I 2 did not shy away from explicitly citing evidence 3 that was not significant. I think it's fair.</p> <p>4 Q. That was my question, is you didn't go out 5 just looking for positive papers in order to form 6 your opinion; right?</p> <p>7 A. No. I looked at all of the papers.</p> <p>8 Q. And I think there was some question of why 9 did you mention some papers in your report that 10 didn't reach positive association. Is that just in 11 fairness or --</p> <p>12 A. I think that is the appropriate way of 13 reviewing the literature and looking at the 14 literature.</p> <p>15 MR. GRIFFIS: Objection to form. Leading. 16 I'm having to object after you answer, 17 because you are answering just a little fast, 18 sir.</p> <p>19 Q. There was, again, a delineation made 20 between hazard and risk and IARC and its conclusions 21 versus what was called real-world human exposure a 22 couple of different times.</p> <p>23 While IARC makes its classification of 24 whether something is of a possible or probable human 25 carcinogen -- you're familiar with that process and</p>

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1 those classifications; right?
 2 A. Yes.
 3 Q. And they draw a line between probable and
 4 possibly; correct?
 5 A. Yes.
 6 Q. And they put glyphosate in that -- in that
 7 former group of probable. Okay. And in order to
 8 look -- we've called that the hazard assessment.
 9 You're familiar with that discussion today?
 10 A. Yeah. We -- we've had an exhaustive
 11 discussion on that.
 12 Q. The Q&A from IARC and all that.
 13 Now, the literature review -- there was a
 14 question about real-world human exposures and risk
 15 assessments.
 16 Does the epidemiological literature give a
 17 feel for real-world human exposures?
 18 A. To the extent possible, it does.
 19 Q. I mean, that's not measuring enormous doses
 20 of some chemical in a lab; right?
 21 A. No. They're just looking at what really
 22 happens in real life. I mean, they take cases and
 23 controls and so forth. It's not -- they're not
 24 necessarily trying to -- you can't because it's
 25 really retrospective. So you're just looking at

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1 what's happening in the real world to the extent
 2 possible. And despite the limitations, it's a good
 3 representation in general.
 4 Q. Okay. I think we used the terms "clinical
 5 significance" or "clinically significant" and
 6 "statistical significance" a few different points
 7 today.
 8 Can you explain, to the best you can, what
 9 the differences between those two is for you?
 10 A. So statistical significance is a pure
 11 number. Right? It's, you know, a P value of less
 12 than 0.05, it says that the findings are -- could be
 13 related to chance in 5 percent of the cases, but we
 14 are 95 percent certain that they are not related to
 15 chance. However, these findings may not really
 16 impact your practice. You may not find them
 17 clinically significant.
 18 And I think for those of us who have done
 19 this for a long time are always -- can cite so many
 20 papers that show the P value of less than 0.05 that
 21 meant nothing.
 22 A pure example was published in the New
 23 England Journal of Medicine, the most prestigious
 24 journal in the world, in a study, randomized trial,
 25 prospective trial in patients with metastatic

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1 pancreatic cancer. They were -- they had two arms.
 2 They were compared prospectively. It was
 3 probably -- it's probably included close to 7, 800
 4 patients. The conclusion of that paper, the
 5 experimental arm that had the novel agent improved
 6 overall survival with a P value of less than 0.0 --
 7 less than 0.05 -- it was actually probably 0.01 --
 8 by 1.5 weeks.
 9 So how often do you believe this novel
 10 agent was used in real life? Not often. And I
 11 think these are examples where you can see certain
 12 things that, based on numbers -- you have enough
 13 numbers, you will see a P value less than 0.05 but
 14 may not be clinically significant.
 15 At the same time, there are situations that
 16 you may not see that P value, you may not see the
 17 0.05, but you see a trend, and you kind of know, if
 18 you had enough numbers, you were going to see
 19 something significant.
 20 So if you take just a small study, 50
 21 versus 50, and you see a trend, you will know that,
 22 if you just had hundred versus hundred, you were
 23 going to reach that P value.
 24 So I think it's very important for us, as
 25 clinicians and researchers, not to take -- not to

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1 just, you know, hold everything on a P value that is
 2 just a simple number, you know. And I can assure
 3 you that statisticians will have a lot of creative
 4 ways to make the P value significant. I call it
 5 funny accounting.
 6 Q. And so -- and I think you've just answered
 7 this, but there are things that are statistically
 8 significant that are not clinically significant to
 9 you?
 10 A. And vice versa.
 11 Q. And vice versa. Okay.
 12 Now, you've said either today or you've
 13 said to me at some point recently that you find
 14 positive studies to be more important than negative
 15 studies. Is that fair?
 16 A. I think it's fair, especially in situations
 17 like this. I mean, you know, if you see -- you
 18 start -- your baseline start is a negative
 19 association. Right? So if you say that this
 20 compound is not associated with this cancer, that's
 21 really the null hypothesis, if you will. That's
 22 really where you're starting from. So if you really
 23 confirm your null hypothesis, okay, that's great.
 24 But if you see a positive association, no
 25 matter how small it is, it is very important to

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1 report for two reasons. Number one, you did not
 2 know about that association before; but, number two,
 3 you have to look at the impact on a population
 4 basis.
 5 You know -- you know, yes, your trial may
 6 have included a couple hundred patients who have had
 7 eight or ten cases. Let's multiply that now by
 8 thousands, thousands, hundreds of thousands in the
 9 U.S., outside the U.S., in Europe, in Asia, in
 10 Australia. All of a sudden you see an epidemic that
 11 is very important for us to identify.
 12 So all of what these small studies are
 13 trying to tell us is there's something there. You
 14 better act on it before it's too late and we see
 15 more patients with this disease.
 16 Q. I'm going to ask you a hypothetical sort of
 17 about all those numbers that we've looked at today.
 18 Imagine if we had taken away the discussion of
 19 statistical significance, those confidence
 20 intervals, and all the things that we've taken
 21 today. The vast majority of results in all of these
 22 papers today, nearly all of them were above 1; is
 23 that correct?
 24 MR. GRIFFIS: Objection. Leading.
 25 A. That's correct.

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1 Q. Okay. And so if something truly doesn't --
 2 you are familiar with what a forest plot is?
 3 A. I am familiar with forest plot.
 4 Q. Okay. So in a forest plot, if you were
 5 plotting all of those results and you had a center
 6 line of 0, those will all be on the right or
 7 positive side of that line; right?
 8 A. Yes.
 9 MR. GRIFFIS: Objection. Leading.
 10 Q. And if you -- if something truly didn't
 11 cause cancer at all, you'd expect to see results
 12 of -- suggesting it was cancer protective, an amount
 13 of -- first of all, it's on the left side of that
 14 line too; is that fair?
 15 MR. GRIFFIS: Objection. Leading and
 16 foundation.
 17 A. I'm not sure about cancer protective. I
 18 would say it would be negative association with
 19 cancer. It would be on the left side or crossing
 20 the middle -- mid line, but I wouldn't go as cancer
 21 protective.
 22 Q. But here what we're seeing is a lot of
 23 results on the right side of the line, just not all
 24 of them reach the P value that statisticians have
 25 decided is the confidence interval that we use as a

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1 standard?
 2 A. Correct.
 3 MR. GRIFFIS: Objection. Counsel is
 4 testifying, not the witness.
 5 Q. You said earlier today -- we were talking
 6 about different -- differing between the subtypes.
 7 You said that some types have -- their causes remain
 8 unknown or they're unknown.
 9 Are you talking about abstract subtypes of
 10 non-Hodgkin's lymphoma, or do you mean particular
 11 clinical presentations of particular patients?
 12 A. No. We know enough about how patients
 13 present and how to treat them and the prognosis, I
 14 think we've done a great job in understanding
 15 subtype, subtypes of lymphoma, as well as
 16 prognostication.
 17 We do a good job in treating the disease;
 18 we can always do better. But I think I meant by
 19 saying is that we -- it's very difficult to
 20 subclassify in these studies every particular trial
 21 to go look at the subtypes. And I already, I think,
 22 outlined why that is the case.
 23 Q. And that was -- so you said you disagreed
 24 with a quote that was read about etiological
 25 heterogeneity among NHL subtypes.

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1 Is that that topic that we've just
 2 discussed?
 3 A. We discussed that. I think it's very --
 4 you cannot just have one etiologic factor that
 5 affects one type of non-Hodgkin's lymphoma, not the
 6 other. I don't think we are able to say that at
 7 this point. There are some types -- some subtypes
 8 of non-Hodgkin's lymphoma that we can actually tell
 9 what's causing them, but we don't have that for
 10 every single subtype.
 11 And I think, if my memory serves me right,
 12 I provided an example as HIV that causes several
 13 types of lymphoma. And there are many other
 14 examples I could give, but at the same time, we
 15 don't have causation for every type.
 16 Q. Okay. You figured out the particular
 17 histological subtype of a person's lymphoma. Does
 18 that give you more information about its etiology as
 19 a clinician, or does that give you more information
 20 about the treatment and prognosis of that disease?
 21 A. More, really, treatment than prognosis.
 22 Like I said, for some subtypes you can talk about
 23 the etiology and you -- you always try. You always
 24 ask questions about occupational exposure, family
 25 history, all of these things, viral association,

1 et cetera. But the reality, as a clinician, this
2 really aids more in prognosticating as well as
3 recommending treatment option.

4 Q. There was a question toward the
5 beginning -- you see I'm getting almost to the very
6 beginning -- about what you could add to a
7 toxicologist or an epidemiologist in terms of
8 expertise on this issue.

9 You are and have been, for most of your
10 career, a clinician; correct?

11 A. Yes.

12 Q. Okay. And what disease do you specialize
13 in?

14 A. Lymphoid malignancies and a little bit of
15 prostate cancer.

16 Q. Do you consider yourself a non-Hodgkin's
17 lymphoma specialist?

18 A. I would say lymphoma specialist, because I
19 do take care of Hodgkin as well. When I was at the
20 University of Chicago, I would see close to 50
21 lymphoma patients a week, at least five to six new
22 patients a week. So, I mean . . .

23 Q. Doctor, do you -- do you rely on
24 epidemiology in your interpretation of it in both
25 your clinical and your academic realms?

1 A. To the extent possible. I mean, I look at
2 the literature. I understand the literature. I'm
3 not an epidemiologist, but I can understand
4 epidemiology papers with -- with their limitations
5 and their strengths. So I think I -- I rely on them
6 somewhat. I wouldn't say they are the sole thing I
7 rely on.

8 Q. Okay. At the very beginning, there was a
9 question about when you last treated a cancer
10 patient. I think you said it was around 11 months
11 ago; is that correct?

12 A. That is correct.

13 Q. To be very clear, you're not offering any
14 opinions on the standard of care of a medical
15 oncologist; is that correct?

16 A. No, but I can.

17 Q. Let's not.

18 A. I -- I continue to write in the area and
19 lecture in the area. And I -- in fact, I'm giving a
20 big seminar on non-Hodgkin's lymphoma at the
21 American Society of Hematology in December. It will
22 get at least 3 to 400 people in attendance. So I
23 continue to work in the field.

24 Q. Completely aside from your business
25 practice?

1 A. Yes. And a lot of the papers that I have
2 and continue to submit are in non-Hodgkin's lymphoma
3 and chronic lymphocytic leukemia, so . . .

4 Q. Doctor, we've been here now for eight hours
5 or more, and defense counsel has given you a lot of
6 new things and a lot of arguments.

7 Has anything today knocked you off your
8 opinion that exposure to glyphosate can cause
9 non-Hodgkin's lymphoma?

10 A. No.

11 Q. Do all the opinion -- opinions that you've
12 put in your report here stand at the end of this
13 deposition?

14 A. They do stand.

15 MR. LITZENBURG: Okay. I have nothing
16 further at this time. I may have some in
17 follow-up.

18 EXAMINATION

19 BY MR. GRIFFIS:

20 Q. Doctor, what will your seminar at the
21 American Hematological Society be about?

22 A. Updates on lymphoma and CLL.

23 Q. What about? Will you be updating people on
24 all of the important literature since --

25 A. I'm chairing a panel. I'm chairing a panel

1 with three other people. So each one of us will
2 actually give a talk, and I'm moderating the panel.

3 Q. Will you be updating the audience in the
4 important developments in the literature over the
5 past year, for example?

6 A. Yeah. Usually I try to look at the
7 submitted abstracts to the American Society of
8 Hematology and what's new and choose which are
9 really more relevant factors, both in the clinical
10 connection in what we have known and where we are
11 going. I did that last year, and they have asked me
12 to do it again.

13 Q. Okay. So it's not a seminar in any
14 particular topic?

15 A. No.

16 Q. At this point it's more of a -- an entree
17 and an overview to the --

18 A. Yeah. I'm focusing in my talk on
19 large-cell lymphoma, but I'm also chairing and
20 moderating the seminar with two other speakers that
21 one of them will talk on follicular lymphoma and the
22 other person on chronic lymphocytic leukemia.

23 Q. Who are the other two speakers?

24 A. I'll have to actually check whether I can
25 give you the information, because the program is not

1 out yet.
 2 Q. Okay.
 3 A. So if you don't mind, I'll check with them.
 4 I don't want to -- it may not be up.
 5 MR. LITZENBURG: Don't disclose anything
 6 that you don't know that you are able to --
 7 Q. When is the seminar?
 8 A. December. December 8. You're welcome to
 9 attend.
 10 Q. A 95 percent confidence interval, sir, only
 11 means that the real value is 95 percent likely to be
 12 within that range if the data is accurate and the
 13 data is not confounded and the data is not otherwise
 14 statistically biased; correct?
 15 A. Yes.
 16 Q. The Greim paper that we talked about
 17 earlier, do you know, sir, that there is sworn
 18 testimony in this case that IARC is able to review
 19 unpublished articles that have been accepted for
 20 publication once they have been accepted for
 21 publication?
 22 A. Don't know that.
 23 Q. And the Greim had been accepted for
 24 publication for a full three months before IARC met?
 25 A. Did not have this information.

1 Q. You've been billing at the rate of \$550 an
 2 hour, sir?
 3 A. It's a bargain. Yes.
 4 Q. And Innovative Oncology Consulting, which
 5 you asked Mr. Litzenburg to make the check payable
 6 to, what is that?
 7 A. That is my -- how do I call it? I formed
 8 an LLC, but I'm the sole owner of it.
 9 Q. That's an entity that you use to get paid
 10 through; is that right?
 11 A. Right. I had aspirations to be a
 12 consultant and didn't -- I stuck to my decision.
 13 Q. You're being one right now, aren't you?
 14 A. Yes.
 15 MR. GRIFFIS: That's all I have, thank you.
 16 MR. LITZENBURG: This is an "I'm probably
 17 done" break, but let's have a quick break.
 18 VIDEOGRAPHER: Going off the record at
 19 5:44 P.M.
 20 (Recess taken from 5:44 P.M. to
 21 5:44 P.M.)
 22 VIDEOGRAPHER: And we are back on the
 23 record at 5:44 P.M.
 24 MR. LITZENBURG: And we can go off. We are
 25 finished for today. Thank you, Doc.

1 Q. I want to mark your billing record, sir,
 2 that you were kind enough to submit to us.
 3 (Nabhan Exhibit 24 marked for
 4 identification.)
 5 Q. Since there are two pages, I'm using two
 6 exhibit stickers, Exhibit 24 and 25.
 7 (Nabhan Exhibit 25 marked for
 8 identification.)
 9 Q. And these are labeled as for the first and
 10 second quarter of 2017, sir?
 11 A. It looks like it, yes.
 12 Q. Had you been working on this project of
 13 assessing for plaintiffs' counsel the literature on
 14 the association or lack of association between
 15 non-Hodgkin's lymphoma and glyphosate before the
 16 first quarter of 2017?
 17 A. I did do a little bit of work when I was
 18 first approached last summer where I did my own
 19 research to make a decision whether I would be an
 20 expert or not. I forgot. It was probably 7 to 10
 21 hours type thing. I think it was last year in May,
 22 looks like that. But that's it.
 23 Q. And you billed for that and were paid for
 24 that?
 25 A. I'm pretty sure I did.

1 THE WITNESS: You're welcome.
 2 MR. GRIFFIS: Thank you, sir.
 3 THE WITNESS: You're welcome.
 4 VIDEOGRAPHER: This concludes the
 5 deposition today of Dr. Chadi Nabhan. We are
 6 off the record at 5:44 P.M.
 7 (Time noted: 5:44 P.M.)
 8
 9 _____
 10 CHADI NABHAN
 11
 12 SUBSCRIBED TO AND SWORN BEFORE ME
 13 THIS ____ DAY OF _____, 20__.
 14 _____
 15 (Notary Public) MY COMMISSION EXPIRES: _____
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CERTIFICATE

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 Diplomat Reporter
Certified Realtime Reporter
Certified Realtime Captioner

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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.

Page _____ Line _____ Reason _____
From _____ to _____

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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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