

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

Before The Honorable Vince Chhabria, Judge

IN RE: ROUNDUP PRODUCTS)
LIABILITY LITIGATION,) NO. M. 16-02741 VC
_____)

San Francisco, California
Tuesday , March 6, 2018

TRANSCRIPT OF PROCEEDINGS

APPEARANCES:

For Plaintiffs:

The Miller Firm LLC
108 Railroad Avenue
Orange, VA 22960
(540) 672-4224
(540) 672-3055 (fax)

BY: MICHAEL J. MILLER

For Plaintiffs:

Andrus Wagstaff PC
7171 West Alaska Drive
Lakewood, CO 80226
(720) 255-7623

**BY: VANCE R. ANDRUS
AIMEE H. WAGSTAFF
DAVID JACKSON WOOL**

For Plaintiffs:

Andrus Wagstaff PC
6315 Ascot Drive
Oakland, CA 94611
(720) 255-7623

BY: KATHRYN MILLER FORGIE

Reported By: Lydia Zinn, CSR No. 9223, FCRR, Official Reporter

1 **APPEARANCES:**

2 For Plaintiffs:

Weitz & Luxenberg PC
700 Broadway
New York, NY 10003
(213) 558-5802

3
4 **BY: ROBIN L. GREENWALD**
5 **PEARL A. ROBERTSON**

6 For Plaintiffs:

Baum Hedlund Aristei and Goldman, P.C.
12100 Wilshire Boulevard, Suite 950
Los Angeles, CA 90024
(310) 207-3233

7
8 **BY: MICHAEL L. BAUM**
9 **ROBERT BRENT WISNER**

10 For Plaintiffs:

Lundy Lundy Soileau & South LLP
501 Broad Street
P.O. Box 3010
Lake Charles, LA 70601
(337) 439-0707

11
12
13 **BY: RUDIE RAY SOILEAU, JR.**

14 For Plaintiffs:

Andrus Anderson LLP
155 Montgomery Street, Suite 900
San Francisco, CA 94104
(415) 986-1400
(415) 976-1474 (fax)

16
17 **BY: LORI E. ANDRUS**

18 For Plaintiff Sioum Gebeyehou:

Law Offices of Tesfaye Tsadik
1736 Franklin Street, 10th Floor
Oakland, CA 94612
(510) 839-3922
(510) 444-1704 (fax)

19
20
21 **BY: TEFAYE WOLDE TSADIK**

22

23

24

25

1 **APPEARANCES:**

2 For Defendant Monsanto Corporation:
3 Hollingsworth LLP
4 1350 I Street, NW
5 Washington, DC 20005
6 (202) 898-5800

7 **BY: KIRBY T. GRIFFIS**
8 **JOE G. HOLLINGSWORTH**
9 **JOHN M. KALAS**
10 **ERIC GORDON LASKER**
11 **HEATHER ANN PIGMAN**
12 **STEPHANIE SALEK**

13
14
15 **Also Present: The Honorable Ioana Petrou**
16 **Leonora Lynham**
17 **Scott Duval**
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I N D E X

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12:35 p.m.

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

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4 **THE COURT:** Okay. Welcome back.

5 **THE WITNESS:** Thank you.

6 **THE COURT:** Ready to resume?

7 **MS. FORGIE:** Yes, Your Honor.

8 **THE COURT:** Do you have -- are you still going on
9 direct?

10 **MS. FORGIE:** Yes, Your Honor.

11 **THE COURT:** Go ahead.

12 **MS. FORGIE:** Very briefly, I hope.

13 DENNIS WEISENBURGER,

14 called as a witness for the Plaintiffs, having been previously
15 duly sworn, testified further as follows:

16 DIRECT EXAMINATION (resumed)

17 **BY MS. FORGIE**

18 **Q.** Okay. Dr. Weisenburger, you recall that the Judge had
19 some questions for us at the end of the day, and I'd like you
20 to please address those. Starting with the NAPP Study, can you
21 please explain what the NAPP Study is?

22 Oh. You can't hear me?

23 **A.** I can hear you.

24 **Q.** Is that better? Thank you.

25 **A.** Yeah. So the NAPP Study is a pooling of case-control

1 studies from four states in the Midwest -- all of the states
2 that were in the De Roos 2003 Study -- and the TransCanada
3 Study, which is six provinces in Canada. So it really combines
4 the McDuffie Study with the De Roos Study. It pools the data
5 into one dataset. And the advantage of this --

6 **THE COURT:** When you say it combines the study, it
7 combines the data from the studies?

8 **THE WITNESS:** Yes.

9 **THE COURT:** So it's a pooled analysis; not a
10 meta-analysis?

11 **THE WITNESS:** Yes, yes. It's a pooled analysis.

12 And the reason to do that is you want to increase the
13 power to detect things. And it also gives you an opportunity
14 to adjust for confounders.

15 So the NAPP Study is a study that is still in progress, in
16 the sense that the data -- the final data analysis has not been
17 finished, and the manuscript has not been, as far as I know,
18 submitted for publication; but the data has been presented at
19 three national or international meetings. The first was in
20 2015, in Canada. And that's the data that I presented
21 yesterday on the one slide that I used. It was also presented
22 later that year in Quebec -- no -- in Brazil. And then it was
23 presented a year later in France. And each of these is an
24 iteration on the other, emphasizing different things, and
25 presenting different parts of the findings.

1 The reason I chose the slide I did is because it shows the
2 data in the format that we have been using to talk about the
3 other case-control studies, and it adjusts for all of the
4 variables that need to be adjusted for, including use of other
5 pesticides. So it's an adjusted -- it's a table with the data
6 adjusted for other confounders or potential confounders.

7 **Q.** And, for example, the McDuffie Study in the original
8 publication doesn't necessarily adjust for pesticides, but are
9 you able to adjust for pesticides with the McDuffie Study in
10 the NAPP data? And can you explain how you do that, please?

11 **A.** Yes. So the three core case-control studies, which is
12 McDuffie, Hardell, Eriksson, and De Roos 2003 -- of those four,
13 three did adjust for the use of other pesticides. McDuffie
14 didn't, but McDuffie is part of the NAPP Study. So in that
15 sense, it was adjusted for in the NAPP Study. So really all
16 four of the core studies have been have been adjusted for the
17 use of other pesticides.

18 **Q.** And so just to be clear, when you say "adjusted," you mean
19 adjusted for other pesticides. Correct?

20 **A.** Yes.

21 **Q.** Okay. And then with regard --

22 The Judge also had some questions about recall bias.

23 **THE COURT:** Could you remind me, before you get
24 there --

25 **MS. FORGIE:** Sorry.

1 **THE COURT:** The slide that you showed from the
2 NAPP Study based on the data that was presented at Canada --
3 what -- remind me what that showed.

4 **THE WITNESS:** So what that showed was that there was
5 an elevated Odds Ratio. Here it is. There was an elevated
6 Odds Ratio of about 2 for all of non-Hodgkin's lymphoma, with
7 greater than 2 days per year handling of glyphosate. And there
8 was also a 2-and-a-half-fold increase, which was statistically
9 significant, for diffuse large B cell lymphoma, which is the
10 middle column there.

11 And then the other, if you look at the other subtypes,
12 "FL" is follicular lymphoma, "SLL" is small lymphocytic
13 lymphoma. And then the last column is kind of all of the other
14 uncommon -- less-common ones combined. You can see the
15 Odds Ratios are increased for all of those. The last two --

16 (Reporter requests clarification.)

17 **THE WITNESS:** Follicular lymphoma. Small lymphocytic
18 lymphoma. And the other less-common subtypes grouped together.

19 So that's basically what it shows. And it's adjusted for
20 age, sex, province or state, family history of cancer, use of
21 protective equipment. It's also adjusted for proxy subjects.
22 And then it's adjusted for these three pesticides. And I'm
23 going to talk about this a little bit later when we talk about
24 confounding, but when we -- so we'll come back to this slide
25 again.

1 **THE COURT:** Okay. And then in terms of numbers,
2 approximately how many people are we talking about from
3 these -- from the three -- is it three De Roos pools, and one
4 pool from McDuffie? Right? How many people are we talking
5 about?

6 **THE WITNESS:** Total number? Let me look here. I
7 don't know exactly. It's over a thousand.

8 **BY MS. FORGIE**

9 **Q.** Over a thousand? Is that what you said?

10 **A.** Yeah. Let me look. I have it.

11 **MS. FORGIE:** I should mention while he's looking this
12 up, this is -- the PowerPoint is Exhibit 300. And I also
13 forgot we have a few additions to give to the Judges and the
14 Clerk for their books, and one for Monsanto (indicating).

15 **THE WITNESS:** So there are 1,690 cases of NHL -- so
16 it's a large pooled study -- and over 5,000 controls.

17 **THE COURT:** You said over 5,000 controls?

18 **THE WITNESS:** Yes.

19 **THE COURT:** And of the 1,600 cases, how many were
20 classified as handling glyphosate 2 or fewer days per year?

21 Or sorry. I guess I may have started to ask that question
22 incorrectly. Of the people who handled glyphosate, of the
23 people who were exposed, how many were -- how many handled
24 glyphosate 2 or fewer days per year, and how many handled it
25 more than 2 days per year?

1 **THE WITNESS:** That's a good question. I don't have
2 it on the tables that -- it was -- those numbers weren't
3 actually presented in the paper, so I can tell you total -- a
4 total number of cases.

5 **THE COURT:** In what paper?

6 **THE WITNESS:** I mean, in this -- in this slide
7 presentation, those -- those specific numbers weren't given in
8 that table.

9 **THE COURT:** Okay.

10 **BY MS. FORGIE**

11 **Q.** Meaning the NAPP Study?

12 **A.** Yeah.

13 **Q.** Yeah.

14 **A.** There were a total of 113 cases that were exposed to
15 glyphosate. So even though it was a large study, the number of
16 cases exposed was relatively small.

17 **THE COURT:** There were 113 cases exposed to
18 glyphosate. So in other words, from this universe of --
19 What was it?

20 **THE WITNESS:** 16,000.

21 **THE COURT:** 1,600?

22 **THE WITNESS:** 1,600. Yeah.

23 **THE COURT:** Okay. So from this universe of 1,600
24 people who had NHL, how many of them were exposed to
25 glyphosate?

1 **THE WITNESS:** 116. 113.

2 **THE COURT:** 113 were exposed to glyphosate. So
3 roughly 1,500 of them were not exposed to glyphosate?

4 **THE WITNESS:** Yes.

5 **THE COURT:** Okay. And -- and you don't -- of the --
6 of the 113 people who had NHL and were exposed to glyphosate,
7 do you know how many of those were exposed for 2 or fewer days
8 per year, and how many were exposed for more than 2 days per
9 year?

10 **THE WITNESS:** I don't know that, because the data
11 isn't presented in -- in the slide deck, so I don't know what
12 it is. But in the McDuffie Paper they were split almost
13 evenly. Of the 51 cases in McDuffie, 28 were less than or
14 equal to 2 days, and 23 were greater than 2 days, so I suspect
15 it would be somewhat similar.

16 **THE COURT:** And then going back to Judge Petrou's
17 question from yesterday, do you know how these people were
18 asked about their glyphosate exposure?

19 **THE WITNESS:** Yeah. It was a bit different in the
20 different studies. For example, in the Nebraska Study, it was
21 a telephone questionnaire which -- a trained interviewer walked
22 the farmer or --

23 **THE COURT:** Nebraska was one of the three pools that
24 De Roos looked at?

25 **THE WITNESS:** Yes.

1 **THE COURT:** Okay.

2 **THE WITNESS:** Yeah. All of the -- all of the North
3 American studies had basically the same design, because they
4 were done by the same people at NCI over a period of time. And
5 the Canada study also had a similar design. Some of them got
6 the initial information by telephone. Some of them got it by
7 mailed questionnaire, followed up by telephone.

8 **THE COURT:** And what were they asked about exposure?

9 **THE WITNESS:** So the first thing they would ask, you
10 know, have you ever -- first thing they would do -- at least,
11 we did in Nebraska, is we had them volunteer what pesticides
12 they used. And for each one of the pesticides, they asked a
13 large number of questions.

14 How often did you use it?

15 How many days per year?

16 How many years did you use it?

17 Did you use protective equipment; protective clothing?

18 **THE COURT:** And are they asked to, like, check boxes
19 about how many days per year, or are they asked to write --
20 actually, like, write or respond in the narrative, just coming
21 up with their own --

22 **THE WITNESS:** They would be -- well, they would --
23 yeah. I don't remember that exactly. I think in the verbal
24 questionnaire they would give the answer, and the interviewer
25 would mark the box; but it was an open-ended questionnaire, so

1 they wrote down whatever the number the farmer told them.

2 **THE COURT:** Okay. And then, again, this is something
3 Judge Petrou was asking yesterday, and I want to make sure I
4 have an understanding of it. Why the decision to -- in this
5 slide, to classify between 2 or fewer days per year of handling
6 glyphosate, and greater than 2 days per year handling
7 glyphosate?

8 **THE WITNESS:** That's a good question. I don't know
9 the answer to that. I know what they've done in some studies.
10 They looked at the median days of exposure in the controls, and
11 applied it to the cases. So I know that's what they -- I think
12 that's what they did in Eriksson.

13 **THE COURT:** Okay.

14 **MS. FORGIE:** Okay.

15 **Q.** All right. Doctor, you were -- the Judge had some
16 questions also about recall bias. And can you please, using
17 the Blair Study, which is Exhibit 303, which you discussed
18 yesterday, explain recall bias, please?

19 **A.** Yeah. So recall bias is a form of non-random bias.
20 And -- and basically the idea is that the people in the
21 case-controlled study, the people with the disease --

22 **THE COURT:** If I could interrupt.

23 I understand what recall bias is.

24 **THE WITNESS:** Okay.

25 **THE COURT:** I want to know how important it is in

1 these case-control studies; how significant of a problem it is.

2 Or if it's -- if you don't think it's a significant
3 problem, I want to get a better understanding of why you don't
4 think it's a significant problem.

5 **MS. FORGIE:** I'm sorry.

6 **THE WITNESS:** Well, okay. Good.

7 So I don't think it's a significant problem in these
8 studies, and I'll show you why. So in the -- in this
9 Blair Paper, it's a methodology paper looking at pesticide use
10 in farmers. And it addresses the issue of recall bias. Again,
11 they use the data from Nebraska.

12 And what they did is -- again, Nebraska had an open-ended
13 question. And then it had focused questions. So in the second
14 phase, after the open-ended, the interviewer would ask, "Well,
15 Mr. Smith, have you been exposed to 2,4-D?" if he hadn't
16 volunteered it. And she'd say he -- or she would go through
17 the list of all of the common pesticides, to sort of prompt
18 them to remember, because they may not remember. So that's how
19 the Nebraska Study was done.

20 So what Dr. Blair did in this paper -- he looked at the
21 Nebraska Study. And he looked at: How many pesticides did the
22 farmers volunteer? And then he looked at: How many pesticides
23 did the controls volunteer in the -- in the open-ended part of
24 the questionnaire? And it was about the same. So it wasn't
25 that the farmers were remembering many more pesticides -- not

1 the farmers -- the cases were remembering more pesticides than
2 the controls. And then they did the same thing for the more
3 focused questions about specific pesticides, and the findings
4 were the same.

5 So Blair's conclusion was that -- that there really isn't
6 any recall bias, at least in the Nebraska Study, which is
7 representative of all of the North American studies. And if
8 there was any recall bias or any bias in remembering, it would
9 actually move the -- the Odds Ratio towards the null, because
10 it would -- because he couldn't see any evidence of
11 differential recall. So it's one methodologic study, but it's
12 the one that addresses this issue.

13 And then the other -- and then the other reason that I
14 gave you yesterday --

15 Maybe you could put up the slide about the studies in
16 IARC.

17 To IARC -- this is just the Blair Study. And, yeah, these
18 are some other studies from IARC. And, as you know, IARC
19 reviewed not just non-Hodgkin's lymphoma, but they reviewed all
20 the case-control studies that were done for heme malignancies
21 as well as solid tumors.

22 And so we made a list hear of all of the studies that were
23 done with a case-control model, and which asked questions about
24 glyphosate. And none of these studies shows the statistically
25 significant increase in Odds Ratio, like you see for

1 non-Hodgkin's lymphoma.

2 So -- so if -- if there was a systematic bias, a
3 systematic recall bias, you would expect to see increased risks
4 in some of these studies that were statistically significant.
5 And most of the studies are around the null.

6 There are a couple where it slightly increased. So if
7 there was a systematic bias -- a systematic recall bias in
8 these case-control studies, you should see the bias in some of
9 the other studies. There's no reason why non-Hodgkin's
10 lymphoma patients would remember the use of glyphosate better
11 than people with brain cancer, or soft-tissue sarcoma, or other
12 kinds of lymphomas or leukemias.

13 So that was the other -- I think the slide sort of shows
14 the data that I was talking about. So if you have a systematic
15 bias, you should see it in other studies. And it isn't seen in
16 any of these other studies.

17 **BY MS. FORGIE**

18 **Q.** Okay. Thank you. And then the third issue we were asked
19 to address is confounding factors. And can you explain, using,
20 please, the Eriksson Study -- the 2008 Eriksson Study, which is
21 Exhibit 17, the 2003 De Roos, Exhibit 15, and the
22 IARC Monograph, Exhibit 57, please?

23 **A.** Yes. So a confounder -- a true confounder is an exposure
24 that's correlated with the exposure that you're trying to
25 measure. So it would be another pesticide that's correlated

1 with glyphosate use in this case. And that also is a risk
2 factor for non-Hodgkin's lymphoma. So the second pesticide is
3 also a risk factor for non-Hodgkin's lymphoma. Okay? That's
4 what a confounder is.

5 And so I'm -- I want to show you this table as an example.
6 I think you saw it yesterday, but this is from the
7 Eriksson Study. And this is the multivariate analysis where
8 they did the adjustment for glyphosate and the other chemicals
9 that had elevated Odds Ratios. So if you look for glyphosate,
10 there was a twofold, statistically significant increase.

11 And after adjustment for all of these other chemicals, it
12 went down to 1.5, and it was no longer statistically
13 significant.

14 Now, we know that MCPA and 2,4,5-T and 2,4-D are organo --
15 they're phenoxy herbicides. And they're known to cause
16 lymphoma. Okay? So those were good ones to adjust for.

17 On the other hand, if you look at arsenic, although
18 arsenic's a carcinogen, it doesn't cause non-Hodgkin's
19 lymphoma. So why would you adjust for it? They probably
20 shouldn't have adjusted for it.

21 So the idea -- when you want to do an ideal adjustment,
22 you want to adjust for confounders that are correlated with the
23 pesticide you're trying to measure. And it should be risk
24 factors or have at least some evidence of potential for being a
25 risk factor.

1 **THE COURT:** So is it known that arsenic does not
2 cause NHL?

3 **THE WITNESS:** Yes.

4 **THE COURT:** Okay. Well, what about -- you mentioned
5 MCPA, and 2,4,5-T.

6 **THE WITNESS:** So those are all phenoxy herbicides
7 that have been linked to non-Hodgkin's lymphoma. Yes.

8 **THE COURT:** What about mercurial seed dressing, and
9 creosote, and tar?

10 **THE WITNESS:** Mercurial seed dressing is another
11 exposure that doesn't cause non-Hodgkin's lymphoma.

12 **THE COURT:** Does not?

13 **THE WITNESS:** No.

14 **THE COURT:** It's known that it does not?

15 **THE WITNESS:** As far as I know, it does not. Yes.

16 Creosote and tar, I think, is a bit controversial, because
17 they could be potential confounders, because they're -- they're
18 petrochemical-derived, and so they would have some solvents and
19 other things in them that do increase risk for non-Hodgkin's
20 lymphoma.

21 But the one that I'm sure of is arsenic. Arsenic is not a
22 risk factor for non-Hodgkin's lymphoma. And it had an
23 Odds Ratio of 1.63, but when they adjusted for all of these
24 other real or potential carcinogens, you see it went down
25 almost to null 1.17. So that's what you want to see.

1 **THE COURT:** Why? Why would it have 1.63 Odds Ratio,
2 if it's known not to cause non-Hodgkin's lymphoma?

3 **THE WITNESS:** Well, it wasn't significantly
4 increased. So you have always have some random error in your
5 studies. Okay? You always have some random error in your
6 studies. And this is probably due to random error.

7 So what -- what Eriksson did is he took all of the
8 exposures -- the pesticide exposures -- that had an Odds Ratio
9 of 1.5 or greater, and he said, *I'm going to put them all into*
10 *my multivariate model.* Okay? But he didn't really try to
11 decide whether they were real confounders or not. But -- and
12 when you do that, you decrease the power of the study. You --
13 you decrease the adjusted Odds Ratio lower. And sometimes
14 you -- you adjust it low enough so that it's no longer
15 statistically significant. And that's what happened in
16 Eriksson. That's also what happened in Hardell.

17 **THE COURT:** But I would think that if you -- if
18 arsenic -- if in this study you came up with an Odds Ratio of
19 1.63 for arsenic, and then you, despite that red flag or yellow
20 flag, you didn't adjust for it, you would be subject to a lot
21 of criticism, I would think.

22 **THE WITNESS:** Well, there are two philosophies on
23 that. One, like de Roos -- in the De Roos 2003 she adjusted
24 for all 46 pesticides, which is really, in my estimation -- I
25 discussed this with Dr. De Roos, too. She was overadjusting.

1 Okay? Because she was adjusting for a whole bunch of
2 pesticides that don't cause non-Hodgkin's lymphoma.

3 On the other hand --

4 And she still actually found a statistically significant
5 increase in -- in risk for non-Hodgkin's lymphoma, even with
6 that overadjustment.

7 **JUDGE PETROU:** So in your mind, if there had been a
8 statistically significant p-value of .05, which is not -- which
9 is not there for arsenic, it's not .5. It's 95 percent
10 security level, essentially. If it had hit a statistically
11 significant p-value, that 1.63, does that mean that then you
12 would have found it more important to adjust for it, or not?

13 **THE WITNESS:** Probably not, because it's not really a
14 confounder. It doesn't cause non-Hodgkin's lymphoma, so you
15 wouldn't need to adjust for it. You could do it. Some people
16 would do it. De Roos did it with the others; but if you do
17 this in -- in the most scientific way, you would wouldn't
18 adjust for things that aren't confounders, because the whole
19 idea of adjusting is to get the true value.

20 Yeah. And so if you adjust for arsenic here, you're going
21 to lower the Odds Ratio for glyphosate, even though it's not a
22 confounder.

23 So the other point I wanted to make about this is, you
24 know, one of the points --

25 **THE COURT:** So could I just --

1 **THE WITNESS:** Sure.

2 **THE COURT:** Before you go on to that, could I ask one
3 clarification question?

4 So in your view, the analysis that ought to be done --

5 Well, let me back up and ask another question. So are you
6 saying that we should be looking at the univariate number; the
7 univariate Odds Ratio for glyphosate?

8 **THE WITNESS:** Well, you always look at the univariate
9 number, because it tells you what direction things are going,
10 but I think the most -- the so it's important to look at the
11 univariate number. If it's high, it's probably real. Okay?

12 **THE COURT:** But why? I mean, why would we ever --
13 I mean, I understand your point about not including
14 arsenic. Like, maybe we should take arsenic out of the
15 multivariate analysis.

16 **THE WITNESS:** Right.

17 **THE COURT:** But why would we not do a multivariate
18 analysis with known or potential confounders, and then look at
19 that number? Why would that number ever not be a better number
20 to look at?

21 **THE WITNESS:** Well, it would be a better number. It
22 would be a better number, particularly if the adjustments were
23 done properly. Okay?

24 And, in fact, all of the studies did do that. Okay? The
25 four core studies -- the case-control studies -- all did that.

1 **THE COURT:** All did what?

2 **THE WITNESS:** All did adjustments for exposure to
3 other pesticides. Hardell did it. De Roos 2003 did it. And
4 it was done for McDuffie in the NAPP Study, which I'll show you
5 in a minute. So they all did it.

6 And it's important to do, because you want to see: Does
7 it go down? Does it go down to 1, like arsenic? Well, gee,
8 then probably it's not very important.

9 Or does it only go down to 1.5 or 1.8? That means that
10 there's still -- there's still an effect there.

11 **THE COURT:** But you just told me that 1.63 for
12 arsenic was not statistically significant. So if you're saying
13 that it goes down to 1.5, you're saying that is significant?
14 For --

15 **THE WITNESS:** Well, I was talking about glyphosate.

16 **THE COURT:** What's the difference? I mean, when we
17 were talking about arsenic, you told me that 1.63 was not
18 statistically significant.

19 **THE WITNESS:** Right.

20 **THE COURT:** So now you're telling me for glyphosate,
21 you go down to 1.5, that's still significant?

22 **THE WITNESS:** Well, it's no longer significant here,
23 if you look at it. So I want to get back to what Dr. De Roos
24 said yesterday. You have to look at the numbers and try to
25 make sense of them. And you don't want to place too much

1 emphasis on statistical significance because, you know, if
2 everything has to be statistically significant, you lose a lot
3 of information. So in this kind of a study, where you know
4 that the -- the Odds Ratio's going to decrease, it does go
5 down.

6 **THE COURT:** Then why would we exclude arsenic from
7 the multivariate analysis merely because it's not -- because
8 1.63 is not statistically significant?

9 **THE WITNESS:** Because it's not a confounder.

10 **THE COURT:** And we know that from other
11 epidemiological studies?

12 **THE WITNESS:** Yes, yes.

13 **THE COURT:** Studies of arsenic?

14 **THE WITNESS:** Yes. So it's not a confounder, so you
15 would take it out. And you would take out other pesticides
16 where there's no evidence that it causes non-Hodgkin's
17 lymphoma.

18 **THE COURT:** But when you say there's no evidence that
19 arsenic causes non-Hodgkins lymphoma, I mean, that's different
20 from saying you know that it doesn't cause non-Hodgkin's
21 lymphoma. I mean, might this 1.63 measurement be some
22 indication that we might want to look into whether arsenic
23 causes non-Hodgkin's lymphoma?

24 **THE WITNESS:** I think it's been -- I think it's been
25 well studied. And, you know, when you see Odds Ratios in the

1 primary -- in the univariate analysis that are not very high
2 and are not statistically significant, you usually don't pay
3 much attention to them. Okay?

4 The only reason he picked it is he set an arbitrary
5 number. I'm going to adjust for every confounder or potential
6 confounder or other exposure that had an Odds Ratio of 1.5, and
7 arsenic fell into that category. That's why he did it.

8 But it's not a confounder, so he shouldn't have had to do
9 that. He shouldn't have done it, in fact.

10 **THE COURT:** So wouldn't the best thing to do --
11 wouldn't it be best to do the multivariate analysis again,
12 after removing arsenic?

13 **THE WITNESS:** It would be better.

14 **THE COURT:** Wouldn't be that be a lot more
15 reliable --

16 **THE WITNESS:** Yes.

17 **THE COURT:** -- than using the univariate analysis for
18 glyphosate?

19 **THE WITNESS:** Yes, it would. It would. You're
20 right.

21 **THE COURT:** Okay. Thank you.

22 **THE WITNESS:** So I'd just like to show you the NAPP
23 slide again.

24 **BY MS. FORGIE**

25 **Q.** Let me just ask you other one other question, if I may,

1 please. Is one of the reasons that you do a univariate
2 analysis so that if you get the confounder wrong, so to speak,
3 like arsenic, at least you have a level that you can look at
4 that is just that, alone? And then as you find out that other
5 factors may or may not be confounders, you can include them or
6 not include them; but you have that univariate analysis to work
7 with?

8 **A.** Yes. I mean, that's traditionally how epidemiologists do
9 it. They always look at the -- at each one separately in a
10 univariate analysis. And then they do a multivariate analysis

11 **Q.** Right.

12 **THE COURT:** But if an expert testified that the
13 univariate analysis for glyphosate in this chart was -- shows
14 that glyphosate causes non-Hodgkin's lymphoma, that would be
15 unreliable. Right?

16 **THE WITNESS:** It could be unreliable, yes. If you
17 told me the Odds Ratio was 10, I would say, *Probably not*. And
18 it was statistically significant, I would say, *No, it's*
19 *probably not*. If we do multivariate, it might drop to 8 or 7,
20 but it's still going to be there.

21 The problem is when you get to that low Odds Ratios, and
22 then you overadjust, they drop below being statistically
23 significant.

24 But you can see the data. You can -- you can get a
25 feeling for the data and see what's happening. Even though

1 it's not significant, it tells you something.

2 **BY MS. FORGIE**

3 **Q.** Okay.

4 **A.** So let me show you the first slide last, just to show you
5 the correct way to do it. So, in fact, what was done in the --
6 in the -- in the NAPP Study was they didn't adjust for all of
7 the 44 pesticides that were seen in De Roos, and others that
8 were seen in Cross-Canada. What they did is they said, *Okay.*
9 *What pesticides are closely correlated with glyphosate? Okay?*

10 And they said, *Okay. Now which of those pesticides are*
11 *known or suspected causes of NHL?*

12 And so in the end, they only adjusted for three chemicals:
13 2,4-D, dicamba, and malathion. That's the proper way to do a
14 multivariate analysis like this, so you don't do
15 overadjustment.

16 **JUDGE PETROU:** So going back to the biological
17 plausibility kind of a theory underlying all of this, in the
18 NAPP Study don't they also run the numbers at over
19 7-days-per-year average; not just above and below 2?

20 **THE WITNESS:** I don't know if they did. They looked
21 at number of years of use. I think maybe that's what the 7
22 was: 7 years of use.

23 **JUDGE PETROU:** I don't believe so. Okay. But in any
24 event, you don't have present in your mind data from the
25 NAPP Study relating to use of over 7 days per year?

1 **THE WITNESS:** No, no.

2 **JUDGE PETROU:** Okay. Go ahead.

3 **BY MS. FORGIE**

4 **Q.** Okay. I want to go back for one second to the Eriksson
5 table that we were discussing, with the adjusted Odds Ratios.

6 **A.** Okay.

7 **JUDGE PETROU:** I'm sorry. I'm going to correct
8 myself. Seven lifetime-days.

9 **THE WITNESS:** Lifetime-days. Yes.

10 Go ahead.

11 **BY MS. FORGIE**

12 **Q.** And you were asked some questions about the --

13 No, that's not the one. That's the NAPP. I want to look
14 at the other one. Hold on one second. Let me get the table
15 up.

16 **A.** So, yeah. We've seen this table. We should go on.

17 **Q.** Yeah. I'm looking for that one. Thank you.

18 So you were asked a couple of questions about the
19 glyphosate in NAPP and the Odds Ratio, which was statistically
20 significant at 2.02; confidence interval 1.1 to 3.71. Do you
21 see that?

22 **A.** Yeah.

23 **Q.** And that's just one piece of evidence that -- along with
24 other Odds Ratios from other studies that we've seen, and the
25 toxicology and the biological plausibility that contributes to

1 your opinions. Correct?

2 **A.** Right. And so what happened in Eriksson and what happened
3 in Hardell is that the Odds Ratios that were statistically
4 significant in the univariate analysis -- when they did the
5 multivariate adjustments, they were still elevated, but now
6 they were no longer statistically significant. But the fact
7 that they're elevated tells you that there's still risk there.
8 Okay?

9 **Q.** And so that --

10 **THE COURT:** So there's still risk for arsenic?

11 **THE WITNESS:** If you believe it causes non-Hodgkin's
12 lymphoma, you could say there's a 17 percent increased risk.

13 **THE COURT:** Well, the point of the exercise is to
14 figure out whether something causes non-Hodgkin's lymphoma. So
15 according to your statement, we would look at the number for
16 arsenic, and be concerned that it causes non-Hodgkin's
17 lymphoma, because although it's not a statistically significant
18 Odds Ratio, it's higher than 1? Right?

19 **THE WITNESS:** Yes. If you didn't know anything else
20 about arsenic, you would say, *Well, maybe arsenic does cause*
21 *non-Hodgkin's lymphoma.*

22 But I would say, *Well, the risk is only 60 percent in the*
23 *univariate, and it goes down to less than 20 percent in the*
24 *multivariate, which is -- which is just barely elevated, so you*
25 *wouldn't pay much attention to that -- okay? -- because the*

1 trend -- the trend is down, and it goes down close to 1.

2 **THE COURT:** So the conclusion that 1.2 or 1.3 or 1.4
3 Odds Ratio is not statistically significant is meaningful is
4 based on background knowledge that you have that the
5 substance -- you already have, that the substance causes
6 non-Hodgkin's lymphoma?

7 **THE WITNESS:** In part. And just looking at the
8 pattern, so it doesn't all go away. There's still an effect
9 there, even though it's not statistically significant.

10 **BY MS. FORGIE**

11 **Q.** And so, Doctor, even though it's always hard to -- what's
12 the phrase? -- prove a negative, you're aware of other
13 information that arsenic is not a confounding factor, or not --

14 **A.** Right.

15 **Q.** -- causally associated with non-Hodgkin's lymphoma, in
16 addition to which, with regard to glyphosate, you're aware of
17 lots of other information -- other epidemiological studies,
18 toxicology studies, and biological plausibility -- that
19 contribute to that -- to your opinions. Correct?

20 **A.** So I -- yeah. I would consider all of those things in
21 making my final judgment.

22 **Q.** Right. And that's part of the reason you're taking out
23 the arsenic out of this table --

24 **A.** Right.

25 **Q.** -- and saying it's not a confounder. Is that correct?

1 **A.** Right. Right. Right.

2 **Q.** And then also when the Judge asked you whether it would
3 be -- I believe you said "unreliable" to use that test, you
4 meant to just rely on one study. You don't ever rely on just
5 one study. You look at all of the information. Correct?

6 **A.** Yes.

7 **THE COURT:** Before we go off this chart, if I could
8 ask one more question. How easy or difficult would it be to
9 take arsenic out, and then do the multivariate analysis again?

10 **THE WITNESS:** We could ask Dr. Ritz to do that. I --
11 I don't have the expertise to do it, but she does.

12 **THE COURT:** How about Dr. Neugut?

13 **THE WITNESS:** Dr. Neugut could do it, too. Sure.

14 **THE COURT:** Okay. Wake up, Dr. Neugut.

15 **MS. FORGIE:** By -- wait. We got Dr. Neugut? Did I
16 miss something? Everybody's laughing at me.

17 **THE COURT:** Dr. Neugut might be taking a nap back
18 there.

19 **MS. FORGIE:** I always miss the good stuff. That's
20 why they don't let me out very often.

21 **THE WITNESS:** So I hope that clarifies the
22 multivariate analysis. And the other thing, I think, that
23 people get too hung up on is the words "statistically
24 significant." In epidemiology you want to look at all of the
25 data, and try to make sense of it. And sometimes there are

1 things there that aren't statistically significant, but they
2 tell you -- they tell you important facts.

3 **JUDGE PETROU:** Doesn't that at least go to the weight
4 for you? Whether or not you choose to completely disregard
5 something -- doesn't whether or not you have a 95 percent
6 confidence level mean something to you?

7 **THE WITNESS:** There's nothing magic about 95 percent.
8 Why wouldn't we use 90 percent? So there are things that are
9 borderline significant. So these are all just tools that we
10 use. Okay? And you have to get a feeling for how to use them.
11 They're not arbitrary. I mean, they're arbitrary, but you
12 shouldn't use them as an arbitrary tool. You shouldn't discard
13 something that's, you know, .52, for example. .052.

14 **JUDGE PETROU:** My question didn't go to whether or
15 not to discard something, but whether -- how much weight you
16 give something.

17 **THE WITNESS:** Well, you would weigh it in the context
18 of the all of the other information. Does it make sense?
19 Doesn't it make sense? I mean, that's the best I can answer.
20 You wouldn't discard it. You would at least consider it.

21 **BY MS. FORGIE**

22 **Q.** I'm going to move along quickly, because we do have
23 another epidemiologist who's probably ready to kill me if I
24 don't move along and take up all of his time.

25 A couple of really quick questions. Yesterday you spoke a

1 little bit about latency, and I just wanted to clear up one
2 thing. You're not offering any type of opinion with regard to
3 an absolute minimum latency period for NHL to develop; are you?

4 **A.** No, I'm not. My comments yesterday were about the
5 Eriksson Study. And in the Eriksson Study, what they -- what
6 they found was that in that study, they had to have a latency
7 of at least 10 years to see a statistically increased risk. Of
8 course, many of those people probably have much longer latency,
9 but 10 years was the minimum.

10 So what I was trying to say -- and I probably didn't say
11 it very well -- was that in an epidemiologic study, you have to
12 allow time for the disease to develop. And -- and in Eriksson,
13 the number was 10. Okay? It's not a magic number, but in that
14 number it was 10. So it gives us some information about
15 glyphosate.

16 **JUDGE PETROU:** No. I understand that. My question
17 was going more to kind of the general medical knowledge in this
18 area, since you are clearly very knowledgeable regarding
19 non-Hodgkin's lymphoma. I know with many cancers, it really
20 has not been determined what the latency period really is.

21 **THE WITNESS:** Right.

22 **JUDGE PETROU:** Some there have. Things like
23 mesothelioma, we know there's a minimum. Others, there aren't.
24 So I was curious as to NHL whether there is a generally
25 accepted medical understanding of the latency period, or

1 whether this remains kind of a question mark at this point.

2 **THE WITNESS:** Well, it is a question mark, because
3 latency depends on a lot of things. It depends on the potency
4 of the chemical. If it's a strong carcinogen, the latency
5 would be short, and it would induce cancers early. If the
6 carcinogen was a weak carcinogen, it -- it might take many,
7 many years.

8 **JUDGE PETROU:** In regards to this matter, in your
9 opinion about the connection between glyphosate and
10 non-Hodgkin's lymphoma, I understand from the Eriksson Study
11 that you were talking about they needed the 10 years to start
12 really seeing it; but am I hearing you correctly that you don't
13 really have -- or there isn't in the general medical literature
14 a clear opinion or statement as to the latency period between
15 glyphosate and NHL, presuming that there is a connection?

16 **THE WITNESS:** There's very little known. Very little
17 known. The thing I told you about Eriksson is the only thing
18 we really know.

19 I mean, in general for non-Hodgkin's lymphoma, you know,
20 I've done some work in solvent exposures. Mixed solvents cause
21 non-Hodgkin's lymphoma. And if you look at the various
22 studies, the median time of -- the median latency time is about
23 20 to 25 years.

24 And I did mention yesterday people who get high-dose
25 chemotherapy for cancer are at increased risk for non-Hodgkin's

1 lymphoma. And those people -- the late tenancy period could be
2 quite short; five years or less. So it really depends on the
3 exposures; your intensity of exposure. Is it a strong
4 carcinogen? A weak carcinogen? It -- yeah. There's no magic
5 number.

6 **MS. FORGIE:** Okay. So I'd better -- I think I've
7 doubled my time, which probably puts me at a p-value of
8 2 billion, and a death sentence from my colleagues. So I'm
9 going to get you off the stand. Thank you, Doctor.

10 I'm going to pass the witness.

11 **MR. GRIFFIS:** Binder for you.

12 **MS. FORGIE:** Thank you. This time we won't lose it.

13 **MR. GRIFFIS:** Good.

14 (Whereupon a document was tendered to the Court.)

15 **CROSS-EXAMINATION**

16 **BY MR. GRIFFIS**

17 **Q.** Good morning, sir. Good afternoon.

18 **A.** Good afternoon. Excuse me. Good afternoon.

19 **Q.** You told Judge Chhabria and Judge Petrou yesterday, with
20 one of your first slides, that you used *the same scientific*
21 *method and intellectual rigor that I use* -- I'm quoting the
22 slide -- *in my daily academic practice*. Right?

23 **A.** Yes.

24 **Q.** Now, I took your deposition in September of 2017. Right?

25 **A.** Yes.

1 **MR. GRIFFIS:** Would you put up Slide 45, please?

2 **Q.** And do you remember testifying at your deposition, sir,
3 that the standard you would use for opinions in the medical
4 article that you put your name on and publish in the medical
5 literature would be more rigorous than opinions that you give
6 in a litigation case? Did you give that testimony?

7 **A.** Yes, but that would probably be for a specific case, for a
8 specific causation, where -- I can't think of the legal
9 terminology -- "more likely than not" would be the legal
10 standard. So that's what I was meaning here.

11 **Q.** When you originally gave that testimony, sir, in *Wendell*
12 *versus Johnson & Johnson*, you said, *When I testify to a*
13 *reasonable degree of medical certainty, what I mean is just*
14 *more likely than not, but I would have a more rigorous standard*
15 *when I publish an article.* Right?

16 **A.** Yes.

17 **Q.** More likely than not. Now, you were identified --
18 Slide 1, please.

19 You were identified by plaintiffs' counsel as an expert in
20 pathology and non-Hodgkin's lymphoma. Right?

21 **A.** I don't know.

22 **Q.** Do you see the letter there?

23 **A.** Okay.

24 **Q.** Okay. So I'm going to ask you a couple of questions about
25 non-Hodgkin's lymphoma. You testified at your deposition that

1 70 percent or more of all non-Hodgkin's lymphoma is idiopathic,
2 meaning we don't know the cause. Right?

3 **A.** That's true.

4 **Q.** And that's after increased knowledge that's been gained
5 over the past few decades. Even after that increased
6 knowledge, we're still at 70 percent unknown. Right?

7 **A.** That's true.

8 **Q.** And there was a rising -- famously in this field, there
9 was a rising wave of non-Hodgkin's lymphoma in the
10 United States that was detected starting in the 1950s.
11 Correct?

12 **A.** Yes.

13 **Q.** And you testified at your deposition that that couldn't
14 possibly have been caused by glyphosate, since glyphosate
15 wasn't even on the market until sometime in the 1970s, and
16 wasn't used at a high-enough level to cause anything for some
17 time after that. Correct?

18 **A.** I don't remember that, but I believe it's correct.

19 **Q.** Okay. So whatever was causing that increasing wave of
20 non-Hodgkin's lymphoma, it wasn't glyphosate. Right?

21 **A.** Right.

22 **Q.** And obviously, whatever caused all of the non-Hodgkin's
23 lymphomas before the '50s also wasn't glyphosate. It wasn't
24 around. Right?

25 **A.** Right.

1 Q. Now, you've testified in the past, sir, that epidemiologic
2 studies in humans provide the best and most-convincing data
3 linking environmental exposures to cancer. Correct?

4 A. Well, I don't remember I said that. I think it's
5 important to have studies of humans to have epidemiologic
6 studies to make decisions.

7 Q. I don't want to talk a little bit about the case-control
8 epidemiology studies that you relied on, sir. You showed us a
9 table from your Expert Report with six case-control studies on
10 that. We saw that yesterday and today both, I believe.

11 Could we have Slide 2, please?

12 Those are the studies from that table. Right?

13 A. Yes.

14 Q. And the Cocco Study -- that was a tiny study with just
15 four exposed cases. Right?

16 A. Yes.

17 Q. And the Orsi Study was nonsignificant, with an outside
18 ratio of 1.0. Right?

19 A. Also with a small number of case.

20 Q. Also with a small number of cases. So I'd like to focus
21 on the larger ones: McDuffie, De Roos, Eriksson, and Hardell.
22 And we know now that the first two -- McDuffie and De Roos --
23 were also analyzed in the NAPP that you were a part of. Right?

24 A. Yes.

25 Q. You didn't mention NAPP. You were talking about why it

1 was good, and the improvement on McDuffie and De Roos today.

2 You didn't mention NAPP at all in your Expert Report. Right?

3 **A.** I didn't, but we talked a lot about it in my depositions.

4 **Q.** I brought it up, and we talked about it at the deposition.

5 Right?

6 **A.** Yes.

7 **Q.** Okay. The NAPP supersedes McDuffie and De Roos. You
8 testified to that at your deposition -- right? -- because it's
9 a pooling of the data?

10 **A.** Yes. And I chose -- because the NAPP was not a published
11 study, I chose to include instead McDuffie and De Roos, because
12 those are the primary studies.

13 **Q.** NAPP's not published, but there's been publicly released
14 data. There have been a number of slideshows. Right?

15 **A.** Yes.

16 **Q.** Like the one that you included in your slide deck from
17 June. And there's a later one with improved data -- further
18 data -- from August. Correct?

19 **A.** The data's different. I'm not sure it's improved, but
20 it's different.

21 **Q.** It's later data. Right? Further analysis?

22 **A.** It's later data. Yes.

23 **Q.** Okay. The -- now I want to talk a little bit about the
24 pooling. The reason you were able to pool McDuffie and -- you
25 can't just take two epidemiology studies, and pool them.

1 Right?

2 **A.** No, you can't. They have to be similar.

3 **Q.** The reason you could pool McDuffie and De Roos was that
4 they had reasonably similar methodologies in the relevant ways?

5 **A.** Yes.

6 **Q.** And the epidemiologists on the studies made the assessment
7 that it was poolable as a result of that. Right?

8 **A.** Yes.

9 **Q.** It wasn't your job to figure that part out; it was the
10 epidemiologists'. Correct?

11 **A.** Yes.

12 **Q.** Now, the NAPP results on glyphosate, as we said, were
13 presented by Dr. Pahwa in Brazil in August of 2015. Right?

14 **A.** Yes.

15 **Q.** Can we see Slide 5, please? Now this, sir, is a slide we
16 have not seen yet. This is from the August data. It's later
17 than the June data presented that you presented. And this is
18 the never/ever data; the overall data. This is what you would
19 use --

20 **THE COURT:** Could I interrupt for a second, just for
21 a clarification question?

22 **MR. GRIFFIS:** Yes.

23 **THE COURT:** When you're referring to the June data
24 they presented, you're talking about the June data from the
25 Canada presentation that you presented here this morning?

1 **MR. GRIFFIS:** Yes, sir. It was in the binder, too,
2 that was handed out. It was an earlier slideshow.

3 **THE COURT:** And the August data that you're talking
4 about is from a presentation that somebody gave in Brazil.

5 **MR. GRIFFIS:** That's right. It's Exhibit 1278.

6 **THE COURT:** Okay.

7 **BY MR. GRIFFIS**

8 **Q.** And this is the never/ever data. And this is the data
9 that used in meta-analyses. Correct? Never/ever data is
10 what's used in the meta-analyses?

11 **A.** Yes.

12 **Q.** You talk about meta-analyses in your Expert Report. And
13 if NAPP had been in those, this is the data that would have
14 been used. Right? The never/ever?

15 **A.** It would have been, yeah, never/ever.

16 **Q.** Okay. So let's get oriented and look at this. There's a
17 column of Odds Ratios. And this is Odds Ratio with a
18 superscript A, and then an Odds Ratio B.

19 And the Odds Ratio B -- what it adds is that it's adjusted
20 for other pesticides. Correct?

21 **A.** Correct.

22 **Q.** So the 113 cases -- you told us earlier there were 113
23 exposed cases in NAPP. And here they are. We originally had a
24 1.43 statistically significant, but when it was adjusted for
25 other pesticides, that became an Odds Ratio of 1.13. Not

1 significant. Right?

2 **A.** Right.

3 **Q.** Okay. So when the McDuffie and De Roos data was pooled
4 and adjusted for other pesticides -- something that couldn't be
5 done for all of that data together in either of the two
6 studies -- we got a non-significant result. Correct?

7 **A.** For ever/never.

8 **Q.** For ever/never. Right.

9 And what made this non-significant, again, was the
10 adjustment for other pesticides. Right?

11 **A.** Correct.

12 **Q.** There was a draft publication that we obtained at the
13 deposition of Dr. Blair, and I talked about with you at your
14 deposition. Right? And in that draft manuscript, the authors
15 said that 2,4-D, dicamba, and malathion, in fact, are
16 associated with non-Hodgkin's lymphoma in case-control studies.
17 And you agreed with that today on the stand. Right?

18 **A.** Right.

19 **Q.** Okay. And it was the adjustment for those -- An
20 adjustment for pesticides like that is absolutely appropriate,
21 and a good idea, and it improves the numbers. Right?

22 **A.** Correct.

23 **Q.** Do you agree, sir? It's definitely true that other
24 pesticide exposures can be a major confounder for whether
25 glyphosate can cause non-Hodgkin's lymphoma?

1 A. That's true.

2 Q. By the way, remember when we discussed the De Roos 2003 at
3 your deposition, and you said that there were three Odds Ratios
4 in that paper -- a logistic regression, a hierarchical
5 regression, and a linear regression -- and the only one that
6 was statistically significant was the linear one?

7 A. I think it was the logistic one.

8 Q. Okay. Let's have Slide 81.

9 A. I'll have to go back and look. It was --

10 Q. There was only one that was statistically significant.
11 Right?

12 A. Right.

13 Q. And that's the one you reported on your Expert Report?

14 A. Yes.

15 Q. That's the one Dr. Ritz reported in her slides yesterday?

16 A. Yes.

17 Q. And you left off the other two. Right?

18 A. Yes.

19 Q. And you don't know, sir, which of those three regressions
20 best controls for other pesticides? You so testified at your
21 deposition?

22 A. I -- I don't really know which does it best. The -- I
23 think the hierarchical regression is considered to be more
24 conservative, but it probably overadjusts. I think De Roos
25 overadjusted in her study.

1 Q. At your deposition, sir, you testified that you don't know
2 which one best controls for other pesticides exposure.

3 Correct?

4 A. I don't know which one best does.

5 Q. Okay. Now --

6 THE COURT: Could -- I'm sorry to interrupt. Could I
7 ask a clarification question? Probably a dumb one. What's the
8 difference between a logistic regression and a hierarchical
9 regression?

10 THE WITNESS: I think you have to ask Dr. Neugut.
11 Okay. They're both very --

12 THE COURT: Is he there -- Dr. Neugut?

13 THE WITNESS: Is Dr. Neugut there?

14 So they're both complicated mathematical formulas to do
15 it. And it's -- I don't understand all of the details of why
16 one is different than the other, but they're -- clearly they --
17 they're similar, but the hierarchical has another step of
18 adjustment that it does.

19 THE COURT: Okay. But in your knowledge, at a
20 minimum, are you aware of whether both the logistic and the
21 hierarchical regressions adjusted for other pesticides?

22 THE WITNESS: They both did.

23 THE COURT: Okay. All right.

24 MR. GRIFFIS: Okay, sir. Let's have Slide 94,
25 please.

1 BY MR. GRIFFIS

2 Q. This is, again, from the August data, sir; the August
3 presentation. And this is different data than what you
4 presented from the June presentation, and what you showed on
5 the slide today. Right?

6 A. This is.

7 Q. It's later data?

8 A. Yeah. This is slightly later data. Yes.

9 Q. Subject to further analysis. Correct? And you testified
10 at your deposition, sir, that the -- there's a negative trend
11 that appears when we look at the duration of glyphosate use.
12 Correct? And we see that when we look at the number of years
13 of exposure, and see that the numbers go down; the -- the Odds
14 Ratios go down when we compare the zero, greater than zero, and
15 less than or equal to 3.5, to the greater than 3.5 exposures
16 for overall, for follicular, for DLBCL; not for SLL, but that
17 was not significant; and for other. Correct?

18 A. Correct. So what this says is that --

19 Q. It was --

20 A. -- looking at the number of years of exposure is not
21 really predictive. You would predict that the more years
22 you're exposed, the higher the Odds Ratio. And here it's the
23 opposite.

24 Q. Yeah. This is not consistent with the hypothesis that
25 glyphosate causes non-Hodgkin's lymphoma?

1 A. No, it's just -- this happens in other data, too. We had
2 the same findings for 2,4-D in our Nebraska Study, where
3 intensity of exposure increased risk, but number of years of
4 exposure didn't increase risk.

5 Q. Right, sir. So the slide that you wanted to show us was
6 the intensity one. It's the next slide. Could we have
7 Slide 95? Frequency days per year. Right?

8 A. Okay. Yeah.

9 Q. And --

10 A. So this is a variation on -- this is an update of the
11 slide that I showed earlier.

12 Q. Yes, sir. It's an update of the slide you showed earlier.
13 And this is one that you said shows some statistically
14 significant trends. Correct?

15 A. Yes. And the Odds Ratios are even higher in this than
16 they were in the ones I showed.

17 Q. And -- well, not for all of them, but we'll get to that,
18 sir.

19 And the next one combines the two. The next slide
20 combines the two lifetime-days --

21 **THE COURT:** So could you go back to the previous
22 slide, and just let me stare at it for a second?

23 **MR. GRIFFIS:** Yes, sir.

24 **THE COURT:** All right. Thank you.

25 **THE WITNESS:** This data is not adjusted for

1 pesticides.

2 **BY MR. GRIFFIS**

3 **Q.** Yes, sir. It goes down when you're adjusting.

4 **A.** That's the difference between the slide I showed, and this
5 one. That's the reason I didn't show this slide.

6 **Q.** And this one -- these numbers would all go down, if
7 adjusted for other pesticides. Right?

8 **A.** In fact, they do.

9 **Q.** Okay. Let's -- ah -- let's have Slide 105, sir. I'm
10 sorry. Slide -- yeah. That's it.

11 This is from Dr. Ritz's presentation. Right? Were you in
12 the audience for this when this was being shown?

13 **A.** Yes.

14 **Q.** This is what she showed for the NAPP for greater than 2
15 days per year. And what she was talking about was this -- what
16 you're calling "intensity exposure." Right?

17 **A.** Right. I think --

18 **Q.** And the actual?

19 **A.** -- 2 days per year would be a crude surrogate for
20 intensity of exposure, or frequency of exposure.

21 **Q.** So her bars here are not controlled for other pesticides.
22 Right? It goes down if you do that?

23 **A.** Are you talking -- what set are you talking about now?

24 **Q.** Well, these bars are coming from --

25 **A.** From the original McDuffie? No, that wasn't adjusted for

1 other pesticides.

2 Q. And it's coming from that NAPP data that we're looking at.

3 Right?

4 A. Yeah. The NAPP data's there, too. I don't know which
5 version she used --

6 Q. Okay.

7 A. -- but the NAPP data's there, too.

8 Q. The SLL data goes below the line here. Right?

9 A. It does.

10 Q. So it goes beyond -- so it would be incorrect to say --
11 it's as shown here. Right?

12 A. I have to believe what you're showing me here. I don't
13 have the original one, so --

14 Q. Okay. Yeah, because it's not on your screen anymore. And
15 all of these adjustments reflect the data in this slide that we
16 were just looking at -- correct? -- including the 113, by the
17 way. That's the number you told us earlier is the actual
18 number of exposed cases. Right?

19 A. Correct.

20 Q. Okay.

21 A. So it, in general, depicts the data that I showed you the
22 numbers on. Right.

23 Q. Now, when we combine -- when we go to Slide 96, and
24 combine the duration with what you're calling "intensity," and
25 look at years times days per year --

1 **JUDGE PETROU:** Hold on, Counsel. I just have a
2 clarifying question. You talk about combining, but this slide
3 does not seem to indicate that it's at least 2 days per year.

4 It has over 7 days, which -- that could be, for example, 7
5 days over 14 years.

6 **MR. GRIFFIS:** That's correct.

7 **JUDGE PETROU:** That's correct. So it's not actually
8 combining the two previous slides. One of those was focused on
9 number of days per year?

10 **MR. GRIFFIS:** That is right. And it's lifetime-days.
11 And then it's reached by number of years times number of days
12 per year to achieve that.

13 **Q.** But what we see when we combine the two -- and we said
14 this in your deposition, sir -- is that all of these results
15 are not significant. Correct?

16 **A.** They -- they aren't significant. Yes. But this is the
17 effect of using number of years, because it dilutes that data,
18 and no longer shows you intensity. It just shows you number of
19 years -- number of days over the period of time.

20 **Q.** And at the deposition you said, *Yeah, so that's why*
21 *intensity is better.*

22 And I said, *It's a better way to get statistically*
23 *significant results to report.*

24 And you said, *Yeah, that's what epidemiologists do.*

25 Right?

1 A. Well, you have to look at the data in all of the different
2 ways, which is what they did. And then you have to try to
3 understand the data.

4 And so that's what I did when I reviewed this data. And
5 this is what we found also in the Nebraska Study; that it
6 seemed like intensity of exposure greater than 2 days per year,
7 not using protective equipment -- these all would increase the
8 dose and intensity of the exposure. And those are the things
9 that are the best predictors.

10 Q. The different ways to cut -- so you cut the data a whole
11 bunch of different ways. And what you reported here as your
12 expert testimony is the most significant way that you could cut
13 the data that you could find. Right?

14 A. In the manuscript and in the abstracts, they present all
15 of the data. They don't hide any data. They say --

16 Q. No, sir.

17 A. -- *We did this. We did that. We showed this. And this*
18 *is what we found.*

19 Q. And what you put in your Expert Report and testified to on
20 the stand is the most significant findings that you could find.
21 Right?

22 A. Well, I -- I was -- I was.

23 Q. You didn't show us --

24 A. I was trying to -- to show the data from the NAPP that I
25 thought was the important data.

1 Q. You did not show us the overall finding that reduces
2 McDuffie and De Roos 2003 to a non-significant 1.13, with an
3 Odds Ratio of 0.84 to 1.51. You didn't show us that. Right?

4 A. No. I have could have.

5 Q. I agree.

6 Let's talk about Eriksson and Hardell. Slide 12, sir.

7 Eriksson, you testified at your deposition, shows no
8 statistically significant association between glyphosate and
9 non-Hodgkin's lymphoma, or any subtype that is adjusted for
10 other pesticides. Right?

11 A. I'd have to look at the study again.

12 Q. Let's look at Slide 27. Question and answer from your
13 deposition, sir. *There's no statistical --*

14 *And this was after we went through the study.*

15 *There's no statistically significant association between*
16 *glyphosate and non-Hodgkin's lymphoma or any subtype of*
17 *non-Hodgkins lymphoma in the studies that is statistically*
18 *significant, greater than 1, and controlled for other*
19 *pesticides. Right?*

20 *And you answered, That's correct.*

21 Do you remember that?

22 A. I believe that's correct.

23 Q. For Hardell, Slide 85, there was no Odds Ratio reported in
24 that study that showed a statistically significant association
25 between glyphosate and non-Hodgkin's lymphoma controlled for

1 other pesticides. Right?

2 **A.** That's true, but the risks were still elevated after
3 adjustment. And that's what you would expect. Okay? You
4 can't just focus on what's statistically significant. You have
5 to look at all of the data. You have to make some judgments
6 about what the data tells you.

7 **Q.** Yes, sir. It was still -- it was still elevated. Like,
8 arsenic was still elevated in the data that we were looking at
9 earlier. Right?

10 **A.** Right. It decreased some, but it didn't go to the null or
11 near the null. It stayed elevated.

12 **Q.** The adjusted numbers we were just looking at for the NAPP
13 were below the arsenic level of 1.63 when we started, and they
14 moved even lower when you adjusted for other pesticides.
15 Right?

16 **A.** The question's too complex. I'm not going to answer that
17 question unless you restate it.

18 **Q.** Okay. Sir, I'll move on. Could we move into the McDuffie
19 article? That's Defense Exhibit 1179. Go to page 1161,
20 Table 8. Okay. Sir, I want to just ask you a couple of
21 questions about a question that Judge Petrou asked.

22 It would be nice if we could get the "out-of-range" thing
23 out (indicating).

24 Judge Petrou asked yesterday about how the dates for these
25 studies were picked. And this is -- this is just one of the

1 studies, of course: The McDuffie Study. And we can see when
2 we look at the various substances that were considered in
3 McDuffie that all sorts of different dates -- date cutoffs were
4 used. Correct? The date cutoffs for glyphosate are different
5 than the date cutoffs for 2,4-D; different for MetaCrop, et
6 cetera. Right?

7 **A.** I don't see any dates here.

8 **Q.** And I mean number -- I mean number of days, sir. Days per
9 year of exposure. Do you see that?

10 **A.** I do. Yes.

11 **Q.** Okay.

12 **A.** So what they're doing is they're parsing --

13 **THE COURT:** I'm not sure he's asked you a question
14 yet.

15 **THE WITNESS:** Oh, okay.

16 **BY MR. GRIFFIS**

17 **Q.** Okay. It may be, sir, that if you took the data for any
18 one of these particular exposures, like glyphosate, and it cut
19 it for 3 days instead of 2 days, or 1 day instead of 2 days,
20 you might get a completely different and non-significant
21 result. Right? The numbers might go down sharply?

22 **A.** It's possible. It's not likely, but it's possible.

23 **Q.** It may be the selection of days that enables the
24 Odds Ratios to be as high as they are. Right?

25 **A.** So I don't believe they manipulated the data to do that

1 sort of thing. Epidemiologists are about the most ethical
2 people you could know, so I don't think that they do this kind
3 of thing just to find something. Okay? I think that's
4 overstating -- overstating things.

5 **Q.** They certainly use different cutoffs for these different
6 formulations. Right?

7 **A.** They're trying to learn. They're trying to understand the
8 data.

9 **Q.** Is it kind like what you said earlier? That we are trying
10 to find the most significant results to report? That's what
11 epidemiologists do? They're trying to learn by seeing where
12 they can show the strongest results?

13 **A.** Epidemiologists try to find truth. That's what they try
14 to do.

15 **Q.** Yes, sir. You invoked the Acquavella 2004 Study yesterday
16 for the proposition that 60 percent of farmers had detectable
17 glyphosate in their urine on day of application. Is that
18 right?

19 **A.** That's correct.

20 **Q.** Okay. So that's what that was from. It was from the
21 Acquavella 2004?

22 **A.** It was from the biomonitoring study. Yes.

23 I don't know what year it was, but --

24 **Q.** Okay. I'd like to go to a different part of that same
25 study that you relied on yesterday, sir. This is Exhibit 511.

1 And I believe it's -- I believe it's our Slide 102. This is
2 Tab 16 of our binders, counsel.

3 **MS. FORGIE:** Thank you.

4 **MR. GRIFFIS:** Can we have that, Scott? Slide 102?
5 That's right.

6 **Q.** So this is -- sir, this is Exhibit 511, Tab 16 of the
7 binder, page 324 of the Acquavella Study that you relied on
8 yesterday. And what it said about the dose that these farmers
9 were exposed to -- maximum systemic dose for farmer applicators
10 was estimated to be .004 milligrams per kilogram. Correct?

11 **A.** That's what it says.

12 **Q.** Do you see that?

13 And the distribution of values was highly skewed. The GM,
14 which is geometric mean, systemic dose -- the mean dose -- was
15 .0001 milligrams per kilogram. Right?

16 **A.** That's what it says.

17 **Q.** And the comparison here is to the USEPA
18 lowest-to-no-effect level from glyphosate toxicology studies,
19 which is 175 milligrams per kilogram per day. Correct?

20 **A.** That's what it says.

21 **Q.** Okay, sir. Now I'd like to talk about latency. We talked
22 about latency some at our deposition; didn't we?

23 **A.** Yes.

24 **Q.** At the deposition you were criticizing the AHS data. We
25 had two depositions, actually. We had one where we didn't have

1 the AHS 2018 data yet, and you were criticizing the latency and
2 the De Roos 2005 information. And then later when AHS 2018
3 came out, you criticized the latency in that study. Correct?

4 **A.** What I really criticized was the short follow-up.

5 **Q.** At the deposition you were criticizing AHS 2018 data; at
6 your second deposition, sir, in January. And you told me that
7 18 years of follow-up probably was not enough in an NHL study.
8 Do you remember that?

9 **A.** Yes. And what I meant was that usually for these
10 retrospective and prospective cohort studies, to -- to find
11 truth, you have to follow the -- the people in the cohort for
12 30 or 40 years, or until most of the people are dead. So 18
13 years in a cohort study -- this is a general comment. 18 years
14 in a cohort study is good follow-up, but it may not be long
15 enough to -- to see an effect.

16 **Q.** Yes. And I --

17 **A.** The latency is very long.

18 **Q.** And I asked you -- after you said 18 years isn't enough, I
19 said, *Is that because it takes a long time for non-Hodgkin's*
20 *lymphoma to show up after an exposure?* And you said, Yes. Do
21 you remember that?

22 **A.** Makes sense.

23 **Q.** And you agree that probably 10 years is when you would
24 begin to see cases that are associated with a chemical. Right?
25 That was your testimony?

1 **A.** Well, based on -- I clarified that earlier, because it
2 was -- it wasn't clear when I testified yesterday on what --
3 for the purposes of an epidemiologic study, and -- and this is
4 the Eriksson Study, specifically. So I can't speak for all
5 studies, but in the Eriksson Study they needed to have a -- I
6 don't know the word -- a latency -- a latency of at least 10
7 years. They did see some cases prior to 10 years; but to see a
8 statistically significant increase, they needed a latency of at
9 least 10 or more years. Okay? So that's one study.

10 It's interesting information. I don't know whether we can
11 generalize that to other studies, but it's the only information
12 we have about latency *in vivos* in non-Hodgkins lymphoma.

13 **Q.** Based on your own work, it's quite unlikely for a person
14 to develop NHL after one or two years of exposure. Right?

15 **A.** That's true.

16 **Q.** You were being questioned by Ms. Forgie. And she said, *Is*
17 *it possible to develop non-Hodgkin's lymphoma in one or two*
18 *years?* And you said it is possible after a short exposure, but
19 it would be quite unlikely?

20 **A.** Yeah. I would stand by that.

21 **Q.** And to be more specific -- and this is in your Expert
22 Report, and your own publication, sir. There's some
23 evidence -- your words -- some evidence with very toxic agent
24 at high exposure, like intravenous chemotherapy drugs, that
25 non-Hodgkin's lymphoma can be caused in one or two years.

1 Right?

2 **A.** Well, in a short period of time. Probably the minimum is
3 two years, but a short period of time. Certainly less than
4 five years, you can see cases.

5 **Q.** Okay. So two is a little too short. More like two to
6 five? In that range?

7 **A.** I would think that's a ballpark.

8 **Q.** And -- and to be clear, we're talking about IV
9 chemotherapy, which is designed to -- which is toxic by design?

10 **A.** Right.

11 **Q.** And you're hoping that it -- you're just hoping it kills
12 more cancer cells than other cells. Right?

13 **A.** Yes.

14 **Q.** Nobody's surprised that that can cause cancer?

15 **A.** Yes.

16 **Q.** You say in your Expert Report that the average latency
17 period for the development of non-Hodgkin's lymphoma due to
18 long-term exposure to carcinogen and chemicals is about 20
19 years, with a range of 10 to 30 years. Right? That's from
20 your Expert Report, Slide 13?

21 **A.** Or 30 or more years. So those are very general statements
22 to -- to just state some principles.

23 **Q.** You said, sir, that when you -- when you originally formed
24 your causation opinion, you only had the 2005 AHS data. Fair?

25 **A.** That's correct.

1 Q. There was data from 2013 -- a draft manuscript -- but you
2 didn't see that until I showed it to you. Right?

3 A. You're talking about the update of the -- of the AHS that
4 was published?

5 Q. Right, from 2013. You saw that at the deposition. And
6 then there was a later publication in 2018?

7 A. Right. Actually, I saw it before the deposition, because
8 I thought you had probably ask me about it. I didn't have it,
9 and I had to ask for it.

10 Q. Okay. You saw it -- somebody else being questioned about
11 it in a deposition?

12 A. Yes.

13 Q. And you asked for it. So you got to see it a week or two
14 before your deposition?

15 A. Yes.

16 Q. All right. Now, certainly it's a negative study about
17 glyphosate and non-Hodgkin's lymphoma. The data do not show an
18 association. Right?

19 A. Correct.

20 Q. And could we have Slide 17, please, Table 2 from the AHS?
21 And we're certainly not going to walk through all of this, but
22 this is all of the results and the point estimates for various
23 doses for all cancers: For various solid tumors, for
24 non-Hodgkin's lymphoma, for lymphohematopoietic cancers, et
25 cetera. And some of them are above 1. Some of them are below

1 1. Pretty much everything straddles the line of 1, and is not
2 significant. Correct?

3 A. For non-Hodgkin's lymphoma, almost everything's below 1.

4 Q. Not everything, sir. Some of them are above 1. Some of
5 them are below 1. And pretty much everything straddles 1, and
6 thus is not significant. Correct?

7 A. For -- for what? For non-Hodgkin's lymphomas?

8 Q. For all cancers. For everything.

9 A. Yeah.

10 Q. When you look at the chart as a whole, what I said is
11 true. Right?

12 A. Yes.

13 Q. And I asked you at your deposition, *When you see something*
14 *like this, when you see epidemiology results that show a whole*
15 *lot of values near 1, some above 1, some below 1, pretty much*
16 *everything straddling 1, that is what you would expect to see*
17 *when the substance being tested does not cause cancer?*

18 And you agree with that. Right?

19 A. If the study is well done, that's true.

20 Q. Recall --

21 And you agreed with me that the AHS cohort of data is
22 highly informative. Right?

23 A. It's highly informative for other pesticides. It's not
24 very informative for glyphosate.

25 Q. Now, you said at your deposition that uniquely for

1 glyphosate, it's not informative. Right? And you believe that
2 to be true?

3 **A.** Yes.

4 **Q.** Okay. And recall bias is not a flaw in the NCI 2018 data.
5 Right? The AHS data?

6 **A.** Recall bias is not a flaw, no.

7 **Q.** Recall bias is something that's inherent not to cohort
8 studies, but to case-control studies. Right?

9 **A.** Yes.

10 **Q.** And it's something you try to control for and you try to
11 deal with, but it's just inherent. It's just there. The
12 people who are sick are going to tend to ruminate on their
13 condition and tend to remember exposures better than people who
14 are just going about their lives, healthy, and not worried
15 about what may have caused this?

16 **A.** It's a hypothetical thing that you always are concerned
17 about when you do a case-control study.

18 **Q.** Yes, sir. And the Blair Study that you talked about that
19 tried to assess that was just about the De Roos Study; not
20 about the other case-control studies you relied on. Correct?

21 **A.** It was actually about the Nebraska Study which was part of
22 De Roos.

23 **Q.** Okay. So it was about a subset of the De Roos Study.
24 Right?

25 **A.** Correct.

1 Q. And the AHS data, you agree, is much, much larger; the
2 number of exposed cases.

3 Let's have Slide 104, please.

4 This is your table -- your table from your Expert Report,
5 sir.

6 A. Correct.

7 Q. And we added up the number of exposed cases.

8 Could you show that, please, Scott?

9 There were 140 total exposed cases in all of the
10 case-control studies. Yes?

11 A. If your math is right, I guess I'll agree with it.

12 Q. Compared to 440 exposed cases in Andreotti 2018 AHS data.
13 Correct, sir?

14 A. More or less exposed. We talked at length yesterday about
15 exposure misclassification. So they were exposed. Some of
16 them were exposed. Some of them probably weren't exposed.

17 Q. Okay. We're going to talk about nondifferential bias in a
18 moment, sir; but 440 cases were found to be exposed in that
19 study. Right?

20 A. I don't have it in front of me. I'll take your word for
21 it.

22 Q. Okay. But you told me at the deposition that you gave
23 this NCI 2018 data no more weight than you gave to the De Roos
24 2005 Paper. It didn't move the needle at all for you. Right?

25 A. That's correct.

1 Q. You testified yesterday that the AHS -- the 2018 AHS was
2 too short, with 8.5 median years of exposure time. Right?

3 A. Well, my main criticism of AHS is that exposure
4 misclassification. Okay? That's the major flaw that's not
5 redeemable. And the other is a lesser criticism, but I think
6 also a valid criticism; but I would say the main reason that I
7 discounted the results from the study is the -- the
8 methodologic problems that occurred that led to nondifferential
9 exposure misclassification.

10 Q. Nondifferential exposure misclassification, as we learned
11 yesterday, is something that causes potentially one person to
12 be classified as exposed when they're really unexposed; one
13 person to be classified as unexposed when they're really
14 exposed.

15 And the effect of that overall -- we discussed this at
16 your deposition, too, sir -- is kind of to blur the data a
17 little bit. Right? And --

18 A. Right.

19 Q. -- in epidemiological terms, to "bias towards the null."
20 Right?

21 A. Correct.

22 Q. So if we go back to that chart, Table 2, we were just
23 looking at that showed all of the confidence intervals --

24 Not that one. The one from NCI 2018, showing the
25 confidence intervals.

1 -- some that are slightly above 1, some that are slightly
2 below 1, when you say that it biases the results toward the
3 null, that means that if there really is nondifferential bias,
4 and you remove that nondifferential bias, the true findings
5 would be a little bit farther from the null. Right?

6 **A.** Correct.

7 **Q.** So, for example, if you had a result of 1.1, but there was
8 some nondifferential bias, and you could somehow magically
9 remove the nondifferential bias, you might find a new relative
10 risk of 1.2. Right?

11 **A.** That's true.

12 **Q.** And, contrariwise, if you have 0.9 as you point
13 estimate -- and that's more like what we have for non-Hodgkin's
14 lymphoma -- and you remove the non-differential bias, it might
15 go down to 8.5. Right?

16 **A.** It might go back up to 1. It always goes to 1.

17 **Q.** Yes, sir. But if you have already been biased towards the
18 null, then the true value is farther from the null. Correct?

19 **A.** That's correct.

20 **Q.** So if the value that you measured with your
21 nondifferential bias is .9, true value would be a little
22 farther from the null: .85 or something. Right?

23 **A.** No. It would be closer to the null; not further from the
24 null. It would be closer to the null. It biases to the null,
25 either way you do it.

1 Q. Yes, sir. If you bias towards -- I don't think you're
2 understanding this, sir. If you bias towards the null, then
3 the result that you report is closer to the null than the true
4 value. Right?

5 A. That's correct.

6 Q. So a result of .9 is closer to the null than the true
7 value of .85. Right?

8 A. That's correct.

9 Q. Okay.

10 A. I may be miss -- misunderstood what you said.

11 Q. Okay. Sir. So I'd like to get back to the point I was
12 making earlier before you brought up exposure
13 misclassification. You testified yesterday that the AHS was
14 too short, at 8.5 years of median exposure. Right?

15 A. I testified that it was probably too short.

16 Q. Okay.

17 A. I don't know that it's too short, but it's probably too
18 short.

19 Q. And the median years of exposure in NAPP --
20 Median years of use -- that's what you were talking about.
21 Median years of use in NAPP was five years. Right?

22 A. I don't know. It was short.

23 Q. It was short?

24 A. It was relatively short. I don't know how short it was.

25 Q. Slide 107, please, Scott.

1 It was an average of five years. Right? Median exposure
2 in the NAPP?

3 **A.** So I don't know where this comes from.

4 **Q.** It comes from the draft manuscript from Pahwa that we had
5 produced to us in the Blair deposition --

6 **A.** Okay.

7 **Q.** -- that we talked about at your deposition, sir.

8 **A.** Okay.

9 **Q.** So if it's too -- if median exposure is too short at 8.5
10 years, it's certainly too short to get a reliable result in
11 five years.

12 **A.** So my comment was a general comment that, in general, for
13 cohort studies, you require a long follow-up. Okay? And
14 you -- and -- and in order to see and find all of the cases.
15 It wasn't -- it wasn't a specific statement, necessarily, about
16 any specific study. And my opinion was that I thought that 8.5
17 years of exposure may not be enough exposure, if it wasn't a
18 lot of exposure to give an effect. So I'm making those
19 comments in the context of how do you design an how do you
20 carry out prospective cohort studies.

21 **Q.** If 8.5 isn't enough -- I mean, you were hypothesizing, I
22 guess, that 8.5 might not be enough.

23 If 8.5 might be enough, then 5, even more so, might not be
24 enough. Right?

25 **A.** Yeah, but we saw an effect here.

1 Q. And if 5 isn't enough, and you saw an effect, it must not
2 be a true effect. Right? It must be something else that
3 you're seeing?

4 A. Well, there could be other factors involved.

5 Q. Other pesticides?

6 A. So what happens in a cohort study is that you have an
7 enrollment period. Okay? And you enroll everybody in that
8 enrollment period.

9 If they have a history of cancer or lymphoma that they
10 developed prior to the enrollment period, you drop them out of
11 the study. Okay? So all of those early cases that were --
12 could have been less than 5 years or less than 8 years were
13 dropped out.

14 So you're starting with a clean cohort, with a lot of
15 people already dropped out. These could be people who had high
16 exposures. They could be people who were susceptible to lower
17 exposures.

18 And so the two studies are very different. Okay? The two
19 studies are very different.

20 Q. I want to talk about something else.

21 **JUDGE PETROU:** Can I just interject for one second
22 here? Because the slide you have up says that it was an
23 average of 5 days per year in the NAPP Study. Do you know in
24 the AHS what the average was?

25 **THE WITNESS:** I'm sure --

1 **JUDGE PETROU:** It's in there somewhere.

2 **THE WITNESS:** It's in there somewhere. Yes. I don't
3 have it in front of me.

4 **BY MR. GRIFFIS**

5 **Q.** Okay, sir. I want to ask you another question about NAPP,
6 sir. The NAPP data for glyphosate is years old, and hasn't
7 been published yet. Right?

8 **THE COURT:** Could I -- before you get to that
9 question, I want to ask one more follow-up question on this
10 latency issue.

11 **MR. GRIFFIS:** Yes, sir.

12 **THE COURT:** And then I think it might be time to take
13 a break, depending on how much time you think you have left.

14 **MR. GRIFFIS:** One minute.

15 **THE COURT:** Oh, okay. And then we'll take a break.

16 **MR. GRIFFIS:** Yes, sir.

17 **THE COURT:** So let me ask you a follow-up question on
18 exposure time, or number of years exposed. As I understand
19 it -- and I may be getting the dates somewhat wrong, but as I
20 understand it, the pools in the De Roos study were -- they're
21 from, like, the '80s. Is that right?

22 **THE WITNESS:** The studies? Yes. They -- they --
23 they were done in the '80s, and they included time before the
24 '80s. So --

25 **THE COURT:** When you say they included time before

1 the '80s, what does that mean?

2 **THE WITNESS:** Well, they included exposures that
3 occurred. So they -- we did those studies in the mid 1980s.

4 **THE COURT:** And so people were -- you were looking at
5 exposures from when to when? From, like, the late '70s to the
6 early '80s?

7 **THE WITNESS:** We were looking at all of the exposures
8 prior to the time they got the diagnosis.

9 **THE COURT:** Okay. So people were diagnosed. These
10 people that you were looking at in the mid '80s were diagnosed
11 with non-Hodgkin's lymphoma when?

12 **THE WITNESS:** In the mid '80s.

13 **THE COURT:** In the mid '80s or early '80s?

14 **THE WITNESS:** Mid '80s. Well, for Nebraska it was, I
15 think, mid '80s.

16 **THE COURT:** Okay.

17 **THE WITNESS:** For the other studies it was a bit
18 earlier. Kansas and Iowa and Minnesota, were earlier.

19 **THE COURT:** Okay. And were diagnosed earlier than
20 the mid '80s?

21 **THE WITNESS:** Yes.

22 **THE COURT:** Okay. And glyphosate started to be used
23 in the mid to late '70s. Is that right?

24 **THE WITNESS:** 1974, '75. Yeah.

25 **THE COURT:** Okay. So if people -- based on

1 everything you've said here today, if somebody was diagnosed
2 with non-Hodgkin's lymphoma in the early '80s, shouldn't we
3 assume that it very likely was caused by something other than
4 glyphosate?

5 **THE WITNESS:** Well, that -- that's obviously what the
6 defense is trying to say, but --

7 **THE COURT:** Well, I'm not really paying that much
8 attention to the defense, but I'm paying attention to what
9 you're saying. And it sounds like what --

10 **THE WITNESS:** So that's one thing you would consider.

11 **THE COURT:** Let me just ask my question, if I could.

12 I mean, I think you said that you need, you know,
13 potentially 18 years of follow-up. And a cohort study is not
14 enough. And you made reference to -- I think it was the
15 Eriksson Study, where they didn't see any meaningful spike
16 until after 10 years of exposure. Right?

17 **THE WITNESS:** Right.

18 **THE COURT:** So if -- if those two statements that you
19 made are meaningful, then why wouldn't they cause us to
20 conclude that somebody who came down with non-Hodgkin's
21 lymphoma in the early '80s or late '70s got it from something
22 other than glyphosate?

23 **THE WITNESS:** Well, I think you would have to
24 consider that. And -- because the -- the exposure times were
25 relatively short compared to other things I talked about, but

1 that's why you do the multivariate analysis. And that's why
2 you adjust for the other pesticides, to answer that question.

3 And if the answer was actually true that there was another
4 pesticide that was actually causing the lymphoma instead of
5 glyphosate -- a confounder -- then it should decrease the
6 glyphosate to near the null, which, in the -- which didn't
7 happen in De Roos, and didn't happen in the NAPP.

8 So in both De Roos there's a statistically significant
9 increase of over 2, which was adjusted for all of the other
10 pesticides. Okay? It was over adjusted for all of the other
11 pesticides.

12 And in the NAPP, where they did a more -- what I would
13 say -- a more scientific adjustment, for those who were heavily
14 exposed greater than 2 days, the risks were significantly
15 elevated.

16 So that's why you do the adjustment, to answer that
17 question. It's a good question. You would wonder about that.

18 **THE COURT:** And then the other -- another thing
19 that -- that has been mentioned in the briefs that I think may
20 be related to this issue is the fact that farmers had an
21 elevated incidence of non-Hodgkin's lymphoma before glyphosate
22 ever came on the scene.

23 **THE WITNESS:** Yes.

24 **THE COURT:** But I assume that, for purposes of these
25 case-controlled studies, that is not a problem. Like, that

1 doesn't -- that doesn't infect the case-control studies,
2 because you're looking at farmers -- farmers with non-Hodgkin's
3 lymphoma, and comparing them to farmers without non-Hodgkin's
4 lymphoma. So all of the other -- presumably, all of the other
5 exposures are similar.

6 Did that make sense -- what I said?

7 **THE WITNESS:** Yeah, it did. It did. And --

8 **THE COURT:** Please feel free to say "No" if it --

9 **THE WITNESS:** No, it did. It did make sense.

10 Now I lost my train of thought here.

11 So we've known for a long time that farmers have an
12 increase in non-Hodgkin's lymphoma. And that's what prompted
13 these studies of pesticides. If you were to say, *Well, why did*
14 *they* --

15 And I did the same thing when I moved to Nebraska. And
16 people told me, *We have a lot of non-Hodgkin's lymphoma here.*
17 And so that was how the Nebraska Study came about; but you
18 know, glyphosate isn't the only thing that causes non-Hodgkin's
19 lymphoma. We know other pesticides do: 2,4-D, malathion. So
20 those pesticides were being used prior to when glyphosate came
21 on the market. So I think it could have been the other
22 pesticides, or it could have been other exposures that the
23 farmers had. We don't really know; but probably it was the
24 other pesticides that had been around for longer, like 2,4-D,
25 2,4,5-T.

1 **THE COURT:** But it doesn't matter, because in the
2 case-control studies we are already studying a population.
3 Whether they're the cases or the controls, we're already
4 studying a population that has a higher incidence of
5 non-Hodgkin's lymphoma.

6 **THE WITNESS:** Well, in a case-controlled study, you
7 have farmers and non-farmers. So you have a whole variety of
8 different people. You take all of the --

9 So in Nebraska.

10 **THE COURT:** So your study was not limited to farmers?

11 **THE WITNESS:** No. And we even studied women in our
12 study. Okay? Farm wives and women in our study, which --

13 Don't laugh.

14 It was dramatic, because none of the other studies studied
15 women.

16 **THE COURT:** At the time it seemed very forward
17 looking.

18 **MS. FORGIE:** Because there are so many studies.

19 **THE WITNESS:** Yeah. It was my idea. I had to argue
20 with Aaron Blair.

21 Anyway, we -- we're interrupting you here.

22 **MR. GRIFFIS:** No, you're not. I'm done. Thank you
23 very much, sir.

24 **THE WITNESS:** Thank you.

25 **MR. GRIFFIS:** Thank you, Your Honor.

1 **THE COURT:** Are you going to have any redirect?

2 **MS. FORGIE:** On behalf of all of the women, I would
3 like a break.

4 **THE COURT:** Feel free to think about whether you want
5 redirect. We'll resume at quarter after.

6 (Recess taken from 2:06 p.m. until 2:20 p.m.)

7 **MS. WAGSTAFF:** Apologies, Your Honor.

8 **THE COURT:** No problem.

9 **MS. FORGIE:** I guess he didn't. No. I'm sorry, Your
10 Honor, for being late. And we have no further questions.

11 **THE COURT:** Okay.

12 **MS. FORGIE:** But thank you.

13 **THE COURT:** Thank you.

14 All right. Who's next?

15 **MR. MILLER:** Your Honor, we call Dr. Alfred Neugut to
16 the stand.

17 **THE COURT:** About time.

18 **THE WITNESS:** No pressure.

19 **MR. MILLER:** We have one second to switch our
20 PowerPoint. Thank you, sir.

21 **THE COURT:** I'll ask. How are things in New Jersey?
22 Still in New Jersey?

23 **THE WITNESS:** Thank you, Your Honor. Snowing, I
24 think.

25 **MR. MILLER:** I poured those.

1 **THE WITNESS:** Oh, okay. Sorry. California water.

2 **THE CLERK:** Do we have new binders for the Judges?

3 **MR. TRAVERS:** I just have copies.

4 **MR. LASKER:** Do you have a cross binder?

5 **MR. MILLER:** Yes. Yes, we do.

6 **MS. GREENWALD:** Sure.

7 (Whereupon a document was tendered to the Court.)

8 **THE CLERK:** Ready? Mr. Miller, you ready?

9 **MR. MILLER:** I'm not sure. All set?

10 **MR. ESFANDIARY:** Sorry.

11 **THE CLERK:** Sir, can you please stand and raise your
12 hand are hand?

13 **ALFRED I. NEUGUT,**

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

16 **THE WITNESS:** I do.

17 **THE CLERK:** Thank you. Please be seated.

18 **MR. MILLER:** Thank you, Your Honor.

19 **THE CLERK:** And for the record, please state your
20 first and last name, and spell both of them.

21 **THE WITNESS:** A-l-f-r-e-d I. N-e-u-g-u-t.

22 **DIRECT EXAMINATION**

23 **BY MR. MILLER**

24 **Q.** All right. Dr. Neugut, good afternoon. Your name has
25 been mentioned a few times here, and we'd like to now get to

1 work. You've prepared a PowerPoint. You started with a map of
2 United States in some fashion. What is this about? And then
3 we can move on to your credentials?

4 **A.** It's just letting you know how I feel about coming out
5 here. Thank you. But --

6 **Q.** All right, sir. All right. Let's go on, then. You are
7 from the East Coast. And you are from Columbia. Right, sir?

8 **A.** That's right. So --

9 **Q.** And I want to go over your credentials. Please articulate
10 them in summary fashion. I'll have some follow-up.

11 **A.** So I'm a medical oncologist and cancer epidemiologist. I
12 was Co-Principal Investigator of the Long Island Breast Cancer
13 Study, which was a study of environmental risk factors for
14 breast cancer.

15 **Q.** Not too fast.

16 **A.** I'm Past President of the American Society for Preventive
17 Oncology, which is the leading society for the study of cancer
18 epidemiology in the United States. I was also the Chair for
19 the Veterans Administration of the committee that evaluated
20 compensation for Vietnam veterans who developed cancer
21 following exposure to Agent Orange in Vietnam. And I'm the
22 author of the chapter on cancer epidemiology for the textbook
23 that's used by fellows in medical oncology. I write or I was
24 on the committee for some years who wrote the questions on
25 cancer epidemiology for the boards for the fellows in medical

1 oncology.

2 Q. All right, Doctor. I have a few follow-up. And you have
3 a Ph.D.?

4 A. Yes.

5 Q. And that's in what?

6 A. Chemical carcinogenesis.

7 Q. Okay. And then you have -- you're a medical doctor.
8 Right?

9 A. Yes.

10 Q. And you're an oncologist; I believe the first one we have
11 here in this case?

12 A. Yes.

13 Q. That means -- are you board certified in oncology cancer
14 medicine?

15 A. Yes.

16 Q. Do you actually treat patients --

17 A. Yes.

18 Q. -- in cancer?

19 Do you have clinic next Tuesday?

20 A. Yes.

21 Q. Yes. When you say you've written 500 papers, they're in
22 peer-reviewed literature?

23 A. Yes.

24 Q. And they're on the causes of cancer?

25 A. Many of them are.

1 Q. Yes, sir. Okay. Now, you wrote an article last year, I
2 believe, on this Long Island Breast Cancer Study. Was it one
3 of the most downloaded articles of the year last year by other
4 physicians?

5 A. Yes.

6 Q. Okay. You were the Chair of the Veterans Administration
7 Committee to Evaluate Compensation for Vietnam Veterans. Who
8 appointed you to that, sir?

9 A. Institute of Medicine.

10 Q. Okay. Now we're going to talk a lot about medical
11 articles and the peer-reviewed literature. Were you a peer
12 reviewer, or are you a peer reviewer?

13 A. Yes.

14 Q. Just briefly tell us what that means.

15 A. I review articles for peer review for journals.

16 Q. Okay. And do you -- are you an editor of journals?

17 A. I have been.

18 Q. Okay. And you say you've written. You're author of a
19 chapter. And have you -- are you hired by countries to help
20 them set up their cancer-prevention systems?

21 A. I assisted a couple of countries in trying to set up.
22 They're mostly in southern Africa.

23 Q. Okay. Let's go to the next slide, please. What did we
24 ask you to do when we asked you to look at this case,
25 Dr. Neugut?

1 A. You asked me to determine whether there was a causal
2 association between glyphosate exposure in non-Hodgkin's
3 lymphoma.

4 Q. Before I get to the next bullet, did you use all of the
5 scientific intellectual rigor that you use in your normal
6 practice at Columbia School of Medicine in analyzing this?

7 A. Yes.

8 Q. And did you use the same principles that you would use in
9 teaching your graduate students?

10 A. Yes.

11 Q. And I jumped the point there. Do you teach graduate
12 students in epidemiology?

13 A. Yes.

14 Q. And so you're an epidemiologist, as well as a cancer
15 doctor?

16 A. Yes.

17 Q. Okay. So you've got a formal degree in epidemiology, as
18 well?

19 A. Yes.

20 Q. Okay. All right. And you've reviewed a lot of stuff in
21 order to come to your conclusions here today. Is that fair?

22 A. Yes, yes.

23 Q. Both positive and negative stuff? Is that fair?

24 A. Yes.

25 Q. Okay. And I see your next bullet point. Explain to us

1 the significance of that point, please.

2 **A.** I've been asked in depositions or asked about hazard
3 assessment or risk assessment, but I don't think those are the
4 terms that are relevant in this context.

5 The question that's addressed is whether there's a causal
6 association between glyphosate and cancer. And the agencies
7 that are responsible for these assessments are different from
8 those that are responsible for risk assessment and hazard
9 assessment.

10 **Q.** How do epidemiologist get to the truth of cause?

11 **A.** With great difficulty.

12 **Q.** Yeah. All right. Let's go to your next slide, please.

13 **THE COURT:** One question about that last slide.

14 I get why hazardous assessment is not helpful.

15 **THE WITNESS:** Mm-hm.

16 **THE COURT:** Can you speak in a little more detail
17 about why risk assessment in this context is not helpful?

18 **THE WITNESS:** I'll tell you the truth. I'm not
19 exactly sure what people mean by "risk assessment." To me, it
20 means the same thing.

21 Risk assessment means asking: What's the relative risk?

22 That's the same thing as asking if it's a cause, or it's
23 part of assessing whether it's a cause.

24 But I mean if the things like what the EPA does, in terms
25 of trying to tell us whether a chemical can be put in a

1 drinking water, or how high a level can be put in there --
2 that's independent of the question -- well, not independent,
3 but it's related to the question of whether a chemical causes
4 cancer or not.

5 A chemical can be thought to be dangerous or harmful, and
6 we should be careful of it, whether or not we are aware of --
7 sometimes -- of whether it causes anything. You know. Stuff
8 in our water. You know. We don't know kind what it's about.
9 We know what causes cancer. Certainly, we know, and are
10 careful about it; but the two things are related, but --

11 **THE COURT:** But if I'm asking about -- if I'm --
12 well, let me give you my perhaps incorrect understanding of the
13 term "risk assessment."

14 **THE WITNESS:** Mm-hm.

15 **THE COURT:** I assume that has hazard -- and this is
16 from -- by the way, I should say, this is from reading the IARC
17 materials, primarily.

18 **THE WITNESS:** Mm-hm.

19 **THE COURT:** Okay. And the IARC preamble talks about
20 or distinguishes between hazard assessment and risk assessment.
21 And it describes hazard assessment, essentially, as assessing
22 whether an agent is capable of causing cancer in the abstract,
23 without regard to how much exposure we're talking about.
24 Right?

25 So the IARC may say -- the IARC Working Group may say that

1 something is a carcinogen, known carcinogen or a probable
2 carcinogen or a possible carcinogen, even if nobody in the
3 world today is being exposed to enough of the substance to
4 cause cancer -- right? -- but it's capable of causing cancer?
5 That's how I understand the term "hazard assessment," as used
6 by the IARC is that --

7 **THE WITNESS:** I just know that they talk about things
8 which are so *de minimis*, that nobody on earth -- or it's so
9 rare, or whatever, literally being exposed or --

10 **THE COURT:** There was a sentence in the preamble that
11 says, even if people are not being exposed to enough of it
12 today --

13 **THE WITNESS:** Uh-huh.

14 **THE COURT:** -- to cause cancer --

15 **THE WITNESS:** Uh-huh.

16 **THE COURT:** -- we are still assessing it as a possible
17 or a probable or a known carcinogen --

18 **THE WITNESS:** Uh-huh.

19 **THE COURT:** -- because you never know --

20 **THE WITNESS:** In the future.

21 **THE COURT:** -- when in the future people's exposure
22 could --

23 **THE WITNESS:** Also works the other way around here.
24 Things that they've described as carcinogens which we've
25 outlawed, so that now there is very little or no exposure to

1 them, hopefully -- or maybe in foreign countries there is,
2 where it's not limited, but no longer in the U.S. DDT, for
3 example, or things like that.

4 **THE COURT:** So anyway, that's how I understand --
5 that's how I'm using the term "hazard assessment."

6 **THE WITNESS:** Uh-huh.

7 **THE COURT:** And I'm trying to understand -- get a
8 better understanding of the term risk assessment. I sort of
9 assumed that when people talk about risk assessment, they were
10 assessing the risk that somebody might get cancer --

11 **THE WITNESS:** Mm-hm.

12 **THE COURT:** -- from the certain exposure that they
13 are experiencing.

14 **THE WITNESS:** Mm-hm.

15 **THE COURT:** Is that an appropriate way to think
16 about --

17 **THE WITNESS:** Yes, I think that's fair, but I think
18 what IARC fundamentally does, and what I'm talking about here,
19 is whether -- and I think what you're dealing with here today,
20 I believe, is really just the fundamental question whether, a
21 priori, the chemical can cause cancer under, I'll say, not
22 theoretical, but realistic circumstances, in the real world,
23 you know, not *de minimis* or not -- not as if we were each being
24 treated like a laboratory animal and being given super, super
25 high levels, but like we're exposed under normative

1 circumstances.

2 **THE COURT:** Okay.

3 **THE WITNESS:** That's what I would consider assessing
4 whether something causes a cancer, and that represents the
5 focus of my approach -- of my thoughts today, and how I've --
6 and I think that's what IARC -- and that's specifically what I
7 think IARC is intended to evaluate, as opposed to hazard
8 assessment and risk assessment, although those may be very
9 closely linked and part of their -- play a role in what they
10 do, as well.

11 **THE COURT:** Okay.

12 **MR. MILLER:** With the Court's permission, let's go
13 off of your PowerPoint for one second, and I apologize, I want
14 to look at Exhibit 149, if we could pull that up, please.

15 Is that something we can do, or I can use the overhead.

16 **MR. TRAVERS:** I believe the Elmo.

17 **MR. MILLER:** Elmo, with the Court's permission.

18 **MR. LASKER:** Do you have --

19 **MR. MILLER:** I do, Exhibit 149 (indicating).

20 **MR. LASKER:** -- a copy for me?

21 **MR. TRAVERS:** We're getting some.

22 **MR. LASKER:** Okay, thank you.

23 **MR. WISNER:** It's C.M.E.

24 (Discussion off the record.)

25 **MR. MILLER:** All right. Can we turn this Elmo on

1 then?

2 **THE CLERK:** It's on the side, there. I can't reach
3 it, but it's on the side.

4 **MR. MILLER:** Here? Thank you. I see, thank you.

5 **Q.** And this is -- I think this because I want to address the
6 Court's question, here.

7 These are briefing notes for the IARC Scientific and
8 Governing Council members prepared by the IARC directors in
9 January 2018, and I want to ask you, Dr. Neugut, about page 8
10 of this exhibit, and it says -- is that readable around there?
11 Help me out?

12 **THE COURT:** Right now there's a -- okay.

13 **MR. MILLER:** There it is, great. Thank you.

14 **Q.** It says, "Monograph's evaluations take account of," quote,
15 "'real world exposures,' by evaluation of epidemiological
16 studies."

17 Is that something that you agree with, Dr. Neugut?

18 **A.** Yes, that's what precisely what I said before. It deals
19 with what would be normal exposures under normal circumstances
20 of -- in the real world.

21 **Q.** I mean, that's what epidemiology does, right, Dr. Neugut?

22 **A.** Epidemiology does that's what IARC does.

23 **Q.** Sure. It says here, a charge was leveled at the
24 monographs is that, "evaluations are divorced from the 'real
25 world,'" end quotes, that is, are made without taking an

1 account of realistic human exposures.

2 Does IARC in these bullet points reject that charge?

3 **A.** That's what this letter says, that the -- that they do
4 deal with real world exposures, and so they're defending
5 themselves against that charge.

6 **Q.** Yes, sir. All right. Thank you.

7 Okay, let's go back now, if I could.... Excuse me, I do
8 want to ask about the second-to-last bullet point in that
9 section. Quote,

10 "In addition, when considering
11 scientific evidence of carcinogenicity,
12 including biological mechanisms, the
13 working group placed special emphasis on
14 whether the observations are relevant to
15 humans."

16 Has that been your experience, as you observed IARC, over
17 the years?

18 **A.** To be honest with you, I'm not exactly sure what the
19 sentence means.

20 **Q.** Okay.

21 **A.** I mean, I assume it's, of course, relevant to humans.
22 I don't know what else it could possibly be relevant to.

23 **Q.** Sure, okay. Well, let's go back, and if we can switch
24 back to the PowerPoint.

25 Cause, just real quickly, what is cause in epidemiology?

1 A. Cause is anything which increases the probability that the
2 outcome will occur.

3 Q. All right, and let me cut to the chase, and we'll come
4 back.

5 Did you develop an opinion whether glyphosate and
6 glyphosate-based formulas caused non-Hodgkin's lymphoma?

7 A. Yes.

8 Q. What's your opinion?

9 A. The answer is yes.

10 Q. Okay. So let's go to your -- your next slide, and explain
11 the significance of this, if you would. This is, I believe,
12 the IARC slide. Yeah, okay.

13 Explain to us the significance of this, if you would,
14 Dr. Neugut, this IARC slide.

15 (Whereupon a document was tendered to the Court.).

16 **BY MR. MILLER**

17 Q. Is it back up on your screen? Okay.

18 A. So when I was asked to evaluate whether cancer -- whether
19 glyphosate was a cancer-causing agent, the first source
20 I always go to in these regards is to IARC, because it is the
21 premier source among cancer epidemiologists for what causes
22 cancer, and it's regularly relied upon by experts in the field
23 of cancer.

24 It's really almost the only source I know that cancer
25 epidemiologists recognize for this option, and it's a

1 universally recognized as a source.

2 If you look at the next slide, just as an example, this
3 is -- and I could go to a hundred websites, but I chose just
4 one at random, the American Cancer Society, and their listing
5 for their lay people who go to the American Cancer Society
6 website for information on cancer, and they're giving
7 information about what causes cancer. People want to know why
8 did I get my cancer, my colon cancer, my gastric cancer,
9 whatever. Here they tell you, here's what the causes of cancer
10 are, and the source they go to, as does everybody else in
11 cancer epidemiology, is IARC.

12 Next slide.

13 And likewise, further down the website in the American
14 Cancer Society, they go on to explain what IARC is, so that the
15 readers on their website could know that this is the source.
16 They describe IARC so that their readers know that this is,
17 again, reconfirming the primacy of IARC as the source of
18 information on what is a cancer-causing agent.

19 And I do this because all of this begins with the 2015
20 monograph from IARC that alluded to glyphosate as a
21 cancer-causing agent.

22 IARC, as it says, has looked at 900 agents, and described
23 them in one fashion or another, as carcinogenic or not.

24 Next slide.

25 And even on the American Cancer Society web page, it

1 describes the Group 2A carcinogens that are probably
2 carcinogenic; and it includes glyphosate on the Web page on the
3 ACS website.

4 I would point out to the Court, just so we understand
5 clearly, we don't have the same terminology as the Court does,
6 someone else pointed out earlier. When IARC uses the
7 terminology "probably carcinogenic," in my estimation, that
8 refers to a probability on the order of probably in the range
9 of 70 to 90 percent, as what they would estimate the
10 probability of glyphosate being a carcinogen is. We're not
11 talking "probably" as 51 percent. They're talking something in
12 the range of 70 to 90 percent. Their terminology of likely --
13 of -- not -- or their next level of is too big, is down
14 probably, I would say, in the 45 to 70 percent range, or
15 something like that, depending on the exact agent.

16 So there are 2A and 1 classifications are very powerful
17 from a scientific and epidemiology point of view. These are
18 very powerful statements.

19 We don't talk, like, you know -- we qualify everything we
20 say. We never make a straightforward statement in our lives
21 without setting maybe, possibly, could be, studies indicate
22 that. That's the way we talk, that's the way we write.

23 So when they say, "probably carcinogenic," that is a very
24 powerful terminology, in the cancer epidemiology world.

25 Next slide.

1 So I wanted to go on and indicate to the Court, having
2 said that, again, I'm the third speaker, you know, and so
3 everything that's been said to now, you know, I don't want to
4 waste the Court's time and repeat everything that everybody
5 else has said. If I say anything that you've heard already or
6 are bored with, you know, feel free to say, yeah, let's move
7 along.

8 But I wanted to make a point about specificity. The truth
9 is from the Bradford-Hill criteria, specificity is usually
10 ignored as one of the five criteria. It rarely comes up, as
11 one of the five -- there are five criteria that usually used.

12 Specificity is one of the other two or three, and it's
13 usually ignored. It happens to be relevant in the context of
14 glyphosate, in NHL. Because it happens to be a constant,
15 consistent, specific association of glyphosate and NHL, a rare
16 tumor, we could theoretically find an association between
17 glyphosate, and probably there are a hundred or more kinds of
18 cancer, and there have been dozens of studies looking at
19 glyphosate and various cancers.

20 And every time you look, what comes up? Glyphosate and
21 NHL. You don't see glyphosate and prostate cancer, you don't
22 see glyphosate and colon cancer, you don't see glyphosate and
23 cancer of the left earlobe. What do you see? You see
24 glyphosate and NHL. Okay, there are a half a dozen studies or
25 so, but all of them are glyphosate and NHL. That's

1 specificity.

2 Next slide.

3 Why in a case-controlled studies does glyphosate
4 consistently turn up with NHL? Those are precisely the logical
5 underpinnings of the Bradford-Hill criteria, and that's a very
6 powerful argument and logical argument for a causal link
7 between glyphosate and NHL.

8 Next slide.

9 Recall bias. I bring this up. I was going to mention it
10 anyway. I had a slide on it before even the Court brought it
11 up yesterday, as a specific question, but I expanded the slide
12 because the Court yesterday raised it as a specific question,
13 so I'll put in my own two cents on what I think about recall
14 bias, and in the context of our discussions the last day, but
15 first of all, recall bias presupposes knowledge or suspicion of
16 an association.

17 You ask some farmer, you know, who has NHL, what have you
18 been exposed to? You give him an hour-long questionnaire with,
19 what have you eaten, do you smoke, how often do you go to the
20 bathroom, you know, how often do you play golf, you know, do
21 you do physical activity? Are you fat? And then you throw in
22 a whole list of herbicides.

23 Why on earth is he going to say, out of everything,
24 glyphosate, I've been exposed to glyphosate? Where in the
25 world did he see it? Did he see that call 1(800) LAWYER on

1 television in 1980s or in 1990s?

2 **THE COURT:** Maybe.

3 **THE WITNESS:** Who knows? But the point is, recall
4 bias presupposes knowledge or suspicion and association.
5 Otherwise, why on earth would he think of it.

6 An example is tobacco and lung cancer. You couldn't
7 possibly do a study today of tobacco and lung cancer. If
8 someone had lung cancer, they would say they smoked even if
9 they didn't smoke. They would remember when they were 18 years
10 old, they went behind the woodshed and they would tell you they
11 were a smoker. You know with broccoli and lung cancer, if, as
12 someone who has lung cancer -- if you're doing a study on a
13 dietary and ask them, "Do you eat broccoli?" they would say
14 "Yes" not because they don't have any reason to suspect or to
15 have a recall bias with regard to broccoli.

16 So is there any reason, again, for individuals with NHL to
17 suspect glyphosate in the 1990s when they were doing these
18 studies? So that brought up with what Dr. Weisenburger alluded
19 to earlier in his discussion, was there any reason for any
20 reason for individuals with other cancers, stomach cancers,
21 leukemia, brain tumors, non-Hodgkin's lymphoma, to suspect
22 glyphosate in the 1990s? Whatever the reason is that you would
23 have suspected a link, it would -- to me, it's seems like it
24 would have had equal logic for any tumor, or -- and one would
25 have seen the same recall bias manifesting itself in studies of

1 those tumors.

2 Am I okay now?

3 Next slide, please.

4 So you saw a similar slide earlier. Here's, again, just a
5 few examples of other tumors, and you can see the Odds Ratios
6 were all null, for the most part, and there's no -- as I said
7 before in my suggestion of specificity, there's no other tumor
8 that's popped up with regard to glyphosate.

9 So again, where's the recall bias? There is no recall
10 bias. It's all -- and in fact look, at Hodgkin's lymphoma,
11 another lymphoma. How is how does the farmer know whether his
12 tumor has a Reed-Sternberg cell in it or not? You know, he's
13 such a smart guy?

14 You know, so for the tumor with the Reed-Sternberg cell,
15 he says, "No, I wasn't exposed to glyphosate," but for the
16 tumor that doesn't have a Reed-Sternberg cell, he said, "I was
17 exposed to glyphosate." That's ridiculous.

18 So the only conclusion we can go with is: It's the truth
19 that, actually, there is an association between glyphosate and
20 NHL.

21 Next slide.

22 So we can quibble or nitpick over whether each of the
23 case-control studies has a limitation and its association
24 between glyphosate and NHL. Of course, that's what we're doing
25 these two days.

1 **THE COURT:** Slow down a little bit.

2 **THE WITNESS:** I'm sorry.

3 **BY MR. MILLER:**

4 **Q.** And let me interrupt you for one second. How many years
5 have you been a epidemiologist? How many years? Thirty-eight?

6 **A.** No. From '81. From '81.

7 **Q.** Okay. From '81. Since 1981, have you ever seen a perfect
8 study?

9 **A.** No.

10 **Q.** Okay.

11 **A.** None of them in my life.

12 **Q.** So what's the point here? The active association arises
13 consistently. What's your point, Dr. Neugut?

14 **A.** So we can quibble. We can nitpick over each of the
15 case-control studies. Each one is going to have some
16 limitations. This is what we call "consistency," that this one
17 has a certain limitation, that one has a certain limitation,
18 but we mix them all together, and together they cancel each
19 other's limitations out, yet overall, the association still
20 arises consistently over the mix of studies.

21 Next slide.

22 So here was a good question yesterday, by Your Honor:

23 Why doesn't every study adjust for all of the herbicides
24 and pesticides, which is, I think, what was addressed a little
25 bit earlier today as well.

1 So this reminds me that some years ago, a reporter -- one
2 of my interests is colonoscopy screening, so some years ago a
3 reporter asked me, or told me, that only 50 percent of
4 New Yorkers over the age of 50 have had a colonoscopy. What
5 are we going to do about this crisis? And my response to him
6 was, 50 percent of New Yorkers over the age of 50 have had a
7 colonoscopy? That's extraordinary. Did they all laugh at
8 themselves, too? Whatever. So that's terrific.

9 So on a certain, level it's extraordinary that two or
10 three out of a half dozen case-control studies have adjusted
11 for all of the herbicides.

12 It's extraordinarily difficult to collect high-quality
13 information on a huge number of herbicides the way you've heard
14 it described already today.

15 Doing these questionnaires is no small potatoes. It is
16 very labor-intensive and very difficult. You heard
17 Dr. Weisenburger discuss it earlier in some depth, but it is
18 very difficult to do that. Not every study can do it. All of
19 the studies he described were NCI studies specifically
20 conducted by NCI. Only the government can pay for this, you
21 know, and -- so most of the time, it cannot be done by every --
22 every, you know, investigator to do it. So the fact that
23 almost half the studies actually did it is unusual.

24 So I point out that that is not -- so the fact that most
25 of them didn't do it is really high, high, in my opinion very

1 high, very more than usual.

2 Try doing one of these questionnaires. They take an hour
3 and a half to two hours if you sit down and do it yourself, and
4 it really is an effort.

5 It's almost universal for any exposure and outcome that's
6 studied in epidemiology and that multiple studies will miss
7 selected covariates or confounders, but so long as some studies
8 have them, that's good enough for us.

9 The last litigation I was involved in -- and I have not
10 been involved in too many tort cases of this type -- was the
11 Actos and bladder cancer litigation. Actos toes is a drug for
12 diabetes, and bladder cancer is related to tobacco. Tobacco is
13 a significant risk factor for it. So tobacco is a major
14 confounder. And at least a third to 40 percent of the studies
15 did not have tobacco as a covariate in the studies. Tobacco is
16 like the easiest of covariates of -- to collect.

17 Do you smoke?

18 And yet half of the studies -- well, not half. Let's just
19 say about a third to 40 percent didn't even have tobacco,
20 didn't control for the tobacco in the analysis, and that's an
21 easy one to collect.

22 So it's very common to be missing a confounder in a
23 substantial number of studies when you're looking at a risk
24 factor and an outcome.

25 So I hope that answers that question for you.

1 Next slide.

2 **Q.** Before we go to the Bradford-Hill criteria, which is your
3 next slide, we've talked about forest plots with the other
4 experts, and I don't want to beat the horse to death, if you
5 will, but I want to hear your explanation in regards
6 specificity and forest plots.

7 With the Court's permission, we'll have it on the screen
8 as well, as a blow-up, if that's acceptable, Your Honor.

9 **THE COURT:** Sure.

10 **MR. MILLER:** It's upside-down, Mr. Wisner. That's
11 now your last official job in this courtroom.

12 **THE WITNESS:** Next slide.

13 **BY MR. MILLER**

14 **Q.** Explain to us what this is, please. This is -- yeah.

15 **A.** This is a forest plot from a meta-analysis. So I'm sure
16 the Court has seen it before, and it's basically a compilation
17 of the Risk Ratios from the different case-control studies; and
18 actually, also includes the AHS follow-up from 2005. And this
19 was published, and it gives a summary Risk Ratio of 1.3. So
20 generally speaking, I believe, based on this and other
21 meta-analyses, that the summary Risk Ratios estimated in the
22 1.3, to 1.5 range.

23 **Q.** Okay, but Doctor -- all right. I want to ask now, Doctor,
24 would that line being 1, we all know now, what are the odds of
25 every study being to the right of that line, if these were

1 spurious or chance findings?

2 **A.** So it would be extremely unusual for -- well, for all of
3 the studies to be to the right -- to be to the right, and
4 that's what is part of the argument for -- for the causal
5 association.

6 That -- again, we're not sort of focusing totally, as
7 you've heard for several days, I forget, from the testimony
8 yesterday, and then this morning or earlier today, that the
9 95 percent confidence interval, per se, is not the be-all and
10 end-all; but the fact that all of the risk estimates are to the
11 right of 1 -- I mean, if you did a random analysis for all of
12 the studies, you'd think that half of them would be to the
13 left, and half of them would be to the right; but the fact that
14 they're all to the right is a powerful argument that -- that,
15 as I said earlier, that there is a specificity and this
16 consistency in the fact that there is a statistical association
17 between them.

18 Most of the studies don't have enough statistical power,
19 and have other problems. And again, because the risk estimate
20 is so small and so modest, it is difficult to -- for each of
21 the studies to be statistically significant on their own,
22 and -- but the fact that they're all to the right is a very
23 powerful argument in the causal, I believe -- in the causal
24 argument.

25 **Q.** Okay. And we're going to your next slide now.

1 A. Next slide.

2 Q. So Bradford Hill and his criteria for causality. Explain
3 it again, briefly, and how you employed it.

4 A. Again, I don't know if the Court wants to hear --

5 **THE COURT:** Very briefly.

6 **THE WITNESS:** Very briefly, there is a temporality.

7 There is consistency. There is dose-response, biological
8 plausibility, strength of association, and specificity.

9 As I said earlier, these are the criteria that have been
10 used by -- in chronic disease epidemiology for establishing
11 causal associations. And I think all of the these are
12 fulfilled in the current instance between glyphosate and -- and
13 NHL, and thus that makes an argument for causal association.

14 **JUDGE PETROU:** Is this your rating system, to the
15 right of each one?

16 **THE WITNESS:** That's my rating system on them. Yes.

17 **JUDGE PETROU:** On what scale? Is 5 the top?

18 **THE WITNESS:** Five is the top, yes. So temporality
19 is just, you know, as everyone says, you always have to have
20 temporality; but sometimes there can be ambiguity in
21 temporality. You can be uncertain whether a cause is always --
22 whether a putative exposure is always before the cause in the
23 studies that you see, but I think in the glyphosate and NHL
24 question, all of the studies are very straightforward in terms
25 of temporality. That's why you give it a 5-plus.

1 Dose-response, again, we haven't seen -- there is
2 dose-response in a couple of studies, as you've seen, but it's
3 not very powerful. Really the Eriksson Study, to me, is the
4 one that seems most plausible.

5 By the way, to answer the question one of you asked
6 earlier as to how you define where to put the cutpoint for
7 dose-response -- I was listening -- so the normal -- the
8 standard way to do that is to bifurcate, to dichotomize at the
9 median of the control group, usually, and that's what they did
10 in Eriksson.

11 I couldn't see in McDuffie -- that was the one where they
12 did it in two days versus less than two days -- they don't
13 actually say in the methods section -- they don't describe in
14 the methods section how -- how they how they picked the two
15 days as the cutpoint.

16 **JUDGE PETROU:** Typically, it's the median of the
17 control group, is that --

18 **THE WITNESS:** Usually, the control group is used.

19 **JUDGE PETROU:** Okay.

20 **THE WITNESS:** If it's a big number -- that is, if
21 it's a very large study -- then they'll do tertiles or
22 quartiles. So, you know, they'll compare the top quartile to
23 the lowest quartile.

24 But most of these studies are not that large, so
25 bifurcation -- you know, dichotomy -- is big enough. You know.

1 Less than 10 days versus more than 10 days.

2 But if there were a lot of them, if there was a much
3 bigger study, you might have had less than 10 days; 10 days to
4 50 days; 50 -- greater than 50 days. You know. You might have
5 had more categories; but again, these studies -- the study
6 wasn't -- I don't think it was large enough to get that
7 elaborate.

8 **JUDGE PETROU:** Mm-hm.

9 **THE WITNESS:** So that was done there.

10 And the strength of association, 1.3 to 1.5, while I think
11 that's just still in the range of a moderate association, it's
12 not like -- you know, it's not a huge relative risk. So I also
13 did not give it a huge rating here.

14 But again, but still, all of the Bradford-Hill Criteria
15 were -- the -- one, two, three, four -- these top five, without
16 specificity, are the usual five. If you look at most causal
17 associations, they go through the five that are typically used.

18 Without specificity -- specificity is usually not even on
19 the list. I'm just saying that in our particular in our
20 particular case, specificity applies. So I included it.

21 Next slide.

22 **THE COURT:** Could I ask you one more question about
23 the previous slide?

24 **THE WITNESS:** Mm-hm.

25 **THE COURT:** Can we go back to the previous slide?

1 Thanks. So temporality is at 5.

2 **THE WITNESS:** You know, I make it easy. It's not
3 like I have a little metric somewhere and I'm going, you know,
4 like (indicating).

5 **THE COURT:** I understand, and normally, I gather from
6 all of the reports and all of the testimony that temporality is
7 not something one thinks too much about, because it's an easy
8 question to answer.

9 **THE WITNESS:** Mm-hm, exactly.

10 **THE COURT:** But in this case, remembering about the
11 question that I -- that I was asking earlier today --

12 **THE WITNESS:** Mm-hm.

13 **THE COURT:** -- about the De Roos Study and
14 McDuffie Study --

15 **THE WITNESS:** Mm-hm.

16 **THE COURT:** -- which involved data collected from the
17 late '70s and early '80s --

18 **THE WITNESS:** Mm-hm.

19 **THE COURT:** -- and there's this concern that, you
20 know, there may be a fairly long latency period for NHL, as
21 potentially caused by glyphosate --

22 **THE WITNESS:** Mm-hm.

23 **THE COURT:** -- and glyphosate didn't come on to the
24 market until the mid '70s. Does that create a temporality
25 problem with respect to those studies?

1 **THE WITNESS:** If anything, it sort of obviates the
2 temporality issue, because it's clear then that the glyphosate
3 predated the NHL, no?

4 **THE COURT:** Well, but doesn't it raise a concern that
5 the NHL was caused by something that they were exposed to
6 before glyphosate came on the market?

7 **THE WITNESS:** So that's a different question.

8 **THE COURT:** That's not a temporality issue.

9 **THE WITNESS:** That wouldn't, to me, be a temporality
10 question. That would be a different question.

11 **THE COURT:** Where would you put that concern?

12 **THE WITNESS:** So that's a question that I think there
13 was a discussion about latency, period. And I suppose that
14 would then come under biological plausibility, or mechanism of
15 action applies in terms of how glyphosate causes glyphosate
16 causes -- theoretically, how glyphosate causes NHL.

17 **MR. MILLER:** If I could, then, Your Honor, could we
18 switch to the Elmo?

19 **Q.** I want to ask you about the particular issue, if I could.
20 This is from the McDuffie Study, Exhibit 21. Every study has
21 what they call a "Materials and Methods" section. Is that
22 right, Doctor?

23 **A.** Yes.

24 **Q.** And I think they perhaps answered this question in
25 Helen McDuffie's study, with these other scientists, and I want

1 to ask you about this.

2 She states, on the second page, or page 1156, a whole
3 sentence here, "but incident cases among men ages 19 and over
4 with a final diagnosis of STSHD NHL" -- I guess that's
5 non-Hodgkin's lymphoma?

6 **A.** Yeah, um-hum.

7 **Q.** -- "or MM" -- that's multiple myeloma?

8 **A.** Yes, sir.

9 **Q.** -- "diagnosed between September 1, 1991, and
10 December 31st, 1994 will were eligible"?

11 **A.** Correct.

12 **Q.** So that if Roundup[®] came on the market in '74, then that
13 would be 17 years --

14 **A.** Right.

15 **Q.** -- between the time, right?

16 **A.** If they were looking at exposures to glyphosate that went
17 back to 1974, then of course, that would be more than enough
18 exposure for those cases.

19 **Q.** All right. Thank you, sir.

20 **THE COURT:** And did you say that was the
21 McDuffie Study?

22 **MR. MILLER:** Yes, Your Honor. That's Exhibit 21, the
23 McDuffie Study, page 1156, which is the second page.

24 **THE COURT:** Thank you.

25 **MR. MILLER:** Yes, Your Honor.

1 Q. Let's go back to your PowerPoint, then. You were talking
2 about -- you've completed your discussion on the Bradford Hill?

3 A. Sure.

4 Q. Let's move on.

5 A. So the only real change since IARC -- and, as I say, I
6 think IARC did a bang-up job in terms of evaluating various
7 questions that addressed the case-control studies -- was the
8 changes; the recent follow-up studies and its limitations. And
9 the question is how it affects our overall thinking with regard
10 to what's going on. So the question is: How do we all feel
11 about it? I mean, I know how they feel about it, and I know
12 how we feel about it. So -- and how it may alter the
13 conclusions of IARC.

14 Next slide.

15 So first, just to make a general statement, a
16 well-conducted epidemiological study does not typically need
17 imputation. Imputation is used when a major problem develops
18 with data collection in a study, so it implies a problem. That
19 does not mean it's a bad thing to do. It's the right thing to
20 do, but the fact that you had to do imputation already shows
21 that you're dealing with an issue.

22 Next slide.

23 So what's the issue? So it's really the conflation of
24 several problems together, and let me say that I think the
25 AHS Study, as someone else said, is actually an excellent

1 study. It's been very productive, and it's very good for many,
2 many things.

3 We were talking very specifically about the association
4 between glyphosate and NHL, and the problem arises from the
5 conflation of these problems, the first of which is the figure
6 on top, which is the extraordinary increase in the use of
7 glyphosate that took place between -- in the late '90s, and
8 totally changed the exposure level of glyphosate among farmers.

9 If we could have used the 1995, you know -- the farmers in
10 AHS were collected between 1993 and 1997. If everyone kept
11 using glyphosate at the same rate, look, if we asked them how
12 much do you smoke, and you say, I smoke two packs a day, the
13 smoker could have just kept going along using two-pack-a-day of
14 smoking, and there would have been no change in smoking, then
15 there would have been no problem with everything, but the fact
16 that there was a extraordinary change in usage, so the baseline
17 exposure rate of glyphosate exposure became totally untenable
18 as a measure of exposure.

19 This was different, I assume, from any of the other
20 herbicides, and it makes the whole glyphosate assessment
21 necessary, therefore, to require a second interview. So
22 without the second interview, you're screwed up.

23 So that leads to, then, needing a second interview in
24 2005. Then you have the 37 percent loss to follow. Basically,
25 they couldn't interview 37 percent for one reason or another.

1 I mean, those of us to who get phone calls all of the time
2 asking us to do a survey can totally understand this.

3 And then on top of that, there's the modest association in
4 the first place. Again, if the Risk Ratio is 10, then all of
5 these errors wouldn't really matter, because they would all be,
6 ah, you know, they would all wash out in the mix, but because
7 we're dealing with a Risk Ratio in the 1.3, 1.4, 1.5 range,
8 that's very delicate, and so any -- any errors attenuate
9 towards the null, as you keep hearing over and over and over
10 again, and therefore, these -- the compilation of all of these
11 errors over and over again take you way down to the null.

12 Then there was the 10 percent initial misclassification in
13 the error in the first place, which attenuates towards the
14 null, and -- and as was shown in one of the papers, when they
15 did imputation in the first place, they had a 17 percent
16 imputation error. So we have an imputation. Again, imputation
17 is necessary, but imputation has an error to it, so imputation
18 doesn't work that great. Again --

19 **THE COURT:** What was the 10 percent initial
20 misclassification error?

21 **THE WITNESS:** That's when they asked the people in
22 1995 how much glyphosate they were using. It was estimated
23 that the error in terms of their estimate of use of glyphosate
24 was about -- I remember 10, or might have been 11 percent,
25 which is not a bad error, as epidemiologic analyses go.

1 **THE COURT:** You mean whether they used it at all or
2 not?

3 **THE WITNESS:** Yeah, mm-hm.

4 **THE COURT:** Okay.

5 **THE WITNESS:** But with a Risk Ratio of 1.3 to 1.5,
6 that's almost enough by itself to wipe out a 1.3 to 1.5 Risk
7 Ratio.

8 Think of it for a moment. If I give you an example, if I
9 did the dietary food frequency questionnaire with you and I
10 asked you how many broccoli do you eat, right? You haven't got
11 a clue of how much broccoli you eat. How many times a week do
12 you eat broccoli? You're going to be wrong, whatever you say.
13 I'm going to be wrong, whatever you say. Everybody in this
14 room is going to be wrong with whatever they say.

15 Somehow, it all works out when you do it
16 epidemiologically, but the point is the error rates work out so
17 that they all attenuate towards the null, and it's
18 conservative, and so these misclassification errors all
19 attenuate towards the null. So you can imagine asking someone
20 how much glyphosate they use, a 10 percent error is actually a
21 fairly minor error, if you think about it. It's not so bad.

22 But it's big enough, for a -- for a modest association,
23 for a modest association, and that's why this conflation --
24 it's specifically with glyphosate and NHL that we have the
25 problem, because -- because we have this modest Risk Ratio, and

1 then we have this extraordinary change over the next 10 years
2 which, just on top of it, makes it totally impossible.

3 And then when they did imputation, they themselves showed
4 it as 17 percent error in how the imputation measured the --
5 the estimate of the glyphosate usage, and that's --

6 **THE COURT:** Could you explain that a little more?

7 **THE WITNESS:** Yeah. They took -- so imputation is
8 that they take all of the knowledge that they have about the
9 cohort -- about the, let's say, the 60 percent, the 62 percent,
10 they know the age, race, sex, many, many things about them, and
11 they know -- they have all answered second questions, taking
12 the 60 percent who answered a second questionnaire, and from
13 that, they took a 20 percent random sample of the people who
14 did answer the second questionnaire. So they know the answer
15 to how much glyphosate they used.

16 Twenty percent of the people who took -- they took a
17 20 percent sample of the 60 percent, who -- so they know their
18 use, on the second questionnaire, of the glyphosate.

19 So then they used imputation to see how well imputation
20 estimated their answers to the second questionnaire, and when
21 they did that, the answer was off by 17 percent, 16 percent or
22 17 percent. I don't want to get picky. So even the imputation
23 mis-measured it by 17 percent.

24 **THE COURT:** So you're saying they asked the questions
25 on the second questionnaire one time.

1 **THE WITNESS:** Mm-hm.

2 **THE COURT:** And then they asked 20 percent of those
3 respondents the same question on the questionnaire again.

4 **THE WITNESS:** Uh-uh, no, no, no, no. They -- so
5 there were 30,000 people -- they had 50,000 people to start
6 with. 30,000 people answered the second questionnaire. So of
7 that 30,000, they took 7,000 people who had answered the
8 questionnaire, and they used imputation to estimate what their
9 answer to the usage of glyphosate was, using the imputation
10 methodology, to see how well they guessed or how well they
11 estimated their use of glyphosate but they knew their use of
12 glyphosate from -- because they'd all answered the
13 questionnaire.

14 I'm not saying it well?

15 **THE COURT:** You're probably saying it well, I'm just
16 not understanding it. What are they comparing?

17 **THE WITNESS:** These people have all answered, so --

18 **THE COURT:** They've answered the question.

19 **THE WITNESS:** They've answered the question.

20 **THE COURT:** And so what is -- what are their answers
21 being compared to?

22 **THE WITNESS:** They're using the imputation
23 methodology to -- so their plan is to use imputation.

24 **THE COURT:** So the methodology that's used for the
25 people who didn't answer the questionnaire is applied to the

1 people who answered, and it's compared to their answers.

2 **THE WITNESS:** To see how well it guesses their
3 answer, where they already know -- where they know the answer,
4 to see if they get the right answer, and when they did that,
5 the answer was off by 17 percent.

6 So again, that's not such a terrible answer for
7 imputation. I'm not criticizing. A 17 percent error for
8 imputation is not a bad guess.

9 Fifty-three percent versus 45 percent, I believe, is what
10 they got, just to give you a sense of the numbers, but in terms
11 of how that will translate later into -- into dealing with a
12 Risk Ratio of 1.3 to 1.5, in terms of, then again, that error
13 rate, how that will affect a Risk Ratio of 1.3 to 1.5 -- again,
14 this is an error on top of an error on top of an error, where
15 each error attenuates to the null.

16 And we haven't even discussed the problem that 20,000
17 people are biased. They -- you know, that's the biggest bias
18 in epidemiology is volunteer bias. Who answered the
19 questionnaire and who didn't answer the questionnaire? And we
20 don't even touch that bias. I didn't even mention it here,
21 because it was so obvious.

22 Next slide.

23 Okay, so those are the problems, and that's why I think
24 the AHS Study is basically not so useful.

25 And just to give you a sense, so you can know, that a

1 37 percent loss to follow-up is, you know, pretty humongous.

2 Here's a another cohort study, happens to be from Harvard,
3 and you can see in the line under the follow-up in this study
4 was 94 percent, just to give you a sense of, studies do do well
5 in terms of follow-up.

6 Next slide.

7 So, okay. So you will ask yourself, as will everyone in
8 the room, if I'm saying that this AHS follow-up study is such a
9 pile of -- you know, is really such -- so -- so bad, how did it
10 get published in *JNCI*, which is such a good journal, as
11 Mr. Lasker is going to tell us at some point I'm sure, and it
12 got published there, peer-reviewed, et cetera, et cetera, and
13 *JNCI* is a good study. I've had 20 papers published there, so
14 by definition, it's a good journal, so -- and it's a perfectly
15 valid question to ask.

16 So -- and I don't know the answer, because peer review is
17 confidential. I can only speculate, based on my knowledge of
18 peer review. Mr. Miller asked me before about my experience
19 with peer review, I do a lot of peer review. So I can give you
20 three possible answers, just for your contemplation.

21 The first -- my first possible answer is what I would
22 call, shit happens. You know? You never know, you know?

23 You got yourself an easy peer reviewer, we all hope for
24 it. I don't know how *Harvard Law Review* works but, you know,
25 but you know you -- ah. You know, you send it in to New

1 England Journal, the gods can smile. Okay, I don't think that
2 happens here.

3 The second possibility is, *JNCI* has been going downhill
4 for the past five years. It used to have an impact factor
5 which was how we measure -- one way in which we measure how
6 good a journal is. It was 18, it's down now to about 12.

7 It got itself a new editor, who actually happens to be a
8 very close friend of mine, has been trying to write itself.
9 Here comes a paper that's going to be very, uh, pre- -- I don't
10 know if prestigious, but get a lot of attention and bring it a
11 lot of notoriety, and so it got itself -- it got peer-reviewed,
12 but got an easier pass in a sense of, the editors wanted it to
13 be published here so it would get some attention to the
14 journal, and indeed, this past -- I got an e-mail this past
15 year that this was one of the top 10 downloaded journals --
16 downloaded papers of 2017. It's, in fact, number three. One
17 of my favorites is on the two.

18 **THE COURT:** Just because Monsanto required every
19 single employee to download it.

20 (Laughter.)

21 **THE WITNESS:** So, but a third possibility is
22 something else, which is, when you do peer review, it takes
23 you, you know, to read the paper it takes you a half hour, 45
24 minutes. It's up to the authors to highlight to you what's
25 good and bad about the paper. So here's the abstract from the

1 paper. Take a look at it, if you have a moment, you read
2 through this thing. Mention the loss to follow-up? Anything
3 about it? Imputation? Not a word.

4 Okay. All right. Let's move on.

5 Next slide.

6 So here's the Limitations section. This is the section
7 I read most carefully when do peer review. I expect the
8 authors to write an honest assessment of the weaknesses in the
9 paper, so I can judge how good and bad -- you know, what the
10 problems are in a paper.

11 This paper actually had two findings in it, one of which
12 related to leukemia, that there was a positive association
13 between A -- glyphosate and AML.

14 So every limitation -- there were three limitations given
15 in this paragraph, all of which relate to their findings with
16 regard to AML to leukemia.

17 There is not a single limitation noted in this paragraph,
18 or in the paragraph before or after, with regard to the loss to
19 follow-up, the use of imputation, any of the things I alluded
20 to in my paragraph.

21 In a sense were they being dishonest, unethical? Who
22 knows? I'm just saying it wasn't as open of a paper in terms
23 of talking about its weaknesses. I'm just giving you a sense
24 of what -- I suspect it was really the earlier point about,
25 shall we say, the trendiness and politics that were involved.

1 Next slide.

2 So we could go through the papers again. But anyway, next
3 slide.

4 **BY MR. MILLER:**

5 **Q.** If you --

6 **THE COURT:** Well, I actually think it, in particular,
7 it would be helpful to go through McDuffie.

8 **THE WITNESS:** Okay.

9 **THE COURT:** And De Roos, 2003.

10 **THE WITNESS:** I didn't mean to --

11 **THE COURT:** No, no, that's okay. And one suggestion
12 I might have is, you know, we could go -- you know, we don't --

13 (Discussion off the record.)

14 **THE COURT:** We don't have a hard stop at 4:00. We
15 can go to 4:15, 4:30 whatever.

16 Why don't we take a little break, and then resume.

17 **MR. MILLER:** Great.

18 **THE COURT:** Why don't we resume --

19 **THE WITNESS:** You want to take a break now

20 Your Honor?

21 **THE COURT:** Yeah, yeah, and resume at 3:30.

22 (Recess taken from 3:20 p.m. to 3:30 p.m.)

23 **MR. MILLER:** Well, let's go back -- I'm sorry. If we
24 could go back to the slides?

25 If I could start, then, Your Honor?

1 **THE COURT:** Please.

2 **MR. MILLER:** All right.

3 **Q.** Even though the McDuffie Study was adjusted for age,
4 province and high-risk exposure, it was not adjusted for other
5 pesticides within the original study, right, sir?

6 **A.** No.

7 **Q.** But is it still a piece of the puzzle that you use in
8 weighing the evidence to come to your conclusion that Roundup®
9 causes?

10 **A.** Correct. We discussed this earlier in terms of, some of
11 the studies did, and others said some of the studies did not.

12 By the way, I wanted to answer -- the judge had asked this
13 morning, or earlier, about the hierarchical regression
14 modeling. So I thought I'd just take one second --

15 **Q.** Please.

16 **A.** -- to answer it. So I don't think it has a direct
17 relevance to much that we're -- that we're talking about, but
18 it's a form of regression analysis where -- so in a logistical
19 regression, or most regression models, we just throw in all of
20 the covariates into one equation, sort of like the kitchen
21 sink, so to speak, and then the computer grinds around for a
22 while and the answers come out, and each covariate gets a Risk
23 Ratio assigned to it, with a 95 percent confidence interval.

24 In hierarchical modeling, the idea is you may want to know
25 how much does a certain class of variable affect the

1 association between the exposure and the outcome.

2 So for example, we could even say with regard to
3 glyphosate, we want to know, how much do the demographic
4 variables affect the association between glyphosate and NHL?

5 So in hierarchical modeling, they would do age, race, sex,
6 socioeconomic status, and do a regression analysis using those
7 covariates first, and then you'll get some kind of overall Risk
8 Ratio, I believe, and it will say how much of the overall -- in
9 the end, the overall association between glyphosate and NHL is
10 due to those covariates as a group.

11 And then you might then put in the -- the herbicides, let
12 say, and then you'd -- might say you might end up saying that
13 the association between the two, that 20 percent of the effect
14 is due to the demographic variables, 80, 70 percent is due to the
15 herbicides, 10 percent is due to something else, you know,
16 unexplained, or idiopathic or something like that.

17 So I don't know that it plays that much of a role in these
18 studies.

19 **THE COURT:** Well, tell me why -- you know, you
20 mentioned that you're not sure how much it matters, for
21 purposes of our discussion.

22 **THE WITNESS:** Um-hum, because we're mostly focused on
23 individual covariates, as a rule; glyphosate in particular.

24 I don't know that the hierarchical modeling is that much
25 of an issue in this.

1 **THE COURT:** Well, maybe it's something, you're going
2 through the studies, and maybe we when we get to De Roos, we
3 can --

4 **THE WITNESS:** Yeah tell me -- if it comes up --

5 **THE COURT:** -- we can talk a little bit more about
6 that.

7 **THE WITNESS:** If it comes up, by all means.

8 **THE COURT:** And so there was a McDuffie slide up just
9 a second ago.

10 **THE WITNESS:** Mm-hm.

11 **THE COURT:** And so when it says on the slide,
12 adjusted for age, province, high-risk exposure, what is
13 high-risk exposure? That's just greater exposure to
14 glyphosate?

15 **THE WITNESS:** Mm-hm. Uh, I don't recall what I mean
16 by that, so I'm going to have to --

17 **BY MR. MILLER**

18 **Q.** Would that be found in Table 1, sir?

19 **A.** In Table 1? I think it's just talking about folk that are
20 high risk, things like medical conditions and things of that
21 sort, but I'm not a hundred percent sure.

22 **THE COURT:** Okay.

23 **THE WITNESS:** It's not referring to herbicides or
24 anything of that ilk.

25

1 **BY MR. MILLER:**

2 **Q.** Oh, all right. So, and in --

3 **A.** You'll recall that it's separated by the two-day -- lesser
4 than two days versus greater than two days.

5 **THE COURT:** And then if I could ask --

6 **THE WITNESS:** Mm-hm.

7 **THE COURT:** -- I guess you've answered the question
8 about the McDuffie Study, and when the incidences of NHL were.
9 Right? You said it was, like, '91 to '94, or something like
10 that, people were diagnosed with NHL?

11 **THE WITNESS:** That's when the cancers occurred, when
12 they were diagnosed, yes.

13 **THE COURT:** When they were diagnosed, okay.

14 **THE WITNESS:** Mm-hm, yes. You know, you always have
15 to -- you can only take newly diagnosed patients into a
16 case-control study. That's, like, a rule of epidemiology.

17 **THE COURT:** Okay, and then what about -- an issue
18 that I don't think you've had a chance to talk about yet is --

19 **THE WITNESS:** Mm-hm.

20 **THE COURT:** -- proxy responders.

21 What -- does -- is the issue of proxy responders a concern
22 for the McDuffie Study?

23 **THE WITNESS:** I don't recall, offhand, how many proxy
24 responders there were, if there were, but as long as -- to me,
25 the solution of proxy responders would be that you keep them

1 equally distributed between cases and controls, so you get a
2 similar -- you eliminate the bias, or whatever problems there
3 were in terms of error, or in terms of -- that a proxy
4 responder would introduce, but --

5 **THE COURT:** If you -- if you had an equal number of
6 or roughly equal number of proxy responders for the cases and
7 for the controls, would that eliminate the concern --

8 **THE WITNESS:** Yes.

9 **THE COURT:** -- about proxy response bias?

10 **THE WITNESS:** I mean, you can always worry, but the
11 answer would be, that would be the solution to the problem,
12 yes.

13 **THE COURT:** Okay, and you don't recall, as you sit
14 here, whether there were any issues --

15 **THE WITNESS:** No.

16 **THE COURT:** -- relating to proxy response.

17 **THE WITNESS:** No, and you know, NHL is a pretty
18 well -- people live -- you know, we're not talking about
19 pancreas cancer. There, you end up oftentimes with almost
20 everyone having a proxy respondent, unfortunately, but in NHL,
21 I would say that, you know, that's not the case.

22 And I would say, if you wended up here with a lot of proxy
23 respondents, you might have problems, because occupational
24 exposure, particularly if we're relying on self-report, you
25 really do want to have the person, to the greatest degree

1 possible, give an answer.

2 **BY MR. MILLER:**

3 **Q.** If I could, on that issue, I want to ask you, from the
4 McDuffie Study, the Materials and Methods on the second page,
5 it lists two points, if we could switch over, and I'll quote.

6 "After position -- "

7 **MR. LASKER:** What page?

8 **MR. MILLER:** Excuse me? We're on page --

9 **MR. LASKER:** Page what?

10 **MR. MILLER:** -- 1156 of the McDuffie, top of the
11 corner, here.

12 **MR. LASKER:** Thank you.

13 **MR. WISNER:** There's this weird thing on the screen,
14 so you have to do it up on the corner. There's this thing
15 that's blocking it.

16 **MR. MILLER:** Excuse me.

17 **Q.** This is in the Materials and Methods, and I apologize for
18 that, but,

19 "After physician's consent was
20 received, postal questionnaires and
21 informed consent forms were mailed to
22 potential cases. Surrogates for deceased
23 cases were not contacted."

24 And to put that in context of this point that Your Honor's
25 raised,

1 "Surrogates for deceased persons were
2 ineligible as controls."

3 **A.** Right.

4 **Q.** All of the participating control subjects were used in the
5 statistical analysis of each cancer site.

6 I don't understand what it means, but I think it addresses
7 this --

8 **A.** Basically saying they didn't have proxies in this study.

9 **Q.** Oh.

10 **THE COURT:** Okay.

11 **MR. MILLER:** Unless Your Honors have any more
12 questions on the McDuffie Study....

13 **Q.** Do you have anything else that you feel you need to share
14 on that.

15 **A.** No.

16 **Q.** Do you want to move to Hardell?

17 **A.** Mm-hm. So Hardell was the Swedish study, and it -- I
18 think it also did not --

19 **THE COURT:** It didn't control for pesticides, right?

20 **THE WITNESS:** Right, it did not control for
21 herbicides, but came up with an elevated Odds Ratio that was
22 not statistically significant.

23 **BY MR. MILLER**

24 **Q.** Well, and then I have a few questions, and I don't know
25 the answer, so I probably shouldn't ask it, but you used the

1 1.85 Odds Ratio for non-Hodgkin's lymphoma, and I understand
2 why, and the univariate analysis, I think they call it, it was
3 actually 3.04, but that's without adjusting. Is that what that
4 is?

5 **A.** Yes.

6 **Q.** That's why you chose not to use it? I'm just asking.

7 **A.** I don't use univariate analyses.

8 **Q.** I understand. I understand. So we go over, then, to the
9 one that you did use, from Table 7, and that's what's referred
10 to as a multivariate analysis. Is that the one that you
11 selected?

12 **A.** Mm-hm.

13 **Q.** Yeah, and so --

14 **A.** So the analysis that we used --

15 **Q.** Yes, sir?

16 **A.** -- and that's the figure that, you know, in the
17 subsequent -- that would be the figure that I would you use for
18 a forest plot or for anything else, to think about, in terms of
19 considering causal association here.

20 **THE COURT:** So that 1.85 Odds Ratio does reflect
21 adjustment for pesticides.

22 **THE WITNESS:** Yes. Oh, in Hardell? Let me make
23 sure. After a while, all of these guys....

24 **THE COURT:** I thought I may be wrong about this, but
25 I thought multivariate analysis sort of equals adjusting for

1 other pesticide use, but maybe that's wrong.

2 **THE WITNESS:** It's equal to adjusting for anything.

3 It means that you adjusted for age, race, sex, whatever.

4 But I believe in Hardell it does reflect -- I apologize,
5 Your Honor.

6 **BY MR. MILLER:**

7 **Q.** It doesn't appear as though they let us know, does it,
8 Doctor?

9 **A.** Yes.

10 **Q.** Yeah.

11 **A.** It adjusts for herbicides.

12 **Q.** Oh, it did?

13 **A.** Mm-hm.

14 **Q.** Okay. All right. Now, before we leave Hardell, is that
15 the -- it's based on the Swedish Population Base, or something?

16 **A.** Yes.

17 **Q.** Swedish?

18 **A.** Yes.

19 **Q.** Okay. All right, anything else about that study --

20 **A.** No.

21 **Q.** -- they we need to --

22 **THE COURT:** What about use of surrogates in that
23 study?

24 **THE WITNESS:** Use of...?

25 **THE COURT:** Surrogate respondents.

1 **THE WITNESS:** I don't -- I don't believe they did,
2 no. Oh, no, I'm sorry, they used next of kin. I apologize.
3 They used next of kin.

4 **THE COURT:** Do you know for what percentage of
5 respondents?

6 **BY MR. MILLER**

7 **Q.** From the Materials and Methods section, Doctor, the NHL
8 study encompassed males greater than 25 years with NHL
9 diagnosed during '87 to '90 and living in the foremost northern
10 counties of Sweden and three counties in mid-Sweden. They were
11 recruited from regional cancer registries, and only cases with
12 histopathologically verified NHL were included. In total, 442
13 cases of these 192 were deceased. Would those be proxy
14 responders then?

15 **MR. LASKER:** Mr. Miller where are you reading from?

16 **MR. MILLER:** Excuse me, I'm on page 1044.

17 **Q.** Does that mean proxy responders of 37 percent, 192 dead
18 people?

19 **A.** As I'm sitting there, I'm not seeing how many proxies.
20 Let me take one.... They don't say specifically.

21 **Q.** Look at the Control section, if you would, Doctor. It
22 says, for each deceased case, two deceased controls matched.

23 **A.** No, I didn't see that.

24 **Q.** Oh.

25 **A.** It doesn't say how many.

1 Q. What's the significance of that?

2 A. I can't find anywhere where it says how many deceased
3 cases there were.

4 JUDGE PETROU: Two.

5 MR. MILLER: How many deceased cases?

6 THE WITNESS: You found it?

7 THE COURT: He was pointing it out to you earlier,
8 down in the lower left.

9 THE WITNESS: I'm sorry.

10 BY MR. MILLER:

11 Q. It's on the screen, Dr. Neugut.

12 A. I'm really being dense, huh? Four -- oh, of these, 192
13 were deceased.

14 Q. Yeah.

15 THE COURT: So does that mean that 192 of the
16 responses were from proxies?

17 THE WITNESS: Yeah, that's correct. So 192 out of
18 442. So that's roughly, a little -- almost half.

19 THE COURT: And how big a deal is that?

20 THE WITNESS: Again, from an epidemio- -- from an
21 epidemiologist's point of view, what's important is that the --
22 it introduces error, but the error is balanced between the two
23 groups. So it's unbiased error. But it does introduce some
24 error, and I would say it's a bit of a concern.

25 In other words, how well does your -- I'll assume his wife

1 knows your use of glyphosates, you know? So my guess is that
2 she's going to underestimate -- well, who knows how she's going
3 to know about it, more or less, you know.

4 So it introduces some error; but again, error attenuates.
5 So again, the error here will attenuate towards the null.

6 So the estimate that you're getting in Hardell, if we're
7 talking about purely the error from having proxies, I would
8 say, would therefore have attenuated the risk estimate.
9 Because again, we're introducing what I would consider to be
10 essentially random error. Like, we're going to have
11 misclassification error in the estimate of the glyphosate
12 usage, both in the cases and in the controls, but theoretically
13 it will be balanced between two groups.

14 **THE COURT:** Would that -- I saw that there was a wide
15 confidence interval --

16 **THE WITNESS:** Mm-hm.

17 **THE COURT:** -- for the Hardell Study.

18 Would -- is that -- does the confidence interval
19 incorporate things like use proxies? I mean, is that why you
20 will see a wider confidence interval?

21 **THE WITNESS:** Possibly, yes.

22 **THE COURT:** Do you know if that's the case in this
23 study?

24 **THE WITNESS:** I wouldn't know offhand, but it also
25 may have lowered the risk estimate, as well.

1 **THE COURT:** If it was non-differential?

2 **THE WITNESS:** If it were non-differential, yes. So
3 the 1.85 and 0.3, again, if we're in Table 7 with the 1.85 Odds
4 Ratio and the 0.55 to 6.20, maybe if you didn't have proxies,
5 you would have had a higher risk estimate, and a statistically
6 significant observed association, potentially.

7 **BY MR. MILLER:**

8 **Q.** With strength in the power?

9 **A.** Well, all errors are conservative, or that theoretically,
10 all unbiased errors are conservative.

11 **Q.** I've run way out of my time. Each lawyer gets amounted so
12 much. So I want to go over what's important, Doctor, and
13 I know you want to go back east, but tell me about De Roos, any
14 significant things that the Court wants to hear about De Roos,
15 and Eriksson, and then I'll leave you.

16 **A.** So De Roos, okay. That's the midwestern study, and it did
17 control for the use of the other herbicides, and it did come up
18 with a statistically significant finding.

19 I don't know if there's much less else to say, unless the
20 Your Honor has another question.

21 **THE COURT:** This was the one where there were
22 different Odds Ratios for the logistic regression analysis and
23 the hierarchical regression analysis.

24 And so the first question is: Is this number associated
25 with the logistic regression analysis or hierarchical?

1 **THE WITNESS:** No. We used regression because I think
2 it's a more legitimate -- first of all, it's more consistent.
3 Everybody -- all the other studies use logistic regression. So
4 first of all, to be consistent across all studies, we certainly
5 use logistic regression sort of being consistent. Hierarchical
6 regression modeling is a fancy-schmancy, sophisticated thing
7 you do to look cool, you know.

8 **THE COURT:** Well, so can you now try and explain the
9 difference between the two, to me?

10 **THE WITNESS:** I was afraid you were going to say
11 that.

12 Do you know which table has the hierarchical? Oh. Oh, I
13 see. Here we go. So --

14 **BY MR. MILLER:**

15 **Q.** All right, let's go to Table 3, Doctor.

16 **A.** Table 3? Oh, I was on Table 5. I see, okay.

17 **Q.** And on Table 3 -- and I'll start with the top -- they show
18 effect estimates for use of specific pesticides and
19 non-Hodgkin's lymphoma incidents, adjusting for use of other
20 pesticides, and they have logistic regression, and
21 hierarchical -- I never say that right -- regression.

22 Going down to the list of herbicides --

23 **A.** Mm-hm.

24 **Q.** -- you see glyphosate. 2.1, statistically significant,
25 under logistical, and 1.6, outside of the statistical

1 significance for hierarchical, if I'm pronouncing that right.

2 What's the significance of all of that, sir?

3 **A.** So I'm going to have to say even I can't figure out the
4 Table 3 in terms of why, for example, one logistical regression
5 comes out at 4, and the other one comes out at 1.8, or
6 something like that. Its -- statistical level of analysis.
7 Well, Dr. Ritz could have handled that one better than I can.

8 **THE COURT:** Okay.

9 **BY MR. MILLER:**

10 **Q.** All right, fair enough. Anything else that you want to
11 say about De Roos --

12 **A.** No.

13 **Q.** -- other than, it is a piece of the puzzle on which you
14 formed your opinion?

15 **A.** Yes.

16 **MR. MILLER:** Any other questions the Court might have
17 about De Roos?

18 Last study, and --

19 **THE COURT:** Maybe, yeah, I do have one more question
20 about De Roos, and it's the question about when people were
21 diagnosed with NHL from these pools, and whether the
22 potentially short period of time between the time glyphosate
23 came on the market and the time these people were diagnosed
24 with NHL affects the analysis.

25 **THE WITNESS:** You mean, because glyphosate came on

1 the market shortly before the diagnoses were made?

2 **THE COURT:** You told me that with McDuffie --

3 **THE WITNESS:** Mm-hm.

4 **THE COURT:** -- people were diagnosed between 1991 and
5 1994 --

6 **THE WITNESS:** Mm-hm.

7 **THE COURT:** -- with glyphosate coming on the market
8 in the mid '70s.

9 **THE WITNESS:** And some of these got diagnosed in the
10 '80s.

11 **THE COURT:** That's what I thought. So I was going to
12 ask you --

13 **THE WITNESS:** Mm-hm.

14 **THE COURT:** -- I thought that some of these folks
15 were diagnosed in the '80s or maybe even the early '70s. I may
16 be mis-remembering that part of it.

17 **THE WITNESS:** Mm-hm.

18 **THE COURT:** How big of a deal is that? How big of a
19 concern is that?

20 **THE WITNESS:** So it depends on what is considered
21 latent, that the Court was talking about before, the latency
22 period -- the latency peered between glyphosate and NHL to be,
23 so which is part of really what the Doctors Portier, probably,
24 and Weisenburger referred to one that I've written; the
25 authority refers to the mechanism of action of glyphosate on --

1 on carcinogenesis, or lymphogenesis here.

2 You know, from my point of view, they're all called
3 "promoters" and "initiators." Promoters can enhance the
4 probability of cancer occurring, even if they don't occur way
5 back at the beginning, you know, where it's -- the process of
6 carcinogenesis takes a decade or more, but we don't have to be
7 talking about an agent which is at the very beginning of the
8 process. We can talk about an agent which acts in a middle of
9 the process, which is a very common phenomenon.

10 Most of the cancer-causing agents that we talk about in
11 daily life actually don't occur at the beginning of
12 carcinogenesis; they act in the middle of carcinogenesis or
13 near the end of carcinogenesis. That's why we can act on them
14 for prevention.

15 When we talk about obesity causing breast cancer, there
16 would be no point in losing weight if it acted 30 years ago,
17 because, then, what would be the point? But it acts near the
18 end of the cancer-causing process.

19 So similarly, glyphosate could or could not be acting near
20 the end of the causation of lymphoma to be enhancing, let say,
21 you may already have cells or partially developed, it's
22 partially along the way towards becoming lymphoma cells, and
23 glyphosate somehow causes them to proliferate faster.

24 **THE COURT:** But -- and I understand that.

25 **THE WITNESS:** Mm-hm.

1 **THE COURT:** But if you're doing the study of people
2 who were diagnosed with NHL, say, in the mid '80s --

3 **THE WITNESS:** Mm-hm.

4 **THE COURT:** -- and glyphosate came on the market in
5 late '70s --

6 **THE WITNESS:** Mm-hm.

7 **THE COURT:** -- wouldn't that be a major concern with
8 the study? I mean if the study did not take that into account
9 in a major way, wouldn't that be a very significant concern to
10 the study?

11 And I don't know whether De Roos took it in to account or
12 not, but if it didn't, wouldn't that be -- wouldn't that be a
13 major concern?

14 **THE WITNESS:** So from an epidemiologist's point of
15 view, to some degree -- and I'll say this, and it's sometimes a
16 criticism of epidemiology, epidemiology operates in a black
17 box. It looks at, you're exposed, you get cancer. What
18 happens in between, I'm not sure. I'm looking to see if
19 there's an association between the two statistically.

20 Now, the process -- now, if you ask me, did the cancer --
21 did the five years affect it? I can do things, as follows.

22 I can say, I'll eliminate five years after exposure, and
23 see if there's an increase. If there's still an association
24 between -- if there's an association between the two, I'll say
25 I'll take out all of the cases that there are in the first five

1 years, and see if I still see an association. That will tell
2 me if -- if the latency is five years, or not.

3 So we can play statistical games or, you know, thought
4 processes to investigate what the latency is, and we do that
5 all the time. I don't know if that was done by De Roos or by
6 the others, in terms of looking at it. They don't report it in
7 here. So it's hard to know what it is.

8 If you asked me how long -- if there are studies which
9 show that lymphomas occur in less than five years after
10 exposure to a carcinogen, my answer is yes, there are, but
11 I don't know if they're of the same mechanism of action as
12 glyphosate. So I -- you know, it's possible yes, possible no.

13 So for the latency period is --

14 **THE COURT:** But if you have reason to believe that
15 the latency period is 10 years, let's say --

16 **THE WITNESS:** Mm-hm, approximately, yes.

17 **THE COURT:** -- wouldn't that be a massive mistake not
18 to do something along the lines of what you're talking about,
19 that is --

20 **THE WITNESS:** Or I'd be concerned at least about the
21 first five years of the cases.

22 I mean, if De Roos was collecting cases over a -- I don't
23 know over a how many year period, then I at least want, over
24 the first few years, I'd be concerned about that. That would
25 be true.

1 **THE COURT:** Okay, thank you.

2 **BY MR. MILLER**

3 **Q.** And that concept of causing cancer later in the cell
4 process, is that tumor promoter theory?

5 **A.** Yeah, mm-hm.

6 **Q.** And last point, I'm done. On the -- when they -- and we
7 call it De Roos. Dr. Weisenburger --

8 **THE COURT:** I'm sorry to interrupt, one other
9 question about that.

10 On the point you were making about the agent acting sort
11 of in the middle of the process rather than the end --

12 **THE WITNESS:** Mm-hm.

13 **THE COURT:** -- is that a potential response to a
14 criticism of the AHS Study as well? Do you get my question?

15 **THE WITNESS:** The AHS cohort study.

16 **THE COURT:** Yeah. In other words, there's this
17 criticism of AHS that we only have 18 years of follow-up, and
18 the latency period may be quite long for NHL --

19 **THE WITNESS:** Oh.

20 **THE COURT:** -- as caused by glyphosate, perhaps, as
21 suggested by the Eriksson Study. So what you just said, isn't
22 that a potential response to that criticism of the AHS Study?

23 **THE WITNESS:** Yeah. I don't think 18 years is not --
24 I don't know if 18 years is -- I don't want to -- I don't know
25 if 18 years -- 18 years is not that short.

1 You know, I mean, you know, depends on how long you think
2 the process of carcinogenesis takes. I mean, that's almost two
3 decades.

4 **THE COURT:** But the concept of glyphosate acting in
5 the middle of the process in carcinogenesis --

6 **THE WITNESS:** Mm-hm.

7 **THE COURT:** -- that would apply to the folks in
8 the -- who are being studied in the AHS Study as well as
9 potentially the folks who are being studied in De Roos, right?

10 **THE WITNESS:** I would assume. Mm-hm, yes.

11 **THE COURT:** Okay. All right.

12 **THE WITNESS:** And if, indeed, that is what happens.

13 **THE COURT:** Right.

14 **THE WITNESS:** I'm speculating.

15 **BY MR. MILLER:**

16 **Q.** Last point, and I'm going to sit down.

17 So in the De Roos/Weisenburger Study of '03, in the
18 Methods section, they tell us that the earliest diagnosis was
19 in July of '83, and if glyphosate came on the market in
20 seventy --

21 **THE COURT:** Sorry, which study are you talking about
22 now?

23 **MR. MILLER:** De Roos '03, Your Honor. Thank you.

24 **THE COURT:** Thank you.
25

1 **BY MR. MILLER:**

2 **Q.** And the Methods section tells us that the first diagnosis
3 was in July of '83, so that if Roundup® came on the market in
4 '74 that would be nine years between the first introduction of
5 Roundup® and the first diagnosis.

6 **A.** Yeah, mm-hm.

7 **MR. MILLER:** I've been handed something else. All
8 right. Thank you very much. I yield the witness.

9 **THE COURT:** Okay. Do you want to -- I mean, we're
10 happy to go on. You want to -- I don't know how long you have
11 with this witness, but.

12 **MR. LASKER:** Not long. I don't know that I'll
13 finish, but I can get started, certainly.

14 **THE COURT:** Sure. Of course.

15 **MR. LASKER:** And how do you turn this on? What --
16 I'm sorry, what --

17 (Discussion off the record.)

18 **THE WITNESS:** But don't I get the other guy?

19 **MR. LASKER:** I think Mr. Miller wants to correct his
20 question on De Roos.

21 **MR. MILLER:** The first page started at 93, but my
22 co-counsel is telling me I needed to go to the second page, to
23 see where I now -- here it is.

24 All right, let's go. I want to make sure I'm not -- yes,
25 I stand corrected. It was from '81, in Iowa, and Kansas, it

1 went it went back to '79.

2 I think that's all of it, right?

3 **MR. WISNER:** Yeah.

4 **MR. MILLER:** I just wanted to make sure I didn't
5 misspeak. I thank you. I apologize.

6 **THE COURT:** And Dr. Neugut, you are free to tell
7 Mr. Lasker to slow down.

8 (Laughter.)

9 **MR. LASKER:** This is going to be -- we'll finish the
10 cross. It will take us about five minutes. I do have 35 pages
11 here of questions.

12 **CROSS-EXAMINATION**

13 **BY MR. LASKER**

14 **Q.** Dr. Neugut, just to clarify that final point that
15 Mr. Miller made, because we have diagnoses starting in 1979 and
16 1980, in the De Roos Study, we have cases there where the
17 maximum conceivable latency could be -- would be five years;
18 and that's only if the farmers started using glyphosate the
19 minute it came on the market. Correct?

20 **A.** Yes.

21 **Q.** And we could have -- of those cases, of course, some of
22 them would not have been using glyphosate on Day One. Correct?

23 **A.** Of course; but again, we're talking about -- talking about
24 a sample size of 3,400. So we're talking about, I'm assuming a
25 small subset of the totality. So I don't know how important

1 this is in the overall analysis of the findings of the study,
2 and the risk estimates, and -- et cetera, et cetera, but you're
3 accurate in terms of what you're saying.

4 Q. Well, we actually can tell if we go to Table 2, which is
5 on the fourth page.

6 And do we have this -- which exhibit number are we, just
7 so the record's clear? I have Exhibit 720. Is that our
8 Exhibit number?

9 MR. KALAS: Right, yeah.

10 BY MR. LASKER

11 Q. So it's Defense Exhibit 720.

12 A. Table 2.

13 Q. Yes, Table 2?

14 A. Mm-hm.

15 Q. And we have here, actually, the breakdown for each of the
16 states; how many of the cases and controls that they
17 contributed to this study. Correct?

18 A. How many cases are from each state? That's what you're
19 saying?

20 Q. Right, yes. And the vast majority of the cases and the
21 controls in this study came from either Iowa, Minnesota, or
22 Kansas. Correct?

23 A. Yes.

24 Q. Somewhere in the vicinity of 80 percent of the study.
25 Correct?

1 A. Yes.

2 Q. And if we then go back to our Methods section to find out
3 when those individuals were diagnosed, it's on the second page,
4 in the top left-hand column. For Iowa, the diagnoses were 1981
5 to 1983. Correct?

6 A. Yes.

7 Q. For Minnesota, it was 1980 to 1982. Correct?

8 A. Yes.

9 Q. And for Kansas, it was 1979 to 1981. Correct?

10 A. Yes.

11 Q. So for 80 percent of this study, the maximum period of use
12 would have been within a range of between four to eight years,
13 as far as the maximum latency possible, if every one of those
14 people started using glyphosate on Day One. Correct?

15 A. Yeah. But as I say, I'm not saying I necessarily think
16 that the latency period is five years, or less than that.
17 I didn't define the latency period.

18 Q. And you talked about this possibility of doing a lagged
19 analysis.

20 A. Mm-hm.

21 Q. And epidemiologists would do a lagged analysis in this
22 situation to try and parse out if this is a problem. Correct?

23 A. I -- so I don't know if -- yes.

24 Q. And in fact, there is no lagged analysis that was
25 conducted in the De Roos Study. Correct?

1 A. No.

2 Q. It's correct that they did not do a lagged analysis?

3 A. It's correct that they did not do one.

4 Q. Okay. Now, Dr. Neugut, I want to go back to where
5 Mr. Miller began. Mr. Miller asked you whether you've used the
6 same intellectual rigor to reach your expert opinion in this
7 litigation as you use in your ordinary course of work; and you
8 said "Yes." Correct?

9 A. Yes.

10 Q. And he said that you reviewed a lot of stuff to reach your
11 conclusions. Correct?

12 A. Yes.

13 Q. And your conclusion was that, to a reasonable degree of
14 medical certainty, glyphosate causes non-Hodgkin's lymphoma.
15 Correct?

16 A. Yes.

17 Q. And, in fact, you reached that opinion in this litigation
18 before you had read any of the glyphosate epidemiologic
19 studies. Correct?

20 A. That's correct. After I read the IARC Monograph, and I
21 don't know if I didn't read any studies, but not -- I may not
22 have read all of the studies at that point.

23 Q. Well, let's go to -- and it's at Tab 2 of the binder.

24 And do we have an exhibit number for this?

25 MS. LYNHAM: It's 1523.

1 **MR. KALAS:** Twenty-three.

2 **MS. LYNHAM:** 1523.

3 **MR. LASKER:** This will be Defense Exhibit 1523. I'm
4 sorry.

5 **Q.** And this is a declaration that you signed under oath, and
6 submitted in another case in this litigation. It wasn't in
7 this court. And you submitted this declaration on -- in April
8 of 20-- -- or you signed it, anyway, in April of 2016. Correct?

9 **A.** Yes.

10 **Q.** And in this declaration, you state your opinion, it's
11 paragraph 7.

12 "It is my opinion to a reasonable
13 degree of medical certainty that glyphosate
14 does cause non-Hodgkin's lymphoma in
15 humans."

16 Correct?

17 **A.** Yes.

18 **Q.** And in your deposition in this case, I asked you if your
19 review of the actual underlying epidemiologic studies took
20 place after April 26, 2016, after your declaration, and you
21 stated yes. Correct?

22 **A.** Yes.

23 **Q.** And that was true testimony, correct? Under oath?

24 **A.** What was my testimony?

25 **Q.** It's right up on the screen. The actual review of the

1 underlying studies epidemiological studies would have taken
2 place after your April 2016 declaration. Correct?

3 **A.** I won't say I didn't read any of them. I read some of
4 them before and some of them after.

5 **Q.** You want to say that now, but when I asked you that
6 question in your deposition, you said that. Correct?

7 **A.** When I read the monograph, I did refer back to some of the
8 epidemiological studies, but it's correct that I did not do a
9 complete review of all of epidemiologic literature at that
10 time.

11 **Q.** In fact, for this declaration, you based your opinion --
12 an opinion that glyphosate, to a reasonable degree of medical
13 certainty, your opinion that it causes non-Hodgkin's lymphoma
14 was based upon your review of the IARC Monograph. Correct?

15 **A.** In most part, yes.

16 **Q.** And you stated in your testimony earlier that you view
17 IARC, when it says "probable," that means 70 percent or
18 80 percent. That's what IARC means by that. Correct?

19 **A.** That's my opinion.

20 **MR. LASKER:** Okay. And do we have the IARC preamble?

21 **MR. KALAS:** Yes.

22 (Whereupon a document was tendered to the Court.)

23 **MR. LASKER:** And I'll wait for it to get up to
24 Your Honor. It's Defense Exhibit 1049. Do you have the
25 preamble?

1 (Discussion off the record.)

2 **MR. LASKER:** It's also a Plaintiff's exhibit, but --
3 and I'm going to put this up on the Elmo, if I can figure out
4 how to work it.

5 **MR. GRIFFIS:** You want the actual preamble up?

6 **MR. LASKER:** Yes, if we could do that. Do you have
7 page 22?

8 **THE COURT:** Page 22 of the preamble, you said?

9 **MR. LASKER:** Yeah, page 22 of the preamble, and for
10 Your Honors, it's the paragraph Group 2, and in particular,
11 where we're talking about lines 29 through 32, and you can see
12 that if we can get that out of range off there.

13 **THE COURT:** I think you just have to wait. It will
14 disappear in a couple of seconds.

15 **BY MR. LASKER:**

16 **Q.** Okay. So Dr. Neugut, if you can read on your screen, this
17 is from the IARC preamble, and we're on page 22 lines 29
18 through 32.

19 What IARC states about the word "probable," the terms
20 "probably carcinogenic" and "possibly carcinogenic" have no
21 quantitative significance and are used simply as descriptors of
22 different levels of evidence of human carcinogenicity, with
23 "probably carcinogenic" signifying a higher level of evidence
24 than "possibly carcinogenic." Correct?

25 **A.** Yes, what I stated was it was my opinion that when IARC

1 states that something is "probably carcinogenic," that its
2 probability of being carcinogenic is promptly in the 70 to
3 90 percent range.

4 Q. I understand, and that's the opinion you have, which was
5 the basis for you being able to reach a decision prior to
6 reading and doing a review of the epidemiological studies.
7 What happened was --

8 A. I wasn't referring to glyphosate specifically.

9 THE COURT: You've got to let Mr. Lasker finish his
10 questions.

11 BY MR. LASKER

12 Q. Your understanding -- not what the preamble states, but
13 your understanding of what "probably carcinogenic" means in
14 IARC's terminology was the basis for you to reach an opinion to
15 a reasonable degree of medical -- of scientific certainty, and
16 signing it and submitting it in court as your expert opinion
17 before doing a review of the epidemiologic studies. Correct?

18 A. Correct, but the -- if I go back to this, this says it is
19 my opinion to a reasonable degree of medical certainty that
20 glyphosate does cause non-Hodgkin's lymphoma in humans.
21 I believe the probability that goes with medical certainty is
22 51 percent, and I'm willing to stand by the statement that I
23 wrote on 4/28/16.

24 And in fact, if IARC had made it a limited, to be, based
25 on the evidence that I read in the -- I think in the monograph

1 alone, without reading any other scientific literature,
2 I probably would have been -- in fact, I would have been
3 willing, in fact, I was willing to sign this statement.

4 Q. All right. Just so I'm clear, then, if IARC was to
5 classify -- had classified glyphosate as just "possibly
6 carcinogenic," and you were to review that classification
7 alone, with nothing more, based upon your methodology, that
8 would be sufficient for you to come into court --

9 A. I'm not --

10 Q. -- and testify to a reasonable degree of medical certainty
11 that glyphosate can cause non-Hodgkin's lymphoma in the humans.
12 Is that correct?

13 A. That is not what I said.

14 Q. Okay. Well, we'll have to look at the record on that.

15 Dr. Neugut, when you subsequently reviewed the
16 epidemiologic studies for purposes of your Expert Report in
17 this case, you also sought to adhere to IARC's guidelines as to
18 how those studies should be considered. Correct?

19 A. I didn't hear you.

20 Q. When you subsequently reviewed the actual epidemiologic
21 studies to be able to draft up your Expert Report in this case,
22 you sought to adhere to the IARC preamble and the guidelines as
23 to how that data would be considered by IARC. Correct?

24 A. Yeah.

25 Q. And because of that, for example, we've heard testimony

1 from all the experts -- two experts who have spoken previously
2 about the NAPP Study. The N-A-P-P Study. Correct?

3 **A.** Yes.

4 **Q.** And the NAPP, as we've heard, is a pooled analysis that
5 pools together all of the case-control studies in the U.S. and
6 in Canada, which includes the McDuffie Study and the De Roos
7 2003 Study that you talked about earlier. Correct?

8 **A.** Yes.

9 **Q.** And you would agree that once you've pooled those studies
10 into a larger study, it is the later pooled study that provides
11 all of the data relevant to causation. Correct?

12 **A.** I don't know. I never read the NAPP Study.

13 **Q.** Well, first of all, I'm just asking you the question, and
14 I want to see if you agree, and this is what you testified --
15 and actually, why don't I just put this up. It is Slide 61
16 from your deposition, your first deposition, page 228, 17 to
17 21.

18 It is fair to say that once you pool those studies into a
19 larger study, it's the later pooled study that provides all of
20 the data relevant to a causation theme. Correct?

21 **A.** Well, this is what I said.

22 **Q.** Yes.

23 **A.** If that's what I said, that's what I said.

24 **Q.** But because you're following IARC's methodology, despite
25 the fact that every other expert epidemiologist in this case

1 has talked about the NAPP Study, talked about the ability to
2 adjust for confounding pesticides, how they did that, and has
3 talked in detail about the findings of that pooled analysis and
4 what it shows, you have not read this study at all. Correct?

5 **A.** No, that's not accurate.

6 **Q.** Have you read the study?

7 **A.** No, but it's not because of the reason you gave.

8 The other two experts, if you're referring to Weisenburger
9 and Ritz, were both involved with the NAPP Study. I have no
10 involvement with it, number one. Number two, I wasn't present
11 when the NAPP Study was presented. No one sent me to Brazil,
12 and I wasn't in Montreal.

13 This is an abstract, and I don't -- it's because it's an
14 abstract that I don't review it, not because of -- it's not a
15 peer-reviewed publication. It's not a publication.

16 So that's why I haven't included it in my thinking or
17 analysis. I have no ability to review the full dataset. Even
18 today, Weisenburger said that he didn't have the full data from
19 this study available. How would I have the full data from the
20 study available? And he's a co-author, on the study.

21 **Q.** Dr. Neugut, did you -- you could have reviewed -- in our
22 deposition, I showed you there were slide decks of data from
23 the NAPP. Correct?

24 **A.** Which is what he had today.

25 **Q.** And we walked through some of those findings. Correct?

1 A. And --

2 Q. Well, you could have, in your analysis, looked at that
3 data. It's not just an abstract. Dr. Ritz testified that this
4 is a peer-reviewed presentation that's been presented at
5 several scientific conferences. You could have reviewed the
6 data.

7 A. Well, there's a difference between an abstract and a
8 publication. An abstract is about 20 lines long, versus a full
9 publication, and I can choose not to review a full publication.
10 If you think I'm being nitpicky, then so be it.

11 Q. Dr. Neugut, do you know what data exists and what data the
12 other experts in this case have reviewed for the NAPP?

13 A. No.

14 Q. And at no point in your very thorough review, using your
15 same intellectual rigor as you used in trying to come up with
16 an answer to this question, despite knowing that that data
17 existed, and you decide, *I'm not going to even look at it to*
18 *decide whether it's reliable or not.* Correct?

19 A. Would I have -- so would I have applied it in my academic
20 rigor in my daily life in academia? I'm not sure that I would
21 have. So I apply those standards, as well.

22 Q. Now, let's talk a little bit more about that. You would
23 agree that IARC -- the IARC criteria are used to reach a public
24 health determination. Correct?

25 A. Yeah -- and I didn't hear the question.

1 Q. I'm sorry. You agree that the IARC criteria are used to
2 reach an assessment for a public health determination.

3 Correct?

4 A. Yes.

5 Q. And when you are conducting an assessment of the
6 epidemiological literature for a scientific assessment of a
7 potential causal inference, you might use a different
8 methodology. Correct?

9 Let's put this up. You have already answered this
10 question. That was at 261, line 17 of your deposition, and I
11 asked you that exact question.

12 "QUESTION: When you were conducting an
13 assessment of the epidemiological literature
14 to reach a scientific as opposed to a public
15 health conclusion, you might have a different
16 methodology."

17 And your answer was, "Possibly."

18 Correct?

19 A. Possibly is possibly.

20 Q. Now, an epidemiologist following a scientific method would
21 be formulating hypotheses; then testing those hypotheses to see
22 if they could be validated, and then testing them again to see
23 if they would be replicated. Correct? That's the scientific
24 method.

25 A. Yes.

1 Q. And epidemiologists can design epidemiologic studies to
2 test a hypothesis. Correct?

3 A. Yes.

4 Q. And hypotheses at issue in this proceeding is whether
5 glyphosate or glyphosate-based herbicides can cause
6 non-Hodgkin's lymphoma. Correct?

7 A. Yes.

8 Q. Some epidemiologic studies, though, report out a number of
9 different potential associations relating to different
10 exposures where they do not have a preset hypothesis. Correct?

11 A. So that's still not -- are you arguing that that's not
12 scientific method?

13 Q. Well, I just want an answer to my question, first.

14 It is true that there are epidemiologic studies that
15 report out a number of different potential associations
16 relating to different exposures where they don't have a preset
17 hypothesis. Correct?

18 A. Of course.

19 Q. And those studies are referred to as exploratory studies.
20 Correct?

21 A. Hypothesis-generating, yes, mm-hm.

22 Q. Exactly. Exploratory studies are not actually testing
23 hypotheses, they are generating additional hypotheses.
24 Correct?

25 A. Yes.

1 Q. And the McDuffie case-control study was an exploratory
2 study. Correct?

3 A. Which one?

4 Q. McDuffie.

5 A. Off the top of my head, I don't know, but --

6 Q. Well, I asked you this in your deposition. Let's put this
7 up: Page 214, 11 to 23. And I asked you whether or not
8 McDuffie's study with respect to glyphosate was an exploratory
9 study. And your answer was, "Yes, that's correct." Right?

10 A. Okay.

11 Q. And the Eriksson Study -- we'll be talking more about that
12 probably tomorrow -- that also was an exploratory study.
13 Correct?

14 And let's put up Slide 14. This was your deposition
15 testimony at 267, 8 -- lines 8 to 20.

16 The Eriksson Study, like McDuffie, is an exploratory
17 study. Correct?

18 A. Well, as I sit here, now if the McDuffie Study already
19 generated the hypothesis, then how can the Eriksson Study be
20 exploratory?

21 Q. Well, I'm asking you, because I asked you this in your
22 deposition, and in your deposition, I asked you if Eriksson was
23 exploratory, and you said yes. Correct? It's right up on your
24 screen.

25 Right now the only question is whether you stated that in

1 your -- testified to that in your deposition in this case.

2 **A.** Well, if that's what I said, then that's what I said, but
3 then -- now, if the hypothesis was already generated by one of
4 them, then the second time around, it's no longer
5 hypothesis-generating.

6 **Q.** We also talked about the Cantor Study, and that was a
7 study that we just looked at as one of the main contributors to
8 the De Roos Study. I think it was actually about 60 percent of
9 the De Roos Study. And Dr. Weisenburger testified earlier that
10 all of those studies were basically the same design.

11 I asked you whether that -- those U.S.-based control
12 studies were exploratory studies. And again, you said, yes,
13 those were exploratory studies. Correct?

14 And we can put up Slide 15, talking about the
15 Cantor Study, which was, I think -- let me get this wrong --
16 someone help me -- Iowa and -- hold on a second -- it was the
17 Iowa and Minnesota portion of the De Roos Study, which is
18 67 percent of cases.

19 And again, all of the U.S.-based case-control studies, per
20 Dr. Weisenburger, with same design, you'd agree with that.
21 Correct?

22 **A.** Yes.

23 **Q.** Okay, and for the Cantor Study and the U.S.-based control
24 studies, those are also exploratory studies. They are
25 generating hypotheses, but they're not testing hypotheses.

1 Correct?

2 **A.** Yes.

3 **Q.** Now, let's talk about, when you have an epidemiologic
4 study that is designed, unlike these case-control studies, to
5 test a causal hypothesis, you would not label an exposure as
6 being associated with an outcome, unless there was a finding of
7 an increased risk that is statistically significant. Correct?

8 **A.** Say that again.

9 **Q.** You would not label an exposure as being associated with
10 an outcome, unless there is a finding of increased risk that is
11 statistically significant. Correct?

12 Let's put up Slide 16, because I asked you this exact
13 question.

14 **A.** We've been talking all day about how you can have
15 non-statistically significant associations.

16 **Q.** I understand that you testified here in court today and at
17 your deposition when I asked you this question:

18 **"QUESTION:** You stated you would not label an
19 exposure as being associated with an outcome
20 unless there was a finding of increased risk
21 that is statistically significant, correct?"

22 And you said, "That's correct."

23 That's your testimony. Correct?

24 **A.** You're right, but the point, though, is that there is
25 flexibility in terms of interpreting non-statistically

1 associations, as well.

2 Q. And another thing that we talked about is that even when
3 you have statistical significance, that does not answer the
4 question about whether or not a study has issues of bias and
5 confounding. And there's been a lot of discussion about that
6 in this hearing. Correct?

7 A. Of course.

8 Q. And particularly here -- I believe Judge Chhabria raised
9 this earlier -- there is evidence of an increased risk of
10 non-Hodgkin's lymphoma in farmers that existed prior to the
11 introduction of glyphosate. Correct?

12 A. Yeah.

13 Q. And we know, because of that, that there is something
14 going on with farmers and their exposures that is leading to an
15 increased risk of non-Hodgkin's lymphoma that we know for a
16 fact is not glyphosate. Correct?

17 A. Well, we don't know why it is.

18 Q. We know it's not glyphosate, though. Correct?

19 A. Yes.

20 Q. And at your deposition you agreed because of that -- and
21 you noted it in several places in your Expert Report -- that an
22 epidemiological analysis of glyphosate and non-Hodgkin's
23 lymphoma should control for exposures to other pesticides.
24 Correct?

25 A. Yes.

1 Q. Now, we'll talk about your criticisms of this study
2 probably tomorrow, the 2018 *JNCI* Study, but you agree that, as
3 reported by the ten government investigators, and I think there
4 are two academic investigators, who combined -- they did not
5 find and they did not report association between glyphosate and
6 non-Hodgkin's lymphoma. Correct?

7 A. Who did that?

8 Q. The ten NCI and NIH scientists who collaborated with two
9 academicians, independent academicians, to investigate this,
10 prepare the study, and publish it. Correct?

11 A. Correct.

12 Q. And you stated in one of your earlier slides that the only
13 thing that changed from the time of IARC to the time of today
14 was the 2018 *JNCI* Study; but there also is that NAPP analysis
15 that came out after the IARC preamble, but you haven't read
16 that.

17 A. Correct.

18 Q. Even prior to the publication of the 2018 NCI Study, and
19 even prior to the pooling of all of the U.S.-based case-control
20 studies, and the adjustment for those three pesticides that
21 we've heard about already at length today and yesterday, that
22 brought that Odds Ratio down to 1.13, and not significant --
23 even without that information, you believe that the
24 epidemiologic evidence was not sufficient to show causal
25 association between glyphosate and non-Hodgkin's lymphoma.

1 Correct?

2 **A.** I thought it was not sufficient?

3 **Q.** Not sufficient.

4 Pull up Slide 24.

5 You would agree that the epidemiological -- and this is
6 before you knew about the 2018 Study, obviously, and without
7 reading NAPP. And you agree that the epidemiology, alone, is
8 not sufficient to show a causal relationship between glyphosate
9 and non-Hodgkin's lymphoma. That was your testimony at your
10 deposition that we're reading right here. Correct?

11 **A.** Yeah, but I've been re-thinking that.

12 **MR. LASKER:** Well, Your Honor, I think this is a good
13 time for the break.

14 **THE COURT:** Okay. Sounds good. So we'll see
15 everybody tomorrow at -- we start at 10:00 tomorrow. Is that
16 right? Okay.

17 **THE WITNESS:** Your Honor, I have a 1:30 flight.

18 **THE COURT:** You have a 1:30 flight?

19 We do could one thing. What we could do is a late launch,
20 and the other thing we could do is keep going today, to ensure
21 that Dr. Neugut makes his flight.

22 **THE WITNESS:** I mean, it's up to Mr. Lasker. I don't
23 know what his -- how long --

24 **MS. WAGSTAFF:** Let's keep going on.

25 **THE COURT:** Mr. Lasker, do you have a rough time

1 estimate on how much more you need for cross-examination?

2 **MR. LASKER:** I think I can make it shorter, but if we
3 do it tomorrow, we'll try and cut it down now. I'm going to
4 try and cut it back, because I think Your Honor has addressed a
5 lot of issues in your questions, but I think I'd be able to do
6 that better tomorrow morning. Unfortunately, if I'd try doing
7 it now, I think it will take too much time.

8 **THE COURT:** Okay. Well, I mean, I would like
9 Dr. Neugut to be able to make his flight.

10 **MR. LASKER:** I'm sorry, what?

11 **THE COURT:** Dr. Neugut has a 1:30 flight.

12 **MR. LASKER:** What time, we're starting at 10:00?

13 **THE COURT:** Yeah, that's when we were --

14 **MR. LASKER:** I think I'm not going to be going nearly
15 that long with him. And actually, if I can start tomorrow
16 morning, I'll be able to make it shorter. That's not going to
17 be a problem, unless Mr. Miller has a very, very long redirect.

18 **MR. MILLER:** Very short redirect, Your Honor, I'm
19 sure.

20 **THE COURT:** Okay. You know, that's one of the
21 hazards of testifying in court, is sometimes you have to change
22 your flight. So hopefully you won't have to do that.

23 All right. We'll see everyone tomorrow. Thank you.

24 (At 4:34 p.m. the proceedings were adjourned.)

25

1 I certify that the foregoing is a correct transcript from the
2 record of proceedings in the above-entitled matter.

3

4 *Lydia Zinn*

5 _____ March 7, 2018

6 Signature of Court Reporter/Transcriber Date

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Lydia Zinn