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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

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Also Present: The Honorable Ioana Petrou
Leonora Lynham
Scott Duval

I N D E X

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Tuesday - March 6, 2018 **12:35 p.m.**

P R O C E E D I N G S

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THE COURT: Okay. Welcome back.

THE WITNESS: Thank you.

THE COURT: Ready to resume?

MS. FORGIE: Yes, Your Honor.

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

MS. FORGIE: Yes, Your Honor.

THE COURT: Go ahead.

MS. FORGIE: Very briefly, I hope.

DENNIS WEISENBURGER,

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

DIRECT EXAMINATION (resumed)

BY MS. FORGIE

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

Oh. You can't hear me?

A. I can hear you.

Q. Is that better? Thank you.

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

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

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

THE WITNESS: Yes.

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

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

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

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

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

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

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

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

A. Yes.

Q. Okay. And then with regard --

The Judge also had some questions about recall bias.

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

MS. FORGIE: Sorry.

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

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

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

(Reporter requests clarification.)

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

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

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

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

BY MS. FORGIE

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

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

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

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

THE COURT: You said over 5,000 controls?

THE WITNESS: Yes.

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

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

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

THE COURT: In what paper?

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

THE COURT: Okay.

BY MS. FORGIE

Q. Meaning the NAPP Study?

A. Yeah.

Q. Yeah.

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

THE COURT: There were 113 cases exposed to glyphosate. So in other words, from this universe of --

What was it?

THE WITNESS: 16,000.

THE COURT: 1,600?

THE WITNESS: 1,600. Yeah.

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

THE WITNESS: 116. 113.

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

THE WITNESS: Yes.

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

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

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

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

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

THE WITNESS: Yes.

THE COURT: Okay.

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

THE COURT: And what were they asked about exposure?

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

How often did you use it?

How many days per year?

How many years did you use it?

Did you use protective equipment; protective clothing?

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

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

they wrote down whatever the number the farmer told them.

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

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

THE COURT: Okay.

MS. FORGIE: Okay.

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

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

THE COURT: If I could interrupt.

I understand what recall bias is.

THE WITNESS: Okay.

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

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

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

MS. FORGIE: I'm sorry.

THE WITNESS: Well, okay. Good.

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

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

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

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

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

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

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

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

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

non-Hodgkin's lymphoma.

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

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

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

BY MS. FORGIE

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

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

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

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

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

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

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

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

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

THE WITNESS: Yes.

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

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

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

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

THE COURT: Does not?

THE WITNESS: No.

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

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

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

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

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

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

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

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

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

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

On the other hand --

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

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

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

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

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

THE COURT: So could I just --

THE WITNESS: Sure.

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

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

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

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

THE COURT: But why? I mean, why would we ever --

I mean, I understand your point about not including arsenic. Like, maybe we should take arsenic out of the multivariate analysis.

THE WITNESS: Right.

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

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

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

THE COURT: All did what?

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

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

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

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

THE WITNESS: Well, I was talking about glyphosate.

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

THE WITNESS: Right.

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

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

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

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

THE WITNESS: Because it's not a confounder.

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

THE WITNESS: Yes, yes.

THE COURT: Studies of arsenic?

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

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

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

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

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

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

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

THE WITNESS: It would be better.

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

THE WITNESS: Yes.

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

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

THE COURT: Okay. Thank you.

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

BY MS. FORGIE

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

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

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

Q. Right.

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

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

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

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

it's not significant, it tells you something.

BY MS. FORGIE

Q. Okay.

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

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

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

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

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

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

THE WITNESS: No, no.

JUDGE PETROU: Okay. Go ahead.

BY MS. FORGIE

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

A. Okay.

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

THE WITNESS: Lifetime-days. Yes.

Go ahead.

BY MS. FORGIE

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

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

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

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

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

A. Yeah.

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

your opinions. Correct?

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

Q. And so that --

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

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

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

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

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

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

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

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

BY MS. FORGIE

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

A. Right.

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

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

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

A. Right.

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

A. Right. Right. Right.

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

A. Yes.

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

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

THE COURT: How about Dr. Neugut?

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

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

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

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

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

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

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

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

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

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

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

BY MS. FORGIE

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

A couple of really quick questions. Yesterday you spoke a

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

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

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

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

THE WITNESS: Right.

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

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

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

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

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

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

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

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

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

I'm going to pass the witness.

MR. GRIFFIS: Binder for you.

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

MR. GRIFFIS: Good.

(Whereupon a document was tendered to the Court.)

CROSS-EXAMINATION

BY MR. GRIFFIS

Q. Good morning, sir. Good afternoon.

A. Good afternoon. Excuse me. Good afternoon.

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

A. Yes.

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

A. Yes.

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

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

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

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

A. Yes.

Q. More likely than not. Now, you were identified --

Slide 1, please.

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

A. I don't know.

Q. Do you see the letter there?

A. Okay.

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

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

A. That's true.

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

A. That's true.

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

A. Yes.

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

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

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

A. Right.

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

A. Right.

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

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

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

Could we have Slide 2, please?

Those are the studies from that table. Right?

A. Yes.

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

A. Yes.

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

A. Also with a small number of case.

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

A. Yes.

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

was good, and the improvement on McDuffie and De Roos today. You didn't mention NAPP at all in your Expert Report. Right?

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

Q. I brought it up, and we talked about it at the deposition. Right?

A. Yes.

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

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

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

A. Yes.

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

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

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

A. It's later data. Yes.

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

Right?

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

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

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

MR. GRIFFIS: Yes.

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

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

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

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

THE COURT: Okay.

BY MR. GRIFFIS

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

A. Yes.

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

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

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

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

A. Correct.

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

significant. Right?

A. Right.

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

A. For ever/never.

Q. For ever/never. Right.

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

A. Correct.

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

A. Right.

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

A. Correct.

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

A. That's true.

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

A. I think it was the logistic one.

Q. Okay. Let's have Slide 81.

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

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

A. Right.

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

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

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

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

Q. Okay. Now --

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

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

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

THE WITNESS: Is Dr. Neugut there?

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

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

THE WITNESS: They both did.

THE COURT: Okay. All right.

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

BY MR. GRIFFIS

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

A. This is.

Q. It's later data?

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

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

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

Q. It was --

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

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

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

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

A. Okay. Yeah.

Q. And --

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

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

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

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

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

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

MR. GRIFFIS: Yes, sir.

THE COURT: All right. Thank you.

THE WITNESS: This data is not adjusted for

pesticides.

BY MR. GRIFFIS

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

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

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

A. In fact, they do.

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

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

A. Yes.

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

A. Right. I think --

Q. And the actual?

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

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

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

Q. Well, these bars are coming from --

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

other pesticides.

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

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

Q. Okay.

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

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

A. It does.

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

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

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

A. Correct.

Q. Okay.

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

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

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

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

MR. GRIFFIS: That's correct.

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

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

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

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

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

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

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

Right?

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

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

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

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

Q. No, sir.

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

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

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

Q. You didn't show us --

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

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

A. No. I have could have.

Q. I agree.

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

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

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

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

And this was after we went through the study.

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

And you answered, *That's correct.*

Do you remember that?

A. I believe that's correct.

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

other pesticides. Right?

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

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

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

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

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

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

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

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

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

A. I don't see any dates here.

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

A. I do. Yes.

Q. Okay.

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

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

THE WITNESS: Oh, okay.

BY MR. GRIFFIS

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

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

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

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

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

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

A. They're trying to learn. They're trying to understand the data.

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

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

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

A. That's correct.

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

A. It was from the biomonitoring study. Yes.

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

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

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

MS. FORGIE: Thank you.

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

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

A. That's what it says.

Q. Do you see that?

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

A. That's what it says.

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

A. That's what it says.

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

A. Yes.

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

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

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

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

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

Q. Yes. And I --

A. The latency is very long.

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

A. Makes sense.

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

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

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

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

A. That's true.

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

A. Yeah. I would stand by that.

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

Right?

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

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

A. I would think that's a ballpark.

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

A. Right.

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

A. Yes.

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

A. Yes.

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

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

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

A. That's correct.

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

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

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

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

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

A. Yes.

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

A. Yes.

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

A. Correct.

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

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

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

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

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

Q. For all cancers. For everything.

A. Yeah.

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

A. Yes.

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

And you a agree with that. Right?

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

Q. Recall --

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

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

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

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

A. Yes.

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

A. Recall bias is not a flaw, no.

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

A. Yes.

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

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

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

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

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

A. Correct.

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

Let's have Slide 104, please.

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

A. Correct.

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

Could you show that, please, Scott?

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

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

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

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

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

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

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

A. That's correct.

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

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

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

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

A. Right.

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

A. Correct.

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

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

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

A. Correct.

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

A. That's true.

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

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

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

A. That's correct.

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

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

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

A. That's correct.

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

A. That's correct.

Q. Okay.

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

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

A. I testified that it was probably too short.

Q. Okay.

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

Q. And the median years of exposure in NAPP --

Median years of use -- that's what you were talking about. Median years of use in NAPP was five years. Right?

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

Q. It was short?

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

Q. Slide 107, please, Scott.

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

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

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

A. Okay.

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

A. Okay.

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

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

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

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

A. Yeah, but we saw an effect here.

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

A. Well, there could be other factors involved.

Q. Other pesticides?

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

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

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

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

Q. I want to talk about something else.

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

THE WITNESS: I'm sure --

JUDGE PETROU: It's in there somewhere.

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

BY MR. GRIFFIS

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

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

MR. GRIFFIS: Yes, sir.

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

MR. GRIFFIS: One minute.

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

MR. GRIFFIS: Yes, sir.

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

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

THE COURT: When you say they included time before

the '80s, what does that mean?

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

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

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

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

THE WITNESS: In the mid '80s.

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

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

THE COURT: Okay.

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

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

THE WITNESS: Yes.

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

THE WITNESS: 1974, '75. Yeah.

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

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

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

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

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

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

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

THE WITNESS: Right.

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

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

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

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

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

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

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

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

THE WITNESS: Yes.

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

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

Did that make sense -- what I said?

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

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

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

Now I lost my train of thought here.

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

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

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

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

So in Nebraska.

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

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

Don't laugh.

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

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

MS. FORGIE: Because there are so many studies.

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

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

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

THE WITNESS: Thank you.

MR. GRIFFIS: Thank you, Your Honor.

THE COURT: Are you going to have any redirect?

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

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

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

MS. WAGSTAFF: Apologies, Your Honor.

THE COURT: No problem.

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

THE COURT: Okay.

MS. FORGIE: But thank you.

THE COURT: Thank you.

All right. Who's next?

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

THE COURT: About time.

THE WITNESS: No pressure.

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

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

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

MR. MILLER: I poured those.

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

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

MR. TRAVERS: I just have copies.

MR. LASKER: Do you have a cross binder?

MR. MILLER: Yes. Yes, we do.

MS. GREENWALD: Sure.

(Whereupon a document was tendered to the Court.)

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

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

MR. ESFANDIARY: Sorry.

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

ALFRED I. NEUGUT,

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

THE WITNESS: I do.

THE CLERK: Thank you. Please be seated.

MR. MILLER: Thank you, Your Honor.

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

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

DIRECT EXAMINATION

BY MR. MILLER

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

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

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

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

A. That's right. So --

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

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

Q. Not too fast.

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

oncology.

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

A. Yes.

Q. And that's in what?

A. Chemical carcinogenesis.

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

A. Yes.

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

A. Yes.

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

A. Yes.

Q. Do you actually treat patients --

A. Yes.

Q. -- in cancer?

Do you have clinic next Tuesday?

A. Yes.

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

A. Yes.

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

A. Many of them are.

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

A. Yes.

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

A. Institute of Medicine.

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

A. Yes.

Q. Just briefly tell us what that means.

A. I review articles for peer review for journals.

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

A. I have been.

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

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

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

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

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

A. Yes, yes.

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

A. Yes.

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

the significance of that point, please.

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

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

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

A. With great difficulty.

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

THE COURT: One question about that last slide.

I get why hazardous assessment is not helpful.

THE WITNESS: Mm-hm.

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

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

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

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

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

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

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

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

THE WITNESS: Mm-hm.

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

THE WITNESS: Mm-hm.

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

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

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

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

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

THE WITNESS: Uh-huh.

THE COURT: -- to cause cancer --

THE WITNESS: Uh-huh.

THE COUT: -- we are still assessing it as a possible or a probable or a known carcinogen --

THE WITNESS: Uh-huh.

THE COURT: -- because you never know --

THE WITNESS: In the future.

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

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

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

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

THE WITNESS: Uh-huh.

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

THE WITNESS: Mm-hm.

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

THE WITNESS: Mm-hm.

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

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

circumstances.

THE COURT: Okay.

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

THE COURT: Okay.

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

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

MR. TRAVERS: I believe the Elmo.

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

MR. LASKER: Do you have --

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

MR. LASKER: -- a copy for me?

MR. TRAVERS: We're getting some.

MR. LASKER: Okay, thank you.

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

(Discussion off the record.)

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

then?

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

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

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

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

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

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

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

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

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

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

A. Epidemiology does that's what IARC does.

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

account of realistic human exposures.

Does IARC in these bullet points reject that charge?

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

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

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

> "In addition, when considering scientific evidence of carcinogenicity, including biological mechanisms, the working group placed special emphasis on whether the observations are relevant to humans."

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

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

Q. Okay.

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

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

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

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

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

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

A. Yes.

Q. What's your opinion?

A. The answer is yes.

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

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

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

BY MR. MILLER

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

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

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

universally recognized as a source.

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

Next slide.

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

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

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

Next slide.

And even on the American Cancer Society web page, it

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

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

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

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

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

Next slide.

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

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

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

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

specificity.

Next slide.

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

Next slide.

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

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

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

television in 1980s or in 1990s?

THE COURT: Maybe.

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

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

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

those tumors.

Am I okay now?

Next slide, please.

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

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

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

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

Next slide.

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

THE COURT: Slow down a little bit.

THE WITNESS: I'm sorry.

BY MR. MILLER:

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

A. No. From '81. From '81.

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

A. No.

Q. Okay.

A. None of them in my life.

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

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

Next slide.

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

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

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

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

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

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

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

high, very more than usual.

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

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

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

Do you smoke?

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

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

So I hope that answers that question for you.

Next slide.

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

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

THE COURT: Sure.

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

THE WITNESS: Next slide.

BY MR. MILLER

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

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

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

spurious or chance findings?

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

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

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

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

A. Next slide.

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

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

THE COURT: Very briefly.

THE WITNESS: Very briefly, there is a temporality. There is consistency. There is dose-response, biological plausibility, strength of association, and specificity.

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

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

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

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

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

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

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

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

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

THE WITNESS: Usually, the control group is used.

JUDGE PETROU: Okay.

THE WITNESS: If it's a big number -- that is, if it's a very large study -- then they'll do tertiles or quartiles. So, you know, they'll compare the top quartile to the lowest quartile.

But most of these studies are not that large, so bifurcation -- you know, dichotomy -- is big enough. You know.

Less than 10 days versus more than 10 days.

But if there were a lot of them, if there was a much bigger study, you might have had less than 10 days; 10 days to 50 days; 50 -- greater than 50 days. You know. You might have had more categories; but again, these studies -- the study wasn't -- I don't think it was large enough to get that elaborate.

JUDGE PETROU: Mm-hm.

THE WITNESS: So that was done there.

And the strength of association, 1.3 to 1.5, while I think that's just still in the range of a moderate association, it's not like -- you know, it's not a huge relative risk. So I also did not give it a huge rating here.

But again, but still, all of the Bradford-Hill Criteria were -- the -- one, two, three, four -- these top five, without specificity, are the usual five. If you look at most causal associations, they go through the five that are typically used.

Without specificity -- specificity is usually not even on the list. I'm just saying that in our particular in our particular case, specificity applies. So I included it.

Next slide.

THE COURT: Could I ask you one more question about the previous slide?

THE WITNESS: Mm-hm.

THE COURT: Can we go back to the previous slide?

Thanks. So temporality is at 5.

THE WITNESS: You know, I make it easy. It's not like I have a little metric somewhere and I'm going, you know, like (indicating).

THE COURT: I understand, and normally, I gather from all of the reports and all of the testimony that temporality is not something one thinks too much about, because it's an easy question to answer.

THE WITNESS: Mm-hm, exactly.

THE COURT: But in this case, remembering about the question that I -- that I was asking earlier today --

THE WITNESS: Mm-hm.

THE COURT: -- about the De Roos Study and McDuffie Study --

THE WITNESS: Mm-hm.

THE COURT: -- which involved data collected from the late '70s and early '80s --

THE WITNESS: Mm-hm.

THE COURT: -- and there's this concern that, you know, there may be a fairly long latency period for NHL, as potentially caused by glyphosate --

THE WITNESS: Mm-hm.

THE COURT: -- and glyphosate didn't come on to the market until the mid '70s. Does that create a temporality problem with respect to those studies?

THE WITNESS: If anything, it sort of obviates the temporality issue, because it's clear then that the glyphosate predated the NHL, no?

THE COURT: Well, but doesn't it raise a concern that the NHL was caused by something that they were exposed to before glyphosate came on the market?

THE WITNESS: So that's a different question.

THE COURT: That's not a temporality issue.

THE WITNESS: That wouldn't, to me, be a temporality question. That would be a different question.

THE COURT: Where would you put that concern?

THE WITNESS: So that's a question that I think there was a discussion about latency, period. And I suppose that would then come under biological plausibility, or mechanism of action applies in terms of how glyphosate causes glyphosate causes -- theoretically, how glyphosate causes NHL.

MR. MILLER: If I could, then, Your Honor, could we switch to the Elmo?

Q. I want to ask you about the particular issue, if I could. This is from the McDuffie Study, Exhibit 21. Every study has what they call a "Materials and Methods" section. Is that right, Doctor?

A. Yes.

Q. And I think they perhaps answered this question in Helen McDuffie's study, with these other scientists, and I want

to ask you about this.

She states, on the second page, or page 1156, a whole sentence here, "but incident cases among men ages 19 and over with a final diagnosis of STSHD NHL" -- I guess that's non-Hodgkin's lymphoma?

A. Yeah, um-hum.

Q. -- "or MM" -- that's multiple myeloma?

A. Yes, sir.

Q. -- "diagnosed between September 1, 1991, and December 31st, 1994 will were eligible"?

A. Correct.

Q. So that if Roundup® came on the market in '74, then that would be 17 years --

A. Right.

Q. -- between the time, right?

A. If they were looking at exposures to glyphosate that went back to 1974, then of course, that would be more than enough exposure for those cases.

Q. All right. Thank you, sir.

THE COURT: And did you say that was the McDuffie Study?

MR. MILLER: Yes, Your Honor. That's Exhibit 21, the McDuffie Study, page 1156, which is the second page.

THE COURT: Thank you.

MR. MILLER: Yes, Your Honor.

Q. Let's go back to your PowerPoint, then. You were talking about -- you've completed your discussion on the Bradford Hill?

A. Sure.

Q. Let's move on.

A. So the only real change since IARC -- and, as I say, I think IARC did a bang-up job in terms of evaluating various questions that addressed the case-control studies -- was the changes; the recent follow-up studies and its limitations. And the question is how it affects our overall thinking with regard to what's going on. So the question is: How do we all feel about it? I mean, I know how they feel about it, and I know how we feel about it. So -- and how it may alter the conclusions of IARC.

Next slide.

So first, just to make a general statement, a well-conducted epidemiological study does not typically need imputation. Imputation is used when a major problem develops with data collection in a study, so it implies a problem. That does not mean it's a bad thing to do. It's the right thing to do, but the fact that you had to do imputation already shows that you're dealing with an issue.

Next slide.

So what's the issue? So it's really the conflation of several problems together, and let me say that I think the AHS Study, as someone else said, is actually an excellent

study. It's been very productive, and it's very good for many, many things.

We were talking very specifically about the association between glyphosate and NHL, and the problem arises from the conflation of these problems, the first of which is the figure on top, which is the extraordinary increase in the use of glyphosate that took place between -- in the late '90s, and totally changed the exposure level of glyphosate among farmers.

If we could have used the 1995, you know -- the farmers in AHS were collected between 1993 and 1997. If everyone kept using glyphosate at the same rate, look, if we asked them how much do you smoke, and you say, I smoke two packs a day, the smoker could have just kept going along using two-pack-a-day of smoking, and there would have been no change in smoking, then there would have been no problem with everything, but the fact that there was a extraordinary change in usage, so the baseline exposure rate of glyphosate exposure became totally untenable as a measure of exposure.

This was different, I assume, from any of the other herbicides, and it makes the whole glyphosate assessment necessary, therefore, to require a second interview. So without the second interview, you're screwed up.

So that leads to, then, needing a second interview in 2005. Then you have the 37 percent loss to follow. Basically, they couldn't interview 37 percent for one reason or another.

I mean, those of us to who get phone calls all of the time asking us to do a survey can totally understand this.

And then on top of that, there's the modest association in the first place. Again, if the Risk Ratio is 10, then all of these errors wouldn't really matter, because they would all be, ah, you know, they would all wash out in the mix, but because we're dealing with a Risk Ratio in the 1.3, 1.4, 1.5 range, that's very delicate, and so any -- any errors attenuate towards the null, as you keep hearing over and over and over again, and therefore, these -- the compilation of all of these errors over and over again take you way down to the null.

Then there was the 10 percent initial misclassification in the error in the first place, which attenuates towards the null, and -- and as was shown in one of the papers, when they did imputation in the first place, they had a 17 percent imputation error. So we have an imputation. Again, imputation is necessary, but imputation has an error to it, so imputation doesn't work that great. Again --

THE COURT: What was the 10 percent initial misclassification error?

THE WITNESS: That's when they asked the people in 1995 how much glyphosate they were using. It was estimated that the error in terms of their estimate of use of glyphosate was about -- I remember 10, or might have been 11 percent, which is not a bad error, as epidemiologic analyses go.

THE COURT: You mean whether they used it at all or not?

THE WITNESS: Yeah, mm-hm.

THE COURT: Okay.

THE WITNESS: But with a Risk Ratio of 1.3 to 1.5, that's almost enough by itself to wipe out a 1.3 to 1.5 Risk Ratio.

Think of it for a moment. If I give you an example, if I did the dietary food frequency questionnaire with you and I asked you how many broccoli do you eat, right? You haven't got a clue of how much broccoli you eat. How many times a week do you eat broccoli? You're going to be wrong, whatever you say. I'm going to be wrong, whatever you say. Everybody in this room is going to be wrong with whatever they say.

Somehow, it all works out when you do it epidemiologically, but the point is the error rates work out so that they all attenuate towards the null, and it's conservative, and so these misclassification errors all attenuate towards the null. So you can imagine asking someone how much glyphosate they use, a 10 percent error is actually a fairly minor error, if you think about it. It's not so bad.

But it's big enough, for a -- for a modest association, for a modest association, and that's why this conflation -- it's specifically with glyphosate and NHL that we have the problem, because -- because we have this modest Risk Ratio, and

then we have this extraordinary change over the next 10 years which, just on top of it, makes it totally impossible.

And then when they did imputation, they themselves showed it as 17 percent error in how the imputation measured the -- the estimate of the glyphosate usage, and that's --

THE COURT: Could you explain that a little more?

THE WITNESS: Yeah. They took -- so imputation is that they take all of the knowledge that they have about the cohort -- about the, let's say, the 60 percent, the 62 percent, they know the age, race, sex, many, many things about them, and they know -- they have all answered second questions, taking the 60 percent who answered a second questionnaire, and from that, they took a 20 percent random sample of the people who did answer the second questionnaire. So they know the answer to how much glyphosate they used.

Twenty percent of the people who took -- they took a 20 percent sample of the 60 percent, who -- so they know their use, on the second questionnaire, of the glyphosate.

So then they used imputation to see how well imputation estimated their answers to the second questionnaire, and when they did that, the answer was off by 17 percent, 16 percent or 17 percent. I don't want to get picky. So even the imputation mis-measured it by 17 percent.

THE COURT: So you're saying they asked the questions on the second questionnaire one time.

THE WITNESS: Mm-hm.

THE COURT: And then they asked 20 percent of those respondents the same question on the questionnaire again.

THE WITNESS: Uh-uh, no, no, no, no. They -- so there were 30,000 people -- they had 50,000 people to start with. 30,000 people answered the second questionnaire. So of that 30,000, they took 7,000 people who had answered the questionnaire, and they used imputation to estimate what their answer to the usage of glyphosate was, using the imputation methodology, to see how well they guessed or how well they estimated their use of glyphosate but they knew their use of glyphosate from -- because they'd all answered the questionnaire.

I'm not saying it well?

THE COURT: You're probably saying it well, I'm just not understanding it. What are they comparing?

THE WITNESS: These people have all answered, so --

THE COURT: They've answered the question.

THE WITNESS: They've answered the question.

THE COURT: And so what is -- what are their answers being compared to?

THE WITNESS: They're using the imputation methodology to -- so their plan is to use imputation.

THE COURT: So the methodology that's used for the people who didn't answer the questionnaire is applied to the

people who answered, and it's compared to their answers.

THE WITNESS: To see how well it guesses their answer, where they already know -- where they know the answer, to see if they get the right answer, and when they did that, the answer was off by 17 percent.

So again, that's not such a terrible answer for imputation. I'm not criticizing. A 17 percent error for imputation is not a bad guess.

Fifty-three percent versus 45 percent, I believe, is what they got, just to give you a sense of the numbers, but in terms of how that will translate later into -- into dealing with a Risk Ratio of 1.3 to 1.5, in terms of, then again, that error rate, how that will affect a Risk Ratio of 1.3 to 1.5 -- again, this is an error on top of an error on top of an error, where each error attenuates to the null.

And we haven't even discussed the problem that 20,000 people are biased. They -- you know, that's the biggest bias in epidemiology is volunteer bias. Who answered the questionnaire and who didn't answer the questionnaire? And we don't even touch that bias. I didn't even mention it here, because it was so obvious.

Next slide.

Okay, so those are the problems, and that's why I think the AHS Study is basically not so useful.

And just to give you a sense, so you can know, that a

37 percent loss to follow-up is, you know, pretty humongous.

Here's a another cohort study, happens to be from Harvard, and you can see in the line under the follow-up in this study was 94 percent, just to give you a sense of, studies do do well in terms of follow-up.

Next slide.

So, okay. So you will ask yourself, as will everyone in the room, if I'm saying that this AHS follow-up study is such a pile of -- you know, is really such -- so -- so bad, how did it get published in *JNCI*, which is such a good journal, as Mr. Lasker is going to tell us at some point I'm sure, and it got published there, peer-reviewed, et cetera, et cetera, and *JNCI* is a good study. I've had 20 papers published there, so by definition, it's a good journal, so -- and it's a perfectly valid question to ask.

So -- and I don't know the answer, because peer review is confidential. I can only speculate, based on my knowledge of peer review. Mr. Miller asked me before about my experience with peer review, I do a lot of peer review. So I can give you three possible answers, just for your contemplation.

The first -- my first possible answer is what I would call, shit happens. You know? You never know, you know?

You got yourself an easy peer reviewer, we all hope for it. I don't know how *Harvard Law Review* works but, you know, but you know you -- ah. You know, you send it in to New

England Journal, the gods can smile. Okay, I don't think that happens here.

The second possibility is, *JNCI* has been going downhill for the past five years. It used to have an impact factor which was how we measure -- one way in which we measure how good a journal is. It was 18, it's down now to about 12.

It got itself a new editor, who actually happens to be a very close friend of mine, has been trying to write itself. Here comes a paper that's going to be very, uh, pre- -- I don't know if prestigious, but get a lot of attention and bring it a lot of notoriety, and so it got itself -- it got peer-reviewed, but got an easier pass in a sense of, the editors wanted it to be published here so it would get some attention to the journal, and indeed, this past -- I got an e-mail this past year that this was one of the top 10 downloaded journals -- downloaded papers of 2017. It's, in fact, number three. One of my favorites is on the two.

THE COURT: Just because Monsanto required every single employee to download it.

(Laughter.)

THE WITNESS: So, but a third possibility is something else, which is, when you do peer review, it takes you, you know, to read the paper it takes you a half hour, 45 minutes. It's up to the authors to highlight to you what's good and bad about the paper. So here's the abstract from the

paper. Take a look at it, if you have a moment, you read through this thing. Mention the loss to follow-up? Anything about it? Imputation? Not a word.

Okay. All right. Let's move on.

Next slide.

So here's the Limitations section. This is the section I read most carefully when do peer review. I expect the authors to write an honest assessment of the weaknesses in the paper, so I can judge how good and bad -- you know, what the problems are in a paper.

This paper actually had two findings in it, one of which related to leukemia, that there was a positive association between A -- glyphosate and AML.

So every limitation -- there were three limitations given in this paragraph, all of which relate to their findings with regard to AML to leukemia.

There is not a single limitation noted in this paragraph, or in the paragraph before or after, with regard to the loss to follow-up, the use of imputation, any of the things I alluded to in my paragraph.

In a sense were they being dishonest, unethical? Who knows? I'm just saying it wasn't as open of a paper in terms of talking about its weaknesses. I'm just giving you a sense of what -- I suspect it was really the earlier point about, shall we say, the trendiness and politics that were involved.

Next slide.

So we could go through the papers again. But anyway, next slide.

BY MR. MILLER:

Q. If you --

THE COURT: Well, I actually think it, in particular, it would be helpful to go through McDuffie.

THE WITNESS: Okay.

THE COURT: And De Roos, 2003.

THE WITNESS: I didn't mean to --

THE COURT: No, no, that's okay. And one suggestion I might have is, you know, we could go -- you know, we don't --

(Discussion off the record.)

THE COURT: We don't have a hard stop at 4:00. We can go to 4:15, 4:30 whatever.

Why don't we take a little break, and then resume.

MR. MILLER: Great.

THE COURT: Why don't we resume --

THE WITNESS: You want to take a break now Your Honor?

THE COURT: Yeah, yeah, and resume at 3:30.

(Recess taken from 3:20 p.m. to 3:30 p.m.)

MR. MILLER: Well, let's go back -- I'm sorry. If we could go back to the slides?

If I could start, then, Your Honor?

THE COURT: Please.

MR. MILLER: All right.

Q. Even though the McDuffie Study was adjusted for age, province and high-risk exposure, it was not adjusted for other pesticides within the original study, right, sir?

A. No.

Q. But is it still a piece of the puzzle that you use in weighing the evidence to come to your conclusion that Roundup® causes?

A. Correct. We discussed this earlier in terms of, some of the studies did, and others said some of the studies did not.

By the way, I wanted to answer -- the judge had asked this morning, or earlier, about the hierarchical regression modeling. So I thought I'd just take one second --

Q. Please.

A. -- to answer it. So I don't think it has a direct relevance to much that we're -- that we're talking about, but it's a form of regression analysis where -- so in a logistical regression, or most regression models, we just throw in all of the covariates into one equation, sort of like the kitchen sink, so to speak, and then the computer grinds around for a while and the answers come out, and each covariate gets a Risk Ratio assigned to it, with a 95 percent confidence interval.

In hierarchical modeling, the idea is you may want to know how much does a certain class of variable affect the

association between the exposure and the outcome.

So for example, we could even say with regard to glyphosate, we want to know, how much do the demographic variables affect the association between glyphosate and NHL?

So in hierarchical modeling, they would do age, race, sex, socioeconomic status, and do a regression analysis using those covariates first, and then you'll get some kind of overall Risk Ratio, I believe, and it will say how much of the overall -- in the end, the overall association between glyphosate and NHL is due to those covariates as a group.

And then you might then put in the -- the herbicides, let say, and then you'd -- might say you might end up saying that the association between the two, that 20 percent of the effect is due to the graphic variables, 80, 70 percent is due to the herbicides, 10 percent is due to something else, you know, unexplained, or idiopathic or something like that.

So I don't know that it plays that much of a role in these studies.

THE COURT: Well, tell me why -- you know, you mentioned that you're not sure how much it matters, for purposes of our discussion.

THE WITNESS: Um-hum, because we're mostly focused on individual covariates, as a rule; glyphosate in particular.

I don't know that the hierarchical modeling is that much of a an issue in this.

THE COURT: Well, maybe it's something, you're going through the studies, and maybe we when we get to De Roos, we can --

THE WITNESS: Yeah tell me -- if it comes up --

THE COURT: -- we can talk a little bit more about that.

THE WITNESS: If it comes up, by all means.

THE COURT: And so there was a McDuffie slide up just a second ago.

THE WITNESS: Mm-hm.

THE COURT: And so when it says on the slide, adjusted for age, province, high-risk exposure, what is high-risk exposure? That's just greater exposure to glyphosate?

THE WITNESS: Mm-hm. Uh, I don't recall what I mean by that, so I'm going to have to --

BY MR. MILLER

Q. Would that be found in Table 1, sir?

A. In Table 1? I think it's just talking about folk that are high risk, things like medical conditions and things of that sort, but I'm not a hundred percent sure.

THE COURT: Okay.

THE WITNESS: It's not referring to herbicides or anything of that ilk.

BY MR. MILLER:

Q. Oh, all right. So, and in --

A. You'll recall that it's separated by the two-day -- lesser than two days versus greater than two days.

THE COURT: And then if I could ask --

THE WITNESS: Mm-hm.

THE COURT: -- I guess you've answered the question about the McDuffie Study, and when the incidences of NHL were. Right? You said it was, like, '91 to '94, or something like that, people were diagnosed with NHL?

THE WITNESS: That's when the cancers occurred, when they were diagnosed, yes.

THE COURT: When they were diagnosed, okay.

THE WITNESS: Mm-hm, yes. You know, you always have to -- you can only take newly diagnosed patients into a case-control study. That's, like, a rule of epidemiology.

THE COURT: Okay, and then what about -- an issue that I don't think you've had a chance to talk about yet is --

THE WITNESS: Mm-hm.

THE COURT: -- proxy responders.

What -- does -- is the issue of proxy responders a concern for the McDuffie Study?

THE WITNESS: I don't recall, offhand, how many proxy responders there were, if there were, but as long as -- to me, the solution of proxy responders would be that you keep them

equally distributed between cases and controls, so you get a similar -- you eliminate the bias, or whatever problems there were in terms of error, or in terms of -- that a proxy responder would introduce, but --

THE COURT: If you -- if you had an equal number of or roughly equal number of proxy responders for the cases and for the controls, would that eliminate the concern --

THE WITNESS: Yes.

THE COURT: -- about proxy response bias?

THE WITNESS: I mean, you can always worry, but the answer would be, that would be the solution to the problem, yes.

THE COURT: Okay, and you don't recall, as you sit here, whether there were any issues --

THE WITNESS: No.

THE COURT: -- relating to proxy response.

THE WITNESS: No, and you know, NHL is a pretty well -- people live -- you know, we're not talking about pancreas cancer. There, you end up oftentimes with almost everyone having a proxy respondent, unfortunately, but in NHL, I would say that, you know, that's not the case.

And I would say, if you wended up here with a lot of proxy respondents, you might have problems, because occupational exposure, particularly if we're relying on self-report, you really do want to have the person, to the greatest degree

possible, give an answer.

BY MR. MILLER:

Q. If I could, on that issue, I want to ask you, from the McDuffie Study, the Materials and Methods on the second page, it lists two points, if we could switch over, and I'll quote. "After position -- "

MR. LASKER: What page?

MR. MILLER: Excuse me? We're on page --

MR. LASKER: Page what?

MR. MILLER: -- 1156 of the McDuffie, top of the corner, here.

MR. LASKER: Thank you.

MR. WISNER: There's this weird thing on the screen, so you have to do it up on the corner. There's this thing that's blocking it.

MR. MILLER: Excuse me.

Q. This is in the Materials and Methods, and I apologize for that, but,

> "After physician's consent was received, postal questionnaires and informed consent forms were mailed to potential cases. Surrogates for deceased cases were not contacted."

And to put that in context of this point that Your Honor's raised,

"Surrogates for deceased persons were ineligible as controls."

A. Right.

Q. All of the participating control subjects were used in the statistical analysis of each cancer site.

I don't understand what it means, but I think it addresses this --

A. Basically saying they didn't have proxies in this study.

Q. Oh.

THE COURT: Okay.

MR. MILLER: Unless Your Honors have any more questions on the McDuffie Study....

Q. Do you have anything else that you feel you need to share on that.

A. No.

Q. Do you want to move to Hardell?

A. Mm-hm. So Hardell was the Swedish study, and it -- I think it also did not --

THE COURT: It didn't control for pesticides, right?

THE WITNESS: Right, it did not control for herbicides, but came up with an elevated Odds Ratio that was not statistically significant.

BY MR. MILLER

Q. Well, and then I have a few questions, and I don't know the answer, so I probably shouldn't ask it, but you used the

1.85 Odds Ratio for non-Hodgkin's lymphoma, and I understand why, and the univariate analysis, I think they call it, it was actually 3.04, but that's without adjusting. Is that what that is?

A. Yes.

Q. That's why you chose not to use it? I'm just asking.

A. I don't use univariate analyses.

Q. I understand. I understand. So we go over, then, to the one that you did use, from Table 7, and that's what's referred to as a multivariate analysis. Is that the one that you selected?

A. Mm-hm.

Q. Yeah, and so --

A. So the analysis that we used --

Q. Yes, sir?

A. -- and that's the figure that, you know, in the subsequent -- that would be the figure that I would you use for a forest plot or for anything else, to think about, in terms of considering causal association here.

THE COURT: So that 1.85 Odds Ratio does reflect adjustment for pesticides.

THE WITNESS: Yes. Oh, in Hardell? Let me make sure. After a while, all of these guys....

THE COURT: I thought I may be wrong about this, but I thought multivariate analysis sort of equals adjusting for

other pesticide use, but maybe that's wrong.

THE WITNESS: It's equal to adjusting for anything. It means that you adjusted for age, race, sex, whatever.

But I believe in Hardell it does reflect -- I apologize, Your Honor.

BY MR. MILLER:

Q. It doesn't appear as though they let us know, does it, Doctor?

A. Yes.

Q. Yeah.

A. It adjusts for herbicides.

Q. Oh, it did?

A. Mm-hm.

Q. Okay. All right. Now, before we leave Hardell, is that the -- it's based on the Swedish Population Base, or something?

A. Yes.

Q. Swedish?

A. Yes.

Q. Okay. All right, anything else about that study --

A. No.

Q. -- they we need to --

THE COURT: What about use of surrogates in that study?

THE WITNESS: Use of...?

THE COURT: Surrogate respondents.

THE WITNESS: I don't -- I don't believe they did, no. Oh, no, I'm sorry, they used next of kin. I apologize. They used next of kin.

THE COURT: Do you know for what percentage of respondents?

BY MR. MILLER

Q. From the Materials and Methods section, Doctor, the NHL study encompassed males greater than 25 years with NHL diagnosed during '87 to '90 and living in the foremost northern counties of Sweden and three counties in mid-Sweden. They were recruited from regional cancer registries, and only cases with histopathologically verified NHL were included. In total, 442 cases of these 192 were deceased. Would those be proxy responders then?

MR. LASKER: Mr. Miller where are you reading from?

MR. MILLER: Excuse me, I'm on page 1044.

Q. Does that mean proxy responders of 37 percent, 192 dead people?

A. As I'm sitting there, I'm not seeing how many proxies. Let me take one.... They don't say specifically.

Q. Look at the Control section, if you would, Doctor. It says, for each deceased case, two deceased controls matched.

A. No, I didn't see that.

Q. Oh.

A. It doesn't say how many.

Q. What's the significance of that?

A. I can't find anywhere where it says how many deceased cases there were.

JUDGE PETROU: Two.

MR. MILLER: How many deceased cases?

THE WITNESS: You found it?

THE COURT: He was pointing it out to you earlier, down in the lower left.

THE WITNESS: I'm sorry.

BY MR. MILLER:

Q. It's on the screen, Dr. Neugut.

A. I'm really being dense, huh? Four -- oh, of these, 192 were deceased.

Q. Yeah.

THE COURT: So does that mean that 192 of the responses were from proxies?

THE WITNESS: Yeah, that's correct. So 192 out of 442. So that's roughly, a little -- almost half.

THE COURT: And how big a deal is that?

THE WITNESS: Again, from an epidemio- -- from an epidemiologist's point of view, what's important is that the -- it introduces error, but the error is balanced between the two groups. So it's unbiased error. But it does introduce some error, and I would say it's a bit of a concern.

In other words, how well does your -- I'll assume his wife

knows your use of glyphosates, you know? So my guess is that she's going to underestimate -- well, who knows how she's going to know about it, more or less, you know.

So it introduces some error; but again, error attenuates. So again, the error here will attenuate towards the null.

So the estimate that you're getting in Hardell, if we're talking about purely the error from having proxies, I would say, would therefore have attenuated the risk estimate. Because again, we're introducing what I would consider to be essentially random error. Like, we're going to have misclassification error in the estimate of the glyphosate usage, both in the cases and in the controls, but theoretically it will be balanced between two groups.

THE COURT: Would that -- I saw that there was a wide confidence interval --

THE WITNESS: Mm-hm.

THE COURT: -- for the Hardell Study.

Would -- is that -- does the confidence interval incorporate things like use proxies? I mean, is that why you will see a wider confidence interval?

THE WITNESS: Possibly, yes.

THE COURT: Do you know if that's the case in this study?

THE WITNESS: I wouldn't know offhand, but it also may have lowered the risk estimate, as well.

THE COURT: If it was non-differential?

THE WITNESS: If it were non-differential, yes. So the 1.85 and 0.3, again, if we're in Table 7 with the 1.85 Odds Ratio and the 0.55 to 6.20, maybe if you didn't have proxies, you would have had a higher risk estimate, and a statistically significant observed association, potentially.

BY MR. MILLER:

Q. With strength in the power?

A. Well, all errors are conservative, or that theoretically, all unbiased errors are conservative.

Q. I've run way out of my time. Each lawyer gets amounted so much. So I want to go over what's important, Doctor, and I know you want to go back east, but tell me about De Roos, any significant things that the Court wants to hear about De Roos, and Eriksson, and then I'll leave you.

A. So De Roos, okay. That's the midwestern study, and it did control for the use of the other herbicides, and it did come up with a statistically significant finding.

I don't know if there's much less else to say, unless the Your Honor has another question.

THE COURT: This was the one where there were different Odds Ratios for the logistic regression analysis and the hierarchical regression analysis.

And so the first question is: Is this number associated with the logistic regression analysis or hierarchical?

THE WITNESS: No. We used regression because I think it's a more legitimate -- first of all, it's more consistent. Everybody -- all the other studies use logistic regression. So first of all, to be consistent across all studies, we certainly use logistic regression sort of being consistent. Hierarchical regression modeling is a fancy-schmancy, sophisticated thing you do to look cool, you know.

THE COURT: Well, so can you now try and explain the difference between the two, to me?

THE WITNESS: I was afraid you were going to say that.

Do you know which table has the hierarchical? Oh. Oh, I see. Here we go. So --

BY MR. MILLER:

Q. All right, let's go to Table 3, Doctor.

A. Table 3? Oh, I was on Table 5. I see, okay.

Q. And on Table 3 -- and I'll start with the top -- they show effect estimates for use of specific pesticides and non-Hodgkin's lymphoma incidents, adjusting for use of other pesticides, and they have logistic regression, and hierarchical -- I never say that right -- regression.

Going down to the list of herbicides --

A. Mm-hm.

Q. -- you see glyphosate. 2.1, statistically significant, under logistical, and 1.6, outside of the statistical

significance for hierarchical, if I'm pronouncing that right.

What's the significance of all of that, sir?

A. So I'm going to have to say even I can't figure out the Table 3 in terms of why, for example, one logistical regression comes out at 4, and the other one comes out at 1.8, or something like that. Its -- statistical level of analysis. Well, Dr. Ritz could have handled that one better than I can.

THE COURT: Okay.

BY MR. MILLER:

Q. All right, fair enough. Anything else that you want to say about De Roos --

A. No.

Q. -- other than, it is a piece of the puzzle on which you formed your opinion?

A. Yes.

MR. MILLER: Any other questions the Court might have about De Roos?

Last study, and --

THE COURT: Maybe, yeah, I do have one more question about De Roos, and it's the question about when people were diagnosed with NHL from these pools, and whether the potentially short period of time between the time glyphosate came on the market and the time these people were diagnosed with NHL affects the analysis.

THE WITNESS: You mean, because glyphosate came on

the market shortly before the diagnoses were made?

THE COURT: You told me that with McDuffie --

THE WITNESS: Mm-hm.

THE COURT: -- people were diagnosed between 1991 and 1994 --

THE WITNESS: Mm-hm.

THE COURT: -- with glyphosate coming on the market in the mid '70s.

THE WITNESS: And some of these got diagnosed in the '80s.

THE COURT: That's what I thought. So I was going to ask you --

THE WITNESS: Mm-hm.

THE COURT: -- I thought that some of these folks were diagnosed in the '80s or maybe even the early '70s. I may be mis-remembering that part of it.

THE WITNESS: Mm-hm.

THE COURT: How big of a deal is that? How big of a concern is that?

THE WITNESS: So it depends on what is considered latent, that the Court was talking about before, the latency period -- the latency peered between glyphosate and NHL to be, so which is part of really what the Doctors Portier, probably, and Weisenburger referred to one that I've written; the authority refers to the mechanism of action of glyphosate on --

on carcinogenesis, or lymphogenesis here.

You know, from my point of view, they're all called "promoters" and "initiators." Promoters can enhance the probability of cancer occurring, even if they don't occur way back at the beginning, you know, where it's -- the process of carcinogenesis takes a decade or more, but we don't have to be talking about an agent which is at the very beginning of the process. We can talk about an agent which acts in a middle of the process, which is a very common phenomenon.

Most of the cancer-causing agents that we talk about in daily life actually don't occur at the beginning of carcinogenesis; they act in the middle of carcinogenesis or near the end of carcinogenesis. That's why we can act on them for prevention.

When we talk about obesity causing breast cancer, there would be no point in losing weight if it acted 30 years ago, because, then, what would be the point? But it acts near the end of the cancer-causing process.

So similarly, glyphosate could or could not be acting near the end of the causation of lymphoma to be enhancing, let say, you may already have cells or partially developed, it's partially along the way towards becoming lymphoma cells, and glyphosate somehow causes them to proliferate faster.

THE COURT: But -- and I understand that.

THE WITNESS: Mm-hm.

THE COURT: But if you're doing the study of people who were diagnosed with NHL, say, in the mid '80s --

THE WITNESS: Mm-hm.

THE COURT: -- and glyphosate came on the market in late '70s --

THE WITNESS: Mm-hm.

THE COURT: -- wouldn't that be a major concern with the study? I mean if the study did not take that into account in a major way, wouldn't that be a very significant concern to the study?

And I don't know whether De Roos took it in to account or not, but if it didn't, wouldn't that be -- wouldn't that be a major concern?

THE WITNESS: So from an epidemiologist's point of view, to some degree -- and I'll say this, and it's sometimes a criticism of epidemiology, epidemiology operates in a black box. It looks at, you're exposed, you get cancer. What happens in between, I'm not sure. I'm looking to see if there's an association between the two statistically.

Now, the process -- now, if you ask me, did the cancer -- did the five years affect it? I can do things, as follows.

I can say, I'll eliminate five years after exposure, and see if there's an increase. If there's still an association between -- if there's an association between the two, I'll say I'll take out all of the cases that there are in the first five

years, and see if I still see an association. That will tell me if -- if the latency is five years, or not.

So we can play statistical games or, you know, thought processes to investigate what the latency is, and we do that all the time. I don't know if that was done by De Roos or by the others, in terms of looking at it. They don't report it in here. So it's hard to know what it is.

If you asked me how long -- if there are studies which show that lymphomas occur in less than five years after exposure to a carcinogen, my answer is yes, there are, but I don't know if they're of the same mechanism of action as glyphosate. So I -- you know, it's possible yes, possible no.

So for the latency period is --

THE COURT: But if you have reason to believe that the latency period is 10 years, let's say --

THE WITNESS: Mm-hm, approximately, yes.

THE COURT: -- wouldn't that be a massive mistake not to do something along the lines of what you're talking about, that is --

THE WITNESS: Or I'd be concerned at least about the first five years of the cases.

I mean, if De Roos was collecting cases over a -- I don't know over a how many year period, then I at least want, over the first few years, I'd be concerned about that. That would be true.

THE COURT: Okay, thank you.

BY MR. MILLER

Q. And that concept of causing cancer later in the cell process, is that tumor promoter theory?

A. Yeah, mm-hm.

Q. And last point, I'm done. On the -- when they -- and we call it De Roos. Dr. Weisenburger --

THE COURT: I'm sorry to interrupt, one other question about that.

On the point you were making about the agent acting sort of in the middle of the process rather than the end --

THE WITNESS: Mm-hm.

THE COURT: -- is that a potential response to a criticism of the AHS Study as well? Do you get my question?

THE WITNESS: The AHS cohort study.

THE COURT: Yeah. In other words, there's this criticism of AHS that we only have 18 years of follow-up, and the latency period may be quite long for NHL --

THE WITNESS: Oh.

THE COURT: -- as caused by glyphosate, perhaps, as suggested by the Eriksson Study. So what you just said, isn't that a potential response to that criticism of the AHS Study?

THE WITNESS: Yeah. I don't think 18 years is not -- I don't know if 18 years is -- I don't want to -- I don't know if 18 years -- 18 years is not that short.

You know, I mean, you know, depends on how long you think the process of carcinogenesis takes. I mean, that's almost two decades.

THE COURT: But the concept of glyphosate acting in the middle of the process in carcinogenesis --

THE WITNESS: Mm-hm.

THE COURT: -- that would apply to the folks in the -- who are being studied in the AHS Study as well as potentially the folks who are being studied in De Roos, right?

THE WITNESS: I would assume. Mm-hm, yes.

THE COURT: Okay. All right.

THE WITNESS: And if, indeed, that is what happens.

THE COURT: Right.

THE WITNESS: I'm speculating.

BY MR. MILLER:

Q. Last point, and I'm going to sit down.

So in the De Roos/Weisenburger Study of '03, in the Methods section, they tell us that the earliest diagnosis was in July of '83, and if glyphosate came on the market in seventy --

THE COURT: Sorry, which study are you talking about now?

MR. MILLER: De Roos '03, Your Honor. Thank you.

THE COURT: Thank you.

BY MR. MILLER:

Q. And the Methods section tells us that the first diagnosis was in July of '83, so that if Roundup® came on the market in '74 that would be nine years between the first introduction of Roundup® and the first diagnosis.

A. Yeah, mm-hm.

MR. MILLER: I've been handed something else. All right. Thank you very much. I yield the witness.

THE COURT: Okay. Do you want to -- I mean, we're happy to go on. You want to -- I don't know how long you have with this witness, but.

MR. LASKER: Not long. I don't know that I'll finish, but I can get started, certainly.

THE COURT: Sure. Of course.

MR. LASKER: And how do you turn this on? What -- I'm sorry, what --

(Discussion off the record.)

THE WITNESS: But don't I get the other guy?

MR. LASKER: I think Mr. Miller wants to correct his question on De Roos.

MR. MILLER: The first page started at 93, but my co-counsel is telling me I needed to go to the second page, to see where I now -- here it is.

All right, let's go. I want to make sure I'm not -- yes, I stand corrected. It was from '81, in Iowa, and Kansas, it

went it went back to '79.

I think that's all of it, right?

MR. WISNER: Yeah.

MR. MILLER: I just wanted to make sure I didn't misspeak. I thank you. I apologize.

THE COURT: And Dr. Neugut, you are free to tell Mr. Lasker to slow down.

(Laughter.)

MR. LASKER: This is going to be -- we'll finish the cross. It will take us about five minutes. I do have 35 pages here of questions.

CROSS-EXAMINATION

BY MR. LASKER

Q. Dr. Neugut, just to clarify that final point that Mr. Miller made, because we have diagnoses starting in 1979 and 1980, in the De Roos Study, we have cases there where the maximum conceivable latency could be -- would be five years; and that's only if the farmers started using glyphosate the minute it came on the market. Correct?

A. Yes.

Q. And we could have -- of those cases, of course, some of them would not have been using glyphosate on Day One. Correct?

A. Of course; but again, we're talking about -- talking about a sample size of 3,400. So we're talking about, I'm assuming a small subset of the totality. So I don't know how important

this is in the overall analysis of the findings of the study, and the risk estimates, and -- et cetera, et cetera, but you're accurate in terms of what you're saying.

Q. Well, we actually can tell if we go to Table 2, which is on the fourth page.

And do we have this -- which exhibit number are we, just so the record's clear? I have Exhibit 720. Is that our Exhibit number?

MR. KALAS: Right, yeah.

BY MR. LASKER

Q. So it's Defense Exhibit 720.

A. Table 2.

Q. Yes, Table 2?

A. Mm-hm.

Q. And we have here, actually, the breakdown for each of the states; how many of the cases and controls that they contributed to this study. Correct?

A. How many cases are from each state? That's what you're saying?

Q. Right, yes. And the vast majority of the cases and the controls in this study came from either Iowa, Minnesota, or Kansas. Correct?

A. Yes.

Q. Somewhere in the vicinity of 80 percent of the study. Correct?

A. Yes.
Q. And if we then go back to our Methods section to find out when those individuals were diagnosed, it's on the second page, in the top left-hand column. For Iowa, the diagnoses were 1981 to 1983. Correct?
A. Yes.
Q. For Minnesota, it was 1980 to 1982. Correct?
A. Yes.
Q. And for Kansas, it was 1979 to 1981. Correct?
A. Yes.
Q. So for 80 percent of this study, the maximum period of use would have been within a range of between four to eight years, as far as the maximum latency possible, if every one of those people started using glyphosate on Day One. Correct?
A. Yeah. But as I say, I'm not saying I necessarily think that the latency period is five years, or less than that. I didn't define the latency period.
Q. And you talked about this possibility of doing a lagged analysis.
A. Mm-hm.
Q. And epidemiologists would do a lagged analysis in this situation to try and parse out if this is a problem. Correct?
A. I -- so I don't know if -- yes.
Q. And in fact, there is no lagged analysis that was conducted in the De Roos Study. Correct?

A. No.
Q. It's correct that they did not do a lagged analysis?
A. It's correct that they did not do one.
Q. Okay. Now, Dr. Neugut, I want to go back to where Mr. Miller began. Mr. Miller asked you whether you've used the same intellectual rigor to reach your expert opinion in this litigation as you use in your ordinary course of work; and you said "Yes." Correct?
A. Yes.
Q. And he said that you reviewed a lot of stuff to reach your conclusions. Correct?
A. Yes.
Q. And your conclusion was that, to a reasonable degree of medical certainty, glyphosate causes non-Hodgkin's lymphoma. Correct?
A. Yes.
Q. And, in fact, you reached that opinion in this litigation before you had read any of the glyphosate epidemiologic studies. Correct?
A. That's correct. After I read the IARC Monograph, and I don't know if I didn't read any studies, but not -- I may not have read all of the studies at that point.
Q. Well, let's go to -- and it's at Tab 2 of the binder.
And do we have an exhibit number for this?
MS. LYNHAM: It's 1523.

MR. KALAS: Twenty-three.

MS. LYNHAM: 1523.

MR. LASKER: This will be Defense Exhibit 1523. I'm sorry.

Q. And this is a declaration that you signed under oath, and submitted in another case in this litigation. It wasn't in this court. And you submitted this declaration on -- in April of 20- -- or you signed it, anyway, in April of 2016. Correct?

A. Yes.

Q. And in this declaration, you state your opinion, it's paragraph 7.

> "It is my opinion to a reasonable degree of medical certainty that glyphosate does cause non-Hodgkin's lymphoma in humans."

Correct?

A. Yes.

Q. And in your deposition in this case, I asked you if your review of the actual underlying epidemiologic studies took place after April 26, 2016, after your declaration, and you stated yes. Correct?

A. Yes.

Q. And that was true testimony, correct? Under oath?

A. What was my testimony?

Q. It's right up on the screen. The actual review of the

underlying studies epidemiological studies would have taken place after your April 2016 declaration. Correct?

A. I won't say I didn't read any of them. I read some of them before and some of them after.

Q. You want to say that now, but when I asked you that question in your deposition, you said that. Correct?

A. When I read the monograph, I did refer back to some of the epidemiological studies, but it's correct that I did not do a complete review of all of epidemiologic literature at that time.

Q. In fact, for this declaration, you based your opinion -- an opinion that glyphosate, to a reasonable degree of medical certainty, your opinion that it causes non-Hodgkin's lymphoma was based upon your review of the IARC Monograph. Correct?

A. In most part, yes.

Q. And you stated in your testimony earlier that you view IARC, when it says "probable," that means 70 percent or 80 percent. That's what IARC means by that. Correct?

A. That's my opinion.

MR. LASKER: Okay. And do we have the IARC preamble?

MR. KALAS: Yes.

(Whereupon a document was tendered to the Court.)

MR. LASKER: And I'll wait for it to get up to Your Honor. It's Defense Exhibit 1049. Do you have the preamble?

(Discussion off the record.)

MR. LASKER: It's also a Plaintiff's exhibit, but -- and I'm going to put this up on the Elmo, if I can figure out how to work it.

MR. GRIFFIS: You want the actual preamble up?

MR. LASKER: Yes, if we could do that. Do you have page 22?

THE COURT: Page 22 of the preamble, you said?

MR. LASKER: Yeah, page 22 of the preamble, and for Your Honors, it's the paragraph Group 2, and in particular, where we're talking about lines 29 through 32, and you can see that if we can get that out of range off there.

THE COURT: I think you just have to wait. It will disappear in a couple of seconds.

BY MR. LASKER:

Q. Okay. So Dr. Neugut, if you can read on your screen, this is from the IARC preamble, and we're on page 22 lines 29 through 32.

What IARC states about the word "probable," the terms "probably carcinogenic" and "possibly carcinogenic" have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with "probably carcinogenic" signifying a higher level of evidence than "possibly carcinogenic." Correct?

A. Yes, what I stated was it was my opinion that when IARC

states that something is "probably carcinogenic," that its probability of being carcinogenic is promptly in the 70 to 90 percent range.

Q. I understand, and that's the opinion you have, which was the basis for you being able to reach a decision prior to reading and doing a review of the epidemiological studies. What happened was --

A. I wasn't referring to glyphosate specifically.

THE COURT: You've got to let Mr. Lasker finish his questions.

BY MR. LASKER

Q. Your understanding -- not what the preamble states, but your understanding of what "probably carcinogenic" means in IARC's terminology was the basis for you to reach an opinion to a reasonable degree of medical -- of scientific certainty, and signing it and submitting it in court as your expert opinion before doing a review of the epidemiologic studies. Correct?

A. Correct, but the -- if I go back to this, this says it is my opinion to a reasonable degree of medical certainty that glyphosate does cause non-Hodgkin's lymphoma in humans. I believe the probability that goes with medical certainty is 51 percent, and I'm willing to stand by the statement that I wrote on 4/28/16.

And in fact, if IARC had made it a limited, to be, based on the evidence that I read in the -- I think in the monograph

alone, without reading any other scientific literature, I probably would have been -- in fact, I would have been willing, in fact, I was willing to sign this statement.

Q. All right. Just so I'm clear, then, if IARC was to classify -- had classified glyphosate as just "possibly carcinogenic," and you were to review that classification alone, with nothing more, based upon your methodology, that would be sufficient for you to come into court --

A. I'm not --

Q. -- and testify to a reasonable degree of medical certainty that glyphosate can cause non-Hodgkin's lymphoma in the humans. Is that correct?

A. That is not what I said.

Q. Okay. Well, we'll have to look at the record on that.

Dr. Neugut, when you subsequently reviewed the epidemiologic studies for purposes of your Expert Report in this case, you also sought to adhere to IARC's guidelines as to how those studies should be considered. Correct?

A. I didn't hear you.

Q. When you subsequently reviewed the actual epidemiologic studies to be able to draft up your Expert Report in this case, you sought to adhere to the IARC preamble and the guidelines as to how that data would be considered by IARC. Correct?

A. Yeah.

Q. And because of that, for example, we've heard testimony

from all the experts -- two experts who have spoken previously about the NAPP Study. The N-A-P-P Study. Correct?

A. Yes.

Q. And the NAPP, as we've heard, is a pooled analysis that pools together all of the case-control studies in the U.S. and in Canada, which includes the McDuffie Study and the De Roos 2003 Study that you talked about earlier. Correct?

A. Yes.

Q. And you would agree that once you've pooled those studies into a larger study, it is the later pooled study that provides all of the data relevant to causation. Correct?

A. I don't know. I never read the NAPP Study.

Q. Well, first of all, I'm just asking you the question, and I want to see if you agree, and this is what you testified -- and actually, why don't I just put this up. It is Slide 61 from your deposition, your first deposition, page 228, 17 to 21.

It is fair to say that once you pool those studies into a larger study, it's the later pooled study that provides all of the data relevant to a causation theme. Correct?

A. Well, this is what I said.

Q. Yes.

A. If that's what I said, that's what I said.

Q. But because you're following IARC's methodology, despite the fact that every other expert epidemiologist in this case

has talked about the NAPP Study, talked about the ability to adjust for confounding pesticides, how they did that, and has talked in detail about the findings of that pooled analysis and what it shows, you have not read this study at all. Correct?

A. No, that's not accurate.

Q. Have you read the study?

A. No, but it's not because of the reason you gave.

The other two experts, if you're referring to Weisenburger and Ritz, were both involved with the NAPP Study. I have no involvement with it, number one. Number two, I wasn't present when the NAPP Study was presented. No one sent me to Brazil, and I wasn't in Montreal.

This is an abstract, and I don't -- it's because it's an abstract that I don't review it, not because of -- it's not a peer-reviewed publication. It's not a publication.

So that's why I haven't included it in my thinking or analysis. I have no ability to review the full dataset. Even today, Weisenburger said that he didn't have the full data from this study available. How would I have the full data from the study available? And he's a co-author, on the study.

Q. Dr. Neugut, did you -- you could have reviewed -- in our deposition, I showed you there were slide decks of data from the NAPP. Correct?

A. Which is what he had today.

Q. And we walked through some of those findings. Correct?

A. And --
Q. Well, you could have, in your analysis, looked at that data. It's not just an abstract. Dr. Ritz testified that this is a peer-reviewed presentation that's been presented at several scientific conferences. You could have reviewed the data.
A. Well, there's a difference between an abstract and a publication. An abstract is about 20 lines long, versus a full publication, and I can choose not to review a full publication. If you think I'm being nitpicky, then so be it.
Q. Dr. Neugut, do you know what data exists and what data the other experts in this case have reviewed for the NAPP?
A. No.
Q. And at no point in your very thorough review, using your same intellectual rigor as you used in trying to come up with an answer to this question, despite knowing that that data existed, and you decide, *I'm not going to even look at it to decide whether it's reliable or not*. Correct?
A. Would I have -- so would I have applied it in my academic rigor in my daily life in academia? I'm not sure that I would have. So I apply those standards, as well.
Q. Now, let's talk a little bit more about that. You would agree that IARC -- the IARC criteria are used to reach a public health determination. Correct?
A. Yeah -- and I didn't hear the question.

Q. I'm sorry. You agree that the IARC criteria are used to reach an assessment for a public health determination. Correct?

A. Yes.

Q. And when you are conducting an assessment of the epidemiological literature for a scientific assessment of a potential causal inference, you might use a different methodology. Correct?

Let's put this up. You have already answered this question. That was at 261, line 17 of your deposition, and I asked you that exact question.

> **"QUESTION:** When you were conducting an assessment of the epidemiological literature to reach a scientific as opposed to a public health conclusion, you might have a different methodology."

And your answer was, "Possibly."

Correct?

A. Possibly is possibly.

Q. Now, an epidemiologist following a scientific method would be formulating hypotheses; then testing those hypotheses to see if they could be validated, and then testing them again to see if they would be replicated. Correct? That's the scientific method.

A. Yes.

Q. And epidemiologists can design epidemiologic studies to test a hypothesis. Correct?

A. Yes.

Q. And hypotheses at issue in this proceeding is whether glyphosate or glyphosate-based herbicides can cause non-Hodgkin's lymphoma. Correct?

A. Yes.

Q. Some epidemiologic studies, though, report out a number of different potential associations relating to different exposures where they do not have a preset hypothesis. Correct?

A. So that's still not -- are you arguing that that's not scientific method?

Q. Well, I just want an answer to my question, first.

It is true that there are epidemiologic studies that report out a number of different potential associations relating to different exposures where they don't have a preset hypothesis. Correct?

A. Of course.

Q. And those studies are referred to as exploratory studies. Correct?

A. Hypothesis-generating, yes, mm-hm.

Q. Exactly. Exploratory studies are not actually testing hypotheses, they are generating additional hypotheses. Correct?

A. Yes.

Q. And the McDuffie case-control study was an exploratory study. Correct?

A. Which one?

Q. McDuffie.

A. Off the top of my head, I don't know, but --

Q. Well, I asked you this in your deposition. Let's put this up: Page 214, 11 to 23. And I asked you whether or not McDuffie's study with respect to glyphosate was an exploratory study. And your answer was, "Yes, that's correct." Right?

A. Okay.

Q. And the Eriksson Study -- we'll be talking more about that probably tomorrow -- that also was an exploratory study. Correct?

And let's put up Slide 14. This was your deposition testimony at 267, 8 -- lines 8 to 20.

The Eriksson Study, like McDuffie, is an exploratory study. Correct?

A. Well, as I sit here, now if the McDuffie Study already generated the hypothesis, then how can the Eriksson Study be exploratory?

Q. Well, I'm asking you, because I asked you this in your deposition, and in your deposition, I asked you if Eriksson was exploratory, and you said yes. Correct? It's right up on your screen.

Right now the only question is whether you stated that in

your -- testified to that in your deposition in this case.

A. Well, if that's what I said, then that's what I said, but then -- now, if the hypothesis was already generated by one of them, then the second time around, it's no longer hypothesis-generating.

Q. We also talked about the Cantor Study, and that was a study that we just looked at as one of the main contributors to the De Roos Study. I think it was actually about 60 percent of the De Roos Study. And Dr. Weisenburger testified earlier that all of those studies were basically the same design.

I asked you whether that -- those U.S.-based control studies were exploratory studies. And again, you said, yes, those were exploratory studies. Correct?

And we can put up Slide 15, talking about the Cantor Study, which was, I think -- let me get this wrong -- someone help me -- Iowa and -- hold on a second -- it was the Iowa and Minnesota portion of the De Roos Study, which is 67 percent of cases.

And again, all of the U.S.-based case-control studies, per Dr. Weisenburger, with same design, you'd agree with that. Correct?

A. Yes.

Q. Okay, and for the Cantor Study and the U.S.-based control studies, those are also exploratory studies. They are generating hypotheses, but they're not testing hypotheses.

Correct?

A. Yes.

Q. Now, let's talk about, when you have an epidemiologic study that is designed, unlike these case-control studies, to test a causal hypothesis, you would not label an exposure as being associated with an outcome, unless there was a finding of an increased risk that is statistically significant. Correct?

A. Say that again.

Q. You would not label an exposure as being associated with an outcome, unless there is a finding of increased risk that is statistically significant. Correct?

Let's put up Slide 16, because I asked you this exact question.

A. We've been talking all day about how you can have non-statistically significant associations.

Q. I understand that you testified here in court today and at your deposition when I asked you this question:

> **"QUESTION:** You stated you would not label an exposure as being associated with an outcome unless there was a finding of increased risk that is statistically significant, correct?"
>
> And you said, "That's correct."

That's your testimony. Correct?

A. You're right, but the point, though, is that there is flexibility in terms of interpreting non-statistically

associations, as well.

Q. And another thing that we talked about is that even when you have statistical significance, that does not answer the question about whether or not a study has issues of bias and confounding. And there's been a lot of discussion about that in this hearing. Correct?

A. Of course.

Q. And particularly here -- I believe Judge Chhabria raised this earlier -- there is evidence of an increased risk of non-Hodgkin's lymphoma in farmers that existed prior to the introduction of glyphosate. Correct?

A. Yeah.

Q. And we know, because of that, that there is something going on with farmers and their exposures that is leading to an increased risk of non-Hodgkin's lymphoma that we know for a fact is not glyphosate. Correct?

A. Well, we don't know why it is.

Q. We know it's not glyphosate, though. Correct?

A. Yes.

Q. And at your deposition you agreed because of that -- and you noted it in several places in your Expert Report -- that an epidemiological analysis of glyphosate and non-Hodgkin's lymphoma should control for exposures to other pesticides. Correct?

A. Yes.

Q. Now, we'll talk about your criticisms of this study probably tomorrow, the 2018 *JNCI* Study, but you agree that, as reported by the ten government investigators, and I think there are two academic investigators, who combined -- they did not find and they did not report association between glyphosate and non-Hodgkin's lymphoma. Correct?

A. Who did that?

Q. The ten NCI and NIH scientists who collaborated with two academicians, independent academicians, to investigate this, prepare the study, and publish it. Correct?

A. Correct.

Q. And you stated in one of your earlier slides that the only thing that changed from the time of IARC to the time of today was the 2018 *JNCI* Study; but there also is that NAPP analysis that came out after the IARC preamble, but you haven't read that.

A. Correct.

Q. Even prior to the publication of the 2018 NCI Study, and even prior to the pooling of all of the U.S.-based case-control studies, and the adjustment for those three pesticides that we've heard about already at length today and yesterday, that brought that Odds Ratio down to 1.13, and not significant -- even without that information, you believe that the epidemiologic evidence was not sufficient to show causal association between glyphosate and non-Hodgkin's lymphoma.

Correct?

A. I thought it was not sufficient?

Q. Not sufficient.

Pull up Slide 24.

You would agree that the epidemiological -- and this is before you knew about the 2018 Study, obviously, and without reading NAPP. And you agree that the epidemiology, alone, is not sufficient to show a causal relationship between glyphosate and non-Hodgkin's lymphoma. That was your testimony at your deposition that we're reading right here. Correct?

A. Yeah, but I've been re-thinking that.

MR. LASKER: Well, Your Honor, I think this is a good time for the break.

THE COURT: Okay. Sounds good. So we'll see everybody tomorrow at -- we start at 10:00 tomorrow. Is that right? Okay.

THE WITNESS: Your Honor, I have a 1:30 flight.

THE COURT: You have a 1:30 flight?

We do could one thing. What we could do is a late launch, and the other thing we could do is keep going today, to ensure that Dr. Neugut makes his flight.

THE WITNESS: I mean, it's up to Mr. Lasker. I don't know what his -- how long --

MS. WAGSTAFF: Let's keep going on.

THE COURT: Mr. Lasker, do you have a rough time

estimate on how much more you need for cross-examination?

MR. LASKER: I think I can make it shorter, but if we do it tomorrow, we'll try and cut it down now. I'm going to try and cut it back, because I think Your Honor has addressed a lot of issues in your questions, but I think I'd be able to do that better tomorrow morning. Unfortunately, if I'd try doing it now, I think it will take too much time.

THE COURT: Okay. Well, I mean, I would like Dr. Neugut to be able to make his flight.

MR. LASKER: I'm sorry, what?

THE COURT: Dr. Neugut has a 1:30 flight.

MR. LASKER: What time, we're starting at 10:00?

THE COURT: Yeah, that's when we were --

MR. LASKER: I think I'm not going to be going nearly that long with him. And actually, if I can start tomorrow morning, I'll be able to make it shorter. That's not going to be a problem, unless Mr. Miller has a very, very long redirect.

MR. MILLER: Very short redirect, Your Honor, I'm sure.

THE COURT: Okay. You know, that's one of the hazards of testifying in court, is sometimes you have to change your flight. So hopefully you won't have to do that.

All right. We'll see everyone tomorrow. Thank you.

(At 4:34 p.m. the proceedings were adjourned.)

I certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

March 7, 2018

Signature of Court Reporter/Transcriber Date

Lydia Zinn