UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

BEFORE THE HONORABLE VINCE CHHABRIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION,	)	No. M16-2741 VC
	)	San Francisco, California
	)	Friday
		April 6, 2018

TRANSCRIPT OF PROCEEDINGS

APPEARANCES:

For Plaintiffs:

ANDRUS WAGSTAFF, PC
7171 West Alaska Drive
Lakewood, CO 80226
(720) 255-7623
BY: AIMEE H. WAGSTAFF, ESQ.
DAVID JACKSON WOOL, ESQ.

For Plaintiffs:

ANDRUS WAGSTAFF, PC
6315 Ascot Drive
Oakland, CA 94611
(720) 255-7623
BY: KATHRYN MILLER FORGIE

For Plaintiffs:

BAUM HEDLUND ARISTEI AND GOLDMAN, PC
12100 Wilshire Boulevard, Suite 950
Los Angeles, CA 90024
(310) 207-3233
BY: ROBERT BRENT WISNER, ESQ.

WEITZ & LUXENBERG, PC
700 Broadway
New York, New York 10003
(213) 5578-5802
BY: ROBIN L. GREENWALD, ESQ.

(APPEARANCES CONTINUED ON FOLLOWING PAGE)

Reported By: ***Debra L. Pas, CSR 11916, CRR, RMR, RPR***
Official Reporter - US District Court
Computerized Transcription By Eclipse

APPEARANCES: (CONTINUED)

For Defendant Monsanto Corp.:

HOLLINGSWORTH, LLP
1350 I Street, NW
Washington, DC 20005
(202) 898-5800
ERIC GORDON LASKER, ESQ.
HEATHER ANN PIGMAN, ESQ.
GRANT HOLLINGWORTH, ESQ.

ALSO PRESENT: The Honorable Ioana Petrou

– – –

Friday - April 6, 2018 **9:07 a.m.**

P R O C E E D I N G S

---000---

MR. LASKER: I have a preliminary matter, Your Honor.

Plaintiffs have just provided with us a slide deck that I take it they are planning on presenting through Dr. Portier. It's 30 slides, which I have had just had a chance to look at. There is one slide that's from his expert report. The other 29 slides are new. They are new analyses. They're are new opinions. I've never seen those numbers before. There is calculations I've never seen before.

Obviously, I have had no idea -- I have not had a chance to depose him. I have no idea where we are going on this, but my ability to cross-examine him on any of this is going to be extremely limited. And I thought we had addressed with Your Honor on the call when these were being -- these presentations were being scheduled, the question about whether or not either Dr. Ritz or Dr. Portier would be presenting new opinions. And our understanding from Your Honor was that that was not the intention of this hearing. So we object very strongly to virtually all of the slides in this slide deck.

THE COURT: I mean, for -- for the sort of -- I know that sometimes it may be difficult to figure out exactly where the line is, but I've been operating under the assumption that this testimony would be, you know, further elaboration of,

further explanation for, further detail regarding the opinions that were previously offered. So -- as opposed to new opinions.

So, you know -- do you want to show me a certain example of a slide that you think reflects a new opinion as opposed to further explanation of, further elaboration of, further detail about, you know, the existing opinion?

MR. LASKER: Yes, Your Honor. And, I mean, we can go through them because all of them are new in different ways. There are a series of new forest plots that we've never seen. But if we can just walk through, I guess, the slide deck, the -- and some of them are of more concern to me than others.

MS. GREENWALD: Your Honor, can I just make one comment before? I don't mean to interrupt, Erik, I apologize.

But I just want to make it clear, because we have gone through this with Dr. Portier. These slides are literally illustrative of what's in his report. There is, I assure you, no new opinion here. These are helping to explain some of the questions that Your Honors have been asking from the bench, both the *Daubert* one --

THE COURT: That's fine. But let me hear from Mr. Lasker about his concerns.

MS. GREENWALD: Okay.

MR. LASKER: So some of these I'm less concerned with, although they are new. The forest plot on Slide 2 I've

never seen before but they are numbers we've dealt with. And I'm less concerned also with 3 and 4 just seem to be explanatory of basic concepts.

5 is actually -- I'm sorry. Slide 5 is actually from his forest plot.

Slide 6 I think seems consistent, although it's a different visual, so I'm not sure.

THE COURT: Different visual --

MR. LASKER: I don't think I object. That was my point there.

Slide 7 is dealing with latency, and there are a variety of opinions now that are being expressed about factors that can affect latency that are new. We can hear what those are, and I'll sort of take it as it comes, but those opinions were not offered in his expert report.

Slide -- I'm sorry.

THE COURT: But there will be a question, he mentioned latency briefly in his expert report. He testified about latency a bit more extensively the other week --

MR. LASKER: Right. And as I said, I think some of these are more -- some of these I have to sort of wait for the testimony. I'm sort of walking you through in order.

Slide 8, I've never seen before. This is an analysis comparing numbers that I've never seen before with calculated odds ratios that I've never seen before with a case-controlled

study versus a cohort study. None of these analyses or opinions were expressed. If I can walk through this --

MS. GREENWALD: Can I stop you there for a minute? Number 8 is a hypothetical, just to help explain. It's nothing to do with any of the data in this case.

THE COURT: One of the things that's a little annoying about this is that you only gave the slide deck to Mr. Lasker this morning. I would think that you could have given it to him earlier.

MS. GREENWALD: Your Honor, there is a real problem. I mean, Dr. Portier lives in Switzerland and he flew in, as you know -- well, you don't know this. He flew in -- well, he got in at 1:00 a.m. the night before Dr. Ritz testified. He was in court --

THE COURT: There is -- there is email and you can email slide decks back and forth, and you can talk on the phone, and you can talk by video conference. I mean, anyway --

MS. GREENWALD: I assure you, we did this yesterday. But, okay. I understand.

MR. LASKER: If I could just continue.

Slide 9 I'm not as concerned about. It's a new forest plot, but I've seen all these numbers before.

Slide 10, I'm not concerned about.

And Slide 11, just is an introductory slide.

Slide 12 and 13, again, seem to be introductory. So I

don't have as concerns -- as many concerns about those, but there's a lot of steps that he discusses in here, going into detail about the imputation method and how it was done that he did not have in his expert report. I have -- I'm sort of at a loss of where he is going with these.

THE COURT: But did he -- I may be misremembering this, but in his supplemental report did he not criticize AHS for the manner in which it imputed exposure information and the like?

MR. LASKER: He did. And as I'm looking at Slide 13 and 14, I think these are just describing the Heltshe methodology. I'm reading this as we go along. I don't think those are issues, but I'm sort of doing this on the fly.

THE COURT: I understand.

MR. LASKER: And Slide 16, he had talked about this issue before. We had not had this graphic, but I'm at least familiar with the issue. These numbers and calculations of absolute bias that he has here, I've never seen before. And I'm not sure, sitting here, how they are calculated or whether or not I would have an issue with them if I had had time to look at them. They are just new numbers for me.

He had talked about the fact that the predictions were below the -- the general issue he had raised, but I've not seen any of these calculations before.

The slides from 17 to, I guess it's -- I think these --

I'm not sure where they end, maybe through 25 --

MS. GREENWALD: Yes, that's right.

MR. LASKER: -- I take it may be going to be a hypothetical that is -- that is presenting different calculations based upon different types of non-differential exposure classification. That's what it looks like to me. And then he is going to have some calculations on how that would impact.

Again, I don't know if these calculations are right or wrong. I have not seen them before. And I'm not going to be able to do anything with them today other than just --

THE COURT: You might be able to do something with them today. Thus far, it appears that you know the material better than a number of the experts, so perhaps you will be able to do something with it today.

MR. LASKER: The issue, though, Your Honor, is the calculations. I don't -- these are numbers that he's presenting. He's got calculations and percentages. And I do understand the materials, but I can't do the math and I don't know how he's done the math and I'd have to explore it here today. I have not had a chance to question him about that.

THE COURT: I understand your concern. And I think there is always going to be difficulty in, you know, sort of drawing the line between what's a new opinion and what's an elaboration of an existing opinion.

I've sensed that this is going to be an issue for you. You know, sort of once we wrap up today, that's going to be an issue for you. And I was sort of expecting you to say, you know: We would like the opportunity to file a brief on what is -- you know, what opinions -- what aspects of the opinions that Dr. Ritz and Dr. Portier offered this week are new opinions and unclosed opinions.

MR. LASKER: Right.

THE COURT: And if you want, you can do that. You would have to do it quickly.

MR. LASKER: Yes.

THE COURT: But if you want to do that by, say, Monday, you know, you can file a brief sort of objecting to what you believe are new opinions from these witnesses as opposed to elaborations, I would invite you to do that.

MR. LASKER: Okay.

THE COURT: And explain why it was unfair for Dr. Portier to testify about X or Y on this day.

MR. LASKER: Yes.

THE COURT: And, but one other thing. If you take me up on the invitation to file that brief, I would like you to address something that is related to that, which is -- it's one thing for an expert to disclose an opinion in a report and then testify at deposition in support of that opinion, and then get up on the stand in front of a jury at a trial and testify to

something different, where the -- you know, the -- the other side is entirely sandbagged by that and hasn't had an opportunity to address it. You know, that is the classic sort of scenario where, you know, a judge would exclude testimony because it was an undisclosed opinion. Right?

One question I have been wondering about is -- you know, is the analysis a little bit different in a context like this when we're -- you know, we're engaging in this phase one inquiry that is still far, far away from any jury trial, right? You know, what -- you know, how -- how does a judge deal with arguments about undisclosed opinions in a context like that? And should a judge be thinking about it differently in a context like that? And you could -- you could address that question in your brief.

MR. LASKER: Yes, Your Honor. And there is case law on that that we would be happy to point you to.

THE COURT: If I recall -- I mean, I'm just -- I don't remember the name of the case, but there was a case that the plaintiffs cited, I believe. And I believe it was a District Court opinion from somewhere in the 11th Circuit, maybe Florida or something like that, maybe Miami.

MR. LASKER: Yes.

THE COURT: Where the -- although I wasn't actually that -- it's been awhile since I've read it, but I wasn't that impressed with the judge's conclusion in that case to allow the

expert opinion in. You know, the judge also discussed, you know, this issue that I'm talking about, at least a little bit, and that -- that discussion seemed at least somewhat persuasive to me.

So if you could address that, that would be helpful.

MR. LASKER: Yeah. We will, Your Honor.

And just one more thing and then I'll sit down before...

But Slide 27, Dr. Portier is presenting numbers from the NAPP. And Dr. Portier never mentioned the NAPP in any of his expert reports, so this is not expansion on opinions. It's he had not offered opinions.

And I had asked him briefly whether he looked at it and he said: I was shown some slides, but I don't remember them. So that's a completely new opinion.

THE COURT: And I recall that as well, and that may be a concern. We can let him testify about it now, but I may not consider it.

MR. LASKER: Thank you, Your Honor.

MS. GREENWALD: Can I make one -- couple of clarification points?

So as to the Andreotti, which is Slides 11 through 26, Mr. Lasker deposed Dr. Portier on this very study in London, according to your Honor's order, when you had supplemental depositions and supplemental reports.

I just want to let you know he did have an opportunity.

For example, 16, which Mr. Lasker suggested he's never had an opportunity to talk to Dr. Portier about, which is the Heltshe study. He spent probably almost half of the deposition talking to him about Heltshe. So I just want to let you know. It wasn't fun at all.

And, also, he did elicit questions from Dr. Portier about NAPP. But with that said, I just wanted to clarify that.

And can we talk a little bit maybe at the end of the day about whether we have an opportunity to respond? Because I don't know what they are going to --

THE COURT: You can file a response to them, but, again, it's going to have to be very quick.

MS. GREENWALD: I understand. You're doing Monday, by the end of the day.

Can we do Wednesday morning? Wednesday afternoon? Is that okay?

THE COURT: Wednesday is fine.

MS. GREENWALD: Okay. Thank you.

THE COURT: Okay. Dr. Portier, do you want to come on up?

MS. GREENWALD: Your Honor, do you want to start? Do you want me to start?

THE COURT: I'll let you go ahead and start. I have some questions, but I will have fewer because he hasn't provided the testimony.

MS. GREENWALD: I understand. That's what I assumed.

THE COURT: So I'll let you go ahead and start.

CHRISTOPHER PORTIER,

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

THE WITNESS: I do.

THE CLERK: Thank you. Please be seated.

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

THE WITNESS: Christopher Portier. C-H-R-I-S-T-O-P-H-E-R, P-O-R-T-I-E-R.

THE COURT: It sounded like you just said Portier.

THE WITNESS: Good morning, Your Honor. I'm afraid you were told wrong on Wednesday.

MS. GREENWALD: It was my fault. At the end of the day, I was not thinking clearly, so I apologize.

THE COURT: No problem.

Sorry about that, Dr. Portier.

DIRECT EXAMINATION

BY MS. GREENWALD

Q. Good morning.

Can you just briefly tell the Court about your epidemiological background?

A. I have some education in epidemiology. My PhD had a minor in epidemiology, an application in biostat.

Early in my career I did a little bit of epidemiology. Throughout my career I've spent a lot of time reviewing epidemiology as part of my job and for other reasons.

Recently I've started publishing epidemiology papers again. I had one come out three months ago looking at exposure in Oakland, exposure to air pollution in Oakland. We drove Google Street View cars around Oakland for two years looking at how much air pollution there is. We have another paper coming out in the very near future --

THE COURT: Can you tell me a little bit more about that Oakland paper?

THE WITNESS: Well, this is the same thing, because what we're also doing is following up that up with Kaiser Permanente and our exposure from the City and trying to see what it means to the health of the people in the city --

THE COURT: I am curious. So -- so what exposure are you looking at? Just air pollution generally?

THE WITNESS: Different components of air pollution, NOx, SOx, carbon black, PM2.5. These are things that we worry about.

THE COURT: So you're -- so how are you measuring air pollution?

THE WITNESS: Oh, the Street View cars have instruments --

THE COURT: What cars?

THE WITNESS: Street View. These are the Google cars that go around and measure, take pictures of everything. They have equipment in the trunk that -- GC mass specs and things, and little tubes on the roof that suck in air into the trunk and measure it and send the information to Google, who stores it, and then comes back to us and we analyze it and made a paper about it.

THE COURT: Huh. And so what are you -- what did that paper conclude?

THE WITNESS: Oh, we were interested in the -- the purpose is to see how well we can measure air pollution using a car for -- with cheap systems and compare that to what the regulatory monitors do, and we concluded we did a better job. And we can pick up hot spots in the city and show you why those are hot spots, why they have elevated levels simply because of traffic flow or things that could be fixed.

So the whole purpose was to try to see if there was a way to understand how to use this data to better design cities like Oakland. We're now doing the whole Bay Area. We have now six cars driving around the whole Bay Area.

THE COURT: So does that -- does that -- did that study seek to identify different sources of pollution or just measure the amount of pollution in particular parts of Oakland?

THE WITNESS: We measured the amount of pollution that was predominant, but -- we spent some time looking at

sources of pollution.

I mean, you know you've got the port right there. That's a huge source of pollutants, but both -- all the interstates around the city are extreme sources of pollutants for Oakland. West Oakland is worse than East Oakland. Yeah.

THE COURT: Okay. And where -- and you said that's a paper that's published?

THE WITNESS: Yeah. Journal of Environmental Science and Technology.

THE COURT: Okay. Thank you.

THE WITNESS: I will be happy to get you a copy, if you would like it.

THE COURT: That's okay.

BY MS. GREENWALD

Q. Okay. If we can go to the first slide, please?

THE COURT: But I actually -- were you done talking about his epidemiology experience?

MS. GREENWALD: I didn't want to -- yes, but I can more than happily --

THE COURT: I would like to hear a little more detail about your experience in epidemiology.

I mean, I was going back through your expert report last night. There is not really -- I mean, there is not really any discussion of epidemiology in your qualifications, is there? In the section of your report on qualifications?

I mean you talk about your jobs --

THE WITNESS: Correct.

THE COURT: -- and you -- you know, you talk -- you talk about the things you've studied, but when -- it looked to me like whenever -- whenever you get into specifics about things that you studied, it's always a focus on animal studies.

And so I -- after reading that, I was left wanting to know a little bit more about, you know, in your -- in your various jobs, like at NTP and, you know, at the National Center for Environmental Health, you know, how much of your work focused on epidemiology as opposed to toxicology?

THE WITNESS: Okay.

THE COURT: If you could kind of walk me through your history of that.

THE WITNESS: I'll give you a few examples, and maybe that will help better understand.

It's -- it's a fair statement that most of my personal research is, indeed, in toxicology and mechanisms of cancer, mechanisms of immunotoxicity and other areas like that. That's a fair statement.

In the 1990s, as an example, 1990s I was asked by the U.S. Government to start a research program with Vietnam, U.S.-Vietnam research program on Agent Orange and its effects on people in Vietnam. Brought together a team, designed some studies. Worked through the State Department to try to get it

together. Eventually after seven years ended up funding such a study.

So even though I didn't do the study, I was intimately involved in its design, its organization and getting it put together.

Later, '98, 1998, I was asked by Congress, again, to review the health and safety of power lines and whether or not they caused childhood leukemia. That involved a predominantly epidemiology study. There was very little toxicology worth looking at. There were 18 epidemiology studies that we had to review and comment on and come to some sort of conclusion that would help Congress and regulatory agencies decide to what agree they needed to address that question. So I had to know all the epidemiology in that situation.

At the NTP I was responsible for the report on carcinogens. So even though Dr. Jameson ran the day-to-day operations of the report, when it came down to it, I have to be the one who said: Yes, I agree with the decision that the team has come up with; or: No, I don't. So, again, I have to be able to read and understand all of the epidemiology in detail.

At CDC I had several divisions that were entirely epidemiology that I had to interact on a daily basis looking at their papers and what they were doing. We had a major study going on in Camp Lejeune, North Carolina, where the Marines had been exposed to water, underground water that had been

contaminated with toluene and other things. I helped design that study. I negotiated with the Navy to allow us to do the study. I had to be able to put forth to them why it was an important study, that the design was appropriate and everything else.

That's -- then I've worked on reviews myself for WHO, for IARC, where I've sat on the panels and had to review data. When I worked --

THE COURT: You sat on epidemiology panels?

THE WITNESS: Only once have I sat on an epidemiology -- and only partly sat on the epidemiology panel. I was chairing the meeting. It was on diesel exhaust.

When I was with EPA Science Advisory Panel, they had a meeting on the use of human studies in pesticide approval process. There had been a push to be able to expose people to pesticides and look at not toxicity of it, but the metabolism and everything in people that I had to chair that -- I chaired that meeting and we had a very lively discussion about it. But, again, that's clinical epidemiology, but it's the same thing.

Is that enough?

THE COURT: Yeah. Although it prompted a couple other questions that crossed my mind as you were talking.

Diesel exhaust, so you chaired the IARC Working Group for diesel exhaust?

THE WITNESS: Yes.

THE COURT: What was the conclusion?

THE WITNESS: Diesel is a known human carcinogen.

THE COURT: And --

THE WITNESS: Diesel exhaust, sorry.

THE COURT: Diesel exhaust.

And is that -- what -- did your working group reach any conclusions about what types of cancer the diesel exhaust causes?

THE WITNESS: Yes. Certainly lung cancer. I'm not sure whether there were some smaller other cancers in there, but certainly lung cancer.

THE COURT: What about NHL?

THE WITNESS: That, I do not recall.

THE COURT: Okay. And I -- and then one -- back to your Oakland paper.

Why is that an epidemiology study? I mean, so you -- you've talked so far about the amount of pollution in the air, but just studying the pollution in the air does not strike me as an epidemiology study, unless I don't have the -- you know, I have the wrong definition in my head of epidemiology.

THE WITNESS: There's a -- the purpose of the exposure study was to do epidemiology. So we have -- I built a collaboration with Kaiser Permanente, who provides health services to most of northern California, or about half the

people in northern California. And it was about 45,000 people in and around the Oakland area. And so what we then did was a retrospective cohort study.

Given the exposures, we backed them up in time and said: What if the City looked like this for the last ten years? Now let's look at the health records of all these people, pull them apart and see if we see a pattern, in this case, for cardiovascular disease.

We were able to show that even in this little area of West Oakland, we can detect differences in cardiovascular disease risks across that small population if we do this good of a job with an exposure evaluation. And that is an epi study.

THE COURT: Okay. All right. Thank you.

BY MS. GREENWALD

Q. Can I just ask you one follow-up about the Camp Lejeune? You were looking at health outcomes of service people who lived at Camp Lejeune?

A. Yes.

Q. Were you focusing on the breast cancer association?

A. That was one thing looked at, the male breast cancer risk, yes.

Q. So that was, again, health outcomes in that case as well?

A. I left before they finished.

Q. Okay.

A. And I don't remember. I read the paper when it was done,

but I don't remember.

Q. Okay. Okay. All right. Thank you.

So should we --

MS. GREENWALD: If you can go to the first slide now?

BY MS. GREENWALD

Q. Can you please explain your methodology for evaluating the causality between glyphosate and non-Hodgkin's lymphoma?

A. Certainly. In any evaluation like this, it's common methodology to review all of the data, the studies in the human populations, the studies in the animal populations, and the other mechanistic studies as well.

So my evaluation of causality here really went through all of that data in great detail looking at it very carefully.

The studies of the human population, which I'll now spend some time on, that's a straightforward methodology that most people use. Those methodologies are followed by EPA, followed by the International Agency for Research on Cancer, followed by the European Chemical Agency and the U.S. Report on Carcinogens and others in terms of how you look at that data, how you evaluate it.

You assess study quality to make sure that you really want to include this study in your overall evaluation. And then you have to evaluate the degree to which the study supports a finding of cancer in humans.

And typically that can range from "I don't know if this

study tells me anything" to "the study clearly tells me that there is nothing going on," to "this study clearly tells me there is something here." And there is all the ways in between of that, and I hope to try to express the -- my understanding of that with these data today.

The guidelines, the methodology used by EPA, IARC, U.S. Report on Carcinogens and to some degree EChA, I was involved in the development of all those guidelines so I do have some knowledge of the methodologies that they use.

THE COURT: What is EChA again?

THE WITNESS: European Chemical Agency.

MS. GREENWALD: If you would go to the next slide, please?

BY MS. GREENWALD

Q. What is this summary plot showing?

A. I'm sorry if this appears to be something new. After seeing most of the presentations, I wanted to bring up a plot that puts everything up there. So we're looking at it -- I am a holistic sort of guy. I want to see all of it up there at one time.

So this is the -- in my expert report I identified six core studies I was interested in: The McDuffie study, the Hardell 2002 study, the De Roos 2003 study, the De Roos 2005 study, and the Ericksson 2008 study, and the Orsi 2009. Is that six? Yes, six.

And I mentioned the meta-analysis that I looked at, the analysis Model 1, from Chang and Delzell. This shows you which ones were adjusted for other pesticide exposures, which ones were not. Shows you the exact range in the chart so you can look at it all at once and see how they differ from each other.

We'll come back to this plot.

Q. So do you want to go to the next slide and see if you can explain?

A. Yeah. I'd like to make sure we're all on the same page of what these plots really are.

So if you look at the next page, this is hypothetical confidence bounds. It's what you have been seeing. It's a dot and whisker plot. You get the little whiskers and the dot in the middle.

I changed it slightly because I'm using a log scale from the bottom instead of a linear scale. So it's slightly different, but I do that because I need to be able to show you a distribution.

So here the point estimate of the odds ratio is 1.5 and the range is .9 to 5.0.

Now, these confidence bounds, they are derived from an underlying statistical distribution. I don't know if you -- you've seen these bell curves before and know what a bell curve is, a normal distribution.

So if I could have the next slide?

So there is this underlying normal distribution that leads to these curves. What you're looking at with the whiskers on this plot is the 95 percent confidence interval. And that means 2.5 percent of the mass under this distribution is to the left of the lower one and 2.5 percent of the mass under this curve is to the right of it. And then 95 percent of the mass of the curve is covering that whole area. So that's what this means.

Now, when it crosses the bound of 1, which is the area where people talk about whether it's significant or not significant, if the lower bound crosses 1 or not. We're talking here -- if you look at this plot, you see where there is the -- the red on the left. And then the red line that is 1, there is that little bit of white area right in there. That, in this case, this theoretical case, is about one and a half percent of the mass of this distribution.

So even though this crosses over -- I'm going to give you some other characteristics of this -- only 4 percent of this distribution is below 1. 96 percent of it is above 1.

So even though that 95 percent confidence interval touches it, there is still a lot of distribution, a lot of probability on the right side of this curve pushing you towards seeing potentially an effect with this type of -- of response.

Is that clear? Good?

If we could go back to 2.

MS. GREENWALD: Go back to 2, please.

A. So part of my interpretation in looking at these studies is maybe slightly different than how most people expressed it or I missed it. But I'm going to use the Ericksson paper as an example here.

Now, you might ask yourself, I certainly asked myself: Why do you always see the paper, virtually all of the papers, to an unadjusted analysis and then an adjusted analysis for pesticides? Why do they do that? Why not just give you the adjusted analysis for the pesticides?

It's because one way to interpret this is with the no pesticide adjustment -- remember, it's still adjusted for age and all kinds of other stuff, but with the no pesticide adjustment, I'm looking to see if there is any association.

And then by adding in the pesticide adjustments, I'm looking to see if part or all of this association can be explained by other pesticides. And those other pesticides push this curve back down because they are taking some of that explanation away from the, in this case, glyphosate.

So I look at these two and I don't say it's not statistically significant when it's adjusted. I say in this case the adjustment took about -- it looks like about half of the explanation -- half of the association seen for glyphosate away from that chemical because of the other pesticides were predicting about 50 percent of it themselves.

In epidemiology they talk about something called "attributable fraction." You sort of -- you've heard this. It's -- we heard Weisenburger say that 70 percent of the NHL is unexplained. That means the attributable fraction is 30 percent. I can attribute 30 percent -- for 30 percent of the NHLs out there, I can find something that probably caused it.

So here I'm looking at something like an attributable risk as well when I think of these things. It's a little bit over. If it goes a lot over, I'm not -- I'm going to figure that the pesticides completely washed out the effect.

But if it moves just a little, even if it becomes non-significant, I'm still going to be recognizing that as probably an effect by glyphosate.

Excuse me, my mouth is dry.

Okay?

BY MS. GREENWALD

Q. Okay.

A. Unless there is a question? Good.

MS. GREENWALD: If we can go now to Slide 5, please?

BY MS. GREENWALD

Q. You mentioned just a few minutes ago that you looked at six core studies. Can you explain Slide 5 for the Court please?

A. Yes. This is directly from my expert report. It's

directly from Chang and Delzell's 2016 paper.

These are all of the studies that were looked at by Chang and Delzell, and I think it's most of the ones I commented on in my original expert report.

The red studies are the studies that went into Model 1 of the meta-analysis done by Chang and Delzell. And those are the McDuffie study, unadjusted.

The Hardell study -- I can't remember if that's adjusted -- that's the unadjusted number. I'd have to look -- no, that is an adjusted number.

The De Roos number, that's a fully adjusted number. And that's the De Roos study using the hierarchical model. The De Roos 2005 study, fully adjusted.

The Ericksson study, fully adjusted.

And the Orsi study, which is not adjusted for other pesticides.

And when they did their meta-analysis, they came up with a meta risk of 1.3 with a lower bound 1.03 and upper bound of 1.6. So they saw a positive effect.

The weights that you see on the side are the weights that are given to these papers in the meta-analysis based on their variance and based on the size of the study. So big studies with tight variance get more weight than little studies with wide variance.

The studies with zero weight here just simply were not in

this meta-analysis. So they get no weight.

Meta-analysis Model Number 2 switched the hierarchical model from De Roos and used just the logistic regression model from De Roos 2003. And, again, you see a significant meta-analysis.

Meta-analysis 3, took Hohenadel, 2011, and switched it for Ericksson, 2008. And again you see a significant effect.

And I forget what four is, I'm sorry. I can't remember them all.

THE COURT: Those four models are from Chang and Delzell?

THE WITNESS: Correct.

THE COURT: Okay.

THE WITNESS: They are meta-analyses that Chang and Delzell did and provided the results for.

They did meta-analyses on a number of the individual lymphomas, and I covered that in my expert report as well.

THE COURT: Can I ask a question about that?

There were -- if I'm recalling correctly, there were three meta-analyses of -- or pooled analyses of the case-controlled studies. There were Chang and Delzell. There was the IARC one. And there was one other one I'm not remembering as I sit here.

THE WITNESS: That was Delzell -- I'm blanking on his name.

MS. GREENWALD: Schinasi?

THE WITNESS: Yes, Schinasi. That was done first.

The reason IARC decided to do their own was because Schinabi somebody -- Schinasi --

MS. GREENWALD: Schinasi.

THE WITNESS: Schinasi.

They went ahead and they used the unadjusted for other pesticides. That's all they used. And the IARC group felt that they wanted to see the comparison with the adjusted pesticides to see if it made a difference.

And so that's what they did. And that is, indeed, what Chang and Delzell did. Chang and --

THE COURT: So did IARC use McDuffie, for example?

THE WITNESS: Yes.

THE COURT: Because the McDuffie numbers, as I recall, were not adjusted for other pesticides.

THE WITNESS: That is correct. Chang and Delzell's meta-analysis Model Number 1 is exactly the same as the IARC meta-analysis Model 1 -- or model, period. They only did one.

This here, Chang and Delzell did a good job of looking at a sensitivity analysis. How sensitive is the finding to pulling one paper out and putting another paper in, or pulling one evaluation out and putting a different evaluation in from these papers? So you can see how sensitive it is. That's a very common thing with a meta-analysis.

THE COURT: So Chang and Delzell was post-IARC?

THE WITNESS: That is correct.

MS. GREENWALD: Your Honor, the Schinasi paper is Exhibit Number 23 in the book, in case you want to know where that is.

BY MS. GREENWALD

Q. Anything else on this slide before we move on?

A. No. I think this is covered.

Q. Okay. So I'd like you to just walk through Slides 6, 7 and 8, which were the slides that -- you were here, Dr. Portier, right, correct, during the various discussions on latency?

A. Yes, I was.

Q. Okay. And I believe you put together Slides 6, 7 and 8 to try to explain to the Court your methodology and how you use latency in your evaluations, is that right?

A. Yes.

Q. Okay. So if you can walk through those, please?

A. Thank you.

In the last time I testified, in the first *Daubert* round, I tried to explain that there were three different pieces to latency and I wasn't sure I got a good -- I did a good job of it.

This is a CDC job, CDC picture from ***Principles of Epidemiology***, a 1992 book that they put out. They redo it

every few years, but they continue to use the 1992 picture for this.

You start with exposure, and during the time that you're being exposed, you're susceptible to something from that exposure. And it could be right at the beginning or it could happen after five years of exposure, but sooner or later there is a subclinical change that occurs in your body somewhere. This is for any disease. And that subclinical change, you don't know it's there. But it's starting to work its way through.

At some point if you had someone looking very carefully at you, you could probably detect these changes, pathologically, but very seldom does that happen unless you're in autopsy.

So it does become detectable at that point, but you don't know it's there until you start getting symptoms. When you start getting symptoms, when they get bad enough, you go to the doctor and then you have the time of diagnosis.

So latency refers to the stage of susceptibility, stage of subclinical disease and part of the stage of clinical disease. It involves all of those various pieces and parts.

MS. GREENWALD: Next slide. Thank you.

BY MS. GREENWALD

Q. This is also from that same CDC *Principles of Epidemiology* book. These are factors that they say can affect latency. We talked about most of these.

The important ones here are age at exposure, gender, genetic susceptibility, other cancer risk factors and other medical conditions, like immunosuppression. You talked quite a bit about that.

The thing I want to make important with this slide is that there are many different latency patterns that you see in people. It's not just there is a latency. There are people who have a very short exposure period before they get to a very short pre-clinical period, before they get to a very short chronic disease period, and then they die.

And you've got people who get exposed for a very long time and nothing happens and then they go through a fast path. And you get people who get exposed, the pre-clinical damage begins, but it takes a very long time before it comes out.

So there's many different types of latency here.

THE COURT: Asbestos would be an example of that last one, right?

THE WITNESS: The "last one" meaning?

THE COURT: Exposed. And you have a long period of pre-clinical damage before you're diagnosed.

THE WITNESS: That's correct.

Black lung would be the same. Any of those types of diseases tend to be longer term, but they -- they start and develop over a long period of time. Debilitating you over much of that period.

BY MS. GREENWALD

Q. Next slide?

A. Yes, next slide.

Now, this is -- this is entirely -- I understand Mr. Lasker's concern with this. I'm not trying to make any statements whatsoever about NHL in this. This is still dealing with the latency issue. And I just wanted to make a couple points about case-controlled studies versus cohort studies, and so I made up these. They are sort of supposed to look like the De Roos 2003 study and the Agricultural Health Study, but they are just sort of to look like that.

In a case-controlled study you have an underlying population. Let's take the De Roos case. They did western Nebraska, all of Iowa and parts of Minnesota, excluding the big cities. It was white males above the age of 19 predominantly, although one of them did above the age of 30. You can actually go, look at the population registries for those states, those areas, and you come up with about 2 million people. So they actually drew non-Hodgkin's lymphoma cases from an underlying population of 2 million people.

Now, if -- this is the theoretical part. If NHL occurs in the general population at 15.2 cases per 100,000 population, then -- this is drawing cases for three years: 1995, '96, '97. Each year, just by chance, you would expect to get 274 cases. That's the blue.

Now, some of those cases would be exposed to glyphosate, but they would just be -- the glyphosate didn't cause it. It's just because people were there, they get exposed. I chose a 10 percent exposed here at random, 10 percent of the people were exposed.

So you get 274 unexposed randomly occurring NHLs, and 30 -- is that 30? -- 30 exposed randomly occurring NHLs. And then I choose here an odds ratio of 1.6 just to illustrate what happens if that's the case. You get 18 additional NHLs that were -- that were not spontaneous, that didn't come from other sources. Those were really due to glyphosate in this theoretical situation.

And if you do that, at the end you end up with 55 cancers that are due to the glyphosate, 91 that are exposed to glyphosate but not due to it, and 832 cases that are not exposed to glyphosate at all. So that's how a case-controlled study comes up. The important thing is there is this base population of 2 million people.

So you're sampling from a lot of people; some of which have very short latencies, some of which have very long latencies. And because you're pulling from that large population, you're looking at an entire latency picture.

If we could now go to the cohort study?

This is the same basic thing. But this -- because it's a cohort, it's typically now an occupational exposure. So more

of the people are exposed.

In this case I chose 50 percent of the people were exposed, but it's the same thing. Every year you get some spontaneous NHL cases. Every year you get some NHL cases that are also spontaneous but exposed to glyphosate. And then you get some that are really done because of glyphosate.

And you start accumulating them over the years such that in the end with this example you end up with almost the same number of cases caused by glyphosate in the two examples, the case-control and the cohort study.

But the reason this pertains to latency is that in this cohort study it has to go long enough for you to statistically be able to detect an increase in cases due to glyphosate if it's really there.

And so many times when we were discussing latency from some of the papers, that -- that's -- latency and lag, and it's a combination of being able to see something versus the other latency, which was the theoretical one I laid out for you.

So I just wanted to point out that there are these two different things that play a role in your thinking as you look at these and decide about the latency issue.

THE COURT: I'm not sure I totally understand what you're saying.

I mean, for the cohort study example that you give, the example that you give involves beginning to follow people in

1995, is that right? And so you start to follow people in '95. But is this example -- is this example a prospective cohort study?

THE WITNESS: Yes.

THE COURT: So this is not an example of the cohort study where they start following people in '95, but they ask them about their past exposures?

THE WITNESS: So I --

THE COURT: Only looking at their exposures from '95 going forward, is that right?

THE WITNESS: Yes.

THE COURT: In this example?

THE WITNESS: Correct.

THE COURT: Okay. I understand now.

THE WITNESS: That would be normal.

THE COURT: Okay.

THE WITNESS: The reason you do the two different studies, the case-control study, if you recall, is specific to the disease. So you're only looking at NHL patients in the case-control study.

The Agriculture Health Study, the reason the AHS funded it is because they are looking at the health of 57,000 farmers and pesticide. So they are looking at everything, cardiovascular disease, et cetera, et cetera. You can't do that in a case-controlled study.

But a case-controlled study is less expensive. It covers a broader base of people. 2 million population in this one study. And that's got some advantages over the expensive long-term cohort study. It's not one is better than the other. It's they are used in different contexts to do different issues.

Q. Anything else on that?

A. So in my evaluation I didn't weight one more than the other. I was looking at what they were trying to tell me more than this study is so much better than that.

Q. In the cohort study, Dr. Portier, I understand that you picked this 1.6 number and, therefore, it would take 20 years of following them to see all the true NHL, what you believe to be the true NHL due to glyphosate exposure.

But is that your opinion, that it would take about 20 years if you were to start from day one and look forward?

A. No. This is strictly theoretical. Clearly, the Agricultural Health Study in the De Roos paper in 2005 had already seen some indications of what was going on in that cohort. So no, I don't believe it would have to go that long. I'm using this strictly for lag. Just strictly for this latency question.

THE COURT: But why -- I mean, I guess -- I guess I'm -- I'm trying to understand why you're making this point in the context of this case. Because we have one cohort study in

this case, and it's the AHS study, and it was not -- forgive me. I may be using the terminology wrong, but as I understand it, it's not a prospective cohort study. It's a retrospective cohort study. And they ask about people's exposure to glyphosate and various other potential hazards going way back.

And I understand -- one thing I understand was the criticism of that. We've heard a lot about -- from Dr. Ritz, about what the problems are with asking people to look back and the context in which they were asked.

But because AHS did look back, I'm having trouble understanding why you are -- what is the relevance of the point that you just made about cohort studies, about prospective cohort studies.

THE WITNESS: Again -- suppose every year I went back and asked them about their exposure. I couldn't publish a paper every year because I wouldn't have enough cases. It takes a long time before I get enough cases where now I'm comfortable with reporting out what I'm seeing.

Okay. So we had a lot of discussion about latency, and other sources that came in and said: Well, the latency for solid cancers is this. And the latency for hemapoietic cancers is something like this. And much of that is drawing from cohort studies, where people have gone into the cohort study and done lagged analyses. But those lagged analyses were not only dealing with low latencies, they are dealing with the

potency and how fast you get a big enough cohort to publish on.

So we have to be careful in interpreting whether it's a short latency or a long latency in looking at the context in which that term is being given to us. That's what I'm trying to point out here. It's a minor term, a latency. It was something we discussed when I was here last time. I didn't think I got a very good picture of it for you.

BY MS. GREENWALD

Q. Let me ask you one quick question before we move on. At baseline in the Agricultural Health Study they excluded NHL, correct?

A. Included?

Q. At baseline they excluded NHL, is that correct?

A. In the -- yes.

Q. In the AHS?

A. Anybody who had any type of hemapoietic tumor before was excluded.

Q. What's the consisting of that?

A. Of what?

Q. Of the fact that they excluded the NHL.

A. They are only following new cases that would appear after this point in time when they started the cohort study.

Q. So if you can move to the next slide, please?

A. Again, for completeness, I showed you the ever/never pictures.

These, for those six core studies that I looked at, three of them had other evaluations. And we've talked about them. I don't necessarily need to go over them with you again and again.

But the McDuffie study did less than two years, greater than two years. De Roos did tertiles of exposure. And Ericksson did greater than ten days exposure and greater than ten years of exposure -- exposure starting greater than ten years ago. And what you see here was the patterns you saw from that. The McDuffie study was positive, unadjusted for any other exposures. That's all of McDuffie.

Ericksson was positive for their two exposure metrics that are climbing or trying to indicate exposure response.

And the De Roos study had nothing on any of those.

Q. As to this Slide Number 9, the intensity of response in De Roos, that was not specific to glyphosate, is that correct?

A. The De Roos measure unfortunately was not directly for glyphosate. In order to get the intensity of exposure, they used personal protective equipment as one of the things that changed the intensity. They measured it. But they asked that question only once.

And these are pesticide sprayers and farmers. Glyphosate has no -- at the time there are no requirements to protect yourself from glyphosate spraying. There are for others.

And so when they just ask the question in general, Do you

use PPE, it probably pertains to the other pesticides, not so much to glyphosate. And so the intensity exposure might be a little bit off.

MS. GREENWALD: Okay. Next slide, please.

BY MS. GREENWALD

Q. This is your Bradford Hill criteria analysis, correct?

A. That is correct.

Q. Okay. Can you explain why you want this up here now? Since you're going to have it up again.

A. This is from my expert report. My final conclusions on my Bradford Hill aspects of epidemiological data and related science.

I will go through this again at the end. So I'm not going to go through it now, other than to say I -- I have looked at this point, at all of that epidemiology, and at this point from my viewing it, I found the consistency strong and I found the strength of the observed association strong.

Part of that is the meta-analysis. Part that is just looking at the multiple studies and seeing that they are indeed all greater or equal to 1 in their mean odds ratio. Seeing very little heterogeneity when we do the overall meta-analysis. They are done by different research teams on different continents. They have different questionnaires. And while there is potential for bias or confounding, there is no obvious bias or confounding in these data.

So I'll go through all of this when we get to the end.

Q. So now Slides 11 through 15 are on the Andreotti study and the imputation issues that you foresee. And am I correct that you did a supplemental report on the Andreotti study?

A. Yes, I did.

Q. And you were deposed on that as well, is that correct?

A. Yes, I was.

Q. If you could walk the Court through 11 through 15, if you could explain why you believe Andreotti is methodologically unsound?

A. I will assure the Court everything in here is, indeed, part of my opinion and was used in making my opinion. Whether every single slide and graph is the same as it was in the supplemental, that is not the case because I -- I'm presenting it to you as carefully and as in depth as I possibly can.

So I'm going to explain two things that I find with the Andreotti study that I find method- -- methodologically unsound. I'm going to stumble over that word every time. I think the evaluations that they did with the imputed exposures are entirely unreliable and shouldn't be used. I believe that the only reliable numbers are a complete case analysis. And I'll explain what that is next.

So why are they doing their imputation? You've heard this before. 20,968 participants did not respond to the survey. 8 percent had died. 15 percent refused to participate. And

14 percent just could not be found.

You can do one of two things here. The first thing you can do is say: I don't have those 37 percent people. I don't have their responses. I'm going to throw them out and analyze the data without them.

That can produce selection bias, and you have to be careful and look at that. It reduces your cohort size, so it reduces your statistical power.

The other thing you can do is imputation. You can use what you know about the exposures at the beginning and from the people who responded and try to guess at what the exposures for the others were.

In this case they chose to do a multi-step multiple imputation. Now, multiple imputation is a well-known technique in epidemiology for doing missing values. Multi-step multiple imputation, I couldn't find another case where they used it. So I don't know that it's been used anywhere else, but I want to explain to you what that is.

MS. GREENWALD: Next slide.

BY MS. GREENWALD

Q. So it's a four-step process. In the first step -- I'm going to break these up for you. You've got -- and I'm going to use simpler numbers. 60 percent have responded to both; 40 percent have not.

So now in the 60 percent that responded to both, I'm going

to take 20 percent of those people and I'm going to put them off to the side. Because I need -- I need to check my work and see how well I've done. And the remaining people, I create this model and it's a relationship between any pesticide use, the thing I'm trying to predict, and the survey responses from the people who answered both surveys, who are not my 20 percent who are sitting off to the side, and I build this regression model. And it might use things like age, and were you exposed to pesticides back at the first evaluation, and things like that.

And then for -- then they will take the 20 percent dataset and they will apply that model to that dataset as if you don't know what the people's answer to whether there were any pesticide exposure or not is. And then you compare it to what they actually did. So you can see how often you got it right and how often you got it wrong.

In this case for any pesticide use, 85.68 percent were what they observed in this 20 percent, and they predicted 85.25 percent. So the -- you would think the bias here is .43 percent. But that is the minimum bias, and I'll explain that in a minute. But that's how they do the first stage. You predict if you had any pesticide exposure for the people who don't have the -- haven't responded.

MS. GREENWALD: Okay. Next slide.

BY MS. GREENWALD

Q. This is almost the same thing. Again, 20 percent are pulled off to the side. Now you would model the relationship between pesticide use ever/never for each pesticide using the same process, survey responses and logistic regression for the 80 percent that I had from the 60 percent that responded to both. I apply the model to the holdout dataset.

This is a little more complicated because this is multiple imputation. So they apply the model to the holdout dataset and they get a probability that each individual used each pesticide. So I may get a probability of .9. You may get a probability of .1. But I -- for me to do my regression analysis, my logistic regression analysis and understand what's going on here, I have to turn that into a: Yes, you were exposed; or: No, you were not exposed.

So they then flip a weighted coin. Let's put it that way. If you're at 90 percent and they flip this weighted coin, which half of the time -- 90 percent of the time is heads, 10 percent of the time is tails. If they flip that coin and it's heads, then you're going to have the -- you're going to have the exposure. If it's tails, you're in the 1 percent -- 10 percent chance group that even though you had a high probability, we're going to tell you you didn't have the exposure.

They do that five times. So that -- you now have five datasets for each person. Then they average those datasets to

come up with -- they analyze each one separately. They get an evaluation. And they give you the average of your evaluations. That's why it's multiple imputation. You try to avoid the problem of just doing it once and getting a randomly weird answer.

So they applied it to the 20 percent holdout people. And for glyphosate use there they observed 52.73 percent had glyphosate exposure, but they only predicted 45.42 percent. So now the minimum imputation bias is 7.31 percent. That means if this holds in the larger dataset, 7.3 percent of the people who really are exposed are being put into the unexposed group.

So you have misclassification bias, not just -- not undifferentiated misclassification error. You now have misclassification bias because you are specifically moving people only in one direction.

They also calculated this thing called a Brier score. And I got a lot of questions in the deposition about Brier scores and I felt I really hadn't done a very good job of noting what Brier scores were, so I spent more time on it.

A Brier score is -- for the 20 percent that I held out, each person in that group has either glyphosate exposure or does not have glyphosate exposure. But they also predicted a probability that they had glyphosate exposure or not. And so what they do with a Brier score is they subtract either one -- I'm sorry.

You've got this probability. If you had the tumor, they subtract -- the exposure, I'm sorry. They subtract that probability from one. If you didn't have the exposure, they subtract that probability from zero. They do that for every person. Each time they do that, they raise that to the second power, they add them all together and take the mean. And that's what a Brier score is.

Now, let me give you an example of a Brier score. If -- if instead of going through all this fancy modeling, I give every person .5 chance of having the exposure. So right in the middle. Completely uninformative. Then I would be subtracting .5 from 1, or I would be subtracting .5 from zero, squaring that, which is .25 in every case. Adding it all up and averaging it and I would get .25. That is a random -- a perfectly random Brier score.

Follow?

THE COURT: I'm not sure. But I'm starting to think maybe the point of this is to not be able to follow it.

THE WITNESS: No. I'm sorry. That wasn't my point.

It's hard to explain. One thing I was asked in deposition was: Is this Brier score, .225, bad or good?

And Brier scores typically, you want them to be small. The smaller the Brier score, the better you are in your prediction.

But I couldn't tell if this was -- this .225 was really

bad, marginally bad, what is it? The .25 for the random case and .225 are very close to each other. So you've only done slightly better than just giving everybody a 50 percent probability of being exposed. That was the point.

Good?

MS. GREENWALD: Okay. Next slide.

A. Yes.

There are two more steps in the multiple imputation. Once you've assigned somebody exposure, you have to figure out how many days per use they are going to be exposed and they use something called "stratified random sampling" to generate that. And while I have concerns about that, I have less concerns.

So I -- I will explain it to you, if you want to know what it is, but it seems to be okay.

And for first year of use, they also use stratified random sampling. A little more complicated this time. But, again, I don't have a lot of concerns with that. I have much more concern with yes, no exposure.

MS. GREENWALD: Next slide.

A. This is data straight out of Heltshe.

BY MS. GREENWALD

Q. So what is the Heltshe paper, just for context for the Court, please?

A. I was going to go there.

The Heltshe paper is the paper where the Agricultural

Health Study puts out their imputation approach. They apply it to the dataset and show you how well it worked. So that's what all of that holdout dataset was and everything else.

And if you remember, I told you for glyphosate they observed 5. -- 52.73 percent. Predicted was 45.42 percent. The difference between those is minus .731 percent. And this little dot on the right-hand side all the way at the bottom circled with a circle and glyphosate on top is that data point.

What I have on the X-axis is the percentage of participants with glyphosate exposure and on the Y-axis this bias term, which is the difference between the observed and the predicted for glyphosate.

THE COURT: I take it that all these other dots, all these other points, are all of the other chemicals that AHS looked at?

THE WITNESS: That's correct. And the X-axis is labeled incorrectly when I go to the others because it's the percentage of participants with at exposure. Okay? You can see atrazine, 2,4-D and glyphosate clearly have the biggest bias terms, and the others have much smaller numbers in their bias.

The thing to note with this slide is that if, indeed, this data were unbiased, the dots would go up and down on top of the line that's running horizontal. That's zero. They would just be above and below it at random.

But what you have is a systematic bias. The line that I've drawn through the data shows the curve. And so what happens is the bias gets bigger, the larger the exposure. And since glyphosate is the largest exposure in this dataset, it's got the biggest bias.

Okay. So what does that mean in terms of exposure misclassification?

If we can go to the next slide?

I tried to explain this to several people and they didn't understand it.

BY MS. GREENWALD

Q. I was one of them.

A. So I'm going to do something simple, a little cartoon. So here I've got ten people and they are observed to have no glyphosate exposure or glyphosate exposure, five observed without and five observed with.

I go to predict the exposure using my imputation method. If the imputation method is perfect, really good, I get the five unexposed exactly right and I get the five exposed exactly right. So that's perfect agreement. Okay?

Now, I can't do this with 47,000 people, but I can do it in a table.

And if I could have the next slide?

And the table looks like this. You have the observed exposure --

Q. Slide 19. I'm sorry, I just want to make sure we are on the right slide. Slide 19.

THE COURT: 19?

MS. GREENWALD: Yes. 18 was just the perfect agreement box.

BY MS. GREENWALD

Q. Sorry, Chris.

A. You have got observed exposure; yes, no. It's 50 percent. 50 percent were exposed; 50 percent were not posted.

You have the imputed exposure; yes, no. 50 percent were yes; 50 percent were no.

And every "yes" that was observed is also a "yes" for imputed. And every "no" that was observed is also a "no" for imputed. Perfect agreement.

Next slide.

Now, let's look at what happens when it doesn't work. So, again, we've got observed five with, five without exposure. My predicted exposure for the unexposed, I predicted three of those to be exposed and two to be unexposed. So I missed three. And for the exposed, I missed two. I said they were not exposed when in truth they were exposed.

Next slide.

So this is some agreement. I -- I misspecified five of the people basically. And this table looks as follows.

There. So there is the -- whatever slide we are on.

Q. 22.

A. Now, the thing here that's interesting with this table, so you have observed exposure 50 percent yes, observed exposure 50 percent no. The imputation exposure is 60 percent yes and 40 percent no.

So you think they only missed 10 percent. But, of course, that's not true. For the yes, the truly observed yes, 20 percent were predicted as no. And for the truly observed no, 30 percent were predicted as yes. So you actually got five of the people wrong, which is 50 percent were actually wrong. Even though the difference between the margins, the 50/50, 60/40 is only 10 percent.

JUDGE PETROU: And does it matter at all that you've got two wrong on each side of it? We have been having endless -- not endless. We have been having extensive conversations regarding differential bias, regarding non-clinical bias, and that question pops in my head as you're going through this.

THE WITNESS: Exactly where I'm going next. But I'll explain in it this picture, because it's simpler with this picture.

A. I've got three on the no, yes; two on the yes, no. So that's approximately the same. So this is about -- this would be pretty much non-differential exposure misclassification. I'm flipping them back and forth. And this would work you to

the null.

Next slide.

Okay. Let's look at the exposure to any pesticide case from Heltshe, et al. And I don't know why this came up this way.

Could you go to the next slide?

Okay. We'll do it here. Sorry. That was supposed to be also circles and stuff, but that's okay. We'll just do it this way.

If you remember, they told us that there was 85.68 true observed exposure, and 85.25 percent imputated exposure as to yes. And that leaves 14.75 percent for imputed and 14.32 percent were observed for the nos.

Now, the best-case scenario is where virtually all of the observed yeses are imputed yeses. And we get 85.25 percent in that hole. And then all of the observed nos are predicted nos. Hopefully, as many as you can get. And that tells me that this one block up here, the observed yeses that are imputed as no, has to be .43 percent. And if I do it that way, I get exactly the numbers they gave me: 85.68 and 85.25.

But that's just one case. I can get the exact same numbers by doing it a different way. The worst-case, I'm going to put as much weight as I can in the observed nos being imputed yes and the observed yeses being imputed nos. And this is the worst-case here.

Now, in this case you've got 14.32 percent observed yeses that are predicted as no and 13.89 observed nos that are predicted as yes. So when you add those two numbers up, that's 28 percent error, not .43 percent error. 28 percent. But both of these numbers, these percentages, are approximately equal. This is going to be non-differential exposure misclassification, almost certainly.

Okay? Now let's look at glyphosate, which is the next slide. Here we go.

So same thing for glyphosate. 52.73 percent observed as having glyphosate exposure. 45.42 percent predicted. Fill out the rest of those edges of this box. Now let's make it as good as it possibly can be.

Again, you force as many as you can into yes observed, yes imputed, as many as you can into no observed and no imputed. And you end up with only 7.31 percent error in the observed that are imputed to be no. That is non-differential exposure misclassification. 7.31 percent are being put in the wrong place. And only one way, not both ways.

THE COURT: So it's non-differential or differential?

THE WITNESS: It's differential.

A. But what's the worst-case?

Next slide.

Again, same numbers at the margins, but now you can see the yeses that are really imputed as nos and the nos that are

imputed yeses are much bigger. And you've got roughly 98 percent error here.

Now, I don't believe that's what they had. I really don't. It's somewhere in between here. I'm pretty certain it's not the best case and I'm pretty certain it's not the worst case. If it were the worst case, they shouldn't have even published the paper. But it's somewhere in between. I don't know what it is.

But even still, the difference in the yeses that are imputed nos and the nos that are imputed yes is still 7.31 percent. And so you still have differential exposure misclassification and you could have a lot of non-differential exposure misclassification error.

Next slide.

So that covers the imputation, so if you have any questions about the imputation, we should do them now.

JUDGE PETROU: Can we go back -- let's actually go back. In my head I'm going back to the part where you had all the numbers underneath the line where you were saying that if it were random, there would be some above and some below, and the most severe one was glyphosate.

So did I read that chart correctly or not to basically say that it's your opinion that this multi-step imputation process led to a differential exposure misclassification, all of which skewed on the side of fewer people who actually had glyphosate

exposure being shown as having glyphosate exposure?

THE WITNESS: That's correct. But the bigger problem is the people that actually had glyphosate exposure are in the control group.

JUDGE PETROU: Got it.

THE WITNESS: And the unexposed population.

MS. GREENWALD: I didn't know if that was the slide you wanted up.

JUDGE PETROU: I don't need it up.

MS. GREENWALD: Okay. All right. Thank you.

THE COURT: So this looks like your last slide on Andreotti.

MS. GREENWALD: Correct.

THE COURT: Do you want to go through this and then we'll take a break?

MS. GREENWALD: Sure.

BY MS. GREENWALD

Q. So we're on Slide 26.

A. So there were other problems with the Andreotti study. As mentioned by Dr. Ritz, the exposed responses for the lifetime days and intensity weighted days were compared to the controls, rather than to the lowest quartile. And in the -- if you recall in the De Roos 2005 study, they compared to the lowest quartile because -- tertile in that case, because they felt the unexposed were different than the exposed groups in key

characteristics.

And so they -- they wanted to avoid having a problem by going to the unexposed group when they were doing dose-response.

If they had done that in this case -- let's look at it. For lifetime days, they show an odds ratio of in quartile 1, .76; quartile 2, .87; quartile 3, .85; and quartile 4, .80.

Now, it's a funny thing about these odds ratios. You've got -- you've got something on the top and something on the bottom. The bottom is the referent population. The top is the group that you're comparing it to. So, for example, to get the odds ratio for quartile 1, you're looking at the odds in quartile 1 of the exposure, divided by the odds in quartile 1 of the unexposed group.

Now, if I take the odds of quartile 1 -- of quartile 2 and divide it by the odds ratio of quartile 1, I cancel out the control odds ratios and now I'm looking at an estimate of the odds ratio where I'm using quartile 1 as the referent population. I can't do the confidence bounds that way. It's much too complicated. I can't actually do it. But I can show you what the middle number would look like.

And when you do that you now see an odds ratio, the modified odds ratio -- that's what MOR is -- of 1.1, 1.12 and 1.05.

Now, that partly might reflect upon the bias, the exposure

bias that is caused by the imputation, but it's also -- there was considerable difference between this control group and the exposed group in this population as well.

And so I would have liked to see them do the same thing that De Roos did in 2005. I think it would have given us a completely different picture.

The same is true with the intensity weighted odds ratios, so I just did the exact same thing there. We talked about the -- you talked with Dr. Ritz and others about the dramatically increasing use from 2000 to 2010 of glyphosate in these populations in the United States. I would have liked to see the analysis using the incidents up until 2005, not all the way to 2013.

Now, they did give me that number. They gave me one number. The 1.04 in the highest quartile for the intensity weighted value was 1.04 with a range of .7 to 1.57. This is the complete case. This is where they threw out everybody who didn't have exposures, that didn't answer the second questionnaire, and just worked with the ones who did answer the second questionnaire.

There was a lot of discussion about undifferentiated exposure misclassifications pulling you to the null. If this number were the correct number -- I don't know the rest of them. I can't tell you anything else. But it's above 1 and you're pulling it to the null. That's exactly what I would

expect to see.

So that entire business about the Andreotti paper being -- having odds ratios below 1, I think is partially due to the bias in the imputation and I think it's partially due to, as Dr. Ritz said, potential unused confounders, potential for confounders that you did not put into the model. I think both of those play a potential role in bringing it down to 1.

I think the exposure misclassification in this is severe. I would have expected that the odds ratios from this would all be near 1. I see exactly what I expected in this study, and it's null. I think it was going to be null, given all the problems with it. And so it doesn't enter into -- with a lot of weight into my overall evaluation. It gave me exactly what I expected to see from a study with these problems.

Okay? And I think that's it for Andreotti.

MS. GREENWALD: It's break time.

THE COURT: Okay. Why don't we take a ten-minute break? We'll be back at ten minutes to the hour.

(Whereupon there was a recess in the proceedings from 10:36 a.m. until 10:53 a.m.)

BY MS. GREENWALD

Q. I just have a couple of follow-up and then I want to talk a little bit about Slide 27.

Dr. Portier, if you could look at De Roos 2005, which is up on the screen? It's also Exhibit No. 23 in your notebook.

Oh, your screen is not working?

It was. Okay.

A. There it is.

Q. Now, you testified earlier before the break about the follow-up on exposed and unexposed group, correct?

A. Correct.

Q. Can you explain what you meant by that using De Roos 2005 in Table 1?

A. Yes. This is -- so there were a lot of things in the survey questionnaire that are asked about: State of residence, age, sex, et cetera.

This table is all of the -- or some of the characteristics from the applicators in the AHS study that were significantly different between the most exposed group and the never exposed group. And this was the justification that De Roos gave for not using the control, the never exposed population, in their evaluation of intensity and length of exposure, and instead using the low exposure group. Because it looks more like the high exposure group than the never exposed group.

And so I just wanted to illustrate that it's not just a few things. There are a number of key characteristics here that could play an important role in that type of comparison: Use of other pesticides, alcohol use, smoking use. They are all different between these groups.

JUDGE PETROU: I'm sorry, because I should understand

this and I don't.

What was the justification, therefore, for using the lowest exposed group as opposed to the never exposed?

What are the differences that we see within those two groups that would lead them to say: We're going to use the low exposed rather than the control never exposed?

THE WITNESS: Let's just take one, for example, 2,4-D use. In the control population, 53.3 percent used 2,4-D. In the lowest exposed 75.2, in the highest exposed 85.1. The lowest and the highest are much closer to each other than that than is the controls, the unexposed.

Beyond high school, 31.3 percent in the unexposed, 42.1, 49.9. That's the pattern that they saw that concerned them. It looked like the exposed groups were much more closely related to each other than the unexposed in things that should not matter. And in doing these types of studies, you want those things that should not matter to be about the same in all the groups. That way it makes your assessment of the exposure response stronger because everything else is about the same.

JUDGE PETROU: And things like the use or exposure to 2,4-D should not matter?

I mean, you just said that what you want is that -- the things that do not matter to be similar in the group that you're using as the control, which is not necessarily the control group, and the high exposed group, right? And we went

through a number of factors, including high school degrees and all the rest of that.

But the first one that you pointed out was 2,4-D. Is 2,4-D in that group of things that should not matter?

THE WITNESS: I guess I didn't explain it right. In order to compare apples and oranges -- let's take an example. Suppose the -- 2,4-D is not a good one.

What you would like to see in a laboratory study, when I was talking about the toxicology study, we control everything except dose. And the reason is that makes it a very clean hypothesis.

In an epi study, you, of course, can't control everything, but you match your cases to your controls. You try to make them as similar as possible so that when you make comparisons across the thing you're interested in, glyphosate, those other things aren't confounding it. Even if you take them into effect --

JUDGE PETROU: Or if they are confounding it, it's equally on both sides of the table?

THE WITNESS: Yeah. And if you take them into effect in confounding -- if you see such stark differences across multiple, multiple characteristics, then you start worrying about are there things I missed that are messing up my unexposed group. You -- it's not -- it's not unheard of to use the lowest exposure group as your referent group when doing

these types of analysis.

JUDGE PETROU: And when you looked at this chart, it's your opinion that it was appropriate here to use the lowest exposed group as the control group essentially?

THE WITNESS: Yes.

BY MS. GREENWALD

Q. So I want to ask you a question about AHS again.

You testified that there could be potential selection bias on the complete response in the AHS, is that right? Did you not hear me? I'm sorry.

A. Yes.

Q. Maybe I could ask it another way.

What was the significance to you in the -- in the Agricultural Health Study not allowing people who had NHL into the study?

A. Okay. So when you do a cohort study like this one, a prospective study, you start at one point and start following people who get the disease.

Now, let's say there was exposure prior to the start of the study, so people have been in this industry for 15 years. Some of them could have been exposed for 15 years.

Some of those people might have gotten NHL and died because of that exposure, or quit working. They are no longer in the field.

So by the time you start the study, you've selected out a

subpopulation of those who would have been susceptible to the NHL. So you've got a population which already has some degree of selection on it in -- that's directly related to the thing you're interested in.

So you generally don't go back and use their past exposure history to do the analysis of the current cohort study. You move it forward from day one, hoping that any selection bias that came in because you selected people, as time goes on disappears and you -- you're getting a real good picture of what's happening in this population.

Q. One last clean-up question.

Can you explain why the lag issue for the Agricultural Health Studies is -- is not solved by the retrospective history?

A. I -- I guess I thought I did.

Q. I think -- well, I just want to make sure there isn't anything you want to add to that. I mean, I realize you just somewhat addressed that?

A. If I was asked the question: Can you design a cohort study, prospective study, where you take a retrospective history of somebody's exposure and we go five or six or seven years, and then let's look at it. It still has that selection bias issue, but my -- my response would be: No, I do a case-controlled study. It's more powerful. It's less expensive. I can do it faster. It would be the more

appropriate study when I have to rely upon past historical exposure.

Q. Okay. So by the way, the slide deck is Exhibit No. 461.

MS. GREENWALD: I think, Your Honors, we're going to skip past Slide Number 27, which is the NAPP, because it's not in his report and it's not in his supplemental deposition either. So we're going to just skip that. We'll replace that.

We'll take that slide out in the final version for the Court and we're going to move on to Slide 28.

BY MS. GREENWALD

Q. So what does this summary plot show?

A. We should take this one out and the next one as well. Well, we will take the NAPP part out of it.

Q. Right. Because I think the plot is still relevant, and then we'll replace that.

A. So the -- I've added the Andreotti ever/never to this picture. And they didn't provide it, so I actually can't add it to the picture. So it doesn't change the plot summary of the ever/never exposures.

The next slide.

So now looking at the Andreotti study within the context of all of the other studies looking at exposure, time, response, type, pictures. You can see it clearly is much lower in terms of the response, the odds ratios, than the other studies were, and some below, some above the 1 odds ratio.

But as I said before, this is exactly what I would expect to see. Because of the flaws of the study, I would expect to see uniform odds ratios and because of that, and it's given me what I expected, it doesn't change my opinion. It doesn't contribute to a change in my opinion.

Q. When you say it doesn't show what you expected -- sorry, that it shows what you expected, do you mean by that that because you expected it to have the results it has and you expected it to -- to bias the numbers to the null, that you do not believe that it's relevant to your ultimate overall opinion in this case?

A. So, yes. The numbers were appearing biased towards the null, potentially even below the null. It's still important to my decision. I want to be clear. This is -- this is a good study. Regretfully, a bad exposure metric, but it's a good study. But the problems with the study in the last round make it have such poor statistical power you would expect it to be odds ratios around 1. And that's exactly what you're seeing.

So it's not that I'm discounting the study or throwing it away. I'm evaluating all aspects of the study, seeing what I expect to see, which is nothing, and that has no change on what I already have concluded.

Q. And that comports with your Slide Number 1 on how you evaluate causality, is that right?

A. That's correct.

MS. GREENWALD: And so if you can go to the last slide?

BY MS. GREENWALD

Q. You had this up before on Bradford Hill, and you wanted to look at it again and explain to the Court how this factored into your overall causality opinion.

A. So after looking at Andreotti and everything else, but in looking at Andreotti, I was willing to reconsider Table 18, but nothing changed. This is from my expert report.

I still believe the observations in the epidemiology studies are consistent.

I believe that the strength of the observed association is high enough to warrant a strong opinion there, mostly driven by the meta-analyses.

The biological plausibility is very strong. There is no doubt about it. Multiple cancers, multiple species, not due to chance, increased risk of rare tumors; all the things that make this a very strong category.

Biological gradient, that's moderate. The De Roos 2005 study doesn't see much of a gradient. The other two studies that did this do. That's not a lot of information to drive a statement about biological gradient, so I gave that a moderate.

The temporal relationship of the observed association is, of course, satisfied. The exposure came before the cancers.

Specificity is not needed because NHL has many other

causes.

The coherence here is strong. "Coherence" means can it get into the body? Is it --

THE COURT: Can I have you rewind to specificity for a minute?

THE WITNESS: Yes.

THE COURT: So what you say in this summary is I think different from what you said in your expert report on specificity. In your expert report you said there are other causes of NHL, so this group of cancers is not specific to glyphosate. There is little support for specificity.

So here you say not needed. Here you say -- in your expert report you say there is a little support for.

So I have a couple questions about that. The first question is: Are you defining specificity correctly in the apparently, no.

And so she says that specificity -- if I recall correctly, she says that specificity is actually strong.

THE WITNESS: If we could go to my expert report?

THE COURT: Page 75.

MS. GREENWALD: It's Tab 162.

THE WITNESS: 162. I would like to look at the definition of "specificity."

THE COURT: Sure.

THE WITNESS: It is -- here. Page 5. Take a minute

to look at what I wrote.

THE COURT: I'll get there as well. Page 5?

THE WITNESS: Yes. Bottom of Page 5.

(Brief pause.)

THE WITNESS: To the best of my knowledge, that is the definition I used. And as far as I understood the Hill criteria, that is the definition he was looking at.

If you see a disease that only one chemical, one exposure, seems to cause, mesothelioma and asbestos is a good example of that, then it's very, very specific to that disease. And so causality for that disease is more strongly established because it's the only cause you have.

THE COURT: So if Dr. Ritz says that there is strong specificity because in the studies of glyphosate there is no association shown with other types of cancer, there are only associations shown with NHL, she's using the incorrect definition of specificity within the Bradford Hill criteria.

THE WITNESS: I would have to look at her -- her report and go through the definition that she had in the report.

THE COURT: Okay. Forget about Dr. Ritz. If I articulated the specificity criterion in that way, am I articulating it incorrectly?

THE WITNESS: I -- I would -- I would say probably yes. The issue with -- the criteria are intended to walk you

through what would add to a causality argument.

So if I have benzene, and benzene exposure causes five separate cancers, and all of a sudden I'm looking at benzene and it causes a sixth cancer, the fact that it only causes the one cancer doesn't give me nearly as much added weight as saying: Look, it causes five other cancers. This is one that's like it. So I would add more weight.

So that definition, as you've expressed it, would not be my definition of specificity.

My definition is, if it's really specific, if NHL has no other causes and all of a sudden you've got one, I don't have to worry about confounders, I don't have to worry about other things, because I don't know any. It strengthens the finding.

THE COURT: Okay.

BY MS. GREENWALD

Q. Would the example that the Court gave fall under consistency -- would it fit under the category of consistency of the observed association of Bradford Hill?

A. Partially. I think partially.

THE COURT: Why? I mean, that's -- I mean, I thought that was about multiple studies.

THE WITNESS: But, again, you're -- multiple studies, but also all of them giving you the same result, the same direction. And even if -- if they all deal with -- suppose I have five cohort studies and the only thing I saw in the cohort

study was NHL in all five. It's multiple studies pointing to the same thing. That's -- that's as close as I can come.

THE COURT: It's multiple studies pointing to the same thing, but it's not -- that doesn't --

THE WITNESS: Only thing.

THE COURT: -- that doesn't incorporate the concept of showing an association between the substance and NHL and not showing an association between the substance and other cancers, right?

THE WITNESS: Yeah. That would be right.

THE COURT: So are -- is that to say that it's not particularly useful to learn that, you know, these studies are showing no association between glyphosate and other cancers while they are showing association, potentially showing association between glyphosate and NHL?

THE WITNESS: I haven't thought about it. I would really have to sit back and give that some thought.

THE COURT: Okay. Thanks.

BY MS. GREENWALD

Q. So Slide Number 30, is that the same table that you have on Page 77 of your report? If you can turn to Page 77, Table 18?

A. Yes.

Q. Okay. If you can turn to Page 78, please.

(Witness complied.)

Q. Can you read what you -- the section that starts -- the paragraph that starts "In my opinion," please. It's the next page after 77.

A. (As read)

"In my opinion, glyphosate probably causes NHL and given the human, animal and experimental evidence I assert that to a reasonable degree of scientific certainty the probability that glyphosate causes NHL is high."

Q. What do you mean by "high"?

A. That's -- that's -- it's a hard question. In my own opinion of what I mean by "high," 100 percent would be absolutely undeniably certain it causes glyphosate -- it causes NHL. 50 percent would be, ahh, maybe. I would give high 90 percent chance. So this sits at about 90 percent in my scale.

Q. And when you say "your scale," is that based on, again, going back to Slide Number 1 in your evaluation of causality and how you've looked at the totality of the evidence here?

A. Yes, of course. In any evaluation like this, there has to be some statement at the end that summarizes the degree to which you believe there is a relationship between this and NHL. There is a variety of scales and methods that are used, most of them are about the same as what I'm saying here. Words like "very strong," "strong," "high probability." Those are typical things to use.

Q. Okay. And when you are reaching this conclusion on Page 78, you're using all the different factors, such as epidemiology -- all the evidence, I should say, and the data, epidemiology toxicology and mechanistic data in reaching that conclusion?

A. Yes. Absolutely.

MS. GREENWALD: I don't have any other questions, Your Honors.

(Discussion held off the record between plaintiff's counsel.)

MS. GREENWALD: I might. I'm sorry.

THE WITNESS: Your Honor, if I could respond to one of your questions we didn't get back to?

THE COURT: Sure. Why don't you give them a chance to confer real quick and then you're free to do that?

(Brief pause.)

BY MS. GREENWALD

Q. An important question.

So just to clarify one thing on specificity. Now, you stated -- while you stated that non-Hodgkin's lymphoma is not specific to glyphosate, we understand that, is it your opinion that glyphosate exposure is specific to NHL? In other words, the reverse of that?

A. I have to go through my head and see if I've really taken the time to look at all the literature on this for glyphosate,

the broader array.

Yeah. I would say I don't see any other strong cancer out there that appears to be associated with glyphosate in my experience in looking at this literature.

Q. Other than non-Hodgkin's lymphoma?

A. Other than NHL. Well --

Q. So you wanted to --

A. -- that's not totally correct because -- that's changed. The classification is now NHL.

Q. It's multiple myeloma?

A. Yes, NHL.

Q. So multiple myeloma is now in Lymphoma Society considered a subset -- a subpart, subset non-Hodgkin's lymphoma, correct?

A. Yeah, under ICD-10.

Q. Okay. You wanted to clarify something.

A. You asked me to look at Page 75 and the statement "there is little support for specificity," under "specificity." That's my jargon for saying it's not needed. It doesn't add to the causation argument. I just wanted to close that loop.

THE COURT: One question I have.

So I was going back through your testimony from last time, and you talked a little bit about the IARC's conclusion, the IARC classification. And you make the point that it's important to the folks at IARC that people know that, you know, if IARC says something is a probable carcinogen, it does not

follow that if you are exposed to it, you will probably get cancer. Right?

That -- and just to use -- you look like you had a little doubt on your face, so I'll just read exactly what you said so there is no ambiguity. You said:

"What IARC is saying is that when they" -- and by the way, I'm on Page 542 now.

"What IARC is saying that when they say it's a probable human carcinogen, they don't want the public to think that means if you're exposed to glyphosate, you'll probably get cancer. That's not what it means," is what you said.

"It means that the literature is so strong that we think it's probable that humans will get cancer at some level of exposures to glyphosate."

That's what you said in your testimony.

And we know from the material that the IARC puts out, right, from the preamble, and also from the paper that it put out in response to all of Monsanto's attacks on its classification, that, you know, IARC draws this firm distinction between hazard, assessment and risk assessment, right, and explains that what it does is hazard assessment.

You are offering an opinion that is beyond a hazard assessment opinion, as I understand it, is that correct?

THE WITNESS: There were great discussion on this

between you and several of the others.

Let me give you my definition of hazard assessment, my definition of risk assessment, and why this is slightly different from what -- what I heard in the discussion.

Hazard identification is exactly what you were talking about a minute ago. Is this a hazard to humans under some exposure condition? How much weight of evidence can we put in that statement?

The risk assessment is then much more specific under these exposure -- this exposure scenario. What is the risk to the population of this, of exposure to this compound?

Now, when agencies and many groups do those types of evaluations --

THE COURT: What types of evaluations?

THE WITNESS: Hazard and risk assessments.

They most of time don't even talk about the cancer, because most of the time it's driven by animal data and not epidemiology data. It's -- it's seldom that they include epidemiology data as strong as you have here.

In this exercise, as I understand it, we're not just saying it could be a carcinogen, but I have to speak directly to NHL. And so that is kind of outside of the usual hazard assessment.

But IARC does speak directly to NHL. Their limited evidence in humans is not only that there is an association,

but that a causal association is credible. And the only cancer they looked at is NHL.

So that's clearly, they believe, a causal association is credible for NHL, and so do I.

THE COURT: And so your opinion on the epidemiology is the same as the opinion articulated by IARC on the epidemiology?

THE WITNESS: It hasn't changed. IARC has very rigid classification rules, the limited evidence of carcinogenicity. That is an accurate description of the data.

I think we could have gone further in describing other things, or they could have gone further in describing it, but that's an accurate description of the data.

THE COURT: Okay. So I mean, I think you've testified to this already, but your -- you know, one could view that phrase "limited evidence of carcinogenicity in humans," and we know that in this context when they are talking about carcinogenicity, they are talking about NHL because that's what the studies show us, but when you -- when you -- when you think about that phrase, "limited evidence," you know, you might say: Well, that doesn't sound very powerful, "limited evidence."

And so I gather that what you have said and what you would still say today is that we have this -- this limited evidence. You can't rule out chance, confounding bias, but the -- when you combine it with the animal evidence and the mechanism

evidence that causes us to conclude that the possibility of chance, bias, confounding in the epidemiology data is lower? Is that a fair way to think of your opinion? And if not, feel free, obviously, to correct me.

THE WITNESS: I would more carefully say that my concern for the chance, the bias and the -- the potential chance, the potential bias and the potential confounding in the epidemiology is lowered. My concern for it is lowered. It's still there, if it's there at all, because we can't really measure it.

But my concern for it is less because now I've got all this other evidence saying it's really biologically active. It looks biologically active in the same system, hemapoietic system, with a very similar tumor in the mice.

So, yeah, I'm much more comfortable to say that I'm much more believing to human evidence.

THE COURT: Okay. And I'll let Mr. Lasker take over, but I want to say now, while it's on my mind, that I do want you to address the animal stuff a little bit after the lunch break. I don't know if you spent any time preparing to talk further about the animal stuff.

So I want to kind of flag for now a couple of the things that I want you to address after the lunch break on the animal stuff so you can, you know, sort of gather your thoughts on it.

And, one, I guess this is probably the biggest question

that I have for you, is: If we took out -- if you took out all of the stuff you did on pooling in the animal context, what would your opinion be and what would be the basis of it?

You don't need to go back and repeat everything that you've said, but if you could just sort of summarize that for us.

And then maybe a -- a smaller question that I had on the animal stuff is, you know, in epidemiology, you know, there is this emphasis on, you know, focusing on published studies. And I -- and I think the IARC says we only look at the published studies, the published data, right?

And I wanted to explore whether that -- that same emphasis is supposed to be placed on published studies for the animal data, or if it's sort of more common in the toxicology context to be relying on unpublished but regulatory studies from the agencies.

So those are the two questions I can think of right now that I want you to address after lunch.

But I'll turn it over to Mr. Lasker right now.

MS. GREENWALD: Could I ask one question on the IARC, a closing question?

THE COURT: Sure. Go ahead.

MS. GREENWALD: It's up to you, Your Honor.

THE COURT: Go ahead.

BY MS. GREENWALD

Q. Chris, if I could -- Dr. Portier, if I could just ask a question to wrap up the IARC issue.

Although IARC could determine that a substance is a probable carcinogen even at exposures not occurring in the real world, is that what IARC found in regard to glyphosate here?

A. No. Of course not. The epidemiology studies are evidence of an effect in the real world.

Q. In real people?

A. In real people at current exposures.

MS. GREENWALD: Thank you.

CROSS EXAMINATION

BY MR. LASKER

Q. Dr. Portier, you won't need that binder. We'll give you a new binder so you don't have all that stuff on your desk.

(Brief pause.)

MR. LASKER: Your Honors ready? Okay.

BY MR. LASKER

Q. Dr. Portier, you talked already about your epidemiological experience, so I don't want to rehash that, but in your expert report -- and that's at Tab 1 in your binder and it's at Page 6 -- when you begin your discussion about the epidemiology, and in particular sort of the last sentence of that first paragraph under "Relevant Epidemiologic Studies," you state there that:

"Other experts will be discussing these studies as well as their strengths and weaknesses."

Correct?

A. That's what it says.

Q. And am I correct in my understanding that you were referring there to the epidemiology experts who submitted reports in this case?

A. Probably.

Q. And then you explain that for the purposes of your opinion in your report, you are focusing on using the results of the studies in evaluating causality and because of that you only briefly describe each study, correct? It's in your report.

A. It says:

"I will focus on using the results of these studies in evaluating causality, so I will only briefly describe each study."

Q. And when the new 2018 Andreotti study was published, you relied upon the expert reports that were prepared by Dr. Ritz in communications you had with journalists and some government officials in Europe, correct?

A. I don't know what you're -- you're asking me.

Q. Okay. Well, maybe it will help if we can turn to Tab 4 in your binder. And this is one email that you produced to us in preparation for your supplemental deposition after the Andreotti study. And this is an email dated November 9, 2017,

which is the day the Andreotti study came out in electronic form. Do you recall that?

A. I don't know that that's the exact date, but I do recall that email.

Q. And you explained -- I'm sorry.

A. I do recall the email.

Q. And you explained to me in your deposition that Martin Pigeon is a reporter of some type?

A. He works for the Corporate Europe Observatory and he also wrote a book.

Q. And so he raised questions with you about the Andreotti study and you forwarded to him Dr. Ritz's, I take it, original expert report and her rebuttal expert report in this litigation, correct?

A. That was on the Right to Know website. I sent it to him. He asked for it.

Q. Well, he -- he asked you for your views of the new AHS study, and in response you sent Dr. Ritz's expert reports, correct?

A. No. We talked on the phone and I asked him if he had seen her expert report, and I sent it to him.

Q. Okay. And then if you look at Tab 5, this is on November 12, 2017. You received an inquiry from a Robert -- and I don't know if I'm pronouncing it correctly -- Bellé. And he is a government official in Europe, an advisor to the French

deputy in Europe, correct?

A. I believe that's his position.

Q. And in response to his requests, again, you forwarded the expert reports of Dr. Ritz, correct?

A. That's correct.

Q. And I'm going to get back to some other points you make in this email, but just to continue on the next page behind Tab 5, you also sent Dr. Ritz's report to a Tiffany Stecker, and she is also a reporter, correct?

A. That's correct.

Q. And you also present some other opinions or some opinions you had at that time in both your emails to Mr. Bellé and to Ms. Strecker that are -- or Stecker, I'm sorry, that are set forth in your expert report, correct?

A. No.

Q. Maybe I misspoke. Let me say that again --

A. The opinion as expressed here, as I told you in the deposition, is wrong.

Q. Okay. And I did misspeak. You're going to where I was, and I said expert report. I'm sorry.

You provided some opinions that you had reached with regard to the Andreotti study as of that date, and as we talked about in your expert report -- I'm sorry, I said it again -- in your deposition, you agree with me now that the opinions that you shared at that time based upon your review of the Andreotti

study were, in fact, incorrect; is that right?

A. One specific part of those opinions were indeed incorrect, in the way in which I describe the imputation.

Q. Okay. And I was going to walk through those and we can do that now.

The first opinion that you offered in this email is similar to or the same as one of the opinions you've offered today, which is your recalculation of the odds ratios in the Andreotti study by comparing them to the low exposure group, correct?

A. That's correct.

Q. In your expert opinions here today and in your expert report you actually provide a second calculation in which the numbers are a little bit higher than the numbers you have here, correct?

A. I don't think so. These are numbers for one of the two areas. I don't remember which one this is.

Q. Okay. We'll turn to that in a minute.

The second opinion you offered is that -- is with respect to the sensitivity analyses that were set forth in the Andreotti study, correct? That's, I guess, the fourth paragraph or third paragraph, depending on -- which starts:

"In addition in their discussion of their sensitivity analyses."

A. Uh-huh.

Q. And you were offering an opinion there as to the impact of the sensitivity analyses on your interpretations of the Andreotti study, correct?

A. Yes.

Q. And in my deposition I asked you -- and we walked through some of the findings of the sensitivity analyses, and you agreed with me that the risk ratios that were calculated with the three different sensitivity analyses conducted in Andreotti were all in the same ballpark as the primary analyses, correct?

A. That is correct.

Q. And then the third issue, and I think this is the one you were referring to previously, was with respect to how the imputation methodology works, correct?

A. That's correct.

Q. And in your emails both to Mr. Bellé and to Ms. Stecker, you expressed your view, your understanding, based upon your review of the paper when it first came out, that through the imputation method if an individual was not exposed to glyphosate or indicated they had not been exposed to glyphosate in the phase one questionnaire, and then did not respond to the second phase questionnaire, the imputation methodology would treat them as unexposed in that second phase time period.

That was the opinion you offered in your -- in these emails, correct?

A. And I was correct, and that is wrong.

Q. Okay. And you understand now that that's not how the imputation methodology works, correct?

A. Correct. And my supplemental report is correct in that. I would also point out the reason they are getting Beate Ritz's expert report is because in her expert report she covered the Andriutus.

Q. Andreotti?

A. No, no. She covered the preliminary document that had been obtained from Dr. Blair in that. And they were asking my opinion on that and I really didn't offer an opinion. Here is an opinion you can look at.

Q. You have in a variety of forums --

THE COURT: Can I ask a question about this email?

MR. LASKER: Yes.

THE COURT: You asked this question finally: I wonder why Aaron Blair is not a coauthor on this manuscript.

Why do you ask that question? What's the point of asking that question?

THE WITNESS: Well, his -- his name was on the original draft manuscript, it was from his office. It's unusual to drop a senior researcher like him from the final manuscript. I -- I do not why he was not on the manuscript.

BY MR. LASKER

Q. Dr. Portier, you have -- sorry. Let me drink first.

You have, not including in this litigation, defended the

IARC Working Group's conclusion with respect to glyphosate in a number of different forums and publications and then communications to regulators, correct?

A. I guess I would have to say that's incorrect.

Q. Okay. Well, let me -- let me ask you about --

A. Can I explain why it's incorrect?

THE COURT: Sure.

BY MR. LASKER

Q. Go ahead.

A. So because of the IARC Monograph report there have been a lot of back and forth from various groups of -- of what was done and what was not done.

At first, I was defending the IARC Monograph report. But when it got to the point where I was talking to the German Bundestag or other groups about this, it had gone beyond that because I was really focused on the European Food Safety Authority risk assessment on glyphosate and not so much defending IARC as pointing out some of the limitations to the way they did things.

So, yes, I defended IARC, but that was not the primary issue.

Q. Okay. If I could ask you to turn to Tab 6 in your binder?

(Witness complied.)

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

You're talking about the limitations in the way they did things. What did you mean by that? Can you explain that a little more?

THE WITNESS: Yes. It's -- there were so many of them.

Their evaluation of the human evidence once they got down to all of their arguments about it, they called it very limited.

THE COURT: Who is "they"?

THE WITNESS: The European Food Safety Authority.

THE COURT: You're talking about the limitations of the way they did things --

THE WITNESS: Correct.

THE COURT: Not IARC.

THE WITNESS: Correct.

THE COURT: I misunderstood. I don't need to hear about that.

THE WITNESS: Okay.

BY MR. LASKER

Q. So, Dr. Portier, are you at Tab 6 in your binder?

And this is another email that you provided to -- or this may be actually something that was on a FOIA request. It was a -- it was a public document, a FOIA request that we have, which is communications -- I think we talked about this in your deposition, your initial deposition; that this is

communications you had after you became aware of the fact that the European Food Safety Authority had reached a conclusion or was prepared to reach a conclusion that glyphosate had no carcinogenic potential, correct?

A. This letter occurred after I had some information that it looked like they were going to do that, that is correct.

Q. Okay. And on November 9, 2015 you sent an email to all of the IARC 112 working group members, correct?

A. I don't believe it's all of them.

Q. Many of them?

A. Many of them.

Q. And you also copied Kate Guyton? She's at IARC. What's her to role again?

A. She's one of the people in the Monograph program. And in the review of glyphosate each person in the Monograph program does one Monograph a year, where they run the meeting. It's not always the head of the program that runs the meetings. She ran the glyphosate meeting.

Q. And you're sending them this email to warn them of the fact that the European Food Safety Authority was about to release this conclusion that glyphosate had no carcinogenic potential, correct?

A. No.

Q. In the first -- I'm sorry. If we can go on the bottom half of the front page, which is the email that you sent, the

first sentence is:

"This week the European Food Safety Agency will release their reassessment of glyphosate. In this review they will conclude that glyphosate has no carcinogenic potential."

So you were alerting them to that fact, correct?

A. That's correct.

Q. And you state that this creates two problems. One is that it will weaken the strength of the IARC Monograph program to stimulate change and how some of these agents are reviewed and addressed.

And the second is that:

"It suggests we did not do our assessment adequately and that, had we seen all the data that they saw, we would have gotten a different answer."

Correct?

A. That's what it says.

Q. And you state:

"I do not intend to let that happen."

Correct?

A. Without -- yeah, without somehow addressing it.

Q. And as a result of that, you were defending and you have been defending the IARC working group analysis of glyphosate in a variety of different forum outside of this litigation, in front of regulators and in various other communications,

correct?

A. As I already mentioned, that's not the case. In most cases I was addressing what I saw as efficiencies in those agencies' review of the glyphosate information. I was not writing to those agencies to defend the IARC evaluation.

Q. You've also explained outside of this litigation, and Judge Chhabria was asking you about this just a moment ago, that the disagreement between IARC's conclusion regarding glyphosate and the regulator's conclusions is about all between hazard and risk, correct?

A. Not at all. In the European system, the European Food Safety Authority is required to ban any pesticide that is a hazard for a carcinogen. So the European Food Safety Authority does not do a risk assessment on pesticides. They do a hazard assessment just like IARC.

So it's not a battle in Europe between risk assessment and hazard assessment. The United States EPA does a risk assessment, even if it's a carcinogen. So that's a different issue.

Q. Okay. Well, then, let me ask it that way. In the United States -- let me start again.

You've explained outside of this litigation that the disagreement between IARC's conclusions regarding glyphosate and the U.S. EPA's conclusions regarding glyphosate is a battle between hazard and risk, correct?

A. No, not -- if that's all I said, then it's absolutely wrong.

Q. Okay. Let me ask you to turn to Tab 7 of your binder. And this is a newspaper story that came out and it's discussing the scientific advisory panel that the EPA impaneled to review their initial OPP evaluation that concluded that glyphosate is not likely to cause cancer, correct?

A. This was before the beginning of that meeting.

Q. And if you can turn to page -- or the second page of this article. And one fact that for us is somewhat peculiar, but for you probably it is not, your brother actually was on the advisory panel because he's also a biostatistician or a toxicologist? Biostatistician, correct?

A. Biostatistician. He was chair of the EPA Science Advisory Panel for seven years.

Q. Okay. And about two-thirds down the second page there is this discussion about the fact that you and your brother -- that your brother was on this advisory panel and there is a statement, again, that starts:

"Asked whether he had spoken with his brother about glyphosate" --

Do you see where I am?

A. Yes.

Q. (As read)

"...Christopher Portier said, in broad terms: I

told him it's a battle between hazard and risk, and that he does understand."

Is it your testimony that that is not an accurate quote that you gave to this reporter?

A. It says "in broad terms." It's an accurate quote when you say "in broad terms."

But there is much more specifics here in terms of the quality of the science.

Q. Okay.

THE COURT: What was happening with your brother?

THE WITNESS: I'm sorry. He was -- he was on the EPA Science Advisory Panel and EPA created a document for their risk assessment of glyphosate, a draft document. And because it's such a high profile, they wanted to bring it to their scientific advisory panel.

There was an argument put forth that my brother couldn't be on the panel because he was, obviously, my brother. And I have been, obviously, vocal about the science used by EPA and used by EFSA in evaluating glyphosate, so he would go my way.

But my brother and I never discussed glyphosate, except that very broad term of saying, you know, it's one of these things where there is a battle going on between hazard and risk and how it's done.

BY MR. LASKER

Q. Now, Dr. Portier, outside of this litigation and in a

submission that you gave to EPA, you have taken the position that causality for glyphosate and non-Hodgkin's lymphoma is plausible, but it clearly has not been demonstrated, correct?

A. Say that again.

Q. You have taken the position -- and this was prior to the Andreotti study.

You took the position in a submission to EPA that causality for glyphosate and non-Hodgkin's lymphoma is plausible, but that it clearly has not been demonstrated, correct?

A. I'd have to see the statement and the context in which I used it.

Q. Okay. So this is one of the attachments to your expert report, and it is Tab 1B. You actually identify it as Document 2 in your appendices, but then we would have two different numbers in the tabs.

A. Okay.

Q. And if I could direct you to -- and this is comments that you made to -- that you submitted to EPA on October 4th, 2016, correct?

A. That's what it looks like, yes.

Q. And if you could turn to Page 7, starting at Line 116, you're talking about:

"EPA's conclusions with respect to glyphosate exposure" -- "EPA's conclusions at that time with

respect to glyphosate exposure and the risk of non-Hodgkin's lymphoma."

Do you see that?

A. Yep.

Q. And at the bottom of that paragraph at Lines 125 to 126, you state:

"So is causality plausible here?

"Yes, absolutely.

"Is it demonstrated?

"No, clearly not.

"Are the findings possibly the results of chance, bias and/or confounding?

"Yes, but more unlikely than likely."

Correct?

A. This talks specifically only to the epidemiology data, and that is correct.

Q. Okay. And that is still your opinion today, that the epidemiology data clearly does not demonstrate that glyphosate causes non-Hodgkin's lymphoma?

A. That's not what it says. It says it's "plausible." And it does not, by itself, clearly demonstrate that glyphosate causes NHL.

Q. Okay. So just so I'm clear, we have been clear a number of times now on this question, so it's --

THE COURT: Sorry, what? I didn't hear a word you

said.

MR. LASKER: I know. I starting saying "clear" and then I realized the sentence was going to be awkward. I'll ask it again. I'm sorry, your Honor.

BY MR. LASKER

Q. Is it still your opinion, based upon the epidemiology, that a causal association between glyphosate and non-Hodgkin's lymphoma, if the question is whether that's been demonstrated, the answer is clearly not, correct?

A. Using only the epidemiology data, I cannot come to the conclusion without any reasonable doubt. I can't take myself to the 100 percent. That it is causal for NHL.

Q. Okay. And just -- just to be clear. If you look at your discussion before that conclusion, or those -- the final statement that you make about causality, leading up to that statement you do discuss the animal evidence, correct? And that's at Line 24 -- 124, I'm sorry. Still on Page 7.

A. Umm, yes.

Q. And, in fact, you were going through what looks like an abbreviated version of your Bradford Hill criteria in that sentence from Lines 120 through 125, correct?

A. Umm, it covers some aspects of it.

Q. Now, you've also -- excuse me. Staying in the same document. If you could turn to page -- just a prior page, Page 6?

And actually from Page 5 to Page 6, you are talking about your opinions with regard to the human evidence for glyphosate, correct?

A. Yes.

Q. And --

JUDGE PETROU: I just want to be clear.

Is it your opinion, because I'm just noting on Page 5 at Lines 15 to 16 you write:

"The agency provides many reasons for this finding. I would summarize them as follows."

THE WITNESS: Yeah. The bulleted points were what the agency has said.

JUDGE PETROU: And then the commentary is your commentary, yours, on it?

THE WITNESS: My commentary is on what the agency has said.

BY MR. LASKER

Q. And just to be clear. Going back to Page 7, Line 120 to 125, where you state "I would note," these are your -- these are your opinions as to the various Bradford Hill criteria and how you believe they have been met or not met with respect to glyphosate and non-Hodgkin's lymphoma, correct?

A. I'm sorry. I drifted looking at this while you were asking it.

Q. All right. On Page 7, Line 120 to 127, you are providing

your observations, your opinions based upon the Bradford Hill criteria or at least some of those criteria?

A. I'm countering what EPA did with their criteria.

Q. And offering your view --

A. It's difficult to follow this without the EPA document in my hand as well. Because this is really commenting heavily back to EPA on their document.

Q. Right.

A. And this is clearly not my opinion currently on the evidence here. So I don't know in what context I was making these statements relative to words that were being written by EPA.

THE COURT: Well, what does -- when you say "Is it demonstrated? No, clearly not." I mean, what do you mean by "demonstrated"?

THE WITNESS: Again, I thought here I was talking about the human evidence.

THE COURT: But it doesn't seem like it from the sentences that precede that.

THE WITNESS: I see that. But that's -- this is under my human evidence section here from EPA -- oh, if you go to Page 5 at the very top, this is the detailed technical review of what they wrote. It's human evidence. That's the category. All of these comments are on the human evidence. Until I get to Page 7 at the very bottom and then I do animals.

I -- I'm finding it very difficult to get the context of these comments on a document from two years ago.

THE COURT: Is there a conclusion -- is there kind of an overall conclusion that you articulate at the end of this document?

THE WITNESS: Not -- not for the carcinogenicity. It's an overall conclusion for the quality of the evaluation done by EPA, and it's at the very beginning. Because I have this outlined, but there are the general comments at the very beginning, and each one of these bullets is a comment on their overall evaluation.

THE COURT: Okay. Thanks.

THE WITNESS: If you look on Page 1 of bullet number 8, that's a more credible statement of what I meant with the human data.

THE COURT: Okay.

BY MR. LASKER

Q. Dr. Portier, if I could direct your attention to Page 5, starting at Line 47?

And if I'm understanding correctly how this document is set up, the statement after -- on Page 5, Line 47 through Line 48, which is on Page 6, is EPA's assessment, which is that:

"Control for confounding varied across studies and there was a strong potential for confounding by

co-exposure to other pesticides."

So that would be EPA's finding, is that correct?

A. As I understand this set of comments two years later, yes.

Q. And then the comments starting on Line 50 is your response, in which you state:

"This is correct with some studies doing better than others."

Correct?

A. That's what it says.

Q. And then you go through and discuss the findings with respect to each of the -- the core studies that you've discussed in your testimony here today, as far as which of the findings in those studies were adjusted for other pesticides and which were not adjusted for other pesticides, correct?

A. It seems.

Q. And now you have opined in this -- in your testimony in this court and in your expert report that the logistic regression analysis for De Roos 2003 with a 2.1 odds ratio was adjusted for other pesticides, but at this point what you're stating to EPA was your understanding that the 2.1 odds ratio in De Roos 2003 was unadjusted, correct?

A. It's a mistype, misspelled. I clearly -- clearly, it's adjusted.

Q. And if you could turn to Tab C, which is another submission in your expert report?

THE COURT: Would you mind, before we go off of this document --

MR. LASKER: I was going to say topic, but --

THE COURT: So I want to ask another question about this document, but it's a slightly different --

MR. LASKER: I'll come back to this document.

THE COURT: Okay.

BY MR. LASKER

Q. If you look at Tab C, which is the third attachment to your expert report, you have a Table 1. It's the first page of the document, "Human Epidemiology Studies." Do you see that?

A. Yes, I do.

Q. And, again, for each of those studies you have different odds ratios reported, and then you have a code of "U" or "C" next to each of the odds ratios, correct?

A. Correct.

Q. And as I'm looking at these -- and we can put De Roos 2003 to the side for a moment -- but "C" refers to controlled and "U" refers to uncontrolled, if I'm reading these numbers correctly, correct? Is that -- am I correct in that?

A. Probably.

Q. Okay.

A. I -- I, again, can't be certain. It's a slide deck I used sometimes ago.

Q. And in this slide deck, again at this point in time, it

was your understanding that the 2.1 odds ratio in De Roos 2003 was unadjusted, correct?

A. It's a mistake. It's incorrect. I don't know if that was my view at that time or not or if it's simply a mistake.

Q. And I'm not going to walk through this. You have similar tables in other PowerPoint slides that you presented in other venues.

A. I'm sure they are cut and paste from one to the next without any critical review.

Q. Do you recall at what point in time you came to the contrary conclusion that the 2.1 odds ratio in De Roos 2003 was adjusted?

A. Why is that the contrary conclusion? I haven't -- I'm -- I'm not committing to the fact that I didn't believe it was controlled then. I really don't know why this "U" is here, other than it's probably a mistake. Clearly, it's controlled for other pesticides.

Q. Okay. Just to conclude your discussion, I'm going back to the document, Judge Chhabria, back to Tab B on Page 6, at Line 68, concluding this section dealing with confounding you state:

"It is fair to say that confounding could not be ruled out in these studies?"

Correct?

A. That's what it says.

Q. And that's consistent with the opinion you've expressed here today?

A. That's correct.

MR. LASKER: Your Honor, I will be coming back to this document for other issues, including latency, but I'm moving off of it right now. So if you have further questions.

THE COURT: Yeah. While it's fresh on my mind, if you don't mind.

MR. LASKER: No.

THE COURT: So I -- something that caught my eye in this document was on the bottom of Page 4. And at the bottom of Page 4 you say -- and I think this is -- this is sort of a summary of your position on the matter. It's the last sentence in this section called "General Comments and Overall Summary." And it says:

"EPA should declare glyphosate a probable human carcinogen and go on to do a risk assessment to determine if human exposure is significant to warrant concern."

MR. LASKER: "Sufficient."

THE COURT: Sufficient. Did I misspeak?

MR. LASKER: Yeah. You stated "significant."

THE COURT: Sorry. Let me read it again:

"EPA should declare glyphosate a probable human carcinogen and go on to do a risk assessment to

determine if human exposure is sufficient to warrant concern."

Now, this is something that was written after the IARC Monograph was published, right?

THE WITNESS: That is correct.

THE COURT: And so, to me, like the most natural reading of this sentence is: Okay, it's a probable human carcinogen for the reasons stated by the IARC, and now we have to go on and figure out if it matters in real-world -- in real-world conditions.

And to me, that sentence seems inconsistent -- that does not seem like a sentence that would be written by somebody who has already decided that glyphosate is currently causing non-Hodgkin's lymphoma in human beings in real-world conditions.

And so I want to give you one more chance to address that.

THE WITNESS: Sure. First of all, the -- the EPA has categories who are how they describe the carcinogenic potential, the strength of evidence for carcinogenicity of chemicals, and one of them is probable human carcinogen. The same language as used by IARC. They have a definition for it. And my opinion was that the data I looked at satisfied that definition, and that's where it belonged.

It's not -- was not my intention in this document to tell EPA that the human evidence is already -- that there is already

something happening in the human population. That's their job. And they didn't do it.

What they did was they did the hazard evaluation, concluded there was no reason to be worried about carcinogenicity at all, and they didn't go on and do the risk assessment because they declared in the hazard assessment there's nothing there.

And so this declarative statement is telling them to do their job.

THE COURT: Okay.

BY MR. LASKER

Q. Now --

THE COURT: Should we take a lunch break?

MR. LASKER: Sure. We can do that.

THE COURT: Why don't we break for lunch and come back at 1:00 o'clock?

MR. LASKER: Thanks.

(Whereupon at 12:10 p.m.proceedings were adjourned for noon recess.)

P R O C E E D I N G S

April 6, 2018 **1:17 p.m.**

---000---

THE COURT: You can resume.

THE WITNESS: Your Honors, before we start, can I address the specificity issue again?

THE COURT: Sure.

THE WITNESS: It seems I was wrong and I want to make sure it's correct on the court record.

I went back and looked at the Hill paper.

THE COURT: Sorry, at the what?

THE WITNESS: Hill, Bradford Hill's paper on specificity. It's somewhat vague as to what he actually means.

So then I looked at EPA's draft risk amendment guidelines on how they interpret specificity. They interpret it the same way I do and the same way that Dr. Ritz does. They use either one in their evaluation.

When you look online in the literature some people use mine, some people use the one Dr. Ritz used.

So I want to make it clear that I didn't -- I hope I did not insult Dr. Ritz by saying she had done something that wasn't part of the criteria. In fact, it surprised me. It is part of the overall criteria.

THE COURT: So is the idea that either way of looking at it gives you an answer on specificity or is the idea that

different people disagree about what -- what the specificity criterion means?

THE WITNESS: So in the case of EPA, it's either. If either criteria is met it increases the argument for causality.

I didn't have enough time to go into why this group said this and that group said that. So I don't know if they are arguing with each other or not. I just want to make sure I understood the issue after you brought it up because it confused me. I thought I knew the criteria well.

BY MR. LASKER

Q. Dr. Portier, if I could take you -- if you would go to Tab 1, which is your expert report. And at Page 16 we have the forest plot.

And we can put this up on the screen as well. It's Slide 24.

And this is the forest plot that you also presented -- one of the forest plots you presented here this morning, correct?

A. Technically that's the only forest plot that I presented, but yes.

Q. And for -- on Page 16 in your expert report --

JUDGE PETROU: Sorry. Where was it in the other binder? It's a lot clearer to look at.

MR. WISNER: It's Exhibit 162.

JUDGE PETROU: 162?

MR. WISNER: Yes.

THE COURT: All right. Thank you.

MR. WISNER: Page 16.

JUDGE PETROU: All right. Thank you.

MR. LASKER: Is Your Honor's copy in color?

JUDGE PETROU: No, but I can tell which ones are the red. It's fine.

MR. LASKER: Okay.

BY MR. LASKER

Q. So, Dr. Portier, in discussing this Figure 1 you state -- or that you set forth the most-fully-adjusted risk estimates from selected epidemiology studies and from the meta-analysis of Chang and Delzell, correct?

A. That's what it says.

Q. And those are the six -- six items or six lines in red, correct?

A. That's correct.

Q. And as you explained -- and this is on Page 15, the top of Page 15.

If we could put up Slide 54 as well, but it's the top four lines on Page 15 in your expert report.

Both the IARC Working Group and Chang and Delzell, when comparing studies used the most-fully-adjusted risk estimates, correct?

A. Say that again.

Q. Page 15. The top of Page 15. I'm reading from your

expert report on the first line:

"As noted by both the IARC Monograph 112 and by Chang and Delzell, when comparing studies, the most reasonable comparison is to use the most-fully-adjusted risk estimates."

Correct?

A. That's what it says, but the keyword there is "comparison."

Q. And you state:

"I will mostly limit my comments to these most-fully-adjusted risk estimates."

Correct?

A. That's what it says, yes. But, again, I want to make sure it's clear. The word "comparison" is important there.

This is the most fully adjusted were the most appropriate for the meta-analysis.

Q. Okay. And none of these most-fully-adjusted risk estimates in the six glyphosate epidemiologic studies that were the core studies that existed at the time of the IARC Working Group showed a statistically significant increased risk of non-Hodgkin's lymphoma, correct?

A. None of them had confidence bounds that did not include 1.

Q. And we now have, of course, the 2018 Andreotti study. And that study, although they don't separately break it out from the data we have, has an ever/never relative risk that would be

extra below 1, correct?

A. I don't know. They didn't provide it.

Q. Okay. But you could -- using the data, and you've done some other calculations with the data, certainly you could calculate an unadjusted odds ratio for ever/never and it would be below 1, correct?

A. A crude odds ratio. I haven't done it, so I -- I can't answer the question, but -- I can't answer it.

MR. LASKER: Okay. Well, if we could put up Slide 26.

BY MR. LASKER

Q. And that is Tab 3 in your binder, Dr. Portier. It's from your deposition in January. I can't remember the exact date, but your supplemental deposition.

And it's at Page 50, Lines 10 to 19. So if you'd like to go and look at that in your expert report?

A. Tab?

Q. Tab 3.

THE COURT: Tab 3, Page 50, you said?

MR. LASKER: Yes.

BY MR. LASKER

Q. And in your deposition you testified that an unadjusted -- and uncorrected unadjusted rate ratio can be calculated from these data, and we're talking about the Andreotti study, in that the ever/never risk ratio would be less than 1, correct?

A. I have to say likely less than 1, since I didn't do it.

Q. The 2018 Andreotti study looked at more exposed cases of non-Hodgkin's lymphoma than all of the glyphosate case-controlled studies combined, correct?

A. You've asked me that question before. I believe it is correct.

Q. The 2018 Andreotti study also allowed for a longer latency period for non-Hodgkin's lymphoma development than any other published glyphosate epidemiological study, correct?

A. It depends on what you mean by "latency." But if you're saying the people in that study could have been exposed much longer than the others, the answer is yes.

Q. Okay. And just so we're clear, if you could look at, again, in your deposition at Tab 3, Page 16, Lines 23 through 17, Line 2.

MR. LASKER: And we can put that up on the screen. It's Slide 20.

BY MR. LASKER

Q. And my question is to you, starting on line 23:

"QUESTION: The 2018 National Cancer Institute study" -- and that refers to Andreotti -- "allows for a longer cancer latency period for non-Hodgkin's lymphoma than any other published glyphosate epidemiologic study, correct?"

And your answer was:

"**ANSWER:** Correct."

A. That's what it says.

THE COURT: Didn't he just say that in his actual testimony?

MR. LASKER: I wasn't clear if he was --

THE COURT: I was pretty clear.

MR. LASKER: Okay.

THE WITNESS: I would like to point out that this was not an NCI study. It was a study by Andreotti and coworkers.

An NCI study would be much more carefully reviewed. It becomes the opinion of the agency. So it's really the Andreotti study.

BY MR. LASKER

Q. You would agree that the bottom line from the analysis that was conducted by the investigators in the 2018 study with respect to glyphosate and non-Hodgkin's lymphoma from the analyses they conducted is that they saw nothing, correct?

A. From the faulty analysis that they conducted, they saw nothing.

Q. And the 2018 Andreotti study had no impact on your evaluation of the glyphosate epidemiologic literature, correct?

A. That's not true. It -- it had impact. I've read it. I considered it. I looked at it. It didn't change it.

Q. Okay. If we could -- if I could direct you to Page 53 of your deposition, starting at Line 23.

MR. LASKER: And if we can put up Slide 22?

BY MR. LASKER

Q. And my question to you, starting at Line 22 on Page 53:

"**QUESTION:** Let me ask you this in general. Do you believe that the 2018 National Cancer Institute Journal study strengthens or weakens the epidemiologic evidence in support of your opinion that there is an association between glyphosate-based herbicides and non-Hodgkin's lymphoma?"

There is an objection.

And then your answer:

"**ANSWER:** I believe that the 2018 Andreotti study had no impact on my evaluation of the epidemiology data. It is neither good nor bad. What was seen is almost what one would have expected to see, because of the exposure misclassification."

That was my question and your answer in your deposition, correct?

A. Yes, it was.

MR. LASKER: If you could put back up on the screen the forest plot? That was Slide 24 and is in the expert report at Page 16.

BY MR. LASKER

Q. So we now have the Andreotti study that updated the De Roos study.

So if we were to be looking at the published epidemiology literature now for the core studies, you would have Andreotti instead of De Roos 2005, correct?

A. Not at all. I would not put the Andreotti study in a meta-analysis partly because of the failures, partly plus of the imputation. It's completely different than the other studies in those regard.

When you do a meta-analysis, you try to bring studies together that will at least have something in common.

Q. So you believe it's appropriate to include the 2005 De Roos AHS study in the meta-analysis --

(Court reporter clarification.)

Q. ...2005 AHS study in the meta-analysis, but it is not appropriate to include the 2018 Andreotti study, is that correct?

A. The De Roos study had all of the exposures for all of the people involved. They didn't have to impute for 37 percent of the people.

Yes, it would be correct epidemiologically to include the De Roos study and not include the Andreotti study.

Q. And you believe that it would be appropriate to include the Ericksson study in a meta-analysis, but not to include the Andreotti study, is that correct?

A. Ericksson analysis and not include the Andreotti. That's correct.

Q. And you believe that it would be appropriate to include the Hardell study in a meta-analysis but not the Andreotti study, correct?

A. The 2002 Hardell saw, correct.

Q. And you believe it would be appropriate to put the Orsi studied in a meta-analysis but not the Andreotti study, is that correct?

A. That is correct.

Q. And in your expert report where there was data for epidemiologic studies that were combined into pooled analyses, you no longer consider the earlier studies in your analyses, correct?

A. I -- I didn't consider them in making my decision. I mean, I read them.

Q. Okay.

A. But, yes, I used a pooled analysis as my main driver.

Q. So, for example, for the Cantor study and the Nordstrom study and the Hardell and Ericksson study, those you did not consider or you considered the later pooled analyses for each of those datasets, correct?

A. Well, let's be correct. To make sure we're very correct here. Cantor only -- Cantor did an analysis of glyphosate unadjusted and whether it relates to NHL. The other two did not do glyphosate analyses. So I couldn't have used them in this evaluation.

They were pooled and De Roos was able to get that information on glyphosate use and put it in to the evaluation. And then she adjusted for all the other exposures. So, yes, I would use De Roos.

Q. Okay. I think we may have some confusion on the names of the studies, so let me just go back to your expert report. It's at Tab 1, again. And if you could go to Page 7 of your expert report?

And this is a continuation that starts on Page 6 where you're talking about Cantor. And as we discussed, and as you just stated, the last line of that first page you note with respect to Cantor:

"This study will not be included separately into the evaluation since it overlaps with De Roos 2003."

Correct?

A. That's correct.

Q. And then with respect to the Nordstrom study, that's Page 9 of your expert report. Do you see Nordstrom 1998?

A. Yes, I do.

Q. And the last sentence in that paragraph you note that:

"This study was later used in a pooled analyses of HCL and NHL" -- I think that's the Hardell 2002 study -- "and will not be considered independently in this evaluation."

Correct?

A. In this evaluation for causation, but will be used in the context of the pooled analysis.

Q. Correct?

A. Correct.

Q. And then the same thing with regard to the Hardell and Ericksson study, which is the next study in your report. And go to the last line. Because that was pooled into another analysis, you used the pooled analysis in your evaluation rather than the earlier studies, correct?

A. That is correct.

Q. And I know that -- and I appreciate that you're not offering any opinions with respect to the North American pooled project, but you are aware that that is a pooled analysis that includes data from the McDuffie study and De Roos 2003, correct?

A. Actually, it's more than that. It's when you -- when you look at the studies, they have a lot more cases and controls than any of the -- than all of the studies individually.

So it's not exactly just taking over what was done by De Roos, because De Roos has some other evaluations in here that -- that you can't ignore. It's, to some degree, a unique study, even with the NAPP.

Q. That's fine.

The data that's in the U.S.-based case-controlled studies and Canadian-based case-controlled studies, you understand that

was pooled for the NAPP analysis, correct?

A. I understand that was pooled. But, again, let's see if I can be clear on what I'm saying here.

If you look -- could I have my table? No, it's not there. No.

THE COURT: Page 16 of your report?

THE WITNESS: It's not there. It's not in that table, so I'll let it go.

A. The individual studies, and I don't know the numbers right off my back, but they each had X number of cases and X number of controls according to the individual write-up. And when you add those up and you go look at De Roos, she says that's what they had, 995 cases and some number of controls.

And then De Roos restricted her set. She didn't use women. She didn't use people who had worked on a farm before the age of 17, et cetera. She had some restrictions. So she ended up with 800-something.

Yet, when you add those things up and look at the North American pooled project, there is still 200 cases and 600 controls that are not accounted for that I'm sure were originally there but were not listed in the original paper or in the De Roos paper. So they have something else they have included.

And because I don't know that, I'm concerned with just telling you I could get rid of the De Roos paper because this

supersedes it.

BY MR. LASKER

Q. And, Dr. Portier, in your forest plot, which is still on the screen, you include weights for each of those six epidemiologic studies that were published as of the IARC Monograph, correct?

A. Those weights are provided in the paper by Chang and Delzell. They are automatically generated by the meta-analysis.

Q. And just so the record is clear -- and we can go to the Chang and Delzell study. It's at Tab 12 in your binder.

And if you look at Page 404, which is the third page in Chang and Delzell, I believe this is the table that -- from which you pulled your numbers, correct?

A. Give me a minute to look. I'm just looking to see if they are correct. But I believe, yes, that's the table I -- that's the plot I pulled it from.

Q. And just, again, so the record is clear, you have, it appears, a typo in your expert report with respect to the weight for Orsi.

And Orsi, in fact, would have a weight of 9.5, which is just -- which comes in just below the 11.6 weight for Ericksson, correct?

A. Give me a minute. Excuse me. There is another table I need to look at.

(Brief pause.)

A. There is a typo, but yes.

BY MR. LASKER

Q. And you testified --

JUDGE PETROU: Are you moving to something else? Because if so, I would like to ask a question about this chart that we're just looking at right now.

MR. LASKER: I'm kind of still on it for a few more questions. I'm not sure where your question will be.

BY MR. LASKER

Q. But, Dr. Portier, you testified during your direct that your view of the strength of causation under the Bradford Hill criteria for glyphosate and non-Hodgkin's lymphoma, and I believe I wrote this down exactly, is mostly driven by the meta-analysis. Do you recall that?

A. I might have said something like that, yes.

Q. And you cited in your testimony this morning to the meta-analysis that we're looking at right here by Chang and Delzell with the 1.3 relative risk and the confidence intervals, and there were a few more that you have on your forest plot that are roughly similar, correct?

A. Say that again, the last part?

Q. There are a few other models that Chang and Delzell used that are roughly similar in their findings for a meta-analysis, correct?

A. That is correct.

Q. And you have not calculated a meta-analysis that incorporates the reported findings from the Andreotti paper or, I take it, from the NAPP, correct?

A. I have not done such a meta-analysis, that is correct.

JUDGE PETROU: Simple, but probably easy for you to answer the question, but I'm curious just looking at the relative weights here.

We have six studies, right, totaling 100 percent. So if they are equally weighted, that's, what, something like 16 and two-thirds percent each.

We've got one study here, McDuffie, which clocks in at 38 percent of the total.

Is there anything about that in and of itself that's troubling or problematic to you when you see a meta-analysis and one out of the six has such a disproportionate percentage of the total?

THE WITNESS: You would look at the heterogeneity. You would look at other issues. In this case there was nothing else. It was strictly driven by the low confidence bounds, small confidence backgrounds and the large number of cases and controls.

JUDGE PETROU: I understood that that's how you got to the 38 percent. I was just wondering if that was a red flag of any kind, so much out of this analysis.

THE WITNESS: So if I had been doing this meta-analysis, I probably would have pulled that study out and looked at sensitivity to the inclusion of that study, the McDuffie study. People do that in epidemiology. That's a standard thing to do when you see something carrying a lot of wait.

JUDGE PETROU: We don't know or do we know whether that was done here?

THE WITNESS: They did not do it here in one of the models that I'm aware of.

JUDGE PETROU: Thank you.

THE WITNESS: I want to correct something, a minute ago.

The strong statement for the strength of the evidence is mostly driven by the meta-analysis. If it was not the meta-analysis, then the individual studies would have taken me a little lower. Not as strong.

I want to be clear it's not the whole review. It's taking it up to that too level.

JUDGE PETROU: I understand.

THE COURT: Could I ask a question about this table, this table on Page 16 of your report that comes from Chang and Delzell. I guess Chang and Delzell chose to use the hierarchical regression model from De Roos 2003 rather than the logistic regression analysis and you -- it sounds like you buy

into that choice. Can you explain why?

THE WITNESS: Actually, they used both. The Model 2 --

THE COURT: "They" being Chang --

THE WITNESS: Chang and Delzell. The Model 2 that they put forward --

THE COURT: Wait. Let me go back to Chang. Are you pointing me to something in their paper?

THE WITNESS: We can go to their paper. That would be great.

THE COURT: Okay.

THE WITNESS: On Page 416 of their paper, their Table 3.

THE COURT: 416. Give me a minute.

Okay.

THE WITNESS: So if you look at the seventh or eighth line where it says "meta-analysis model" under Model 1, they are using 1A, 2, 3, 4, 6, 7. And so 1A is the De Roos hierarchical regression. That's all the way in column -- under RR. That's one, two, three, four -- five when you look at Model 1.

And then the second one is 1b, and that's using the logistic regression. So they looked at the sensitivity of the meta-analysis to both of those evaluations. I --

THE COURT: But it seems like they emphasize and you

emphasize the hierarchical regression analysis and I'm curious what -- why they and you decided to do that.

What's the rationale behind their decision and your decision to do that?

THE WITNESS: So I do not favor the hierarchical model over the logistic regression model. They are both legitimate reasonable ways to analyze the data. It's like doing a sensitivity analysis on your analysis method. I could use logistic regression. I could use hierarchical regression.

The problem with the hierarchical regression here, my only minor problem with it is they used a fairly uninformative Brier in their evaluation. It's a -- it's a different area of statistics. It's called Baseyian statistics.

And Dr. Ritz kind of explained this. You have to make some assumptions about how carcinogenic glyphosate is before you do the analysis, and then it goes through this mark-off chain --

THE COURT: Do you have make an assumption about how carcinogenic glyphosate is or how carcinogenic the other pesticides are?

THE WITNESS: All of them. Every individual one of them had a prior for how carcinogenic they were.

And it's known with the technique. If you choose a non-informative prior, it will pull down the regression from what you would get with a logistic regression.

And so, again, you're sort of seeing what you would inspect to see with that analysis compared to what you see against the logistic regression.

THE COURT: But when you pool it with all these other studies, with all this other data, it looks like it's inconsequential from looking at the Chang and Delzell Table 3 that you just showed me, it's the -- am I right that it's the exact same numbers?

THE WITNESS: Chang and Delzell should have given you two significant digits. The U.S. EPA Science Advisory Panel redid this analysis. And so they do differ in the third digit, but that's it.

I think Model 2 is 1.04 is the lower bound and Model 1 is 1.03, but I would have to go back and double-check that.

THE COURT: Okay. Thanks.

BY MR. LASKER

Q. Dr. Portier, I'd like to turn now to some of your testimony about the imputation method that was used in the 2018 Andreotti study.

First, Dr. Portier, you do not have sufficient experience in the field of epidemiology to speak to whether imputation, multiple imputation is a widely accepted methodology in epidemiologic studies, correct?

A. No. At this point I feel I do.

Q. Well, if we could turn to your deposition in January at

Page 82. And this, again, is Tab 3 in your binder.

A. What page?

Q. Page 82, starting at Line 15.

A. Yes.

Q. And my question to you was:

"QUESTION: Do you agree in general" --

MR. LASKER: I'm sorry. We can put this up on the screen. It's Slide 38.

BY MR. LASKER

Q. (As read)

"QUESTION: Do you agree in general -- and we'll get to the 2018 NCI study in a second, but do you agree in general that multiple imputation is a widely accepted methodology for use in the epidemiologic research?"

There was an objection to form.

And your answer:

"ANSWER: I am not sure I have the history in the field sufficient to be able to say it's widely accepted. I just don't think I can answer that appropriately."

Correct?

A. At that point, yes. But as I pointed out in my direct, I -- these questions made me go back and look at this very carefully.

Q. And you had some testimony in direct about trying to find

other studies that used a multiple imputation, correct?

A. No, multi-step multiple imputation. Instead of just -- there's lots of multiple imputation studies out there. This is a very specific twist on that that I -- I couldn't find.

Q. During your direct examination, you testified about a bit about the Brier score that was calculated as part of the Heltshe analysis. Do you recall that testimony?

A. Yes.

Q. Prior to reading the Heltshe paper, you had never heard the term "Brier score," correct?

A. I think that's probably true.

Q. And you had never calculated a Brier score, correct?

A. That is true.

Q. You testified during your deposition that it was your understanding after reading Heltshe that a Brier score of zero shows perfect accuracy, correct?

A. That is correct.

Q. And if we can look to the Heltshe paper, it's Tab 13. And if you could turn to Table 3, which is on Page 413?

MR. LASKER: We can put this up on the screen also. It's Slide 41.

JUDGE PETROU: Before you ask a question about that, is it your testimony today that a Brier score of zero means perfect?

THE WITNESS: Yes.

JUDGE PETROU: Okay.

THE WITNESS: As far as I'm aware.

THE COURT: I take it it wasn't a change because your question was specific to your deposition testimony.

Go ahead.

BY MR. LASKER

Q. If we look at Table 3, the very first pesticide that is named, which is methyl bromide. Do you see that?

A. Yes.

Q. And that pesticide has a Brier score, which is the second-to-last column in this table, of 0.004, correct?

A. That's what it -- that's what it says.

Q. And the Heltshe investigators on Page 412 --

A. Where?

Q. On Page 412.

A. Okay.

Q. And this is in the second column right above "Days Per Year Use of Specific Pesticides," there is six lines at the very bottom. They state:

"For only a few of the rare pesticides, less than 1 percent usage, used in phase two does the imputed prevalence differ from the true prevalence by more than 20 percent."

Do you see that?

A. I see that, yes.

Q. And one of the pesticides that they identify where the imputed prevalence is more than 20 percent different from the imputed prevalence is methyl bromide, correct?

A. That's correct. But we discussed that in the deposition as to why that occurs. I would be happy to discuss it again.

Q. We can. I understand -- and I think we'll be getting to that.

But just to continue with this analysis or this discussion. The investigators, the Heltshe investigators, also used their imputation methodology, and I think you discussed this a bit in your direct examination, to look at any pesticide exposure; not just specific pesticides but a category for any pesticide exposure, correct?

A. That is correct.

Q. And for the any pesticide exposure, and you mentioned this in your direct and it's in the abstract on the first page of this paper.

MR. LASKER: And you can put this up. It's Slide 44.

BY MR. LASKER

Q. The investigators noted that:

"The observed and imputed prevalence of any pesticide use in the holdout dataset were 85.7 percent and 85.3 percent, respectively."

Correct?

A. That's what it says, yes.

Q. And you agree, though, that the Brier score for their any pesticide imputation was among the highest of all the Brier scores that they calculated in their analysis, correct?

A. Can you show me where that is?

Q. Well, we can go first of all to your deposition. And this is again, Tab 3, Page 104. It's Line 1 through Line 5.

MR. LASKER: You can put this up on the screen as Slide 45.

BY MR. LASKER

Q. And we were discussing the Brier score here for any pesticide use, correct?

A. No, we're talking about relative Brier scores. We're not -- we're not -- you were talking -- never mind. Ask the question again.

Q. We're talking about -- I don't know what relative Brier scores are. We're talking about the Brier score here, correct, for the any pesticide use imputation?

A. There is no Brier score that I know of for any pesticide use. The numbers you have in your question, starting at Line 11:

"QUESTION: In fact, the Brier scores for the -- the Brier score for any pesticide use which we just talked about, the 85.3 and 85.7" --

Which are not Brier scores. Those are the estimated prevalences.

"**QUESTION:** The Brier score for any pesticide use is higher than the Brier score for almost all of the 38 individual pesticides, correct?

"**ANSWER:** Where is that Brier score?"

So I'm asking the same question again. Where in the text is that Brier score?

Q. Okay. Let's go to Tab 13.

A. Thank you.

Q. And in that first column, and this is where we went during your deposition as well, on the left they have the -- I'm sorry.

THE COURT: Page?

MR. LASKER: I'm sorry. Page 412.

THE COURT: Okay.

BY MR. LASKER

Q. The column, the left-hand column they have "Results," "Imputation Assessment," correct?

A. Oh, the titles, yes.

Q. And they are discussing here the any pesticide imputation, correct?

Do you see the 85.25 and the 85.68, which rounds out to 85.7 and 85.3?

A. Correct.

Q. And then as we talked about during your deposition at the bottom of the column they provide, and it's about one, two,

three, four, five, six, seven -- seven lines from the bottom of that column they have the Brier score and the Brier reference score. Do you see that? 1.0 -- .1092 for the Brier score and .1227 for the Brier reference -- or the reference Brier, correct?

A. No. I'm not seeing it. Where is this?

Q. It is, if you go above Figure 1 and you count up seven lines.

A. Oh, yes. Now I see it.

Q. And so that was the Brier score for any pesticide use?

A. Correct.

Q. And that Brier reference score, as we discussed in your deposition, was among the highest of all the Brier scores that were calculated if you look at the list of all 38 individual pesticide Brier scores, correct?

A. The Brier reference score?

Q. The Brier score.

A. Oh, the Brier score. Let me take a look again.

(Brief pause.)

A. Yes, it's one of the higher.

Q. And so we have an imputation of 85.3 percent versus 85.7 percent with one of the highest Brier scores that were calculated in the Heltshe paper, correct?

A. That's correct. The Brier score is affected by the prevalence of the exposure in the population you're looking at.

So it would not be surprising that the Brier score for any pesticide use would be large, just like it is for glyphosate, malathion and 2,4-D.

Q. And just because the Brier score was large for any pesticide use and for the specific pesticides like glyphosate that had the highest exposure rate does not mean, as we see for any pesticides, that the imputation method does not provide an estimate that, in the case of any pesticide, was an almost complete match, correct?

A. It does not indicate it's almost a complete match. That is what I showed with the tables during my direct.

It indicates the best you can do is .41 -- point, what is it? The difference? .4-something percent difference. But it could be as high as, I forgot, a 28 percent difference. And so you could easily get that Brier score with a 28 percent difference.

Q. And so you testified during your deposition that you believe that the proper comparison for the imputation of pesticides should be based upon absolute error, correct?

A. Between the -- yes.

Q. The authors and the investigators of the Heltshe paper, however, presented the data based upon relative error as between the imputation and the actual prevalence, correct?

A. They reported it as well as reporting the individual numbers.

Q. And they have a table, Figure 2, on Page 414 where they list out all of the individual pesticides based upon relative error, correct?

A. Correct.

THE WITNESS: Just for the judges, your Honors. Relative error means that you take the error that I was looking at and divide it by the prevalence of the exposure in the population. So as glyphosate has the largest exposure, it has the largest divisor. And since methyl bromide has the lowest exposure, it has the smallest divider.

BY MR. LASKER

Q. When the Heltshe investigators looked at relative error and reported it, as they did in their paper, they reported that glyphosate fell basically in the middle of the pack, correct?

A. It -- the bottom third or something. Right at the edge of the bottom third.

But as I pointed out, when you're doing the logistic regression that you're going to do to evaluate these data, it's the absolute that's going to make a difference in terms of the bias and not the relative.

Q. I understand the opinion you were offering earlier today, but I want to just make sure we're clear on what the authors of this paper stated.

And if you look at Figure 2 and we're talking about relative error, there is relative error both on the high side

and on the low side, correct?

A. That's correct.

Q. And glyphosate -- if you include also the relative error on the high side, glyphosate falls right in the middle of the pack for these pesticides, correct?

A. I'm sorry. Say that again? Of the ones on the high side, it falls towards the middle of the high side? It's still toward the bottom.

Q. Okay.

A. Bottom third.

Q. Let's just be clear. If I could direct you to your testimony at your deposition again. And this is at Page 106, Line 13.

A. 106?

Q. Uh-huh. Yes.

MR. LASKER: And we can put this up on the screen. It's Slide 49.

MS. GREENWALD: Your Honor, my objection is that he asked and answered all these questions already. I just want to make that clear, that maybe Dr. Portier can look above that as well.

MR. LASKER: We can go -- what do you want, 105/19? He can start reading from there. It's basically the same discussion we just had.

MR. WISNER: Specifically Page 105 starting at

Line 19.

MR. LASKER: Yeah, that's fine.

(Brief pause.)

THE COURT: Can we move on to something, please?

MR. LASKER: We can, Your Honor. That's fine.

BY MR. LASKER

Q. Dr. Portier, I'd like to ask you about your calculations of -- or your recalculations of the relative risks in the 2018 NCI study. And you presented some of this data during your direct examination. I just want to make sure I understand how you did these calculations.

MR. LASKER: So if we could put up Slide 29.

JUDGE PETROU: Just to be clear, because he's already said it's not an NCI study. When you say 2018, you're referring to?

MR. LASKER: Yeah, the JNCI study or the Andreotti study. I'll say Andreotti study, Your Honor.

If you could put up Slide 29?

BY MR. LASKER

Q. This is the relative risks that were reported in the Andreotti study for cumulative days of exposure for non-Hodgkin's lymphoma. And if you have --

MR. LASKER: Do we have the science binder up there for him to look at the Andreotti study?

BY MR. LASKER

Q. Do you need to look to the study to confirm these data points or do you recognize them?

A. I will reserve the right to go look when you tell me what you want to tell me. How is that?

Q. That's fine.

A. It's slowing you down. Let's --

Q. I'm just going to confirm that these were the numbers from the study.

My next question is: If I understand how you calculated your -- your recalculated odds ratios or relative risks, if we can go to Slide 55, you did not -- you took out the unexposed control numbers of people who are unexposed, you took the lowest exposure group and made that your reference and then you calculated your relative risks compared to that lower exposure group, correct?

A. Yes.

Q. And similarly for intensity of exposure.

MR. LASKER: We could put you have Slide 56.

BY MR. LASKER

Q. This is from the primary analysis that's in the paper for cumulative -- for intensity weighted cumulative days, correct?

A. That is correct.

Q. With all the relative risks reported as below 1. And then you did the same calculation.

MR. LASKER: And we can put up Slide 57.

BY MR. LASKER

Q. You removed, or you no longer considered the unexposed group and you recalculated the relative risks using the lower exposed group as your reference, correct?

A. And it's an approximate calculation. I will say that right upfront.

Q. Right.

A. I can't do the adjustments or anything because I don't have the real data. This is approximately what would happen.

Q. Okay. And obviously -- well, let me ask.

Your recalculation does not change the number of farmers that are in each of these five groups, correct?

A. No, it wouldn't.

Q. And it doesn't change the fact that there is a higher incidence of non-Hodgkin's lymphoma, at least as adjusted, in the unexposed group than there is in any of the four exposed groups, correct?

A. Say that again.

Q. Your recalculation does not change the fact that there was a higher incidence, at least as adjusted, of non-Hodgkin's lymphoma in the group that is not exposed than there was in any of the groups Q1 through Q4 where there was exposure?

A. In the adjusted analysis the incidents in the unexposed was larger than the incidents in the exposed, yes.

Q. And you did not say that any of your related relative risks comparing higher exposure to lower exposure shows a statistically significant positive association between glyphosate and non-Hodgkin's lymphoma, correct?

A. I can't use -- I can't create confidence bounds. That's correct.

Q. And you also are not saying that your recalculated relative risks show evidence of a dose-response, correct?

A. That is correct.

Q. Dr. Portier, last topic. Latency.

You agree that because the latency period for cancer can take years, evaluation of epidemiologic studies should consider whether the exposure occurred sufficiently long ago to be associated with cancer development, correct?

A. Say it again? I'm sorry, it's...

Q. Okay. You agree that the cause, the latency period for cancer take years, evaluation of epidemiologic studies should consider whether the exposure occurred sufficiently long ago to be associated with cancer development, correct?

A. Correct.

Q. You agree that cancer latency is one of the things that you must consider in evaluating the epidemiologic literature, correct?

A. Must? Umm -- umm, not really. Not always.

Q. Okay. If I could ask you to turn again to Tab 3, which is

your deposition in January of 2018.

A. Which tab is that?

Q. Tab 3.

A. Okay.

Q. And if you could look at Page 11, starting at Line 6.

A. 11?

Q. Page 11, yes.

(Witness complied.)

Q. And I asked you the question:

"QUESTION: It is your opinion that because the latency period for cancers can be long by years, evaluation of studies should consider whether the exposure occurred sufficiently long ago to be associated with cancer development, correct?"

And then your answer:

"ANSWER: I will put it in my own words. Cancer latency is one of the things you must consider in evaluating the epidemiological literature."

Correct.

A. That's what it says.

Q. And that's what you said, correct, obviously?

A. That's what I said.

Q. And you also then state that:

"ANSWER: In this case I referenced a paper that looked at the estimates of how long it took for

non-Hodgkin's lymphoma to form, and 6.7 years was a little short."

Correct?

A. That's correct.

Q. And in your submission to EPA, and we can look at --

A. Can I do one thing in here, just tell you why I said no today about --

THE COURT: Sure.

THE WITNESS: Good. I was a little too soft here. If I have an epidemiology study of a constant exposure chemical that's been in the environment for 50 years and people were looking at it and I am now going to do a case-control study, I don't worry about latency. So I don't necessarily have to look at it in situations where I have long-term exposure.

THE COURT: But you have to at least look at whether latency is an issue when they are doing such a study, right?

THE WITNESS: Correct. You look at whether it's an issue or not, but you don't necessarily have to know what the latency is.

BY MR. LASKER

Q. And if I could direct you again to Tab 1B, which was -- to your expert report. Your submission to EPA, which we were discussing earlier today.

A. Tab 1B.

Q. B as in "boy," I'm sorry.

And on Page 6 starting at Line 89, you refer to a study by Kato in 2005, correct?

A. Which line do you start on?

Q. Line 89.

A. Yes.

Q. And you state that:

"This is a high quality population-based incidence case-control study looking at the relationship between organic solvent exposure and NHL in women."

MR. LASKER: And I'm sorry. We can also put this up. It's Slide 36.

BY MR. LASKER

Q. And continuing:

"Looking at the relationship between organic solvent exposure and NHL in women found statistical significance only for women occupationally exposed prior to 1970. And then note that cases and controls were recruited between 1995 and 1998. And cited two other studies with similar results (no reference given). They concluded this long latency was either due to higher exposures prior to 1970 or at least a 25-year latency period is required for NHL induction by these exposures."

Correct?

A. That's what it says. That's correct.

Q. And you also cited to two studies that looked at NHL latency for patients who received radiation or chemotherapy for Hodgkin's disease, correct? And you talk about that on Page 7.

A. Yes.

Q. And you explain that one of these studies reported latencies ranging from one year to 11 years, with a median latency of 5.5 years. And that the other reported latencies of up to 16 years, correct?

A. Can I have a minute to read this to make sure I know what I'm looking at?

Q. Sure.

(Brief pause.)

A. Okay. That's what it says, yes.

Q. And you state that:

"These types of radiation and chemotherapy exposures are rather extreme relative to those from glyphosate, and it would not be surprising for the glyphosate lag time to be longer than that that from chemotherapy and radiation treatment."

Correct?

A. That's what it says.

MR. LASKER: I have no further questions, Your Honor.

THE WITNESS: But it says "lag time." I want to be

clear what we're talking about here.

There's -- again, we're playing this game with the latency term. The shortest time from exposure to getting NHL is one year. That's quite clear in this literature. Not everybody will get that in one year. That's also clear from this literature.

The first part about the 1970 study, I'd have to go back and look at that again to explain it to you, but my recollection is that in that study this is a prospective study, very long prospective study with a small group. They were looking for statistically significant increases. It look a very long time for it to get there.

They interpreted it as latency, and that's all I'm reporting here.

MR. LASKER: Thank you.

No further questions, Your Honors.

MR. WISNER: Your Honor, I'm going to be doing the redirect.

THE COURT: Okay.

REDIRECT EXAMINATION

BY MR. WISNER

Q. Good afternoon, Doctor.

Just since it's right in front you, you're looking at the EPA submission that you made as a comment to the EPA, is that right?

A. Yeah, that's right in front of me here.

Q. Okay. In that section --

MR. LASKER: Is this in your binder now?

MR. WISNER: It's the document you were just reading.

A. Tab B. Is that it? Tab B?

BY MR. WISNER

Q. Now, Doctor, in this submission there is some discussion of latency and lag. Are you referring to cohort studies in this section or are you referring to case-control studies?

A. Case-controlled study.

Q. What -- why is that important, if at all?

A. Well, again, as I stated this morning, you have to accumulate enough patients to see a statistically significant effect, to talk about: Okay, we've got an effect here. That's a function of how big the cohort is and how long you wait.

The smaller the cohort the longer you have to wait; the bigger the cohort the faster you see that statistical significant.

Q. Specifically you discussed earlier today this issue about latency and how case-control studies have access to a very large population to pull cases from, is that right?

A. That's correct.

Q. Specifically you have discussed De Roos '03, is that right?

A. I discussed a theoretical looking one of those, yes.

Q. Let's just talk about De Roos 2003.

A. Okay.

Q. What was of the size of the population they pulled cases from?

A. Approximately 2 million.

Q. And when you're pulling from millions of potential people versus in a cohort study which, for example, in the AHS, you know, we're talking about 50-, 60-, 70,000, how does that affect your interpretation of the results as it relates to the lag issue?

A. I certainly -- if -- if we go to the picture I had this morning about latency time, and recognize that a single exposure could send somebody who's susceptible into that pathway to getting disease, then by choosing such a large population, you should pick up some of those. And so you stand a better chance of seeing those types of things.

So latency becomes less of a problem in a big study like that than in a cohort study where, especially one like this, this is an outdoor population. So they are healthy -- the AHS, they are generally healthier than the rest of it. So it could even take longer in that cohort because they are generally healthier.

And so it's hard to make these easy judgments on latency between any of these studies. I just look for the minimum and see if it makes sense.

Q. Would it be fair to say that in an assessing the latency issue with any epidemiological study, you're not just looking at the potential lag in the study, but you're also looking at the size of the study as well?

A. Correct.

Q. And so, for example, if the AHS followed 2 million people from day one, would there be as much of a concern about latency in that context?

A. No, no. Not at all. You would probably see something much sooner.

Q. We talked specifically just now about AHS and you said that these people are healthy --

THE COURT: Can you hold on a second?

MR. WISNER: Sure.

THE COURT: I want to ask a follow-up question about the latency issue.

I had a chance to discuss this with Dr. Ritz, but I don't think we've quite discussed this question yet, or maybe we have and I'm just not remembering.

But with the case-controlled studies, as you saw and we discussed with Dr. Ritz the other day, you do have these situations where people have been diagnosed with NHL in the early '80s and glyphosate didn't come on the market or wasn't introduced or wasn't approved until '75. And Dr. Ritz said -- and so there is automatically, I think, an alarm bell or a

warning sign about latency.

Would you agree with that, that it's something that you need to look into?

THE WITNESS: Definitely you want to look at it very carefully.

THE COURT: Okay. And Dr. Ritz said that as it relates to exposure to other pesticides, the latency is not a concern in De Roos 2003 because she adjusted for other pesticide use.

Do you agree with that, or is there anything that you can -- you would like to say about that?

THE WITNESS: I don't think I understood. If you could try me again.

THE COURT: Sure. So you might say: Let's look at these pools of people who were diagnosed with NHL in 1981. And glyphosate didn't come on the market until '75.

We -- with these studies we tended to prefer a longer period between exposure and diagnosis. And if we don't have a longer period between exposure and diagnosis, we are concerned that perhaps the disease was caused by something else.

And so the first question you might ask when you see the numbers that I just described, the dates that I just described is: Well, maybe the pesticide that the people were using before they started using glyphosate is what caused the non-Hodgkin's lymphoma rather than glyphosate, because after

all, they were presumably exposed to that, you know, in the 5- to 15-year-period before diagnosis.

THE WITNESS: Now I understand your question.

THE COURT: And so, you know, the -- and so Dr. Ritz had a response to that and I wanted to hear your response to it as well.

You know, is that a concern with these studies? Is it a concern that maybe some other pesticide exposure that occurred during the 5- to 15-year-period before diagnosis is more likely to have caused NHL in these people than glyphosate.

THE WITNESS: Okay. Now I -- now I get it.

Because De Roos adjusted for every other pesticide she could possibly adjust for, unless there is a phantom pesticide out there or a phantom exposure causing the NHL, then seeing NHL should worry you.

If you hadn't seen NHL in that study you might argue: Okay, the latency wasn't long enough.

But having seen it and having adjusted for everything, I would have to conclude that that's a real NHL finding.

THE COURT: I mean, you said having adjusted for everything. I mean, it -- so I get the point about pesticides, right? She adjusted for all these other pesticides.

But there are other things that could be causes of non-Hodgkin's lymphoma that are not pesticides, right? And I -- I don't know what they are. But a couple of -- a couple

of the ones that have been thrown out are excessive sun exposure, exposure to diesel fumes. Who knows what else.

I mean, given the dates on which these people were diagnosed and given the date that glyphosate was introduced into the market, don't we -- isn't it still an alarm bell? I mean, isn't it still a real concern that, you know, geeze, we normally want there to be, like, a ten-year latency period at least for an epidemiological study.

Don't we need to be really concerned that, you know, these cases of NHL were caused by something else that we haven't thought about and not glyphosate?

THE WITNESS: I would argue not in this case. Given -- given the body of evidence -- I can't look at one study at a time. I have to look at the body of evidence.

So let's take a theoretical case. Five years after the introduction of a pesticide, I do a case-controlled study and I see something. That flags. The latency period clearly flags on that and I would mention it at the end of my study.

Five years later I do a new case-controlled study and it's still there. Well, I might not comment on latency anymore because now I've seen it twice in two studies.

And then I do a third study five years later and I see it there as well. Now, I'm not worried about latency in the first study because I've got a consistent picture coming across here. And so --

THE COURT: We don't really have that for glyphosate, right? I mean, we don't really have -- we don't have -- we don't have a group of people that were diagnosed in, you know, the first happen of the '80s and then a group of people that were diagnosed five years later, and then five years after that, and then five years after that, right?

I mean, the case-controlled studies stopped before we could get to the point that you're describing, right?

THE WITNESS: I wouldn't say that, no. I would say that you have 10, 12 years of exposure for some of the people in these cohorts, in these case-controlled studies.

And when you look again at the size of the population --

THE COURT: I guess Ericksson would be an example of that.

THE WITNESS: Yeah.

But, again, when you look at the size of the base population that these case-controlled studies represent, all of them as a conglomerate -- I don't know what it is, but it's going to be in excess of 2 or 3 or 4 million people. And from such a large draw, you can get the people who have really short latencies and you can actually see the effects.

Because, I mean, it's clear to me that even if somebody comes in and says the latency should be six years on average for glyphosate, that's -- for NHL, that's still average. 50 percent of the people were less, 50 percent of the people were

more, if it's a bell curve. And so when you get that big population, you can see those short latency people.

THE COURT: Okay. Thank you.

BY MR. WISNER

Q. I would like to follow up on two little points the judge raised. The first is we talked about diesel, sunshine, things like that.

While those -- putting aside whether or not those, in fact, are something that can cause NHL, that's the first aspect of a confounder.

Let's look at the second one. Is there any reason to believe in De Roos 2003, that the control group was being exposed to less diesel or less sunshine than the study group?

A. No.

Q. And so when we talk about potential other causes of a signal that we're seeing in De Roos 2003, can you think of anything that would have differentially impacted just glyphosate users in the study?

A. Other than pesticides. Farm use, farm -- working as a farmer might have made a difference, but that was controlled for in some of the studies.

No, I -- I can't think of anything right off the bat. Right there. Just doesn't come out. No.

Q. So conceivably we could sort of conjure up other potential explanations for things that cause NHL. But unless we can show

that it's differentially impacting these two groups, it's a non-issue because we would expect to see it in both?

A. It would be non-differential, that's correct.

Q. And the second issue on this latency point, and I think this came out with Dr. Ritz but I want to get your thoughts on it.

When we're talking a risk that we're seeing in a study, if there is insufficient latency and we see no risk, does that mean we should study it longer?

A. It depends on how much you believe that there is other data suggesting you really need to study this, this agent. Then you would indeed study it some more.

Q. But if it's the opposite, where in a relatively short period of time you see a fully adjusted risk, does latency in that context invalidate the data you're seeing?

A. No. I -- even if I was told the latency was a median of six years or ten years, I still don't think it would invalidate it because it's an entire distribution.

And if I'm corrected for everything I really need to just for, then I have to believe it's the exposure that I'm really looking at. That has to be my hypothesis moving forward.

Q. And while we're talking about De Roos, a couple of follow-ups on it.

Mr. Lasker asked you some questions about whether or not NAPP sort of subsumes De Roos. Do you recall that?

A. Yes.
Q. In De Roos, how many pesticides did she control for?
A. Forty-eight I think, something in that neighborhood.
Q. How many did the NAPP control for?
A. I don't really recall -- oh, I think it was on -- it was in the draft manuscript, it was three, I believe.
Q. So it would be fair to say then that the adjusted estimate in De Roos is fundamentally different than the adjusted estimates in the NAPP study?
A. That's correct.
Q. I also understand that in De Roos 2003 the researchers actually excluded anybody who said I don't know about their exposures to the 47 different pesticides, is that right?
A. That is correct.
Q. And so what they were left with in the De Roos study is just those people where they had complete exposure information for every single person, is that right?
A. That's correct.
Q. Did they do that in NAPP?
A. Don't know.
Q. Okay. Assuming they didn't, would that also make the De Roos analysis different?
A. Assuming they threw out people who --
Q. Sorry. I'll ask that question again.
Assuming they didn't do that in NAPP, that they just used

everybody, regardless if they used -- said I don't know --

A. And then imputed some sort of exposure?

Q. Precisely.

A. They would be different.

Q. Okay. Would it be fair to say then that De Roos 2003 stands on its own?

A. Yes. That would be fair to say.

Q. And would it be fair to say that you should ignore De Roos 2003 because of NAPP?

A. I didn't -- I've not looked at NAPP close enough to be able to say that. Other than the characteristics of the NAPP study are different in terms of analysis, interpretation, and cases and controls than in De Roos. And so I would be extraordinarily cautious in ruling out, throwing way De Roos and keeping NAPP.

Q. Okay. Let's take a look quickly at the Andreotti study. I believe it's Tab 13 in the binder in front of you -- or is it 12?

Let me find it. One second.

THE COURT: Your binder or --

MR. WISNER: My binder. I was trying to keep it simple. I will give you my binder.

A. 13 is Heltshe.

BY MR. WISNER

Q. Here is my binder --

A. I have a copy.

Q. Do you have Andreotti in front of you?

MR. WISNER: In our binder, Your Honor, it's Exhibit 12. It's the first thing.

BY MR. WISNER

Q. Do you have it, Doctor? Do you have Andreotti in front of you?

THE COURT: Do you want to take a break?

THE WITNESS: No, no. We're moving along.

BY MR. WISNER

Q. I know you just got back from Europe a few days ago.

A. I'm ready to go home.

Q. Okay. So if you look at Andreotti, there is actually -- if you just go to Table 1, I believe it's on Page 3 of 8. Do you see that, Doctor?

A. Yes, I see it.

Q. Okay. And so one of the things that we talked about on direct and was alluded to by Mr. Lasker was that there was different characteristics between the -- to people who never used glyphosate in the AHS and the people who did, right?

A. Correct.

Q. And in De Roos 2005 they actually, because of those differences, chose to analyze the -- the risks relative to the lower exposed than to the unexposed, is that right?

A. That is correct.

Q. All right. So if we look in Andreotti on Table 1, we also have a list of the characteristics of the two -- of the different groups. Do you see that?

A. That's -- yes.

Q. And one thing that I want to just point out here, you see highest level of education. Do you see that?

A. Yes.

Q. High school or less. And it looks like about 70 percent of the never users of glyphosate had high school or less, is that right?

A. That is correct.

Q. Whereas, about 60 percent, or 57.9 percent of the median users had a high school or less. Do you see that?

A. Correct, yes.

Q. And for the heavy users that's less than 50 percent, right?

A. Right.

Q. Now, putting aside issues of pesticide exposures or whatnot, are there such things like socioeconomic differences that might lead certain groups of people to be more prone to cancer?

A. Oh, absolutely. It's certainly well known. The people with high school or less education tend to have worse jobs, truck drivers, offloading and onloading things. So if we talk about diesel exposures, they are probably going to have more

than those with a high school education.

Q. And so would you agree then that when you have really different groups of people and the cases and the controls are so different, that it makes sense to sort of try to find a group that are more similar so you can actually see a fair comparison?

A. When -- when you're doing an exposure response comparison, yes.

Q. Now, you tried to calculate the crude -- the crude odds ratios, in doing that sort of approach with the Andreotti numbers, correct?

A. I wouldn't call them true, but the odds ratios against a different -- the lowest quartile.

Q. I meant "crude," not "true."

A. Oh, crude.

Q. You generate some crude numbers based on what was in the paper, is that correct?

A. Correct.

Q. Obviously, you can't generate odds ratios because you don't have the data and you can't look at the variances and the standard deviations and stuff, right?

A. Correct.

Q. But you can kind of get a sense of what the numbers would be, is that right?

A. Yes.

Q. Now, you recall during the cross-examination Mr. Lasker showed you some emails that you sent to reporters?

A. Yes.

Q. Do you remember that?

A. Yes.

Q. And he pointed out -- if you have their binder -- is his binder up there or no?

A. I have their binder.

Q. You have their binders? Go to, like, Tab 5. This is one of the emails they showed you.

(Witness complied.)

Q. Do you see the email right here, Doctor?

A. Yes.

Q. I don't believe this has an exhibit number, but it's Tab 5 in the binder.

MR. LASKER: We will have to do the exhibits, but go ahead.

BY MR. WISNER

Q. Here you have list Q1 1, Q2 1, Q3 1.6. Do you see that?

A. Yes.

Q. And Mr. Lasker suggested that those numbers were incorrect. Do you recall that?

A. He had questions about them and he stopped questioning. I don't know where he was going with it, but he did mention it.

Q. He said we'll get back to it and we never got back to it,

did we?

A. Yes, correct.

Q. Okay. Well, let's take a look. Let's go to your supplemental report, which is Exhibit 164 in our binder.

A. I don't have "our" binder.

MS. WAGSTAFF: We should do a joint binder next time.

THE COURT: There is never going to be a next time.

(Whereupon document was tendered to the witness.)

A. Thank you.

BY MR. WISNER

Q. Do you have Exhibit 164? Do you find it? 164?

A. Yes.

Q. Okay. All right. And if you turn to page -- do you know where you calculated the numbers? I think it's on Page 2, second-to-the-last paragraph on Page 2. Do you see that?

A. Yes, I do.

Q. And you list the numbers, and the second-to-the-last sentence says:

"This would lead to rate ratios for the quartile analysis of lifetime days."

And then you list the lifetime days numbers. Do you see that?

JUDGE PETROU: I'm sorry. Where are you?

MR. WISNER: I'm sorry. The second paragraph, Page 2, bottom of that paragraph. It's "This would lead to

rate ratios." Second-to-the-last sentence on Page 2.

THE COURT: Exhibit 164?

MR. WISNER: Yes, Supplemental Expert Report of Dr. Chris Portier.

THE COURT: No. This is Dr. Ritz's supplemental report. Exhibit 164?

MR. WISNER: Oh.

THE WITNESS: Not mine.

MR. WISNER: There was a printing error. I apologize.

BY MR. WISNER

Q. Doctor, can I just approach you with your report?

THE COURT: It sounds like he has it and we do not have it.

MR. WISNER: Your Honor, may I approach?

THE COURT: Sure.

MR. WISNER: This is the supplemental report.

(Whereupon document was tendered to the Court.)

BY MR. WISNER

Q. Do you see the part where you discuss the numbers, Doctor?

A. Second paragraph from the bottom, last of that paragraph.

Q. Yeah, last two sentences. Do you see that?

A. Yes.

Q. And then you list the numbers for the -- can you just read those last two sentences?

A. This would lead to rate ratios for the quartile analysis of lifetime days of Q1 equal 1; Q2 equals --

(Court reporter clarification.)

A. Q1 equals 1; Q2 equals 1.096; Q3 equals 1.118; and Q4 equals 1.053. And for intensity Q2 equal 1; Q3 equal 1.06; and Q4 equals 1.048.

Do you want the last sentence too?

Q. That's fine, Doctor.

So those are actually the numbers, although I believe in the email you rounded 1.048 to 1.05, is that right?

A. Yes.

Q. Okay. So have you ever reported different numbers to the best of your knowledge?

A. No.

Q. Okay. I'd like to go back to --

THE COURT: Can I ask? I'm trying to figure out if we should take a break or plow ahead. Do you have a rough estimate of how much longer you have?

MR. WISNER: In my head, ten minutes. But realistically 20.

THE COURT: Let's take a break then. We'll take a break until 3:00 o'clock.

(Whereupon there was a recess in the proceedings from 2:47 p.m. until 3:00 p.m.)

MR. WISNER: May I proceed, Your Honor?

THE COURT: Sure.

BY MR. WISNER

Q. Let's address some quick points from De Roos, 2003. Please turn to Exhibit 15 in our binder.

(Witness complied.)

Q. Do you have in it front of you, Doctor?

A. Sorry?

Q. Do you have it in front of you?

A. Yes.

Q. All right. So this is the 2003 De Roos publication, is that right?

A. That's correct.

Q. All right. I just want to talk about a couple quick points just to clean up the record on this. On Page 3 do you see Table 1?

A. Yes.

Q. And this reflects the Bayesian assumptions made in doing the hierarchical analysis, right?

A. Yes, that's correct.

Q. And to be clear, Bayesian statistics takes the approach that we take what we know in the world and then see how the data comports with what we know, is that fair?

A. Yeah, to some degree.

Q. Whereas, logistical regression, which is sort of more commonly used, just says what does the data show me, is that

right?

A. Yeah. That's a good simple explanation, yes.

Q. That's what my statistics professor taught me.

So if we look at this carcinogenic probability, it says for glyphosate .03. Do you see that?

A. .3.

Q. Yes, sorry. .3, I apologize. 0.3.

And based on the footnote here, that assumption about the potential carcinogenicity of glyphosate is based on the assertion that it's not assessed by IARC or U.S. EPA IRIS or deemed unclassifiable in one or both assessments.

Do you see that?

A. Yes.

Q. Now, we know today that that assumption is wrong, right?

A. Yes.

Q. And classified by IARC, right?

A. But it used this paper to do it so it gets a little circular, but yes. It's now classified by IARC.

Q. Fair enough.

But if we were to redo this analysis today, that assumption about the carcinogenicity of glyphosate would be a higher number, right?

A. Yes.

Q. And I mean, I don't know what number it would be, but it would probably be somewhere between .8, which is a probable

human carcinogen in one assessment and possible human carcinogen in another assessment, or .6, probable human carcinogen in one assessment and unclassifiable in the other. Is that fair to say?

A. Yeah, that's the weight they would give it.

Q. Okay. And if you make the assumption that there is a much higher probability that, in fact, glyphosate is a carcinogen, does that increase -- would that likely increase the odds ratio that we see for the hierarchical analysis?

A. Yes, it would, likely. It would almost certainly.

Q. Okay. Now, there has been some questions about whether or not De Roos 2003 controlled for other pesticides in the logistic regression, is that right?

A. There were some questions, yes.

Q. Let's just first be very clear. Do you have any doubt that De Roos 2003 controlled for other pesticides in the logistical regression?

A. None at all.

Q. Okay. And if we turn to -- we read some portions of it earlier, so I'm not going to read those portions. But I want to point out a few more sentences in here that might be helpful.

If you turn to Page 7, and we're looking at the left column, the paragraph -- the second full paragraph, this is adjustment. Do you see that?

A. Second full -- yes.

Q. And it reads:

"Adjustment for multiple pesticides suggested that there were few instances of substantial confounding of pesticide effects by other pesticides."

Do you see that?

A. Yes, I do.

Q. What do you understand that sentence to be saying?

A. That you adjusted for all of the multiple pesticides they were looking at.

Q. And if you turn the page, Page 8, again the first sentence of the first full paragraph, do you see that?

A. Yes.

Q. And it reads:

"This pooled study of multiple agricultural pesticides provided an opportunity to estimate the effect of each specific pesticide and certain pesticide combinations on NHL incidents adjusted for the use of other pesticides."

Do you see that?

A. That's what it says.

Q. And what do you understand that sentence to mean?

A. They adjusted for other pesticides.

Q. Okay. You were at IARC, correct?

A. I have been at IARC several times.

Q. Let me be more specific. You were at the IARC meeting that assessed glyphosate, right?

A. The working group meeting, yes, I was.

Q. And to the best of your knowledge, did people within IARC when they were looking at this data think that De Roos had adjusted for other pesticide use?

A. Yes.

Q. Okay. All right. Let's turn to Exhibit -- Tab 1B in their binder. It's this EPA submission that you made.

A. Okay.

Q. And actually before we go there, I just want to clarify something, Doctor.

The opinions that you've given about what IARC concluded and what it did not conclude as it relates to glyphosate, is that based both on your reading of the monograph as well as your own personal experience at the actual working group?

A. Would you say the question again?

Q. Sure. You've offered some opinions about what the IARC classification means, specifically with related to real world exposures.

A. Correct.

Q. Okay. That opinion, is that not just based on how you read the IARC Monograph, but also based on the fact that you were actually there in the discussions when they were making this decision?

A. Yes. I have been to roughly eight IARC Monographs and I helped to draft the preamble that sets the rules for what they are doing and how they express it. So, yes, I do understand what they intend this to mean.

Q. And just putting aside your personal opinion for a second. The IARC classification of glyphosate, just by itself, does that support the conclusion that, in fact, glyphosate can cause non-Hodgkin's lymphoma?

A. Yes.

Q. And does it support the conclusion that it can cause non-Hodgkin's lymphoma as it's occurring and being used in the real world today?

A. Yes.

Q. Now, turning to Exhibit 1B in the -- sorry, Tab 1B in their binder. Do you see this, Doctor?

A. Yes.

Q. And this is an attachment that was included as part of your expert report, is that right?

A. That is correct.

Q. Now, I notice this is dated October 4th, 2016, right?

A. Correct.

Q. And your expert report, that's dated -- well, it's dated afterwards, right?

A. Yes.

Q. So to be clear, what exactly are you doing in this EPA

submission or comment?

A. So EPA was evaluating the carcinogenicity of glyphosate, and I spent almost my entire work career writing up how to evaluate and how to interpret studies. I helped EPA write their guidelines. And when I saw the document they had put together, I knew it was not following their guidelines, which are good scientific guidelines. And I have concern that they are not doing what they had said they were doing, what the law requires them to do.

And so my comments here are specific to that document and the deficiencies in that document.

Q. I notice on the first page of this submission you have a disclaimer. Do you see that?

A. Yes.

Q. It says essentially that you're doing this on your own dime, is that right?

A. That's correct.

Q. Why?

A. Because I care. Science -- science is supposed to be there to improve the decision-making process; to protect public health, if at all possible. It wasn't being used here in a proper way. It was being used to -- I don't know why they were doing it, but it was clearly the wrong way to evaluate the data. I felt I had to comment.

Q. Now, the time you prepared this comment to the EPA, had,

you conducted a full Bradford Hill analysis of causation?

A. No, no.

Q. What were you doing then when you submitted this to the EPA?

A. Again, I'm commenting specifically on what EPA did in certain parts of their report. It's certainly not this -- this is not an entire evaluation of all the literature for glyphosate. This is a very specific document.

Q. After you submitted this, did you then sit down and do that full Bradford Hill analysis?

A. Yes, I did.

Q. And in doing it, did you look at a lot more material and a lot more studies and data than you looked at for preparing this comment?

A. Yes. That is true.

Q. Okay. So would it be fair to say then that your opinions as they exist today are accurately described in your expert report but they aren't fully encapsulated in the attachments to it?

A. That is correct.

Q. Okay. Now, in this document there was a couple questions that were asked of you, and I just want to sort of explore them a little bit more.

Now, turning to Page 6 -- I'm sorry, Page 7 of the document. Starting at Lines 116 through Lines 127, you kind of

do a summary of the human evidence section, is that right?

A. 116 to 127?

Q. Yes.

A. Yes.

Q. At the very end of it you ask some rhetorical questions and then answer them, is that right?

A. Yes, that is right.

Q. Okay. And you said here:

"So is causality plausible here?"

And you write:

"Yes, absolutely."

Right?

A. Correct.

Q. What does that mean?

A. That means that there is no reason to suspect from these data in the humans that it is not causal for glyphosate causing non-Hodgkin's lymphoma.

Q. And when you say here yes, that it's absolutely plausible, causation is, can you give me a weight of what you believe that would be, if you can?

A. Based upon my current understanding of all of this and -- I -- I thought I said it this morning. It's about 90 percent. I'm 90 percent there for -- of the way there for absolute undeniable causation.

Q. But you're not 100 percent?

A. I'm not at 100 percent.

Q. And the 100 percent is exactly what the next sentence refers to, when it says: Is it demonstrated? No, clearly not."

Is that right?

A. That clearly refers to the 100 percent.

Q. Okay. And this is also in the context of the human evidence, right?

A. Correct.

Q. Now, when you take the human evidence, which is epidemiology, and you start combining it with the extensive amount of toxicology data, the mechanism data, and what we know about cancer in humans, how does that affect your opinion?

A. That's what got me to the 90 percent, was using all of the information in front of me.

Again, the animal data supports what you're seeing in the human data. The mechanism data supports what you're seeing in the human data. All of it pushes you in the direction of causality.

Q. Now, on Page 4 of this submission, the last sentence -- we talked about it briefly on cross -- it says:

"EPA should declare glyphosate a probable human carcinogen?"

Do you see that?

A. Yes.

Q. And then you go on to say:

"And then conduct a risk assessment."

Right?

A. That is correct.

Q. All right. What exactly is an EPA risk assessment? I think there has not really been a lot of testimony about that. What is that?

A. Okay. So EPA's mission is to protect the public health from exposure to environmental issues, and chemicals are one of them. Pesticides are one of them.

When EPA does a risk assessment, they -- ignoring anything to do about human exposure at this point, they are going to look at the evidence they have in front of them and they are going to try to make a dose-response curve, something they believe links human exposure to the probability of getting cancer for -- sometimes specific cancers, but very seldom is it a specific cancer because most times they build those curves from and animal data.

The reason for that is because very few times do you have human epidemiology data with enough exposure information that you can do a good dose-response curve. So they use other kinds of extrapolations to do that.

So they build that curve. Human dose across the bottom. Probability of cancer across the Y-axis.

Then they say: What are we willing to accept as risk to

the population? One in 100,000? One in a million? And so they take their curve and they estimate what dose will give them that level of risk in the population. And that's probably where they set their standard.

Standards can be very different in this field because it might be in an eight-hour workday no more than this. Over a month no more than that. So standards get a little complicated. But that's the basic gist of it. They are trying to set a standard.

Q. And in setting that standard they look at stuff like absorption rates and how -- how it's being used in an occupational setting and stuff like that?

A. Correct.

Q. Is that really relevant to the question we have here, which is: Does this stuff cause cancer?

A. I -- I guess. I'm always a little vague on what the question is here. But if it's -- if it's an issue of does it cause cancer or not, that is different than the risk assessment issue, absolutely.

Q. And to be clear, even in the context of a risk assessment, I mean the EPA is actually assuming people are going to get cancer, is that right?

A. The expectation is that one in a million people in the country would get cancer if a million people were exposed to this.

Q. So really what they are doing is they are setting a policy of what we're willing to accept people's exposures to this otherwise known carcinogen?
A. It's -- sort of. It's a policy. It's a regulation. It's -- it's society's way of trying to protect themselves.
Q. Now, you understand that, obviously, governments in Europe do things a little differently, right?
A. That's correct.
Q. In fact, you've helped develop some of those standards as well, is that right?
A. Some of the ways they do things, yes.
Q. Are you familiar that the State of California has a set of standards as well?
A. Some of them. I'm familiar with some of them.
Q. You do understand that the State of California has determined that glyphosate is a substance known to cause cancer. Are you aware of that?
A. I was aware of that.
Q. And you are aware that they are currently looking at exactly the question you brought up. What exposure is the minimum allowed? Do you understand that?
A. Yes.
Q. In fact, I believe you submitted a comment to the Office of Environmental Health and Human Hazard Assessment here in California, is that right?

A. That's correct. It was a very short note. They were only considering the animal cancer data that was considered by IARC, and I wanted to suggest that they look a little broader.

Q. And that's because you wanted to make sure they were getting the right exposure levels adjusted for -- in the mice and rat studies as it relates to, for instance, humans, right?

A. Correct.

Q. But that was, to be clear, after they had determined that it, in fact, causes cancer?

A. I don't know that process well enough.

Q. Fair enough.

A. I can't answer that question.

Q. Fair enough.

THE COURT: You don't know how they came to the determination -- whoever "they" is in California, you don't know how they came to the determination that glyphosate is, quote/unquote, known by the State of California to cause cancer.

THE WITNESS: Oh, I do know that.

THE COURT: Okay. How is that?

THE WITNESS: If a -- what's the term they use -- a respected entity or something along those lines declares it, then they will put on it their list for Prop 65. And IARC is on that list. And so when IARC declared it, they took the action of putting it on the list.

THE COURT: I was always a little confused about that. I mean, if a respected body like the IARC concludes something is a probable carcinogen or a possible carcinogen, then the State of California all of a sudden announces that the chemical or the substance is known by the State of California to cause cancer.

How did -- do you have any idea how the State of California makes that leap from "possible carcinogen" or "probable carcinogen" to "known by the State of California" to cause cancer?

THE WITNESS: If I understand California bureaucracy, and I might not, Prop 65 set up a committee that decides who these respected entities are. And once that's done, the rest of the process becomes somewhat regimented.

The actual wording for California has -- knows this, whatever it is.

THE COURT: "Known by the State of California."

THE WITNESS: I think that's in the law. I think that's in the Prop 65.

THE COURT: So this law says that when the IARC concludes that something is possibly carcinogenic or probably carcinogenic, the requirement then is that companies have to declare that it's known by the State of California to be a carcinogen?

THE WITNESS: I think it's almost that. I think when

a respected entity defined by this committee and the committee also decides is it probable or possible, I don't know if Prop 65 works for possible human carcinogen.

THE COURT: It does.

THE WITNESS: I don't remember. I don't know. But I know it is probable.

MR. WISNER: I would be happy to brief the issue if the Court would like.

THE COURT: You don't need to.

MR. WISNER: There is good case law on it.

Okay. Sorry, I went down this rabbit hole of the California EPA. I apologize.

BY MR. WISNER

Q. But I -- I just kind of want to point out something that I think came out just now, this idea of IARC being a respected organization.

You actually -- this came up in cross-examination. There was about this publication that you were looking to have published relating to the glyphosate analysis and comparing it to what EFSA did, is that right?

A. The emailed referred to a letter that we were writing that eventually got published as a publication. But it referred to the letter criticizing EFSA on the way they did their evaluation.

Q. How many people signed that letter with you?

A. I think it was 96.

Q. We've heard a lot of testimony that when you have four epidemiologists you'll have 25 opinions or something. Is there any significance to you, Dr. Portier, that 95 other world-renowned scientists would join you in your statement?

A. They all agreed with the statement. They very carefully looked at it. I don't know what more to say about that.

It was -- we all feel the same way. We spend a lot of time and effort developing methods and evaluating literature. We want our governments to do the same -- we want them to do it right. And so, yeah, they were all very much into this.

Q. I guess my last question or my last follow-up question -- I never say last one because you never know if it will be your last.

When IARC's 17 scientists, the voting members, got together to discuss glyphosate in 2015, did they have anything to gain or lose by classifying glyphosate as a probable human carcinogen?

A. No.

Q. Now, let's contrast that to, for example, the EPA. The EPA has classified glyphosate as a non-carcinogen since the '70s, right?

A. Yes. That's its current classification. In fact, it's slightly different. I think it's unlikely to be carcinogenic in humans.

Q. We talked briefly about the SAP, or the Scientific Advisory Panel, in the EPA's recent assessment.

If, in fact, the EPA were to say tomorrow: You know what? It does cause cancer. They would have to effectively say that they were wrong for 40 years, is that right?

A. I guess that's correct.

MR. WISNER: No further questions, Your Honor.

THE COURT: What about my questions?

MS. GREENWALD: Toxicology.

THE COURT: Just go ahead and answer those questions?

MR. WISNER: I can lead you a little bit.

BY MR. WISNER

Q. The first question was if you get rid of the pooled analysis, how does that effect your toxicology opinions?

A. The pooled analysis is just a tool for me to better understand the strength of the evidence across multiple studies. Like a meta-analysis or the pooled analysis in epidemiology.

Not having it doesn't change the core meaning of the data. And so my opinion of the animal carcinogenicity data wouldn't change just because I couldn't use the pooled analysis.

THE COURT: I don't have a good memory of the discussion that you had with the lawyers about this last time, but there was -- I seem to recall there was some suggestion that this pooling in this context is like unprecedented. And I

think you said: Well, one of the reasons it's rare or unprecedented -- I can't remember the words that were used, but one of the reasons it's rare or unprecedented is that we have so many studies in this context and it's very rare to have so many animal studies regarding the same chemical or substance.

Am I remembering that correctly?

THE WITNESS: That's correct.

THE COURT: Is there really no other, you know, substance that people study for carcinogenicity, where, you know, a similar number of animal studies have been done?

THE WITNESS: Radiation. But radiation is already so heavily regulated nobody changes it. So ionizing radiation.

That's the one that pops to mind. I'm not sure a lot more will pop to mind. DDT and DDE, there were a good many studies on that a long time ago.

Dioxin. There are now two studies on dioxin. Someone could do a pooled analysis there.

But other than that there aren't that many.

THE COURT: So as far as you know, nobody has done a pooled analysis for any of those other chemicals that you just identified?

THE WITNESS: That's correct.

THE COURT: Okay. And what about -- I mean, so every once in a while during this case I get a flashback to a case that I had when I was a lawyer. It was a case -- I represented

the City of San Francisco, and San Francisco passed this law requiring retailers to warn customers about RF energy from cell phones. And there was a challenge to that and I defended that, not successfully.

And I seem to remember there were a lot of studies of both human animal on RF -- on the effects of RF energy.

And, as a matter of fact, if I recall correctly, your friends Hardell and Ericksson did some epidemiological studies on RF energy from cell phone use.

But would that be an example of something where there were lots and lots of animal studies done? Or do you not know?

THE WITNESS: First, to be absolutely above-board on this, I have been retained by a law firm about RF radiation. I don't know if that changes -- you want me to give you the answer?

THE COURT: Not at all.

THE WITNESS: Okay.

THE COURT: Although I'm curious who retained you, what case.

MR. WISNER: Dr. Portier, I would just advise you if you're not violating any confidentiality issues.

THE WITNESS: No, no. I'm just as an expert. I'm not testifying. Just to help in background.

THE COURT: Oh, okay.

THE WITNESS: So in answer to your question, there

are five or -- five or so animal studies. The problem is you can't pool them. One study is in transgenic animals, so it's a completely different type of animal. It's a genetically modified animal.

One study was done --

THE COURT: Monsanto modified the animal?

(Laughter.)

THE WITNESS: One study was done with the animals in a giant wheel pushing their heads towards the middle of the wheel and exposure right on the back of their heads in the middle of the wheel.

One study was done with antennas in the roof of the cage, so they get a uniform exposure to the animals.

And, finally, a third one was done with little antennas glued to the back of the head of the animal.

It would be impossible to pool those together and feel you were doing something reasonable.

BY MR. WISNER

Q. Quick follow-up to the Court's question, although, as you stated, it's not commonly done.

The scientific principles and procedures that you used in conducting the pooled analysis with this unique glyphosate database, are those the same that others use in your field?

A. Yeah. The methodology is the same when you're looking at studies that you can pool.

THE COURT: Again, I don't want to get into the -- whether the methodology is sound or not. I just want to -- my main question was what -- what was left if we took that out?

MR. WISNER: Just wanted a clear question on the record and then I'll move on.

BY MR. WISNER

Q. And the second question related to publications and relying on data that is not published in the context of toxicology.

I mean, I guess if you remember his question, if you could answer it to the best of your ability.

A. Certainly. Certainly.

So first of all, IARC accepts all publicly available information, not necessarily just peer reviewed. The --

THE COURT: Just overall or just in the context of toxicology?

THE WITNESS: Overall.

THE COURT: Okay.

THE WITNESS: Overall. The reason for it is they have in this case 17 experts sitting in a room. They can peer review anything.

And so if somebody gives them a document with far enough lead time, they can peer review that document and decide to use it or not use it. As long as it's publicly available, that's good.

In general, regulatory issues are handled with non-publicly available data. And they -- they do it as best they possibly can or with some definite problems based upon my comments to them. But they do get into it.

Now, different regulatory agencies do it different ways. EPA sometimes actually goes in and looks at the individual animal data and redoes the analysis. But most of the time they take the analysis that's given to them by the contractor that worked for industry and they use that in their risk assessment, as well as all the peer reviewed data.

The same holds true in Europe. They seldom go and look at the actual studies. They take the analysis done by industry and use that to generate their evaluation.

Most people outside of that proprietary framework don't have access to those studies, so they wouldn't use them.

If they were available and I were doing something, publicly available, then I would definitely use them. And I would think that's a sound methodology, to use something you can look at and somebody else can look at it and say: You got this right or you got this wrong.

That's the scientific way. That's -- so transparency is important to scientists like me. But in the real world, the regulatory authorities toe a line between transparency and privacy.

BY MR. WISNER

Q. Doctor, you mentioned briefly industry-sponsored studies. Did you look at the Greim article in this case?

A. Yes, as well as the individual reports from many of the industry-sponsored studies.

Q. That's actually my next question. Did you just rely on the summary estimates in the Greim article?

A. No, because the -- the Greim article was very wrong. The bottom line is, I went in and analyzed everything all over myself to make sure that I fully understood what the animal evidence was telling us. And I think that's normal for anybody wanting to do this type of work who has the time. That would be the methodology you use.

THE COURT: So the only thing you didn't do, it sounds like, was go to the reading room.

THE WITNESS: I'm -- I'm not a big fan of wasting my time. And that was my opinion of the reading room.

BY MR. WISNER

Q. I guess my last question is, and this has sort of been covered already, but I just want to make it very clear: The procedures used by IARC, are those procedures and methodologies generally accepted in the scientific community?

A. Definitely.

Q. And the procedures and methodologies that you used in arriving at your opinion, did you use those methods that are

generally accepted in your scientific community?

A. Yes. Generally accepted in the scientific community, except it seems they don't like my pooling, some of them.

I'm joking. I think they are generally accepted in the scientific community.

MR. WISNER: Thank you, your Honor. No further questions for real this time.

THE COURT: Anything further?

MR. LASKER: Nothing further, Your Honor.

THE COURT: Thank you very much, Dr. Portier, for coming back. I appreciate it.

THE WITNESS: Thank you.

(Witness excused.)

THE COURT: Okay. I think you don't have to deal with us again for quite a while probably.

Is there anything else anybody needs to discuss before we head out and try to catch our airplanes and whatnot?

MS. GREENWALD: I have one question. Do you want any page limits on the submissions for next week or -- I didn't know. We didn't ask that earlier.

THE COURT: I really would prefer that you not address anything other than the issue that I asked you to address.

MS. GREENWALD: Correct.

THE COURT: So I can't imagine that it will take you

a lot --

MS. GREENWALD: I agree.

THE COURT: -- to do that. But I will let you take the space that you need, okay?

MS. GREENWALD: Have a nice vacation. Thank you.

THE COURT: Thank you.

THE CLERK: Court is adjourned.

(Proceedings adjourned.)

I N D E X

Friday, April 6, 2018 - Volume 1

– – –

CERTIFICATE OF REPORTER

I certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

Debra L. Pas, CSR 11916, CRR, RMR, RPR

Friday, April 6, 2018