SUPERIOR COURT OF CALIFORNIA

COUNTY OF ALAMEDA

BEFORE THE HONORABLE WINIFRED Y. SMITH, JUDGE PRESIDING

DEPARTMENT NUMBER 21

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COORDINATION PROCEEDING SPECIAL TITLE (RULE 3.550) ROUNDUP PRODUCTS CASE	**JCCP No. 4953**
THIS TRANSCRIPT RELATES TO: Pilliod, et al. vs. Monsanto Company, et al.	**Case No. RG17862702** **Pages 2420 - 2662** **Volume 16**
	Pages 2424 - 2430 FILED UNDER SEAL BY ORDER OF THE COURT AND BOUND SEPARATELY

Reporters' Transcript of Proceedings

Monday, April 8, 2019

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(Multiple other counsel present as reflected in the minutes.)

I N D E X

Monday, April 8, 2019

E X H I B I T S

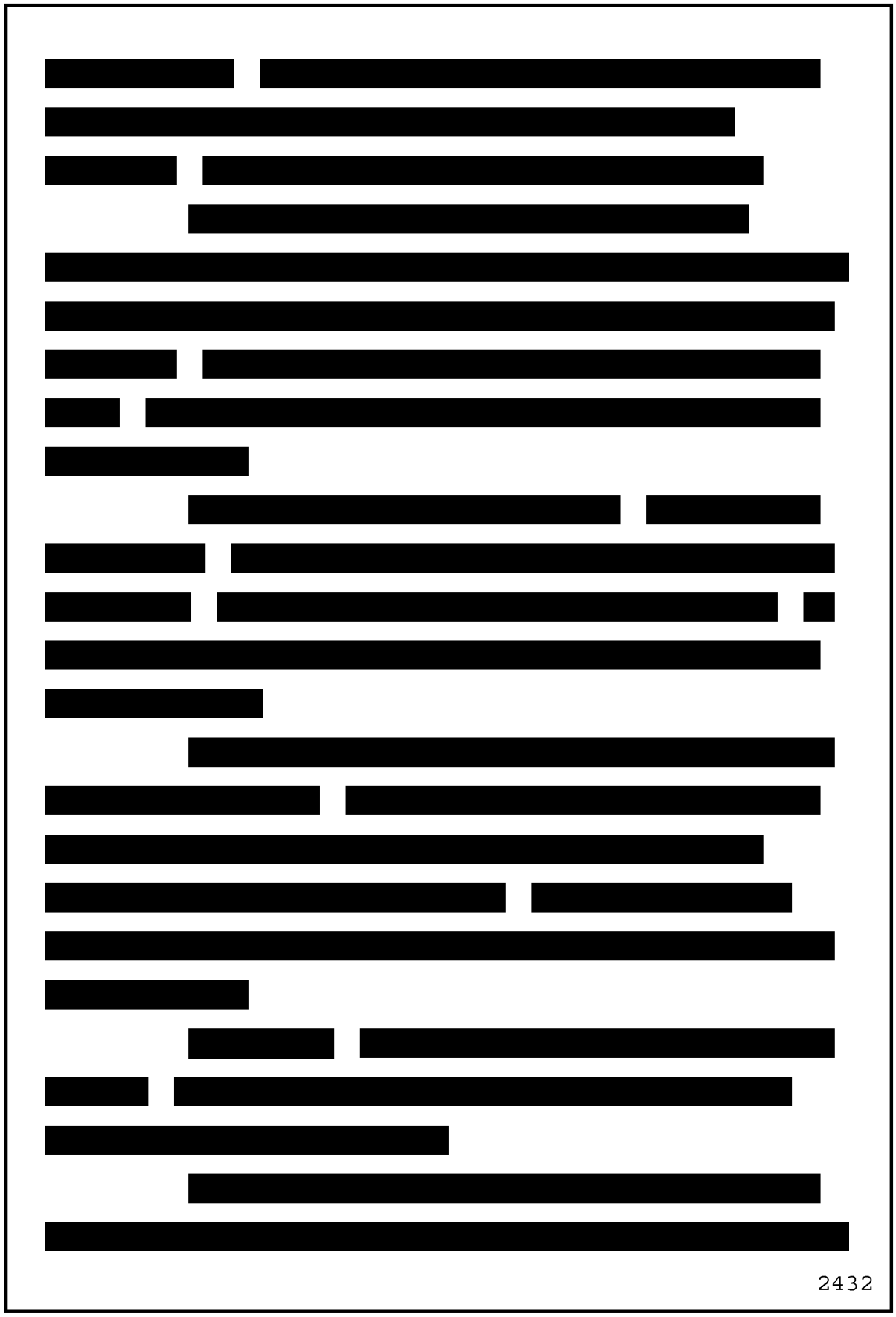
Monday, April 8, 2019 9:01 a.m.

(Proceedings held in chambers outside the presence of the jury.)

(Pages 2424 through 2430 were placed under seal by Order of the Court and bound separately.)

(The following proceedings were heard in chambers outside the presence of the jury:)

[redacted]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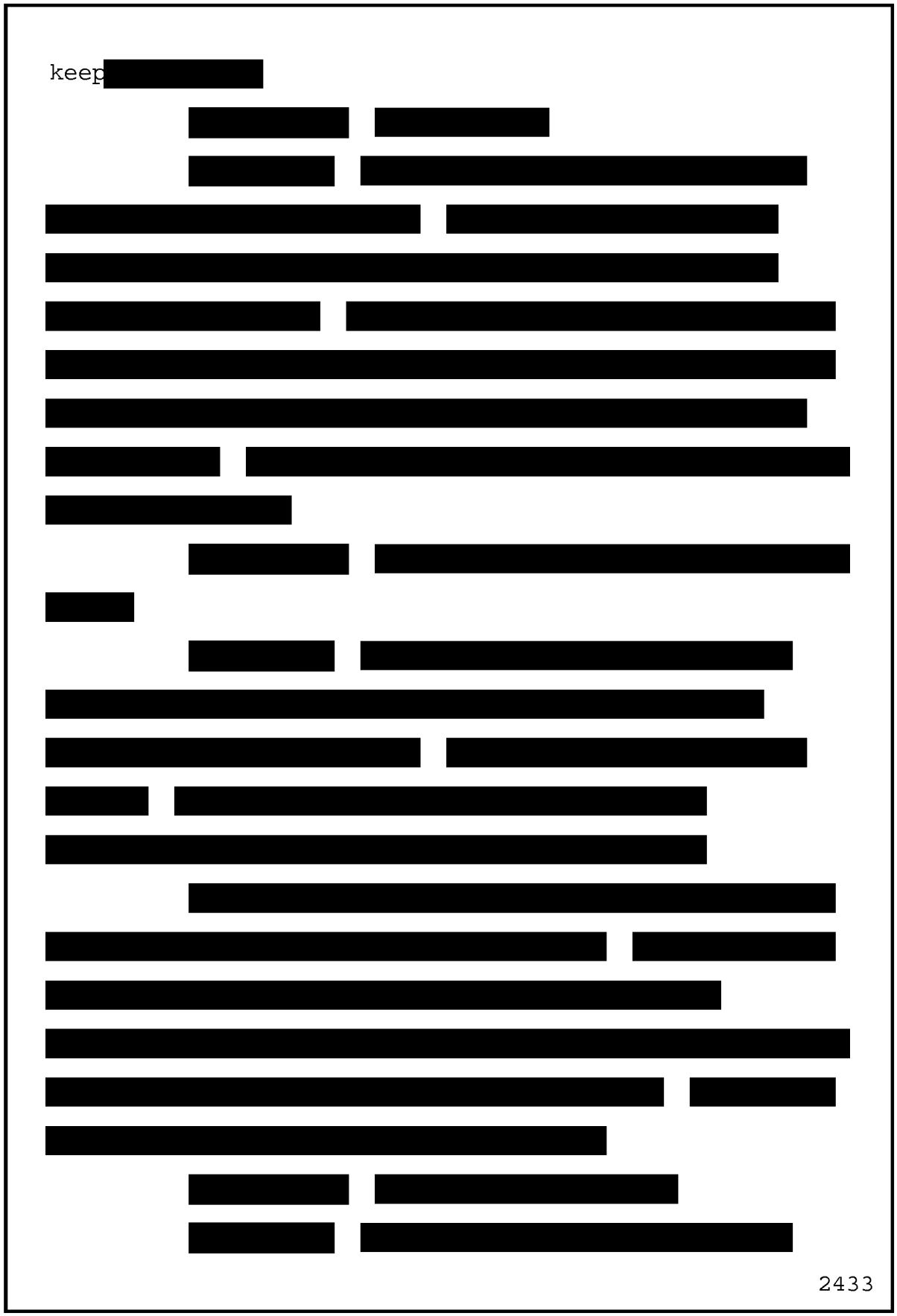
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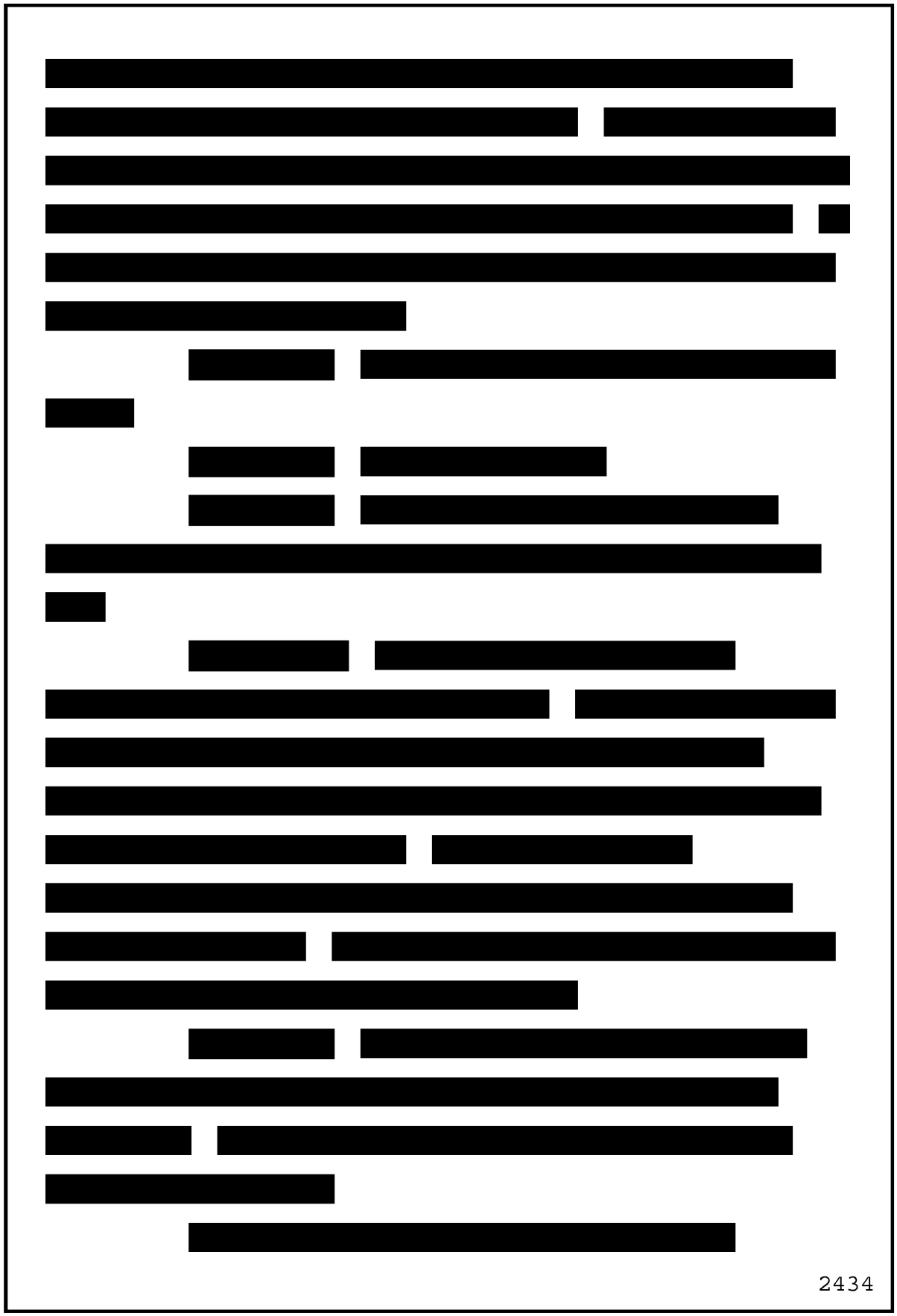
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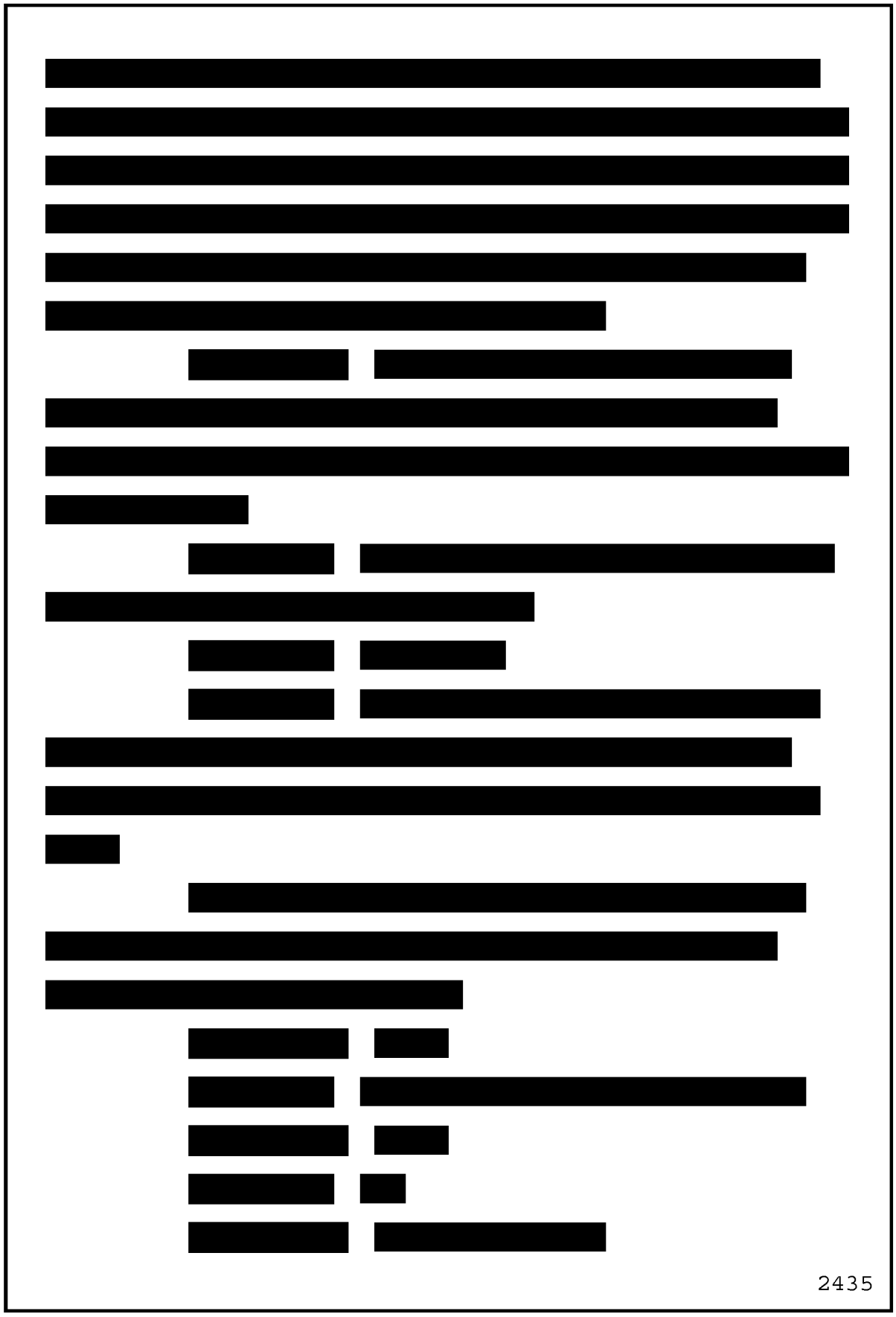
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[redacted]

(Recess taken at 9:05 a.m.)

(Proceedings resumed at 9:08 a.m.)

(Proceedings continued in open court in the presence of the jury.)

THE COURT: So we're back. Hope you had a good, long, restful weekend and forgot you were jurors and had juror amnesia. We're back with evidence. Plaintiffs are going to continue their case.

And Mr. Miller, you may proceed.

MR. MILLER: Thank you, Your Honor. Good morning.

THE COURT: Good morning.

MR. MILLER: And good morning to you folks. First thing we're going to do is read a request for admission. And it's Request for Admission Number 31:

"Admit that Monsanto has never conducted an epidemiological study to study the association between glyphosate containing formulation and non-Hodgkin's lymphoma."

And Monsanto's answer: Admitted.

MR. MILLER: Now we would call our next witness, Beate Ritz.

BEATE RITZ,

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

THE CLERK: Would you please state and spell your name for the record.

THE WITNESS: My name is Beate Ritz. B-E-A-T-E, last name, R-I-T-Z.

DIRECT EXAMINATION

BY MR. MILLER:

Q. Good morning.

A. Good morning.

Q. Who are you?

A. My students call me Dr. Ritz.

Q. Okay.

A. I teach at UCLA. I'm a professor of public health in the Department of Epidemiology. But actually, my salary comes from the Center for Occupational and Environmental Health, which is a State of California paid position to investigate occupational and environmental health risks.

Q. We're going to get into that in more detail. Let's go back to the beginning.

You're a medical doctor?

A. Yes.

Q. You're a neurologist?

A. I wouldn't call myself a neurologist. But I got trained in Germany, and I got trained in psychology and neurology, and I do a lot of neurotoxin-related studies.

Q. And then -- and I'm going to put your CV, Exhibit 3055, I'm going to publish it with permission from the Court.

THE COURT: Any objection?

MR. EVANS: No objection.

THE COURT: Granted.

BY MR. MILLER:

Q. So I want to walk through this with you a little bit.

This is your curriculum vitae, right?

A. Yes.

Q. And what does curriculum vitae mean?

A. That's -- we call it CV, in short. It's a statement of my educational and professional background and all of -- it's a summary of all of the -- bibliography of all the work I did and projects I conducted.

Q. And as you just explained to us, you're a professor, and you're in the Department of Epidemiology and Environmental Health at the University of California Los Angeles School of Public Health?

A. Correct.

Q. And how long have you been there?

A. I came in 1989, because I actually studied there. I got a Ph.D. in epidemiology before they hired me in 1995 as an assistant professor, based on my degree from Germany, actually.

Q. And after we go through your qualifications, we're going to ask you opinions about the issues in this case.

You understand that, right?

A. Yes.

Q. When you give us opinions, will you apply the same analytical skills you apply and the same constructs you use when you teach medical students or graduate students or interns?

A. Yes, absolutely.

Q. All right. Let's go through them.

You have a Ph.D. in epidemiology from UCLA?

A. Right.

Q. How many years has that been now, 1995?

A. Twenty-some.

Q. I lost count myself.

And you have a doctoral degree in medical sociology. That's actually a medical degree in Germany?

A. Yes. That's actually a doctoral degree, in

addition to the M.D. So I have two Ph.D.s.

Q. And you're the chair of the Department of Epidemiology.

What does it mean to be the chair?

A. I was the chair of the department, which is an administrative function. But mainly what we do is, we decide what the curriculum for Ph.D. students and master-level students in epidemiology should look like.

So the chair is deciding mostly on the teaching curriculum.

Q. How many epidemiologists have you had under and with you there at UCLA?

A. Oh, boy. We have 200 students per year. Half of them are doctoral students. And I personally mentored at least 50.

Q. Okay. Let's talk about editorial boards. We haven't heard about that yet, I don't think.

What does it mean to be on the editorial board of a journal?

A. It means that you have the tasks, the work of an editor. So when a paper gets submitted for publication, then you are one of the people who are looking at it first, deciding whether it belongs in this journal. And then also deciding who should review it.

In my function, at the time, on the board at

EPIDEMIOLOGY, you would also do the review yourself. So you would get the most appropriate articles to review in your area.

Q. Okay. And I've heard the phrase before, "impact journal."

What does that mean?

A. So high impact journals are the journals that are very well-respected in our field, that we all volunteer to review, because none of this is paid. And we are very proud of that peer-review process, because that's how science works.

We are criticizing each other. We are asking questions of manuscripts. Only if the reviewers are really satisfied and the editor is satisfied with what they see, will a paper be accepted for publication.

Q. Is EPIDEMIOLOGY a high impact journal?

A. Yes.

Q. And for six years, you were on the editorial board at EPIDEMIOLOGY?

A. Yes. Actually, it is the official origin of the International Society for Environmental Epidemiology, and has been since its inception.

Q. And we're going to talk more about that.

I want to look now, if I could, since 2001, you've been the chair of the external advisory committee

for NCI/NIEHS. Goodness gracious.

What is that?

A. National Cancer Institute is NCI, and NIEHS is who gives me all my money for my research. That's the National Institute of Environmental Health Sciences, which is located in North Carolina, right opposite of the EPA.

Q. And you're also a member of the EPA Science Advisory Board for Human Health Research Strategy?

A. Yes. That was one of my advisory committee duties at one point.

So these are temporary, interim advisory boards that you get put on. And you do your job, and then you're off again.

Q. Sure. But to be invited, you consider it an honor?

A. Oh, yes. That's why we do it, and it's all unpaid. It's service.

Q. I understand. All right.

Now, we're not going to go through everything on your CV, but there are some things we want to talk about. Tell the jury about grants, funded research.

What does all that mean?

A. Right. So, I mean, I can teach at UCLA and mentor my students. But what I also need to do is

research. UCLA is a research university, not just a teaching university.

When I do research, I do human research, epidemiology. That means we actually have to approach human beings, and we have to interview them, assess their health.

So when I do a study of Parkinson's disease in the Central Valley, neurologists who work with me go out with me and see every single patient multiple times. And then my students who are trained by me are interviewing these people, taking their blood, bringing home samples of urine, soil, whatever we need.

And all of that costs money. My students need to pay their tuition and need to live, so I usually pay them for this work. And we get this kind of money from the National Institute of Health, which the National Institute of Environmental Health Science is part of. So I have to write a grant. It gets peer-reviewed in a big committee. And then I get scored.

And if I'm better than 90 percent of all the people who submitted grants, in the 10th percentile or less, then I get money.

Q. There you go.

And you've been successful in convincing these federal agencies that you ought to be doing research for

these purposes?

A. Yes. And I have a long list of funded grants.

Q. We're not going to go through all of them, but let's look at a few.

So you do quite a bit with the environment.

Is that fair?

A. Yes.

Q. And I see you're doing one down here for NASA.

What is that about?

A. That's actually a really interesting grant. I do air pollution and pesticide work. And this is the part that I consider my air pollution work.

So this NASA grant, I was approached by somebody from JPL, Jet Propulsion Lab, who designed an instrument, a camera, that actually takes photos of polarized light in the air column. So you have a satellite, a camera on it, and they are looking at the Earth. And they are measuring particle and composition of particles in the air column.

That satellite will go up in 2020. And in the meantime, what we are doing as epidemiologists -- I'm not a satellite person, but what I'm supposed to do is assemble all the health data for Southern California, Ethiopia, Chile, and Taiwan to see if different levels of air pollution in these different areas are actually

related to health effects. And we will be doing that throughout 2020.

Q. I see you had grants to study pesticide?

A. Yes. That's one of my smaller grants on pesticides.

Q. I understand you had more than a few grants to study pesticides?

A. That's correct.

Q. Is that fair?

A. Yes.

Q. Would it be fair to say that pesticide exposure and its impact on humankind has been an area of research for you over your professional life?

A. Yes. In 1995, I decided that pesticides for California are a very important environmental and occupational health risk. And that as an official in my center, I should better be studying this, just like air pollution is.

And I've been pursuing NIH for funding for many, many years. And I've studied neurotoxins, childhood cancers, autism, Parkinson's; many, many outcomes.

Q. So pesticides and childhood cancers, you got a grant from --

A. NIEHS, yes.

Q. -- the National Institute of Environmental Health Sciences?

A. Correct.

Q. That was in 2011 to 2013?

A. Right.

Q. Before Roundup, you were never an expert witness before?

A. No. No.

Q. So you've been doing this long before we ever asked you to look at this case, right?

A. Absolutely. Twenty years.

Q. Sure.

I want to ask you about this $7 million grant from the National Environmental Institute for Health Sciences.

A. Yes. So that was actually a center that I co-directed with Dr. Chesselet, who was a neuroscientist, and it was specific to look at neurotoxins and the combined effect of pesticides and genetic predisposition to Parkinson's.

And that was a center that was actually funded twice. And the 7 million is, I think, only part of what we got. We got about 15 million to do this. In total, I think was -- all of the different fundings we got from this, it was about 15 million.

But more people than me worked on it. I did all the human work, but there was a lot in animals. Yeah.

Q. As a scientist, do you study the effects of pesticides on animals and cells, as well as humans?

So you look at all three areas of science?

A. Well, I myself don't do animal studies, and I don't look at cells either. I just look at humans.

But I collect cells from humans. I collect blood cells from humans, and we use these many different ways in a lab. I don't have a wet lab myself, because I can't do everything; I'm not an expert in everything.

But I work very closely with basic scientists who do all this work. We collect the samples, we decide on hypothesis, and we look at the data together that comes out of this lab experiment.

In fact, this center you showed a minute ago was specifically funded so people like me can do human research. M.D.s and more clinical people work better with basic scientists who then look at animal studies and cell studies, and we actually learn to talk with each other.

That was a great experience. And I learned, over 15 years, to actually understand these studies, maybe 80 percent.

Q. As part of your job, high-ranking epidemiologist for the State of California, when you come to conclusions about hazards to California citizens, you look at the epidemiology -- you're an epidemiologist, but do you also factor in the animal studies and cell studies?

A. Absolutely.

Q. Is that the way good scientists do that?

A. Yes. And I specifically learned to do that.

Q. You're trained that way, correct?

A. Yes.

Q. Let's go to page 11 of your CV, another grant you received to study prostate cancer and pesticide exposure in diverse populations in California's Central Valley.

Tell us a little bit about that.

A. Yes. This is with my long-term collaborator, Dr. Cockburn, from University of Southern California. And we designed a pesticide exposure model that is based on a very, very unique tool that the State of California has, and which is called the Pesticide Use Report System.

So every farmer in California who uses and applies pesticides to a field has to actually report what he applied, when he applied it, how much he

applied, and where he applied it on a field.

And that goes into a central database called the Pesticide Use Report System, and we can actually get this data, it's public data. And we can use it to map where pesticide use happened in California.

So when we know in our studies where people lived, we can actually see where they worked, where they lived, and how much pesticide was applied right where they lived and worked. And that's what we did for this study.

Q. Now, there are epidemiologists out there in the world, the scientific community, that are not environmental epidemiologists, right?

A. That's correct.

Q. And the distinction I would like you to articulate for us: Do generic epidemiologists know, train, study, and work every day with exposure assessment models like you folks do?

A. Absolutely not. That's the specialty that I teach and that I was trained in.

So rather than -- you sometimes hear somebody is a cancer epidemiologist or a reproductive epidemiologist. So these people define themselves according to the disease they study.

Q. Sure.

A. I call myself an occupational and environmental epidemiologist because my profession is grounded in doing exposure assessment and doing that right.

Of course, I'm an M.D.; of course I understand diseases. But really what our specialty is, is get the exposure assessment right.

Q. And isn't that the hardest part of deciding what, if any, pesticides cause what, if any, problems? An accurate exposure assessment model?

A. Absolutely. That's what I teach my students in the classroom. Because we have so many beautiful medical tools to actually define diseases. And I can send my neurologist to examine the Parkinson's patient, and I know it's a Parkinson's case and not something else. But to get the exposure assessment right is a real science.

Q. You work for the California Air Resources Project?

A. Board, yes.

Q. Board, excuse me.

A. I had a project for them. The California Air Resources Board gives out some money for scientific studies of air pollution. So I had a few of those types of funding from the State, as well.

Q. Who appointed you to that?

A. Oh, and I'm actually a scientific advisor on the Air Toxics Board for the State of California, and that's a governor appointee.

Q. And let's go back now to your CV and look at it some more. We'll get to your opinions in a second.

Page 14, you did another pesticide exposure modeling to look at long-term health effects in 1999?

A. Yes. That was actually the beginning of my career in pesticide epidemiology.

We first had to set up the exposure model, and we had to actually convince people that we could model exposures. And that's what we did at that time.

Q. And the objectives of this grant in 1999 were to:

"Develop geographic model for pesticide exposure of California residents between 1950 and 1990 using satellite images of crops, aerial photographs, and pesticide use reporting data from the California Department of Pesticide Regulations."

A. Correct.

Q. Something you've been studying for a long time?

A. Yes. And improved over the years.

Q. That's what science does, right?

A. Yes. And actually, we could only do this because of the explosion of computer technology and imaging technology.

So what we had to do on paper maps in 1999, it's all digitized now. And I employed undergrads at UCLA for five years to digitize maps.

Q. Not only do you receive grants, but you've been asked to review other scientists' grants to decide if their scientific hypotheses are worthy of federal funding?

A. Yes, I do that regularly.

Q. You've done that a lot, haven't you?

A. Yes.

Q. Do you consider that an honor?

A. Yes. I consider it an honor. You're considered a peer, I guess.

Q. And we've highlighted some of those scientific organizations that you've decided whether other scientists should be allowed to study what they propose?

A. Right. In more recent years, I've been also internationally reviewing mostly pesticide grants. One for India, and I think one for South Africa.

Q. For other countries?

A. For other countries, yes.

Q. Wow. Okay.

I'm not sure we've heard yet or not, but what is a journal reviewer?

A. A journal reviewer does the job of actually reading a paper that is being sent to a journal. After the editor, he's the first person to read it.

And we are asked our professional opinion whether the scientific methods applied in this paper and the conclusions by the authors are correct. Or if we have questions, we ask a lot of questions.

Q. I'm not going to ask about every journal you review for, but a few.

American Journal of Epidemiology?

A. Right.

Q. And a second journal called EPIDEMIOLOGY?

A. Yes.

Q. Then the International Journal for Epidemiology, right?

A. Yes.

Q. The Annals of Epidemiology?

A. Yes.

Q. Environmental Health Perspectives?

A. Right. That's actually the official journal of the National Institute of Environmental Health Sciences.

Q. Occupational and Environmental Medicine?

A. Yes.

Q. JAMA. What's JAMA?

A. Journal of the American Medical Association. It's considered a very high-level medical journal.

Q. And you're a reviewer for their articles?

A. Yes.

Q. That means you edit, reject, accept, or recommend --

A. I recommend, yes. I ask questions, I review, and I'm asked by the editors to say whether I would accept or reject a paper.

Q. And finally, The Lancet?

A. Yes.

Q. Which is --

A. It's the British equivalent to JAMA.

Q. I've heard British doctors tell me they think it's better than JAMA?

A. Yes.

Q. We'll leave that discussion for later.

We're not going to go through every peer-reviewed article because we want to get on to the heart of the matter.

But you've published how many peer-reviewed articles?

A. I think now, it's about 278.

Q. Would it be fair to say that a significant number of them deal with the issues of pesticide and their effects on mankind?

A. Yes, absolutely.

Q. You also signed on to a letter that I think we've heard about in this case, and it's in your CV. That was in the scientific journals, 40 years of IARC?

A. Correct.

Q. Why did you feel strongly enough to support IARC after they were challenged?

A. I actually consider this a real privilege, because I consider the International Agency for Research on Cancer the worldwide authority on establishing whether an agent is a carcinogen.

I spent one year, 2006, at IARC as a visiting professor. I was part of one of their Monograph reviews, and I saw the whole process. And I was extremely impressed by the rigor and the science that was applied, and the independence of the researchers who participated.

Q. Finally, there are medical textbooks out there in the world, right?

A. Yes.

Q. And you've written chapters in books?

A. Yes.

Q. And I'm not going to pronounce the first one. It looks like that might have been in German?

A. Yeah. When I started publishing, that was in the '80s, we were still writing books. Now we are writing articles.

Q. All right. A few more questions about your qualifications, and then we'll move on.

So you have been repeatedly asked to advise the State of California on the issue of pesticides and their effects?

A. Yes. Actually, at the Air Toxics board meeting, we assessed whether or not to classify pyrophosphate as an air toxin.

Q. Now, we've heard about the Agricultural Health Study in this trial.

A. Yes.

Q. Okay. And you were on the advisory panel that oversaw that study?

A. Correct.

Q. In fact, at one point, you served as the president of the advisory board?

A. I served as a chair one year when we still had meetings, yes.

Q. And before this old lawyer ever called and

asked you to look at this stuff, you had been teaching your students about some of the strength and weaknesses of that body of data, hadn't you?

A. Right. So I teach methods, applied methods in epidemiology, and it's always good to have an example.

I like to use examples that I know a lot about, and the Agricultural Health Study is one I know a lot about because I was sitting on the advisory panel. So I use that study in my class teaching, and I've used it for decades.

Q. And you mentioned at the beginning of our qualification discussion, the International Association of Environmental Epidemiologists?

A. Correct.

Q. Tell us a little more about that organization.

A. So this is an organization that was founded in the late '80s. And it was -- it actually started in California, but it was meant to be an international society and very quickly became very international. So it is now covering every continent.

And it is a society of the professionals who do the kind of work I do, assessing environmental and occupational hazards worldwide. A large part of it is air pollution, but pesticides are another large part of what we professionally investigate.

Q. Do they have a president of that organization?

A. Yes, they do.

Q. Who is it?

A. It's currently me.

Q. So you're the president of the International --

A. Yes.

MR. MILLER: Your Honor, at this time, I move Dr. Ritz in as an expert on the causes of non-Hodgkin's lymphoma as relates to pesticide and pesticide exposure.

THE COURT: Any voir dire?

MR. EVANS: Subject to prior motions, we'll reserve for cross, thank you.

MR. MILLER: All right. Exhibit 1144 has been published to the jury before.

BY MR. MILLER:

Q. We've asked you to look at some issues in this case about Roundup, haven't we?

A. Yes.

Q. And you're going to give me your opinions to a reasonable degree of medical certainty?

A. Yes.

Q. And this jury has heard a lot. But let's cut to the chase. Let's get it clear.

Does Roundup cause tumors in mammals?

A. According to my readings, yes, it does.

Q. Does Roundup cause malignant lymphoma in mice?

A. Yes.

Q. Does Roundup cause genetic damage in human lymphocytes?

A. Yes.

Q. Does Roundup cause oxidative stress in human cells?

A. According to the research, yes, it does.

Q. Does Roundup cause non-Hodgkin's lymphoma in humans at real world exposures?

A. Yes. According to the epidemiologic literature, I would say yes.

Q. Let's talk about methodology first, a little bit about what it is that epidemiologists do that makes them so unique.

So what is an odds ratio?

A. An odds ratio is what it says. A ratio. A ratio of odds.

Easier -- it is trying to actually capture what we call a risk ratio, and a risk ratio is a little easier to understand. It is the number of people who are exposed and get the disease over the number of people, the ratio of people, who are unexposed and get the disease. So you have a ratio of exposed and disease

over unexposed and disease.

So you can see that if this ratio is above 1, then the exposed have more disease than the unexposed. That's as easy as that.

If the ratio is below 1, then it means the exposed actually have less than you would expect, because the unexposed have more cancers.

Q. What does it mean, adjusted findings versus unadjusted findings?

A. That is a concept that relates to what we call confounding or confounders.

So when we try to find out whether one factor causes a disease, we cannot apply a pesticide to a human population and wait 15 years and see what happens. That's unethical and unpractical.

Q. So if somebody wanted to do a test now and give 1,000 people -- say these thousand people will be exposed to Roundup for the next three years, take this 1,000 people and not expose them to Roundup, and see who got the most non-Hodgkin's lymphoma.

Would that be ethical?

A. No.

Q. Why not?

A. I mean, if you are suspecting or knowing, as we now do after many years of epidemiologic studies,

that there's harm involved, and we're expecting NHL to happen, that's highly unethical to expose human subjects in that way.

So that would be a controlled experiment in humans. We only do this by taking things away. Like putting water filters in people's homes to clean the water rather than putting toxins in, right?

So we would not go and put toxins on anyone.

Q. You can't paint the playground monkey bars with lead at one park and not with lead in the other, and see what happens to these children.

That would just be unethical?

A. It even would be unethical not to remove the leaded bars now because we know what happens, right.

Q. So before we get to the actual studies upon what your opinions are based, I want to look at Exhibit 1093.

MR. MILLER: It's been published before.

MR. WISNER: It actually hasn't.

MR. EVANS: No objection.

MR. MILLER: Exhibit 1093.

Can you blow up the top half of that. Thank you.

MR. EVANS: Your Honor, hold on just a second.

Can you take that down.

MR. MILLER: Yes. It's down.

MR. EVANS: I do object to this, Your Honor. It's outside the scope of what this witness has talked about before. It's not in any of her reports or prior testimony.

MR. MILLER: That's simply not true.

We can pull out her depositions and pull out her report, Your Honor. But I don't know if the Court wants us to do it standing right here.

THE COURT: Sidebar.

(Sidebar discussion not reported.)

BY MR. MILLER:

Q. We're going to talk to you about this, but not publish it.

Do you agree with the State of California when they've listed glyphosate as a known cause of cancer?

A. Yes.

Q. And who are the men and women scientists in the State of California that make that decision?

A. It's an office called OEHHA, Office of Environmental Health Hazard Assessment.

They also help put all the documents together that we review on the Air Toxics Board. So I know that office quite well.

Q. Are you aware of any of the men and women

scientists in California who have not agreed with the positions stated by the great State of California that Roundup is a known cause of non-Hodgkin's lymphoma?

MR. EVANS: Objection. Your Honor. Hearsay.

THE COURT: Sustained.

BY MR. MILLER:

Q. Let's talk about how you reached the opinion that Roundup causes non-Hodgkin's lymphoma. And if you go to your book --

MR. MILLER: And with the Court's permission, we're going to publish Exhibit 0031, the first Hardell study.

No objection to Exhibit 0031. All right. We can blow up the top third of that, please.

BY MR. MILLER:

Q. Now, this is -- we've talked about peer-reviewed studies, right?

A. Correct.

Q. This is a peer-reviewed study?

A. Yes.

Q. And it's in the American Cancer Society Journal, and it's published in 1999?

A. Yes.

Q. Was this the first study that dealt with the issue of Roundup and non-Hodgkin's lymphoma?

A. No, it wasn't the first. But there were several American studies prior to this.

Q. And what is the significance of this study of Dr. Hardell and Dr. Eriksson?

A. This is the first study not on the American continent, and it's based on a Swedish database that we all consider, as epidemiologists, to be very solid. The Swedes, the Norwegians, the Danish have medical records you can actually rely on for these studies.

Q. And does this study help inform you about whether this is an association between Roundup and non-Hodgkin's lymphoma?

A. It is part of my assessment, yes.

Q. And this is a case-control study?

A. Yes.

Q. Okay. So tell the jury, if you could, what a case-control study is.

A. A case is a case of cancer; in this case, a case of non-Hodgkin's lymphoma. And so what we're trying to do is, we are assessing a case -- first of all, we make sure it's a case, it's really non-Hodgkin's lymphoma. So this is a cancer registry, but they also go back to the medical records and pathology to make sure it is non-Hodgkin's lymphoma and not something else.

And then what you're doing is, you're going to these other registries that the Scandinavian countries have and you pull a control.

So a control is anybody else who is alive and living in the same district or department, whatever they call it here, who has maybe a similar age, the same sex, and some other characteristics. That's what we call, sometimes, matching.

And then what we do is, we try to interview all of these people. So we interview the cases about their lifestyle factors, their occupations, whether they applied pesticides, how they applied them, when they applied them.

And the controls serve as -- I told you, what is the rate of disease among the exposed over what is the rate among the unexposed, right?

So the control, actually we can turn that around and say, what is the rate of disease -- yeah, I said it -- in the exposed versus unexposed?

So we can actually, then, the controls, what they're doing is giving us the exposure rate among those who were not diseased.

So if the cases were exposed in the same way as the controls, my ratio would become 1, right? Because we would see exactly the same number of cancers

among the exposed and the unexposed.

So the controls are really giving us the other side of the coin, which is: What's the disease rate if you're not -- under -- what's the exposure rate? And then, is it the same in the controls, those who didn't have the disease, as it is in the cases. Right?

So it's just giving us that comparison group that we need. Because otherwise, we just have cases, and we know what the exposures were. They're not all 100 percent exposed, right? A certain percent is exposed, another percent has NHL for other reasons.

But we use the controls to compare and say, well, if there is more exposure among the cases than among the controls -- if it's double, for example -- then we have a doubling of risk.

Q. Let's look at the last page of this first Hardell study, 1999. I want to ask you about the paragraph, "Other much-used pesticides." I want to read this to you and ask you what we should learn or take away from this:

"Other much-used pesticides -- that is, glyphosate -- also might be of concern. In fact, in this study, four cases and three controls were exposed to this herbicide. Odds ratio 2.3, confidence interval .4 to 13."

Now, tell us what all that means, so we know.

A. So this odds ratio gives us the full increase in risk, 2.3-fold increase in risk when you are exposed to glyphosate to have non-Hodgkin's.

However, this is based on four cases and three controls who were exposed to glyphosate. What that tells you is, it correlates to what we call the 95 percent confidence interval. And you probably want to look at that slide.

And that confidence interval is very wide. It's .4 to 13. And I told you what this ratio does. A 1 means there's nothing, less than 1 means it's protective, more than 1 means there's an increase in risk.

So this says it may be protective or it could increase my risk 13-fold. The central estimate is 2.3. So I know very little, except that my best guess is 2.3. But this data is not sufficient.

Q. Okay. If we can switch to Exhibit 0109, published by agreement.

Tell us what this means in the context of what we were just discussing, the statistical significance, confidence interval.

A. You see the 1.5. It's not a 2.3, it's a 1.5. That's the central estimate, the dot.

That's the estimate that, if you're just comparing the risk in the exposed versus the risk in the unexposed, that's what you get. The 1.5 means it's greater than 1. So 1.5 is 50 percent increase in risk, right? Or 1.5-fold increase in risk, sometimes we say it that way.

However, you can see that sometimes we have these brackets. And that's the confidence interval. I don't have enough data to say this is statistically significant. Why? Because the .9 is below 1.

So, if anything is below 1 in this confidence interval, it means it's not statistically significant according to the p-value that they state, which is usually .05.

On the other hand, you see that the upper limit of the confidence interval is 5. So what we know here is, if we repeat this experiment, this study, multiple times, then we could -- the true effect could be as low as .9, so a 10 percent protection, or as high as a 5-fold risk increase. But from this data, I can't say.

Q. Great time to ask you about the area under the curve. Let's introduce that concept.

A. This is saying what I said before. It explains these confidence values to you.

MR. EVANS: Objection.

THE COURT: Why don't you hold on and give me a clear objection.

MR. EVANS: Yes. We -- if you could just show it to me and tell me what exhibit it is before it is put on the screen, that would be great.

MR. MILLER: Yes, fine. Of course.

BY MR. MILLER:

Q. Let's talk about it.

A. Okay. So here we have the same picture, but now we have this curve above it. And all it tells you here is the 95 percent goes from .9 to 5-fold.

And then you see these little tails in the end of 2.5 percent. That's the missing -- when you add them up, you get 5 percent. So 95 percent plus 5 percent is 100 percent, right?

So all of your data -- all of your estimates that you expect to be estimating should be under that curve. So if anything is lower than .9, it would be in that lower end of the 2.5, and anything that is above the 5 would be in that upper tail, right?

So all this tells you is -- this is an image of what a 95 percent confidence interval conveys to you. It is that the mass of the data supports that the actual estimate is somewhere between .9 and 5, but most likely

around 50 percent increase.

You can say, I throw all of this data out because that lower tail goes below 1. I teach my students not to do that. Because we can -- we can only wait so long before we make up our mind, and statistical significance testing is actually not state of the art anymore.

We want to look at the data in the way I'm showing you. We want to look at the data in its completeness. And the lower confidence interval below 1 doesn't mean I throw all of this data out; it just means my study was slightly too small to say it's statistically significant.

Q. And applying these epidemiological principles in the real word setting of the Hardell study so that you've got a -- if you go back to page 7, and blow up that same paragraph.

We see that we have a 2.3 odds ratio. That is not statistically significant, right?

A. Correct.

Q. Do we throw that data out or keep that data and continue to study?

A. Since this is one of the early studies, it's a warning flag that I definitely take seriously. Even so, I cannot say anything with statistical significance

about the relationship. But I will put it in context of everything else I know, and other studies.

Q. And do you teach your students about the tyranny of statistical significance?

A. Yes, that is part of my class.

Q. What does that mean?

A. So medical students who don't like statistics very much need tools to make a quick decision. One of the tools is significance testing. However, that comes more out of the philosophy of industrial testing of lightbulbs. And, you know, we can test lightbulbs many, many times, and it doesn't hurt anyone.

When we do these studies in humans, we throw a lot of data out if that's our only tool. So the tyranny of statistical testing is that people say a study like Hardell should not be looked at because it's not statistically significant.

I agree that this is not the evidence to say glyphosate causes NHL. But it is one piece of the puzzle that I need to put in with all the other pieces that I have.

MR. MILLER: Permission to show Exhibit 0121?

MR. EVANS: No objection.

BY MR. MILLER:

Q. Tell us what this --

A. So this is a study published by colleagues in the European Journal of Epidemiology that actually addresses that unknowing tyranny of statistical significance testing in biomedical research. So this shortcut is often used to dismiss studies.

And I teach my students that it's a luxury we really don't have, okay. We should not discard data, because every data point we collect is blood, sweat, and tears of us and our patients.

So if you just look at the -- can I stand up and show it?

THE COURT: Sure.

THE WITNESS: So just look at this. Ignore this. Just look at this side, okay?

So here you have what we looked at before. We have here an incident rate ratio, it's the same as an odds ratio, of 2.0. This now tells you that it's a 2-fold risk for whatever that agent was. And you see the 95 percent confidence interval, it's .9 to 4.2.

So this tells you -- it goes below 1, so it's not statistically significant at the .05 level that they used. But the upper level goes to 4.2. So it could be as much as a 4-fold risk increase.

This data alone, not statistically significant, wide confidence interval. I look at it and

say it's a hint; there could be a 2-fold increase, but I need more data. I don't draw a conclusion yet.

But now let's see what happens when we go to the next graph. Here is my data, my original data. And now I look -- I tell my students, go to the literature, find out what other people did. You're not the only one, probably, who did this, right? And then list all of these results from previous studies, and this is what you get.

So somebody saw a 2.5-fold, somebody saw a 3-fold, and somewhere between 2 and 3, all of these point estimates. But you see that the whiskers are different widths. What these whiskers can weigh is how large the study was or how small the variance.

So this study has a large variance, and it goes below 1. This study has a large variance, but it is all above 1. But what you can see is the pattern; all of the central estimates are above 1.

And if you would take all of these studies together and compare it with this one, this fits very well into your prior knowledge. If you summarize across all of this data, you probably get smaller because now you have a lot more data, smaller confidence intervals, and it will settle somewhere around 2. That's my estimate, right?

So if you don't know this, you say, I don't know; we need more data. If you know this, you can put this study now in the context and actually be much more confident and say, this study shows what previous studies have shown, which is a 2-fold risk increase. And it's completely consistent with previous literature.

I would not dare to do this without prior studies or other information. But in the context of what we already know, we can do this.

BY MR. MILLER:

Q. Stay where you are. I want to ask you, we've heard the phrase "forest plot."

Is this a forest plot?

A. Yes. We would call that a forest plot.

Q. And the vertical axis at 1, anything to the right of 1 there would be evidence of cause of whatever that relationship is?

A. Yes. This is a positive association. Above 1, the exposed have a higher risk of disease than the unexposed.

Q. And anything below 1 would --

A. Would be preventive.

Q. Yes. So, and whatever -- this example, whatever that agent is, for whatever that condition, all of the central estimates are on the right side of 1?

A. Correct.

Q. What's the significance of that in epidemiology?

A. Well, we call it consistent results across studies. I'm sure that these are all very different types of studies and various researchers, various time periods, various methods of assessing the exposure. But they're all showing kind of the same tendency.

Q. And cutting to the chase: At the end of this examination, we're going to talk about one of the most recent studies, Dr. Zhang from here in Berkeley, right?

A. Yes.

Q. And Dr. Zhang's study that came out in February of this year, from Berkeley, contains a forest plot about the association between Roundup and non-Hodgkin's lymphoma?

A. Yes. And it looks about like this, yeah.

Q. And we're going to get a chance to look at that. If we can go back to page 7 now.

I just want to finish some questions about this paragraph, and we'll move on to the next study.

So we know about the odds ratio, and we know it's not significantly significant in this first Hardell study.

These scientists go on to say:

"Since the time period for diagnosis in this study" -- this is 1999 -- "the use of glyphosate has increased dramatically, especially during the 1990s."

Is that your observation?

A. Yes.

Q. There was a dramatic increase in the use of glyphosate?

A. Yes. About mid-1990s.

Q. And to be fair, when they say "glyphosate," no one sprays pure glyphosate, they really mean a formulated product?

A. Yes, correct.

Q. Okay.

"It is now the most common herbicide used in Sweden," which is, of course, where this study was performed.

Correct?

A. Correct.

Q. Quote:

"Gene mutations and chromosome aberrations have been reported in mouse lymphoma cells exposed for glyphosate."

Do you agree with that?

A. Yes.

Q. Last sentence in the paragraph:

"For these reasons, glyphosate deserves further epidemiologic studies."

A. Yes.

Q. Now, at this time, 1999, was the data then available, if Monsanto had chose to do an epidemiological study, for them to have conducted one?

A. Absolutely.

Q. Do you see any evidence that Monsanto conducted their own epidemiological study?

A. Not on NHL, no.

THE COURT: Excuse me.

If you have any electronics or your phone turned on, please turn them off. Thank you.

BY MR. MILLER:

Q. So that was 1999. And then the next study, 2001, two years later, in peer-reviewed literature.

MR. MILLER: Permission to publish 1568?

MR. EVANS: No objection.

BY MR. MILLER:

Q. Now, this is another peer-reviewed study about the relationship between non-Hodgkin's lymphoma, right?

A. Correct.

Q. With Roundup?

A. Uh-huh.

Q. And one, two, three, four, five, six, seven, eight, nine scientists authoring this article, right?

A. Correct.

Q. And Cancer Epidemiology, Biomarkers & Prevention is a peer-reviewed journal?

A. Yes.

Q. Before we get into the nuts and bolts of the study, tell the jury what a dose relationship is.

What does that mean?

A. It's simply what it says. The more dose you have, the more you expect the outcome to increase.

So the more exposure to pesticides, the more cancers we would see. That's what we call a dose-response relationship.

Q. And do these scientists in this peer-reviewed journal show a dose response for exposure to glyphosate?

That is to say, the more glyphosate you're exposed to, you increase the risk?

A. They show a very crude one, but it's an increased response.

Q. And how important is that to your opinions in this case, Doctor?

A. Very important.

Q. Why?

A. Because of the assumption that I would expect

more cancers if the dose is higher.

Q. All right. Why don't you tell these folks how this study was constructed.

A. So this is actually the Canadian study of pesticides. Again, different from the U.S., where we don't have a national cancer registry, the Canadians have long-term cancer registries. And in these -- and medical records.

So they can actually go and pull out the non-Hodgkin's lymphoma from these records, and that's what they did in these years. And then they went back and tried to interview every single case they found.

And again, they also have registries -- other types of registries of their citizens. So they can go to those registries and then find the control subjects, the subjects that never had an NHL, and then they ask exactly the same questions they ask the cases of the controls to see whether there's a difference in exposures.

MR. MILLER: And let's go, if we could, to page 7, please, of this exhibit. Table 8. Blow up Table 8.

And these seven scientists in this peer-reviewed journal -- if we can highlight the glyphosate section. Go all the way across that line, if

you would.

BY MR. MILLER:

Q. Explain to us what we're reading here and what is the significance.

A. Here, we actually have a table where what the cases and controls reported is shown.

And you can see that we have a line called "Unexposed," and then a line more than 0 and less than or equal to 2. So these are people who use glyphosate once or twice a year. Very low dose, right?

And then the next line is more than 2. So these are the people who used glyphosate more than two days per year.

And you can see that we have 23 exposed -- 23 non-Hodgkin's lymphoma cases who said they used glyphosate for more than two days per year. And that's about 4.5 percent among the cases.

And then when we look at the controls -- and I tried to tell you before that we use the controls to see how much exposure they had. Because if they're exposed, and they're controls, they're not sick, and it's exactly the same, then our ratio is 1, right?

But you can see here that among our controls, we had 36 exposed, which relates to 2.4 percent.

And if you want to cheat, you just use the

percent 4.5 divided by 2.4. But that's too simple; we do it a little more careful because we adjust for age and sex and other things. Then you get the odds ratio of 2.12.

So in this study, if you use for more than two days per year, then your risk of having non-Hodgkin's lymphoma is 2.1-fold increase.

And you can see that confidence interval is 1.2 to 3.73, meaning it's on the right side of the 1, so it's statistically significant, right? And it is a wide confidence interval because we only have 23 exposed cases and 36 exposed controls.

But clearly it's showing that if you have more than two days of exposure per year, and you report that in this Canadian study, your risk is more than 2-fold increase.

MR. MILLER: I would like to publish Exhibit 0118.

MR. EVANS: No objection.

MR. MILLER: Your Honor, I have a blowup.

With the Court's permission, I'm going to lean it up here.

THE COURT: Okay.

MR. MILLER: This is a blowup of that exhibit. All right. Here we go.

BY MR. MILLER:

Q. What we've done here is blow up a couple of -- well, a lot of studies you're going to talk about here today. We've already gone by Hardell, and that's why I wanted to put it up now.

Looking at Hardell, we talked about a not statistically significant, but an increased odds ratio.

And it was 2.3, right?

A. Correct.

Q. Which is a doubling of the risk. Okay. So that would be 2.3. Not statistically significant.

A. But I prefer the confidence interval.

Q. Very wide one. Absolutely. I feel like I'm in your class now.

And it's .4 to 13. Okay.

A. Yeah.

Q. Absolutely.

Is that right?

A. Yes.

Q. Okay. Now, we've gone to the second study in our journey here, and that's the McDuffie study. And we were looking at Table 8.

I want to make sure we get it right. For greater than two days use, we have 2.12?

A. Correct. And it's 1.2 to 3.7.

Q. 1.2 to 3.7.

A. Uh-huh.

Q. Statistically significant?

A. Yes.

Q. Doubling the risk. All right.

Other things I wanted to ask you about this study was, it included home and garden users.

Is that correct?

A. Yes. But you can actually see that occasional use for less than two days did not increase the risk.

The occasional users, more than 0 or equal to 2, so that one or two per day, you see no increase in risk.

Q. So the good news is, according to the study, if you use it one day a year, even two days a year, you should not be at increased risk for non-Hodgkin's lymphoma?

A. According to this study, yes.

Q. And according to this study, if you use it greater than two days a year, you're at double the risk of non-Hodgkin's lymphoma?

A. Yes.

Q. Statistically significant.

And if we could look at page 6, there's a note there I wanted to ask you about. Table 7, if you blow

up that note, right there on the right side there.

What does that mean about --

A. So here, they say something about what they call a multivariate model.

So we can just look one by one at every variable we're interested in, whether it causes NHL or not in my study. Or we can put all these variables together in the same model.

And what they've been doing here -- because you run out of numbers very quickly when you throw everything into your model, and then your model doesn't work anymore.

What they did is, they looked at a number of pesticides individually to see whether they made a difference when they put them in the model, and then did not consider those anymore for variables that should be included if they made no difference.

Here, they are saying that these individual pesticides -- carbaryl, lindane, DDT, malathion, and captan fungicides -- were excluded from the multivariate model because they were not contributing to the risk of NHL.

Q. So if they don't contribute to the risk of non-Hodgkin's lymphoma, the criticism that Monsanto levels that they should have been adjusted for, where

does that go?

A. This is a typical beginner's mistake in my classes, as well. That when you say, well, it's always better to try everything and see whether your risk factor survives. That's not how we do -- that's maybe a statistician who doesn't know anything about the field who says, well, okay, let's see what the numbers tell me. The numbers tell you all sorts of things. You need to know something about what you're studying.

What we teach our students is -- and this is state of the science epidemiology. In order for a variable to have to adjust for -- first of all, it has to be a risk factor for the outcome. If this factor is not a risk factor for the outcome, it should not bias my results. Only things that actually cause the disease can be confounders.

So they have -- the first rule is: Is the risk factor responsible for the outcome? If it's not, I don't have to consider it a confounder.

The second question is: Is that risk factor associated with my exposure of interest? If both are correct, then, yes, I have a confounder; I better adjust for it, I have to put it in my model.

However, if I can exclude that something is causing NHL, I don't have to consider it anymore.

Q. Monsanto is going to look at this study and say that it proves that first-degree relatives with cancer have an increased risk of getting non-Hodgkin's lymphoma.

Is that the finding of the study or an incidental observation?

A. At this point, I would say that's an incidental observation because it wasn't what they were investigating. They're just showing all of the associations that they found in this study.

Q. And by doing that, these scientists mixed up hemopoietic cancers with general cancers?

A. Yeah. This is any cancer.

Q. Right. And tell the folks what a hemopoietic cancer is.

A. So that's a blood-related -- a blood system cancer.

Q. Is there any debate in the science that if your relative has hemopoietic cancer, if your relative has a non-Hodgkin's lymphoma or a Hodgkin's lymphoma or melanoma, that you are at increased risk?

A. Yes. You probably would want to check for that, but not for any cancer.

Q. Let's continue our journey. We've just gone from 2001. We go back now to 2002 and Dr. Hardell,

again.

Tell us what he's doing now. He has another scientist join him.

MR. MILLER: If we can move to publish 1575?

MR. EVANS: No objection.

BY MR. MILLER:

Q. So what do we have here?

A. This is the second publication by Hardell and Eriksson, and also somebody by the name Nordstrom.

And this is now a pooled analysis of two Swedish case-control studies. So this is the original study plus a new study.

Q. And I think it's time for us to learn, what is a pooled analysis?

A. That is when we're putting data together from different studies and analyzing them together.

Q. So he's taken his data from his 1999 study, got some new data, and come up with a larger --

A. Sample size.

Q. What do we mean by "power of the study"?

A. This is a statistical concept. It tells you whether or not I expect something to reach statistical significance.

So the more cases I have, the more controls I have; and the more exposures I have, the more powerful

the study is.

If I don't have enough cases, not enough cases are exposed, then I'm having a really hard time making any conclusions.

Q. This is published in May of 2011.

Any indication that after this was published in the peer-reviewed literature, that Monsanto began its own epidemiological studies with these issues?

A. Not that I know. 2002.

Q. I'm sorry, 2002. That's when I downloaded it. Oh, okay. Let's go to page 2.

I want to ask you about the first sentence in the first paragraph:

"Non-Hodgkin's lymphoma is one of the malignant diseases with the most rapidly increasing incidence in the western world."

This is again -- thank you, Counsel, 2002.

What's going on?

A. Yes. This has actually been going on for quite a while, for several decades, that non-Hodgkin's lymphoma was increasing worldwide. Which is unusual, because most cancers haven't been showing that same pattern.

Q. Well, good.

Let's go, if we could, to page 6. And look at

Table 7, please. Now, this has got some answers and some questions for us.

It looks like they're looking at four pesticides -- three pesticides and then a category of other pesticides, correct?

A. Yes.

Q. Glyphosate, our subject of interest, if you can highlight that.

They have a univariate and multivariate analysis?

A. Yeah, they call it univariate. Even so, it's not truly univariate because I think they're adjusting for sex, race, and province. But they only put one pesticide at a time in the model.

So in this case, they would only put glyphosate in the model and ignore the other pesticides.

Q. So we have an odds ratio of 3.04?

A. Correct.

Q. With a statistically significant confidence level?

A. Yes. 1.08. And again, it's over 1, and it goes up to 8.5.

Q. And that would be equivalent to a tripling of the risk?

A. Correct.

Q. And then there's also a multivariate analysis?

A. Correct.

Q. And that's 1.85?

A. Right.

Q. But not statistically significant?

A. Right. Because the lower bound goes below 1, but the upper point is 6.2, so it could be as much as 6-fold.

Q. And the authors tell us, if you go down and highlight the last sentence in that paragraph:

"The results in the multivariate analysis."

Do you see that with me? They say:

"The results of the multivariate analysis must be interpreted" with what, Doctor?

A. With caution.

Q. Do you agree with that?

A. Yes.

Q. So should I put both of those odds ratios on the board, or one? You tell me.

A. Well, we like to see both.

Q. Okay.

A. But when we interpret, we are not just going with one. Because as I told you, if I put another variable into this model, and that other variable is a pesticide, and for some reason, it's a pesticide that

doesn't cause NHL, but every farmer who is now using glyphosate used that pesticide before, then that pesticide becomes the perfect indicator for glyphosate use.

It has nothing to do with NHL, but it indicates that you later use glyphosate.

So what I'm doing by putting them both in the model is something called split the variance.

So each of these, one is a perfect indicator for the other. Both explain half, and that's what's happening in that multivariate model. It's split from one to two.

But we have to decide whether both pesticides put in the same model truly just contribute 80 percent of risk, or one is just a perfect indicator for people having also used glyphosate?

Q. All right. So I'll put down both of them from the Hardell two, the 3.04, and I'll say univariate.

A. Uh-huh.

Q. And a confidence level of 1.08 to 8.52.

A. Right.

Q. And then I'll put down the multivariate analysis 1.85 with a .55 to 6.2. All right.

If we go to page 6 on the study, I want to ask you if you agree with the authors here, bottom left:

"Glyphosate is now mostly used in Sweden. In this study, exposure to glyphosate was a risk factor for non-Hodgkin's lymphoma."

Do you agree with that?

A. Yes.

Q. And this was in 2001?

A. '-2.

Q. I'm having trouble with that, aren't I? All right. Let's go on.

2003, we see another study on the issue.

MR. MILLER: Permission to publish, Your Honor, 1588?

THE COURT: I think it's a good time to take our morning break.

Ladies and gentlemen, we're going to take a 15-minute break, and we're going to resume at 20 of the hour.

Same admonition. Please don't talk about anything you've heard, please don't talk about any of the evidence, and we'll resume in 15 minutes.

(Recess taken at 10:24 a.m.)

(Proceedings resumed at 10:41 a.m.)

(The following proceedings were heard in the presence of the jury:)

THE COURT: You may resume.

MR. MILLER: Thank you, Your Honor.

BY MR. MILLER:

Q. All right. Doctor, did you get a little break?

A. I'm fine.

Q. Before our break, we had talked about how it was important to consider in your overall evaluation, not only animal and cell data, but even not significant epidemiological data.

Do you remember that?

A. Absolutely.

Q. I think I failed to follow up.

How important is it if you do get a statistically significant finding, say, as McDuffie did, of doubling the risk for non-Hodgkin's lymphoma?

A. Well, this is the lazy man's way of looking at data, and I would not suggest it.

Q. Okay.

A. But it makes doctors feel good when they can call something statistically significant.

I really would look at the study, at the possible biases, the size of the effect, more than twofold, and the confidence interval that tells us something about how informative the study was. That it's statistically significant is an added bonus, but

it's not what I would be looking for.

Q. So the end-all, be-all, you would need to use your education, experience and calculate it with everything else that you're looking at?

A. Correct.

Q. Okay. We were looking at the De Roos study before our break. And let's do that again.

MR. MILLER: Permission to publish 1588?

MR. EVANS: No objection.

MR. MILLER: Thank you.

BY MR. MILLER:

Q. This was the De Roos study. That was, I believe, in 2003.

And we have one, two, three, four, five, six, seven scientists, right?

A. Yes.

Q. Including Aaron Blair; we've heard a lot about Dr. Blair?

A. Yes.

Q. And he went on to be the head of the IARC committee that concluded Roundup was a probable human carcinogen.

Are you aware of that?

A. Yes.

Q. And it also includes Dennis Weisenburger?

A. Yes.

Q. He's our witness tomorrow, so we'll talk about this. But since he's one of the authors, we'll talk to him about it, too.

And you understand that you haven't looked at the Pilliods' medical records, right?

A. No.

Q. And Dr. Weisenburger has?

A. Yes.

Q. So we'll save that for him.

Tell us what the De Roos/Weisenburger/Blair study looks at and what its findings are.

A. Right. So at the time this study was published -- and it's, again, what we call a pooled study, so it actually pools data from several other studies.

And these other studies were all initiated by a group of National Cancer Institute investigators, including Dr. Blair, in the 1980s when it occurred to them that non-Hodgkin's lymphoma seemed to be at an increasing trend. But also found more among farmers, and they were starting to get really worried about these occupational exposures in farmers.

So the National Cancer Institute, which Dr. Blair was an internal scientist for, he was paid --

his job was to be studying cancer for the National Cancer Institute, and he was given money to conduct studies. And so they initiated three studies. And the data from these three studies now pooled into this one study.

Each of these studies had different states. One was Kansas, one was Minnesota, Nebraska, and Iowa. And you can already tell why, right? These are farm states. And because they are farm states, they have a lot of farmers, and they have a lot of pesticide use. So that's why they targeted these states.

But they also targeted them because these states already had some kind of a cancer registration system going on. So they started working with the people from the regional cancer registries to pull out all of the NHL cases and do exactly what we heard about our Swedish colleagues doing.

They then pulled all the cases and went to telephone records and tax records to pull out control subjects, people of similar age, sex, et cetera. And then went and interviewed them by phone to get all of the information on their use of pesticides in home, gardening, and farming.

Q. And I forgot to ask, I want to be clear.

This is a peer-reviewed paper?

A. Absolutely, yes.

Q. Published in the scientific journals?

A. Yes.

Q. Let's look now to Table 3 in the De Roos/Weisenburger/Blair paper. And if you blow up that table.

So what they're doing, they're looking at the effect estimates for the use of specific pesticides and non-Hodgkin's lymphoma incidence, and they're adjusting for the use of other pesticides, right?

A. Yes. That's what they do.

Q. So every study we've looked at adjusts. They adjust for age, for sex, some adjust for race. They adjust.

This one adjusts for all those things, and adjusts for pesticides, right?

A. Yes.

Q. And if we look at the whole table now -- we can blow the whole table up.

Before we look at just glyphosate, the point is that there's over 45 various pesticides, herbicides that they looked at, right?

A. Yes. Because these were really the first studies ever to look at pesticides and NHL. This is a 2003 publication, but these other studies pooled here

were published earlier.

And they weren't sure what pesticide to look at, so they asked about 49.

Q. And it looks like -- you can explain -- it looks like out of the 44, only three have a doubling of the risk for non-Hodgkin's lymphoma, right?

A. Yes, double or more.

Q. And one of those is glyphosate, isn't it?

A. Correct.

Q. Let's blow up the glyphosate findings. All right. So we have glyphosate there.

It's a doubling of the risk under the standard logistical regression, right?

A. Yes.

Q. And then there's a new sort of computer program called hierarchal regression?

A. Yes.

Q. And that's 1.6, not quite statistically significant, right?

A. Right. So all of the other studies you looked at would have used something called a logistic regression.

It's a regression model that uses a logit term to predict the probability of the outcome, which here is NHL. And that's what we use when we have a yes/no

outcome. Cancer, yes/no. So that's the usual model we use.

And that model allows us to not only put pesticide, yes/no, amount of pesticide, into the model to predict outcome, but it allows us to put variables such as sex, age, region of the country or state, having a family history of lymphoma, et cetera. You can put all these variables into the model and see how they relate to the outcome, how they predict the outcome.

And an adjusted model would be one where we put a pesticide and then all these other factors that we're worried about that they are biasing: Sex, age, maybe region, and maybe other pesticides.

Q. So all the other studies we looked at so far, they all do logistic regression?

A. Correct.

Q. Would it be fair to say that's the standard model?

A. Absolutely.

Q. Then we have this hierarchal.

But should I write down both of these or one of these on our chart?

A. We want to probably write them both, because I like to look at all data. But I need to explain what that hierarchal means.

Q. So it's 2.1, statistically significant?

A. Right. It's a 1.1 to 4. So, again, that lower confidence interval is above 1, which now tells you it's statistically significant. And the upper one is 4, so our true effect could be anywhere from 1.1 and 4.

But the most likely estimate is the central estimate, the 2.1, so a 2-fold.

Q. A 2-fold risk for glyphosate, only one out of 44 pesticides studied, right?

A. Yes.

Q. And the hierarchal, that's 1.6; about a 60 percent increased risk?

A. Yes.

Q. Not statistically significant, it's .9 to 2.8.

We want to look at all of the data, right?

A. Right.

Q. Explain to us, what is this new hierarchical regression.

A. So this, in 2003, I used hierarchical regression myself because it was a new kind of method that was proposed by my colleague, Dr. Sander Greenland, who is an epidemiologist. He wrote the big book on epidemiology methods.

And he said that we're always looking at one

study by itself, and we're never integrating prior knowledge, meaning what other studies have shown or what we could be maybe teasing out of what we get from other pesticides being related to NHL in this case.

And so hierarchal regression does something called weighting. It's like when I give a mid-term exam and a final exam. I can do different things to come up with a final grade. I can use a 50/50 -- half the points from the mid-term, half the points from the final -- and then your final grade is the weighted average of both exams. And they're weighted in the same way, right, 50/50.

But I can also say, by the mid-term, I only have half the material taught, and the final is a summary final; I ask my students everything, so that should weigh more. And they probably also are a little more strong in the final, so I give that a 60 percent weight, and I give the mid-term only 40 percent.

So they can improve. I want them to be able to improve, and that improvement to be weighted more. So I give a higher weight to the final. So if the student got a B on the mid-term and an A on the final, they have a chance to actually get an A, between a B and an A.

So this hierarchal regression does something

similar. It uses the estimate from the logistic regression, the 2.1 from glyphosate, and says, what else do we know from this data?

Well, we know what other pesticides do, in terms of being related or associated with the outcome of NHL.

If I don't know which of 49 pesticides causes NHL, and every single one has the same likelihood of causing NHL, then all the estimates for all the pesticides should look similar, right?

So I'm using the overall estimate for all pesticides, and we saw that most of them are null -- meaning 1, no effect -- and say, most likely, my glyphosate estimate should look like that of all other pesticides.

And now I do a weighted average. You see what happens? It reduces the estimate of 2 towards the other estimate because I'm weighing them. I'm putting them together.

But that assumes that I am correct. That, really, every single pesticide or most of the pesticides in this model actually cause NHL. And that, truly, glyphosate should just behave like all these other pesticides and I correctly weighted this.

But there's a lot of belief that goes into

that, and a lot of discussion among experts about how I weight this. Do I give it 30 percent? Do I give it 70 percent? 80 percent? How much do I believe glyphosate is the same or not the same?

Q. And in fairness to De Roos and her team, in 2003, we didn't know what we know now?

A. Correct.

Q. So, by way of example, in 2003, we didn't know IARC concluded glyphosate was a probable human carcinogenic?

A. No. That was 2015, so she couldn't know.

Q. Right, of course she couldn't.

But had she known that then, what would have been the weighting difference in the hierarchal?

MR. EVANS: Objection.

Your Honor, objection.

THE COURT: Sustained.

BY MR. MILLER:

Q. Is there a mathematical formula by which any scientist would have weighted it differently?

Let's look at page 3, the footnote to Table 1.

Is that the weighting algorithm for weighting on this hierarchal regression?

A. Yes. That's why she published this table, because she's one of the scientists who wants to be as

transparent as possible. So she wanted to really show what she did when she generated these weighted estimates, so she gives two weights.

Q. Explain to us how this table works.

A. Right. So you actually see all of the different pesticides listed. And on the right side, you see a column called "Carcinogenic Probability."

So she's giving every pesticide a carcinogenic probability. And probabilities are from 0 to 1; 1 is 100 percent; 0 is no percent.

So you can see that Aldrin, for example, gets a 60 percent chance for being a carcinogen. And there's one that gets 100 percent, and some get 80 percent, and some get 30 percent.

We want to know what she gave glyphosate.

Q. Let's scroll down and put it up.

She weighted glyphosate at?

A. She weighted it as low as possible, .3. That's the lowest weight she gave.

Q. Which, in fairness to her --

A. Sorry, it's a .1. But it's in the lower range.

Q. And let's go back to the table. Very bottom.

So you would weight that higher if you knew that IARC declared it a probable human carcinogen per

this table, right?

A. Yes. Because she explains to you what these probability weights refer to. For example, she says .9 -- so 90 percent probability is probable human carcinogen in both assessments, which is EPA and IARC assessment.

And then you can say .8, probable human carcinogen in one assessment and possible human carcinogen in the other. So only had to say it's probable, the other maybe.

And then there's a .6 for probable human carcinogen in one and unclassified in the other.

Q. Knowing what we know now, what rating ought it give for glyphosate in a hierarchal regression?

A. At least .6 but not .3.

Q. Which would do what to this 1.6 number?

A. Increase it. Because you're weighing the actual glyphosate amount, not pulling it down to the others.

Q. Let's go to page 8 of the De Roos glyphosate study. I'm looking at the bottom left.

These scientists say:

"Second, the fact that there were few associations suggests that the positive results we observed are not likely to be due

to a systemic recall bias for pesticide exposures, or selection bias for the subgroup included in the analyses of multiple pesticides."

What are they telling us there, Doctor?

A. So this is what we typically do in our discussions as epidemiologists. We consider all possible biases and give the reader an idea of what we believe a bias could or couldn't be.

And here, Dr. Anneclaire De Roos says, from looking at these results, there's certainly not a systematic recall bias, meaning all these people with NHL just reported having been exposed to every single pesticide.

We asked them for 49. And if they really thought pesticide caused it, they would have just over-reported. And they would have systematically over-reported every one. But she didn't see that happen. So she says, it's unlikely that kind of bias existed here.

And there's selection bias, which is a bias in case-control studies where you're not sure the control group is absolutely adequate because people may have not answered. Cases are more likely to participate in research; controls are a little more reluctant. They

don't have time, et cetera. So you're always worried that there's a slight bias to selection into a study.

For example, you could imagine that controls would say yes to a study that wants to look at pesticides because they were pesticide-exposed, and they're interested in what pesticides do.

You can also imagine that farmers who spray a lot of pesticides are busy and don't want to be bothered.

So in the control group, certain folks would select themselves out with more or less exposure, and that's what we call selection bias. And she says there's no sign here that that happened.

Q. That's a good thing? Meaning the results are what they think they are?

A. Quite solid.

Q. Let's move on.

Is there anything else we should talk about on De Roos?

A. I don't think so.

Q. This is 2003.

Was there any reason that Monsanto could not have done a study in 2003?

A. I don't see a reason.

Q. Okay. Now, let's go to the North American

Pooling Project.

You've heard about that?

A. Yes.

Q. Called the NAPP?

A. Yes.

Q. What is the NAPP, North American Pooling Project?

A. Every time we do these studies, we worry we don't have enough data. And pooling gives you more and more data, which also gives you more and more opportunities to look at the data in different ways.

So in one small study, you cannot distinguish between short-term users and long-term users because you don't have enough people to do that.

However, the more data you pull together, the more chances you have. You can look at short-term users, long-term users, years of use. You can split them up, look at men or women or sub types of cancer.

These things, you can't do when the study is too small. So we like to pool, and then we can look at the data in that way. And the NAPP study pooled everything that's in the De Roos study, so those North American studies, plus the Canadian study.

Q. And again, we have one of the authors of the NAPP study here tomorrow, Dr. Weisenburger.

But we still want to talk to you about it a little bit, if it helped inform your opinion.

A. Yes, it did.

Q. All right. So in this larger study, the NAPP study, it's been presented three times at professional meetings?

A. Correct.

Q. Does it have to be peer-reviewed at some level to be allowed to be presented at a professional meeting?

A. Absolutely.

Q. And -- all right.

So you reviewed the three presentations from the three different professional meetings it's been held at?

A. Yes.

Q. We have all three here today. If defense counsel wants to talk to you about all of them, fine. I want to talk to you about the one that explains this best.

A. Right.

MR. MILLER: Permission to publish 2082?

MR. EVANS: No objection.

BY MR. MILLER:

Q. Now, this is the NAPP study?

A. Yes. That's what's called the NAPP study.

Q. And this was presented at a professional conference in Ontario, it looks like, in June of 2015?

A. Yes.

Q. And it's a detailed evaluation of glyphosate use and the risk of non-Hodgkin's lymphoma?

A. Correct. Go to page 2, if you would.

Q. NHL is a cancer that starts in the lymphocytes, right?

A. Uh-huh. Right.

Q. We all agree that's where it starts?

A. Yes.

Q. "Heterogeneous," what does that mean?

A. That means it's varied, various different types.

Q. Glyphosate, a broad-spectrum herbicide --

A. Yes.

Q. -- commonly known as Roundup.

And by the time this study was done, the most frequently used herbicide in the world?

A. Correct.

Q. And there's estimates for glyphosate use in 2012.

Do you see that?

A. Yes.

Q. It looks like the Central Valley of California

uses its fair share?

A. Yes.

Q. And the corn and soybean belt in the middle of the country?

A. Yes. And if you know geography, you can probably see Iowa and Nebraska, where the NAPP study was done. And they're all brown, meaning there's no place where it's not used.

Q. And we go on to the next page.

And this tells these doctors -- if someone says that people aren't telling other doctors about this, that's not accurate, is it?

A. No.

Q. So this is at a medical --

A. Professional --

Q. -- seminar of some sort, where these professionals, these scientists who wrote this study are sharing with as many doctors who will attend that seminar, that Roundup is a possible carcinogenic, pursuant to the IARC evaluation, right?

A. Yes. That's what this says.

Q. And the next page, please.

These are the states where -- and the provinces where they pulled the data from?

A. Correct. So we see four states in the U.S.,

in the Midwest, and then the Canadian provinces.

Q. In an effort to save time, I won't go through every page. Let's go to page 12.

What is this?

A. So this is a table where we have the non-Hodgkin's lymphoma subtypes, and an overall estimate for all of them together.

And we see how many cases reported using glyphosate -- any glyphosate use, 113. And we now see an odds ratio reported of 1.22, with a confidence interval of .91 to 1.63. So it's not statistically significant, but it shows a 22 percent risk increase for all non-Hodgkin's together.

Q. And that's ever versus never use?

A. Yes. Ever or never. You could have used it for a half a day or an hour, you're included.

Q. And then go to page 14:

"Frequency. Days per year of glyphosate handling and the risk of non-Hodgkin's lymphoma."

Explain to us the significance of these scientific findings.

A. Here, you see exactly what I tried to say, why we want to pool data. Now you can split the data in many little boxes and still have information in the box.

If you do that with not enough data, then you have zeros everywhere. Here, we can estimate because we have a really large study.

So what we estimate here is, when you use glyphosate for a number of days per year, one or two, then you can see there's not much risk increase. All these odds ratios are .8, .5, .77, 1.4, 1.38 --

Q. Let me stop you, Doctor.

MR. MILLER: With the Court's permission, may the doctor be allowed to go up to the board again? I think it might be easier.

THE COURT: Yes.

THE WITNESS: What you're seeing here is that it wiggles around the 1, right? I would not pay any great attention to these estimates because they're from very low use, less than -- two or less days per year.

And overall, they -- some are on one side of the 1, and some are on the other side, and all of the confidence intervals are including 1, meaning they're not statistically significant, right?

That's fine, but we don't want to look at that alone. We actually want to look at what happens when you use glyphosate more than two days per year. So not two days, but two days per year.

And you can see that overall -- that's all

non-Hodgkin's together -- we now see a 1.98; we can round it to 2. It's a 2-fold risk increase. Our confidence interval here is 1.16 to 3.4. So, clearly, statistically significant; clearly above the 1, this lower value.

And now we have the luxury to actually look at subtypes, right? We have follicular lymphoma, large B-cell, small lymphocytic leukemia, and then the others.

BY MR. MILLER:

Q. Well, Doctor, we're particularly interested in diffuse large B-cell lymphomas.

What are the findings?

A. First of all, we see they're all above 1. But some -- again, the confidence intervals include the 1, we are not really sure.

But the one that really sticks out here is the 2.49, so a two-and-a-half-fold risk increase with confidence intervals of 1.23 to 5, clearly statistically significant, right?

So for those diffuse large B-cell lymphomas, we see a two-and-a-half-fold risk increase if you use glyphosate for two or more days per year.

Q. So if someone told this jury that there was not a statistically significant finding by peer-reviewed scientists that diffuse large B-cell has a doubling

risk, would that be accurate?

A. No. We see it here.

Q. Which numbers should I write down on my board?

A. It depends on whether you want to go the overall, that's the most comparable. But if you're interested in B-cell, you want to put that one, as well.

Q. I'll put both.

A. Okay. 1.98; confidence interval, 1.16 to 3.4.

And then large B-cell, 2.49; confidence interval, 1.23 to 5.04.

Q. Does this help inform your opinion that Roundup causes non-Hodgkin's lymphoma?

A. Yes. This is more data, so we're putting it together and looking at it from different angles.

Q. Anything else we need to talk about regarding NAPP before we move on to other case-control studies that informed your opinions that glyphosate causes non-Hodgkin's lymphoma?

A. I think that's it.

Q. Let's go to Eriksson.

MR. MILLER: Court's permission, Exhibit 1703?

MR. EVANS: No objection.

THE COURT: Okay.

BY MR. MILLER:

Q. I want to ask one more question about NAPP.

In NAPP, they showed, obviously, dose exposure, right?

A. Yes. That's what -- we have three levels. We have unexposed, fairly low exposure, occasional exposure, and then higher exposure.

Q. So now we've had several studies where you've seen dose-response.

How significant is that to you as a scientist?

A. That's very important.

Q. Why so?

A. Because we are always presuming that higher exposure should be causing more cancers.

Q. And although they don't do two days a year, four days a year, 20 days a year, should we assume that the more you're exposed, the more your risk is?

A. Yes.

Q. Let's go to Eriksson, 1703, yet another study on the risk of non-Hodgkin's lymphoma from exposure to pesticides, right?

A. Correct. Another Swedish study.

Q. And these scientists, Dr. Eriksson, Dr. Hardell, Dr. Carlberg, and Dr. Akerman?

A. Correct.

Q. Published in a peer-reviewed journal, International Journal of Cancer, 2008?

A. Yes.

Q. Does this help inform your opinion?

A. Yes.

Q. Tell us about this study. What were they looking at? What's your finding?

A. This is actually a study where the cases occurred later in time. They occurred between 1999 and 2002. The cases we looked at before all occurred in the early '80s and the late '80s and the early '90s. So this is really a different period for the cases.

But they all are in Sweden, and it's very similar to the studies we looked at in Sweden before, but it's a different time period for the cases.

Q. And did they find dose-response in this study?

A. Yes, they did.

Q. Let's look at some of their findings. If you can please go to page 3, Table 2.

All right. Now, here we have exposure to various herbicides, one of them is glyphosate, right?

A. Correct.

Q. And if you are exposed less than ten days over a lifetime, what are their findings? And what are their findings for greater than ten days?

A. First of all, if you're just never, ever exposed, the finding is 2.2, and the confidence interval

is 1.1 to 3.7.

But then they were actually able to look at more or less than ten days per year and split this data, more or less, into groups. And you can see that for the people who used glyphosate, but less -- up to ten days per year -- you see a 70 percent increase. But that confidence interval does include the 1, so it's not statistically significant.

Q. But for greater than ten days?

A. You now see that -- I see what we call dose-response. It's now 2.36, or 2.4 if you want to round.

So you're going from a 70 percent increase to a 2.4-fold risk increase, and you also see it's statistically significant. It's 1.04 to 5.37, so clearly statistically significant at higher levels of exposure. And a dose response pattern, which I like.

Q. Sure, sure. That's what we're going to get to in a minute.

One of the Bradford Hill criteria, correct?

A. Correct.

Q. Which of those numbers should I write down here for the Eriksson study?

A. I would write down 1.67 -- well, we want to write all three down, I think. Then it makes it more

comparable.

2.02 for the overall, with a 1.1 to 3.7 confidence interval.

Q. Got it.

A. And then for less than/equals ten days, 1.69, with a confidence interval of .7 to 4.07.

And then for more than ten days, 2.36, and a confidence interval of 1.04 to 5.37.

Q. All right. Got it.

A. And you can see here, the 2.20, it's 12 plus 17 equals 29 exposed cases. You have a statistical significance because you have 29 over 18, cases and controls.

When you split that up, you're increasing the variance. You have smaller groups, less number of people exposed, and therefore you need -- you have less data, so your confidence intervals widen.

You see how that happens? You get the .724 and the 1.04 to 5, which is much wider than the 1.1 to 3.7. So you're adding in your understanding if you see a dose response.

Q. And fortunately, this study had enough people in it where you could actually see whether ten days or greater increased the risk?

A. Right. And what's different from less than

ten days.

Q. And you saw that dose or exposure response?

A. Right.

Q. Let's go to page 6, the last page of this. I want to look at some of the things the scientists had to say.

That first sentence, these scientists report that:

"Glyphosate was associated with a statistically significant odds ratio for lymphoma in our study. And the result was strengthened by a tendency to dose-response effect, as shown in Table 2."

So these scientists agree with you that there is a dose response?

A. Yes.

Q. Last sentence before acknowledgments:

"Furthermore, our earlier indication of an association between glyphosate and non-Hodgkin's lymphoma has been credibly strengthened."

Do you agree with that?

A. Yes. Because they have a lot more cases to look at that were exposed, and they were able to split it into a seeming dose response.

Q. And this is in 2008?

A. Yes.

Q. Okay. Now, I want to talk about a study we didn't put on our chart, but in fairness, talk about it for a minute.

MR. MILLER: It's 1899. Permission to publish?

MR. EVANS: No objection.

BY MR. MILLER:

Q. This is the Cocco study. And there's lots of scientists involved in this.

Tell us about the Cocco study, please.

A. That's a lymphoma study out of a consortium called the Epilymph. But it's not a -- so it's pooling data from lots of European studies -- six European countries, in fact.

However, these studies were not focused on farming communities or farmers. So most of these cases would have actually lived in urban areas.

And you can see what happens. Very few people here are glyphosate-exposed, because most of the cases come from urban areas.

Q. Let's look, if we can, at Table 4, which is on page 4, the bottom left there.

It's a very small study?

A. Correct.

Q. What does it find in regards to the risk of a B-cell lymphoma and glyphosate?

A. Here, we really have few exposed cases and controls; four cases exposed and two controls exposed, but B-cell lymphoma cases.

And you can see the odds ratio here is 3.1. But since we have so few exposed, and it's mostly urban cases, we have really, again, very wide confidence intervals. Our whiskers around those points are very broad. So we have a .6 to 17. Clearly includes 1, not statistically significant. But certainly an odds ratio of 3.

Q. So what do you take away from that?

We're not going to put it on our board because you don't want us to, right?

A. No.

Q. Is there anything we learn from this?

A. Well, it's one extra small piece in the puzzle confirming what we have seen before.

Q. Let's move on to 1746.

MR. MILLER: Permission to publish?

MR. EVANS: No objection.

BY MR. MILLER:

Q. Tell us about the Orsi study. This is a

hospital-based study, right?

A. Yes. We actually distinguish case-control studies that were based on cancer registries, where we find every single case from hospital-based studies.

So here, we go to a hospital, and everybody who comes to that hospital and is a case of non-Hodgkin's lymphoma gets enrolled in a study.

These studies have problems because -- before, I told you, how do we get controls? We go to tax records, we go to citizen registries, to insurance registries, we call people randomly by the phone, right?

And we know, since we have every case of lymphoma, anybody else who lives in that community is fine to be a control. When we go to a hospital, we don't really know who the cases are who end up in this hospital and this hospital alone.

And then we don't know -- so what are the real controls for these cases? And there's a lot of debate in my research area about what the right way is to actually sample controls.

And the easy way is to just use other patients. So other people who came for some other disease to the same hospital, but they don't have lymphoma.

So that is one problem. If these other

diseases were also related to pesticide use, then you're underestimating the effect of pesticide on the cases. Because you're comparing one sort of cases to another sort of cases.

So there's a lot of debate about studies based just on hospital patients, if they're the correct thing.

Q. If we can look at the conclusion of the Orsi study real quick, it says they do not rule out a relationship of non-Hodgkin's lymphoma.

Do you see that?

A. Yes.

Q. And that's what you -- how do you feel about that? You just talked about hospital-based studies.

A. Right. So they want to be careful and say they're not sure if, actually, their control section was adequate to really generate an unbiased estimate for pesticides.

Q. Okay. So the previous study we looked at, Cocco, had a tripling of the risk; but you had problems, we didn't want to include it, right?

A. Right. That was also hospital-based.

Q. This study has no increased risk, but it's a hospital-based study; you don't want to include it?

A. Correct.

Q. Before we get to the large studies that have

come out recently, one in February and one while we were actually picking this jury, we want to talk about those.

But before we do, we want to talk about the Agricultural Health Study that you oversaw and that Monsanto relies upon in this case.

You're familiar with it, obviously?

A. Obviously, yes.

Q. You read it and considered it in your opinions, but you don't give it much weight.

Why not?

A. No. The Agricultural Health Study is a very valiant effort to estimate pesticide cancer risk from pesticides. But from every pesticide that farmers in Iowa and North Carolina used.

And glyphosate is very special in that lineup; they have more than 50 that they assessed. Because different from every pesticide they used, glyphosate use changed rapidly in the middle of their first questionnaire, their baseline assessment in their cohort.

And the exposure assessment -- the very first exposure assessment they did was really just a questionnaire that farmers who came to get their licensing exams to be a licensed pesticide applicator in the state of Iowa or North Carolina filled out on

20-some pesticides.

There are 21 pages. These pages were put in front of them, bubble them in. And they were asked about behaviors, age, family history, and then 21 pesticides. And for every pesticide, they had to report -- on the spot -- how much they have used, and in what decade throughout their lifetime. And they probably used about half an hour to do that, and reported every pesticide they used.

Some may have thought it was a part of the exam. Other people were just interested in the research, different reasons, they bubbled in. And that way, they got about 56,000 farmers that came to these licensing exams to bubble in these 21-page questionnaires. And among those questions was glyphosate.

So one of the pesticides they were asked about was glyphosate. And what they also asked -- they asked them to report, have you ever used? And if yes, in what decade? How many days on average per decade, and how many years? When did you start and when did you stop using this pesticide?

And they did that between 1993 and 1997, because it took them five years to get 56,000 people to answer these questionnaires.

Well, you have now some people who answered on glyphosate in 1993, '-4, and '-5; about 30,000, that was the first batch. And then you have another 20,000 or 26,000 who answered in '95, '-6 and '-7, right? And there was a huge change in glyphosate use right in the middle of that period.

So you get some people who report in 1993 what they used, lifetime, and some in 1997. Guess what? Those in 1997 report what they changed. Those in 1993, you have the baseline in 1993, you absolutely don't know what they did in 1997.

Q. I want to ask you about that a little more.

So in 1993 -- and I've used my high-tech graphics here. Let's call this guy Farmer Tom, okay?

Farmer Tom is going to go in and fill out the agriculture health form, right?

A. Yes.

Q. And he has to answer yes or no for Roundup use, right?

A. Yes.

Q. So in 1993, he says no, he hasn't used Roundup?

A. Correct.

Q. In 1994, he joins the ever-growing crowd of people that are using Roundup, just like there's an

ever-growing crowd of people using cell phones, right?

A. Correct.

Q. He uses glyphosate in 1994, 1995, 1996, and then this fellow comes down with non-Hodgkin's lymphoma, diagnosed in 2002.

Does he go down in a "I used glyphosate" or a "I didn't use glyphosate" category?

A. In the no use category.

Q. So he would have used glyphosate for three years, gotten non-Hodgkin's lymphoma, and in their study, they're calling him a non-user, right?

A. Unless he reported in the second round.

Q. How many people failed to report in the second round?

A. 38 percent.

Q. Which is about 18,000 people?

A. Yes.

Q. You have a fancier name for this problem than I do. It's called non-differential exposure misclassification.

A. Yes.

Q. What does that mean?

A. That means we are making the same mistake in assigning exposures, whether or not this person later develops non-Hodgkin's lymphoma. That's the

non-differential part.

So we are making a lot of mistakes in assigning exposures, but they're not mistakes we're making only for the cases or the controls. We're making it for both.

Q. The other thing we need to point out is that this is not something you've said recently.

You teach your medical students the problem with exposure?

A. Misclassification, yes.

Q. Using that as an example, don't you?

A. Correct. Because I teach biases. I teach about confounding, selection bias, and disease misclassification.

And from exposure misclassification, that's a great example.

MR. MILLER: Permission to publish 1209?

MR. EVANS: No objection.

BY MR. MILLER:

Q. All right. What is this, Doctor?

A. This is one of my slide decks, six in a page, from fall 2012 in my master's class.

Q. "Slide deck," meaning something you show and use when you lecture medical students to teach them how to become --

A. Public health students.

Q. And let's go, if we can, to page 5 of that.

A. Some of them are medical students, too.

Q. Bottom left, you talk about the disadvantages of the cohort method?

A. Correct.

Q. To be clear, the Agricultural Health Study is a cohort study?

A. Yes. It's a cohort study because we're starting with individuals who are undiseased. So all of these farmers should not have had a cancer when they enrolled and told you about the pesticides.

Actually, some of them had had cancers, but in the analysis, then they are excluded. We are not using those who had cancer at baseline.

So we are starting in a cohort with people who have no cancer, no disease of interest, no non-Hodgkin's lymphoma. They may have had another cancer, but not NHL.

And then we ask them, what was your exposure? So they report all the exposures they've had, and then we watch them passively through cancer registries.

That's why, actually, the Ag Health Study is quite brilliant; they did it in Iowa and North Carolina farmers, first of all. But also, they have cancer

registries.

So we don't have to really find these people. And that's why this was funded, because they could passively follow them over time. The cancer registry would pick up every farmer who developed a cancer and every farmer who developed NHL over time.

Very elegant, right? You don't rely on people coming back to you; you just use the cancer registry. But, yeah -- yeah.

Q. Go ahead.

A. And that's why epidemiologists love this. Because first of all, we know that at baseline, people report their exposure, and then we can follow them. They can't drop out because we find them in the cancer registry.

The only way to drop out is to move to California, and then we find them in the California registry, right? So that is a really elegant method.

The problem is what I'm discussing here with my students. We have a large number of people we're following, and we need to have a large number because cancer is a rare disease, NHL is a rare disease. We need 56,000 farmers and follow them over many years to have enough cases occur.

Very different from a case-control study,

where I start with the cases, right? I already have NHL. I assemble 500 of those people. It took them almost 20 years to get 500 cases.

So you have to be committed, in a cohort study, to follow these people over a very long time, and to do it right.

So we have large numbers of people. We need to follow them. It's relatively expensive. This study would not have been funded to someone like me because the NIH only funds you for five years, and then you really have to scramble to get the next five years. It can only be done within the NIH, where money is easier to come by, and you can maintain the follow-up.

So it's really expensive. You need a long duration of follow-up. So you have to have the money, you have to have the sample size. You have to watch them for a long time.

And the next one is the real disadvantage of this type of study if all you want to do is find cases from the cancer registry, because what you're ignoring is exposures that change. You have absolutely no problem with exposures that have already happened at baseline. And they're fixed at baseline, right?

However, if now exposures change over time, you better ask them again. And that's what, actually,

these authors realized. Five years later, they had another round approaching these people to ask, oh, by the way, what changed in the last five years? Did you use different kinds of pesticides?

Because if you're committed to follow them for 20 years, you better know what's happening in those 20 years. Because pesticide use may rapidly change. Some do, others don't. Glyphosate use changed a lot.

Q. And let's cut to the chase.

They lost track of 17,000 people?

A. In the first round, and another 17,000 in the second round.

Q. So they had to scientifically guess -- or I think we called it multiple imputation -- about what these people might or might not have done?

A. Yes.

Q. And only Farmer Tom knows if he actually used Roundup after 1993?

A. Right. If he never reported again, that's all we know.

Q. Let's go back up to the page.

You used the Agricultural Health Study as an example of these problems to these studies in 2012?

A. Exactly. Right. This is my introduction of what it is. This just says who funds it and what it is.

It's farmers who have pesticide exposures.

Q. I want to go back, if I could, to the first one. I jumped a little too quick.

The disadvantages of the cohort method.

The bottom bullet point.

"The cohort is generally not representative of the general population."

That's also true here, isn't it?

A. Absolutely. When we do these beautiful cohort studies, we're getting a group of people who are willing to participate not only once, but willing to participate over a long period.

And these people are generally different than the general population. Because in the general population, you have all these people who say not me, right? And even among the farmers who were asked at the pesticide licensing exam, there were some who refused.

So we could presume that not all farmers wanted to actually participate. Certainly not all farmers wanted to. They did it when they were in person at the licensing exam. For example, they also were given a take-home with more questions on more pesticides. Fourteen thousand already did not send in that take-home. You know that those 14,000 didn't really want to be in the study, they just didn't want to

say no the first time.

So cohorts are special. They're people who want to be studied and want to remain in the study. They're never the general population.

Q. And licensed pesticide applicators know to wear Tyvek suits or boots or gloves, masks?

A. Yes.

Q. It's what you learn to become --

MR. EVANS: Objection, your Honor. Speculation.

THE COURT: Sustained.

MR. MILLER: I'll rephrase.

BY MR. MILLER:

Q. What do licensed pesticide applicators learn about --

A. Well, that's why they come for the licensing --

MR. EVANS: Same objection, Your Honor.

THE COURT: Sustained.

BY MR. MILLER:

Q. All right. They were there to take a licensed pesticide applicator exam?

A. Correct.

Q. So they're learning to become professional pesticide applicators?

A. Correct.

MR. MILLER: With the Court's permission, we would like to publish Exhibit 120 and Exhibit 119.

MR. EVANS: No objection, Your Honor.

THE COURT: Granted.

BY MR. MILLER:

Q. Exhibit 120 tells us the amount of -- I'll let you explain.

A. This is actually downloaded from an EPA website, where they are showing you how much glyphosate is being used in one year.

In this case, they're using the highest possible amount. That's called EPest-high. In that year, 1993, you can see that the different shading gives you the pounds per square mile that are applied in these different states.

Q. That's the first year you could fill out the forms?

A. Right. And I think Iowa, if I'm correct, is right here, and North Carolina over there. So we can see there are still pockets of Iowa where no glyphosate is used in 1993, and there are also high use and low use areas. And definitely for North Carolina, it looks very sprinkled.

Q. Let's look at Exhibit 119, 2013 use.

A. We can't see any difference in application in Iowa, and it's much darker in North Carolina. So we clearly say glyphosate use increased enormously.

Basically, we have nobody unexposed anymore in Iowa. We couldn't even do a study in Iowa, because everybody would be exposed.

MR. EVANS: Objection, Your Honor.

THE COURT: Overruled.

BY MR. MILLER:

Q. What -- that amazing expansion of the use of glyphosate, is that what you were talking about, the problem with exposure misclassification?

A. Yes. What we have here is the period 1993 to 2013. That's exactly the period that's covered by the Agricultural Health Study. And you can see here how use changed. And you are asked to relate pesticide exposure that was assessed in 1993 through 1997 and it's effect through 2013.

There was one additional questionnaire in 1999 through 2004, where they tried to find people again, but 63 percent or 62 percent were found and reported. But they also were only asked to report one year.

Q. Wait a minute.

So the second reporting, the same problem again, Farmer Tom says what he does in that one year.

But if he used glyphosate for four years before he filled out that questionnaire, he still gets reported as a nonuser?

A. Correct. What they actually asked is, the last year of farming, what did you use?

So if that farmer used in 2001, but -- did not use glyphosate in 2001, but used it in '96, '97, '98, '99, 2000, he would say, no, in 2001, I didn't use.

So he says it once at baseline because he doesn't use yet. He uses for five to eight years. He doesn't use anymore -- maybe because he's starting to feel sick or he retires or whatever -- and he only reports on the last year that he farmed. And that's the data they have.

Q. That's the second AHS study, called Andreotti?

A. Yes.

Q. So now we have the exposure misclassification problem in the first AHS study, done by Dr. Blair and Dr. De Roos.

And now Dr. Andreotti does the second AHS study, and he's got 37 percent loss to follow-up, and he still has the exposure misclassification for the ones he can get ahold of?

A. Yes.

Q. You have, I think, a very good demonstrative.

MR. MILLER: Permission to publish 0123 about this increased use and its effect?

MR. EVANS: No objection, Your Honor.

BY MR. MILLER:

Q. Explain to us what you're telling us here.

A. This is just put in an image what I already explained to you. Saying that, you know, depending on when Farmer Ted or Farmer Tom was actually enrolled at his pesticide licensing exam and asked, what is your lifetime use of glyphosate, you call him a user or nonuser or user of X amount. Because they asked about, how many years and how many days per year did you use?

So if you say, in 1994, I never used or I -- you know, I sprayed a little bit here and there, I sprayed maybe three days a year in one decade, then you're locked into the low use or no use category.

But if that same farmer then decides, I'm jumping on the bandwagon and spraying glyphosate at a much higher rate because my neighbors do it and this is now the hottest herbicide in town that really is helpful for my crop production, then it changes that. In 1996, we wouldn't know that from the baseline.

And we wouldn't know that for 30,000 people who reported prior to 1996. He's one of them, right?

If he comes back and is asked, so in the last

five years, the last year you farmed, what was your farming? What did you use? Then it's kind of luck whether that last year actually reflects what happened in the meantime or not, right?

He could have started to use glyphosate, still use it, and reported. Okay. We're okay.

But he could have started and stopped and reports, no, I stopped. We keep him in the low exposure, even though he might have exposed himself for five years to large amounts. We don't know.

That happens to lots of people in this study. Especially the ones we don't know anything about, the 38 percent who never came back. And all we have is the baseline, the first time they are questioned, and we are using that to guess what the exposure is over the next 20 years.

And that's similar to what would happen if you would ask people to report their iPhone use. Clearly, before 2007, there weren't any iPhones, so you would say cell phone use, right? Pesticide use, cell phone use.

So you get people reporting, yeah, I use cell phones. But iPhones weren't on the market yet. So you wouldn't be knowing whether it was an iPhone or something else, right?

So if you accrue people between 2007 and 2010

when iPhones came on the market, some may be early adopters and report, I already have my iPhone, right? But others take two or three years, and you get the answer in 2007, no, I'm not an iPhone user.

But you don't follow them again or you're not having a follow-up, so you're categorizing the one person as a user and the other person who adopted a year later as a nonuser. And then you follow them to see if whether the iPhone gives them any health hazards, makes them distracted and get in a car crash.

But you have completely misclassified users because you asked at one time, when all the use was changing rapidly. And one person, by chance, was asked before, and one was asked after.

Q. I used to be a house painter before I was a lawyer. Pretty good one. I like to use analogies of paint cans.

If you have nonusers as a can of white paint and users as a can of red paint, you have to keep those paints separate in order to get the right colors on the wall.

What does exposure misclassification do to my white and red paint?

A. Ideally, you want to know whether somebody is a user or nonuser; a user is red, a nonuser is white.

If you misclassify, it's like dipping a spoon in red and putting it into white, and dipping a spoon into white and putting it in red.

If you do this often enough, what do you get? Two pink cans, no difference.

Q. This problem about the exposure misclassification and winding up with a lot of pink paint, it was discussed before the results came out from the AHS study, right?

A. Absolutely.

MR. MILLER: With the Court's permission, we have four articles summarized on Exhibit 0122 that we would like to publish at this time.

MR. EVANS: No objection, Your Honor.

MR. MILLER: We can put that up. Let's go one at a time.

These are one, two, three, four, five studies, which comment before -- some before -- this problem of exposure misclassification.

BY MR. MILLER:

Q. Let's look at Gray first. That was in the year 2000, the top one.

What is this saying? You reviewed this?

A. Yes.

Q. Does it help support your opinion that, in

fact, that's a real problem in the AHS study?

A. Yes. They actually refer to this strange non-differential exposure misclassification that we described; exposure misclassification, white and red mixed. And it's non-differential, meaning we're doing it for cases and controls.

Q. And it says:

"Non-differential exposure misclassification will produce bias towards the null."

What does that mean?

A. This is what everyone who learns epidemiology learns. The more you are mixing the paints, the less you can distinguish the white from the red. Because in the end, they're pink.

You can also say it's a signal-to-noise ratio. If you have a signal, and the noise is really high, you don't see the signal, right? You have to have a quite strong signal for the noise not to cover it. So what non-differential exposure misclassification does is hide the signal.

Or when you look at my points and the whiskers, it draws the points to 1.

Q. That was in 2000, before the AHS results were reported?

A. Right.

Q. Let's look at the next scientist, that's Acquavella.

Dr. Acquavella. You know him, right?

A. Yes.

Q. Who was he employed by?

A. He was employed by Monsanto at this time.

Q. At the time he wrote this, he was employed by Monsanto, right?

A. Yes.

Q. So what is he telling us here?

A. In 2006, he is as concerned about exposure misclassification as I just described, and said that there is possible substantial exposure misclassification in the study.

Q. This is before the results came out?

A. Yes.

Q. So before the results came out, Monsanto criticized and was concerned about the AHS results?

A. Yes.

Q. Let's look at the third one.

This is Weichenthal, "A Review of Pesticide Exposure and Cancer Incidence in the Agricultural Health Study Cohort."

A. This is a nice review paper in the Environmental Health Perspectives, which is that very

famous journal by the National Institute of Environmental Health Sciences.

And this is a Canadian colleague who looked at all of the published results from the AHS study. And in his conclusion, concluded that exposure misclassification undoubtedly has an impact on AHS findings reported to date.

Q. Do you agree with that?

A. Yes.

Q. Let's look at Blair.

This is interesting, because Dr. Blair was one of the original authors of the original AHS study and also the chairman of the IARC committee?

A. Yes.

Q. And he writes on the subject, as well.

What does he tell us?

A. He specifically is concerned about pesticide exposure misclassification for the same reason I always am. Because everybody thinks we are just generating wrong results, meaning results that indicate risk. The dirty little truth is that, most of the time, we see nothing.

Because when we do a bad job, what we do is, we see nothing. We actually drown the signal in the noise. It's not as intuitive as saying, you guys got it

all wrong, so you must have produced these enormous effects.

No. Most of the time, we don't see anything. And then we have to say, there is nothing. When public health makes us scratch our heads or public health concern makes us scratch our heads and say, maybe I did a lousy job in exposure assessment.

So he was as concerned as I was about pesticide misclassification and what it does to these dots and whiskers. And that's what he says here.

Q. Here, we have the actual author of the AHS -- one of the authors -- telling us there's a false negative. Findings could be common because of the problems with the AHS.

A. Correct.

Q. And he went on to vote with IARC to find that Roundup is a probable human carcinogenic?

A. As far as I know. He actually goes further and says the true relative risk -- so the true increase in risk -- could be threefold, and you see absolutely nothing.

Q. What's a false negative?

A. False negative means you see nothing negative, but there is truly something.

Q. And the last one, and we're going to talk

about that in more detail in a while, but that's Sheppard. At the bottom.

Dr. Sheppard talks about this, as well, doesn't she?

A. Yes. She's a statistician, and she was also concerned about biases in the Agricultural Health Study. And she reviewed what happened there and came to the following conclusion: Due to all of the nonresponse over time --

Q. That's the 37 percent that never bothered to fill out the questionnaire?

A. Right. And the way they then guessed these people's exposure, they might have meaningfully attenuated the cancer risk estimates.

Q. What does that mean?

A. Attenuation means there's less than there should be, and meaningful means there's quite a bit. So, again, that dot is drawn towards nothing.

Q. This scientist who is published in this says:

"We probably meaningfully attenuated, or made too low, the risk."

A. Correct.

Q. So that's how you process the case-control and then the critiques for the AHS, right?

A. Yes.

Q. Have I missed anything, or have we covered it?

A. No, we covered it.

Q. Okay. Let's go then to Exhibit 2333.

THE COURT: Are we changing to a new topic, new study?

MR. MILLER: Whenever Your Honor wants to take lunch, I'm never a man that says no to lunch.

THE COURT: If you're going to start something new, maybe we should go ahead and break for lunch.

MR. MILLER: It's two more case-control studies and then some Bradford Hill. I have about half-hour to 45 minutes left.

THE COURT: Why don't we take a break now for lunch. We'll come back at 1:00.

MR. MILLER: Sure.

(Luncheon recess was taken at 11:55 a.m.)

AFTERNOON SESSION 1:03 p.m.

(Proceedings resumed in open court in the presence of the jury.)

THE COURT: Ready to continue.

MR. MILLER: Thank you, Your Honor.

Q. Doctor, good afternoon.

A. Good afternoon.

Q. Did you have a good lunch?

A. Yes.

Q. Good. I hope everybody did.

I wanted to just wrap up a couple things. I think we can finish the direct exam in about a half an hour. So that's my goal.

I forgot to ask you. This NAPP study that we were talking about, the one that Anneclaire De Roos and Dr. Weisenburger and Dr. Blair, that shows the doubling of the risk statistically significant, that was adjusted for the pesticides?

A. Yes, they adjusted for the pesticides they thought they should be adjusting for because other studies previously had indicated there could be a risk, but IARC hadn't made the same classification for them as for glyphosate.

Q. Okay. And we talked about the Cocco study that had triple the risk but it was so small and the confidence level was so wide, they don't put it on there.

A. Right.

Q. We did put the Orsi study on that you had criticisms of but wanted to put it on here for completeness. It was one; right?

A. Yeah, it was basically one.

Q. Now, we talked, I think at length, about the AHS studies which are a De Roos '05 and Andreotti 2018.

A. Right.

Q. That's I guess AHS 1 and AHS 2; correct?

A. Correct.

Q. And you talked about the problems with the studies?

A. Yes.

Q. Yeah, but I wanted to put the odds ratios down from them even though you articulated the problems with them. Okay?

A. Yes.

Q. And looking at Exhibit 1629, I think they had an odds ratio of 1.2.

On page 3, Table 2.

A. Yes.

Q. Okay. Not statistically significant?

A. Right.

Q. The numbers again?

A. And it's .7 to 1.9.

Q. .7 to 1.9.

And on the Andreotti or AHS 2 where they lost 17,000 people, did they actually conclude that glyphosate prevented non-Hodgkin's lymphoma?

A. No, they didn't.

Q. What was the odds ratio that they used?

A. They did not give us one for the overall, from

what I recall, but they gave us five -- where is it?

Q. It's Exhibit 2230, is the Andreotti study.

A. Yes. And there's a Table 2 continued on page 513, and they are giving us categories of exposure and they start with none. So no exposure at all, which is the comparison group. And then they --

MR. MILLER: We can put that on the screen, if there's no objection, 2230.

MR. EVANS: No objection.

BY MR. MILLER:

Q. Table 2.

A. So this is B-cell, but we can also look at non-Hodgkin's lymphoma overall. But B-cell is fine as well. So what we can see here is that compared to the reference group, all of these estimates are below 1. Right?

And, yes, the confidence interval does include the 1. So none of these are statistically significant. But there is a pattern of all four exposure categories. Quarter 1, 2, 3, 4 means low, medium, higher, highest exposed according to their intensity of exposure.

You see that all of them are below 1 for non-Hodgkin's lymphoma, and they're even further below 1 for B-cell lymphoma.

So if we believe these, then we would say

there's a 24 percent decrease in non-Hodgkin's at that second quartile. The .76. It's 1 minus .76. So it's a 24 percent decrease, lower risk in non-Hodgkin's if you are exposed at that level 2.

If you're exposed at the level 4, your risk is about 14 percent lower than if you're not exposed. So that is really against our expectations. We would expect that maybe it's 1, meaning there's no difference. But why would glyphosate use prevent non-Hodgkin's lymphoma?

Also what makes us a little bit suspicious is that all of these are consistently on that side. If it was just random fluctuation, then one estimate would be below the 1 and another above the 1 and you would have -- you would just see, oh, this is random. You know, there's random noise. Some estimates are slightly below 1, some are slightly above 1, but on average they are null.

This study suggests that there is actually a benefit from having glyphosate exposure. I don't think I would like to believe that. And what the next step is when you -- when something is so against your expectations, you're worried about bias, a systematic bias that introduces that flipping of estimates to the other side.

Q. And that's nondifferential --

A. No. This couldn't be nondifferential misclassification. This is a systematic bias in another direction than expected.

Q. Okay.

A. So there's something else going wrong here. So it could be that all of my comparisons against those we call not exposed are wrong, that the people we're putting in the not exposed group are really not not exposed. Right?

Q. We've talked about the white paint and the pink paint and the red paint.

A. Right. Exactly.

Q. We got pink paint here?

A. Yes.

Q. Now, moving on.

The Zhang study came out in February of 2019; right?

A. Uh-huh.

Q. And you reviewed it?

A. Yes, I did.

Q. And does it help inform and confirm the opinions that you already held in this case?

A. Yes.

MR. MILLER: If we could, with the Court's

permission, publish 2333.

MR. EVANS: No objection.

(Exhibit published.)

MR. MILLER: All right. If we'd blow up the title, please.

Q. All right. Now this was released I guess about a month before trial started, and it's entitled "Exposure to Glyphosate-based Herbicides and the Risk for Non-Hodgkin's Lymphoma: A meta-analysis and supporting evidence."

What's a meta-analysis?

A. So a meta-analysis pretty much brings together all of the studies that were done up to that point in time, uses their summary estimates, and then generates a weighted average of all of them.

Q. And it says "and supporting evidence." Did Dr. Zhang, Dr. Rana, Dr. Shaffer, Dr. Taioli, and Dr. Sheppard, did they all use, then, those three pillars of evidence when they analyzed this issue of glyphosate and the risk for non-Hodgkin's lymphoma?

A. Yeah. This is actually a very interdisciplinary group of authors. I believe Dr. Zhang is a toxicologist. So she knows best about animal studies and cell studies.

So this group of people came together to write

not only about the epidemiologic human data, but put that human data into the context of the animal and the mechanistic knowledge we have about glyphosate and NHL.

Q. This is a peer-reviewed study?

A. Yes.

Q. Okay. Let's turn to page 34 of the document, please, of this study and look at the declaration of interest, if we could pull that out.

All authors have no financial conflicts of interest to declare. We disclose Dr. Zhang, Dr. Taioli, and Dr. Sheppard served as a science review board members for the United States Environmental Protection Agency Scientific Advisory Board.

A. Yes.

Q. Are you aware of that?

A. Yes.

Q. And that was the meeting that was held in December 2016?

A. Yes.

Q. And we've heard about that here.

So these three scientists served on that board. And then independent of that after being asked by the EPA to look at this issue, went out and published this 30 -- 55-page analysis in the peer-review literature; is that right?

A. Correct.

Q. Let's look if we could now, please, at page 3. I'd like to go to that last two sentences in the first paragraph, if we could.

I just want to cut to the chase here. "We documented," start there.

Here's what these three scientists who had been tapped by the EPA to look at this issue:

> We documented further support from studies of malignant lymphoma incidence in mice treated with pure glyphosate.

Right?

A. Uh-huh.

Q. You observed that as well, haven't you?

A. Yes, I saw those.

Q. (Reading from document:)

> As well as the potential links between glyphosate-based herbicide exposure and immunosuppression, endocrine disruption, and genetic alterations that are commonly associated with non-Hodgkin's lymphoma.

A. Correct.

Q. It's what the animal data tells us, doesn't it?

A. Yes, that's what they're putting here.

Q. "Overall in accordance with evidence from experimental animal and mechanistic studies, our current meta-analysis of human epidemiological studies suggests the," what, Doctor?

A. The compelling link between exposures to GBHs and increased risk for non-Hodgkin's lymphoma.

Q. Do you agree with these three scientists who had been tapped to be on the Environmental Protection Agency Science Advisory Panel that in fact as we stand here today in 2019, there is a compelling link between exposure to Roundup and an increased risk for non-Hodgkin's lymphoma?

A. Yes, that's the conclusion I came to myself.

Q. And what they did, why don't you explain to the folks how they did the study.

A. So they went back to the literature, just like we did this morning. And they pulled out all these estimates. And you can see them in a table lining up. And then they do exactly this weighing approach where they weigh according to the size of the study, the number of people who were exposed. So if there's a small study, it gets very little weight. If there's a big study, it gets a lot of weight.

But they also selected according to the best

exposure assessment or the best estimate from each study that they trusted the most, and they said that would be the estimate with the highest exposure.

So they did not want to combine an ever/never which some of the previous studies had done. They just looked at one estimate from every study which was ever/never. So somebody could have had half a day of glyphosate use, it was ever.

They really went for the best exposure assessment that they could identify, the best estimate from each of the studies. For each study, they used one of these estimates and then combined across and generated that summary estimate with a confidence interval that tells us 95 percent confidence interval, how much if I repeated this 100 times, 95 percent of the time, my estimate would fall into those -- into those brackets, right, into those whiskers.

Q. And to be clear, what they did is they took the data from the case-control studies that we've been looking at.

A. Right.

Q. And they mixed it in a scientific way with the data from the Agricultural Health Study; right?

A. Yes, they also used date from the Ag Health.

Q. And even with the criticisms about the AHS

data, when they did that, when they mixed the case-control studies with the Agricultural Health Study, did they get a statistically increased risk of getting non-Hodgkin's lymphoma if you're exposed to Roundup?

A. Yes, they did.

Q. Let's go to page 5, if we could, of the Zhang report. I want to ask you about the last sentence in the second full paragraph.

It says:

> Given that more than 6 billion-kilograms of Roundup have been applied in the world in the last decade, glyphosate may be considered ubiquitous in our environment.

MR. EVANS: Your Honor, objection.

THE COURT: Sustained.

MR. EVANS: Move to strike.

MR. MILLER: I'll withdraw.

MR. EVANS: Move to strike.

THE COURT: It will be stricken.

MR. MILLER: Okay. I'll move on.

Q. Let's go to page 28, go to the first full paragraph. All right, see where we are?

This is what Dr. Zhang from Berkeley and her colleagues report in this peer-reviewed article:

Together all of the meta-analysis conducted to date, including our own, consistently report the same key finding: Exposure to glyphosate-based herbicides are associated with an increased risk of non-Hodgkin's lymphoma.

A. Yes.

Q. And is that consistent with your opinion?

A. Yes, and it's consistent with the previous meta-analyses by other authors.

Q. If you would please go to page 34. All right. Top paragraph, and looking at the second full sentence. This is in their conclusion.

Using our high-exposure *a priori* hypothesis -- now, what is an *a priori* hypothesis?

A. *A priori* means even before you do any analyses, you're actually stating what your hypothesis is. That's why it's *a priori*. Before you do your analysis, you say, "Well, you know, I presume that the highest exposure causes the most cancer." And that's pretty much the *a priori* hypothesis they use.

Q. More than two days, more than ten days --

A. Ten days. Right.

Q. (Reading from document:)

Using our high exposure *a priori*

hypothesis and including a recently updated AHS cohort in a meta-analysis for the first time, we report that glyphosate-based herbicide exposure is associated with an increased risk of non-Hodgkin's lymphoma.

And that's what they did; right?

A. Right.

Q. They mixed the AHS with --

A. Uh-huh.

Q. They go on to say:

The totality --

Down in the middle of the paragraph:

The totality of evidence from the six studies on glyphosate-exposed mice support this association in humans.

A. Correct.

Q. These are the three scientists who had been previously tapped to be on the EPA Scientific Advisory Board?

A. Yeah. So they bring the second pillar of science, the animal studies, into the evidence here for their conclusion.

MR. MILLER: Exhibit 0105.

MR. EVANS: No objection.

(Exhibit published.)

BY MR. MILLER:

Q. We looked earlier in our testimony today at forest plots in the abstract. Do you remember that?

A. Yes.

Q. This forest plot has been pulled from Dr. Zhang's published meta-analysis.

A. Uh-huh.

MR. MILLER: With the Court's permission, could I have the Doctor come down and explain to us what these dots mean?

THE COURT: Actually, if we have a pointer I think it might be better.

Do we have a pointer?

MR. WISNER: Yes.

MR. MILLER: Great.

THE COURT: Actually you might want to pull it a little closer.

MR. MILLER: I think we can put it up actually on the board then. Exhibit 0105 and the Doctor could talk about it from up there.

THE COURT: I just suggest using a pointer so we're not standing in front of the material as she's talking about it.

MR. MILLER: Understand. Sure.

Q. This is a forest plot, Doctor?

A. Yes.

Q. And it's from -- I'll give you this, I have no idea how to use this.

A. Oh, boy.

MR. WISNER: The green dot on the top.

THE WITNESS: On the top, yeah.

THE COURT: It's fine to stand over there and point if you want. But I think going there would be a little disruptive. If you were to stand on the other side and point with or without the pointer, that's fine.

BY MR. MILLER:

Q. Is this a forest plot?

A. Yes.

Q. Okay. And is this from the Zhang article?

A. Yes. It's just turned around, I guess.

Q. Okay. So tell us what that blue line means.

A. So the blue line, we are at 1. That's our ratio measure. If the rate of cancer in the exposed is the same as in the unexposed, we get a ratio measure of 1. Same number. Right? Dividing two numbers with each other that are the same, we get 1.

So this is where there is no effect.

Q. Okay. So every dot to the right of that blue line indicates an association?

A. Correct. So all of these dots indicate an increase. And this is on a log scale so it's a little bit wider to the 2 and then it decreases to the 10. That is because the confidence intervals otherwise because we're going only from zero to 1 would be unequal, they wouldn't be the same lengths and they should be. That's the only technical thing.

Otherwise you can see here that's 2. So most of these hover around 2.

Q. Okay. So I'm not an epidemiologist. I'm looking at this, I see a lot of dots on the right, one dot on the blue line, one dot to the left. These are all about the issue of Roundup and non-Hodgkin's lymphoma?

A. Yes. All of these are measures of association for Roundup and non-Hodgkin's lymphoma, and they come from the different studies.

And this one was that hospital-based study in France that had very few exposed people. Remember most of these people were from urban areas.

Q. Orsi.

A. Yeah, Orsi. And you can also see that these whiskers are really wide. So we have very little information in that study, but the little bit of information we have says there's no association.

Q. Okay. So what we want to ask is what are the odds of all of those dots being on the right and this association being by chance with this multiple studies the vast majority on the right?

A. Well, we like to look at patterns. And when we see a pattern like this across a lot of different studies from different continents, from Canada, from the U.S., from Sweden, then we start thinking, hmm, there mights be something here because look at this, the pattern is pretty clear except for one type of study, and that's the one in red, that's our cohort study.

In fact, we wouldn't be in a real forest plot that we use for a meta-analysis, we're not supposed to use all of these because they're coming from the same data. We have to make up our mind which one to use.

But this was just to illustrate that, yes, the Agricultural Health Study has not very wide whiskers because it has a lot of cases, it has a lot of exposed cases. And these dots are very close to the 1. This dot is on the other side, and it's the only dot that's on the other side of the equation.

Q. Now, does this forest plot, showing the vast majority of those dots on the right, does it support your opinion that Roundup causes non-Hodgkin's lymphoma?

A. Absolutely.

Q. Thank you very much. You can have a seat.

Now the jury has been very patient with me.

A. Oh, by the way. These are all the meta-analyses. So all of these people have actually used this data except out of these, they only used one estimate, and then they came up with these estimates. And you can see how powerful the meta-analysis is because all of these lower confidence intervals are now above 1 meaning it's statistically significant.

So no matter who put this data together in whatever way they wanted, they always came up with a low is 27 percent increase, high is 45 percent risk increase for an ever/never, or in case of Zhang, high exposures to glyphosate and NHL risk.

Q. I almost forgot to ask you. 2016 Monsanto did fund a study. They funded the Chang and Delzell; correct?

A. Yes.

Q. And that study funded by Monsanto showed a statistically increased risk --

A. Yes.

Q. -- of non-Hodgkin's lymphoma if you're exposed to Roundup?

A. Yes. We see that because even this confidence interval is close to 1.

Q. 40 years after the product was on the market?

A. Yes.

Q. Now literally while I'm over introducing myself to these folks in the big building across the street, the last article we're going to talk about came out, the Leon study came out recently last couple weeks. February it was accepted. We found about it about then.

Okay. Tell us what on earth is the Leon study? And we'll ask permission to publish it.

A. Yes. So this is really the latest study and it brings in new data. It is another, they call it a pooled analysis, but it's really again one of these meta-analyses because for each study they're creating one estimate and then they're combining them.

But this time they're combining the Agricultural Health Study. So we know about that one. Then they're combining that with a study in France of more than 140,000 farmers and a study in Norway with about 140,000 farmers as well.

Q. Okay. So I want to stop you there.

AHS was about 50,000.

A. Yes.

Q. And they lost the 17,000 and went down to about 33,000.

A. Correct.

Q. You're now telling me that that data, for whatever that data was worth, is combined with a French study?

A. Yes.

Q. And a Norwegian study. How big was the French study?

A. About 140,000, I think.

Q. How big was the Norwegian study?

A. Also. But the Norwegian study has more weight because they had a longer follow-up time. The French study only had five years of follow-up. So we calculate the number of people times the year of follow-up. That's what we call person-time. So if it's 100,000 people followed for five years, we have 500,000 years of follow-up time. If it is 100,000 people followed for 20 years, we have 2 million years of follow-up time.

So the Norwegian study had about two-and-a-half million years of follow-up time. The French study only about 400,000. So the Norwegian study is what weighs the most.

Q. And this is not an abstract thing for you, you're intimately familiar with the Norwegian database --

A. Yes.

Q. -- and the French database?

A. Yes. Because the -- yeah. The reason is these are groups who are doing pesticide exposure assessment in farmers. That's something I do. So I know people who work with this data. I know papers because I review them. I probably have reviewed that crop exposure matrix they used.

MR. MILLER: Permission to publish 2984, Your Honor.

MR. EVANS: No objection.

(Exhibit published.)

BY MR. MILLER:

Q. So this is the study. It was in the *International Journal of Epidemiology* recently; right?

A. Yes.

Q. And I think you have been or are currently an editor?

A. No, not of *International*, no. But it's a very well-known journal.

Q. Very well. And it's about pesticide use and the risk of non-Hodgkin's lymphoma combining agriculture cohorts from France, Norway, and the United States.

A. Correct.

Q. Okay. And it's not a dose response study, it's an ever versus never study.

A. Correct.

Q. What's the significance of that?

A. So in this case, we have a combination of the U.S. data where they actually tried in the Agricultural Health Study to come up with an intensity. And I showed you those five estimates, 1 and then .8, .8, .8, .8. Right? They could do that because they asked the farmers how many years they used and then created this intensity measure.

What I didn't tell you yet about the Ag Health Study is how they created intensity by combining the years with how these farmers applied pesticides.

So in the baseline questionnaire of the Agricultural Health Study, they asked one question: How are you applying pesticides? And they could report with a hand sprayer, with a rig on a tractor, whatever they had.

And then they also asked: Have you used pesticide -- have you used protective equipment such as face masks or whatever to cover yourself? But they only ask that once.

They had a list of 21 pesticides they answered to, and then one question saying what did you -- how did you apply, what did you do to cover yourself?

And then that information was presumed to be valid for every pesticide they reported on. So if a

farmer, for example, said they used glyphosate and they used an OP, an organic phosphate pesticide, which can be acutely toxic, and said, "Yeah, I use a respirator and I spray from an enclosed cab on a tractor," then they would presume they do that for the glyphosate as well as the highly toxic OP. And we don't know that that's the case.

So the Ag Health Study created this intensity measure with data that they didn't really know that it applied to glyphosate.

Q. And because they had so many numbers, so many people in three different areas, they were able to look not only at non-Hodgkin's lymphoma but diffuse large B-cell?

A. Yes. So this study. The combined study.

Q. Yes.

A. Yes.

Q. In the Leon study?

A. Yes.

Q. Can we please turn to page 7.

A. But what I haven't told you yet --

Q. Please go ahead.

A. -- there was a completely different exposure assessment in the French study and the Norwegian study, and that's important to understand.

So the French study is a study of individuals who are all insureds through a French system of farmers insurance. The 140,000 individuals are all in that farmers insurance. They were pulled from that listing and then were asked about their pesticide use lifelong.

Half of the people they -- not pesticide use. Sorry. About what they farmed, what crops they farmed, what animals they farmed, how long they had farmed, and whether they had ever used pesticides but not which pesticide.

So 140,000 people reporting that, but they were already half of them were retired. On average they were 67 years old when they got to them.

So these French farmers reported that. And then the researchers went back to records about all the different crops in France and what was applied in terms of pesticides on these crops in what years that these farmers reported having farmed these crops.

That's called a crop exposure matrix. So you're not asking people what they are applying, you're asking them what they're farming.

Then you're making the best guess you can which is, oh, if you had this crop in this year and you said you used pesticides, you probably used A, B, and C pesticide. But there's no guarantee that they actually

did.

And they also didn't know how much they used and how long they used because it was presumed that by farming the crop, reporting you used pesticide, you did it. And not everybody did.

Q. Okay. Let's turn to page 7. And I appreciate that explanation.

So, and let's look at diffuse large B-cell. That's where our interest lies.

The cohort specific hazard ratios for every use of glyphosate and diffuse large B-cell were --

A. The overall.

Q. -- overall were 1.6. And the AGRICAN?

A. No, that's CNAP --

Q. Huh?

A. That's the Norwegian study.

Q. The Norwegian study --

A. The overall is above it.

Q. I'm sorry. Excuse me.

There was an elevated -- what's MHR?

A. That's the meta hazard ratio.

Q. Okay.

A. Again, it's just a ratio, same as an odds ratio. And meta because we're combining three studies.

Q. With every use of glyphosate, 1.36

statistically significant; right?

A. Yes. Confidence interval 1 to 1.85.

Q. Okay. So with -- although they did not find it for overall non-Hodgkin's lymphoma in this very large study, they found it for diffuse large B-cell?

A. They did.

Q. Right? And when they looked at the separate databases, the AGRICAN database showed a 1.67 statistically significant for diffuse large B-cell; right?

A. No. No, no, no. The 1.06 in AGRICAN, that's the French one.

Q. Okay.

A. And a 1.67 in CNAP, that's the Norwegian --

Q. Ah, thank you.

A. -- one.

Q. That's why you're the expert and I'm not. Okay. All right.

A. And then interestingly, in the Agricultural Health Study, they're finding a 1.20.

Q. So they go back and reanalyze the Agricultural Health Study for diffuse large B-cell --

A. Right.

Q. -- and see an increased risk but not statistically significant.

A. Correct.

Q. All right. Does this large study lend support to your proposition that it certainly with diffuse large B-cell, there's an increased risk of non-Hodgkin's lymphoma?

A. Yes.

Q. From Roundup?

A. Yes.

Q. All right. This came out during the start of trial. There's been no studies since then, has there?

Okay. All right.

A. We don't know.

Q. So we're kind of wrapping this up. We've talked now about the studies that have informed your opinion?

A. Yes.

Q. Okay.

A. So all of the studies we showed are informing my opinion, including the Agricultural Health Study, but especially now this new study that just came out that used yet another very different way of assessing glyphosate use and came to the same conclusion, and pretty much almost exactly the same estimate as we saw in the meta-analyses of all the previous studies, and statistically significant.

Q. And we talked about a Bradford-Hill, I think he was a knight in England, Sir Bradford-Hill. Why is he so famous? And why do we use him to assess --

A. So he came up with terms, criteria, guidelines for assessing scientific evidence. And the guidelines he came up with, a lot of people have tried to improve over the last 50 years. But nobody has. And it really allows science to be evaluated across scientific silos and put them together.

Q. Scientific silos?

A. Yes.

Q. All right. And have you done a Bradford-Hill analysis in this case?

A. I did.

Q. Let's take a minute to look through that, and then I'm done.

All right. Consistency of association. What's that mean?

A. That means that these studies that I looked at were consistent in showing an association. And the ones that weren't consistent, I can -- I can kind of understand why that is the case.

So for the Agricultural Health Study, I kind of guess why that is the case because of the huge exposure misclassification.

So generally, including the latest study that mostly was driven by the Norwegian results, I would say there's strong consistency across the case-control studies and then also the latest pooled analysis that includes Norway.

Q. Strength of association, what's that? And how did you rate it in this case?

A. So that's how high that odds ratio is. And we saw odds ratios that were 1.3 and we saw odds ratios of 2 and of 3. But whenever we are looking at a higher dose, the odds ratios seem to move above 2.

So strengths of association, we usually say we're comfortable if we see something higher than 2. I actually, in environmental epidemiology, I'm very happy with a 1.2. And that is because the more common exposures are, the less able you're actually to see very strong effects.

You are able to see a 20 percent, a 50 percent. It's a statistical issue. It's really complex why that is the case. But definitely here we are seeing 2.3-folds when we're going to the higher levels. That's strengths of association. But if we say ever/never, then it's moderate.

Q. Moderate for ever/never?

A. Because it's around 1.5.

Q. Moderate for ever/never, and then for longer use it's what?

A. It's strong.

Q. Is it greater than 10 days?

A. Yeah.

Q. All right. Tell us about biological plausibility.

A. So that is when we're starting to actually look at our colleagues' results in animals and in mechanistic studies of cells, lymphocytes in human beings. So if we see that glyphosate also has an effect in that mechanistic sense by generating genotoxicity, oxidative stress, endocrine disruption, and that makes sense with the cancer that we're looking at in humans, we say there is biologic plausibility.

So we're moving from the experimental mechanistic side to the humans. And, yes, there is absolutely biologic plausibility here.

Q. Yes.

A. Yes.

Q. All right. What is gradient?

A. That's our dose response. So we saw it.

Q. Okay. How should I characterize it?

A. Yes, there is a gradient.

Q. Gradient or dose response.

A. Right.

Q. Meaning more exposure, more risk?

A. Right.

Q. Temporality?

A. Absolutely because these farmers were exposed prior to their occurrence of NHL.

Q. So, yes?

A. Yes.

Q. Specificity?

A. By the way, that's the only criterion, temporality, that is absolutely necessary to assess causality. If we know that something came after the fact, it's not causal; right? But with everything else, we have more leeway. But temporality is established and has to be established.

Q. Okay. What is specificity?

A. That means that it's not just causing every disease and every cancer, that it's causing a specific cancer and that there's biologic plausibility for that. So it's given since we're looking at NHL.

Q. So, yes?

A. Yes. And we saw in other papers that glyphosate was not linked to other types of cancer.

Q. What's coherence?

A. Coherence is kind of everything taken together

and do I see anything that kind of rubs me wrong and is questionable in the evidence from humans, from animals, and mechanistically. So does a coherent picture emerge or not? It does.

Q. It does.

Last question. You've been very patient, as has everyone.

Does Roundup cause non-Hodgkin's lymphoma in real world exposures?

A. Yes, it does.

Q. Does the more you're exposed increase your risk in non-Hodgkin's lymphoma?

A. Yes.

MR. MILLER: Thanks, everyone. I'm done.

THE COURT: Thank you.

Do you need a minute to change the technology, or are you ready to go?

MR. EVANS: I think we can get started, and then we'll probably take a break whenever Your Honor wants, but I can probably get started without changing much.

THE COURT: Great.

We'll have cross-examination by defense counsel, Mr. Evans.

///

CROSS-EXAMINATION

BY MR. EVANS:

Q. Good afternoon, Dr. Ritz. My name is Kelly Evans. Good to talk with you.

MR. EVANS: Good afternoon, everyone, ladies and gentlemen of the jury, and Your Honor.

Q. I guess I just wanted to start by just making sure I have some understanding definitionally of a couple of things you talked about. And I wanted to just start --

MR. EVANS: If I can have the ELMO, please.

Q. This was your example; correct?

A. Yes, this graph, yes.

Q. Okay. And just trying to make sure I understand. If we are talking about an absolute risk, let's just pick a random number, say 10 in a million, okay, that's the actual absolute risk. Does that make sense?

A. Yes.

Q. Okay. And so just to make sure I understand what the -- this is, you said, an odds ratio or a confidence interval; correct?

A. That's an odds ratio.

Q. Odds ratio or relative risk.

A. Yes.

Q. So if you have the actual risk of 1 -- or 10 in a million, that's the background or baseline risk. Do you understand that?

A. I'm not sure that I understand that, no.

Q. Okay. Do you -- there's such a thing that's called a baseline risk; is that fair?

A. It's usually the risk in the unexposed, yes.

Q. Okay. Risk in the unexposed.

A. Yes.

Q. And in this example, let's assume whatever we're studying the risk is 10 in a million.

A. Okay.

Q. All right? Now, if you then look at exposed group --

A. Yes.

Q. -- with a relative risk or odds ratio of 1.5, the number would be -- go from 10 to 15?

A. Yeah, 15 in a million.

Q. Okay. And regardless of what that baseline risk is, that's what the relative risk of the odds ratio results in. So if you had, for example, 10 in a thousand, you would just go from 10 in a thousand to 15 in a thousand.

A. Correct.

Q. Is that right?

A. Yes.

Q. Okay. And if you're looking then at a group of individuals who all were exposed to whatever you're studying, and in this case there are 15 of them who have the disease or condition you're looking at, you would say with that 15 that you have exposure and disease, that 10 of them were the background, that's what you would expect with unexposed, and then the five of them you would say that's the additional that you've got from the exposure; correct?

A. That's what we usually call the excess risk due to exposure.

Q. Okay. Okay. But that 10 that's in the background group doesn't go away, you still have to account for that 10 that originally exists; correct?

A. That's why we have a reference group, yes.

Q. Okay. All right. Thank you.

Now, you --

MR. EVANS: Can I just have my boards -- where are my big boards at? Over here?

Q. So, Dr. Ritz, I'm just showing here a printout of the definition of limited evidence of carcinogenicity from the IARC preamble. Are you familiar with that?

A. Yes.

Q. Okay. And just so I --

MR. EVANS: Let me scoot by here, sorry.

Just so I make sure -- can you see okay?

Q. The definition by IARC with respect to limited evidence of carcinogenicity says:

> A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the working group to be credible but chance, bias, and confounding could not be ruled out with reasonable confidence.

Did I read that correctly?

A. That's correct.

Q. Okay. Now I want to just talk for a minute about those three: Chance, bias, and confounding.

A. Yes.

Q. Okay? And chance we talked about today. I think you talked about it with respect to statistical significant; is --

A. Yes, that's what chance is. So by chance, would I, if I repeat this 100 times, find this estimate or something that is beyond the whiskers on either side.

Q. Right. And IARC is using that 95 percent confidence interval --

A. Yes.

Q. -- that most all these studies are using; correct?

A. Correct.

Q. Now. Bias, we talked about a little bit I think with Dr. Jameson and maybe with Dr. Portier. But could you tell the ladies and gentlemen of the jury what bias means.

A. Yes, actually that's my class I teach, ten weeks, six hours a week.

Q. Well, why don't we not take 10 weeks --

A. So it could take a while.

Q. Okay.

A. But basically it is confounding. Confounding is one bias, selection bias, and exposure and disease misclassification bias. So those are the three big ones in epidemiology that we need to consider.

Q. And where is recall bias? What is that?

A. That's a selection bias.

Q. Okay. And I think Dr. Jameson mentioned something about if, for example, you're studying --

A. Oh, sorry. I misspoke. It's an exposure misclassification.

Q. Okay.

A. Yeah.

Q. Dr. Jameson said something about if you're

studying individuals who have NHL and you ask them whether they've used Roundup before, the fact that you're asking that question may result in someone having an incorrect recall of what they actually were exposed to. Is that a fair example?

A. We have always worried in studies where we're starting with case status, that people who have the disease are reporting in the same way as the controls, and that's what we would call a recall bias if they are systemically reporting differently.

But so far that has never been shown to be the case in these farmer studies.

Q. And then confounding, again, I know you may have a class that goes on for 10 weeks, but what is the -- what's an example of confounding? What does that mean in this context?

A. So confounding bias is a bias where I'm confounding two factors' effect with each other. So I'm thinking it's one pesticide, but it's another pesticide.

So the first thing I need to know is: Do I know about any pesticides that are causing NHL? If I know one, then I need to be concerned because if that pesticide really truly causes NHL and my pesticide that I'm interested in is also applied whenever the other pesticide is applied, then I don't know which one of the

two is actually causing the effect. So it's a mixing of the effects of two different factors.

But for something to be a confounder, the first question always is: Is it a risk factor for the outcome? If it's not, it's not a confounder.

Q. All right. And the confounding issue here -- and again, I think Dr. Jameson and Dr. Portier talked about that there are other pesticides that they believe are confounding, confounding with respect to the risk of NHL. Do you agree with that?

A. No. Because according to IARC, I don't think there is a pesticide that we are as concerned about in terms of carcinogenicity. But in order to be careful, yes, if there is even some probable cause for thinking that a pesticide could cause it, then, yes, I would want to put it in the model and see whether it changes the effect or not.

Q. And do you think controlling for confounding is an important part of epidemiology?

A. It is important, but not as important as getting the exposure assessment right. And that has often been misstated in the literature on -- and misunderstood by individuals.

Q. But if you have confounding, that's a separate issue from statistical significance. Agree?

A. Yes.

Q. Okay. And so before you really look at whether something is occurring by chance or is statistically significant, it's important to control for confounding if you have a reason to control for it.

A. Yeah. So one could say chance or statistically significant testing which tests for chance occurrence, that's a random error. So how much is there random error in my data. While bias is systematic. Bias is what draws systematically my estimate towards the null, across the null, or sometimes away from the null.

Q. And confounding, in a lot of the studies we looked at or at least some of the studies, they looked at trying to look at other specific pesticides and controlling for that; correct?

A. Yes.

Q. And some did not; correct?

A. Well, yes and no. Some studies put all sorts of pesticides in without considering that first element of: Do all of these pesticides really cause NHL? And I would say they generated confounding rather than corrected for confounding. Because you can actually generate confounding by putting something into a model that is not a confounder, that's not a risk factor for

the outcome.

So you have to be very careful when you're saying I'm adjusting for a confounder because if it doesn't follow the rule of truly being a risk factor for the outcome, you generate confounding.

Q. And a good example of that I think you've talked about before is the De Roos 2003 study where it controlled logistical regression for all 47 different pesticides; right?

A. That one did not -- the 2.1, is that what you're talking about?

Q. Correct.

A. That controlled for all pesticides, correct. But it was not -- it wasn't -- what are you saying? They didn't compare it to a crude.

Q. Right. I'm just saying I believe you said before that that wouldn't be the way you would do that study.

A. I wouldn't do that, yes.

Q. Right. Because you want to look -- you said before you want to actually use your brain to figure out which ones potentially or likely will be actually confounding as opposed to just doing them all; right?

A. That is the preferential treatment. But you can actually -- there's a trick. You can actually

generate the crude estimate. You can even do it -- I could even do it here with a calculator from the numbers that De Roos gave and then compare the adjusted -- fully adjusted one to the crude, and you would see it's very close. And when you know it's very close, then there's no confounding.

Q. All right. Now I want to step back. I want to just get some of those definitional issues regarding the IARC statement and then making sure I understood what a relative risk is and what the background rate is.

Just a couple baseline things.

You're not here to talk specifically about Mr. and Mrs. Pilliod; correct?

A. No.

Q. You haven't looked at their medical records, you don't have specific opinions about their case; correct?

A. No.

Q. Okay. So I said "correct?" I think that's "yes."

A. Yes.

Q. Okay.

Now I want to go back and start by looking just a little bit at your CV. And I have a couple of questions about that. This is the exhibit that

Mr. Miller showed you earlier.

MR. EVANS: If I could have the ELMO back.

Q. And this is what I believe is Exhibit 3055 in your binder. Do you have that there?

A. Yes.

Q. Okay. And so this is as of January 2019 the CV you prepared; correct?

A. Yes.

Q. Okay. Now if you look at the second page, you have an entry here that Mr. Miller touched on briefly that says that from 2001 to current --

A. Oh, that's a mistake.

Q. Right.

A. Yeah. It shouldn't be "current."

Q. It says -- well, this is your CV that you prepared; correct?

A. Well, I don't go over it every month.

Q. Okay.

A. Except for adding papers.

Q. All right. Just want to make sure the ladies and gentlemen of the jury understand, though --

A. Yes.

Q. -- you have on your CV that from 2001 to current, and then you say --

A. It should state 2018.

Q. Okay.

A. Sorry.

Q. 2018?

A. Because this was January 2019. But it should say 2018. I was a member of that board until 2018.

Q. All right. And you -- it goes on to say that you were the chair since 2005.

A. Yes.

Q. Is that accurate or not?

A. That's accurate.

Q. Okay. And you were a member since 2001 of the External Advisory Committee for the NCI/NIEH Agricultural Health Cohort Study. That's the AHS study that we've been talking about today?

A. That's correct.

Q. Okay. Now in that role, you were able to have input into the study; correct?

A. Since 2001, yes. And it ended in 2000 -- I think '8 because after that they never convened an advisory panel anymore.

Q. Okay. But you just told us, I thought, that you were the actual chair until 2018; correct?

A. Yes. They never told us that they disbanded it.

Q. Okay.

A. So, yes, they from time to time told us, "Oh, when we have money again, we'll see each other." So, yeah.

Q. But it's accurate to say that until actually you were hired by plaintiffs' counsel in this case, you were the chair, in your mind, of the AHS Advisory Committee; correct?

A. Of a defunct advisory panel that hadn't been meeting in almost a decade, yes.

Q. Okay. Well, I'm just trying to understand what you have on your CV.

A. Yes, I understand. Yeah.

Q. Okay. All right.

And at no time while you were in that position did you actually raise any of the criticisms that you raise today regarding the AHS study; correct?

A. No. I actually mentioned that about a year and a half or two years ago to them.

Q. After you were hired by plaintiffs' counsel and no longer on the -- chairperson on the committee; correct?

A. Well, after the paper had come out, yes.

Q. All right.

A. And I had grounds to argue, yes.

Q. Right. After the Andreotti paper came out in

2018, you were actually hired by plaintiffs' counsel in 2016; correct?

A. That's correct.

Q. Just so the ladies and gentlemen of the jury are clear, though, prior to the time that you were hired by plaintiffs' counsel, you did not communicate any of the criticisms that you've talked about today to the investigators, to the people who were actually running the AHS study; correct?

A. I don't think this is correct because there were multiple manuscripts that were kind of in circulation before that since 2013, and I did mention criticism when they asked me to talk about those specific papers.

Q. Dr. Ritz, were you previously asked whether you had had any discussions with any of the agriculture health scientists regarding any study data on glyphosate and non-Hodgkin's lymphoma, and did you answer that that you had not?

A. It wasn't glyphosate. It was the exposure assessment I was talking about with them. It wasn't any results on glyphosate. It was generally -- because we meet at meetings and we talk, we are colleagues.

Q. Right.

A. And I asked them about their exposure

assessment, about their follow-up problems, about their imputation, because that's my profession, exposure assessment. Not specifically to glyphosate, but to all pesticides that they were analyzing. Yes, we discussed those.

Q. Now, specifically you've talked about today that you were concerned about the glyphosate exposures because of the increased use; correct?

A. Well, the change in use.

Q. Right.

A. The change.

Q. You've talked about that it went up dramatically which could result in misclassification; right?

A. Yes.

Q. Right. But you never told them, because you didn't talk about glyphosate, you never told that to any of the scientists at the committee while doing the study; correct?

A. Not specifically because that wasn't -- glyphosate wasn't something I was very interested in.

Q. Right. And in fact, before you were hired in this case, you had actually never studied glyphosate or its relationship or possible relationship to NHL; correct?

A. To NHL, no.

Q. Right.

A. However, I did study glyphosate for other outcomes in the State of California.

Q. Just not NHL which is what we're talking about; correct?

A. Correct. Yes.

Q. Now, you talked about that the committee didn't meet for, you said since the mid 2000s; is that what you said?

A. Yeah, I think either 2008 or 2009. That was -- I know I was chair once.

Q. And the classification or misclassification issue you talked about, you didn't talk about glyphosate specifically, but did you say to them, "Hey, we ought to do another questionnaire, and let's have a more comprehensive questionnaire"?

Did you send any e-mails to anyone saying, "Let's" -- "We've got to do that"?

A. Well, what I told them a lot of times is they should have done the study in California because we have records of pesticide, yes, and they wouldn't have had this problem because it would all be documented.

Q. Okay. Well, my question is a little different, Dr. Ritz, which was: The studies ongoing,

when you joined it as the -- you became the chair of the advisory committee.

A. Yes.

Q. Right? The study is ongoing in North Carolina and Ireland. So saying you should have gone to California probably doesn't exactly help the investigators do much, does it?

A. Exactly. That's why we said it kind of too bad.

Q. Okay. But the U.S. government National Cancer Institute is spending tens of millions of dollars doing a study that you don't feel like you should tell the investigators, "Hey, there's a way we could fix this"?

A. We couldn't --

(Simultaneous colloquy.)

BY MR. EVANS:

Q. We --

A. We couldn't fix it because they had already done everything and started everything. They had done their baseline in 1993 through 1997. I came on the advisory board in 2001. They were in the middle of the follow-up. There was nothing I could do.

They were already doing everything they thought was the best to do at that time. And I got stuck with that. And that's when I kind of smilingly

said, "Well, maybe you should have done this in California because you would actually have had records, sorry."

Q. And so instead of suggesting, "Hey, why don't we send out another questionnaire that's more comprehensive" -- I mean, you knew what the questionnaire was; correct?

A. Yes.

Q. Right. And instead of saying, "Hey, why don't we send out another questionnaire and get the data we need to make this study that's ongoing meaningful according to Dr. Ritz," you just said, "We should do it in California"?

A. No. But you cannot change course in the middle of a second assessment. They already had started the follow-up in 1999. I came as an outsider in 2001. It took me a year to realize what they were doing. It was already 2002. By 2003-4, they were done. There was nothing I could do or propose anymore that would have changed what they were doing in the middle of doing.

Q. Is the AHS still ongoing?

A. I'm not really sure.

Q. Okay. So you don't know --

A. They're following, yes. What I know is that whenever they have money to link these individuals to

cancer registries, they do it. However, I think they gave up after the third round of trying to reach people and having lost yet another 15,000 people who didn't want to answer, they gave up sending out questionnaires and trying to reach these people.

Q. Now, I want to go back to you talked about the class that you teach at UCLA; right? You showed a PowerPoint that you use.

A. Yes.

Q. Okay. I just want to go back to that.

And the slide deck is titled "Introduction to Cohort Studies"; correct?

A. Yes.

Q. And this is in the fall of 2012; right?

A. Yes.

Q. Okay. And in this class that you teach, you actually teach -- in Table 1 -- in Table 1 you've got something called "validity for etiologic inference according to study design."

Did I read that correctly?

A. Yes.

Q. And you listed from your perspective, from an epidemiologic perspective, the validity of studies -- and by the way "etiologic" is causation; right?

A. Yes.

Q. Okay. And you've listed here randomized clinical trial is the highest form of evidence; right?

A. That's what this table lists.

Q. Well, this is from your class?

A. It's not a table I made.

Q. Okay.

A. It's a table from a publication, and you can see the citation at the bottom.

Q. Right. Clearly you didn't make it. I've certainly seen this before.

And the question is: In your class, you're teaching students that this is the well understood and recognized hierarchy of epidemiology studies; correct?

A. No. Absolutely false. This is a table that I use to dispel a myth that this is the right ranking. Okay? That's how I use this table.

Because I tell my students that what you call a prospective cohort study is maybe not really prospective. Because the AHS study is called the prospective cohort study because we're starting in 1993 to follow prospectively for the outcome. However, we are going retrospectively assessing exposures because we're asking them to report lifetime exposures that have already happened.

So is this now prospective or retrospective?

It's both.

Q. Okay.

A. And what I also teach them is that a nested case-control study has exactly the same validity as a cohort study and they should not make the mistake to use this ranking.

Q. All right. Just to be sure, whether you call the AHS either prospective -- and "prospective" meaning you start today and you just look forward; right?

A. Correct.

Q. Or if you include a retrospective component to it, either one are both higher on this ranking than a case-control study; true?

A. In this table. And as I said, I and others completely -- including Sander Greenland who taught me and wrote the textbook on epidemiology, completely disagree with this kind of ranking. A nested case-control study has the same validity as a cohort study if done properly. Actually it can be better than a cohort study.

Q. And a nested case-control study, as I understand it, is actually a case-control study that's conducted within a cohort study; right?

A. No, not -- that is one way. But another way, it can be nested also in a population based on a

population registry just like all the cancer registry studies we've seen.

Q. All right. And then your course goes on for several different PowerPoint slides to talk about the design of the cohort study. You go on for several slides on design, cohort study examples. Right?

A. Correct.

Q. And then you've got causal inferences in cohort studies, experimental versus observational studies.

A. Correct.

Q. And then you've got several other slides. And then you finally get down to where you talk about -- after you go through the cohort studies, you talk about what you showed the jury earlier today which is just what you called the disadvantages of the cohort method; right?

A. Correct.

Q. Now, right above that, you've got a section called advantages of the cohort method; correct?

A. Right. Because this is my lecture on cohort studies so I have to present both.

Q. Right. But what you didn't do -- and then you have an example. The Agricultural Health Study.

A. Yes.

Q. And none of your slides that talk about the AHS specifically -- and there are one, two, three, four, five, six -- and then you go the Ag Health Study topics. None of those slides actually document back in 2012 before you were hired by plaintiffs' counsel the criticisms that you said today; correct? None of those slides say that?

A. I wouldn't think so. Why would I give the answers that I ask my students to figure out? I don't do that. I don't put that on slides. They have to write it down.

I ask them questions, we discuss. I give them material to discuss. And then, you know, after that discussion, they can write the answer.

Q. All right.

MR. EVANS: I think now is a good time to take a break, Your Honor, if it works for you. Or should I keep going?

THE COURT: Can you keep going for another 15 minutes?

MR. EVANS: Sure.

THE COURT: Okay.

BY MR. EVANS:

Q. I wanted to look for a few minutes at the same data that you had on the chart that Mr. Miller used with

you.

Now, what I did here was I just -- the forest plot that was on this side. I didn't recreate because I wanted to have these numbers big enough and I wanted to have a column here to talk about whether these studies were actually adjusted for other pesticides or not.

Do you understand?

A. Yes.

Q. Okay. Now, with respect to whether Andreotti and De Roos, those first three were adjusted for other pesticides, which may be confounders; do you agree with me they were adjusted?

A. Yes, they were.

Q. So I'm going to put yes --

A. They were adjusted for pesticides. Whether I agree that they adjusted for the correct ones is a different question.

Q. I understand.

Now, De Roos 2003 --

A. Was fully adjusted.

Q. Okay. And that's the one you talked about earlier which you have the two different adjustments, one you called -- I don't know who put this up here, but you talked about the hierarchal regression.

A. Yes.

Q. Bayesian.

A. The Bayesian is not an adjustment. The Bayesian brings in other knowledge. It's a weighing scheme. It's not -- it has nothing to do with adjustment.

Q. Right. So --

A. So only the first one is the one we should use and should call fully adjusted.

Q. So that's yes.

A. Yes.

Q. And now just to be clear, the De Roos 2003 study was actually looking at, you said, three earlier studies; correct?

A. It was the combination of data from three earlier studies.

Q. And those -- that same data -- and I think we're going to connect the dots here in a second, but that same data from De Roos 2003 has been reanalyzed in the NAPP study you talked about; true?

A. Together with McDuffie.

Q. Right. And so we're going to put down here the NAPP study just to keep track of this.

But if you bring De Roos -- I'm just going to draw an arrow down here that goes into the NAPP study along with McDuffie; correct?

A. Yes.

Q. Here. So those two go into there.

Now, just so we can hopefully be done today, the NAPP study and the De Roos study, Dr. Weisenburger, who's going to be here tomorrow, was one of the investigator authors on those studies; correct?

A. Yes.

Q. Okay. So we're going to leave the NAPP study for him to talk about.

Now with respect to the Eriksson 2008 and the chart that was prepared by plaintiffs' counsel, it has two numbers here, one says most adjusted and one says is not. Was this number here, you agree, not adjusted for pesticides?

A. It wasn't.

Q. Okay. So that Eriksson -- so, I'm sorry. So the Bayesian, you want to put "no" on that?

A. No. The Bayesian was fully adjusted, but it's not a way of adjustment. It's a different kind of analysis.

Q. All right. So what do you want me to put there? Yes, no, or not applicable?

A. Not applicable.

Q. Okay. Now, Eriksson, this is not adjusted; correct?

A. Yes. Not adjusted for pesticides.

Q. For pesticides, which is what I'm talking about.

And the most adjusted, the 1.51, that was adjusted?

A. Adjusted, yes.

Q. Now, the 10 days number here on Eriksson, that was not adjusted; correct?

A. No, because they wouldn't have had the numbers to do that.

Q. And Hardell and Eriksson, I think this is just a typo. This says 1999. I know there were actually two Hardell studies, but I think this is actually from -- I'm actually not sure what it's from --

A. 2002.

Q. -- the 3.0, that's a 2002; right?

A. Yes.

Q. All right. So I'll just correct that. 2002. And was that -- this 3.0 number not adjusted; correct?

A. I don't believe so.

Q. All right. And the most adjusted, the 1.85 was?

A. Yes.

Q. And McDuffie was not adjusted; correct?

A. Yeah, the ever/never wasn't.

Q. Well --

A. And the more than two days, I don't think was either.

Q. Right. You talked about earlier that they did a multivariate assessment up front and decided that they were not going to adjust because it didn't need to be adjusted; correct?

A. Correct. And then later McDuffie made it into the NAPP, and the NAPP actually did adjust.

Q. Okay, right. And again --

A. Yes.

Q. -- tomorrow we're going to talk about the NAPP. I promise it's going to be riveting and very exciting, but we'll do it.

And Orsi I believe was not adjusted; true?

A. I'm not sure about that. But I would imagine they couldn't adjust because they had three cases.

Q. Okay. And then again the chart prepared by plaintiffs' counsel, it actually talks about the studies that make up these different meta-analyses; you see that?

A. Yes.

Q. Okay. And M here is the Orsi study which is not adjusted.

A. But has a very small weight, almost no weight

at all.

Q. Understand.

And then K here --

So I'm just going to put not adjusted.

And then K and L, those are the two McDuffie numbers which were also not adjusted; correct?

A. Yes.

Q. And then if we look at the rest of these, the -- on each of these meta-analyses use the -- either the Andreotti or the De Roos data; correct?

A. Yes.

Q. And that's adjusted.

Now C, it's the highest exposure, that's in Zhang.

D and E. D and E, those are --

A. Adjust.

Q. -- adjusted.

F, G, and H.

A. Depends on what they used.

Q. Right. So G was adjusted, but F was not; correct?

A. Looks like it.

Q. And then J, most adjusted, was adjusted; is that fair?

A. Yes.

Q. Okay. So putting aside the issue of chance, which again is one of the things IARC was concerned about, if you're looking at the confounding potential by pesticides, these are what these studies adjusted or did not adjust for pesticides; right?

A. Yes. But we have to agree that pesticides are actually risk factors for NHL and which ones are and which ones aren't.

Q. Okay.

A. Because, you know, if we don't, then we don't have to adjust. And adjustment doesn't do anything.

Q. I'm just talking about what the study did; right?

A. Yes.

MR. EVANS: I actually do need to set up now for the next session.

THE COURT: That's okay. We'll take a 15-minute break.

(Recess taken at 2:22 p.m.)

(Proceedings resumed in open court in the presence of the jury at 2:45 p.m.)

THE COURT: Continue, Mr. Evans.

MR. EVANS: Thank you, Your Honor.

THE COURT: We may need to take another quick five-minute break at some point.

MR. EVANS: Just --

THE COURT: We'll just have a short break in an hour.

MR. EVANS: Okay. Sure.

Q. So, Dr. Ritz, I got a little ahead of myself. I wanted to just make sure. You also referenced the Leon study; correct?

A. I didn't hear you.

Q. The Leon study.

A. Oh, yes.

Q. That's the last one that just came out recently; correct?

A. Yes, correct.

Q. Now you told the ladies and gentlemen of the jury about the DLBCL number.

A. Yes.

Q. This is the study that has the 130,000 or whatever from France, and this is the big --

A. Yes.

Q. -- big study; right?

A. Yes.

Q. You didn't tell the ladies and gentlemen of the jury what the actual overall NHL number is, did you?

A. I wasn't asked.

Q. Okay. And that's -- that number is actually

in this big study .95; correct?

A. Yes, I think so. Do you want me to look it up?

Q. Actually, let's put it up here on the ELMO.

This is again the study you were shown, Exhibit 2984, and that's the Leon study; correct?

A. That's correct.

Q. And this is the Table 2 that you were looking at; right?

Confusing when I turn it around in circles.

Right?

A. Yes, it is.

Q. And the .95, that is for non-Hodgkin's lymphoma; correct?

A. That's correct.

Q. Okay. And the confidence intervals there are .77 and 1.18; correct?

A. Correct.

Q. Just write that on here. .77. And what was the second one? 1.18?

A. Yes.

Q. Now, the NAPP numbers that you put up with Mr. Miller, those were from the June report; correct?

A. I believe so.

Q. Right.

And you know, in fact, that there have been subsequent reports that have different numbers, lower numbers, and in fact lose statistical significance; correct?

A. Would you want to show me those?

Q. Well, do you know or not know?

A. There were three different versions.

Q. Okay. Again I'm going to --

A. But what numbers are you referring to?

Q. Well, I just want to --

A. Yeah.

Q. Dr. Weisenburger is going to talk about NAPP. I just want to make sure. You talked about the June numbers; correct?

A. I talked about a study that was presented in Ontario in June -- on June 3, 2015, yes.

Q. And you know that there were subsequent reports from the same study that have different findings; correct?

A. They don't have different findings. They're all very consistent actually. But they have slightly different numbers because they're doing different things, yeah.

Q. Okay. So I'm just going to put down here June is the Dr. Ritz report that you were using.

And, again, Dr. Weisenburger tomorrow will talk about, I assume, the later numbers.

(Pause in the proceedings.)

BY MR. EVANS:

Q. Now I want to talk a little bit about the AHS study. And I want to make sure I understand what your testimony is.

Do you have a problem with the 2005 report?

A. Yes.

Q. And you think that that is also not a valid report, valid study?

A. The dose response analyses, no, I don't consider them valid for glyphosate. For other pesticides, that's a totally different issue, yes.

Q. And 2018, the second study, you've also expressed your criticisms; you think both of them are flawed fundamentally.

A. Yes.

Q. And when the 2005 report came out when you were the chair of the advisory committee in 2005, again, you didn't talk to any of the investigators about glyphosate and NHL; true? It's what you testified to earlier; right?

A. Yeah, yeah, yeah.

Q. So you thought it was a flawed report. And

you didn't bother to pick up the phone, send an e-mail, and say, "You know what, this is really mistaken," because of the criticisms you've expressed today?

A. Well, I had hoped that they could actually improve upon what they were showing in 2005 with the follow-up data, and unfortunately they couldn't.

Q. Right. But as the chair of the committee that's advising the study, you didn't actually tell anybody about your criticisms; correct?

A. Well, what would have that -- what would have been changed by that? Because they had already done all of their data collection; right? And so the next step is to do the analyses and see what happens.

Q. Okay. My question is a little different, though; right? Which is you actually didn't tell anybody about your criticisms; correct?

A. I don't remember actually looking specifically at the study because that wasn't what we were asked to do when we got together at these meetings.

What we were asked to do at the meetings is to evaluate the whole process of what NIH was doing, not their results. Okay. And by the time in 2005, they had already done all of their process and what they mostly were presenting to us were kind of nested case-control studies, additional exposure assessment, a little

smaller studies.

I remember that one session of this meeting -- or several days were just talking about studies where EPA would and NIOSH would be going out into the fields trying to actually measure pesticides and trying to find out more about application methods.

So these were the kinds of things that we were discussing at these meetings, not what they had already done and couldn't be changed anymore. And not results either. It was really about process.

Q. And my question, I think, was a little simpler. And I'm sorry --

A. Sorry.

Q. Which was: The criticisms that you've talked about today when you were chair of the advisory committee, you did not tell your colleagues then; correct? Very simple question.

A. No, it's not simple. Because first of all, nobody asked me to review the specific paper. And second, I wasn't sure what -- you know, what would it have helped to go there and say, well, well, well, what did you do and can't change anymore; right?

What I was hoping was that the second set of data collection would actually improve upon what they had done in the first round, and unfortunately it

didn't.

And they were still hoping they would actually find more farmers. Right? Every year we were told, "Oh, we're doing everything to actually get to over 90 percent." But they couldn't.

And what do you tell people who put their blood and tears into trying to do this and, you know, in the end, end up with something that, at least for glyphosate, doesn't work. Do you pound them over the head with it when it's already done? No, you don't. Plus I wasn't asked.

Q. Are you done?

A. Yeah.

Q. Okay.

A. Thank you.

Q. So my question again, if you could answer it, is: Did you tell the investigators when you were chair of the scientific advisory panel, did you tell them the criticisms that you -- as expressed today after you are being paid $500 an hour to give these criticisms; correct?

A. Incorrect. Because who am I supposed to tell something when a study is not even -- when I'm not even asked to actually be evaluating that specific manuscript? Anneclaire De Roos did this for her

dissertation work. Okay? She was a junior colleague who was partially employed by NCI who was doing this work for that kind of purpose to advance her career. And she did some really nice work generally. The 2003 paper is wonderful, the 2005 paper maybe not so great. But I was hoping that she would improve upon it.

Yes, and we did discuss exposure assessment, but, you know, what do you do when they already did everything that they did?

What I was hoping was that they could actually go in and assess these exposures in a nested case-control study in the future. But by the time that could have been said, there was no more advisory panel meeting. Because the advisory panel meetings I was at were all about the cohort, nothing else.

Q. All right. I think I understand the answer to my question which is, no, you did not tell anybody. I understand you've got lots of reasons that you articulated, but the simple answer is, no, you did not tell anybody the criticisms that you told the jury about today; correct?

A. Well, the Andreotti paper came out, when? 2018? So how can I criticize a paper that hasn't come out until 2018?

Q. But you also have criticisms that you just

said in the 2005 paper; correct, Doctor?

A. For the baseline assessment, yes. But I was hoping that that could be overcome with the second -- with the second repeat in the field, yes.

Q. You were hoping, but you weren't actually telling any of those invest --

A. We were all hoping. We were all hoping that we would find all of these farmers again.

Q. Can I finish my question, please?

A. Yes, go ahead.

Q. Right. You were hoping, but you weren't actually communicating to the investigators the criticisms that you talked about today; true?

A. This is really the wrong question. And there's no answer to it. They were just doing the second assessment. If they had gotten 98 percent of the people back to report what happened in the meantime, they would have been able to do a bang-up job. I couldn't know that in 2005 that that wouldn't be the case.

Q. All right. Let's -- let's move on.

Let's take a look at Exhibit 4106, which is the Andreotti paper that's been previously published.

A. What number?

Q. 4106. I've got a copy here. It's a little

different number than what you've got. I'll just hand it up if you'd like.

A. Yes.

Q. That might be a little bit easier.

MR. EVANS: May I approach, Your Honor?

THE COURT: Yes.

THE WITNESS: Thank you.

BY MR. EVANS:

Q. And I want to talk about just first with respect to the Andreotti paper, the methods. Do you see that described on the first page?

A. Yes.

Q. Okay. And it explains the AHS is a prospective cohort of licensed pesticide applicators from North Carolina and Iowa.

A. Yes.

Q. (Reading from document:)

Here we updated the previous evaluation of glyphosate with cancer incidence from registry linkages through 2012 in North Carolina, 2013 in Iowa.

Lifetime days and intensity weighted lifetime days of glyphosate use were based on self-reported information from enrollment from '93 to '97.

Did I read that right?

A. Yes.

Q. And follow-up questionnaires from '99 to 2005; right?

A. Yes.

Q. (Reading from document:)

> We estimate incidence rate ratios and 95 percent confidence intervals using Poisson regression --

A. Poisson. Poisson.

Q. -- controlling for potential confounders including use of other pesticides. All statistical tests were two-sided.

Did I read that right?

A. Yes.

Q. And then it goes on to give the results. And it states:

> Among the 54,251 applicators, 44,000 used glyphosate including 5,779 incident cancers, 79 percent of all cases. In unlagged analyses glyphosate was not statistically significantly associated with cancer at any site.

Did I read that correctly?

A. Yes.

Q. (Reading from document:)

However, among applicators in the highest exposure quartile there was an increased risk of acute myeloid leukemia compared with never users though this association was not statistically significant. Results for AML were similar with a 5-year and 20-year exposure lagging.

Right?

A. Yes.

Q. And we're not here talking about leukemia; correct?

A. I don't think so.

Q. And one of the things that you didn't talk about today was the lagging that they actually have looked at; right? Which is they looked at 5- and 20-year reported out different lagging results; correct?

A. Yes.

Q. And let's take a look at those.

And if you turn to Table 3 and the non-Hodgkin's lymphoma. Do you see that?

A. Yes.

Q. And there's the 5-year lag. This is on 4106.0006.

A. Yes.

Q. Okay. The 5-year lag of non-Hodgkin's lymphoma. And then they've got it broken down into quartiles. Do you see that?

A. Yes.

Q. And the different quartiles, I think you explained this earlier, they actually went through a process whereby they tried to group the applicators depending upon how much they actually used and were exposed to glyphosate; correct? To Roundup?

A. Yes. The intensity weighted process that I described as completely faulty.

Q. And I understand. But the authors and investigators here reported out these numbers. They don't think it's completely faulty; correct?

A. They are reporting numbers. I don't know what they think. They are reporting what they see, yes.

Q. Now, when you look at whether you're in the lowest exposure group or the highest exposure group, none of them show an increased risk that's statistically significant; correct?

A. Correct.

Q. And they don't show a protective effect that's statistically significant; correct?

A. Nothing is statistically significant.

Q. And if you look over to the right, there's actually a 20-year lag. Now what does a 5-year lag and a 20-year lag in this study mean?

A. That's a good question because this is an intensity-weighted scheme so it's really hard to say what it really means. But generally it means we are taking out the last five years of exposure prior to the onset of disease because we think it's not relevant to the disease. So we're making the assumption what happened in the last five years before somebody was diagnosed is irrelevant.

Q. And it has something to do with the latency of a disease; correct?

A. That's correct.

Q. And so if, for example, I'm exposed to something today, and I'm diagnosed with something tomorrow, depending on the latency of that condition, it may or may not be related; right?

A. Yes.

Q. Okay. And the -- right below that there is a B-cell lymphoma; correct?

A. Yes.

Q. And those also, for both the 5-year and 20-year lags, do not show a statistically significant increased risk; correct?

A. They're almost the same because they're almost the same number.

Q. Now, if you go back to page 2 of the exhibit and it's the second column and it looks like the second sentence starting with "Using this information."

And the article says:

> Using this information three metrics of cumulative lifetime exposure were created for each pesticide. Ever/never use, lifetime days of use (days per year times number of years), and intensity-weighted lifetime days (lifetime days times intensity score.) The intensity score was derived from an algorithm based on literature-based measurements and information provided by the applicator, specifically whether the participant mixed or applied pesticides, repaired pesticide-related equipment, used personal protective equipment, and application method used.

Right?

A. Yes.

Q. Okay. And so they are there, at least, trying to explain, and I know it's not to your satisfaction,

but they're explaining the method they went to to put people in different quartiles of exposure and assess the results. Fair?

A. This is their way of explaining how they've generated these quartiles. However, what they're not saying here is how they derived this data. They're not saying that they asked for 21 pesticides one question: Did you repair? We don't know whether they repaired when they used glyphosate. We don't know whether they used personal protective equipment and which kind when they actually used glyphosate.

If they used more than one pesticide, it could be any pesticide. So what they are reporting is what any pesticide. We're assuming that what they're reporting in one question applies to glyphosate. A huge problem.

Q. A huge problem that until you were being paid by plaintiffs' counsel, you didn't bother to tell them with respect to glyphosate; correct?

A. We have discussed exposure assessment ad nauseam at these meetings. How they translate into papers depends on the first author.

Q. Now --

A. I would not have done these quartiles, but they like them.

Q. So you disagree with the authors?

A. I disagree with the way they analyzed this data, yes.

Q. All right. And then if you go to page 7, first full paragraph says "In our study." Are you with me?

A. Yes.

Q. (Reading from document:)

> In our study, we observed no association between glyphosate use and NHL overall or any of its subtypes. This lack of association was consistent for both exposure metrics, unlagged and lagged analyses, after further adjustments for pesticides linked to NHL and previous AHS analyses, and when we excluded multiple myeloma from the NHL grouping.

Did I read that correctly?

A. Yes.

Q. Now, one of the criticisms you have raised today relates to the issue of imputation of data for individuals who didn't respond; correct?

A. That's correct.

Q. Okay. And if you go to page 4 of the study, the second -- I guess it's the first full paragraph on

the left, down below the numbers it says: To evaluate the impact -- do you see that?

A. Not yet.

Q. Second paragraph down a little bit with all those numbers. Keep going. There you go. Right there.

A. Okay.

Q. Down.

A. Thank you.

Q. "To evaluate the impact," below the numbers. There we go. Great.

> To evaluate the impact of using imputed exposure data for participants who did not complete the follow-up questionnaire, we limited the analysis to 34,698 participants who completed both questionnaires, reducing the total number of cancer cases to 4,699.

Right?

A. Yes.

Q. And then it says:

> Glyphosate use was not associated with NHL.

Did I read that correctly?

A. Yes, you did.

But that doesn't negate my criticism of their

baseline questionnaire or their follow-up questionnaire.

MR. EVANS: Move to strike, Your Honor. No question pending.

THE COURT: Granted.

BY MR. EVANS:

Q. Now, Dr. Ritz, you have previously stated that you actually admire your colleagues for doing the AHS study; correct?

A. Yeah, it took a lot of courage.

Q. Right. And you think there's a lot of useful data that's resulted from this study; correct?

A. Yes, for other pesticides.

Q. I understand. You today are saying that with respect to glyphosate this study is hopelessly flawed, but, again, didn't bother to tell people that before you actually started being paid by the attorneys; correct?

A. I don't know what I should say.

Q. Okay. Well, you can answer "yes" or "no."

A. Well, if somebody had asked me about glyphosate, I would have said exactly what I'm saying today, yes.

Q. Even though you said earlier that you hadn't actually looked at NHL and glyphosate; right?

A. If somebody had asked me to look at it, I would have made exactly the same criticism and no

exception.

Q. Now, there are over 250 peer-reviewed publications based on the AHS data; correct?

A. Yes.

Q. And you would characterize that study as being a very productive study with respect to the amount of results that have been generated?

A. It's a very productive study, yes.

Q. And you also think you've said before the AHS is in fact a beautiful study; correct?

A. I have said that, yes.

Q. And in fact, Dr. Andreotti actually won an award in 2018 for the publication that you've been criticizing; correct?

A. I don't know.

Q. You don't know that she won an award by --

A. No.

Q. -- the National Cancer Institute for the very publication that you are now criticizing?

A. I don't know it, no.

Q. Have you looked for that?

A. Why would I?

Q. Well, you were questioned about it before; right?

A. I didn't know. I'm not looking up people for

their awards. That's not what I do. I have other things to do. Sorry.

But, yes, maybe she did. That's fine. They honor and award awards to people who do a lot of work, and she probably did a lot of work. That doesn't mean I have to agree with her results or I have to agree with how she did the work.

Q. And it doesn't mean that she has to agree with your criticisms; right?

A. What?

Q. It doesn't mean that she has to agree with your point of view either; correct?

A. But there are others there agreeing with mine as well.

Q. Now, just to be clear, the Agricultural Health Study has not been in any way funded by Monsanto or industry; correct?

A. No.

Q. They did not have input or control over the study; correct?

A. As far as I know, Monsanto criticized this study many, many, many times.

Q. Okay. And my question was a little different, which is: Did they have input or control?

A. Well, they certainly tried to exert control in

these meetings that I was at by criticizing this study and by publishing criticisms about the study.

Q. Now, the evolution of the AHS over time, you understand that there have been several papers, and we talked about 250 different papers, we talked about two of them with respect to NHL.

A. Yes.

Q. But there've been a lot of updates to the results of the AHS; correct?

A. I'm not sure I know what you mean.

Q. Well, the evolution of the understanding of the methodologies that were being used is, now in 2018, understood where it wasn't perhaps 20 years ago; right?

A. Those methodologies have not changed. I mean, they asked their questions in 1993 to 1997, that was their methodology and that's -- you know, there's nothing we can change about that. We would probably do it differently now if we designed another study, yes.

Q. Now, you talked about the 2005 results.

And one of the authors --

MR. EVANS: May I approach, Your Honor?

THE COURT: Yes.

BY MR. EVANS:

Q. You said a minute ago that Dr. De Roos apparently was not experienced when she did this, but

this was also Dr. Blair who we've heard a lot about who ended up being on IARC was actually on the 2005 paper; correct?

A. He was on this, yes.

Q. Okay. Let's take a look at it for a minute. And just look at the abstract at the top. It says:

> Glyphosate is a broad spectrum herbicide that is one of the most frequently applied pesticides in the world. Although there has been little consistent evidence of genotoxicity or carcinogenicity from in vitro animal studies, a few epidemiologic reports have indicated potential health effects of glyphosate. We evaluated association between glyphosate exposure and cancer incidence in the AHS, a prospective cohort study of 57,311 licensed pesticide applicators in Iowa and North Carolina. Correct?

A. That's what it says.

Q. (Reading from document:)

> Detailed information on pesticide use and other factors was obtained from a self-administered questionnaire completed

at the time of enrollment from '93 to '97. Among private and commercial applicators, 75.5 percent reported having ever used glyphosate of which 97 percent were men. In this analysis, glyphosate exposure was defined as, A, ever personally mixing or applying products containing glyphosate, B, cumulative lifetime days of use or cumulative exposure days, years of use times days per year, and C, intensity-weighted cumulative exposure days, years of use times days by years times estimated intensity level.

Do you see that?

A. Yes, the same method Andreotti reported.

Q. Okay. So they are looking at, again, the different usage of Roundup -- of glyphosate by these different agricultural workers; correct?

A. What was that?

Q. They're looking at the use of glyphosate or Roundup by these agricultural workers, and they're trying to assess which of them are being exposed a lot, which of them are being exposed not so much, and then in between; right?

A. Well, they asked them between 1993 and 1997

about their lifetime use of glyphosate, and then they used that to turn it into these exposure estimates.

Q. And if you turn to page 4, the discussion, first sentence there says:

> There was no association between glyphosate exposure and all cancer incidence or most of the specific cancer subtypes we evaluated, including NHL, whether the exposure metric was ever used, cumulative exposure days, or intensity-weighted cumulative exposure days.

Right?

A. Yes.

Q. Now, one of the benefits of a prospective cohort study is that you can measure people's use going forward; correct? You're following a group of people and you can see what they're doing going forward; right?

A. You wish that that's what they had done, but they didn't.

Q. I'm talking about in general. A cohort study, that's one of the things that you can do.

A. That's one of the things that studies like the Harvard Nurses' Health Study does by sending questionnaires every two years to 100-some-thousand

nurses who every two years report and have a follow-up of over 90 percent in these nurses. That's a different type of study.

This study lost in the first five years 38 percent of the cohort.

Q. Understand. I think --

A. Yeah.

Q. -- with respect to the Andreotti paper and the analysis, I think we all understand you have some serious criticisms you're expressing today; right?

A. Yes.

Q. Okay. And the benefit of being the chairman of the Science Advisory Panel would be that you could have expressed them and hopefully have changed how it's being done; correct?

A. No, I couldn't.

MR. MILLER: Your Honor, argumentative. Asked and answered now eight times.

THE COURT: So I'm going to sustain the objection to the extent that it does cover ground that's been covered. But I would also ask Dr. Ritz just to answer exactly what's being answered -- asked. Not answered.

THE WITNESS: Yes.

///

BY MR. EVANS:

Q. Now, in contrast to the prospective cohort, you can also have a retrospective and look back; correct?

A. Yes.

Q. And one of the problems with the look back potentially in a cohort -- and I'm not talking about the AHS, just talking about in general --

A. Yes.

Q. -- is this whole issue about recall bias that we talked about earlier; right?

A. No, that's not what is the problem with retrospective cohort studies.

Q. Okay. If you're looking backwards and trying to remember what you used 10 or 20 or 30 years ago, you don't think that could be a problem?

A. That's not what we call a retrospective cohort study technically.

A retrospective cohort study starts in the past. For example, I start with a worker cohort in 1950 for whom I have records of having worked in an industry, and I follow them forward up to now in terms of every exposure and their outcome. That's what we call a retrospective cohort study. There's no recall. There are records.

Q. Okay. When does a recall bias become an issue for you in epidemiology, or does it?

A. It is. So it is an issue in case-control studies because we're asking people to remember what they did throughout their whole life. And it's the same issue in cohort studies if you're asking people at the beginning of the study to recall everything they did during their whole life. And then you're not following them every two years with the same question to update this. Because you can remember much easier in a two-year period than you can your whole lifetime.

Q. And if you are in a study like a case-control study and if you're someone who has the condition that is being analyzed and you're asked to look back 10 or 20 or 30 years, that data may not be reliable; fair?

A. It may or may not be reliable. However, we have techniques to make it more reliable by spending a lot more time and effort on every case and every control, going over records with them, and giving them a lot of time to remember and look at their records, talk to their coworkers, talk to their wives, and then report.

These workers in the AHS came to take a test and had half an hour to bubble in. They were not able to go back and do all of the very intense records search

that in a case-control study you can actually afford because you have a limited number of people and you can guide them through it. You can't do that with 56,000 individuals.

Q. Again, I wasn't even asking about the AHS. I was just asking about in general. So I move to strike --

A. Well, yes, but I'm trying to put this in context for you.

MR. MILLER: That answer is responsive. And I object to that.

MR. EVANS: Well, I move to strike as nonresponsive.

THE COURT: Well, the question was -- I'm going to strike everything regarding AHS. The question was about generally the cohort or the case study. Just the general question about the way which a case study operates. So I'm striking the response regarding the AHS study which he was not asking about.

So just listen very carefully to the question.

THE WITNESS: Okay.

MR. EVANS: Can I have Exhibit 6625, please.

Q. Now you showed the ladies and gentlemen of the jury a summary of several articles that have been written that talked about some issues regarding the AHS

study; correct? That was one of the things --

A. What was that? Which study?

Q. Well, there were several letters and several comments that were summarized on one sheet, and you just had like one paragraph. Do you remember that?

A. Yes. Yes.

Q. And I'm just going to show you one response to one of those letters.

MR. EVANS: And may I publish, Your Honor?

MR. MILLER: No objection.

THE COURT: Yes.

(Exhibit published.)

BY MR. EVANS:

Q. And this is the last of those criticisms was something that was written by, I think, Sheppard and Shaffer; correct? That was one of the ones you referred to.

A. Yes.

Q. And this is the response to those criticisms by Dr. Andreotti and the other investigators on the study; correct?

A. It looks like it, yes.

Q. Okay. And if you go down in the first paragraph, it looks like the third sentence, "Although we agree."

It reads -- and this is, by the way, a response that's published in the National Cancer Institute Journal; correct?

A. I believe it must be the same journal that also published the criticism.

Q. Right.

A. Yeah.

Q. But again you showed the criticism, you didn't actually show this response; right?

A. I didn't show the criticism. There was one quote on a, you know, general slide.

Q. Okay. It says:

> Although we agree that this method could theoretically bias risk estimates toward the null, based on sensitivity analysis that we conducted and reported in the manuscript and described more fully below, we demonstrate that our imputation likely did not materially impact risk estimates.

Did I read that correctly?

A. Yes, you did.

Q. And then if you look at the last paragraph right before the funding section on the next column. It says:

Overall we believe that these data --

And that's talking about the Andreotti paper that we looked at; correct?

A. Yes.

Q. (Reading from document:)

-- demonstrate that not including outcome information our imputation of glyphosate exposure did not introduce meaningful bias in our cancer risk estimates associated with this pesticide.

Did I read that correctly?

A. Yes.

But that wasn't about my criticism.

Q. I understand.

And on the next page, there's a table with evaluation imputation method; right?

A. Yes.

Q. And so, again, one of the criticisms you talked about today was the imputation of data. And the authors have actually responded and evaluated that criticism and said they don't believe, given their methodology, their statistical approach, that it had an impact; true?

A. They believe, yes. And I believe something else.

Q. I think I'm done with the first binder, and I only have part of another one. So we should be getting there.

MR. EVANS: Now, okay?

MR. MILLER: Fine, Your Honor.

BY MR. EVANS:

Q. I think we're on the same page here. I just want to make sure and so we can move through this quickly.

Again, the same issue regarding adjusting. We talked about the McDuffie and the Eriksson studies that -- numbers with respect to those response that you talked about earlier, those are unadjusted numbers, they're not adjusted for other pesticides; correct?

A. For other pesticides, yes.

Q. Okay.

A. In those two studies, yes.

Q. Right, understood.

And just so I do this quickly, so the Eriksson and McDuffie numbers with respect to those response were not adjusted.

Now the NAPP studies, again, we're just going to punt that till tomorrow when Dr. Weisenburger will be here. These are actually different numbers from subsequent report that you didn't talk about so we're

going to leave that.

And then the De Roos, which is the 2005 AHS, and the Andreotti, they report out again several different analyses of the dose response issue which is they have these different quartiles and they did not see a dose response in their studies; correct?

A. Well, if anything, they saw a protective effect, yeah. It went to the other side of the 1. So there's no dose response there.

Q. All right, thank you.

See? That was quick.

MR. MILLER: No objection to publication, Your Honor.

THE COURT: Okay.

(Exhibit published.)

BY MR. EVANS:

Q. And, again, Dr. Ritz, just want to -- these, as I understand, are all the studies that you've talked about today regarding the DLBCL results; correct?

A. I would have to look up the Eriksson and Orsi, but I believe you.

Q. Okay. And, again, the Eriksson number, do you know whether that was adjusted or not?

A. It's probably not adjusted because it must be a very small number of cases that were exposed.

Q. And Orsi, I think you said earlier, was not adjusted; correct?

A. Did they even have more than one case? Could they do this?

Q. I just put out they reported --

A. I doubt that they have done that.

Q. And then, again, this is the NAPP that we're going to talk about with Dr. Weisenburger.

And then there are a number of different reports out, both 5-year, lifetime, and 20-year lag with respect to DLBCL in the Andreotti study. And those are adjusted numbers. And these are -- these are the numbers reported out; is that accurate?

A. Yeah, it looks like.

Q. And then Leon study you talked about here, the 1.36; correct?

A. Yes.

Q. And then the Chang study 1.1 with a confidence interval between .5 and 2.3. Does that sound right?

A. I would have to look at that study.

Q. Okay. And do you know whether those were actually adjusted or not?

A. What? Andreotti is adjusted. Leon was also adjusted.

Q. And what about Chang, do you know if it is?

A. No, I don't know.

Q. So these are adjusted?

A. Chang may or may not be because that's a meta-analysis; correct?

Q. Right. I think we looked at it earlier, which is --

A. And if they included studies with nonadjusted estimates, then some are adjusted, others aren't.

Q. Right. Okay. And so we looked at that earlier with respect to that, including some data from unadjusted studies; correct?

A. Yes.

Q. And Leon is adjusted.

A. Uh-huh.

Q. Okay. I just wanted to confirm that.

I think this has been shown before so it should be okay. This is the summary of the regulatory conclusions.

And I just want to ask you, Dr. Ritz, first of all, have you reviewed the regulatory conclusions by these different agencies that are up there?

A. I looked at the EPA one, EFSA, yes. Health Canada, I guess. Australia, no.

Q. Okay. And do you agree or disagree with each of those statements?

A. I certainly disagree with EPA, and I read that in detail. And I know exactly why I disagree with them. And I certainly disagree with EFSA. And I'm not the only one.

Q. And since you're the one who's here testifying, I'm going to ask for you. So with respect to the ECHA statement that's up here:

> Based on epidemiologic data as well as the data from long-term studies in rats and mice, taking a weight of the evidence approach, no hazard classification for carcinogenicity is warranted.
>
> Do you agree or disagree?

A. Absolutely disagree. And that's the chemical agency.

Q. And glyphosate -- this is EFSA.

> Glyphosate is unlikely to pose a carcinogenic hazard to humans.
>
> Do you agree or disagree?

A. I disagree. And what they're evaluating is diet-related to carcinogenicity. So not farmers.

MR. EVANS: Move to strike.

THE COURT: Sustained. Granted.

BY MR. EVANS:

Q. Next question.

Based on all the available data, the weight of the evidence clearly do not support the descriptors (carcinogenic to humans) and, quote, likely to be carcinogenic to humans at this time.

Do you agree or disagree?

A. I disagree.

Q. All right. And Health Canada:

Glyphosate is not genotoxic and is unlikely to pose a human cancer risk.

Do you agree or disagree?

A. Disagree.

Q. And Australia. You say you haven't seen this before, but I'll read it:

Scientific weight of the evidence indicates that exposure to glyphosate does not pose a carcinogenetic or genotoxic risk to humans.

Do you agree or disagree?

A. Disagree.

Q. And each of those different regulatory agencies have reviewed epidemiology; correct?

A. They review epidemiology, yes.

Q. In their assessments, they're reviewing the epidemiology that existed up to the time that they did

it; correct?

A. They do their best, yes.

MR. EVANS: No further questions.

MR. MILLER: Very brief, Your Honor.

REDIRECT EXAMINATION

BY MR. MILLER:

Q. If anybody else here is old enough to remember Paul Harvey.

MR. MILLER: It's been shown to the jury before. This is the EPA report, Your Honor, Exhibit 2112. If I might use the ELMO, please.

Q. Counsel for Monsanto just put up that the EPA says there's absolutely no carcinogenic risk for non-Hodgkin's lymphoma. Did you hear him ask those questions?

A. Yes.

Q. All right. Well, here is the report written by Mr. Jess Rowland and the committee in the Office of Pesticide Programs.

MR. MILLER: We have the ELMO on? We do. All right.

Q. And although he says, look, I can't say cancer all over. When it comes to non-Hodgkin's lymphoma, they just don't know.

MR. MILLER: Can you blow that up?

Excuse me. Okay.

Q. Due to study limitations and contradictory results across studies of at least equal quality, a conclusion regarding the association of glyphosate exposure and the risk of non-Hodgkin's lymphoma cannot be determined.

So they don't say it doesn't cause cancer. They say they just don't know there's conflicting studies.

A. Yes.

Q. Right? That would be a more accurate statement of what Mr. Rowland and his committee had to say; right?

A. Yes.

Q. All right. That's that.

It will take about five minutes.

So let's go to the Gray study. We've already published it before. It's Exhibit 1548. Okay.

Please go to page 22, the bottom paragraph.

And just to put this in context, the Gray study was the federal government's Agricultural Health Study, a critical review with suggested improvements, which this was published in the year 2000; right?

A. I don't have it in here but --

Q. Well, let's go back to the front page.

MR. MILLER: Your Honor, if I could approach?

THE COURT: Sure.

MR. EVANS: Your Honor, may I look at it before they publish it?

MR. MILLER: We'll take it down. Sure.

(Pause in the proceedings.)

MR. EVANS: No objection.

BY MR. MILLER:

Q. Okay. So we'll put that back up on the screen.

Counsel repeatedly asked you why didn't somebody tell them that they were off on the wrong course with this one-shot application thing. And the federal government here --

Let's go to the front page. Let's put this in context.

Okay. So this is year 2000, right, federal government, Agricultural Health Study, A Critical Review with Suggested Improvements, by Gray and others; right?

A. Yes.

Q. Now Gray is at Harvard; right?

A. Yes.

Q. Okay. So he's telling them in the year 2000 what kind of problems they have.

Let's look at page 22.

A. Actually, he's not alone. He has very illustrious occupational epidemiologists with him on this.

Q. These are people with a lot of gravitas?

A. Yes.

Q. And it's those environmental epidemiologists that understand exposure --

A. Occupational epidemiologists, yes.

Q. Occupational. All right.

MR. MILLER: So if we can blow up that bottom paragraph.

Q. Exploring the reliability and validity of pesticide use data.

Since pesticide use data will be the basis of categorizing potential pesticide exposure in the AHS, the validity of these data is also critical.

That's true, isn't it?

A. Yes.

Q. They've got to be valid data?

A. Yes.

Q. (Reading from document:)

A simple and pertinent step would be to re-administer the questionnaire to a sample of respondents to see how much the

answers change.

A. Correct.

Q. That was the recommendation in the year 2000; right?

A. Yes.

Q. And they never did that, did they?

A. Well, they re-administered between 1999 and 2004 to 62 percent.

Q. Okay.

A. But not -- they didn't ask the same question. They asked a different question.

Q. It says:

> Other studies to validate reported pesticide use, for example, by comparison with purchase records, are also essential.

A. Yes.

Q. That's the recommendation from Dr. Gray at Harvard and others. Did they do that?

A. No.

Q. Dr. Gray and his fellow scientists go on to say:

> A relatively simple check would consist of questions about number of acres for each specific crop for which a specific pesticide was used.

Did they do that?

A. Not in the papers that we looked at.

Q. So all these recommendations that were made 19 years ago to hopefully try to make this data more valid, none of them were followed?

A. Not in these papers, no.

Q. Counsel asked you about whether IARC said even though it was a probable human carcinogen it couldn't completely rule out chance or bias. Do you remember that line of questioning?

A. Yes.

Q. Since we have the Zhang study now and the Leon study, are you comfortable ruling out chance or bias?

A. Actually, the Leon study, the Norwegian results really do make me more comfortable, yes. Because these people did not recall pesticide use in Norway. They actually reported to their agriculture census every five years while they were farming, what they were farming, whether they were using pesticide equipment, and what crops they were farming. And in Norway, that's a very limited number. It's potatoes, grains, fruits and vegetables and meadows. And only on grains and maybe sometimes on meadows they actually apply glyphosate.

And then they looked when glyphosate was

registered, and they assigned these exposures according to what these farmers reported every five years between 1969 and 1989. And I think that's a pretty good exposure assessment.

Q. Very good. You've been very patient, and I thank you for your time. The last series of questions.

Back that up a little bit.

Anything that you were asked on cross-examination, or shown, did it in any way change your opinion that Roundup causes tumors in mammals?

A. No.

Q. Did anything that Monsanto's lawyer showed you change your opinion that Roundup causes malignant lymphoma in mice?

A. No.

Q. Anything that he showed you change your opinion that Roundup causes genetic damage in human lymphocytes?

A. No.

Q. Same question. Anything he showed you or discussed with you change your opinion, Dr. Ritz, that Roundup causes oxidative stress in human cells?

A. No.

Q. Last question, and I'll sit down. Anything that he showed you change your opinion that

non-Hodgkin's lymphoma in humans at real world exposure can cause non-Hodgkin's lymphoma?

A. In my professional opinion, yes, real world exposures can cause non-Hodgkin's lymphoma.

Q. And if I was a graduate student in your class and I asked you the same question, would I get a different answer because you've been retained by a lawyer?

A. No.

MR. MILLER: I have nothing further.

THE COURT: Is that it?

MR. EVANS: Just one question.

RECROSS-EXAMINATION

BY MR. EVANS:

Q. The Leon study that just came out actually showed no increase risk of .95 for NHL; correct?

A. Well, what we didn't discuss for --

Q. Could you answer the question?

A. Yes.

Q. Thank you.

THE COURT: Are we done?

FURTHER REDIRECT EXAMINATION

BY MR. MILLER:

Q. Just last question. But it does tell us that in diffuse large B-cell, there's a statistically

increased risk for non-Hodgkin's lymphoma?

A. Yes, absolutely.

MR. MILLER: Thank you for your time, Dr. Ritz.

THE COURT: Are we done?

MR. EVANS: Yes.

MR. MILLER: Yes, Your Honor.

(Witness excused.)

THE COURT: We're going to take a five-minute break. That's to the bathroom and back.

(Recess taken at 3:45 p.m.)

(Proceedings resumed in open court in the presence of the jury at 3:52 p.m.)

THE COURT: All right. So, ladies and gentlemen, we're going to resume.

And what you're going to see next is the videotaped deposition of a witness who will not be here live, but his testimony on the videotape is as though he were sitting here. So the evidence that he presents will have the same quality of any other type of evidence that you will consider when you begin deliberating.

MR. WISNER: Your Honor, just before the video, I'm going to read two admissions into the record.

Counsel has seen them.

THE COURT: Okay. That's fine.

MR. WISNER: Admission number 8. Request: Admit that in 1999 Monsanto hired Dr. James Parry to evaluate studies on the genotoxicity of glyphosate and provide a report on those studies.

Response: Monsanto admits that in 1999 Monsanto entered into a consulting relationship with Dr. James Parry to review research evaluating whether glyphosate and glyphosate-based products were genotoxic.

Admission number 9. Request: Admit that Dr. James Parry was a recognized genotox expert in 1999.

Response: Monsanto admits that Dr. Parry was recognized as having significant experience in the area of genotoxicity in 1999. To the extent that plaintiffs suggest Dr. Parry was retained as an expert for purposes of this or any other litigation, Monsanto otherwise denies this request.

At this time, Your Honor, we call Dr. Mark Martens by video deposition.

The deposition was taken on April 7th, 2017, in Washington, D.C. The total run time is two hours and 22 minutes. I don't expect to finish that today. Of that, approximately two hours of that is the plaintiffs' and half an hour of it is Monsanto's.

THE COURT: So we will break at 4:30. So just find a natural breaking point at or around 4:30.

Mr. Wright, will you turn off a couple of the lights so we can see the screen. I think that's probably fine.

(Video excerpts from the deposition testimony of Mark Martens played in open court; not reported herein.)

THE COURT: Thank you. All right.

So, ladies and gentlemen, it's 4:30. We are done for the day.

Thank you for your time and attention today. Please do not read, talk about, or otherwise communicate about this case with anyone. And I want you to forget you're a juror when you go home. Have a good evening and we'll see you here tomorrow at 9:00 a.m.

MR. ISMAIL: Your Honor, do you want to give the jury a heads-up about Wednesday's schedule?

THE COURT: There's no change.

9:00 o'clock tomorrow morning. We'll be here tomorrow, Wednesday, and Thursday. So we'll have a full week of evidence.

(Jury excused for the evening recess.)

(Proceedings continued out of the presence of the jury:)

THE COURT: Dr. Weisenburger is up tomorrow.

MR. WISNER: Yes, Your Honor.

THE COURT: Is he all day?

MR. WISNER: All day. And probably into the next day as well.

THE COURT: Okay.

MR. MILLER: Your Honor, if I could, Judge Chhabria wanted me on a phone call tomorrow at 1:00 o'clock. I don't think it will be a long phone call, but I think he set it at 1:00 o'clock so I could be on it. With the Court's permission, if we could do lunch so I could do that, that would be great.

THE COURT: Sure. How long do you anticipate that conversation might take?

MR. MILLER: 15 minutes or less.

THE COURT: Okay, we'll figure it out. Remind me tomorrow as we're going through the morning so I can time our breaks and break for lunch.

MR. WISNER: By stipulation, Your Honor, we move Exhibits 25, 26, 27, and 34 into evidence, and I have a copy for the clerk.

THE COURT: Okay. That's fine. And those were all attached to -- were those all attached to --

MR. WISNER: No, this is from a different depo.

THE COURT: This is from something else?

MR. WISNER: Yes.

THE COURT: Because we haven't started that process yet so as long as you guys agree, that's fine.

MR. WISNER: This is from Reeves.

THE COURT: That's fine.

(Plaintiffs' Exhibits 25, 26, 27, 34 were received in evidence.)

THE COURT: All right. So I'll see you tomorrow at 9:00 a.m.

(Proceedings adjourned at 4:30 p.m.)

State of California)
)
County of Alameda)

We, Kelly L. Shainline and Lori Stokes, Court Reporters at the Superior Court of California, County of Alameda, do hereby certify:

That we were present at the time of the above proceedings;

That we took down in machine shorthand notes all proceedings had and testimony given;

That we thereafter transcribed said shorthand notes with the aid of a computer;

That the above and foregoing is a full, true, and correct transcription of said shorthand notes, and a full, true and correct transcript of all proceedings had and testimony taken;

That we are not a party to the action or related to a party or counsel;

That we have no financial or other interest in the outcome of the action.

Dated: April 8, 2019

Kelly L. Shainline
CSR No. 13476, CRR

Lori Stokes
CSR No. 12732, RPR