Exhibit 13

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION,	)
This document relates to:	) Case No. 16-md-02741-VC
ALL ACTIONS	)

VIDEO DEPOSITION OF
BEATE RITZ, MD, PHD
Los Angeles, California
Monday, September 18, 2017

Reported by:
LISA MOSKOWITZ, CSR 10816, RPR, CRR, CLR,
NCRA Realtime Systems Administrator
JOB NO. 128477

September 18, 2017
9:05 a.m.

Video deposition of BEATE RITZ, MD, PHD, held at the offices of Baum, Hedlund, Aristei & Goldman, PC, 12100 Wilshire Boulevard, Suite 950, Los Angeles, California, before Lisa Moskowitz, California CSR 10816, RPR, CRR, CLR, NCRA Realtime Systems Administrator.

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BY: ELYSE SHIMADA, ESQ.

ALSO PRESENT:
SCOTT MCNAIR, Videographer
LEEMON McHENRY

---------- I N D E X ----------

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LOS ANGELES, MONDAY, SEPTEMBER 18, 2017
9:05 A.M.

THE VIDEOGRAPHER: Good morning. This is the start of tape labeled number 1 of the videotaped deposition of Dr. Beate Ritz in the matter of Roundup Products Liability Litigation. This case is before the United States District Court for the Northern District of California bearing MDL Number 2741 and Case Number 16-MD-02741-VC. This deposition is being held at 12100 Wilshire Boulevard in Los Angeles, California. Today's date is September 18, 2017. The time is approximately 9:05 a.m. 09:04 09:04 09:05 09:05

My name is Scott McNair from TSG Reporting, Incorporated. I'm the legal video specialist. The court reporter today is Lisa Moskowitz also in association with TSG Reporting.

Will counsel please identify yourselves for the record.

MS. FORGIE: Kathryn Forgie for 09:05

the plaintiffs with Andrus Wagstaff.

MR. BAUM: Michael Baum for plaintiffs.

MR. WISNER: Brent Wisner for plaintiffs.

MR. ESFANDIARY: Pedram Esfandiary for plaintiffs.

MR. McHENRY: Leemon McHenry for plaintiffs.

THE VIDEOGRAPHER: On the phone?

MS. FLAHERTY: Yvonne Flaherty, Lockridge, Grindal Nauen for plaintiffs.

THE REPORTER: And the other two counsel for the record on the phone?

MS. FORGIE: Jeff, Mike, you guys? Are you there?

THE REPORTER: Can you please identify yourselves for the video record?

MR. MILLER: Michael Miller and Jeff Travers.

MS. FORGIE: For plaintiffs.

MR. LASKER: Eric Lasker for Monsanto, Hollingsworth, LLP.

MS. SHIMADA: Elyse Shimada for Monsanto, Hollingsworth, LLP.
THE VIDEOGRAPHER: Thank you. Will the court reporter please swear in the witness. 09:06

Beate Ritz, MD, PhD,
called as a witness, having been duly sworn, was examined and testified as follows:

EXAMINATION
BY MR. LASKER:
Q. Good morning, Dr. Ritz.
A. Good morning. 09:07
Q. As you just heard, my name is Eric Lasker. I represent Monsanto. I'll be asking you some questions today.
Have you had your deposition taken before? 09:07
A. Once in, I don't know, 1991 or '2.
Q. I'm sure your attorneys have told you the process, but your deposition is being videotaped, and we have a court reporter. I will try and speak slowly for 09:07

the court reporter's benefit, although I'm not very good at that. I'll warn you. And if we can just wait for the question to be completed before you answer, that makes it easier for the court reporter. Okay? 09:07
A. Yes.
Q. If you have any uncertainties about my question or my question is poorly worded, just let me know. Okay? Great.
Let's start by marking your CV. 09:07
This will be Exhibit 19-1.
(Exhibit Number 19-1 was marked for identification.)
BY MR. LASKER:
Q. So Dr. Ritz, you received your 09:08 medical training in Germany; correct?
A. Correct.
Q. And you received what is identified on your CV as a medical certificate and then a doctoral degree in medical sociology. 09:08
A. Correct.
Q. I'm just trying to understand the terminology here. What is a doctoral degree in medical sociology?
A. It's a PhD equivalent. 09:08

Q. What was your specialty? What was your area --
A. Medical sociology which includes occupational health. So mine was in occupational health. 09:08
Q. Okay. And the medical certificate, is that --
A. That licenses you to be a physician.
Q. Okay. Did you ever -- have you 09:08 ever practiced as a clinical physician?
A. Yes.
Q. Where did you practice?
A. At the University Hospital Hamburg psychiatric department. 09:09
Q. Have you ever provided medical care for patients with -- well, did you ever provide medical care for cancer in patients with cancer?
A. Yes. 09:09
Q. When was that?
A. That was during my final year in medical school at the University of Hamburg pediatrics ward that was filled with children with leukemia and brain tumors. 09:09

Q. And that was somewhere around 1982?
A. '3.
Q. '83.
Other than that, have you provided clinical care for patients with cancer? 09:09
A. No.
Q. You're not an oncologist; correct?
A. No.
Q. You came to UCLA in 1991 to pursue a master's degree and then a PhD in 09:09 epidemiology; correct?
A. No. 1989.
Q. 1989. Thank you.
In 1995, you became an assistant professor of epidemiology at UCLA; correct? 09:09
A. Correct.
Q. One of your responsibilities in that position was advising and mentoring doctoral students; correct?
A. Correct. 09:10
Q. The first doctoral student you mentored was Kurt Straif; correct?
A. Correct.
Q. Had you known Dr. Straif before you became his mentor in 1997? 09:10

A. I knew him as a student. He was a student in the epi department, and he was actually mentored by a different faculty, Dr. Krause, who left UCLA and because of that, Dr. Straif had to be reassigned to another advisor. 09:10

Q. Had you known Dr. Straif back in Germany?

A. No.

Q. Did you continue to have a professional relationship with Dr. Straif after he received his PhD? 09:10

A. Not a professional relationship but a personal one.

Q. Okay. So you and Dr. Straif are friends? 09:10

MS. FORGIE: Objection.

THE WITNESS: I don't know how you would characterize it, but we're collegially affiliated. So he invited me, for example, to spend a visiting year at IARC. 09:11

BY MR. LASKER:

Q. Okay. That's where I was going next; so you anticipated that. 09:11

A. Yeah.

Q. Beyond -- first of all, just so the record is clear, Dr. Straif is now the head of the IARC Monograph program; correct?

A. As far as I understand, yes. 09:11

Q. Was he the head of the Monograph program when he invited you to become a visiting scientist at IARC?

A. No.

Q. What was his position then? 09:11

A. He was a senior scientist in the program, as far as I remember. And he was not the official person inviting me. He just recommended to me that I should come to IARC, and it was Dr. Boffetta who invited me officially. 09:11

Q. What did you do as a visiting scientist at IARC?

A. Well, my role was to work with -- to mentor and work with junior colleagues who were in the epidemiology program. Actually, one of the senior scientists -- we have a very regular exchange of doctoral students who go for internships to IARC. That is actually under the -- not my own 09:11 09:12

students but the students of our cancer research are at UCLA, Dr. Zhang, and one of his former students was actually a member of the epidemiology group at IARC at the time, Mia Hashibe, and she was the one who was helping all the students integrate into the IARC program, and my role as visiting scientist was to actually help her but also mentor a lot of junior scientists there because, at the time, I was considered a senior scientist. 09:12 09:12

Q. So I didn't understand this. UCLA and IARC have a --

A. A mentorship program.

MS. FORGIE: Wait for him to get the question out before you answer, please. 09:12

BY MR. LASKER:

Q. And how long has UCLA had this mentoring program with IARC? 09:13

A. I believe it is as long as Dr. Zhang was a faculty member at UCLA because he came -- he had a time where he, in his own professional career, actually spent time at IARC. 09:13

Q. So when would that -- a year, what year would that program have started?

A. 1997.

Q. Does that continue to the present?

A. I don't believe so because Dr. Hashibe left IARC, and Dr. Zhang is not very active anymore in terms of research. 09:13

Q. Were you paid for your work as a visiting scientist at IARC?

A. I got a stipend that helped me pay for rent. It was not considered pay. 09:13

Q. Did you continue to receive pay from UCLA during that period?

A. I was on a sabbatical officially, and yes, during that sabbatical, you're entitled to payment. 09:13

Q. How long did you work as a visiting scientist at IARC?

A. I started, I think, in August of 2006, and I left to go back to UCLA in July of the next year, 2007. 09:14

Q. I've seen some documents that identify you as also serving during this period as a member of the IARC secretariat; is that right? 09:14

A. Not that I recall that that was an official title, however, I was an observer -- a member of the group that was in charge of putting the 100s volume together or the ideas for the 100s volume, and I was an observer at several events that were led by the Monograph group.

They always have observers from visiting professors, junior scientists, but I was not a member of any of the groups. 09:14

Q. And the Volume 100, what is that?

A. That is -- that was a special memorial volume in which they decided which agents to re-review that they had previously reviewed. So the 100 carcinogenic compounds and groups that were previously reviewed in the 100s volume they decided what to re-review. 09:15

Q. Gotcha.

You were working for IARC during the same years that one of the other plaintiffs experts Christopher Portier was also over at IARC, I believe, working on an advisory group to recommend amendments to the preamble. 09:15

Are you familiar with that?

A. No.

Q. Did you have any dealings with Dr. Portier when you were at IARC?

A. None. 09:15

Q. Do you have any professional relationship with Dr. Portier?

A. None.

Q. Do you have any collegial relationship? If that's the word we use -- 09:15

A. I don't.

MS. FORGIE: Careful there.

MR. LASKER: I'm using her word. Trying to find the right word there.

BY MR. LASKER: 09:16

Q. I take it you did not work on any of the amendments to the IARC preamble?

A. No.

Q. Now, I was looking at your CV, and I don't see it. Maybe it's just an oversight, your work for IARC on your CV. 09:16

Is that listed here, and I just missed it?

A. That was a sabbatical. I don't list every sabbatical I take. 09:16

Q. Okay. So I didn't miss it. It's not on your CV?

A. No.

Q. Okay.

A. There may be some talk -- no. I don't know. 09:16

Q. Have you had any discussion with Dr. Straif about IARC's review of glyphosate?

A. None. 09:16

Q. Have you had any discussion with Dr. Straif about any of your work as a plaintiff's expert in this litigation?

A. None.

Q. Your CV mentions that you are a member or originally were a member of the external advisory committee for the Agricultural Health Study and then in 2005, you became the chair of that committee; correct? 09:16 09:17

A. Correct.

Q. And you're currently still serving as the chair of the AHS --

A. Normally but that committee hasn't met since. 09:17

Q. When was the last time that committee met?

A. I think I was the chair once; so it must have been in 2006 or '7.

Q. Okay. How did you first get appointed to the advisory committee? 09:17

A. I was approached, as far as I recall, by Dr. Alavanja at a professional meeting, and he asked me whether I would be interested in this kind of appointment. 09:17

Q. How were you selected in 2005 to become the chair of the committee?

A. Because the chair stepped down, and they thought they needed somebody else to chair. So they asked me, but it was, at the time, already not clear whether this advisory panel would really have much to do in the future. 09:17

That was one reason why I said yes because I knew it wouldn't be much work. 09:17

Q. For the period 2001 to 2005 then, was that a period where there was more work on the advisory committee?

A. Yes.

Q. What was the role of the advisory 09:18

committee during that period of time?
A. That was a very active time for the cohort because they were in the second phase of going out there and interviewing and trying to interact with the farmers. 09:18
And so from year to year, they would present their progress, but at the same time, they were also using the baseline data that they had collected between 1993 and 1997 to do the first analyses and 09:18 produce the first results that came out of this cohort.
So it was a very, very busy time of investigators presenting first results, presenting first ideas on how to do exposure 09:18 assessments and to bang ideas around, and that's what the advisory committee was charged to do, which is to not only follow the fieldwork and make recommendations that was ongoing but also to evaluate those first 09:19 analyses and results coming out of the study.
Q. So this was during the period of time when the De Roos 2005 publication came out which looked at glyphosate; correct? 09:19

A. Correct.
Q. In your role on the advisory committee, would you, then, have received the initial results of that analysis? Have that presented to you for discussion? 09:19
A. Not necessarily. That was actually up to the authors and depended on whether they wanted input from the advisory panel or certain members of the advisory panel, and I can't remember seeing that manuscript. 09:19
Q. Would the advisory committee review the publications that came out of the AHS after they appeared in the --
A. That was not our task. Our task was really to be there for those who wanted 09:20 a pre-review.
Q. Did the advisory committee consult on the methodologies that were being used by the Agricultural Health Study group during that period in preparing their analyses for 09:20 publication?
MS. FORGIE: Objection.
You can answer.
THE WITNESS: There was not one publication that we would ever review. 09:20

Part of what was done at the advisory panel meetings was present to us studies within the Agricultural Health Study that helped us evaluate the exposure assessment methods. 09:20
I remember presentations by Dr. Curwin, by the NIOSH group that went out and did field measurements, and I also remember presentations by Dr. Acquavella from Monsanto. They had 09:20 a relatively close relationship during that time in trying to evaluate exposures in the field.
BY MR. LASKER:
Q. Do you recall then did you review 09:21 Dr. Acquavella's analyses of urinary biomarkers for glyphosate in other pesticides?
A. We did not review it, but we were made aware of it. 09:21
Q. Did you actually have the opportunity to question Dr. Acquavella about his -- and his team about their analyses?
A. Maybe one or two questions. I 09:21

mean, we are in a room with 35, 50 people, and, you know, if you can get your hand up fast enough, you can ask a question.
Q. Do you recall during that meeting whether anybody raised, from the advisory 09:21 committee, raised any concerns about the validity or reliability of the analysis this Dr. Acquavella was conducting?
MS. FORGIE: Objection.
THE WITNESS: I do not. I cannot 09:21 remember.
BY MR. LASKER:
Q. So that -- you mentioned that was from the period before 2005, and you have one meeting that you recall after 2005, 09:22 sometime in 2006 and 2007. Have you had any activity as a member of or as a chair of the external advisory group for AHS since that time?
A. What would happen is from time to 09:22 time we would get a small report of activities that are ongoing in writing. We would have maybe one or two conference calls where we could ask questions about the ongoing activities, and I've been informed 09:22

that there will be a two-day meeting coming up in February, but I can't attend it because I'm teaching.

Q. Did you have, during that time period, calls addressing the second phase questionnaire to gather more information on exposure information from the cohort? 09:22

MS. FORGIE: Object to form.

THE WITNESS: That was done. There was no more questions about that. 09:23

BY MR. LASKER:

Q. So during the period -- that would have been completed in 2003 or 2004.

A. Yeah, yeah.

Q. Were you advising, or was your committee advising the AHS on the procedures to use during the second phase in gathering additional information from the cohort? 09:23

A. Well, that was already decided prior to them going out in the field; so there was nothing you could change. You don't change methods in the middle of assessments in the field because you get in trouble. 09:23

Q. We'll be talking a little bit later

about the response rate for the exposure assessment for the AHS and how the AHS group has addressed that in their studies.

Were there any discussions with your group about methods that could be used to address the issue of non-responders in phase 2? 09:23

A. Only insofar as they were trying to come up with field methods to get more people to respond. 09:24

Q. Have you had any discussions with any of the Agricultural Health Study scientists regarding any study data on glyphosate and non-Hodgkin's lymphoma?

A. No. 09:24

Q. Have you had any discussions with anyone at the AHS regarding research into pesticides more generally?

A. Oh, yes.

Q. What discussions -- I know this may be a broad topic. I don't know exactly how to break this down. What discussions have you had with the AHS group about conducting pesticide cancer epidemiology? I assume that's the general category. 09:24 09:24

A. That's --

MS. FORGIE: Wait for the question.

THE WITNESS: That's very broad; so the discussions would have been quite broad. 09:25

BY MR. LASKER:

Q. I realized that as I was asking the question. Have you had conversations with the AHS scientists about how to conduct their dose response analyses of pesticides and non-Hodgkin's lymphoma? 09:25

A. No.

Q. Have you had discussions regarding -- with the AHS scientists about how to deal with issues of selection -- potential selection bias in the -- if there is any in the AHS study? 09:25

MS. FORGIE: Object to form.

THE WITNESS: Selection bias would be a differential bias due to loss to follow-up. Are we talking about cancer, or are we talking any outcome? 09:25

BY MR. LASKER:

Q. Cancer.

A. In terms of cancer we would not

necessarily expect selection bias. We would expect selection to -- well, we would suspect loss to follow-up only if we cannot find cancer cases in the registries that were being searched for, and that was actually part of the assessments in the -- when I was in the room at those meetings was what search algorithms they were using broadly to find cancer cases, and they included not only the cancer registries in the States but mortality registries and other means including following up with the participants. So in terms of cancer, we would expect them to have been able to find all the cancers. 09:26 09:26

Q. Did you have any discussions with AHS scientists about the possibility of misclassification -- exposure misclassification bias in the study?

A. The study is a very broad term. The study has many, many sub studies including a Parkinson's study I'm very interested in because that's what I do. And yes, there could be selection bias in that Parkinson's study, and it could be very 09:27 09:27

severe so I'm sure we've had a lot of discussion around that.
Q. Let me back up because you used selection bias, and I thought we were talking about something different but maybe I misstated. I was talking about exposure and misclassification bias. That's a separate issue than selection bias. 09:27
A. Yes.
MS. FORGIE: Wait for a question. 09:27
BY MR. LASKER:
Q. Have you had conversations with AHS scientists about exposure misclassification bias particularly with respect to pesticides? 09:27
A. That was an ongoing discussion that we had at just about every meeting because in pesticide epidemiology, we are generally aware that that's a big problem. Exposure misclassification is always a problem with when you have time varying exposures, and you have lifelong exposure periods that you have to evaluate. So it's not like, for example, I do a lot of pregnancy studies. You have a nine months period, and that's 09:28

rather easy to recall for the women, or you can even sample urine every month from a pregnant woman. You cannot sample urine over lifetime from the farming population of the size of the AHS. So it's an ongoing debate. 09:28
Q. It would be fair to say that the Agricultural Health Study has made significant efforts through the way it interacts with the cohort and the way that it formulates the questionnaires, including with advice from your committee to minimize the potential for exposure misclassification bias? 09:28
MS. FORGIE: Object to form. 09:28
THE WITNESS: That's a very relative term. Again, when it comes to lifelong exposures, misclassification of exposure gets more and more -- to be more and more problem the older the enrollees are and the longer back they have to recall. It also is a big problem if you're not reassessing exposures every single year. 09:29
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BY MR. LASKER:
Q. Did the advisory committee make recommendations to the AHS scientists on methods to address exposure misclassification or potential for exposure misclassification that the AHS scientists did not accept? 09:29
A. I can't recall.
Q. Dr. Matthew Ross of Mississippi State is also a member of your advisory committee for the AHS group; correct? 09:29
A. As far as I remember, yes.
Q. Have you had any conversation with Dr. Ross about glyphosate?
A. No. 09:29
Q. Have you followed the AHS outside of this litigation -- have you followed the AHS's findings with respect to potential risk factors in the agricultural community for non-Hodgkin's lymphoma? 09:30
MS. FORGIE: Object to form.
THE WITNESS: I have been following the AHS over many years. The focus for me was always my Parkinson's interest. However, since Dr. De Roos was a 09:30

candidate for faculty at UCLA, I have been very interested in her publication; so I'm very aware of her publications.
BY MR. LASKER:
Q. When was Dr. De Roos being considered for a faculty position at UCLA? 09:30
A. A few years ago. Two or three years ago right before she went to Drexel.
Q. And through that process, I take it you then reviewed all of her studies for -- 09:30
A. More or less, yes. Especially the ones I'm familiar with.
Q. What different exposures or risk factors has the AHS through its research associated with non-Hodgkin's lymphoma that you can recall? 09:31
A. It has found diesel, and it has -- there's a small risk increase in certain animal husbandry and solvent exposures, but the one that I recall the most is diesel exposures. 09:31
Q. Your CV also mentions that you are a Fellow at the Collegium Ramazzini. I guess you became that in 2007; correct?
A. Correct. 09:31

Q. What is a Collegium Ramazzini?
A. It's a boys' club. That's one reason why I'm not often there. It is a group of occupational and environmentally interested health professionals who are meeting once a year in a small place near Bologna in Italy. Ramazzini was 1700's the first occupational physician credited with finding several occupational disorders or diagnosing them for the first time. So in his honor, this is a society. You can only be invited to become a member, and it has a limited number of members. So only when a member expires or leaves can a new one be inducted. 09:32
BY MR. LASKER:
Q. What is the numerical limit?
A. I think it is 189 for some reason.
Q. Do you know who invited you for membership? 09:32
A. Yes. It was Dr. Phillip Grandjean from Denmark.
Q. Where does -- to the extent that you know the Collegium Ramazzini receive funding for its scientific endeavors? 09:32

A. I'm not sure they even have any scientific endeavors, and I wouldn't know where they're getting their funding from, but certainly they are not paying you to go there. 09:33
Q. Are you aware of that the Collegium Ramazzini has announced the intention to conduct research into glyphosate?
MS. FORGIE: Objection.
THE WITNESS: I have no -- I have not followed them for a while. 09:33
BY MR. LASKER:
Q. So the answer is no?
A. No.
MS. FORGIE: Objection. 09:33
BY MR. LASKER:
Q. Dr. Straif is a Fellow of the Collegium Ramazzini; correct?
A. I think he is, but I'm not really certain. I've never met him there. 09:33
Q. Dr. Blair is a Fellow of the Collegium Ramazzini; correct?
A. I think that's true. Again, I don't recall seeing him there.
Q. And Dr. Portier is a fellow of the 09:33

Collegium Ramazzini; correct?
A. I wouldn't know that.
Q. In 2009, you were elected as a counselor for the International Society for Environmental Epidemiology; correct? 09:33
A. Correct.
Q. What is the role of a counselor for the ISEE?
A. Well, that's kind of like a board member, and what you do is you're on a phone call once a month with all the other members including the president and the president elect and the treasurer, and you're conducting business of the society. 09:34
Q. One of the things that you've done -- at least I see from your CV -- is that you have been a member of the ISEE's conference organizing committee. 09:34
A. That's correct.
Q. What does that committee do? I think it's halfway self-evident but . . . 09:34
A. Yes, it is self-evident. So we are the ones who are reviewing the applications that come in from members for conducting the conference every year, and we also are 09:34

trying to help the conference organizers in every way we can. And we have guidelines for conference organizers. So that's pretty much it.
Q. Okay. In your expert report, you discuss what you describe as some of the peer review that's conducted in connection with abstracts and presentations at the ISEE conferences; correct? 09:34
A. Correct. 09:35
Q. Can you describe that peer review process?
A. Yes. Every year when the conferences are being conducted, we elicit peer reviewers from among the council as well as from the membership. So we have a call for the membership out to nominate peer reviewers for the abstracts and then we appoint the -- the council appoints these peer reviewers with the help of the conference organizers, and they then are tasked with peer reviewing the abstracts. And there are guidelines for that. There is a point system for that, and it's always at least three reviewers who review, and then 09:35

it's being summarized and discussed in the conference committee or better with the conference organizers.

Q. So the abstract obviously is going to be a fairly short document. Does the peer review process involve reaching out and talking to the investigators about their work? What actually is done as part of that peer review? 09:36

MS. FORGIE: Object to form. 09:36

THE WITNESS: What we're trying to do is match the abstracts with people in the specific areas of knowledge so that we have expertise in terms of the outcomes assessed, the exposures 09:36 assessed, the type of studies conducted. So the peer reviewers are not reaching out, but they are to evaluate whether there is enough information to make this a scientifically solid abstract. 09:36

BY MR. LASKER:

Q. And did you attend the ISEE conference in Brazil in 2015?

A. I did.

Q. Did you sit in on the presentation 09:36

or any of the presentations of the NAPP investigators?

A. Unfortunately not.

Q. Dr. Ritz, let's talk about some of -- let's get your expert report as the 09:37 next document. I don't know that we'll be dealing much with your CV so you can set that aside.

(Exhibit Number 19-2 was marked for identification.) 09:37

BY MR. LASKER:

Q. So this will be Exhibit 19-2. Dr. Ritz, on page -- you address some of the methodological issues with epidemiology and epidemiological studies in your report; 09:38 correct?

A. Yes.

Q. I'd like to take you to page 6 and carrying over to page 7 you're discussing what you identify as the null hypothesis; 09:38 correct?

A. Yes.

Q. The null hypothesis is an essential concept in scientific methodology not only in epidemiology but in all areas of 09:38

scientific endeavor seeking to analyze cause and effect; correct?

MS. FORGIE: Object to form.

THE WITNESS: Yes, we generally formulate something of a null hypothesis 09:38 in science, yes.

BY MR. LASKER:

Q. The scientific method is based upon generating a hypothesis and then testing to see if they can falsify -- if that 09:39 hypothesis can be found to be not true; correct?

MS. FORGIE: Object to form.

THE WITNESS: Actually a null hypothesis is one specific hypothesis. 09:39 It's the hypothesis that there's no difference.

BY MR. LASKER:

Q. Right.

A. And that is actually in scientific 09:39 circles being discussed as probably not the best way to go about science all the time. Sometimes you actually want to specify a hypothesis of a certain type of difference.

However, there is a multitude more 09:39

ways of specifying that difference in terms of size or extent so that people can't easily agree to that kind of hypothesis. But one in science could decide to hypothesize something that's not a null 09:39 hypothesis, but the convention is to start with a null hypothesis.

Q. If we are using a null hypothesis, the process of a scientific method is to generate a hypothesis to see if that null 09:40 hypothesis could be shown to be not true; correct?

A. I would not state it that way. We are starting with a null hypothesis, and then we are trying to provide data that 09:40 either confirms or refutes the null hypothesis.

Q. Got it. Better.

In epidemiology and in cancer epidemiology, for example, the null 09:40 hypothesis would be that an exposure being studied is not a cause of cancer; correct?

MS. FORGIE: Object to form.

THE WITNESS: We would, yes. A null hypothesis we would state as no 09:40

difference in risk.
BY MR. LASKER:
Q. Epidemiologists will then design studies to test that null hypothesis; correct? 09:41
A. Well, we are testing the hypothesis whether or not that agent contributes to the disease. The null hypothesis would be that it doesn't.
Q. And when you design an 09:41 epidemiological study, you are designing the study to be able to test that null hypothesis; correct?
A. We can't really -- as I said, we are testing whether an agent adheres or 09:41 whether the exposure to an agent falls under the null hypothesis, or we can generate data that refutes that null hypothesis, yes.
Q. All right. So in designing an epidemiologic study, you are designing the 09:41 study to try and generate data that would at least -- would allow you to test the null hypothesis?
A. That would allow me to test whether there is a difference or not. 09:41

Q. Correct. In epidemiologic studies, the null hypothesis is reflected in an odd ratio or risk ratio of 1.0; correct?
MS. FORGIE: Object to form.
THE WITNESS: Well, that is one 09:42 measure. We are using different measures: odds ratio, risk ratios, rate ratios. And these ratios have point estimates and confidence intervals. The null hypothesis is that, yes, there's no 09:42 difference in the risk among the exposed compared to the risk among the unexposed or the rate in the exposed compared to the rate in the unexposed. And since the ratio measure when there's no 09:42 difference is one, that would be considered no effect.
BY MR. LASKER:
Q. Epidemiologists will then analyze the data to determine whether that null 09:42 hypothesis can be rejected from that data; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Modern epidemiologists would not go out to test 09:43

a null hypothesis or that kind of null hypothesis in the term of statistical testing. What we're trying to do is estimate parameters. So we estimate the parameter of interest which in this case 09:43 is the relative risk, the risk ratio, or the odds ratio.
BY MR. LASKER:
Q. We'll be talking about exactly how to test that. I'm not talking about how 09:43 they would test it, but as a threshold epidemiologists using whatever approach -- and we'll talk about this in a moment. But epidemiologists will analyze the data from their study to determine whether the null 09:43 hypothesis can be rejected; correct?
MS. FORGIE: Objection. Asked and answered.
You can answer it again.
THE WITNESS: Again, I would not 09:43 formulate it in this way. It's an estimation problem. We are trying to estimate a relative risk, a rate ratio, or an odds ratio which are parameters that tell me something about the risk in 09:43

the exposed compared to the risk in the unexposed. Along with that goes statistics, but, in essence, we are estimating parameters.
BY MR. LASKER: 09:44
Q. The process of estimating parameters in epidemiologic study is to determine whether that data would provide evidence against a null hypothesis; correct?
MS. FORGIE: Object to form and 09:44 asked and answered.
THE WITNESS: Again, I would want to estimate this parameter and then also see in statistical terms how informative this parameter is. 09:44
BY MR. LASKER:
Q. Right. And the -- what you're looking for with respect to that parameter is whether or not the data you are analyzing would exclude the null hypothesis, if you're 09:44 going to reach a causation opinion; correct?
MS. FORGIE: Object to form, asked and answered.
THE WITNESS: There's a lot more to that than just a null hypothesis. 09:44

There's a lot more that we're doing in epidemiology to convince ourselves that there is causation.

BY MR. LASKER:

Q. That's fair enough. One step in the process to determine whether or not there is causation through an epidemiologic study is whether or not the data is -- allows one to exclude the null hypothesis; correct? 09:45

MS. FORGIE: Object to form, asked and answered.

You can answer it again.

THE WITNESS: Again, we are trying to estimate parameters. These parameters have point and interval -- point and interval estimates, and a lot more goes into evaluating the validity of that parameter.

BY MR. LASKER: 09:45

Q. I agree with that, and we'll be talking about that. But the purpose of determining the point estimate and the parameters for the statistical analysis part of that -- and we'll talk about the other 09:45

parts as well -- is to determine whether or not at that step the null hypothesis of 1.0 is at least not due to chance. Is that fair?

MS. FORGIE: Objection. Wait. 09:46

Object to form and asked and answered.

You can do it again.

THE WITNESS: Chance is one -- is just one criterion we are considering as epidemiologists, and I teach bias 09:46 analysis in the basic methods class at UCLA. What I teach my students is that what we have to make sure is that there's no bias and that before everything else we are ever considering. 09:46 So I would not even consider data unless we would go through a rigorous analysis of all the biases.

BY MR. LASKER:

Q. Fair enough. In your analysis of 09:46 the issues of chance, the issues of bias, the issues of confounding, when you're looking at all of those issues together, what you are trying to, as an epidemiologist, is to determine whether or 09:47

not those factors can be at least addressed efficiently so that together that would allow you to determine that the null hypothesis has been rejected in that study. Is that fair? 09:47

MR. LASKER: Object to form.

THE WITNESS: I do not formulate my research ever in that way. I'm estimating parameters, and I'm assessing validity of studies. 09:47

BY MR. LASKER:

Q. What would you need to -- what steps would you need to go through then in an epidemiological study in analyzing the issues of chance and bias and confounding to 09:47 reach a conclusion in your mind that that study demonstrates a positive association between the exposure at interest and the outcome at interest?

MS. FORGIE: Objection to form. 09:48

THE WITNESS: That is a very long lecture. I don't know whether we want to have it here. It takes me ten weeks and four hours a week to do that. So the short form is that you start with 09:48

the study design, that you start with the exposure assessment validity, that you start with the outcome assessment validity, that you then go into a sample size, exposure prevalence, any kind of 09:48 bias you can think of, and once you have wrapped it all together, that's when you're doing a lot of sensitivity analyses in your data to convince yourself that no way -- no matter how 09:48 you look at your data, there is a signal.

BY MR. LASKER:

Q. And I think that you mentioned this -- you may have mentioned it in your 09:48 report. I know you mentioned it in some of your class materials -- that the fundamental question that an epidemiologist must ask before reaching a causation opinion is is there any other way of explaining the set of 09:49 facts before us, is there any other answer that is equally or more likely than cause and effect; correct?

MS. FORGIE: Object to form.

THE WITNESS: We generally like to 09:49

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challenge each other. Epidemiologists are extremely critical of their own work and that of their colleagues. So we are asking many, many questions trying to debunk a positive result that we might be seeing in a study. We're coming up with causal models, with -- yeah, bias analyses, sensitivity analyses, and after we've done all of that, there might be a positive association; there might not be a positive association. Whether that's causal, we would usually want more than one study to decide. 09:49 09:49

BY MR. LASKER:

Q. And the underlying -- the fundamental question that you're trying to answer when you look at an epidemiologic study or a body of epidemiologic literature is whether there is any other way of explaining the facts before you other than cause and effect; correct? 09:50 09:50

A. That would be any one way because there's always one way or another in any type of study that I can think of that you can find alternative explanations, but what 09:50

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you are looking for then is just as consistent a pattern that would explain everything else.

Q. And if you are not -- if you find that there is some other explanation that could explain the findings, then you would not be able to reach an opinion of cause and effect. Is that fair? 09:50

MS. FORGIE: Object to form.

THE WITNESS: That would depend. So I would want to see that -- there could be an alternative explanation in one study but not in another. So what we would like to see is studies done on different continents, in different counties, by different investigators with different methods. If they all show the same results, then I'm pretty happy because there's probably not one explanation that explains it away. 09:50 09:51 09:51

BY MR. LASKER:

Q. The null hypothesis in this case is that glyphosate is not associated with non-Hodgkin's lymphoma; correct?

A. It's either glyphosate or 09:51

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glyphosate-related formulations.

Q. For epidemiology, it would actually be glyphosate-based herbicides; correct?

A. Correct.

Q. There are no epidemiology studies that are just pure glyphosate. It's all the formulate herbicide product? 09:51

A. Epidemiology is done in the real world. So what is out in the real world is what we're studying. 09:51

Q. Okay. And the question, then, on to the scientific method and the question for you, I take it, in this case is whether the epidemiologic studies provide data that would allow you to exclude -- well, strike that. 09:52

You have reviewed, as part of your work in this case, IARC's assessment of the glyphosate epidemiology; correct?

A. I have read that monograph, yes. 09:52

Q. And in your expert report -- I think it's on page 16 -- it's actually the last sentence on page 16, you state that you "concur with the IARC conclusions after conducting my own independent analysis of 09:52

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the studies included in the IARC review"; correct?

A. Yes, that's what it says.

Q. Okay. And that's the opinion that you are -- you'll be presenting in this litigation; correct? 09:53

A. I will be presenting my own conclusions.

Q. And your own conclusions concur with the IARC's conclusions; correct? 09:53

MS. FORGIE: Object to form.

THE WITNESS: It concurs with the overall IARC conclusions.

BY MR. LASKER:

Q. And just to be clear, when you're talking about the IARC's conclusions in your report, you're talking about IARC's conclusions with regard to epidemiology; correct? 09:53

MS. FORGIE: Object to form. 09:53

THE WITNESS: I am meaning the overall IARC conclusions.

BY MR. LASKER:

Q. Okay. In this section in your report where you state that you concur with 09:53

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IARC's conclusions after conducting your own independent analysis of the studies, first of all, what studies did you review in connection with your work on this case?
A. What studies did IARC review? 09:53
Q. No, did you review. Because you state, "After conducting my own independent analysis of the studies included in the IARC review," which studies are we talking about there? 09:54
A. That overlap with IARC's? They should be all in IARC plus I looked at several others.
Q. But IARC looked at studies dealing with genotoxicity and dealing with 09:54 toxicology and all the like.
A. Yes.
Q. Did you review the genotoxicology studies that IARC reviewed?
A. I did review several papers on 09:54 genotoxicity as well as animal studies, yes.
Q. And did you conduct an analysis, your own independent analysis of the animal toxicology studies?
A. As far as I'm able to do that, I 09:54

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did.
Q. That's not your area of expertise, I take it?
MS. FORGIE: Objection. Object to form. 09:54
THE WITNESS: Well, in effect, I'm a member of the interdisciplinary program in molecular toxicology at UCLA. So I teach toxicologists. So yes, I do know how to read toxicology literature. 09:55
BY MR. LASKER:
Q. With respect to the conclusions that can be reached with respect to the animal toxicology studies, would you defer to the other experts that have been put 09:55 forth by the plaintiff's counsel on those issues?
MS. FORGIE: Object to form.
THE WITNESS: I'm sure that a toxicologist can read these papers in 09:55 different ways, but since I am -- I have been working with toxicologists for 25 years. I'm a member of this teaching program, I would say that I have a certain ability to draw my own 09:55

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conclusions. Plus I'm medically trained, and I know animal pathology because it's very close to human pathology.
BY MR. LASKER: 09:55
Q. So if I were to ask you questions about the Sugimoto rodent study, would you be in a position to answer those questions here today?
MS. FORGIE: Object to form. 09:56
THE WITNESS: You would have to show me those papers, and I would tell you.
BY MR. LASKER:
Q. In your expert report up until the 09:56 line -- up until page 16, you do not discuss any studies other than the epidemiologic studies; correct?
A. Correct.
Q. And in your discussion on page 16 09:56 when you're talking about the conclusions that IARC reached, you are talking about IARC's -- the only thing you discussed is IARC's conclusion with regard to the epidemiology; correct? 09:56

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MS. FORGIE: Object to form.
THE WITNESS: Well, as a scientist, you read everything, and as a scientist, I did go back to the toxicology and genotoxicity literature, and I did read 09:56 the IARC Monograph on that. So when I come to a conclusion, it's in the totality of everything I have reviewed.
BY MR. LASKER:
Q. I understand that, but your expert 09:57 report in discussing the IARC conclusions that you concur with, the only conclusions that you discussed up to this point in your report are IARC's conclusions with respect to the epidemiology; correct? 09:57
MS. FORGIE: Object to form. Asked and answered.
You can answer it again.
THE WITNESS: Again, I cannot exclude what I know and what I've read 09:57 and what I've evaluated. So even if I just refer in this report to the epidemiology, which, of course, I consider myself most an expert, when I make that comment, I'm referring to the 09:57

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whole IARC conclusion which included the toxicology and the genotoxicity.
BY MR. LASKER:
Q. Do you concur with IARC's conclusions with respect to the epidemiology? 09:57
MS. FORGIE: Object to form.
THE WITNESS: Well, IARC's conclusions are IARC's conclusions. They are very categorical. As a scientist, I wish it wasn't as categorical, and I may or may not confer with the way they are drawing these categorical conclusions. I think the epidemiology is quite strong. 09:57 09:58
BY MR. LASKER:
Q. Let me be clear, though. When you state in your expert report on page 16 that you concur with the IARC's conclusions, do you concur with IARC's conclusions with respect to the epidemiology? 09:58
MS. FORGIE: Object to form and asked and answered.
You can answer it again.
THE WITNESS: Well, I'm concurring 09:58

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here with the overall IARC conclusion.
BY MR. LASKER:
Q. I understand that, but that's not my question.
MS. FORGIE: Wait, wait. 09:58
BY MR. LASKER:
Q. When you state here that you are concurring with the IARC's conclusions, you state that at page 16 of your expert report, after talking to the epidemiological literature, my question to you is: Do you concur with the IARC's conclusions regarding the glyphosate epidemiology? 09:58
MS. FORGIE: Object to form, asked and answered twice before. 09:58
You can answer it again.
THE WITNESS: IARC drew conclusions based on three criteria. I read the IARC Monograph. I went back to some of the literature on the genotoxicity and on the animal studies, and I concur with IARC's conclusions. 09:59
BY MR. LASKER:
Q. Okay. Again, I want to be clear for the record so that the court understands 09:59

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and the answer can be yes or no. That's obviously your answer.
With respect to IARC's conclusions, with respect to the epidemiological literature of glyphosate, and you know that IARC separately analyzed the epidemiology; correct? 09:59
MS. FORGIE: Object to form.
THE WITNESS: IARC has several groups that are evaluating pieces of science. One is an epidemiology group. One is a genotoxicity -- one is a mechanistic group. Genotoxicity is part of it. One is an animal group. Each of them evaluate the literature independently, come up with conclusions, but then they are meeting together and discussing with each other the literature and possible conclusions from it. 09:59 09:59 10:00
So every scientist in the room gets to know what the other group is doing and how they are reaching possible conclusions, and they may disagree. A toxicologist may disagree with an 10:00

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epidemiologist or the epidemiologist group and vice versa. But --
BY MR. LASKER:
Q. I understand --
MS. FORGIE: Wait let her finish, please. 10:00
THE WITNESS: In the end, they have to come together with a conclusion, and the conclusions are very categorical, and they are balance of evidence type of conclusions. 10:00
BY MR. LASKER:
Q. I understand that. But my question to you is specific to the epidemiology subgroup in IARC, and they reached a conclusion with respect to the epidemiological literature; correct? 10:00
MS. FORGIE: Objection. Asked and answered.
You can answer it again. 10:00
THE WITNESS: Actually, the epidemiology group alone isn't who comes up with these conclusions. It is everybody in the room at the overall meetings who agrees on these. 10:00

BY MR. LASKER:
Q. Okay. And everybody in the room came to a conclusion with respect to the epidemiologic literature; correct?
MS. FORGIE: Object to form. 10:01
THE WITNESS: They came to a balanced evaluation that then was put into the Monograph and got a category number which is 2A possible carcinogen.
BY MR. LASKER: 10:01
Q. Okay. And that is the overall assessment of glyphosate. I understand that. There is also a separate assessment in the Monograph for the epidemiology, and there's a separate assessment for the animal 10:01 toxicology, and there is a separate assessment for the mechanisms; correct?
A. Yes.
Q. What I am asking you is specific to the conclusion that IARC reached with 10:01 respect to the epidemiology. Okay?
MS. FORGIE: Objection.
THE WITNESS: Again, the epidemiology group made their conclusion. I'm not going to question 10:01

their conclusion. I make my own conclusion, but my conclusion as a scientist is based on reviewing all of the literature. I'm more than an epidemiologist. I have medical 10:02 training, and I have been working with toxicologists and animal experimentalists for 25, 30 years.
BY MR. LASKER:
Q. Right. I understand all of that, 10:02 but my question for you is specific to the epidemiology. The IARC working group came to a conclusion that the glyphosate epidemiology with respect to non-Hodgkin's lymphoma fit into their category of limited. 10:02
You understand that; correct?
MS. FORGIE: Object to form. Asked and answered.
You can answer it again.
THE WITNESS: I understand the 10:02 categories that IARC is using, and they have some unfortunate language including the word "limited" because it's not -- it's a common language word that is very easy to misunderstand. 10:02

BY MR. LASKER:
Q. Okay. Well, let's just be clear on what IARC means by "limited" with respect to epidemiology.
IARC defines limited as: "A 10:02 positive association has been observed between glyphosate" -- "between exposure to glyphosate in this instance and NHL for which a causal interpretation is credible but chance, bias, or confounding cannot be 10:03 ruled out with reasonable confidence."
Correct?
A. Correct.
MS. FORGIE: Object to form.
BY MR. LASKER: 10:03
Q. And IARC determined that the glyphosate epidemiology -- epidemiologic literature fit within that definition; correct?
MS. FORGIE: Object to form, asked 10:03 and answered.
You can answer it again.
THE WITNESS: The working group gave the label 2A which is this kind of definition, yes. 10:03

BY MR. LASKER:
Q. Okay. Let's just be clear about this. 2A is the overall assessment. We're talking about the epidemiologic studies.
A. Uh-huh. 10:03
MS. FORGIE: Wait for a question.
BY MR. LASKER:
Q. With respect to the epidemiologic studies, IARC concluded for glyphosate and non-Hodgkin's lymphoma that a positive 10:03 association has been observed for which a causal interpretation is credible but chance, bias, or confounding cannot be ruled out with reasonable confidence; correct?
MS. FORGIE: Object to form, asked 10:04 and answered.
You can answer it again, but you're getting --
THE WITNESS: That is --
MS. FORGIE: Wait, let me finish. 10:04
You're getting to a point where you're badgering the witness.
THE WITNESS: That's the IARC definition.
///

BY MR. LASKER:
Q. And you state in your expert report -- and I'm just trying to understand what this means -- you state in your expert report that you concur with the IARC conclusions. 10:04

My question to you -- and the answer can be yes or no -- is whether you concur with IARC that for glyphosate and non-Hodgkin's lymphoma and the epidemiological studies, a positive association has been observed for which a causal interpretation is credible but chance, bias, or confounding cannot be ruled out with reasonable confidence? 10:04

MS. FORGIE: Object to form. Asked and answered. Also mischaracterizes the IARC, as you know, the IARC categories.

THE WITNESS: Again, on page 16 of my document what I'm referring to is the overall IARC conclusion. 10:04

BY MR. LASKER:
Q. My question to you is, independent of whatever you mean or you're interpreting the sentence on page 16 in your expert 10:05

report to mean, the question to you is very simple. Do you agree with IARC in its classification of the epidemiological literature for glyphosate and non-Hodgkin's lymphoma that a positive association has 10:05 been observed for which a causal interpretation is credible but chance, bias, or confounding cannot be ruled out with reasonable confidence?

MS. FORGIE: Object to the form, 10:05 asked and answered. Also you're deliberately misreading the IARC categories.

THE WITNESS: Again, IARC has unfortunate wording in their categories. 10:05 One of the unfortunate words is "limited." They are expanding on it in a way that to non-epidemiologists is problematic, and I'm not going to argue with IARC about this. 10:05

BY MR. LASKER:
Q. My question is not about use of the word "limited" or whatever word they use. My question is the substance of what IARC concluded, and you may agree or you may 10:06

disagree, but you haven't told me yet which of those things it is. That's all I'm trying to find out. It's a simple question, and if we need to mark this and the judge can answer, that's fine. We'll do that. 10:06 But it's a simple question, yes or no.

Do you agree with IARC in its review of the glyphosate and Roundup epidemiological literature for non-Hodgkin's lymphoma that a positive association has 10:06 been observed for which a causal interpretation is credible but chance, bias, or confounding could not be ruled out with reasonable confidence?

MS. FORGIE: Objection. Object to 10:06 the form. You're mischaracterizing and misreading the categories of IARC, as you know, and it's been asked and answered at least five or six times now.

You may answer it again. 10:06

THE WITNESS: Again, IARC does their evaluation the way they do. I'm a scientist. I did my independent evaluation. I used my words. They used theirs. I may not agree with the kind 10:07

of wording they are using. I think the epidemiology is extremely strong.

BY MR. LASKER:
Q. Do you believe based upon your review of the epidemiological literature for 10:07 glyphosate and non-Hodgkin's lymphoma that a positive association has been observed for which a causal interpretation is credible but chance, bias, or confounding could not be ruled out with reasonable confidence? 10:07

MS. FORGIE: Object to form. Asked and answered.

You can answer it again.

THE WITNESS: My reading of the literature is that the epidemiology is 10:07 very strong especially since there was additional literature since IARC conferred in 2015.

BY MR. LASKER:
Q. Okay. Is your analysis, then, of 10:07 the epidemiological literature, your conclusions, informed by epidemiological data that has come out subsequent to the IARC working group meeting?
A. I reviewed the NAPP, yes. 10:08

Q. Okay. And so in reaching your conclusions about the strength of the epidemiology for glyphosate and non-Hodgkin's lymphoma -- strike that.

Let me just circle back. Including your analysis of the glyphosate literature and the NAPP data, do you believe that a positive association has been observed between exposure to Roundup and non-Hodgkin's lymphoma for which a causal interpretation is credible but chance, bias, or confounding could not be ruled out with reasonable confidence? 10:08

MS. FORGIE: Object to form. Asked and answered.

You can answer it again.

THE WITNESS: I believe there's a positive association for which causal association is quite credible.

BY MR. LASKER: 10:08

Q. Do you believe that chance, bias, and confounding can be ruled out with reasonable confidence?

MS. FORGIE: Objection. Asked and answered. 10:09

You can answer it again.

THE WITNESS: Again, I think that a causal association is quite credible, and I, as a scientist who is not just an epidemiologist, put this in context with everything I know, and I agree with IARC that it's a 2A. 10:09

BY MR. LASKER:

Q. My question, though, is with respect to the epidemiologic literature. With respect to the epidemiologic literature for the glyphosate and non-Hodgkin's lymphoma, do you think that chance, bias, or confounding can be ruled out with reasonable confidence? 10:09

MS. FORGIE: Object to form, asked and answered. This is like the tenth time.

You can answer it again.

THE WITNESS: Okay. I think the epidemiology is quite strong. I think that there is enough reason to make causal associations. However, I put this in the context of the animal data and the mechanistic data. As a 10:09

scientist, I cannot split my mind into three different parts, and that's also not what IARC does.

IARC sits in a room and discusses this with everyone and comes to their conclusion overall. However, there's additional data that came out since IARC met, and that strengthens the evidence. 10:10

BY MR. LASKER:

Q. Let's talk about chance. 10:10

MS. FORGIE: If you're at a reasonable breaking point, just let us know.

MR. LASKER: Sure. How long have we been? Over an hour? 10:10

MS. FORGIE: An hour and ten minutes.

MR. LASKER: That'll be fine.

THE VIDEOGRAPHER: We're off the record at 10:10 a.m. 10:10

(Recess taken from 10:10 a.m. to 10:27 a.m.)

THE VIDEOGRAPHER: We are back on the record at 10:27 a.m.

///

BY MR. LASKER:

Q. Hello, Dr. Ritz. During the break I was looking through your expert report, and I did not see any mention in your report about any of the animal cancer bioassays regarding glyphosate. Am I correct that there's no mention of those animal cancer bioassays in your expert report? 10:29

MS. FORGIE: Object to the form.

THE WITNESS: Well, they are mentioned, but I am not critiquing them in the way that I would critique an epidemiology study. But I certainly reviewed them. 10:29

BY MR. LASKER: 10:29

Q. Can you point in your expert report where you mentioned any of the animal cancer bioassays?

A. Under biologic plausibility and where I say what I searched. Where is that? 10:29

Q. I think that's your literature review.

A. Literature search, yeah.

Q. Okay. So let's start with the biological plausibility because I read that 10:29

through a number of times. Maybe I missed it. There are some discussions of a handful of genotoxicity studies, and you cite them. But I don't see mentioned anywhere in these two paragraphs of the animal cancer bioassays. Is that correct? 10:30

MS. FORGIE: Object to form.

THE WITNESS: Well, the animal studies I mention on page 25.

BY MR. LASKER: 10:30

Q. Which animal studies?

A. Animal experiments.

Q. With regard to cytotoxic and genotoxic effects. I see that. Where do you mention any animal cancer bioassays? 10:30

A. That says models. Correct. What are you referring to now?

Q. I'm asking if there's any mention anywhere in this section of biological plausibility to an animal cancer bioassay 10:30 because I'm not seeing it.

MS. FORGIE: Object to the form.

THE WITNESS: Well, has been confirmed by laboratory experiments listed above is what I was referring to, 10:31

and the listed above are mentioned in my search algorithm.

BY MR. LASKER:

Q. First of all, the listed above, just so we're clear in the section of 10:31 biological plausibility, is referring to studies of genotoxicity and oxidative stress; correct?

MS. FORGIE: Object to the form.

THE WITNESS: No, that's a compound 10:31 sentence, and what I was referring to here is, one, the oxidative stress and genotoxicity as a mechanism and, two, the lab experiments that also confirmed carcinogenicity. 10:31

BY MR. LASKER:

Q. Can you point anywhere -- first of all, in biological plausibility -- we'll go to your literature search as well, but anywhere in biological plausibility in those 10:32 two paragraphs where you mention an animal cancer bioassay?

A. To me the lab experiments are exactly that. That's what they mean.

Q. You state, "The lab experiments 10:32

listed above," and the lab experiments listed above are dealing with cytotoxic and genotoxic effects.

MS. FORGIE: Wait. Is there a question? 10:32

BY MR. LASKER:

Q. Where is there a reference anywhere in these two paragraphs to an animal cancer bioassay?

A. No, the listed above does not refer 10:32 to the mechanisms. The listed above is in terms of the whole document.

Q. Your whole expert report?

A. Uh-huh.

Q. And you believe that you mentioned 10:32 the animal cancer bioassays in your literature search?

A. Yes.

Q. Let's go to the literature search then. Now, the literature search, just so 10:32 the record is clear is at pages 8 and 9 which is some 16 -- 15 or 16 pages before that sentence in the biological plausibility section; correct?

A. I can't see it right now. Oh, 10:33

here. Yes. Page 8. It starts on page 8.

Q. Where in pages 8 and 9 do you mention animal cancer bioassays?

A. Animal and mechanistic literature. It's on page 9. 550 articles for animal and 10:33 mechanistic literature and 600 citations for cancer. So that includes the oncology of animals.

Q. And the bracket after that says, "Most citations were not immediately 10:33 relevant to the present question due to their focus on topics such as effects in fish resulting from runoff, effects on present pregnancy and child development, or effects on other cancer types." 10:33

Do you see that?

A. Yes.

Q. In your discussion of the literature search, you stated that you were looking to obtain all published studies on 10:33 the relationship between non-Hodgkin's lymphoma and glyphosate; correct?

A. Yes.

Q. And --

A. And ingredients. The active 10:34

ingredient in Roundup. So it included Roundup.

Q. And your statement to then is that this reference to the fact that you conducted a literature search that yielded over 550 articles for animal an mechanistic literature was a disclosure that you had reviewed the animal cancer bioassays and were rendering an opinion on them in this case? 10:34

MS. FORGIE: Object to the form.

THE WITNESS: This disclosure means that yes, everything that's out there in the literature I am willing and able to look at and select from and form my opinion on. That's what I do as a scientist.

Actually as a scientist I often spend Sundays doing exactly this, searching the literature broadly to find animal and other types of studies that then give me an hint in terms of what I'm doing as an epidemiologist, and it's great fun. I like it.

///

BY MR. LASKER:

Q. First of all, is it your understanding that you will be proffering any opinions in this case with respect to animal cancer bioassays? 10:35

MS. FORGIE: Object to the form.

THE WITNESS: Well, my -- what I understand is that I'm here as an expert epidemiologist but also as a scientist. As an expert epidemiologist, I rendered you with my evaluation of the epidemiology. As a scientist I'm curious. I go beyond epidemiology. I look at other types of literature. And I disclosed this here because I was told that I'm supposed to disclose that.

MR. LASKER: Okay. For the record we'll state there is nothing in this expert report that mentions an animal cancer bioassay. There is no disclosure as required under the federal rules of any opinion being proffered on animal cancer bioassays, and unless counsel is here to represent that this witness will not be offering opinions with respect to

animal cancer bioassays, we will petition the court for a second deposition of this witness because we were not prepared to question the witness on those issues because of the expert report she submitted. And we would also move to strike because those opinions have not been properly disclosed.

MS. FORGIE: Well, we're not going to agree to a second deposition, of course. I would say she clearly has stated in there that she has looked at over 550 articles for animal and mechanistic literature. There's another reference in there about the effects in rodents of glyphosate and she's talked about the CARC report and the IARC Monograph all of which, as you well know, do discuss animal literature.

MR. LASKER: Well, to be quite clear, that is not what her expert report is, and the judge will be able to read her expert report; so we don't need to debate that. But my question to

you just so I understand -- we have to have motions practice. Is it plaintiff's intention to proffer Dr. Ritz to offer expert opinions with regard animal cancer bioassays?

MS. FORGIE: She intends to give her opinion --

MR. WISNER: Objection. Kathryn, you don't have to answer questions in a deposition. Are we off the record?

MR. LASKER: We are on the record.

MR. WISNER: You can't question attorneys. That's ridiculous. Let's go off the record if you want to ask that question.

MR. LASKER: I certainly can. If we have to get on record with the court and call the court right now, we can do that as well. I need to know right now because I'd like to move on. If the plaintiffs' counsel are not willing to state on the record that Dr. Ritz will not be offering opinions on animal cancer bioassays, then we'll have an issue with the court including a motion

to strike and a motion for leave to seek additional deposition.
MS. FORGIE: You can bring whatever motions you want. You can bring whatever motions you want. She's made it very, very clear that she has expertise in toxicology. You have a copy of her CV. She's talked about studies and the effects in rodents of glyphosate which for whatever reasons you haven't found. She's talked about the IARC Monograph. She's talked about the CARC report. She's talked about the 550 articles on rodents, and she's talked about the fact that she intends as a scientist in epidemiology to look at the totality of sciences, and that's exactly what's in her report. Make whatever motions you want to make. I'm not going to argue about this with you.
MR. LASKER: Just to be clear, the statements in her report with respect to animals which you want to talk about are specific to genotoxicity and cytotoxicity. They do not mention

cancer bioassays, and the 550 articles that you are referencing are the ones talks she about from her initial search which she excluded.
MS. FORGIE: I'm not going to argue with you.
MR. WISNER: Objection. How are you testifying? What's going on here?
MR. LASKER: We will file a motion with the court as necessary to strike this witness' testimony and also to seek a second deposition.
MS. FORGIE: You do whatever you think is appropriate. She has clearly stated in her expert report that she intends to give full opinions including all kinds of science.
MR. LASKER: We will submit and, in fact, the judge has a full expert report in front of him, and he can look at that himself.
BY MR. LASKER:
Q. Dr. Ritz, in your report you provide a definition of a number of terms that epidemiologists use to try to address

the issue of chance; correct?
A. Uh-huh. There are definitions in there in terms of chance and bias, yes.
Q. We'll get to bias. I want to talk about the terms you identify with respect to chance. You provide definitions of the terms "P-value" I believe on page 11 in your report; correct?
A. It's the -- the title says "Statistical Significance," but the P-value is mentioned.
Q. Okay. And you explain in your expert report -- and we're going to get into some of the issues with this, but epidemiologists at least present P-values in trying to address the issue of whether or not a reported odds ratio or relative risk might be due to chance; correct?
MS. FORGIE: Object to form.
THE WITNESS: Epidemiologists are trained -- modern epidemiologists -- and those are the ones who drive the methods in epidemiology -- are trained to at least rely on one parameter. P-value is one parameter.

BY MR. LASKER:
Q. Okay.
A. So epidemiologists are taught what a P-value is and how to evaluate it, but they're also taught never to use just a P-value to evaluate a study or chance.
Q. And that's what I'm going to be getting to right now in my next questions. You mention in your report at pages 11 to 12 that the -- there is a convention of using a P-value of less than .05, but some studies will use P-values such as less than .01 or P less than negative 10 to 7 which is one in 10 million; right?
MS. FORGIE: Object to the form.
THE WITNESS: So what is the question?
BY MR. LASKER:
Q. It is correct that epidemiologists in various studies will use different P-values including P less than .05 but sometimes P less than .01 or P less than 10 to negative 7; correct?
MS. FORGIE: Object to the form.
THE WITNESS: These type of

P-values have been defined and used in studies, but a P-value has a very different meaning depending on the type of test you are conducting. For example, there are test of pair-wise comparisons. There are tests of trends. There are tests of heterogeneity. There are many, many testing situations in which we use P-values, and they have a very different meaning. 10:42

BY MR. LASKER:

Q. One of the articles that you use in teaching your epidemiology students about P-values is an article by Charles Poole entitled "Low P-values or Narrow Confidence Intervals: Which are More Durable?" Correct? 10:42

A. Yes, I love that article.

Q. Good. I have some questions about that. This will be Exhibit 19-3. 10:42

(Exhibit Number 19-3 was marked for identification.)

BY MR. LASKER:

Q. In this article, Dr. Poole, as you explain in your report, in your expert 10:43

report, talks about the fact that a P-value -- and this is on page 293, but you've been using this article in your teaching for a long time. I think you probably know better than I do. 10:43

Dr. Poole mentions that a P-value cannot be read as a probability of obtaining a particular result if there is no true association between an exposure and disease; correct? 10:43

A. Where is that?

Q. I may be paraphrasing but hold on a second. Well, let me just ask it from your report because you state this as well. I think it's in here somewhere, but I'm not 10:44 going to find it as quickly. You state in your expert report that a P-value should not be interpreted as a probability that glyphosate -- in this instance, glyphosate causes NHL; correct? 10:44

MS. FORGIE: Object to the form.

THE WITNESS: I would never use a P-value to say anything about causation. A P-value is a parameter, one of many types of parameters we are using in 10:44

evaluating data in order to reach causal conclusions, but it's really just one. It is a knee-jerk reaction in the medical field unfortunately, and that's what this article is all about, to just 10:44 look at P-values and not the data overall to draw conclusions on the validity or reliability of data and come to a conclusion.

And at UCLA we are taught not to do 10:45 that, and we are teaching our students not to do that.

BY MR. LASKER:

Q. And you agree that it is not proper scientific methodology to point to a P-value 10:45 alone as providing evidence that data -- of the data being analyzed substantiates a conclusion of causation?

A. Well, a P-value alone is nothing any epidemiologist worth their salt would 10:45 consider for coming to causal conclusions. What we do is we look at the data overall in the context of the study design, the biases, the size of the study, the effect we are trying to estimate, the effect size. 10:46

So a P-value could be highly statistically significant, and that 10 to the minus 7 is one of those genomic studies have P-values of 10 to the minus 10, and still the effect size is an odds ratio of 10:46 1.03. So that gene contributes 3 percent increase to a disease. Is that meaningful clinically? Can we do something with that? Is that even useful? We need to debate that. But the P-value is the P-value. It's 10:46 10 to the minus 10, and it's huge. Does it point to something? We need a lot of other reasoning to make use of that.

Q. I think one of the things that Dr. -- first of all, let me make sure that 10:46 I'm clear. The -- if a test result -- a test statistic results in a P-value of .05, that does not mean that there's only a 5 percent likelihood that the null value is correct; correct? 10:47

MS. FORGIE: Object to the form.

THE WITNESS: A P-value doesn't refer to a likelihood. That's a likelihood ratio test.

///

BY MR. LASKER:
Q. So where the test on a glyphosate and carcinogenicity, a P statistic of .05 does not mean that there is a 95 percent chance that glyphosate caused the observed cancers; correct? 10:47
A. It means that if you repeat a trial a hundred times, 95 percent of the time you may find a result as large or larger than what you're seeing. 10:47
Q. Okay. But my question was a little bit different. A P-value of .05 in a glyphosate cancer study does not mean that it is 95 percent likely that glyphosate caused the observed cancers; correct? 10:47
MS. FORGIE: Object to the form. Asked and answered.
Go ahead.
THE WITNESS: That was a double negative; so I have to restate this. A 10:48 P-value alone will not be used for causal evaluation, and a P-value of .05 means that if a hundred times I repeat this experiment in the same population, 95 percent of the time I would see a 10:48

result as large or larger than what I've seen.
BY MR. LASKER:
Q. But a P-value of .05 does not mean there's a 95 percent likelihood that 10:48 glyphosate caused the observed cancer being analyzed; correct?
MS. FORGIE: Object to the form. Asked and answered.
You can answer it again. 10:48
THE WITNESS: This is not a way I would ever express the meaning of a P-value.
BY MR. LASKER:
Q. And that's because, as I think you 10:48 explained, the P-value does not tell us anything about the study's internal validity in being able to accurately identify a causal association if it exists; correct?
MS. FORGIE: Object to the form. 10:48
THE WITNESS: A P-value is not a measure of validity. A P-value is a measure of randomness or chance.
BY MR. LASKER:
Q. And Dr. Poole explains -- and this 10:49

time I think I do have the quote for you.
MS. FORGIE: What page are you?
MR. LASKER: On page 293.
BY MR. LASKER:
Q. That -- and this is on the left 10:49 column, the second paragraph from the top, that "Statisticians who have examined these questions in detail have found under widely ranging conditions that P-values on the order of .05, .01, and even lower provide 10:49 much less evidence against the null value than they appear to provide at face value." Correct?
A. That's what it states.
Q. And Dr. Poole explains that 10:49 P-values in the vicinity of .05 provide almost no evidence against the null hypothesis at all; correct?
A. It says as a general matter P-values in the vicinity of .05 provide 10:49 almost no evidence against the null hypothesis at all.
Q. And that's what you teach your epidemiology students; correct?
MS. FORGIE: Object to the form. 10:50

THE WITNESS: What I teach my epidemiology students is to take these statements and put them in the context of how we use P-values in epidemiology as one parameter and not the end-all of 10:50 causal reasoning.
BY MR. LASKER:
Q. And you agree with Dr. Poole that a P-value in the vicinity of .05 generally provide almost no evidence against the null 10:50 hypothesis -- well, I put the "generally" in the wrong place. Let me put it exactly how he says it.
You agree with Dr. Poole that as a general matter P-values in the vicinity of 10:50 .05 provide almost no evidence against the null hypothesis at all; correct?
MS. FORGIE: Objection. Asked and answered.
You can answer it again. 10:50
THE WITNESS: Well, this sentence is taken out of context. What I interpret him to be saying here is that a threshold of .05 because he continues by talking about a P of .04, which is, 10:50

you know, the next from .05, that keeping decision-making at a threshold of .05 is a pretty ridiculous experiment -- way of arguing.

What you really want to do is look at the P-value distribution, and that's what this sentence refers to that, you know, thresholds are thresholds. Whatever evidence you think you can draw out of them, why this threshold and not the next? So we should look at distributions and not thresholds. 10:51 10:51

BY MR. LASKER:

Q. In fact, the next sentence that you refer to, Dr. Poole states that a P-value of .04, for instance, is typically found to be almost equally probable under the null and alternative hypotheses; correct? 10:51

A. Correct. That's what it states.

Q. And you agree with that; correct? 10:51

A. It refers to the structure of a P-value being a distribution -- coming from a distribution, but we are deciding arbitrarily what threshold to use, yes.

Q. And you agree that P-values of .04 10:52

are typically found to be almost equally probable under the null and alternative hypotheses; correct?

MS. FORGIE: Object to the form.

THE WITNESS: Again, this is taken out of context. This can be misunderstood. Since this sentence is taken out of context, what I think he's referring to is the misuse of thresholds such as .05. And what he's trying to argue here is that there's no real difference between a P-value of .05 and a P-value of .04 or a P-value of .06. It's just that we as a scientific community or the medical community has agreed that P .05 is it. That does not necessarily make sense if you want to look at data in a much more comprehensive way, you should look at a P-value distribution, and the P-value has a continuum. 10:52 10:52 10:53

And insofar as we're trying to have a scientific dialogue, we should use the most data we can and not just the threshold for decision-making. Human 10:53

lives are not light bulbs. P-values of .05 come out of light-bulb testing that statisticians used -- right -- in industrial settings. And why it's a simple matter. We like to think without having to go back to all the data, and that's a bad habit, and we are trying to teach our students not to get into those bad habits. 10:53

THE REPORTER: Counsel, excuse me. I just had a technical difficulty. I need to go off and restart very quickly. 10:53

MR. LASKER: Okay.

THE VIDEOGRAPHER: We're off the record at 10:52 a.m. This marks the end of videotape number 1. 10:53

(Recess taken from 10:52 a.m. to 10:57 a.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 10:57 a.m. This marks the beginning of videotape number 2 in the deposition of Dr. Beate Ritz. 10:57

BY MR. LASKER:

Q. Dr. Ritz, going back to the Poole 10:57

paper that you use in teaching your epidemiologic students, I'd like to return to this sentence that Dr. Poole has in his article that P equals .04 is typically found to be almost equally probable under the null and alternative hypothesis. 10:57

Do you see that?

A. Yes.

Q. And so in our circumstance, in this case, the null hypothesis is that glyphosate does not cause non-Hodgkin's lymphoma, and the alternate hypothesis would be that glyphosate does cause non-Hodgkin's lymphoma; correct? 10:57

MS. FORGIE: Object to the form. 10:57

THE WITNESS: Actually, there's usually more than one alternate hypothesis. So the alternate hypothesis could be it is tenfold more probable to suffer from non-Hodgkin's lymphoma. It's twofold more probable. So these are all parameter estimates of an effect size, meaning the alternative is not just one alternative. The alternative is a continuum. 10:58 10:58

That's what I tried to explain an hour ago when I said why we are usually going with the null hypothesis is because that is one point while alternative hypotheses are many fold. 10:58

BY MR. LASKER:

Q. Understood.

What Dr. Poole is stating then is that a P-value of .04 would be almost equally probable under the null hypothesis 10:58 here that glyphosate doesn't cause non-Hodgkin's lymphoma and the alternative hypotheses of various possible measures in which glyphosate does cause non-Hodgkin's lymphoma; correct? 10:58

MS. FORGIE: Object to the form.

THE WITNESS: Well, what he's trying to say here, as I interpret this, is that he is emphasizing that we should not be using one P-value of .04 or .05 10:59 or .06, but we should be evaluating the data, and that's how I teach it, in terms of what the overall picture in terms of chance, bias, et cetera, is, and if we are just talking P-values, 10:59

what the picture is in terms of a P-value distribution and you can actually find that in Dr. Greenland's book where he discusses on the P-value is the P-value distribution as an 10:59 alternate to this threshold kind of experiment.

BY MR. LASKER:

Q. When you use that distribution, you find that a P-value of .05 generally 10:59 provides almost no evidence against the null hypothesis; correct?

MS. FORGIE: Object to the form.

THE WITNESS: No, that's not the right interpretation. It means it's 11:00 almost equally probable. It doesn't say that I'm rejecting or not rejecting either the null or the alternative hypothesis.

BY MR. LASKER: 11:00

Q. Understood.

Okay. So then if you have a P equals -- and to use Dr. Poole's specific quote here -- if you have a P equals .04 then in a study, you will find it is equally 11:00

probable that here glyphosate, in fact, caused the cancer or that glyphosate did not cause the cancer; correct?

MS. FORGIE: Object to the form. Mischaracterizes, asked and answered. 11:00

THE WITNESS: The P-value here says nothing about glyphosate. What he says here is that a P of .04 is typically found to be almost equally probable under a null alternative hypothesis. He 11:00 speaks about a P-value, not about a null hypothesis that glyphosate is or isn't causing NHL.

BY MR. LASKER:

Q. I understand that. We can take it 11:00 from both steps, but we want to discuss the fact that -- and I think you mentioned this before -- in the context of this case, the null hypothesis that we're looking at is whether or not glyphosate causes 11:01 non-Hodgkin's lymphoma?

A. And what I would be --

MS. FORGIE: Wait, wait. There's no question.

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BY MR. LASKER:

Q. So the null hypothesis would be the glyphosate does not cause non-Hodgkin's lymphoma here, and the alternative hypothesis might be a variety of other 11:01 things with respect to the nature of glyphosate's association with non-Hodgkin's lymphoma.

What I'd like to understand here, and I think I'm reading this as it's stated 11:01 here, but if that is our understanding of the null hypothesis here, a P-value of .04 would typically be found to be almost equally probable under that null hypothesis or under an alternative causation 11:01 hypothesis; correct?

MS. FORGIE: Object to the form. Asked and answered.

And you can answer it again.

THE WITNESS: This is about the 11:02 P-value. It's about threshold. It's about null hypotheses and alternative hypotheses. It's not about how I assess causation.

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BY MR. LASKER:
Q. I'm not saying it is. I'm just trying to understand P-values, and I think it's consistent with what you said, but a P-value of .04 in the context of a glyphosate study or glyphosate cancer study you could be equally likely to find that P-value if glyphosate actually was a cause of cancer or if glyphosate was not a cause of the cancer; correct? 11:02
MS. FORGIE: Object to the form. Asked and answered.
You can answer it again.
THE WITNESS: No. It means you have to state your null hypothesis or you have to state your alternative hypothesis. Under those hypotheses, you are able to calculate a P-value. If it is .04, then it might be equally probable under both types of hypotheses. 11:02 That what this means.
BY MR. LASKER:
Q. Okay. So if you were to do a test, and you were testing the null hypothesis of whether glyphosate causes cancer and you get 11:03

a P-value of .04, what Dr. Poole is stating is that that result would be equally likely if, in fact, the glyphosate had caused those cancers or the glyphosate had not caused those cancers? 11:03
MS. FORGIE: Object to the form. Asked and answered.
You can answer it again.
THE WITNESS: No, that's not how I would interpret this. 11:03
BY MR. LASKER:
Q. If you're doing a test in which the null hypothesis is glyphosate does not cause cancer and the alternative hypothesis is that glyphosate does cause cancer and you 11:03 get a P-value of .04, that would make the null hypothesis and the alternative hypothesis equally likely; correct?
MS. FORGIE: Object to the form. Asked and answered. 11:03
You can answer it again.
This is like the fifth time on the same question, Eric.
THE WITNESS: Again, the P-value of .04 that he refers to here is the 11:03

threshold P-value, and he calls this threshold P-value equally probable under the null and alternative hypotheses. We have to state all these hypotheses. We then can calculate P-values. 11:04
We can calculate P-value distributions, and we can see how likely the P-values are, not the associations, not the causation, not everything else.
BY MR. LASKER: 11:04
Q. And the P-value is equally likely under the null and the alternative hypothesis; correct?
MS. FORGIE: Object to the form. Asked and answered. This is like the 11:04 eighth time.
You can answer it again.
THE WITNESS: Again, as I understand what Dr. Poole is trying to say here is to avoid thresholds such as 11:04 P-values of .04 because they are always referring to one type of hypothesis, and we are rarely ever asking the other alternative hypotheses. So we are usually just testing one hypothesis. We 11:05

could, of course, then decide to also test other hypotheses, and we could get for or against those hypotheses with a similar equal chance of P-value of .04. That's what it says. 11:05
BY MR. LASKER:
Q. Dr. Poole also notes that one upshot of this work has been a statistical research program devoted to calibrating, standardizing, conditioning, and adjusting 11:05 low P-values to make them higher so that they reflect more realistically the limited statistical evidence they provide against null hypothesis; correct?
MS. FORGIE: Object to the form. 11:05 That's misread.
But you can answer.
THE WITNESS: He's referring to Bayesian methods being developed here, yes. 11:05
BY MR. LASKER:
Q. And you agree that's appropriate; correct?
A. I'm not a Bayesian.
Q. So you don't know one way or the 11:05

other?
MS. FORGIE: Object to the form.
THE WITNESS: No. I'm saying that Bayesian versus frequentist statisticians have a lot of things in common, and I would not want to be on one side or the other. I think they're useful for different purposes.
BY MR. LASKER:
Q. You do agree, though, that statistical methods devoted to calibrating, standardizing, conditioning, and adjusting low P-values to make them higher so that they reflect more realistically the limited statistical evidence they provide against a null hypothesis is a good idea? 11:06
MS. FORGIE: Objection. Asked and answered.
You can answer it again.
THE WITNESS: I'm saying I'm not of either statistical camp, frequentist or Bayesian. I believe that they are both useful. They have appropriate purposes and when needed, I use either one of them, and what he says here is that 11:06

there are developments in statistics that, you know, we should be looking out for, and this is 2001. So some of these might have happened.
BY MR. LASKER: 11:06
Q. You also talk about confidence intervals in your expert report; correct?
A. Correct.
Q. And, again, the standard methodology or the standard measure used by epidemiologists to exclude chance using confidence intervals is the 95 percent confidence interval; correct? 11:07
MS. FORGIE: Object to the form.
THE WITNESS: The 95 percent confidence interval is a convention just like the P-value of .05.
BY MR. LASKER:
Q. Under that convention, a confidence interval is considered statistically significant if it excludes the null hypothesis of 1.0; correct? 11:07
A. The confidence interval projects similar types of data as the P-value in this case. You are correct that if a P-value of 11:07

.05 is what I'm looking for, then a 95 percent confidence interval would exclude the 1.
Q. And, again, you would not state that a statistical significance -- if a test is significant at the 95 percent confidence interval, that would not mean to you that you can have 95 percent confidence that the value that you see in a given study is not due to chance; correct? 11:08
MS. FORGIE: Object to the form.
THE WITNESS: That's not how we interpret confidence intervals. Confidence intervals have similar information but also more information than a P-value. So I have to first decide on the confidence limit, which is 95 percent, which is also similar to a P-value of .05.
So if I use a confidence interval in the same bad manner as a P-value, meaning as a threshold, then that's all I get out of it. However, I teach my students that a confidence interval actually tells them a lot more than what 11:08

a P-value would. A singular threshold P-value, not a P-value distribution.
BY MR. LASKER:
Q. One thing you teach your students to look at is what's called the confidence limit ratio; correct? 11:09
A. Yes, we can look at that as well.
Q. And the confidence limit ratio is the ratio between the upper and the lower end of the confidence interval; correct? 11:09
A. Correct.
Q. So if we have a study that reports an odds ratio of 1.5 and, let's say, a confidence interval of 0.8 to 3.2 -- do the; math work well -- the confidence limit ratio would be 4' correct? 11:09
MS. FORGIE: Object to the form.
THE WITNESS: What would the ratio be.
BY MR. LASKER: 11:09
Q. If it's a 95 percent confidence level of 0.8 to 3.2, then your confidence limit ratio is 3.2 divided by 0.8 or 4; correct?
A. Right. 11:09

Q. You can use the CLR -- we'll call it CLR for confidence limit ratio -- you can use the CLR calculation to compare the power after the fact of different studies to exclude chance as the explanation of a potential association; correct?

MS. FORGIE: Object to the form.

THE WITNESS: Well, this is just one way of looking at confidence intervals again. So what actually Dr. Poole does when he shows his Table 1 is that he -- that's what I teach my students is that you should not use any one of these parameters whether it's a relative risk, a 95 percent confidence interval, a P-value, or a 95 percent CLR as just one piece of information to decide anything.

You should use each piece of that puzzle to put it -- and put them together and evaluate the data appropriately within that context. And these are one, two, three, four types of ways of doing that.

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BY MR. LASKER:

Q. Right. I understand that.

But with respect to the CLR, the CLR calculation allows you to compare the power of the different studies to either exclude or not exclude a potential causal association; correct?

MS. FORGIE: Object to the form and asked and answered.

You can answer it again.

THE WITNESS: Actually, it doesn't really because the CLR, as we have just done here. As an example, you are dividing an upper limit above one by a lower limit, the low one. So that ratio alone doesn't tell you anything about whether the P-value actually would be above or below a threshold.

So his example here is when you see the last one, part D, that a relatively narrow confidence limit ratio then reflects a P-value that under conventional statistics would not be considered significant; however, the CLR tells you you have a fairly nice

confidence interval width.

BY MR. LASKER:

Q. Right. I'm just trying to understand what that means. I recognize it's not going to tell you about statistical significance.

My understanding of a CLR was that it would give you some indication of the power of the study to find or not find an effect; is that correct?

MS. FORGIE: Object to the form. Asked and answered.

You can answer it again.

THE WITNESS: Again, it is one way of looking at the confidence interval widths. That's all it is. However, confidence intervals can and cannot include the null value. They can be close to the null value. They can be far away from the null value. They can be very wide but very far from the null value, and anybody would then jump and say that's a study that proves. Okay.

So each part of that equation of parameters cannot be taken out of

context. What I'm trying to teach my students is use everything, every bit of information you can get. Calculate all of these values. Look at them with an informed mind and don't exclude one in favor of the other.

BY MR. LASKER:

Q. Can we go to the 2010 PowerPoint.

MS. FORGIE: Are we putting 19-3 aside?

MR. LASKER: We can just keep it. We might refer back to it.

MS. FORGIE: Okay. Thank you.

(Exhibit Number 19-4 was marked for identification.)

BY MR. LASKER:

Q. Dr. Ritz, I'm not sure if you remember --

MS. FORGIE: Is this 4?

THE REPORTER: 4.

MR. LASKER: 19-4.

BY MR. LASKER:

Q. Dr. Ritz, these are PowerPoint slides of yours we found on the internet. One of the slide decks that you use in your

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lectures, at least this was in 2010; correct?
A. I imagine. If nobody played with it.
MS. FORGIE: I don't know about that. 11:13
BY MR. LASKER:
Q. On pages 123 -- actually, 124 and 125. The one thing we did do is we put numbers on these slides. So it's actually in the bottom right-hand corner. It's the only change we made; so we can actually do this in a somewhat efficient manner. 11:14
MS. FORGIE: What number again on what page? 11:14
MR. LASKER: 124 and 125. This is the same slide actually that appears in Dr. Poole's article.
BY MR. LASKER:
Q. On page 125, you make the point that the estimates with a smaller CLR -- here it's B and D -- mean the width of the confidence intervals is tighter -- are findings that stand the best chance of holding up in the context of existing and 11:14

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future research; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Conditional on their validity.
BY MR. LASKER: 11:14
Q. Correct.
A. Uh-huh.
Q. And those studies with the tighter confidence limit ratio would weigh more heavily into a meta-analysis; correct? 11:15
MS. FORGIE: Object to the form.
THE WITNESS: Not necessarily. It depends on the study size. So we could have -- it depends.
BY MR. LASKER: 11:15
Q. Okay. In your lecture notes to your class, you state that "Estimates B and D would weigh more heavily into meta-analysis and would exert stronger influences on probability distributions in properly conducted Bayesian analyses"; correct? 11:15
A. Yes.
Q. And that is correct; right?
A. Yes, that is correct. 11:15

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Q. And you also state that these estimates with the more narrow CLR are the results that should be put forth for emphasis as the most statistically stable results that this study has to offer; correct? 11:15
MS. FORGIE: Object to the form.
THE WITNESS: What was the question? That I state this?
BY MR. LASKER: 11:15
Q. You state that these estimates B and D with the more narrow CLR are the results that should be put forth for emphasis as the most statistically stable results this study has to offer; correct? 11:16
MS. FORGIE: Object to the form.
THE WITNESS: Actually, it doesn't refer to the CLR. It refers to the whole of the data provided under B and D. 11:16
BY MR. LASKER:
Q. Okay. And the data with a narrower CLR, one of the points you're making here is that even though, for example, your category C is statistically significant to the P, it 11:16

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was .02 level, because it has a wider CLR than, for example, number D or letter D, which is not statistically significant, it is -- has less chance of holding up conditioned on its validity in the context of existing and future research; correct? 11:17
MS. FORGIE: Object to the form.
THE WITNESS: Indeed that is one thing I try to explain to my students to not rely just on the P-value, P less than .05, which in the C row, we see is the case, but we also have a wide CLR, and we have a very strong point estimate and a wide confidence interval. 11:17
So when you're taking all of that into consideration, then the estimate D would be at least, if not more, valid, might prove more valid in the end or more reproducible in the end than the estimate C. However, you know, all this depends on validity, as I said. 11:17
BY MR. LASKER:
Q. Okay. And you state in your expert report -- and it's on page 12 in your expert report -- that -- the last sentence of the 11:18

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first full paragraph that starts "Importantly, however."
A. Which page?
Q. Page 12.
A. Yes. 11:18
Q. And you state that "The odds ratios or the risk ratios least likely to be influenced by chance are not those with low P-values, but those with narrow confidence intervals or low CLRs." Correct? 11:18
MS. FORGIE: Object to the form.
THE WITNESS: Where was that?
BY MR. LASKER:
Q. The last sentence of the second paragraph. 11:18
A. "Importantly, estimates least influenced by chance are not those with low P-values but those with narrow confidence intervals."
Q. That's correct; right? 11:18
A. In the context of this, yes.
Q. Okay. And when we talk about narrow confidence intervals, the measurement that you provided for us that I'd like to be able to use is the CLR; correct? 11:19

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MS. FORGIE: Object to the form.
BY MR. LASKER:
Q. That's a measurement of the width of the confidence interval?
A. It's a measurement of the width of 11:19 the confidence interval; however, the CLR does not tell you anything about the placement of the confidence interval.
Q. Understood.
MS. FORGIE: Wait. Let her finish. 11:19
THE WITNESS: What I've been trying to say is we should not rely solely on a P-value especially a P-value threshold or a confidence interval or a CLR or a point estimate. 11:19
So don't be fooled by a high point estimate but a confidence interval that goes from .5 to 200 because that data is pretty much uninformative.
BY MR. LASKER: 11:19
Q. Now, on page -- in your expert report on page 15, you provide a table listing of different publications with epidemiological data in glyphosate and non-Hodgkin's lymphoma; correct? 11:20

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A. Where is this?
Q. Page 15 in your report.
A. Yes.
Q. And just so it's clear, this table does not tell you or does not provide you 11:20 with a -- the relative -- a sense of the relative power of the listed studies to identify a causal association between glyphosate and non-Hodgkin's lymphoma; correct? 11:20
A. This table shows what it says in the sentence above. "I show the sample size of each human study of glyphosate in NHL." That's exactly it. It shows the sample size. 11:20
Q. Okay. This table did not tell you -- did not tell you which of these studies is the most powerful study in being able to assess an association between glyphosate and non-Hodgkin's lymphoma; 11:20 correct?
MS. FORGIE: Objection. Asked and answered. That's the exact question you just asked.
You can answer it again. 11:21

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THE WITNESS: Well, powerful has many meanings. If we're talking about statistically powerful versus powerful in a sense of validity, then, you know, those are different discussions. 11:21
BY MR. LASKER:
Q. This table does not tell us anything about which study is the most statistically powerful in determining whether there is a causal relationship 11:21 between glyphosate and non-Hodgkin's lymphoma; correct?
MS. FORGIE: Objection. Asked and answered. This is the third time.
You can answer it again. 11:21
THE WITNESS: This table was meant to show sample size.
BY MR. LASKER:
Q. It does not tell you anything about the power of the study to determine a causal 11:21 association between glyphosate and non-Hodgkin's lymphoma; correct?
MS. FORGIE: Objection. Asked and answered. This is the fourth time.
THE WITNESS: Wrong. Sample size 11:21

is one element of the power of a study.
BY MR. LASKER:
Q. Okay. The top listed study on your table is the Cocco study 2013; correct?
A. Yes. 11:21
Q. And the Cocco study is the least powerful of all the epidemiologic studies to be able to assess the association between glyphosate and non-Hodgkin's lymphoma; correct? 11:22
MS. FORGIE: Object to the form.
THE WITNESS: This table shows sample size. It has nothing to do with statistical power in the sense that it's a complete evaluation of statistical 11:22 power. However, sample size is part of what we use in calculating statistical power.
BY MR. LASKER:
Q. My question, though, you have Cocco 11:22 listed as the top study on this table, and the Cocco study is, in fact, the least powerful study in assessing a potential causal association between glyphosate and non-Hodgkin's lymphoma; correct? 11:22

MS. FORGIE: Objection. I object to the form, and asked and answered.
THE WITNESS: You don't like my table?
BY MR. LASKER: 11:22
Q. I'm just asking you a question.
A. The Cocco study is what the Cocco study is, and I actually explain the Cocco study a few pages later. The study by Cocco was limited in how much we can glean from 11:22 its results as only four cases and two controls had ever used glyphosate.
Q. So the Cocco study is, because of that fact, not powerful in assessing an association between glyphosate and 11:23 non-Hodgkin's lymphoma; correct?
MS. FORGIE: Object to the form and asked and answered. This is, like, the fifth or sixth time.
You can answer it again. 11:23
THE WITNESS: The Cocco study has been evaluated by me. It's also been listed in this table. This table shows sample size. The Cocco study is definitely the largest study we have in 11:23

terms of sample size of cases, not controls. The AHS has a lot more controls. So in terms of case number, it is the most -- it is the study with the most cases. However, as I said a 11:23 few pages after on page 18, it is limited because of low exposure prevalence.
BY MR. LASKER:
Q. And just so I understand, the Cocco 11:23 study is the, I believe, least powerful study in being able to answer the question of whether glyphosate is causally associated with non-Hodgkin's lymphoma; correct?
MS. FORGIE: Object to the form. 11:24
Asked and answered. This is number six.
You can answer it again.
THE WITNESS: The Cocco study has a large sample size in terms of cases. The AHS study has the largest sample 11:24 size in terms of controls. One is at the top; the other is at the bottom. We could turn it around if you'd like. However, power, statistical power is determined by a number of parameters. 11:24

One of those is the number of cases. The other is the number of controls. Yet another is the prevalence of exposure, and then power cannot be distinguished on a playing field without 11:24 saying what effect size you actually want to estimate. So once we have agreed what the effect size is, then we can talk about power.
BY MR. LASKER: 11:24
Q. It would not be appropriate for somebody to look at this table on page 15 and conclude that the Cocco study was more powerful than the De Roos study with respect to assessing whether there is an association 11:25 between glyphosate and non-Hodgkin's lymphoma; is that fair?
MS. FORGIE: Object to the form and asked and answered.
THE WITNESS: Again, if we're 11:25 talking statistical power and not validity of the study, which, you know, is another criterion that I would put actually much higher here, the Cocco study has the most cases. The De Roos 11:25

study has the most controls. Both are powerful because of that part of the equation that goes into a power analysis. However, there are more parameters than the number of cases, the number of controls. One of them is exposure prevalence. I explain that when I talk about the Cocco study as not being able to tell us much because it has low exposure prevalence. On the other hand, De Roos has a very high exposure prevalence. 11:25 11:25

BY MR. LASKER:

Q. Dr. --

MS. FORGIE: Wait. Let her finish. 11:26

MR. LASKER: We're going to be here all day, and I'm going to have to mark this and go to the judge because I can't get a yes or no answer to any question I ask. I asked a very simple question, and she's going into a monologue. We're not going to have that happen here. So if the witness is not going to answer the questions, then we'll have to go to the court again to either get 11:26 11:26

instruction for the witness to answer the questions or to provide us more time. I ask yes or no questions and I get a speech.

MS. FORGIE: You know, first of all, part of the problem is you keep putting these long declaratory statements before everything. She is not required to give a yes or no answer. She has answered it very clearly -- 11:26 11:26

MR. LASKER: You're not the witness.

MS. FORGIE: Let me finish.

MR. LASKER: You're not the witness. 11:26

MS. FORGIE: Neither are you. So, you know what? If you want to call the judge, I think you should go ahead.

MR. LASKER: Okay. Well, we're going to start marking these and at a certain point we'll go -- let me mark the last question and answer. I'm going to ask the question again. 11:26

MS. FORGIE: Are you going to call the judge? 11:26

MR. LASKER: I will eventually if this keeps up. I'm going to mark them and we'll come back to the judge if we have to.

BY MR. LASKER: 11:26

Q. Table 15, the table you present on page 15 of your report. It would not be appropriate to look at this table alone to reach a conclusion as to the relative power of the listed studies to determine whether glyphosate is associated with non-Hodgkin's lymphoma; correct? 11:27

MS. FORGIE: Object to the form. Asked and answered. This is like number 7. 11:27

You can answer it again.

THE WITNESS: This is a table that refers to sample size. Sample size is part of statistical power.

BY MR. LASKER: 11:27

Q. It would not be --

A. So --

MS. FORGIE: She is entitled to finish.

THE WITNESS: It is appropriate to 11:27

show parts of statistical power, but, of course, I would not want to infer statistical power from just this table. But it is part of it.

BY MR. LASKER: 11:27

Q. And another way one could look at this would be to calculate the CLRs for each of these studies; correct?

MS. FORGIE: Object to the form.

BY MR. LASKER: 11:27

Q. For the endpoint of Roundup and non-Hodgkin's lymphoma?

A. CLRs is something that we calculate after we have the data and the parameter estimates. 11:28

Q. Right. And we have the data and the parameter estimates for each of these studies; correct?

A. Yeah, but that is not how we're calculating statistical power. Statistical power is something that we are calculating prior to conducting the study. 11:28

Q. Understood. So now it's after the fact we have the data. We could actually calculate a CLR for each of these studies; 11:28

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correct?
A. If we can agree on which results to use and, yeah, we can do that.
Q. Have you done that exercise?
A. In my head. 11:28
Q. With respect to -- let's move on. The interpretation of confidence intervals in observational studies requires the assumption of no bias; correct?
MS. FORGIE: Object to the form. 11:28
THE WITNESS: It is correct that confidence intervals and observational studies do not include -- are not estimates of bias.
BY MR. LASKER: 11:29
Q. So the interpretation of confidence interval and observational studies requires the assumption of no bias; correct?
MS. FORGIE: Object to the form. Asked and answered. 11:29
You can answer it again.
THE WITNESS: We make assumptions when interpreting confidence intervals of observational studies, and one of the assumptions is no other biases, yes. 11:29

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BY MR. LASKER:
Q. In other words, even if a study reports a positive association and reports a 95 percent confidence interval that excludes 1.0, that study cannot be interpreted as 11:29 evidence of a causal association if there is bias in the study; correct?
MS. FORGIE: Object to the form.
THE WITNESS: It depends on the kind of bias, the size of bias. We are 11:29 talking about bias as a category. We at UCLA try to teach bias in terms of quantitative and so the bias can be so minimal that it's not to be a concern.
BY MR. LASKER: 11:30
Q. One type of bias that you identify in your expert report is recall bias; correct?
A. Yes.
Q. And you also teach your students 11:30 about recall bias, your epidemiology students; correct?
A. Correct.
Q. Let's get the 2017 slide deck on informational bias. 11:30

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(Exhibit Number 19-5 was marked for identification.)
BY MR. LASKER:
Q. And, Dr. Ritz, I've handed you as Exhibit 19-5, I believe this is a slide deck 11:31 that you used either last year or you're using currently with your epi 200 B students; correct?
A. I don't know. I haven't reviewed it. It looks like it. 11:31
Q. This is a document I'll represent that you produced in response to our --
A. Oh, okay. Then it must be.
MS. FORGIE: Did you add pages to it? 11:31
MR. LASKER: She's updated.
THE WITNESS: I learn.
BY MR. LASKER:
Q. So at page 61 in your slide deck, you talk about this issue of recall bias. I 11:31 just want to make sure I understand the terminology. So as you explain --
MS. FORGIE: Wait a minute. I don't see page 61.
MR. LASKER: It's actually page 60, 11:31

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and then there's no page number on 61.
MS. FORGIE: Right. I don't see the pages.
MR. LASKER: It is the page after 60 which I've called 61 in my simplistic 11:31 way of counting.
BY MR. LASKER:
Q. So you see the slide that has recall bias listed at the top; correct?
A. Correct. 11:32
Q. And recall bias is a form of differential misclassification bias of particular concern in interview-based case control studies; correct?
A. Correct. 11:32
Q. And the issue with recall bias is that cases who are diseased may ruminate about prior exposures and report it more completely than controls; correct?
MS. FORGIE: Object to the form. 11:32
THE WITNESS: It says that that is one way how differential recall can occur.
BY MR. LASKER:
Q. And the other thing that you 11:32

mention and you teach your students is that cases might exaggerate exposure while subjects without the disease under investigation. And I guess there's something missing here. 11:32

A. Yeah. That's why this --

Q. Let me understand this correctly.

A. No, this is an appendix to the class, so it's not edited.

Q. But I think the point -- and let me make sure I'm correct -- the point that without the typo you would be making here is that cases might exaggerate exposure compared to subjects without the disease under investigation; correct? 11:33

A. Yes.

MS. FORGIE: Object to the form.

THE WITNESS: Well, that is one way how differential recall bias can occur and why I'm teaching it is to say that when we do our fieldwork have to avoid that this is going to happen. 11:33

BY MR. LASKER:

Q. And the other issue that you teach your students is that in the case control 11:33

study, controls might not recall exposures since they do not have an incentive to do so; correct?

A. Correct. And, again, that is under the premise that we are doing whatever we can to have everybody recall in the same way. 11:33

Q. A recall bias -- well, recall bias can create another -- there can be another issue with recall bias if a study relies upon next of kin or proxy respondents to provide exposure information; correct? 11:34

MS. FORGIE: Object to the form.

THE WITNESS: That's not -- we can call it recall bias, but it is usually being less informed about the exposure so it's kind of information bias. 11:34

BY MR. LASKER:

Q. As a general matter, exposure data provided by proxies is considered less reliable than exposure information provided by the actual cases and controls; correct? 11:34

MS. FORGIE: Object to the form.

THE WITNESS: That is relative.

For example, if it is an exposure that a 11:34

case would not want to report but the wife then tells us, it's actually more reliable. So it really depends on the study.

BY MR. LASKER: 11:34

Q. I'll give you that one. I know that you do this a lot in your work, but with respect to pesticide exposures, as a general matter, exposure data provided by proxies would be considered less reliable than exposure data provided by the cases or controls themselves; correct? 11:35

MS. FORGIE: Object to the form.

THE WITNESS: Again, it depends on the circumstances. For example, if the proxy would be a co-worker, he might be just as able to report the work practices and the type of exposures. If the proxy is a son of the farmer who worked alongside the farmer, he would be very well capable of responding. If it is a wife who never goes out in the fields and doesn't talk to her husband at night at the table -- at the dinner table about what he's been doing all 11:35

day, yes. But if it's a wife who quizzes her husband on how did your day go and what did you do and what are the expenses about these kind of pesticides that I'm seeing on the ledger here because she does the books, she knows very well. 11:35

BY MR. LASKER:

Q. That's why I didn't want to ask in every case because obviously case-by-case can be different. But as a general matter overall, exposure data provided by proxies is considered at least potentially less reliable than exposure data provided by the cases and controls themselves; correct? 11:36

MS. FORGIE: Object to the form and asked and answered.

You can answer it again.

THE WITNESS: Again, it depends. It depends on who the proxy is, how close the proxy is to the individual, how much they communicate, how much they work together, and whether it is actually a proxy who counts while the individual doesn't count, meaning, well, 11:36

we paid so much for all of these pesticides in the last year and the husband doesn't care. He just uses what's there. So sometimes we find in our studies of elderly especially that 11:36
the wives are much more reliable sources. So you can't really say that it's always the proxy that misreports.
BY MR. LASKER:
Q. And I understand that. I'm not 11:36
trying to nail you down on every instance.
MS. FORGIE: There's no question.
BY MR. LASKER:
Q. But let me -- one of the things you've done, and I've seen this in some of 11:37
your publications is you can conduct a sensitivity analysis to determine whether or not the inclusion of proxy data affects the results of the study; correct?
A. Correct. 11:37
Q. And one of the things you're concerned about when you do that analysis is a possibility that the use of a proxy may have introduced some misclassification bias into a study; correct? 11:37

MS. FORGIE: Object to the form.
THE WITNESS: The use of proxies versus the individual themselves may or may not result in information bias, and it may or may not result in differential 11:37
information bias. So if we are using proxies in cases and controls, then whatever they misreport for cases and controls might be at the same level, and that would be a non-differential 11:38
misclassification.
BY MR. LASKER:
Q. And when you do your sensitivity analysis, you're looking to see whether there's a differential or non-differential 11:38
including the proxy data; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Not exactly. If I want to establish the validity of a proxy, I would actually need a gold 11:38
standard like a record, then interview the case, interview the proxy, and then compare both to the gold standard.
BY MR. LASKER:
Q. Another type of bias that can arise 11:38

in observational studies is selection bias; correct?
A. Correct.
Q. And case control studies are vulnerable to selection bias and their 11:39
validity to a large degree hinges on the choice of proper controls; correct?
MS. FORGIE: Object to the form.
THE WITNESS: It is correct that at -- that there is selection bias in 11:39
case control studies. There is also selection bias in cohort studies. However, case control studies are usually evaluated more critical in terms of selection bias because we try to 11:39
avoid it as much as we can.
BY MR. LASKER:
Q. And, in fact, in your own publications, you've talked about the fact that this problem of selection bias can be 11:39
circumvented in the cohort study; correct?
MS. FORGIE: Object to the form.
THE WITNESS: It's a different kind of selection bias in a cohort study as well as in a case control study. A 11:39

cohort study does not have the kind of selection bias that a case control study has. But it has another type of selection bias that a case control study doesn't have. 11:40
BY MR. LASKER:
Q. And what is that?
A. That's loss to follow-up, differential loss to follow-up.
Q. With respect to loss to follow-up 11:40
for disease outcome, that is not, as I understand it, correct me if I'm wrong, an issue with the Agricultural Health Study; correct?
MS. FORGIE: Object to the form. 11:40
THE WITNESS: Not necessarily. The Agricultural Health Study may have selection bias depending on whether or not there's differential loss to follow-up with respect to the exposed 11:40
and the unexposed.
BY MR. LASKER:
Q. Okay.
A. So it would depend on what the outcome it is we are talking about. 11:40

Q. The other issue you mention in your expert report is confounding. A confounder is an exposure that is associated both with the exposure of interest and the outcome of interest; correct? 11:41

MS. FORGIE: Object to the form.

THE WITNESS: That is one part of how we define a confounder.

BY MR. LASKER:

Q. So, for example, there was a study a few years back now that reported a positive association between coffee and pancreatic cancer? It's somewhat of a well-known -- 11:41

A. Favorite example. 11:41

Q. And when the investigators looked more closely at that data, they discovered that the reported positive association was actually due to the fact that, if I have this correctly, coffee drinkers were more likely to be smokers and the smoking increased the risk of pancreatic cancer? Do I have that right, or do I have it backwards? 11:41

MS. FORGIE: Object to the form. 11:41

THE WITNESS: That was part of the argument, however, that's not how we are defining confounding. Confounding is an independent risk factor for the outcome that also has an association with the exposure and is not an intermediate in the pathway to disease. 11:41

MS. FORGIE: When you get to a good breaking point.

MR. LASKER: Okay. Let's get through this. 11:41

MS. FORGIE: Thanks.

BY MR. LASKER:

Q. With respect to coffee drinkers and pancreatic cancer, smoking was a confounder; is that correct? 11:42

A. Assuming that smoking really causes pancreatic cancer which I'm not completely sure it's true, but I'm not a pancreatic cancer researcher, and depending on what population we're talking about, for example, there are populations where you have a lot of coffee drinking but no smoking, and there are populations where you have a lot of smoking and no coffee drinking, meaning the 11:42

two are independent.

Assuming that we are in a population where the two are actually dependent and we know that, that coffee drinkers smoke more or vice versa, then that could be defined as a confounder. However, in a cohort study, you can actually assess that. 11:42

Q. In your studies, your epidemiologic studies, you will try to address the possibility of confounding; correct? 11:42

A. Definitely.

MR. LASKER: Why don't we take a break now.

MS. FORGIE: Great. Thank you. 11:43

THE VIDEOGRAPHER: We are off the record at 11:43 a.m.

(Recess taken from 11:43 a.m. to 11:55 a.m.)

THE VIDEOGRAPHER: We are back on the record at 11:55 a.m. 11:55

BY MR. LASKER:

Q. Back on the record.

Dr. Ritz, we were talking about confounding, and I think one of the points 11:55

you made in your report, I think elsewhere, is in analyzing or conducting a study, you'd want to identify as best you can other risk factors for disease that you're studying to be able to see whether or not those are confounders; correct? 11:56

A. It is correct that you're always very worried about confounding no matter what and that you're identifying strong risk factors for the disease that also is associated with exposure. 11:56

In the second step, you have to see whether there are possibly intermediates in the pathway and/or proxies for the exposure, and that's a very important assessment. 11:56

Q. And that can be even more difficult in a situation where you have a disease that has unknown causes; correct?

MS. FORGIE: Object to the form.

THE WITNESS: It's actually not more or less difficult. A disease that has known causes such as lung cancer, we know that we have to control for smoking, and we may or may not have that data. So that's a very difficult study 11:56

to do if we don't have smoking data.
So difficult in a sense, if I don't have a strong risk factor, then it also cannot be a strong confounder. So I'm actually a little bit out of the woods when there's no risk factor because I can assume that if there was a really strong risk factor, I would know about it. 11:57
So if there was a really strong confounder, I probably would know about it. 11:57

BY MR. LASKER:

Q. You read the deposition of Dr. Blair in this case? 11:57

A. Yes.

Q. Dr. Blair has been studying agricultural exposures and cancer going back probably 40-some-odd years; right?

MS. FORGIE: Object to the form. 11:57

THE WITNESS: I'm not sure, but I know that he's been publishing in the '80s on industrial workers, that he's worked at the NCI and that he was generally also interested in 11:57

agricultural work, and he's a coauthor of some of these early papers.

BY MR. LASKER:

Q. Do you agree with Dr. Blair that there was an association that was found 11:58 between farming -- farmers and non-Hodgkin's lymphoma that existed prior to the time that glyphosate was on the market?

MS. FORGIE: Object to the form.

THE WITNESS: Did he say that 11:58 anywhere in the document?

BY MR. LASKER:

Q. Yeah. If you want, I can show it to you if you want.

A. Yeah, yeah, please. 11:58

MR. LASKER: We are not going to mark it as an exhibit. It's a transcript.

MS. FORGIE: I think we should mark it. 11:58

MR. LASKER: You want to mark it?

MS. FORGIE: Yeah.

MR. LASKER: Where are we then?

THE REPORTER: 6.

///

(Exhibit Number 19-6 was marked for identification.)

BY MR. LASKER:

Q. On page 80 --

A. Is it the page numbers down here? 11:59

Q. Yeah, the actual --

A. Yeah, okay.

Q. I'm sorry. Page 90. I don't know if you can see the highlighting. And at pages 90, we're talking with Dr. Blair about 11:59 this issue of an increased or an association between farming and non-Hodgkin's lymphoma dating back to the 1960s.

Do you see that?

A. Yes. 11:59

Q. And do you agree with Dr. Blair that there was this epidemiological literature pointing to an association between farming and non-Hodgkin's lymphoma dating back to before glyphosate was on the 11:59 market?

A. Well, he seems to be saying that. I know those very old studies are very, very broad; so they would ask somebody have you ever farmed, and, you know, find an 12:00

association but, yeah, at the level of that broad types of exposure, it might be the case.

Q. Okay. So with respect to farmers and non-Hodgkin's lymphoma, there is at 12:00 least something going on that would not be related to glyphosate exposure; correct?

MS. FORGIE: Object to the form. Asked and answered.

You can answer it again. 12:00

THE WITNESS: I agree that there is a difficulty in assessing exposures that vary over time. So when have we started in agriculture using chemicals? After World War II. Before that, they used 12:00 arsenicals, et cetera; right? But really manmade chemicals for pest control were introduced during World War II and after World War II and took off in the U.S. in the 1950s. So general 12:01 exposure to agricultural chemicals dates back to the 1950s.

Among those chemicals may have been carcinogens. We know that there were waves of chemicals that were being used. 12:01

We started with organic chlorines until we decided that that was a bad idea because they bioaccumulate. And then the organic phosphates got their trial run almost parallel. They were quite acutely toxic; so there were some restrictions on those, and the herbicides, the early ones were 2,4-D.

2,4-D is, for example, a 2B IARC possible human carcinogen. So definitely farmers have been exposed to carcinogens at least since World War II.

BY MR. LASKER:

Q. And you also mentioned earlier that diesel fuel might be associated with non-Hodgkin's lymphoma in farmers; correct?

A. Yes, that has been shown in the AHS. I mean, one study does not make a summer -- one swallow. So we would never just rely on one study, but there's reason to think that certain hematopoietic cancers, possibly also some cancer subtypes of NHL might be related to what is in diesel.

Q. And various types of animal husbandry like chicken farming or certain

types of farming have been, at least in the AHS, associated with non-Hodgkin's lymphoma; correct?

MS. FORGIE: Object to the form.

THE WITNESS: There could be risk factors for Hodgkin's lymphoma, but it has to be reevaluated.

BY MR. LASKER:

Q. For non-Hodgkin's?

A. For non-Hodgkin's lymphoma. However, that doesn't make them a confounder. We now have to also consider whether or not they're related to the exposures.

MS. FORGIE: Wait, let her finish.

MR. LASKER: Understood.

BY MR. LASKER:

Q. So an epidemiologic study, and I think your studies are like this as well, will often report different odds ratios with different levels of adjustment to account for potential confounding; correct?

A. We would try different levels of adjustment for multiple reasons, but the main reason would be to assess confounding.

Q. In your expert report at page 16 -- and this is -- if you have your expert report in front of you, on page 16. In the last paragraph which starts "The IARC working group's monograph on glyphosate."

Do you see that?

A. Yeah.

Q. You state in the second sentence "The most highly adjusted estimates, also known as fully adjusted models, are the estimates that adjust for as many confounding variables as possible such as adjusting for age, sex, race, and also sometimes other pesticide exposures"; correct?

A. Yes.

Q. And then you state that "This is relevant because these fully adjusted models give the reader confidence that the findings are most likely due to glyphosate Roundup exposure instead of other potential causes that act as a confounder"; correct?

A. Correct.

Q. And on page 14 of your report, you present what's called a forest plot of the

various odds ratios or rate ratios in some of the epidemiological studies for glyphosate; correct?

A. You can call it a forest plot. I would just call it a visual representation of results from different studies.

Q. In your visual depiction of the results from different studies, you do not provide or list the most highly adjusted odds ratios or risk ratios from the studies; correct?

A. Not correct. De Roos 2003 is a very highly adjusted for 43 different pesticides.

Q. The most highly adjusted estimate in the De Roos 2003 paper had a report odds ratio of 1.6.

A. No.

MS. FORGIE: Object to the form.

THE WITNESS: Would you show me that?

MS. FORGIE: I don't think there's a question.

THE WITNESS: Yeah, is there a question.

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BY MR. LASKER:
Q. There is a question. There are two -- actually, three odds ratios in the De Roos 2003 study.
A. Yes. 12:05
Q. You have reported one of those odds ratios and not the other odds ratio; correct?
A. It's the odds ratio from the logistic regression. 12:05
Q. We'll come back, and we'll circle back to that later when we talk about De Roos 2003, but with respect to the other studies in this paper, for example, in the Eriksson study, you do not provide the most 12:06
highly adjusted odds ratio from the Eriksson study in your chart on page 14; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I would need to see the Eriksson paper because there was a 12:06
multi-varied adjusted odds ratio, and I imagine that we looked at that at some point.
BY MR. LASKER:
Q. Okay. Well, let's pull out the 12:06

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Eriksson study right now.
MS. FORGIE: Are we done with these guys?
MR. LASKER: Yeah, for now.
So the Eriksson is -- we'll mark it 12:06
as --
MS. SHIMADO: 7.
(Exhibit Number 19-7 was marked for identification.)
BY MR. LASKER: 12:07
Q. I think you're talking about the multi-variate analysis that's on page 1661 Table 7; correct?
A. Yes.
Q. And the multi-variate odds ratio 12:07
for glyphosate in the Eriksson study is an odds ratio of 1.51 with a confidence interval of 0.77 to 2.94; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Correct. 12:07
BY MR. LASKER:
Q. That is not the odds ratio that you present in your visual depiction on page 14 of your expert report; correct?
A. That is not. 12:07

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Q. And if we look at the Hardell study for 1999 -- you have Hardell 2003 listed for hairy cell leukemia. I'm looking at the bottom of your table here.
Do you see that? 12:09
A. Yes.
MR. LASKER: Let's mark Hardell 2002.
(Exhibit Number 19-8 was marked for identification.) 12:09
MS. FORGIE: Are we done with Eriksson?
MR. LASKER: For now. We'll go back to it.
BY MR. LASKER: 12:09
Q. In your visual depiction for Hardell, you're depicting an odds ratio of slightly above 3. That is listed as statistically significant; correct?
MS. FORGIE: Object to the form. 12:09
BY MR. LASKER:
Q. At least as it's depicted on your page 14?
MS. FORGIE: Object to the form.
THE WITNESS: It has a wide 12:09

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confidence interval and about 3 from what I see, yes.
BY MR. LASKER:
Q. And if you look at Exhibit 19-8 and you look at page 1047, which is Table 7, 12:09
again, the most adjusted odds ratio in that study is 1.85 with an odds ratio of 0.55 to 6.2; correct?
A. That's what they call them, multi-variate model. 12:10
Q. So again for Hardell, you do not present the most fully adjusted odds ratio according to that study; correct?
MS. FORGIE: Object to the form.
THE WITNESS: For good reasons. 12:10
BY MR. LASKER:
Q. I'm just asking the question in your Table 14 --
A. Yes.
Q. -- for Hardell, you do not present 12:10
the most adjusted -- highly adjusted odds ratio reported by the authors of the study; right?
MS. FORGIE: Object to the form.
Asked and answered. 12:10

You can answer it again.
THE WITNESS: So I'm presenting the odds ratio that I believe has the most validity given what they presented in their paper. 12:10
BY MR. LASKER:
Q. And for the NAPP -- and we'll get to that in a second -- you also have elected in your visual depiction of the study results to report an odds ratio that was not 12:11 adjusted for three pesticides that the NAPP investigators adjusted for in their study; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Again, what I strive 12:11 to do is present odds ratios on confidence interval for what I believe the most valid model is because we're now talking about evaluating the data overall. That does not necessarily mean 12:11 the most fully adjusted model.
BY MR. LASKER:
Q. Just so I understand this, although you state in your expert report that the most highly adjusted estimates reported in 12:11

these studies -- just so we're clear, the -- your comment with respect to the most highly adjusted estimates is specific to the meta-analysis that were conducted of the glyphosate studies; correct? 12:12
MS. FORGIE: Object to the form.
THE WITNESS: It refers to what others considered as their criteria for pulling estimates, not mine, yes.
BY MR. LASKER: 12:12
Q. And you were stating in here that IARC's adjustment or their analysis -- their meta-analysis using these most highly adjusted estimates from the studies was appropriate because it gave the reader 12:12 confidence that the findings are most likely due to glyphosate Roundup exposure instead of another potential cause that acts as a confounder; correct?
A. I'm making no statements about 12:12 appropriateness of these estimates. What I'm saying here is that they did something we call conservative, which is throw the kitchen sink into a model and see what falls out on the other end. 12:12

That is not what I consider the most valid approach.
Q. Okay. The visual depiction that you have of the studies on page 14, you did not -- I mentioned it as a forest plot. You 12:13 weren't --
A. Happy with it.
Q. -- happy with that terminology.
Forest plots, if I understand correctly, are usually depicted on a 12:13 logarithmic scale; correct?
A. Uh-huh.
Q. And the issue with a logarithmic scale, so, for example, in your visual depiction of the Orsi study -- and we can 12:13 look at the actual odds ratios if you want in that study -- but that was a study that had an odds ratio of 1.0 and a lower confidence interval was about 0.5 and the upper confidence interval was about 2.0. 12:13
If you presented that in a forest plot, your line would be about equal distance on both sides of --
A. There --
Q. -- one; correct? 12:14

MS. FORGIE: Wait. Object to the form.
THE WITNESS: There are different ways of depicting results visually, and in a forest plot, you are trying to show 12:14 confidence intervals that are symmetrical, and you can only do that when you use a logarithmic scale.
BY MR. LASKER:
Q. And by using the depiction that you 12:14 use, which is not a logarithmic scale, the visual effect of that is that the confidence intervals will go further out to the right or will appear in this depiction to go further out to the right than if you were 12:14 presenting a forest plot on a logarithmic scale; correct?
MS. FORGIE: Object to the form.
THE WITNESS: That is only the case when you go below 1. As long as you're 12:14 above 1, they are actually symmetric, and you can see that down here Eriksson 2008.
BY MR. LASKER:
Q. Right. 12:14

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But with the -- and we have in this -- in your visual depiction, numerous lines that go below 1 and above 1. When you present it the way that you have in a normal scale as opposed to the way you do it on a forest plot with a logarithmic scale, that has the effect of making those lines extend out further or appear further out to the right than to the left; correct? 12:15

MS. FORGIE: Object to the form and asked and answered. 12:15

You can answer it again.

THE WITNESS: This is not a forest plot. This is just a visualization. I'm giving point estimates and confidence intervals, and the reason for doing this is to have an easy reminder myself, as well as the reader, what the point estimates and the confidence interval widths is. 12:15

It was not to say whether or not it is going more or less beyond the null value, but it clearly indicates when it goes below the null value.

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BY MR. LASKER:

Q. With respect to confounding -- and this is going to be a general question, I think, but epidemiologists use different methods to control for potential confounding; correct? 12:16

A. Yes.

Q. So epidemiologists can control for confounders through model fitting techniques like a regression analysis; correct? 12:16

A. That is one way.

Q. And epidemiologists can also control for confounding by conducting a stratified analysis; correct?

MS. FORGIE: Object to the form. 12:16

THE WITNESS: That is one other way of looking at control for confounding.

BY MR. LASKER:

Q. So in a stratified analysis, an epidemiologist will calculate an odds ratio for subjects with concurrent exposures to two potential risk factors, and then they'll separately calculate the odds ratios for the subjects having only one of those exposures; correct? 12:16

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A. Not necessarily. You can subgroup, but in the end, you want a summary effect estimate that you weigh by the strata. So you're standardizing your estimate according to the weights of the strata in which these individuals fall. 12:17

Q. So in your stratification, for example, you would have if there is current exposures or potential for current exposures, you would have one strata that is exposed only to one of those risk factors, one strata that's exposed to both of those risk factors, and one strata that's exposed to the other risk factor; correct? 12:17

MS. FORGIE: Object to the form. 12:17

THE WITNESS: If you're lucky, you have people in all of those strata, but you have to define the strata, and that's one reason why we use that tool not necessarily when we have better data that's not categorical because, otherwise, within those strata, still have confounding because of categorization. 12:17

So we're trying to use 12:17

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multi-variate models rather than stratification.

BY MR. LASKER:

Q. Just so we can agree what -- how this works, let's turn back to 19-4 which is your 2010 slide deck. 12:18

MS. FORGIE: Wait. Let me get it. Okay.

THE WITNESS: Page?

BY MR. LASKER: 12:18

Q. 98. And as you teach your students then, a stratified analysis is a method for controlling for confounders. "We estimate the exposure disease association within categories or strata of the confounders as in the examples given previously or and derive a summary estimate of this association across the strata which often assumes that the association does not vary across strata." Correct? 12:19

A. Correct. That's exactly what I just tried to explain.

Q. In your rebuttal expert report, you state that "Controlling for confounding by other pesticides in the glyphosate NHL 12:19

studies could make it harder to identify an association between glyphosate and NHL."

Do you recall that?

MS. FORGIE: Object to the form.

THE WITNESS: Where do I say that? 12:19

MS. FORGIE: Are we done with 4?

MR. LASKER: For now, yeah. Where are we now?

MS. SHIMADO: 9.

(Exhibit Number 19-9 was marked for identification.) 12:20

BY MR. LASKER:

Q. So pages of 6 and 7, I think and maybe I'm misunderstanding, but I thought what you were stating in pages 6 and 7 of your report is that controlling in the glyphosate NHL studies controlling for confounding by other pesticides can make it harder to identify an association between glyphosate and NHL; correct? 12:20

MS. FORGIE: Object to the form.

THE WITNESS: Well, it depends on what we mean by "make it harder." So what I am trying to say here, what I do remember -- I'm not reading it right 12:20

now -- is that I was trying to identify confounders which is a different concept.

It's the underlying scientific concept behind control for confounding. 12:21 Confounding is something I can assess in data. Confounder is a scientific concept that I need to presume, and that's what we're doing with directed basic little graphs. You saw a lot of 12:21 them in my slides.

And so what that means is we have to convince ourselves that a variable is a confounder, meaning, there's an underlying true association between that 12:21 variable and the outcome as well as that variable and the exposure of interest and that that variable is not just a proxy measure of the exposure that I'm actually trying to evaluate. 12:21

And any kind of proxy measure of the exposure should not be treated as a confounder.

BY MR. LASKER:

Q. I think I was actually looking at 12:22

page 7 where you're talking about this issue of smoking, lung cancer and whether or not radon exposure adds to the background instance of lung cancer. So I think we're talking past each other. 12:22

A. Yeah.

MS. FORGIE: There's no question.

BY MR. LASKER:

Q. We agree in any event.

MS. FORGIE: No, I don't agree that 12:22 we agree. All this smoking stuff is just putting me right off smoking.

BY MR. LASKER:

Q. My question actually goes to the point I think you're trying to make on 12:22 page 7, and maybe I'm misunderstanding it.

But in your example on page 7, you discuss the possibility of another confounder, in this case, I think it's radon, making it more difficult to identify 12:22 an association between an exposure and outcome; correct?

MS. FORGIE: Object to the form.

THE WITNESS: This is really funny in a way because that's exactly what I'm 12:22

trying to teach my students that they should not confuse confounders and effect modifiers. In this case, it's an effect modification and not a confounding. That said, the same factor 12:23 can be an effect modifier and a confounder and/or a proxy. That's why I'm saying confounding is something we do mathematically. We have the data. We throw something in; we take something 12:23 out. But confounder is at the conceptual level. I need to decide is this a confounder? Yes or no? We have our rules for that. Is that a proxy for an exposure and not a confounder, or is 12:23 it acting as an effect measure modifier, and in this case, that was an effect measure modification.

BY MR. LASKER:

Q. So if I understand correctly, 12:23 effect measure modifier in this case is radon?

A. Uh-huh.

MS. FORGIE: Object to the form.

BY MR. LASKER: 12:23

Q. You have to say yes or no, obviously, for the court reporter.
A. Oh, I think that's how I build it. It could be either smoking or radon that I -- but I think it was radon that I called it the effect measure modifier. I'm not saying it, but I think that's correct. 12:24
Q. And the reason that in this example radon was an effect measure modifier that could impact the ability to conduct the analysis of smoking and lung cancer was because in your analysis the radon could result in ten extra cases of lung cancer per 100,000 miners; correct? 12:24
A. Yes. 12:24
Q. And it's the size of that association, if you will, that will determine the extent to which this effect modification could be -- could introduce a problem in conducting your epidemiological analysis; correct? 12:25
MS. FORGIE: Object to the form.
THE WITNESS: It is insofar a problem as effect measure modification comes into play when you're comparing 12:25

across populations. So you could in one population estimate a relative risk of 2 and another relative risk of 5, and we both would probably agree those are very different numbers. In one population you have an effect modifier present; in another you don't. So it is not that the association was the agent of interest is really different but that the comparison you're making are comparisons to a population at a different risk, baseline risk. And the extent to which the effect modifier could influence the odds ratio that -- of interest in a study will depend on how powerful an effect modification you have; correct. 12:25 12:26
MS. FORGIE: Object to the form.
BY MR. LASKER:
Q. In other words, let me just reword this. Maybe this would be easier. If the radon exposure added one extra case of lung cancer for 100,000 miners instead of ten extra cases of lung cancer for 100,000, that would have a fairly minimal impact on the 12:26

odds ratio that would be reported for smoking and lung cancer; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Fairly minimal is relative, but the number would be smaller. 12:26
BY MR. LASKER:
Q. Okay. And in -- and I think you can probably calculate it. It would probably be -- we'd be looking at -- 12:26
A. 5.05.
MS. FORGIE: There's no question.
BY MR. LASKER:
Q. Instead of -- the 20 out of four, you'd be looking at 31 out of 5 over 5; correct? In that scenario? Or I'm sorry. 12:27
MS. FORGIE: No, object to the form.
THE WITNESS: 31 over --
MR. WISNER: 21 over? 12:27
MR. LASKER: I think that's right.
MS. FORGIE: What's the question?
BY MR. LASKER:
Q. Instead of 21 over 4 it would be 31 over 5? 12:27

MS. FORGIE: Object to the form.
THE WITNESS: Well, 20 over 4 is ignoring radon.
BY MR. LASKER:
Q. Right. 12:27
A. So that's the fivefold increased risk due to smoking. So now if radon affects non smokers and smokers in the same way, then we would be adding one case to each. 12:28
Q. Right.
A. So we would have 21 over 5.
Q. Okay. 21 over 5?
A. Uh-huh.
Q. So then it would be 4.25 as opposed to 5. It would be a much smaller difference. 12:28
MS. FORGIE: Object to the form.
THE WITNESS: 4.25 is pretty big, but there's a difference to 5, yeah. 12:28
BY MR. LASKER:
Q. And so to be able to determine or to be -- if the issue is whether other pesticides are effect modifiers in conducting -- in looking at a glyphosate 12:28

non-Hodgkin's lymphoma association, one of the issues you can look at is how powerful of an association there is between these other pesticides and non-Hodgkin's lymphoma; correct? 12:28

MS. FORGIE: Object to the form.

THE WITNESS: That is not the only thing I would look at. I would also look at how correlated the exposures are with glyphosate. 12:29

BY MR. LASKER:

Q. But in this instance -- this example is not talking about a correlation?

A. No.

Q. I'm just trying to get the exposure 12:29 modification aspect of it.

MS. FORGIE: There's no question.

BY MR. LASKER:

Q. Are we on the same page here?

MS. FORGIE: Objection. 12:29

You're asking if you guys are on the same page?

MR. LASKER: I have to be able to ask the question without you objecting in the middle of it. Let me ask the 12:29

question again.

BY MR. LASKER:

Q. I want to focus on the effect modification point that you're making here, and that does not rely upon any correlation 12:29 between, in this case, radon and smoking; right?

MS. FORGIE: Object to the form.

THE WITNESS: This is an example where I'm trying to show in the first 12:29 part of this example how when you have one risk factor only assessment and you're comparing -- and you're calculating a so-and-so fold risk in the exposed over the unexposed, and you're 12:29 going to another population where now you have an additional risk factor for the outcome that adds to the baseline risk, and it adds in the same way in the exposed and the unexposed how you would 12:30 see a different odds at risk or rate ratio.

BY MR. LASKER:

Q. And my only point here, I guess -- and my understanding maybe I'm missing it 12:30

was that you were raising the possibility that other pesticide exposures might have an effect modification on glyphosate studies if you're looking at a population that has those other pesticide exposures and that 12:30 increases the background instance of NHL; is that correct?

MS. FORGIE: Object to the form.

THE WITNESS: Well, if we agree which pesticides are related to NHL and 12:30 one population of farmers is exposed to those, then we would presume that those farmers have a larger background rate of NHL.

BY MR. LASKER: 12:30

Q. Okay. And to be able to assess the extent to which that could create an exposure modification, we would need to consider the strength of that association between the other pesticides and 12:31 non-Hodgkin's lymphoma; correct?

MS. FORGIE: Object to the form.

THE WITNESS: No. What we need is enough sample size to then evaluate the effect of glyphosate. 12:31

BY MR. LASKER:

Q. Okay. But if the other pesticide exposures were resulting in one extra case of non-Hodgkin's lymphoma over -- out of a hundred thousand, that would have less of an 12:31 effect modification than if they were resulting in ten cases of non-Hodgkin's lymphoma out of a hundred thousand; correct?

MS. FORGIE: Object to the form.

THE WITNESS: That would depend on 12:31 the correlation of the exposures in this dataset. So the correlation of the pesticides was glyphosate.

BY MR. LASKER:

Q. And I guess so the effect 12:31 modification you present on page 7 depends upon the correlation between radon and smoking?

A. Yes.

Q. Okay. Moving on, we can take a 12:32 break for lunch now or go on for a little bit longer.

MS. FORGIE: It's up to you guys. I don't eat; so it doesn't matter to me.

MR. LASKER: Why don't we have 12:32

lunch now. It's a little bit of a short session, but it's probably a good time.
THE VIDEOGRAPHER: We're off the record at 12:32 p.m.
(Recess taken from 12:32 p.m. to 12:33 p.m.) 12:32
THE VIDEOGRAPHER: We are back on the record at 12:33 p.m.
BY MR. LASKER:
Q. Dr. Ritz, let's walk through some of the epidemiologic studies that you discuss in your report. I think the first study you talk about is the Cantor study from 1992. Why don't we mark that. 12:33
(Exhibit Number 19-10 was marked for identification.) 12:33
THE WITNESS: Actually, the Eriksson study that I mentioned first. Doesn't matter.
BY MR. LASKER: 12:34
Q. We'll get to Eriksson as well. 19-10. So the Cantor study reported an odds ratio for glyphosate and non-Hodgkin's lymphoma, and it's on page 2450 in this study in Table 6 of 1.1 with a confidence 12:34

interval of 0.7 to 1.9; correct?
A. Correct.
Q. And the odds ratio was adjusted as indicated in the footnote to the table for vital status, age, sex, smoking, family 12:34 history of lymphopoietic cancer, high-risk occupations, and high-risk exposures; correct?
A. Yes.
Q. And as Cantor is defining high-risk 12:35 exposures, if it meets a certain criteria, those could include exposures to other pesticides; correct?
A. As far as I remember, but I'm just looking for that definition. 12:35
Q. I think it is page 2448, top of the right-hand column just above "results."
MS. FORGIE: Where did you see it?
MR. LASKER: 2448, top of the right-hand column. 12:35
THE WITNESS: Yeah, it's the odds ratio of 1.5 plus. Is that it?
BY MR. LASKER:
Q. I believe so.
A. Yeah. 12:35

MS. FORGIE: Thank you.
BY MR. LASKER:
Q. Just so the record is clear, in the Cantor study the odds ratio was adjusted for vital status, age, sex, smoking, family 12:36 history of lymphopoietic cancer, high-risk occupation and high-risk exposures which can include other pesticides; correct?
A. Other substances it says, but I imagine it's pesticides included. 12:36
Q. And the CLR, if we were to calculate that confidence limit ratio for the glyphosate and non-Hodgkin's lymphoma, is 1.9 to 0.7. So that is slightly below 3.0; correct? 12:36
A. Yeah.
Q. And this is -- and you said you'd done this in your head. I don't know if you recall it in your head, but the CLR for the Cantor study is the smallest CLR for any 12:36 odds ratio, report odds ratio, where the odds ratio has been adjusted for other pesticide exposures; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I would need to check 12:37

the other -- De Roos, for example, which includes Cantor. I would imagine that De Roos is at least as powerful as Cantor; so it should actually be shorter. 12:37
BY MR. LASKER:
Q. If you look in your -- and this is an abbreviated short form, but De Roos 2003. You have -- we can get to the actual number if you want, but you have it on -- the 12:37 number that you used at least has a CLR that is well above 3; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I wouldn't be able to do that in my head without the numbers 12:37 right now. I have to guess where this is coming out, and I also need to -- oh, and this is a differently adjusted estimate, plus it's from a larger study. So it doesn't just include Cantor. It 12:38 also includes the Nebraska and some other study.
BY MR. LASKER:
Q. We'll look at the CLR for De Roos when we get there. We can just compare 12:38

them, but I think you stated that you thought the De Roos study might be at least as powerful as the Cantor study. Are there any other case control studies that you believe would be as powerful as the Cantor study, any measuring glyphosate in non-Hodgkin's lymphoma? 12:38

MS. FORGIE: Object to the form.

THE WITNESS: It depends on what the comparison is that I want to do. For example, ever handled is a very bad exposure assessment. So this 1.1 for ever handled I would judge as not very valid because the exposure is probably strongly misclassified non-differentially. 12:38 12:39

BY MR. LASKER:

Q. Except for three of the studies I believe -- let's strike this. Let's strike this. The odds ratio that you present in your expert report on page 14 are for ever/never exposure; correct? 12:39

MS. FORGIE: Object to the form.

THE WITNESS: Page 14? Which one? This? 12:39

BY MR. LASKER:

Q. Yeah.

A. I have to check it whether it's always ever/never. Did I not show any others? No, I guess they would be mostly ever/never. 12:39

Q. Okay. So with respect to that assessment that you have or that measure that you have on page 14 of your expert report, are you aware of -- and I'm going to give you -- talk also, and we'll put it in the NAPP which is a further pooling of the Cantor data and some other data from Canada. But other than that, is there any study that has greater power than Cantor with respect to the ever/never odds ratio for glyphosate-based herbicides in non-Hodgkin's lymphoma? 12:39 12:40

MS. FORGIE: Object to the form.

THE WITNESS: Actually I'm realizing something that I didn't realize before. This table actually says "odds ratios for ever having handled specific herbicides prior to 1965." I thought glyphosate was not available prior to 1965. 12:40

BY MR. LASKER:

Q. That would be the right column of the table, the left table. Left column is upper. 12:40

A. Oh, okay.

Q. Going back to the question then, other than the subsequent studies that pooled Cantor and included Cantor in the pooling, which would be De Roos 2003 and the NAPP, are you -- are you aware of any study that had a greater power to assess ever/never exposure to glyphosate in non-Hodgkin's lymphoma? 12:41 12:41

MS. FORGIE: Object to the form.

THE WITNESS: I wouldn't be able to tell off my head because I consider ever/never the lowest common denominator across all these studies, and I would hope that we have better measures to assess exposure than ever/never. 12:41 12:41

MS. FORGIE: Just so you know, it looks like the lunch is here. I'm not saying we have to break now.

MR. LASKER: We'll probably just continue through this. 12:41

MS. FORGIE: I agree.

BY MR. LASKER:

Q. And if I understand you correctly, that is because it's your opinion that ever/never analyses are not as informative on whether or not there is an association between glyphosate and non-Hodgkin's lymphoma as measures that try to look at the amount of exposure of glyphosate; correct? 12:41 12:42

MS. FORGIE: Object to the form.

THE WITNESS: An ever/never exposure presumes that any type of exposure I had can be handled in the same way. So somebody looking at a bottle of pesticides and spraying it once gets to be thrown in the same category as somebody applying pesticides on a regular basis in an occupation. And that is the least informative and the most capable of inducing non-differential exposure misclassification by people recalling wrongly. 12:42 12:42

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BY MR. LASKER:
Q. The -- in your expert report you opine, and I think this is at page 17 of your report. I'm sorry. On page 18 of your report. At the bottom of page 18 -- and you were right. This is the bottom of my head. I got it backwards as to which study you were doing first in your report. So bottom of page 18 you're talking about the Cantor study, going over to page 19; correct? 12:43
A. Yes.
Q. And you state that the Cantor study is less informative because the cases are diagnosed with non-Hodgkin's lymphoma between 1980 and 1983 which you state was at 12:43 most only six to ten years from the first potential glyphosate exposure; correct?
A. Correct.
Q. And you explain that this would be -- and just so the record is clear, we 12:44 are talking about here is the concept of latency; correct?
A. This talks about latency, yes.
Q. And the issue of latency is that you would need to have a certain period of 12:44

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time elapse from the time of exposure until the measure of non-Hodgkin's lymphoma for the biological process to take place that would lead to exposure to diagnose disease; correct? 12:44
MS. FORGIE: Object to the form.
THE WITNESS: Latency -- the word "latency" is used in different ways and in epidemiology we are trying to figure out the minimum time between an exposure 12:44 happening and causing the disease. So in a time-changing exposure and a cumulative or a -- not an exposure like the A bomb that's one time -- right? -- you kind of have to decide when the 12:45 potential for carcinogenicity has occurred, and from that point of time to when you're actually diagnosing the disease. That may be very different depending on many factors including age 12:45 of the subject.
BY MR. LASKER:
Q. Right. And the point that you're making with respect to Cantor, and I think you state this on page 17 of your report 12:45

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about in the middle paragraph -- I'm sorry, in the first paragraph about halfway down, you state that typically we would generally expect a five to ten-year minimum latency between exposure and disease onset for blood 12:45 system-related cancers; correct?
A. That's read correctly.
Q. So what that means is even if you have -- let's say if you have a known carcinogen that causes NHL, it would take a 12:46 minimum of five to ten years from the date of exposure for the regression from cellular insult to result in a diagnosable case of non-Hodgkin's lymphoma; correct?
MS. FORGIE: Object to the form. 12:46
THE WITNESS: No. I'm using this in terms of epidemiologic latency time which we are estimating was in groups. So we are never estimating for one person. So in one person, it could be 12:46 happening within a year or two. In another person, it might not be happening until 35 years out. That's why I also refer to age. For example, somebody who is already age 60 and is 12:46

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more susceptible to exposures, that cancer might just happen earlier after exposure than in somebody where the cancer cell is dormant and kept in check by the immune system and other factors 12:47 for 20 more years. So the latency period is really an average or minimum dependent on what population I'm looking at and whether I allow for that population to age into the time when the 12:47 cancers would occur.
So mostly I would imagine I have higher power in my study when the people are aged into that age when they actually have cancer. 12:47
BY MR. LASKER:
Q. And the concern that you're raising with respect to the Cantor study is that -- well, actually let me just take a step back here. You state -- and I think this is on 12:47 page 19. You state that one would prefer for NHL cancer epidemiology study, one would prefer a minimum latency period of on average ten years; correct?
MS. FORGIE: Object to the form. 12:48

THE WITNESS: That's what this says.
BY MR. LASKER:
Q. This is you.
A. Yes, yes, this is what the sentence says. So what I was meaning by this is that a study would be more powerful if we allowed for longer latency because we then would capture more cases due to the exposure. Because if you're only allowing for two years, you would only capture those people who was in those two years come down with the cancer. If you allowing for five years, you can see how that number would increase and then ten years, 20 years out.
So depending on how long we have between the first exposure or the minimum exposure necessary to cause cancer and the events that later occur, the longer the latency, the more chance I have to capture every single case that was actually caused by the exposure because there are these dormant cells.
Q. Just so I understand also because I think there's a couple things going on, but

correct me if I'm wrong. One issue is that you want to be measuring the exposures that could have, in fact, resulted in the outcome; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I'm not sure I understand, but yes, we want to measure exposures as carefully as we can to estimate whether they are causing the outcome.
BY MR. LASKER:
Q. So, for example, and just take an extreme example, if you were to do an epidemiologic study and you measured an exposure on Tuesday and the individual came -- was diagnosed with non-Hodgkin's lymphoma on Wednesday, whatever the exposure was on Tuesday wouldn't have been a cause of the NHL because there hasn't been a sufficient time that has elapsed for the causal mechanism to take place; correct?
A. If I'm assuming that the only exposure the person ever had was on Tuesday.
Q. Right?
A. Yes.

Q. And one of the issues you're raising in the Cantor study is if you're not looking back sufficiently far in time, then you are not capturing exposures that could have had sufficient time to go through that process whereby they would result in a diagnosable non-Hodgkin's lymphoma; correct?
MS. FORGIE: Object to the form.
THE WITNESS: So what I'm trying to say here is that exposures have to occur a certain number of, let's say, days, years, months prior to the onset of a cancer before I would think that it is biologically possible or plausible. But that could be a year in a certain circumstance, two years in another, and on average, it might be very different depending on the population I'm looking at.
BY MR. LASKER:
Q. And the point you make here on page 19 is you could have traits that vary but for a study of non-Hodgkin's lymphoma, you'd prefer a minimum latency period of on average ten years to make sure that you are

capturing the biologically plausible exposures that could account for any reported non-Hodgkin's lymphoma; correct?
MS. FORGIE: Object to the form.
THE WITNESS: That's not correct. That's really not what this says. What this says is that there is an exposure lag time that I would like in order to capture every single case and not just the ones that are the early birds.
BY MR. LASKER:
Q. If you have, though, an early bird if you will, one of the issues that you're trying to account for is the possibility that that earlier diagnosed non-Hodgkin's lymphoma would have been related to something that predates any exposure; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Well, when I have a study that only has a two-year minimum follow-up and no more, then I always have to raise that possibility. That's why I would like a study that has a longer period of time between the

exposure and the outcome so I can estimate what an average mild latency might be. And if I have a study that only follows for one year, I would probably be concerned. With a study following two years, less, three years, less, et cetera, et cetera. 12:53

BY MR. LASKER:

Q. What you're mentioning here with respect to Cantor is that you have a concern because only six to ten years have elapsed between a potential first glyphosate exposure and an NHL diagnosis; correct? 12:53

A. Well, my concern is not with respect to the biologically relevant latency period but with respect to having really captured all NHLs that might have been caused by the exposure because I presume that, in this case, I only captured the early birds, the people who got their cancer relatively soon after exposure. 12:53

Q. You would have to, though, in determining that those non-Hodgkin's lymphomas that you see are attributable to the exposure, one factor that you would also 12:54

want to consider is whether or not those exposures took place during the time period sufficiently before the diagnosis that you could attribute the exposure to the outcome; correct? Because before you did the study, you don't know there's an association; right? 12:54

MS. FORGIE: Object to the form.

THE WITNESS: Well, it depends on which study I'm conducting, but before this study was conducted, I don't think there was much known about glyphosate. So I agree. So this is certainly a study that is trying to evaluate something we know very little about, and of course, we always want the most information we can get and the longest period between exposures. 12:54

But as a public health official, I want to look right away. I want to look after two years and three years and four years, but if I don't see something after two years or three years, then I want to look after five years because it doesn't mean there's nothing when I 12:54 12:55

don't see something after two years.

And in epidemiology, what we often do in order to remove exposures that are irrelevant is we are discounting exposures within the year before diagnosis, and that's a tool one can use. 12:55

BY MR. LASKER:

Q. And one of the things that you talk about with another study, with the Eriksson study is a lag period of ten years because in that study, that was the demarcation; correct? 12:55

A. Yes, that's correct.

Q. Okay. And that goes to the same issue that you're raising which is that for hematopoietic cancers, you might need a period of ten years before the exposure could actually give rise to diseases so that you can actually measure an effect; correct? 12:55

MS. FORGIE: Object to the form.

THE WITNESS: That's incorrect. That's actually stating the opposite of what I said. What I'm saying is that you want that -- actually for 12:55

hematopoietic cancers, it's generally in the radiation literature -- and that's where I wrote my dissertation in -- assume that it's two-year minimum. And so what we would do is we would look carefully and critically maybe at around one year or two year, but these are all presumed. 12:56

And they come from the medical literature on radiation effects -- side effects. They are not coming from population studies and workers and the general population. So what we think the case is is that if you say one day or a month, everybody would shake their head. Maybe even one year we would shake our heads and say I'm not really sure. But anything beyond one year would definitely raise concern. 12:56

Because we are also now talking about initiation of cancer or promotion of cancer, and initiation of cancer might take longer than promotion. Promotion might be the last step in the chain of events, and that might be very 12:56 12:57

soon.
So again, what I'm saying is that I would like to move out from the time of exposure that is relevant for the cause of the disease. I would like to move out as long as I can in order to capture as many cases caused by that exposure as possible.
So ten years out is a good time frame because it makes me more comfortable that I'm not only capturing early birds but that I'm really looking at the chronic consequences of that exposure.
BY MR. LASKER: 12:57
Q. Understood.
So with respect to the Cantor study then, if I'm understanding you correctly, your concern was -- with respect to latency was solely a concern about power? 12:57
MS. FORGIE: Object to the form.
THE WITNESS: No, it was not about power, but it was a concern about this study not -- being a little bit early in the sense that the chronic effects could 12:58

not be assessed as comprehensively as I would have liked to and later studies do a better job.
MR. BAUM: Is this a good time to switch over to lunch? 12:58
MR. LASKER: Almost.
BY MR. LASKER:
Q. Now, in your analysis, you were assessing the start date, if you will, of glyphosate as a potential exposure in 1974; 12:58 is that correct?
MS. FORGIE: Object to the form.
THE WITNESS: Well, we don't really know unless the author tells us exactly when the exposure happened, but the 12:58 potential for exposure starts in '74, yes.
BY MR. LASKER:
Q. Do you know when glyphosate was first approved for use in agricultural 12:59 settings?
A. I thought that was about that time.
MR. LASKER: Let's just mark the next exhibit in line.
MR. BAUM: Eric, it's 1 o'clock. 12:59

MR. LASKER: We're going to be about five minutes. It's still all in the context of this.
(Exhibit Number 19-11 was marked for identification.) 12:59
MS. FORGIE: What number are we on?
MS. SHIMADO: 11.
BY MR. LASKER:
Q. And this will be, and I'll -- obviously, you're going to have to -- well, 12:59 I'll represent and I'm going to ask you a question on the assumption my representation is correct. I'll represent to you that this December, 1975, letter from EPA marks the first date on which glyphosate-based 01:00 formulation was approved for use in agricultural settings.
A. Uh-huh.
MS. FORGIE: There's no question.
BY MR. LASKER: 01:00
Q. If that assumption is correct for farming studies, and these are -- the Cantor study was specific to farming exposures in calculating that latency period, would I be correct, then, that December, 1975, would be 01:00

the starting point for that calculation?
MS. FORGIE: Object to the form.
THE WITNESS: We are presuming that this is the only way to get glyphosate use. 01:00
BY MR. LASKER:
Q. This is the first approval for agricultural settings. It would be used as sort of right of way and roadways for road crews. It could have been used before then, 01:00 but the first approval for farmers for use of glyphosate was in December of 1975.
A. And that --
MS. FORGIE: Wait. There's no question pending. 01:01
BY MR. LASKER:
Q. With that assumption in mind, if you're trying to measure farming exposures, which was the exposures in the Cantor study which was the farmers exposure, I think by 01:01 its definition and by its terms, would December of 1975, then, be the proper start point for determining the potential latency period between exposure and disease outcome?
MS. FORGIE: Object to the form. 01:01

Asked and answered. She just answered that exact question.
You can answer it again.
THE WITNESS: Well, I have to make certain assumptions. One was that they actually didn't ask other occupations, such as road worker, and also that these farmers weren't given glyphosate in trial runs because there's a difference, and I thought I'd seen that somewhere listed that actually glyphosate was being tried out in certain farming populations prior to general approval. 01:01 01:01
BY MR. LASKER:
Q. Okay. I'm not sure where you've seen that, but for the purpose of this question, if we assume that December, 1975, was the first date where glyphosate was approved for agricultural uses, for farm uses, and that none of the farmers here were using it for some trial purposes before its official approval, would December, 1975, then, be the proper starting point for then calculating the latency period for the Cantor study? 01:02 01:02

MS. FORGIE: Object to the form. Asked and answered.
You can answer it again.
THE WITNESS: Well, if that is what they are actually assessing, then you would have potential exposure starting at the time this agent became available to the farmers, and then you could use that for a latency period calculation. 01:02
MR. LASKER: Why don't we take a break for lunch. 01:03
THE VIDEOGRAPHER: This marks the end of videotape number 2 in the deposition of Dr. Beate Ritz. We're off the record at 1:03 p.m. 01:03
(Lunch recess taken from 1:03 p.m. to 1:46 p.m.)
THE VIDEOGRAPHER: We are back on the record at 1:46 p.m. This marks the beginning of videotape number 3 in the deposition of Dr. Beate Ritz. 01:46
BY MR. LASKER:
Q. Dr. Ritz, let's move on to the De Roos 2003-case control study. We'll mark that as the next exhibit in line. 01:46

(Exhibit Number 19-12 was marked for identification.)
BY MR. LASKER:
Q. Dr. Ritz, we can walk through this if you'd like, but I feel you probably already have done that. The median latency time for the NHL cases in this study is roughly equivalent to the median latency time for the cases in the Cantor study; correct? 01:47 01:47
A. As far as I know, it went out a little bit longer in Minnesota.
Q. No, I think you're talking Nebraska was longer and Kansas City was shorter.
MS. FORGIE: Wait. Is there a question? 01:48
MR. LASKER: I'm working my way through it.
THE WITNESS: Nebraska is the longest followed by Minnesota and then Kansas. 01:48
BY MR. LASKER:
Q. And Kansas was shorter?
A. Correct.
Q. And Nebraska was longer. So 01:48

roughly the median -- most of the data was the same as Cantor, and then you have some shorter and some longer; right?
MS. FORGIE: Object to the form.
THE WITNESS: It depends on how many people were in each of those studies. 01:48
BY MR. LASKER:
Q. You can look on Table 2.
A. Yeah, Iowa and Minnesota is the biggest chunk of it. 01:48
Q. And then the other two are both about the same?
A. Yeah.
Q. So can we agree the median latency period for the De Roos 2003 study is roughly equivalent to the median latency period for the Cantor study? 01:48
MS. FORGIE: Object to the form.
THE WITNESS: We can calculate it, but it probably would come out similarly, but it's important that we also have longer latency in there, in Nebraska. 01:48
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BY MR. LASKER:
Q. Right. But the median latency is the same. We have shorter latency for the roughly 15 or 16 percent from Kansas and slightly longer latency for the 17.4 percent in Nebraska; correct? 01:49
MS. FORGIE: Object to the form.
THE WITNESS: 21.5 percent in Nebraska.
BY MR. LASKER: 01:49
Q. I was looking at the analysis of multiple pesticides.
A. Oh.
Q. Correct?
MS. FORGIE: Object to the form. 01:49
THE WITNESS: 17.4, yes.
BY MR. LASKER:
Q. Okay. With respect to the Nebraska data which is, as you mentioned, is data that's somewhat longer, that goes out from July 1983 to June 1986? 01:49
A. Correct.
Q. Even in that sub population litigation --
(Interruption in the 01:50

proceedings.)
MR. LASKER: Back on the record.
BY MR. LASKER:
Q. Even for the 17 percent of the data that came from Nebraska, you still would not have a median latency period for glyphosate for ten years; correct? 01:50
MS. FORGIE: Object to the form.
THE WITNESS: That makes assumptions that we're starting to count in 1975 which may or may not be correct. But that gives us eight years, I guess. 01:50
BY MR. LASKER:
Q. Whether it's '74 or '75, the maximum latency period would be -- maybe the maximum would be 12 years, but we're talking the median latency period. The median latency period even for this Nebraska subgroup would be less than ten years; correct? 01:50
MS. FORGIE: Object to the form.
THE WITNESS: About ten years.
BY MR. LASKER:
Q. Let me make sure I understand the median latency period. This would allow -- 01:51

if everybody had taken glyphosate the very first day that it was available, that would be the latency period, but, of course, that's not going to be the reality in the study; correct? 01:51
A. I don't know --
MS. FORGIE: Object to the form.
THE WITNESS: I don't know what the reality in the study is because it's not stated exactly when these farmers started, and if we are presuming that the EPA date is the earliest one, and you said yourself there were other uses for glyphosate, so who knows? Farmers do all sorts of things including buying things that are not EPA approved. So I don't know. 01:51
BY MR. LASKER:
Q. So there are two parts of this: When you talk about median latency, there is, in this case, a maximum latency period of whenever you want to start measuring 1974, 1975 through to the date of diagnosis. That would be the maximum latency period possible. 01:51 01:52

A. Correct.
Q. The actual median latency for the population that's being studied would be less than the maximum latency period; correct? 01:52
A. It would be somewhere in between the diagnosis dates, and the diagnosis dates are July, '83, through June, '86.
Q. I understand that. That would be when diagnosis was. The exposure -- the median period of exposure would not be ten years before that. It would be somewhat less. At some point in time prior to diagnosis that they're exposed, not the very first day; correct? 01:52 01:52
MS. FORGIE: Object to the form and asked and answered.
You can answer again.
THE WITNESS: Well, it depends what we are presuming about the exposure. So if we are presuming that they really only started using in 1975, and they were using a certain amount of glyphosate that needed to be used in a certain way, they might have used, you 01:52 01:53

know, a huge amount the first time around because they were told it's very non-toxic and maybe all of the relevant exposure were in the first year. I don't know. They did not investigate that. 01:53

BY MR. LASKER:

Q. Okay. I understand that.

But with respect to, as an epidemiologist if you're looking at this study and you don't have the data on when exposures took place, would you assume then in your analysis of the Nebraska data for purposes of assessing the data that all of the exposures to Roundup took place on the first date that exposures were possible? 01:53

MS. FORGIE: Object to the form. Asked and answered.

You can answer it again.

THE WITNESS: Well, I would probably look at a range of possible times, and then you can, you know, use that in your analysis. 01:53

BY MR. LASKER:

Q. Okay. And if you were to do that 01:53

analysis, the median latency period, even of the Nebraska data, would be less than ten years; correct?

MS. FORGIE: Object to the form. Asked and answered. 01:54

THE WITNESS: Not necessarily because the Nebraska diagnosis median is 1985. So that's ten years after 1975.

BY MR. LASKER:

Q. I understand that. Let me just make sure I understand this. You mentioned that you had used some sort of range that determined likely first exposure date. 01:54

It wouldn't all be assumed to be 1975; correct? 01:54

MS. FORGIE: Object to the form. Asked and answered. She's testified --

THE WITNESS: That would be a kind of sensitivity analysis you might want to play with. 01:54

BY MR. LASKER:

Q. And if that analysis were conducted, the median latency period for even the Nebraska, 17 percent in this study could be less than ten years; correct? 01:54

MS. FORGIE: Object to the form. Asked and answered.

You can answer it again.

THE WITNESS: Well, I could define a range that would make it less than ten years, but if I subtract 1985 and 1975, I have ten years on average. 01:54

BY MR. LASKER:

Q. Okay. And you talked earlier about the issue -- we were talking about this in connection with the Cantor study about the power of this study to be able to identify association. So I'd like to ask you about that. 01:55

I'd asked you about the CLR for De Roos, and we now have that data; so I'd like to return to that discussion. The glyphosate data is presented on Table 3; correct? 01:55

A. Correct. 01:55

Q. And for the logistical regression analysis which is the analysis that you report on in your expert report, we have a confidence interval that ranges from 1.1 to 4.0; correct? 01:56

A. Yes.

Q. So that confidence interval is -- I'm sorry, the CLR for that, and I've done the math, but it's going to be about 3.6, and you can sort of eyeball that; right? 01:56

A. Yeah.

MS. FORGIE: Object to the form.

BY MR. LASKER:

Q. And for the hierarchical regression odds ratio, we have 2.8 over 0.9; so the CLR for the hierarchical regression would be slightly above 3; correct? 01:56

A. Yes.

Q. So the CLR for both of the De Roos 2003 odds ratios for glyphosate are larger than the CLR for the Cantor 1992 study; correct? 01:56

A. What did we have for that again?

Q. You can go back. It's 2.7, but why don't you look at it just to confirm for yourself. 01:56

MS. FORGIE: Do you remember what exhibit it is?

MR. LASKER: It's probably the last one we just did. 01:56

MS. SHIMADO: 10. Exhibit 10.
BY MR. LASKER:
Q. You should have it right there.
A. Yeah.
Q. For the record, I'll ask the question again while you're looking at this. 01:57
The CLR for both of the logistic regression analysis and the hierarchical regression analysis in the De Roos 2003 study is actually larger than the CLR for the Cantor study; correct? 01:57
A. That is correct.
Q. Am I correct, though, in my understanding that the -- your concern -- while you're concerned about the latency period in the Cantor study as making that study less informative, you do not have that same concern for the De Roos 2003 study? 01:57
A. Well, first to the '95 percent confidence interval, the confidence interval widens with the number of adjustments I make. Obviously, De Roos makes a lot more co-adjustments than Cantor, and that's probably the reason why these confidence intervals are wider. So in a way, actually 01:57 01:58

her estimate would be the more fully adjusted compared to the Cantor.
With respect to latency, the same rules apply. However, she added some studies that actually had longer latency. 01:58
Again, the latency issue is an issue because I'm missing cases that are truly caused by the exposure, if I believe exposure causes disease, and so it has to do with early studies where I'm catching these early cases and not yet the later ones. 01:58
Q. Let me just sort of step back, though, because there's a lot in that answer, and I want to make sure I understand that fully. 01:58
Is it your testimony that the logistical regression analysis in De Roos 2003 had more controls, adjusted for more factors than the hierarchical regression?
MS. FORGIE: Object to the form. 01:59
THE WITNESS: No, that's not what I said. The hierarchical regression makes additional assumptions that we can debate and that are debated. You will not see many -- she is actually one of 01:59

the first people to ever use hierarchical regression in a systematic way in the literature.
There are a few more papers here and there. I did it myself in 2002. 01:59
Somehow hierarchical regression has fallen out of favor because you have to make a lot of assumptions, and reviewers actually constantly fight with you over those assumptions whether they're correct or not. So generally, we would go back in a consensus manner to a normal logistic regression in which we are adjusting for as many variables that we think make validly sense to adjust for. 01:59 01:59
And this estimate of 2.1 was the confidence interval of 1.1 to 4, had wider confidence interval even though there are more cases and more controls in the analysis. The only way this happens is if there is more full adjustment for cofactors to widen these confidence intervals. 02:00
So from that, I conclude that she 02:00

must have adjusted for a lot more than Cantor.
BY MR. LASKER:
Q. Let me just step back here because that was my question. The confidence interval for the hierarchical regression is narrower than the confidence interval for the logistic regression analysis? 02:00
A. Correct, and that's by method. By making more assumptions, you're narrowing confidence intervals. That's how hierarchical regression works. 02:00
Q. Let me step back so I make sure I understand the question -- understand the answer to my question. 02:00
In the Cantor 1992 study, you raised concerns about a median latency period of less than ten years as making that study which had a 1.1 adjusted odds ratio, in your mind, less informative. And I'm just trying to understand if that same concern about the median latency period of less than ten years makes the De Roos 2003 study which has that hierarchy ratio that you cite less informative. 02:01 02:01

MS. FORGIE: Objection. Object to the form. Asked and answered.

You can answer.

THE WITNESS: Cantor is part of the study; however, the beauty of pooled studies is that they pool across different studies with different strengths and different weaknesses. It helps for the sample size. It helps for the statistical power. In this case, it helps even to adjust for more variables that you would be happy to adjust for, and overall, it's more powerful because of all of these reasons. 02:01

BY MR. LASKER: 02:02

Q. That wasn't my question. My question was that you, in your expert report, cited to a median latency period for NHL of less than ten years as a reason why the Cantor study was less informative, and the 1.1 odds ratio in that study was less informative to you. 02:02

The De Roos 2003 study has a median latency period of less than ten years. My question to you is whether that fact makes 02:02

the De Roos 2003 study less informative?

MS. FORGIE: Object to the form. Mischaracterizes her testimony and asked and answered.

You can answer it again. 02:02

THE WITNESS: Again, the latency period in Cantor cannot be different from what the latency period of the part of the data that is Cantor data in this pooled analysis is. So it is what it is. 02:02

However, adding additional states and additional data improves what this study can do over the Cantor study. Plus it overall increases the latency because we have the Nebraska study as well. 02:02

BY MR. LASKER:

Q. Okay. But we also have the Minnesota study which has a shorter latency period; correct? 02:03

MS. FORGIE: Object to the form.

THE WITNESS: It's likely shorter. Yes.

///

BY MR. LASKER:

Q. Just to clarify, the Kansas study has a shorter period?

A. Kansas, yes.

Q. So again, my question is -- and it may or may not -- but does the fact that the De Roos 2003 study has a median latency period of less than ten years, in your assessment, does that, in your mind, make the De Roos 2003 study less informative? 02:03

MS. FORGIE: Object to the form. Mischaracterizes her testimony. Asked and answered.

You can answer it again.

THE WITNESS: I think De Roos is a really excellent study that did everything we can do in terms of pooling data in terms of relating the exposures that she had access to to the outcomes in adjusting and trying different methods and in actually lengthening the overall latency by including Nebraska. 02:03

MR. LASKER: Mark that answer. I'm going to ask the question again.

///

BY MR. LASKER:

Q. In your opinion, does the fact that the De Roos 2003 study has a median latency of less than ten years make that study less informative? 02:04

MS. FORGIE: Objection. Object to the form. Mischaracterizes her prior testimony, asked and answered. This is, like, the fifth time you've asked the same question. 02:04

THE WITNESS: Now I'm really confused because I don't know anymore what you mean by "less informative."

BY MR. LASKER:

Q. Okay. Well, that was your terminology with respect to the Cantor study. 02:04

A. Correct.

Q. And you stated that the Cantor study was less informative because it had a median latency period of less than ten years. My question is: Do you believe that the De Roos study is less informative because it has a median latency period of less than ten years? 02:04

MS. FORGIE: Objection. Object to the form. I object to the mischaracterization of her prior testimony. Asked and answered six times. 02:05

You can answer it again.

THE WITNESS: So the De Roos study generally is a better study than the Cantor study because it pools data. So it's not less informative. It's 02:05 actually more informative, that it cannot go beyond the latency period of one of the studies included for that data is a no-brainer.

However, she added data with a 02:05 longer latency; so she is actually now covering all sorts of latency periods that we can look at. And the longer, of course, we would have a latency period, the more powerful. If she had another 02:05 study to add, it would become more powerful, but it is an incremental step going from one study that may be less informative to two studies that are more informative to three studies that are 02:05

even more informative.

BY MR. LASKER:

Q. And the Nebraska data is from a case control study that was published by Dr. Zahm; correct? 02:06

A. Yes, Sheila.

Q. And Dr. Zahm in her published case control study did not report any association between glyphosate and non-Hodgkin's lymphoma, did she? 02:06

A. Can you show me that?

Q. Sure.

(Exhibit Number 19-13 was marked for identification.)

BY MR. LASKER: 02:06

Q. Again, my question is Dr. Zahm, in her paper, does not report any -- specifically any association or positive association between glyphosate and non-Hodgkin's lymphoma; correct? 02:07

MS. FORGIE: Take as much time as you want reading it.

THE WITNESS: It looks like this is a study specifically analyzed for 2,4-D and some more general pesticide 02:07

exposures.

BY MR. LASKER:

Q. My question to you is: In the published paper addressing the Nebraska data that was pooled in De Roos 2003, the 02:07 investigators, Zahm, et al., do not report any association between glyphosate and non-Hodgkin's lymphoma; correct?

MS. FORGIE: Objection. Object to the form, and asked and answered. 02:08

You can answer it again.

THE WITNESS: So the beauty of pooled studies is that I can do things that I can't do in a single study. I presume that Sheila thought she could 02:08 not analyze certain types of pesticide based on what is 201 cases.

So that would be normal procedure to then make this data available for a larger pooled study for pesticide 02:08 exposures that are less common.

BY MR. LASKER:

Q. My question was -- and I still I'm not sure I've gotten -- I still haven't gotten an answer. Dr. Zahm, in her 02:08

published case control study, looking at that Nebraska data that was then pooled into De Roos 2003, does not report any association between glyphosate-based herbicides and non-Hodgkin's lymphoma; 02:08 correct?

MS. FORGIE: Objection. Object to the form, asked and answered. This is the fifth time she's answered.

You can answer it again. 02:09

THE WITNESS: So the pooled data is not what is being reported on here. There's a difference between a study and a study report. Usually when you do these studies, they're very expensive. 02:09 You collect a lot more data than what you can report in one paper, and for your career, you better publish more than one paper.

There's always the issue of common 02:09 and less common exposures; so when I collect as extensively as I can any kind of occupational exposure, I might or might not have the statistical power to investigate every of those exposures in 02:09

my study that is relatively limited since there are only 201 white males as cases.

So in that case, I provide this data for a collaborative effort and Dr. De Roos' paper is such a collaborative effort where then I provide them with a lot more data than I would be -- you see that she is the second author here, and Dr. Blair is the last author. So they would have had access to more data than this paper is actually reporting on. 02:10

MR. LASKER: I'm going to have the reporter mark that answer again. I'm going to ask the question one more time to see if I can get an answer. If not, we'll just have to address this with the Court later. 02:10

MS. FORGIE: I object to the statements about not getting an answer -- 02:10

MR. LASKER: That's fine. Just object.

MS. FORGIE: It's unfair. 02:10

BY MR. LASKER:

Q. Dr. Ritz, in her published paper, case controlled paper, looking at the Nebraska data that was subsequently pulled into De Roos 2003, Dr. Zahm does not report any association between glyphosate and non-Hodgkin's lymphoma; correct? 02:10

MS. FORGIE: Objection. Object to the form and asked and answered. This will be, like, the eighth or ninth time she's answered the same question. 02:10

You can answer it again.

THE WITNESS: This data in the Zahm publication from 1990 is not the data that was pooled into this pooled study. This is data specifically for one type of application. What I imagine Dr. Zahm provided to Dr. De Roos is a much more extensive dataset and the De Roos study is based on that more extensive dataset. 02:11

BY MR. LASKER:

Q. The De Roos study is looking at 187 cases in its pooled analysis and 113 cases in its analysis of multiple pesticides from Nebraska; correct? 02:11

A. 113, yes.

Q. And the Zahm published paper had, would you say, over 200 cases of non-Hodgkin's lymphoma; correct?

A. 201. 02:11

Q. Okay. De Roos and her co-investigators in the 2003 paper discuss their findings with respect to glyphosate in their conclusion -- in the concluding section; correct? Or I guess in their discussion section? 02:12

A. Yes.

Q. And on page 7 of 9, the paragraph -- sort of the second paragraph from the end of the bottom of the second column on page 7 is where De Roos and her co-investigators discuss their findings with respect to glyphosate; correct? 02:12

A. This one? The second to the last.

Q. Glyphosate -- 02:12

A. Yeah, yeah.

Q. In that discussion, they talk about the -- they cite to the Hardell paper, and they cite to the McDuffie paper; correct?

MS. FORGIE: Objection. 02:13

THE WITNESS: I see a citation to a Williams paper and a Hardell paper.

BY MR. LASKER:

Q. Number 51 --

A. And 51. 02:13

Q. -- and number 8 is the McDuffie paper; correct?

A. Oh, 8, yes.

Q. So they cite to the McDuffie paper and the Hardell paper; correct? 02:13

A. Yes.

MS. FORGIE: Objection.

BY MR. LASKER:

Q. And they state that these few suggested findings provide some impetus for further investigation into the potential health effects of glyphosate; correct? 02:13

A. It seems like they are citing Williams here.

Q. I understand that. 02:13

The conclusion that De Roos and her co-investigators provide in their discussion in their paper after reviewing the other epidemiological studies they cite, Hardell and McDuffie, after they've done their 02:14

analysis as well for the pooled data from the U.S. case controlled studies, was that these were suggested findings that provide some impetus for further investigation into the potential health effects of glyphosate; correct? 02:14

MS. FORGIE: Object to the form.

THE WITNESS: The way I read this is that they are commenting on Hardell and McDuffie. 02:14

BY MR. LASKER:

Q. They do not -- De Roos and her co-authors do not anywhere in their paper state that their study in combination with the earlier epidemiological studies supports a conclusion that there has been shown a causal association between glyphosate and NHL, do they? 02:14

MS. FORGIE: Object to the form.

THE WITNESS: Well, they're actually saying, "Our results indicate increased NHL incidents by number of pesticides used only for the subgroup of potentially carcinogenic ones," and then they list them. 02:14 02:15

BY MR. LASKER:

Q. They do not list glyphosate; right?

MS. FORGIE: Wait. She hasn't finished her answer. Please let her finish. 02:15

THE WITNESS: I'm looking for the glyphosate. No, that's the general statement.

BY MR. LASKER:

Q. Okay. 02:15

A. But you would need to look at the list of what she considers potentially carcinogenic which is on Table 1, and you will see that glyphosate was one of them because it got a .3. 02:15

Q. In her -- in De Roos' discussion, if I can direct you to page 6 of 9, she has data there for combined pesticide use, Table 5.

Do you see that? 02:16

A. Yes.

Q. And one of the analyses that they conduct is a combined analysis of atrazine and dicamba; correct?

A. Yes. 02:16

Q. And as it happens, their findings for their logistic regression and their hierarchical regression for atrazine and dicamba combined are almost identical to their findings for glyphosate alone; correct? 02:16

MS. FORGIE: Object to the form.

THE WITNESS: I don't know what you mean by "identical."

BY MR. LASKER: 02:16

Q. Well, for atrazine and dicamba in their logistical regression, they had an odds ratio of 2.1 which is the same odds ratio as glyphosate had in logistical regression; correct? 02:17

A. Yes, but odds ratio of 2.1 or .7 or .3 you can find all over this table.

Q. And the confidence interval for the logistic regression analysis for 2.1 was marginally significant and very similar to the confidence interval for glyphosate alone; correct? 02:17

A. Correct. But you can see that it is based on very different data. It's based on 31 cases and 60 controls in that category 02:17

versus 36 and 61 for glyphosate. So it's not the same people.

Q. Right. I wasn't suggesting it's the same people.

The hierarchical regression analysis, the conclusion for atrazine and dicamba combined was a 1.6 odds ratio which is the same odds ratio reported for glyphosate; correct? 02:17

MS. FORGIE: Object to the form. 02:17

THE WITNESS: Well, yeah, I mean, when we do these kind of analyses, a lot of odds ratios might be the same.

BY MR. LASKER:

Q. And the confidence interval for the hierarchical regression analysis for atrazine and dicamba combined is, again, virtually identical to the odds ratio for the hierarchical regression analysis for glyphosate; correct? 02:18 02:18

MS. FORGIE: Object to the form.

THE WITNESS: Not surprising given the assumptions they made for the hierarchical regression.

///

BY MR. LASKER:
Q. And in discussing those odds ratios, 2.1 for the logistic regression analysis that is just statistically significant and a 1.6 for the hierarchical regression analysis that's not significant in connection with atrazine and dicamba on page 6 in their study, and it is in the text right above the words "Discussion," De Roos states that those findings were "probably misleading due to imprecision of estimates noting that these results did not hold up following shrinkage and hierarchical regression analysis according to our prior distribution of complete exchangeability"; correct? 02:18 02:18 02:19 02:19
A. That's what this says. I mean, the text.
Q. And to the extent that -- I take it you would not view the identical -- or not nearly identical odds ratios reported for glyphosate in the same study as being probably misleading; correct? 02:19
MS. FORGIE: Object to the form.
THE WITNESS: You are comparing two 02:19

tables you absolutely cannot compare. The result for atrazine and dicamba both, it's what we call an interaction term, and what she is comparing here is they seem to be indicative super additivity and results from logistic regression. 02:19
And what this next sentence is referring to, such as for atrazine and dicamba, were probably misleading. So the misleading is the super additivity and not the effect estimate. 02:19
BY MR. LASKER:
Q. Let's go on to the Lee study just briefly. That's Lee 2004. 02:20
MS. FORGIE: Are we putting these away?
MR. LASKER: For now, yes.
(Exhibit Number 19-14 was marked for identification.) 02:20
BY MR. LASKER:
Q. The Lee study is another pooled analysis here using two of the three studies that were used in De Roos 2003; correct?
A. Correct. 02:20

Q. The Lee study reporting its results does not adjust for exposures to other pesticides; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I have to check that. 02:21
BY MR. LASKER:
Q. Table 3 on page 300.
A. The Lee study does not give you an effect estimate for glyphosate. It gives you a stratified analysis by asthmatics and non-asthmatics for glyphosate. 02:21
Q. And in that stratified analysis, they do not adjust for exposures to other pesticides; correct?
MS. FORGIE: Object to the form. 02:21
Asked and answered.
You can answer it again.
THE WITNESS: That seems to be correct, and I would be very surprised if they did because they had only six cases among asthmatics. If you throw any more variable into that model, you will explode it. 02:21
BY MR. LASKER:
Q. Well, the adjustment model is based 02:22

upon all the exposures. It's not specific to glyphosate; correct?
A. No. The one for glyphosate has six exposed cases and 12 exposed controls, and you already have age, vital status, and state in there. So if you do it two by two by two table, then you have no more subjects -- 02:22
Q. I'm sorry --
A. -- in one of these. 02:22
Q. We're not connecting here --
A. Table number 3.
MS. FORGIE: Wait, let her finish.
BY MR. LASKER:
Q. All of the adjustments in this entire study, and there's a whole lot of adjustments they do with stratification on Tables 2 and Table 3, none of the odds ratios anywhere in this study are adjusted for exposures to other pesticides; correct? 02:22 02:22
MS. FORGIE: Objection. Object to form. Asked and answered.
You can answer it again.
THE WITNESS: The glyphosate estimates are estimates among 02:22

non-asthmatics and asthmatics. When you split your data in that way, you limit the way you can adjust. In this case, when you have asthmatics with six glyphosate exposed cases and 12 controls, there's absolutely no way -- I don't even know how they adjust for age vital status and state without exploding their model. 02:23

BY MR. LASKER: 02:23

Q. Okay. Dr. Ritz, that wasn't my question, and that doesn't answer my question in the slightest.

MS. FORGIE: I object to that commentary. She's answered it twice. 02:23

MR. LASKER: We'll mark this answer as well.

BY MR. LASKER:

Q. It's a very simple question. There's two tables here, Table 2 and Table 3 with a whole lot of reported odds ratios, not only for glyphosate, but for other pesticides, for other exposures, for combined herbicides. None of those odds ratios include any adjustment for exposure 02:23

to other pesticide; correct?

MS. FORGIE: Objection. Object to the form. Asked and answered.

You can answer it again.

THE WITNESS: None of the pesticide results are concomitantly adjusted, and it's not a surprise because they are stratifying by asthma status, and in order to compare one model with another, they have to adjust for exactly the same variables or else you can't compare the models. 02:23 02:24

And the intent here is to compare models for asthmatics with models for non-asthmatics. If you put different adjustments variables in there, you don't know whether you see a difference or not. 02:24

MR. LASKER: We're going to have to mark that answer again and ask one more time because I can't get a yes or no answer to a question. I'll ask it one more time. 02:24

BY MR. LASKER:

Q. None of the odds ratios in the Lee 02:24

study were adjusted for exposure to other pesticides; correct?

MS. FORGIE: Objection. Object to the form. Asked and answered. As you just stated, this is like the seventh time. 02:24

You can answer it again.

THE WITNESS: This study intends to look at a stratified analysis of non-asthmatics and asthmatics. If I really want to compare the effects estimates between these two groups of people and I want to assess whether glyphosate has the same effect in one group than in the other, I have to automatically adjust for the same variables. They already are adjusting for age, vital status, and state, therefore, there is no way they could also adjust for everything else. 02:24 02:25 02:25

BY MR. LASKER:

Q. So if the answer is, yes, that's fine, but I need an answer for the record. Am I correct that the Lee study does not adjust for exposure to other pesticides? 02:25

Yes or no?

MS. FORGIE: Objection.

No. She's not required to give a yes or no answer, and you know that.

MR. LASKER: Frankly, she is. 02:25

MS. FORGIE: No, she's not. Don't do this. Objection. Object to the form. Object to asked and answered for the seventh time.

You're not required to give a yes or no answer. You can answer again. 02:25

BY MR. LASKER:

Q. I'm asking for a yes or no answer. If you can't give a yes or no answer, you can just state that and we'll move on and we'll deal with it later for the judge. 02:25

MS. FORGIE: Objection.

THE WITNESS: My answer will not change.

BY MR. LASKER: 02:25

Q. My question to you is am I correct that the Lee study in reporting the odds ratios for all the odds ratios reported does not adjust for the exposure to other pesticides? 02:25

MS. FORGIE: Objection. Object to the form and asked and answered.
You can answer it again.
THE WITNESS: This is such a general question that it's not answerable. But in order to inform you what is done in this study, I state it again. This study intends to compare effect estimates between asthmatics and non-asthmatics. In order to do so, the authors had to adjust for exactly the same variables in the pesticide models. The variables they adjusted for are age, vital status, and state. 02:26
MR. LASKER: Mark that and we'll move on. 02:26
BY MR. LASKER:
Q. The issue with latency that you raised and we've discussed before from the same pool data would also exist to the extent that it concerns you or not with the Lee study; correct? 02:26
MS. FORGIE: Object to the form.
THE WITNESS: I'm not sure what you mean by issue. However, this study 02:26

includes Nebraska and we seem to have agreed that that has a longer latency and gives you more opportunity to investigate this question.
BY MR. LASKER: 02:27
Q. And it also includes the data in Cantor that has the latency period that you believe is too short; correct?
A. I never said that I believed it is too short, but it does include the Iowa and Minnesota data that's in the Cantor study. 02:27
Q. Let's move on to the McDuffie study.
MS. FORGIE: Are we finished with this? 02:27
MR. LASKER: Yeah.
(Exhibit Number 19-15 was marked for identification.)
BY MR. LASKER:
Q. Dr. Ritz, for the record this is the McDuffie study which is the case control study from Canada; correct? 02:27
A. Yes.
Q. And the authors describe McDuffie, et al., describe their analysis in this 02:28

study as exploratory; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Where do they say that?
BY MR. LASKER: 02:28
Q. On page 1161 in the second column about two-thirds of the way down. Do you see the sentence starting "We report results"?
A. Yes. 02:28
Q. "We reported results."
A. Uh-huh.
Q. And McDuffie, et al., describe their analysis in this study as exploratory; correct? 02:28
MS. FORGIE: Objection. Object to the form.
THE WITNESS: What they're stating is that they investigated a number of different chemicals and exposures and, therefore, some of the analyses to unspecified agents should be considered exploratory. I don't know what they mean by unspecified agents. 02:28
///

BY MR. LASKER:
Q. During the point in time, and I think you mentioned this -- well, at -- in the method section -- strike that.
Do you know based upon your review of this study whether glyphosate was specified in the hypothesis when they were conducting this study? 02:29
MS. FORGIE: Object to the form.
THE WITNESS: I wouldn't know that. 02:29
BY MR. LASKER:
Q. Okay. So you cannot state, then, whether or not the glyphosate findings would be considered by the investigators McDuffie, et al., to be exploratory; correct? 02:29
MS. FORGIE: Object to the form.
THE WITNESS: That's not correct because what I -- when I design a study and a study questionnaire, I have to decide which chemical agents to specify, meaning, to name or to want to investigate. So in my preparation for a study, I have to be very clear about what kinds of pesticides I'm asking about, and I would call that 02:29 02:30

specification.
So if they hadn't been interested in glyphosate, they wouldn't have investigated it, and they wouldn't have asked it in a questionnaire. 02:30
BY MR. LASKER:
Q. They state, however, in presenting the data, and they do present data on various different chemical agents, and they have a whole list of them, that they are 02:30 presenting results for chemical agents and exposures that were not specified in the hypothesis; correct?
MS. FORGIE: Object to the form. Asked and answered. You can answer it 02:30 again.
THE WITNESS: They refer to a number of chemical agents and exposures that were not specified. The way that might happen is that when you have a 02:30 questionnaire, you have open questions and you don't specify the name of the chemical, but people decide to write them in. I have no idea what they mean by unspecified, but that's one way of 02:30

reading it.
BY MR. LASKER:
Q. And you don't know sitting here today whether or not based upon this and based upon however they prepared this 02:31 information, whether the findings that they report with respect to glyphosate should be considered exploratory; correct?
MS. FORGIE: Objection. Asked and answered. Object to the form. 02:31
You can answer it again.
THE WITNESS: All I can tell you I don't consider this exploratory.
BY MR. LASKER:
Q. Okay. The McDuffie case control 02:31 study did not adjust for exposure to other pesticides; correct?
A. In what table?
Q. Any of the tables.
A. That's not correct. Table 6 and 7 02:31 seem to be adjusting for chemicals.
Q. 6 and 7 are dealing with various medical variables?
A. And dicamba and Aldrin and Mecoprop. 02:32

Q. With respect to the tables that report any findings with respect to glyphosate, none of those findings are adjusted for exposures to other pesticides; correct? 02:32
MS. FORGIE: Object to the form.
THE WITNESS: Which table are we talking about?
BY MR. LASKER:
Q. Well, for glyphosate, it would be 02:32 Tables 2 and Table 8 as far as I know. But you should make sure that you agree with that. Take your time.
A. 2 and --
MS. FORGIE: 8. 02:33
THE WITNESS: These tables seem to adjust for age and province.
BY MR. LASKER:
Q. Just so the record is clear in the odds ratios that are reported for glyphosate 02:33 in the McDuffie study, the investigators do not adjust for exposure to other pesticides; correct?
A. That seems correct.
Q. The -- as you note in your expert 02:33

report and just so the record is clear, for the two ever/never odds ratios for the glyphosate that McDuffie reports, they find odds ratios of 1.26 in one model and 1.2 in the other model, and neither of those odds 02:34 ratios are statistically significant by the 95 percent confidence interval; correct?
A. Well, if we want to play the P-value game, that's correct, but the values are 1.26 and 1.20. One adjusted; one not. 02:34 But that's an ever/never.
Q. Right. And the -- you mention in your report that there was separate analyses of the McDuffie data that, first of all, separated out association for glyphosate 02:34 with and without malathion; correct? I think that's your expert report at page 18.
A. Where's that?
Q. In your expert report at page 18.
MR. WISNER: Do you want to go off 02:35 the record while we fix this?
MR. LASKER: If we can. I don't know that we can. Let's wait until the end of this question.
MS. FORGIE: What was the question 02:35

again?
MR. LASKER: Now I'm losing track of these things. Oh, okay.
BY MR. LASKER:
Q. So in your expert report you note that there was a separate analysis of the McDuffie data that separated out the association for glyphosate with and without co-exposure to malathion; correct? 02:35
A. Yes, that's the Hohenadel paper. 02:36
Q. The Hohenadel study is a stratified analysis like we were discussing earlier in your testimony here today; correct?
MS. FORGIE: Object to the form.
THE WITNESS: It's not a stratified analysis. It's what we would call an interaction model testing. 02:36
BY MR. LASKER:
Q. In that interaction model testing when, and I think you report this, you note this in your expert report, when Hohenadel looked at the McDuffie data and looked at exposures -- farmers who were exposed to glyphosate alone without co-exposure to malathion, they found or they reported an 02:36

odds ratio of 0.92 with a confidence interval of 0.54 to 1.55; correct?
A. Correct.
Q. And in your report you also point to a separate analysis that you say McDuffie conducted which looked at glyphosate exposure mixed with dicamba exposure; correct, in your expert report? 02:37
A. Where is that?
Q. Right above -- 02:37
A. Above? Yes.
Q. Okay. And I take it that that -- your discussion there is based upon -- and correct me if I'm wrong -- Table 2 in the McDuffie paper? 02:37
A. It's the McDuffie paper.
Q. Look at Table 2.
MS. FORGIE: You can look at whatever you want.
BY MR. LASKER: 02:38
Q. You'll see the numbers that you cite in your expert report on Table 2 for dicamba and dicamba individual. Do you see those?
A. Yes. 02:38

Q. So in your report when you are stating that there was an elevated odds ratio for dicamba exposure mixed with glyphosate exposure, that is relying upon that footnote G in Table 2; correct? 02:38
A. Correct. That's what it was.
Q. And footnote G states that the odds ratio that you cite for mixed exposure for dicamba and glyphosate also involves mixed exposures to dicamba and 2,4-D and Mecoprop; 02:39 correct?
A. That's what it says in the footnote.
Q. And unlike for glyphosate, McDuffie reported statistically significant increased 02:39 risks of non-Hodgkin's lymphoma separately associated with exposures to each of the three pesticides 2,4-D, dicamba, and Mecoprop; correct?
A. That's in table -- 02:39
Q. It's actually in Table 2. They have separate odds ratios reported for 2,4-D that is statistically significant in their --
A. Yes. 02:39

Q. -- model, second model for Mecoprop?
A. Yeah, but it's an effect estimate of 1.26 and 1.32, and it's only statistically significant after the 02:39 adjustment.
Q. Okay. And then for Mecoprop there is a 2.23 or 2.33 odds ratio --
A. Correct.
Q. -- statistically significant to 02:39 both measure and for dicamba even in the dicamba alone for their more highly adjusted odds ratio it's 1.68 marginally statistically significant; correct?
A. Yes. 02:40
Q. And you cannot tell from this data when you're looking at the mixed exposures for dicamba when they're mixed for 2,4-D Mecoprop and glyphosate, you cannot attribute the difference between dicamba 02:40 alone and this dicamba mixture to glyphosate, can you?
MS. FORGIE: Object to the form.
THE WITNESS: You can never do that in an individual anyhow. When you're 02:40

doing these kind of analyses, you have mixed exposures. If a person is exposed to two compounds, then it can be either one compound or the other or both together that are responsible for the event. 02:40

BY MR. LASKER:

Q. But in this case, it's not one or the other or two. There's actually four different chemicals when you're stating that there was in your expert report -- and let's go back to your expert report. You state that McDuffie reported that when glyphosate exposure was mixed with dicamba, the risk was increased. 02:41

Do you see that?

A. Yes.

Q. And, in fact, what McDuffie was reporting is that when dicamba exposure also included mixed exposures to glyphosate, 2,4-D and Mecoprop, there was an increase as compared to the dicamba alone; correct? 02:41

MS. FORGIE: Object to the form. Mischaracterizes.

THE WITNESS: That's not what I'm 02:41

saying. I'm saying there is dicamba that is of the kind Banvel and Target which includes glyphosate and then there's dicamba overall. So one is a subgroup of the other. And you can actually see that when you're looking at the number of exposed cases and exposed controls. Dicamba is the all-encompassing over label and then they're breaking it down with and without glyphosate, et cetera, mixtures. 02:41 02:42

BY MR. LASKER:

Q. The et cetera is the important point, but let me make sure I understand. Is it your testimony that or Banvel or Target is a mixed exposure with glyphosate? 02:42

MS. FORGIE: Objection. Object to the form and mischaracterizes her testimony.

THE WITNESS: So it says in the footnote, "dicamba is a major chemical class, includes Banvel and Target and a mixture of dicamba glyphosate, Rustler, or a mixture of dicamba 2,4-D and Mecoprop. 02:42 02:42

BY MR. LASKER:

Q. And then Dynel, Killex; correct?

MS. FORGIE: Object to the form.

THE WITNESS: Dynel DS, and Killex.

BY MR. LASKER: 02:42

Q. So the mixed exposure would be in Rustler for dicamba and glyphosate; correct?

A. There are several mixtures. There's the mixture of dicamba and glyphosate in Rustler and then there's the mixture of dicamba with 2,4-D and Mecoprop. 02:42

Q. So for the 1.68 odds ratio, that's dicamba alone; correct?

A. That's the overall dicamba. That's not dicamba alone. That's not -- that's dicamba with everything. 02:43

Q. And your understanding is dicamba with everything is 1.68 and dicamba alone is the 1.88?

A. No. 02:43

MS. FORGIE: Object to the form.

THE WITNESS: It's the opposite.

MS. FORGIE: Okay. That's what I thought.

THE WITNESS: Dicamba overall is 02:43

1.88, and the dicamba, Banvel and Target is 1.68.

BY MR. LASKER:

Q. And the difference -- in your expert report you state that the difference going up to that higher number is because there was including mixtures with glyphosate, but that higher number actually also reflects exposures to 2,4-D and Mecoprop; correct? 02:43 02:43

MS. FORGIE: Objection. Object to the form and asked and answered.

You can answer it again.

THE WITNESS: I'm not sure that I understand what you're trying to get at. In this table, dicamba exposure was the footnote G is the overall encompassing -- all-encompassing exposure. The individual dicamba herbicide Banvel or Target is the one that's reported below. The number of cases is lower, and the number of controls is lower, but, in essence, the number of 26 and 50s included in the larger category above which is 73 and 02:43 02:44

131.
BY MR. LASKER:
Q. My question is very simple. In your expert report, you state that the odds ratio of 1.92 was an odds ratio of glyphosate exposure mixed with dicamba. And am I correct in my reading of this table that that 1.92 odd ratio is, in fact, dicamba with mixtures that include glyphosate but also Mecoprop and 2,4-D? 02:44
MS. FORGIE: Objection. Object to the form and also asked and answered.
You can answer it again.
THE WITNESS: The larger group encompasses everything including glyphosate. 02:44
BY MR. LASKER:
Q. And Mecoprop and 2,4-D; correct?
A. It's the largest group.
Q. Yes. And you have to answer the question or there's no answer on the record. 02:45
A. Yes. It's the larger group.
MS. FORGIE: Wait, wait. So get a format back that's question and answer so I can get my objections in. 02:45

BY MR. LASKER:
Q. So the odds ratio of 1.92 that you cite in your expert report as glyphosate exposure mixed with dicamba is the odds ratio that McDuffie reports for dicamba and dicamba mixtures including glyphosate 2,4-D and Mecoprop; correct? 02:45
MS. FERGIE: Objection. Object to the form. Also asked and answered.
You can answer it again. 02:45
THE WITNESS: Dicamba here is a super category for several mixtures, and it's stated under footnote G. And we can see that that's the case because there are more NHL cases and more controls in that category than in the category below. 02:45
MR. LASKER: I'm going to mark this answer as well.
BY MR. LASKER: 02:45
Q. I'm going to ask the question again because I think it's a simple question, but I'm not getting an answer?
MS. FORGIE: I'm objecting to that commentary. You're badgering the 02:46

witness when you do that.
MR. LASKER: You can object as much as you want.
MS. FORGIE: I will.
BY MR. LASKER: 02:46
Q. The odds ratio of 1.92 that you report in your expert report as the odds ratio for glyphosate mixed with dicamba is as reported, in fact, in the study McDuffie an odds ratio for dicamba and dicamba mixtures with glyphosate but also with 2,4-D and Mecoprop; correct? 02:46
MS. FORGIE: Objection. And I object to the form. And I object to the fact this is the eighth time you've asked her. You're badgering this witness. It's not fair. 02:46
You can answer again.
THE WITNESS: The reason why I'm referring to this is because this is a mixture exposure, and that's very clearly stated in my report. 02:46
BY MR. LASKER:
Q. Your report --
A. The mixture includes dicamba and 02:46

glyphosate under heading G in this footnote.
Q. The mixture also includes which you don't mention in your report 2,4-D and Mecoprop; correct? 02:46
MS. FORGIE: Objection. Asked and answered. Object to the form.
You can answer again.
THE WITNESS: It is a mixture exposure. Some people were exposed to a mixture of dicamba and glyphosate. 02:47
Others might have been exposed to a mixture of dicamba with something else, but it says the major chemical classes included Banvel and Target, and it refers to these two as major and being a mixture of dicamba and glyphosate. 02:47
BY MR. LASKER:
Q. Banvel and Target do not have glyphosate in them, do they?
MS. FORGIE: Objection. Asked and answered. 02:47
You can answer it again.
THE WITNESS: The way it states it dicamba is a major chemical class, includes Banvel and Target and a mixture 02:47

of dicamba and glyphosate. That's what I said.
BY MR. LASKER:
Q. So is it your understanding and the basis of your expert report that Banvel and Target include glyphosate? 02:47
MS. FORGIE: Objection. Object to the form. Asked and answered. You're badgering the witness. This is completely unfair. 02:47
I'll let you answer it again.
THE WITNESS: What I said is that dicamba is a major chemical class and what they refer to here is that dicamba wasn't dicamba alone, but it was under 02:47 this rubric of dicamba G exposed. They subsumed multiple agents that were mixed with dicamba.
BY MR. LASKER:
Q. McDuffie provides an analysis in 02:48 her expert report. I'm not sure that fully answered on the last question but I'm going to move on so I can get through this deposition for now at least.
McDuffie provides an analysis on 02:48

Table 8, I believe, of exposures based upon days, less than two days or more than two days for purposes for glyphosate; correct?
A. Yes.
Q. You do not cite to this analysis, 02:48 unless I missed it, anywhere in your expert report; correct?
A. I think I'm referring to it in my Bradford Hill analyses. Yes. However, the effect as to -- 02:49
Q. Can you show me where you are?
A. Yes. Page 23. Bradford Hill evaluations.
However, the effect estimates for longer or more extensive use in several 02:49 studies were larger between two and three, and that includes this estimate.
Q. So if you were referring to this at page 23, you would need to refer to the McDuffie paper? 02:49
A. Yes.
Q. You do not in your discussion of the McDuffie paper --
A. Point that out.
Q. Point that out; correct? 02:49

A. I guess I didn't.
MS. FORGIE: When you get to a good breaking point, let's take a short break, please.
MR. LASKER: Okay. Let's just get 02:50 through this.
MS. FORGIE: That's fine.
BY MR. LASKER:
Q. In your opinion, does this analysis on Table 8 of less than or equal to two days 02:50 versus greater than two days provide evidence of a dose response for glyphosate?
A. This is not supposed to give a dose response. This is an analysis where you're trying to separate out people who are 02:50 completely unexposed to this agent and people who had minimal exposure versus reasonable exposure two days per year. And in doing so, you can actually see that there's very little confounding due to any 02:50 other variable because for minimal exposure the effect estimate is 1. So even if I would compare as done in De Roos, the people with more than two days of exposure to the people of less than two days, I would still 02:51

get the same kind of effect estimate.
Q. I'm not sure I got the answer to my question, though.
In your opinion, does the analysis that McDuffie provides in Table 8 of less 02:51 than or equal to two days' exposure versus greater than two days, in your opinion, does that provide evidence of a dose response for glyphosate?
MS. FORGIE: Objection. Object to 02:51 the form. Also asked and answered. She just answered that.
You can answer it again.
THE WITNESS: The intent of this analysis is not dose response. The 02:51 intent of this analysis is to distinguish between types of people who use and did not use glyphosate.
BY MR. LASKER:
Q. And do I understand correctly then 02:51 that you do not interpret the data reported in this table as providing evidence of a dose response?
MS. FORGIE: Objection. Asked and answered. 02:51

You can answer it again.
THE WITNESS: I see this as an indicator that a better exposure assessment that defines glyphosate use not as ever/never which is the worst or the most simple category you can get but as a reasonable amount, more than two days per year, we don't know how many days those are, but that that category provides you with some indication that there is an effect. 02:51 02:52
BY MR. LASKER:
Q. So I think I understand you, but I just want to make sure that I'm clear. Am I correct then in my understanding that you do not interpret the data on Table 8 in McDuffie as presenting evidence of a dose response glyphosate and non-Hodgkin's lymphoma? 02:52
MS. FERGIE: Objection. Object to the form. Also asked and answered. 02:52
You can answer it again.
A. There's no formal analysis of a dose response. However, the more than two days per year category suggests that there 02:52

is a dose effect.
BY MR. LASKER:
Q. And so I get your opinions because that what we're here for. In your opinion, does the data presented on Table 8 for glyphosate provide evidence of a dose response for glyphosate and non-Hodgkin's lymphoma? 02:52
MS. FERGIE: Objection. Asked and answered. This is the fifth time. 02:53
You can answer it again.
A. So, again, this is not a formal dose response analysis, but it is a very clever analysis and one that I really enjoy looking at because, first of all, they are splitting up people who don't use glyphosate and then the group of people who do use it and the casual users, whether -- versus the more frequent or more intense users, and in that sense, you can say that at the higher doses there is actually an effect. 02:53 02:53
BY MR. LASKER:
Q. Okay. I'm still trying to get an answer to this question because I don't think I have it. 02:53

In your opinion, does the data presented in Table 8 in the McDuffie paper provide evidence of a dose response for glyphosate and non-Hodgkin's lymphoma?
MS. FORGIE: Objection. I object to the form, and especially I object to the fact that she's answered this five or six times now. Again, you're badgering the witness just because you don't like the answer. 02:53 02:54
You can answer it again.
THE WITNESS: Okay. So clever analysis, splitting up unexposed and exposed, selecting out people who are maybe occasional users, looking at those who have probably regular intense use. Among those with regular and intense use, we see an effect for glyphosate. 02:54
BY MR. LASKER:
Q. That wasn't my question. My question is: Does this data in Table 8 from McDuffie, in your opinion, present evidence of a dose response for glyphosate? 02:54
MS. FORGIE: Objection. Asked and answered. 02:54

THE WITNESS: I have criteria for those response. You may have your own. In this case, there is a high use of glyphosate associated clearly with an odds ratio of 2.12 with NHL. 02:54
BY MR. LASKER:
Q. Does this Table 8 in the McDuffie meet your criteria to be interpreted as providing evidence of a dose response for the glyphosate in non-Hodgkin's lymphoma? 02:55
MS. FORGIE: Objection. Asked and answered.
THE WITNESS: This results provides evidence that with intensity and frequency, whatever this means, two days per year, there is indeed an effect for glyphosate compared to people who are using either none or using occasionally less than two times a year. 02:55
MR. LASKER: I'm going to mark this answer, and again, I'm going to ask the question again because I still don't get answers to my questions. 02:55
BY MR. LASKER:
Q. Based upon your criteria, whatever 02:55

criteria you use in your professional work, does the data presented in Table 8 in the McDuffie paper provide evidence of a dose response effect for glyphosate in non-Hodgkin's lymphoma? 02:55

MS. FORGIE: Objection. This is, like, the eighth time you've asked the same exact question, and she's answered it seven or eight times. This is really badgering the witness. I'm going to let 02:55 her answer it one more time.

THE WITNESS: I just repeat myself. We are distinguishing unexposed people from irregular users, minimal users, and regular users. In the regular use 02:56 group, we see an effect.

MR. LASKER: Okay. Mark that answer.

Let's take a break.

THE VIDEOGRAPHER: We are off the 02:56 record at 2:56 p.m.

(Recess taken from 2:56 p.m. to 3:13 p.m.)

THE VIDEOGRAPHER: We are back on the record at 3:13 p.m. 03:13

BY MR. LASKER:

Q. Dr. Ritz, we were talking about Table 8 in the McDuffie paper, and I'm correct, am I not, that the McDuffie paper does not provide any analysis of the 03:13 intensity of the exposures to glyphosate in this population; correct?

MS. FORGIE: Object to form.

THE WITNESS: That is incorrect. They are actually distinguishing between 03:14 irregular and regular users, and in the category of regular users, they see an increased risk.

BY MR. LASKER:

Q. So regular users is greater than 03:14 two days per year; correct?

A. Yes.

Q. So if somebody were to use glyphosate for a half-hour in the spring in the driveway and then a half-hour in the 03:14 fall and another half-hour in the summer, that would be three times a year, and they would be greater than two days a year; correct?

A. I don't venture to say that because 03:14

they're measuring here in days, and when I do my pesticide studies, we actually ask how many hours per day, and then we average across to come to eight-hour workday and add all of that up. How they exactly did that 03:14 is not described here, but that's how we would do it.

Q. Okay. But you don't know how the investigators in this study calculated day of exposure; correct? 03:15

MS. FORGIE: Objection. Asked and answered.

THE WITNESS: These investigators give you a more than two day per year category, and I imagine they did this in 03:15 order to distinguish between irregular users who they classify as more than zero and less than two days.

BY MR. LASKER:

Q. My question, though, is these 03:15 investigators do not indicate and you don't have any information as to how they determine a day of exposure; correct?

MS. FORGIE: Objection. Asked and answered. 03:15

You can answer it again.

THE WITNESS: These investigators asked people to report occupational exposures, and when you ask about occupational exposures, you usually 03:15 refer to a workday. So I would interpret this as two workdays per year.

BY MR. LASKER:

Q. Okay. So your interpretation -- it's not set forth in the study, but your 03:15 interpretation of this table is that greater than two days means a full two-day -- each day would be a full workday of exposure?

MS. FORGIE: Objection. Asked and answered. Also mischaracterizes her 03:16 testimony.

THE WITNESS: I, as a pesticide exposure assessment epidemiologist, would specifically ask people to report how many hours, how many days, how many 03:16 weeks, how many years they would be having used these specific agents and then categorize it according to the days or hours or years.

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BY MR. LASKER:
Q. I understand what you would do. That's not my question. I'm trying to find out what McDuffie and her group did.
They do not state in their paper -- they do not define a day as being an eight-hour exposure day, do they? 03:16
MR. FORGIE: Objection. Asked and answered.
THE WITNESS: I have to check. They actually asked extensive questions including histories, pesticide spill, protective equipment, et cetera. So given that they asked all this, and they were after workplace exposures, I would interpret this as two workdays. 03:16 03:17
BY MR. LASKER:
Q. McDuffie does not, anywhere in this paper, state that they define a day as a workday of exposure, do they? 03:17
MS. FORGIE: Objection. Asked and answered. She just testified as to exactly how she interprets that meaning.
MR. LASKER: Okay. That's not the question I asked. I'll ask the question 03:17

again.
MS. FORGIE: Yes, it is.
BY MR. LASKER:
Q. McDuffie and her investigators in this published paper never state that they defined a day of exposure as a full workday of exposure; correct? 03:17
MS. FORGIE: Objection. Asked and answered. You're badgering the witness. She's already told you how she interprets it. 03:17
You can answer it again.
THE WITNESS: Yes, actually they're saying on page 1157, "We created dose response levels based on days per year of personally mixing or applying selected herbicides, insecticide, fungicides, and fumigants." 03:17
So days per year of personally mixing or applying, that's workplace types of exposures. 03:18
BY MR. LASKER:
Q. I understand, but they don't state a minimum time period in a day for it to be quantified as a day of exposure; correct? 03:18

MS. FORGIE: Objection. Asked and answered. She's told you exactly two or three times how she interprets that.
You can answer it again.
THE WITNESS: I think I answered it. 03:18
MS. FORGIE: You can answer it again.
THE WITNESS: So they are trying to distinguish between regular users and occupational regular users who are mixing and applying pesticides and people who might be for one day in their life applying glyphosate. 03:18
BY MR. LASKER: 03:18
Q. Dr. Ritz, let's talk about the North American pooled project analysis by Pahwa in 2015.
MS. FORGIE: Are we putting this away? 03:18
MR. LASKER: For now, yeah.
BY MR. LASKER:
Q. And this is analysis which was a pooled analysis of the case control studies that were pooled in De Roos 2003 and also 03:19

the Canadian case control study that was analyzed by McDuffie; correct?
A. Can I have the exhibit?
Q. Sure.
A. Thank you. 03:19
Q. This is 19-16.
(Exhibit Number 19-16 was marked for identification.)
THE WITNESS: It's called the North American Pooled Project. On page 5, we see that it is encompassing those states, yes. 03:19
BY MR. LASKER:
Q. And this is the analysis at that 2015 ISEE conference that you cite to in your expert report; correct? 03:19
A. Yes.
Q. When did you -- you provided this slide deck or at least it was provided to us as an additional material considered after your rebuttal expert report. 03:20
When did you first see this slide deck?
A. I saw it after the deposition of Dr. Blair, and there was reference to this. 03:20

Q. Did you -- had you seen this slide deck prior to the time you prepared your initial expert report in this case?
A. No.
Q. Okay. And I take it you saw it then sometime before you reviewed the rebuttal -- I'm sorry, before you prepared your rebuttal exert report, your second expert report? 03:20
A. Yes. 03:20
Q. Have you read Dr. Neugut's deposition?
A. Yes.
Q. Did you see this slide deck before you read Dr. Neugut's deposition or after? 03:20
A. I wouldn't be able to tell.
Q. So may have been before or may have been after, you're not sure?
A. I don't know.
Q. If I can refer you to page 10 of this presentation, the NAPP presentation, they provide data or odd ratios for their ever/never analysis both overall for the glyphosate and non-Hodgkin's lymphoma and also for various subtypes of NHL; correct? 03:21 03:21

A. Correct.
Q. And they have two analyses that they present in this table. Their odds ratio A which is adjusted for age, sex, state, province, emphatic or hematopoietic cancer in a first-degree relative, use of a proxy respondent, and use of personal protective equipment; correct? 03:21
A. Yes.
Q. And then odds ratio B would adjust for those factors just listed and also adjusts for 2,4-D, dicamba and malathion; correct? 03:22
A. Correct.
Q. For the ever/never analysis of the pooled data from the U.S.-based and Canadian-based case control studies, when adjusted for the use of 2,4-D, dicamba and malathion, they report an odds ratio of 1.13 with a confidence interval of 0.84 to 1.51; correct? 03:22 03:22
A. Yes.
Q. And for their various subtypes of non-Hodgkin's lymphoma, in their adjusted model adjusting for the use of 2,4-D, 03:22

dicamba, and malathion, they report varying odds ratios, one of which is below 1, three of which are above 1, but all of which are not statistically significant; correct?
A. Well, I wouldn't evaluate this according to statistical significance especially in a subgroup analysis where I'm splitting the data in this way. The way I would evaluate it is whether there's considerable change in effect estimates and width of the confidence interval. 03:23 03:23
Q. Okay. So follicular lymphoma for their odds ratio that's adjusted for the use of 2,4-D, dicamba, and malathion, they find an odds ratio of 0.69; correct? 03:23
A. That's what they state, yes.
Q. And that was a reduction in the odds ratio when they adjusted for these exposures to other pesticides; correct?
A. Correct. 03:23
Q. For diffuse large B cell lymphoma when they adjusted for 2,4-D, dicamba, and malathion, they report an odds ratio of 1.23. That's not statistically significant; correct? 03:24

MS. FORGIE: Objection. I object to the form.
MR. LASKER: That's fine.
THE WITNESS: That's an odds ratio that's lower than 1.6 and the confidence interval includes the 1. 03:24
BY MR. LASKER:
Q. Okay. So when they adjusted for the use of 2,4-D, dicamba, and Malathion, their odds ratio for diffuse large B cell lymphoma went down and was no longer statistically significant; correct? 03:24
MS. FORGIE: Objection. Object to the form.
THE WITNESS: It fluctuated. It went from 1.6 to 1.23, but the confidence interval basically overlapping. 03:24
BY MR. LASKER:
Q. And for the odds ratio with adjustment for 2,4-D, dicamba, and Malathion, the confidence interval went from .81 to 1.88 including a null hypothesis of 1.0; correct? 03:24
A. Including the null value in a 03:24

formal statistical test.
Q. And SLL, I knew I was going to get to this one. What does SLL stand for?
A. Small lymphocytic lymphoma.
Q. For that odds ratio there is not a meaningful change when they adjusted for exposures to other pesticides; correct? 03:25
MS. FORGIE: Objection. Object to the form.
THE WITNESS: It almost -- it basically stays the same. The confidence interval widens as one would expect when you put additional variables in a model. 03:25
BY MR. LASKER: 03:25
Q. And then for the other category you have an odds ratio that drops from 1.66 to 1.51 with adjustments for 2,4-D, dicamba, and Malathion, and that adjusted odds ratio is 0.87 to 2.6 which includes the null value of 1.0; correct? 03:25
MS. FORGIE: Object to the form.
THE WITNESS: Well, the odds ratio changes from 1.66 to 1.51 which is almost the same. And as I stated 03:26

before, the confidence intervals widen when you add other variables into the model, and it does include null to null value.
BY MR. LASKER: 03:26
Q. And in your original expert report before you had seen this data, you had discussed the fact that the Pahwa NAPP data should be considered in conducting any meta-analysis of the website data; correct? 03:26
MS. FORGIE: Object to the form.
THE WITNESS: Where is that stated?
BY MR. LASKER:
Q. That is on page 16, 15 and 16, where you're talking about the NAPP data. 03:26
And, first of all, just to be clear, in your expert report for the NAPP data you are reporting data that is not adjusted for exposures to 2,4-D, dicamba, and Malathion; correct? 03:27
A. I have to go to the abstract to confirm that.
So what's the question?
Q. In your expert report before you had seen the data adjusted for exposures to 03:27

2,4-D, dicamba, and Malathion, you had suggested that the NAPP data had not been included in the meta-analysis that had been performed for glyphosate and non-Hodgkin's lymphoma; correct? 03:27
A. That is correct. They have not been included anywhere, and that's what this sentence says.
Q. And under the methodology that both Chang and Delzell used and that the IARC scientists used in conducting their meta-analyses, when there was a subsequent pooled analysis of case control data, they included that subsequent study, and they removed the earlier studies from their meta-analysis; correct? 03:28
MS. FORGIE: Object to the form.
THE WITNESS: That would usually be how you do it.
BY MR. LASKER: 03:28
Q. And in both the Chang and Delzell meta-analysis and the analysis that IARC did with its working group for their meta-analysis, they used the odds ratios that were -- where they had them that were 03:28

adjusted for exposures to other pesticides; correct?
A. I think they did, but can you show me where that's stated.
Q. In your expert report actually at page 16. We went through that earlier. 03:29
A. Okay.
Q. Correct?
A. Yes.
Q. If we were to conclude the NAPP data into the meta-analysis using the methodology that was used by Chang and Delzell and using the methodology that was used by IARC, we would use the odds ratio for the NAPP of 1.13; correct? 03:29
MS. FORGIE: Object to the form.
THE WITNESS: No. This is not a valid model in my mind because you have to show me that 2,4-D, dicamba, and Malathion are actually related to glyphosate use and also are independent risk factor for NHL. So if you're telling me dicamba is an independent risk factor for NHL, then yes. Also it should be removed. 03:29 03:30

Also I would not accept this model because we would not want to adjust for the use of proxy respondents or personal protective equipment because those two variables are indicators for exposure mismeasurement. You cannot adjust a model for exposure mismeasurement. These are confounded and shouldn't be in the models. 03:30

BY MR. LASKER: 03:30

Q. I understand, and I'm going to get to your opinions about the NAPP and how they did their analysis. The IARC in conducting its meta-analysis did not reach any conclusions with respect to the individual studies as to whether or not they found those studies to be internally valid; correct? They just used the data that was presented? 03:30

A. I don't -- 03:30

MS. FORGIE: Object to the form.

THE WITNESS: I don't believe that IARC would use estimates that they don't believe are valid. I wouldn't.

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BY MR. LASKER:

Q. In their methodology the both for the IARC meta-analysis and for the NAPP, they used the data point presented in each of the studies that were available for glyphosate and non-Hodgkin's lymphoma; correct? 03:31

A. That's how you conduct meta-analysis.

Q. They did not exclude any of the analyses; correct? 03:31

MS. FORGIE: Object to the form.

THE WITNESS: They did not exclude one of the studies.

BY MR. LASKER: 03:31

Q. And they did not -- so for their purposes -- and I understand you will have your own interpretation how you do a meta-analysis when we talk about that in a moment, but following their methodology, if this study was available to them, they would use as they did with every other study what was reported as the most adjusted odds ratio which in this case was reported as 1.13; correct? 03:31 03:31

MS. FORGIE: Object to the form.

THE WITNESS: I don't want to venture into what people would be doing if. I would not recommend to use this preliminary data that has obvious problems to replace studies that have been published and peer-reviewed. 03:32

BY MR. LASKER:

Q. I'm sorry. This is the data except for the fact that we now have adjusted odds ratios which you had not seen when you prepared your expert report. This is the same NAPP analysis that you had put forth as a basis for your expert opinion; correct? 03:32

MS. FORGIE: Objection. Mischaracterizes her report. 03:32

THE WITNESS: I have not used these slides. I have used an abstract.

BY MR. LASKER:

Q. But it was an abstract that resulted in the presentation at the exact same conference where the abstract was presented, and this is -- the exhibit we have, 19-16, is a presentation that went along with that abstract at that conference; 03:32 03:32

correct?

MS. FORGIE: Object to the form. Mischaracterizes.

THE WITNESS: When we are scientists to present results, we sometime like to present results that are provocative and also have discussions. So I would consider this one of those slides where we can then discuss how to run the analysis one way or another. 03:32 03:33

These kind of discussions often feed into final analyses that are published in the literature because the authors then are aware of criticism from the scientific community. That's the whole reason to present these. 03:33

BY MR. LASKER:

Q. I'm just a little confused now because prior to seeing this data adjusted for the pesticides, you were opining, and you had earlier in this deposition I thought, that the NAPP data presented at Brazil at that ISEE conference should be considered as part of the analysis of the 03:33 03:33

epidemiologic literature, didn't you?
A. The abstract I saw, yes. But I'm not referring to this table.
Q. Okay. So while you believe that the NAPP data that was prepared and 03:33
presented in a one-paragraph abstract for this presentation should be considered, you do not believe that it would be appropriate to consider the full data that was actually presented at that conference because it is 03:34
preliminary; is that correct?
MS. FORGIE: Object to the form.
THE WITNESS: So any data that we are presenting and not putting into a paper version is preliminary including 03:34
the abstract that went to this conference. The only reason why I like the abstract is because it referred to existing data, existing studies that I had read that I understood. The 03:34
methodology and the way they were performed. However, when we are presenting tables at conferences, what we are doing is allowing input into analyses from a scientific audience that 03:34

is informed. That's what this table is all about, and had I been there, I would have made comments about this kind of table.
BY MR. LASKER: 03:35
Q. I just want to be clear now if I understand your position. Is it your position, then, that the NAPP data is too preliminary to be considered as part of an expert analysis, or is it your opinion that 03:35
the NAPP data in the abstract that came out before this conference should be considered but that the data presented at the conference should not?
MS. FORGIE: Objection. 03:35
Mischaracterizes her testimony.
THE WITNESS: It's all the same data. It's just a question of which analyses you believe more or not.
BY MR. LASKER: 03:35
Q. And is it my -- is it your testimony then that while you believe in the data that was presented in the abstract and you think that should be considered as reliable evidence, epidemiological evidence 03:35

for glyphosate and non-Hodgkin's lymphoma, you do not believe that the data that was actually presented at that conference should be considered as reliable evidence, separate epidemiological evidence regarding 03:35
glyphosate and NHL?
MS. FORGIE: Object to the form.
THE WITNESS: Again, I want to say the same data.
BY MR. LASKER: 03:36
Q. So in your expert report, you stated that we should consider the NAPP data in our analysis; correct?
A. Yes.
Q. Okay. And so it's fair to say that 03:36
you also agree that we should consider the data that was actually presented from the NAPP in its conference in our analysis; correct?
MS. FORGIE: Object to the form. 03:36
THE WITNESS: That's different. The data, the way it's presented, contains a lot of what we would call sensitivity analyses and ways of presenting the data that I would as a 03:36

reviewer agree or not agree with.
BY MR. LASKER:
Q. And am I correct in my understanding that your concern with respect to presenting the data from the NAPP for -- 03:36
as compared to data that controls for 2,4-D, dicamba, and Malathion versus data that does not control for 2,4-D, dicamba, and Malathion, that you believe it is more reliable to look to the data that does not 03:37
control for 2,4-D, dicamba, and Malathion?
MS. FORGIE: Object to the form and object to mischaracterizing her testimony.
THE WITNESS: I never talked about 03:37
reliability. That's not at issue here. What is at issue is validity of the model, and I disagree with the validity of this model, and I would suggest additional sensitivity analyses 03:37
concerning this.
BY MR. LASKER:
Q. And am I correct in my understanding that -- well, let me ask this: Do you have concerns of the validity of the 03:37

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NAPP model for all of the data presented or only for the data presented that adjusts for exposures to 2,4-D, dicamba, and Malathion?
MS. FORGIE: Object to the form.
THE WITNESS: I have validity concerns about this one table, and I would like to see additional analyses before I would make up my mind. 03:37
BY MR. LASKER:
Q. Do you have validity concerns for the data presented in the abstract that you relied upon in your expert report before you saw this data? 03:38
A. The validity concerns are not considering the data. The validity concerns are with respect to this one subanalyses that I consider a sensitivity analysis. 03:38
Q. Which subanalyses are you talking about?
A. The one adjusting for three additional pesticides. 03:38
Q. So that's -- so I understand. So you do not have -- I'm just making sure I understand this. You do not have validity concerns with respect to the NAPP data that 03:38

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does not adjust for dicamba, 2,4-D, and Malathion; is that correct?
MS. FORGIE: Object to the form.
THE WITNESS: I have validity concerns about this whole table as I just told you because I would suggest that, first of all, proxy respondents and personal protective equipment should not be entered in the model to begin with. 03:38
BY MR. LASKER:
Q. That information, and I'll just -- I don't have time to go through this, but if that information was in the abstract that they controlled for that, would you have concerns with the data and the information presented in the abstract that you relied upon in your original expert report? 03:39
MS. FORGIE: Object to the form and also asked and answered. 03:39
You can answer it again.
THE WITNESS: I can only refer to this table in front of me that states very clearly what they adjusted for, and I would have asked as a conscientious 03:39

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reviewer to remove these two variables and tell me whether it makes a difference.
BY MR. LASKER:
Q. And do you have greater concern for the validity of the odds ratios that adjusts for 2,4-D, dicamba, and Malathion than for the odds ratios that do not? 03:39
MS. FORGIE: Objection. Object to the form. Asked and answered. 03:39
You can answer it again.
THE WITNESS: That's a question I cannot answer because I don't know what the results would be if we did this differently. 03:39
BY MR. LASKER:
Q. Okay.
A. And that's what we do in epidemiology. We try all sorts of things and see how the data behaves. 03:39
Q. Okay. For the analysis for duration of exposure and days of exposure, the NAPP basically had data on duration -- if you look at page 7.
A. Page 7? 03:40

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Q. Yeah.
A. Oh, yeah.
Q. So the duration and frequency and lifetime days analysis for the NAPP is drawn from the Nebraska and the Canadian case control data because we don't have all -- we don't have the full data for Iowa, Minnesota. We don't have any data for Kansas to conduct those analyses; correct? 03:40
MS. FORGIE: Object to the form. 03:40
THE WITNESS: If those Xs mean there's no data, then that seems to be the case.
BY MR. LASKER:
Q. Okay. If we can go then to page 26, and I want to start just with the first column which is proxy and self-respondents, and we'll talk about the self-respondents only in a second. But for the -- they provide information in this table for frequency with respect to days per year, duration, and also lifetime days; correct? 03:41
A. Yes.
Q. And when we do the frequency 03:41

analysis -- and this is not particularly surprising since the Canadian case control study was a large driver of this -- we have a somewhat similar finding to what is reported in the McDuffie paper; correct? 03:42

MS. FORGIE: Object to the form.

THE WITNESS: Frequency more than two days per year and odds ratio of 1.73 or 1.77 counts as similar to 2, yes.

BY MR. LASKER: 03:42

Q. For duration -- so it's a different measure -- correct? -- of how many years they actually used glyphosate; correct?

A. Yes.

Q. McDuffie does not provide any 03:42 indication of the duration of use in her analysis in her study; correct?

MS. FORGIE: Object to the form.

THE WITNESS: She doesn't provide tables. That doesn't mean that they 03:42 didn't have it. Did they have it?

BY MR. LASKER:

Q. In the McDuffie paper?

A. No. In the data.

Q. They did have it in the data, yes. 03:42

A. Yes.

Q. But in the McDuffie paper they don't report duration; correct?

A. No.

Q. When they look at that data for 03:42 duration, we find that there is a lower incidence of NHL with a, at least numerically, with greater duration of use of glyphosate; correct? Goes from either 1.28 to 0.94 or 1.17 to 0.78; correct? 03:43

A. There's basically no difference.

Q. When we look at lifetime days, so this is actually figuring out the total amount of exposure that an individual in the study would have -- correct? -- that last 03:43 category?

A. It's not the total amount. It's duration times intensity, and that could be seven years used minimally or -- and that would give you a seven or seven days used at 03:43 the two workdays per year as we discussed. We don't know.

Q. Just to be clear because your answer had the word "seven years," and I want to make sure we understand this. The 03:44

lifetime days analysis is less than seven days in a lifetime of exposure to glyphosate or greater than seven days of exposure to glyphosate in the lifetime; correct?

MS. FORGIE: Object to the form. 03:44

THE WITNESS: What they call lifetime days is similar to pack years. So it's a product of the number of years times the days per year.

BY MR. LASKER: 03:44

Q. And when they did this analysis using that same McDuffie data and also the Nebraska data was added to it, and they looked at total lifetime days of exposure to glyphosate and they looked at that higher 03:44 category, the highest category they reported of greater than seven lifetime days of exposure to glyphosate, they had an odds ratio of either 1.08 or 1.06 for glyphosate and non-Hodgkin's lymphoma; correct? 03:44

A. They call it lifetime days, but it's not days in a lifetime. It's this product of years times number of days per year; so it's more like a pack year, and I'm not surprised because duration, number of 03:45

years, had no effect. So if you're using duration as number of years, you are very likely to wipe out any intensity effect.

Q. Well, the intensity just to be fair, the duration would include all the 03:45 days within each year -- the lifetime days has both factored into it. It has the days per year, and it has the duration of time; correct?

MS. FORGIE: Objection. Object to 03:45 the form.

THE WITNESS: It's not correct because number of days per year has two categories. It has the greater than zero and less than two which we agreed 03:45 on were the occasional users and then the two or more or better two -- more than two. So when you're calculating number of years times number of day per year, you're actually mixing a lot of 03:45 different things together. It's a really bad measure. So if you don't believe it is duration low level chronic exposure, if you think it's intensity, you have to have a high level of 03:46

exposure, then lifetime days is really not a good measure.
BY MR. LASKER:
Q. Is it your opinion that there could be intense exposure to glyphosate that is less than seven days of exposure in a lifetime? 03:46
A. Yes.
Q. And, in your opinion, when you look at this analysis -- 03:46
A. It's not seven days per lifetime. It's seven lifetime days as defined by this product.
Q. Okay. And you would agree that when this data is analyzed for pack year type analysis or lifetime days analysis, there's no indication of any greater risk of non-Hodgkin's lymphoma in the group that has the greater than seven days lifetime exposure; correct? 03:46 03:47
MS. FORGIE: Object to the form. Mischaracterizes her testimony.
THE WITNESS: Well, lifetime days seem to be a measure that doesn't show a dose response here. However, frequency 03:47

of years -- days per year which is not really a frequency but an intensity, seems to have an effect.
BY MR. LASKER:
Q. And your belief that this is an intensity is based upon your understanding of what a day of exposure means? 03:47
A. Correct.
MS. FORGIE: Objection.
BY MR. LASKER: 03:47
Q. And for day of exposure, would that be different -- defined differently for a lifetime day, each day and that day of exposure as compared to a frequency day?
MS. FORGIE: Object to the form. 03:47
THE WITNESS: So these frequencies go from zero to who knows what; correct? Number of days per year. And when you multiply those by years, then you could have very high intensity days with a low number of years landing in the lower category, or you could have the opposite. So there's a lot of potential for exposure misclassification in terms of who's a regular user and who is not. 03:48 03:48

BY MR. LASKER:
Q. And without knowing more about how a defined exposure for frequency days, there could be exposure misclassification throughout this entire analysis in duration, in frequency, and in lifetime days; correct? 03:48
MS. FORGIE: Object to the form.
THE WITNESS: Well, duration is defined as duration, but we don't know what the intensity is. So that would just be a measure of duration. It could be a very low intensity; it could be a very high intensity. It's just duration. 03:48
On the other hand, frequency which I call intensity in this case distinguishes the high use from the low occasional use. There's no duration in this. We can only assume how it relates to duration, but they're not showing us data that relates frequency and duration. And then this made-up lifetime days is a product of years, number of years times number of days per year. So a product of the two above. 03:48 03:49

And the one with the larger span will weight the other to nothing or to whatever that is.
So what we're seeing in duration year gets reflected in lifetime years only in lifetime years it's even more misclassified because it mixes intensity with duration. 03:49
BY MR. LASKER:
Q. At the time you prepared your original expert report in this case, were you aware of the fact that the NAPP had conducted this further analysis of duration and lifetime days exposure to glyphosate? 03:49
MS. FORGIE: Object to the form. 03:49
THE WITNESS: At what time?
MS. FORGIE: Asked and answered.
BY MR. LASKER:
Q. At the time you prepared your expert report in this case. 03:49
A. I hadn't seen this.
Q. Okay. Also on this page there is a sensitivity analysis for proxy respondents, use of proxy respondents; correct?
A. You mean the same table? 03:50

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Q. Yes.
A. The same table distinguishes between proxy and self and self-respondents. So it's not really a stratified analysis. It's a sensitivity analysis. 03:50
Q. Right. That's what I said. It's a sensitivity analysis; correct?
A. Yeah, yeah.
Q. When they conducted their sensitivity analysis, they found that for 03:51
the never/ever category the odds ratio for self-respondents only for glyphosate and non-Hodgkin's lymphoma and all of the case control studies pooled in North America, U.S. and Canada, was 0.95 with a confidence 03:51
interval of 0.69 to 1.32; correct?
A. That's what they're reporting.
Q. And that is, in fact, the -- if we're looking at the -- just a second. Okay. Let's talk about the Eriksson paper. 03:52
Let's change. I'm sorry. I got this note. I just completely ignored it.
THE VIDEOGRAPHER: This marks the end of videotape number 3 in the deposition of Dr. Beate Ritz. We're off 03:52

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the record at 3:51 p.m.
(Recess taken from 3:51 p.m. to 4:02 p.m.)
THE VIDEOGRAPHER: We are back on the record at 4:02 p.m. This marks the 04:03
beginning of videotape number 4 in the deposition of Beate Ritz.
BY MR. LASKER:
Q. Dr. Ritz, I'd like to direct you to Exhibit 19-7, which is the Eriksson study. 04:04
I just have a few questions.
MS. FORGIE: Do we have it?
MR. LASKER: She's got it.
BY MR. LASKER:
Q. We previously discussed the fact 04:04
that --
MS. FORGIE: Hold on a second.
MR. LASKER: Let's go off the record.
THE VIDEOGRAPHER: We're off the 04:04
record at 4:03 p.m.
(Recess taken from 4:03 p.m. to 4:03 p.m.)
THE VIDEOGRAPHER: We are back on the record at 4:03 p.m. 04:04

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BY MR. LASKER:
Q. Dr. Ritz, we were talking about the Eriksson study. I think earlier we established that the only odds ratio in this paper or the only table that includes odds 04:04
ratios in this paper that were adjusted for the pesticide exposure is table 7 where the multi-variate analysis is presented on page 1661; correct?
A. Yes. 04:05
Q. Now, when you look at the other odds ratios in these other tables that are not adjusted for other pesticide exposures, virtually every odds ratio for every compound and every chemical that is analyzed 04:05
is reported at above 1.0; is that correct?
A. That's a very simplified statement because a lot of the odds ratios are right around 1.
Q. Virtually every single one of the 04:05
odds ratios that are reported in this paper are above 1.0; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Again, there are lots of odds ratio hover above 1. There are 04:05

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odds ratio below 1, and there are odds ratios above 1, and there are lots of analyses that are including the same subjects. So if you want to do odds ratio counting, you need to discount the 04:06
ones that are using the exact same data on the exact same people.
BY MR. LASKER:
Q. Correct. And when you do that, the vast majority of these odds ratios reported 04:06
in Eriksson are above 1.0; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Again, that's not how I look at this. I look at this as odds ratios reported for different agents for 04:06
different purposes. One is a yes/no, ever/never. Other purposes are intensity or duration measures, and splitting up groups into less and higher intensity, you can see how nicely dose 04:06
response patterns are starting to emerge. And the lower odds -- the lower exposure odds ratios usually include a close to 1, and the confidence intervals include 1. 04:07

BY MR. LASKER:
Q. Let me ask you this question generally: If you have a case control study, and you are -- I think you refer to this in your expert report at page 8 when you're talking about the fact that the De Roos 2003 study had odds ratios below 1 and above 1. And one of the things you stated there is that if you have odds ratios in a case control study for multiple agents and they're all above 1, you would have a concern for -- about recall bias; is that correct? 04:07
MS. FORGIE: Object to the form.
BY MR. LASKER: 04:07
Q. And you can look at page 8 on your expert report.
A. Where is it?
Q. At the very top you stated, "If recall bias existed, you would expect all pesticides reported to show an association with the outcome and not just one among many since the tendencies to recall better and more exposures than controlled would not be expected to be specific to one chemical." 04:07 04:08

Correct?
A. Yes.
Q. So if you have all chemicals in a study where you have elevated odds ratios, one of the things you would be concerned about, in general, is the possibility of recall bias; correct? 04:08
MS. FORGIE: Object to the form.
THE WITNESS: In general, if it's all chemicals, yes, but in this study I see a lot of odds ratios that are around 1 or even below 1 reported, and many of the odds ratios are duplicate analyses in terms of a dose response. So there's an analysis of an ever/never, and then for the same people we are now categorizing them in several categories to explore a dose response. 04:08 04:08
In that case I would expect that the overall estimate is somewhere a weighted average of the categories that I'm looking at. And in many cases you can see that the specificity increases. That's why we do this. So the specificity of exposure increases with 04:09 04:09

intensity or duration of use, and that's informative. When it doesn't, then it actually dissuades me that this agent is actually contributing.
BY MR. LASKER: 04:09
Q. Dr. Ritz, if you look at Table 5 in the Eriksson study which looks at insecticides total, DDT, mercurial seed dressing, pyretrine, other, every single odds ratio reported in that table is above 1; correct? 04:09
MS. FORGIE: Object to the form.
THE WITNESS: The confidence intervals, many of them include the 1, and it is a table of subtypes meaning we're now going into very, very small subgroups with very low exposures. So essentially a lot of these estimates are non-informative. 04:10
BY MR. LASKER: 04:10
Q. Let's skip over to --
A. And some are actually below 1. Clearly below 1.
Q. Let's skip over to the De Roos 2005 cohort study. First of all, I'd like to 04:10

mark as --
MS. FORGIE: Are we putting this away?
MR. LASKER: Yeah.
MS. FORGIE: Thank you. 04:10
MR. LASKER: So this is 19-17.
(Exhibit Number 19-17 was marked for identification.)
BY MR. LASKER:
Q. Dr. Ritz, this is a slide deck that unfortunately we received in this form. It's a little bit difficult to read, but this is a slide deck you produced to us in response to our document subpoena. 04:11
I take it this is a slide deck you've used in training in teaching of your class; correct? 04:11
A. Yes.
Q. And the glyphosate case control studies that we've been discussing are what are called retrospective in that they take individuals with NHL or without NHL, and then they look back in time and ask them about their prior exposures; correct? 04:11
MS. FORGIE: Object to the form. 04:11

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THE WITNESS: They are case control studies in which cases and controls report their lifetime use of pesticides.
BY MR. LASKER:
Q. So retrospective analyses; correct? 04:12
MS. FORGIE: Object to the form.
THE WITNESS: It's not an analysis that's retrospective. It's the exposure assessment that's retrospective.
BY MR. LASKER: 04:12
Q. So the exposure amendment in the case control studies are retrospective; right?
A. Correct. Not always. In this one. In these because they're questionnaire 04:12 based. They're case control studies that follow records, and they not retrospective.
Q. In the case control studies, is it your testimony that there are glyphosate case control studies that are not 04:12 retrospective in their gathering of exposure data?
A. Not in the literature that I reviewed.
Q. Okay. Strike that. Or don't 04:12

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strike that. Let me just make clear. In the literature you reviewed, in the case control studies you reviewed for glyphosate, are all of those containing exposure information retrospective? 04:12
MS. FORGIE: Object to form. Asked and answered.
You can answer it again.
THE WITNESS: They had questionnaire that were sent out to 04:13 cases and controls asking them about lifetime exposure. In that sense it's a retrospective exposure assessment.
BY MR. LASKER:
Q. And it is true as you teach your 04:13 students -- and this is on page 2. It's the top slide on the right -- that retrospective often is considered a less reliable design in an epidemiologist study; correct?
MS. FORGIE: Object to the form. 04:13
THE WITNESS: Well, that is a very broad statement.
BY MR. LASKER:
Q. I'm just asking about the statement you make in your slide presentation -- 04:13

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A. Where is it?
Q. -- to your students. It is the top slide on the left on page 2. "Retrospective is often considered a less reliable design." Is that correct? 04:13
A. Yes. And that does not refer as a judgment to case control studies but to the term "retrospective," and this is not to say that it really is a lesser way and a less reliable design. That's why it's in quotes. 04:14 This is to stimulate my students to think about the advantages of this kind of exposure assessment.
Q. And on page 5 in your slide deck for your students in the top right for 04:14 discussing cohort studies, you state that cohort studies are generally most accepted in scientific community; correct?
A. Again, that is to stimulate discussion about is that really a criterion 04:14 we should be using as epidemiologists even if the scientific community equates cohort studies with higher study quality. One of the things I do in my class is I start with this where there is that validity ranking 04:14

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highest to lowest, and I try to debunk it.
Q. And just to be clear, the "this" because that won't be on the record, you start on page 1 with your Table 1, which is a listing of validity ranking from highest 04:15 to lowest, and this is, I take it, what is generally presented in the scientific literature as the ranking of study designs by validity; correct?
A. Correct. 04:15
MS. FORGIE: Object to the form.
THE WITNESS: Well, this is how many people think about epidemiologic or medical trials and designs, yes.
BY MR. LASKER: 04:15
Q. And in this ranking, randomized clinical trials are the highest, and prospective cohort studies are directly below that; correct?
A. That's correct. 04:15
Q. And there is a term for "nested case control study." That is a case control study that is conducted within a cohort; correct?
A. Yes. Sometimes it's used for 04:15

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population-based case control study as long as you know what the source of controls was.
Q. Okay. And in this sort of general ranking in the scientific community of design validity, where would a non-nested case control study fit in this ranking? 04:16
A. Right below case control study.
Q. So a case control study would be below nested case control study and above time series analysis? 04:16
A. Correct.
Q. Okay. The one cohort study that we have for glyphosate and non-Hodgkin's lymphoma or the one cohort analysis is from the Agricultural Health Study; correct? 04:16
A. Correct.
Q. So let's look to that now.
A. Just for the record, I'm using this to stimulate discussion because I disagree with this ranking presented in Table 1. 04:16
Q. So this is 19-18.
(Exhibit Number 19-18 was marked for identification.)
BY MR. LASKER:
Q. And for -- so Exhibit 19-18 is the 04:17

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2005 published AHS study of glyphosate by De Roos; correct?
A. Yes.
Q. You mentioned this study in your report at page 21. You can go to that. And 04:17
you present right above that chart the odds ratio for the De Roos 2005 study for glyphosate and non-Hodgkin's lymphoma as 1.2; correct?
A. Yes. 04:17
Q. And if you look at De Roos in Table 2 on page 51, the odds ratio that you report in your expert report is the odds ratio that is minimally adjusted, only adjusted for age; correct? 04:18
A. I report two -- I report 1.2 and next to it the 1.1.
Q. I'm sorry. Got it. My mistake.
And you mention in your expert report that the confidence interval for the 04:18
finding in the De Roos study is wide, 0.7 to 1.9, which you describe as a wide confidence interval; correct?
A. Yeah. And they're exactly the same. 04:18

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Q. And this confidence interval, if you were to calculate the CLR for the De Roos study to measure the width of the confidence interval, for the De Roos study 1.9 to 0.7. That is, again, somewhat below 04:19
3; correct?
A. Slightly, yeah.
Q. And that confidence limit ratio is actually narrower than the CLR for the case control studies for adjusted odds ratios 04:19
that we've been reporting that we've been talking about; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Again, that's not the only criteria to evaluate statistical 04:19
significance or confidence interval or any meaning that these estimates might have.
BY MR. LASKER:
Q. I understand. I'm just trying to 04:19
get an understanding because in your report you discuss this confidence interval as being wide, and, in fact, this confidence interval is narrower than the confidence interval that appears in the case control 04:19

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studies that we've been discussing; correct?
MS. FORGIE: Object to the form.
THE WITNESS: For a cohort study this is a rather wide confidence interval especially if you look at some 04:19
more common cancers. It should be better. Yes, the one for all cancer. It's .9 to 1.1. That's a nice confidence interval.
BY MR. LASKER: 04:20
Q. I understand that. But I'd like to ask you with respect to the case control studies. Would it be correct to my understanding that the confidence interval for glyphosate and non-Hodgkin's lymphoma in 04:20
the De Roos 2005 study is not wide as compared to the odds ratios for glyphosate and non-Hodgkin's lymphoma reported in the case control studies?
MS. FORGIE: Object to the form. 04:20
Asked and answered.
You can answer it again.
THE WITNESS: These confidence intervals might be comparable. However, it's even more important that the 04:20

confidence interval safely includes the overall meta-analytic point estimate of 1.45.

BY MR. LASKER:

Q. I'm sorry. I have no idea what that is. It seems like a meta conference interval that was reported by -- 04:20

A. No, I'm talking about the point estimate falling nicely into this wide confidence interval for NHL. So this study does not contradict the meta-analysis. That's what I'm saying. 04:21

Q. So the meta-analysis number you're reporting, you're discussing here, is the meta-analysis number from the -- 04:21

A. From several --

MS. FORGIE: Wait for the question.

BY MR. LASKER:

Q. -- from the IARC meta-analysis and the Chang and Delzell meta-analysis that did not include the NAPP data; correct? 04:21

MS. FORGIE: Object to the form.

THE WITNESS: Yes, that's correct.

BY MR. LASKER:

Q. And the De Roos 2005 study in and 04:21

of itself fair to say does not report a positive association between glyphosate and non-Hodgkin's lymphoma; correct?

MS. FORGIE: Object to the form.

THE WITNESS: A 1.2 to 1.1 is still a positive association. 04:21

BY MR. LASKER:

Q. In your opinion, does the De Roos 2005 cohort study provide evidence that supports the hypothesis that glyphosate causes non-Hodgkin's lymphoma? 04:22

A. It contributes very little.

Q. Okay. But that's not quite answering my question.

Do you believe that the De Roos 2005 cohort study provides some evidence, even if you think it's little, in favor of an opinion that there's an association between glyphosate and non-Hodgkin's lymphoma? 04:22

MS. FORGIE: Object to the form.

Also, asked and answered.

You can answer it again.

THE WITNESS: This study does not, in the way it's reported here and in the 04:22

way I see these data, does not contribute very much to the discussion.

BY MR. LASKER:

Q. Okay. And the Table 3 analysis, I take it, which sets forth the various risk ratios based upon two measures of exposure, either cumulative exposure days or intensity weighted exposure days, am I correct in my understanding that you do not believe this data contributes much to the analysis of glyphosate and non-Hodgkin's lymphoma? 04:22 04:23

MS. FORGIE: Object to the form.

Also, asked and answered.

You can answer it again.

THE WITNESS: These tables are much more complex to analyze because we're now getting into a discussion over appropriate exposure assessment which I don't think is -- the exposure measures that are used here to derive these total cut points are most likely misclassified. 04:23

BY MR. LASKER:

Q. Now, these exposure measures are the same exposure measures the Agricultural 04:23

Health Study has used in numerous different epidemiological studies that were being published at the same time that you were serving on that outside advisory committee; correct? 04:23

MS. FORGIE: Object to the form.

THE WITNESS: An exposure measure for one pesticide is not exactly the same as an exposure measure for another pesticide, and I think we agreed today that it depends on when these pesticides were used and where they were used and whether use changed. There's no other pesticide for which use changed in the same way that I can think of, at least as for glyphosate, during the general baseline enrollment of these farmers. 04:24 04:24

THE REPORTER: I'm so sorry. My computer just rebooted.

MR. LASKER: How much did we miss and what do we have to do?

THE REPORTER: No, I've got it all the writer. I just need to go off and reboot.

MS. FORGIE: Why don't we take a

short break.
THE VIDEOGRAPHER: We're off the at 4:23 p.m.
(Recess taken from 4:23 p.m. to 4:47 p.m.) 04:47
THE VIDEOGRAPHER: We are back on the record at 4:47 p.m.
BY MR. LASKER:
Q. Dr. Ritz, we were looking at De Roos 2005. I'd like to actually direct you to Table 1 on page 50. 04:47
A. Yeah, I'm there.
Q. And that table, at the bottom, presents data from this cohort on co-exposures for glyphosate and other common pesticides or exposures in individuals not exposed to glyphosate; correct? 04:47
A. Yes.
Q. Okay. And for every pesticide in this cohort, they found that as there was increased use of glyphosate, there was also increased use of these other pesticides; correct? 04:48
MS. FORGIE: Object to the form.
THE WITNESS: I'm confused. Should 04:48

I answer.
BY MR. LASKER:
Q. Yes.
MS. FORGIE: If you understand the question, you can answer. 04:48
THE WITNESS: So you're saying there's correlation between pesticide use and the AHS?
BY MR. LASKER:
Q. I'm saying that for every pesticide that they looked at, and there's, I think, ten pesticides listed on Table 1, they found that with glyphosate use and with greater glyphosate use, there was greater use of these other pesticides; correct? 04:48
MS. FORGIE: Object to the form.
THE WITNESS: These pesticides correlate with glyphosate, yes.
BY MR. LASKER:
Q. So you have a correlation between increased glyphosate use and use of these other pesticides; correct? 04:49
A. That's how it looks like.
Q. And if I understand correctly, if any of these other pesticides are, in fact, 04:49

risk factors for NHL, that would introduce a differential confounding so that you'd have a greater confounding of your glyphosate measure with higher glyphosate exposure as compared to lower glyphosate exposure; correct? 04:49
MS. FORGIE: Object to the form.
THE WITNESS: Not necessarily. This really depends on how you look at glyphosate data in terms of, first of all, is it -- is any of these other pesticides really a -- you said that, NHL risk factor. 04:49
(Simultaneous cross-talk interrupted by the reporter.) 04:50
MS. FORGIE: Wait, wait.
THE WITNESS: Are they correlated with glyphosate exposure, but then couldn't you imagine that even a true risk factor for NHL that's correlated with glyphosate has two different meanings. One, it might be a risk factor that's on its own, but it also could be an indicator for pesticide use, glyphosate, and that's what this is also 04:50

showing.
So all of these pesticides are perfect indicators of glyphosate use.
BY MR. LASKER:
Q. Okay. My question -- I'm going to try to understand this, your answer, but let me just make sure I understand this. 04:50
Given this data showing that there is increased correlation between glyphosate exposure and exposure -- strike that. 04:50
Given this data that there's an increased correlation with use of other pesticides and glyphosate with increasing use of glyphosate, is one possibility given this data that there is -- if any of these other pesticides are associated with non-Hodgkin's lymphoma, that there is increased confounding for higher doses of glyphosate exposure? 04:51
MS. FORGIE: Object to the form. 04:51
THE WITNESS: So it's not increased confounding. It's some -- it can be some type of confounding. It can also be a proxy for the exposure. It was all highly correlated exposures. That's the 04:51

case. You have to decide whether it's a confounder or a proxy.

BY MR. LASKER:

Q. Okay. And if the pesticides are confounders and we determined that, for the purposes of this question, that they are independent causes of non-Hodgkin's lymphoma, and you were to compare the odds ratio for glyphosate exposure for the lowest exposed to the highest exposed, you could have confounding -- if you don't control -- adjust for those other exposures, you could have confounding that would inflate the odds ratio for the higher glyphosate exposure as compared to the lower glyphosate exposure. 04:51 04:51 04:52

That's possible; correct?

MS. FORGIE: Object to the form.

THE WITNESS: So confounding is always a possibility especially with highly correlated exposures. So the intellectual challenge here is to decide how to treat these variables. Are they truly confounders in the sense that we are assuming that glyphosate has no effect and all the effect comes from the 04:52 04:52

other pesticide, or are there one or two or three carcinogens, all of them contributing to the risk of NHL, and how do we put those together in a model if we -- if they're highly correlated, we put them all three in the model, then they will just split variance, and none of them will show anything. 04:52

BY MR. LASKER:

Q. And if we have that situation, the real challenge we have, if I understand you correctly, is, let's say, if we have four pesticides, we have glyphosate and we have three other pesticides, and they are often used together, and you have this situation with a correlated, and you have positive associations popping out with each of the different chemicals, then am I correct in my understanding that it is difficult to reach a determination as to whether all of them are, in fact, associated with increased risk of NHL or one of them is and which one is; correct? 04:52 04:53 04:53

MS. FORGIE: Object to the form.

THE WITNESS: That's not what I'm 04:53

saying. I'm saying that the data and the mass will not help you. What you have to do is design a study in which you can distinguish between these three exposures -- four exposures, and make up your mind what to call these individual agents. Are they truly risk factors increasing the risk of NHL, or are they not. 04:53

If all four of them are risk factors, and they are highly correlated so every time one person is exposed to one, they're also exposed to all three others, then you don't have a study that you can actually -- from which you then can come with a conclusion on one of them. 04:54 04:54

All you can say is all four of them seem to increase risk of NHL.

BY MR. LASKER: 04:54

Q. And has there been a study, to your mind, that has allowed -- that would allow one to parse that out?

A. Yes.

Q. Which study would that be? 04:54

A. That would be the hypothetical study in glyphosate production workers.

Q. I'm sorry. I misspoke. My question was: Has there been, in fact, an epidemiological study conducted that you've reviewed that would allow you to tease out that fact between the different pesticide exposures? 04:54

MS. FORGIE: Object to the form.

THE WITNESS: That depends on which study we are talking about because confounding is a study-specific issue. So in some studies, one of these pesticides may be a confounder. In another study, it might not be, and that would depend on the timing of exposure. 04:54 04:55

So for this study, the AHS where we only have farmers who are coming for a pesticide exam at baseline. Right? That's how they were enrolled. They came to an exam in Iowa or North Carolina to get their pesticide application license. 04:55

So we know from the beginning that this is a cohort that will have multiple 04:55

pesticide exposures, and a lot of them will be highly correlated. In other populations, it might not be as much of a problem because certain farmers may just use glyphosate and nothing else. 04:55

BY MR. LASKER:

Q. I understand.

My question to you, though, is: You've reviewed all the epidemiologic literature; so if there is a study, that's 04:55 fine. You can let me know what study that is.

Is there an epidemiological study that you've identified in the literature that allows you to distinguish between 04:55 glyphosate and other pesticides that are potentially being used by that population to determine whether all of them are risk factors, one of them is a risk factor, or distinguish between them? 04:56

MS. FORGIE: Object to the form. Also asked and answered.

THE WITNESS: Well, I think the De Roos 2003 study is actually a very good example where even after we adjust for 04:56

40-some pesticides, the effect of glyphosate is still apparent.

BY MR. LASKER:

Q. And is that in the hierarchical regression analysis? 04:56

A. That is in the logistic regression, and I stated before that I do not think that the hierarchical is the way to go for many reasons because it makes all these assumptions about carcinogenicity of 04:56 substances we don't know anything about.

Q. Other than De Roos 2003, is there a study that you believe allows you to tease out the effects of glyphosate versus another pesticide to determine which of those are 04:56 risk factors and which of those are just correlated?

A. I believe that the Eriksson study also made multiple adjustments and glyphosate survived those, but it is real 04:57 study to study. We could go through all of them.

Q. The De Roos 2005, in their dose response analysis, as they performed their analysis for cumulative exposure days, they 04:57

reported risk ratios of below 1 for the higher tertiles of exposure for cumulative exposure days and also intensity-weighted exposure days; correct?

A. That's how it looks like. 04:57

Q. The number of days of exposure to glyphosate in the exposed members of the AHS cohort in the highest exposure group was significantly higher than the reported days of exposure to glyphosate in any of the case 04:58 control studies; correct?

MS. FORGIE: Object to the form.

THE WITNESS: I'm actually very surprised to see this number. I can't imagine anybody was spraying glyphosate 04:58 on a daily basis for seven years.

BY MR. LASKER:

Q. The data in this study for De Roos would span 27 years of potential glyphosate use; correct? 04:58

MS. FORGIE: Object to the form.

THE WITNESS: It would be -- no. It would be use between -- let's see.

BY MR. LASKER:

Q. Between -- 04:58

MS. FORGIE: Wait, let her finish.

THE WITNESS: -- whatever we're assuming is the introduction of glyphosate and the first person in this cohort having used it. Some of these 04:58 farmers -- or actually the bulk of these farmers were less than 45 -- 40 years -- 50 years of age when they were enrolled. So I don't think they might have used glyphosate -- well, depends on the age 04:59 they started farming; right?

BY MR. LASKER:

Q. Yes.

A. So it could be 1975 to enrollment. So that would be -- the latest enrollment is 04:59 1997; so we have 22 years maximum.

Q. Okay. And is it your testimony that you believe that the data presented in this table with the maximum, and it is the single maximum exposure of 2,678 days, do 04:59 you believe that that data point is incorrect?

MS. FORGIE: Object to the form.

THE WITNESS: I have no idea, but I'm very surprised to see it. On the 04:59

other hand, these are farmers who are high intensive users of pesticides; so maybe there's something to it.

BY MR. LASKER:

Q. Am I correct that the 2005AHS data presents data for exposures that are significantly more intense than any of the exposures that are assessed in any of the case control studies that we've talked about; correct? 04:59 05:00

MS. FORGIE: Object to the form.

A. So now we are coming to the exposure assessment that was done in 1993 to 1997. As we know in 1995-'6 there was a big change in glyphosate use due to genetically modified crops. So the individuals who were enrolled in 1993 would report general use among farmers where glyphosate is just one among several herbicides; right? Could be 2,4-D. Could be atrazine, could be all sorts of thing. And then we have this big, big switch in 1995, and you're still enrolling these farmers, and now they have started to use modified crops, and they're using glyphosate at a huge amount. And what 05:00 05:00

you're now having is a situation where you don't know anything about what people in 1993 did. You know who changed in 1995 to glyphosate-intensive farming, but you would not know who was interviewed in 1993 also changed to glyphosate-intensive farming. You would keep them in the low exposure even though they may have changed to a much higher level. 05:01

Q. My question was not that, though; so let me ask my question again and see what the answer is. Am I correct in my understanding that the cohort that was analyzed in the De Roos study had significantly more intense exposures both by cumulative exposure days and to intensity measure to glyphosate than any of the individuals who were assessed in the case control studies we've been discussing? 05:01 05:01

MS. FORGIE: Object to the form. Also asked and answered.

You can answer again.

THE WITNESS: So I'm having a hard time comparing them because the other studies had more than two days. That 05:02

could also be a hundred days; right? So plus those were days per year. Here we have a cumulative exposure meaning this could be an average that's actually less than what was reported in the other studies depending on the number of years. 05:02

BY MR. LASKER:

Q. The two data points we have from Eriksson, it was ten days -- more than ten days or less than ten days; correct? 05:02

A. Yes, but I'm not sure that it was ten days per year or ten days cumulative.

Q. Okay. I'll represent, and if I'm wrong, the court will know and everybody will know that it was ten days cumulative. 05:02

The NAPP data we just looked at reported seven days cumulative as the cutoff point; correct?

MS. FORGIE: Object to the form. 05:02

THE WITNESS: That was the cumulative, yes.

BY MR. LASKER:

Q. So we have a cutoff of seven days cumulative for the NAPP U.S.-based case 05:03

controls. We have a cutoff of 10 days cumulative for the Eriksson study, and we have a cutoff in the De Roos 2005 cohort that goes 1 to 20 days cumulative for the low exposure group, 21 days to 56 days for the mid exposure group, and 57 days to 2,678 days in the high exposure group; correct? 05:03

A. Correct.

MS. FORGIE: Object to the form. 05:03

THE WITNESS: Over 22 years.

BY MR. LASKER:

Q. And my question -- and for the Eriksson study, you'd have that same time period generally, the number of years of exposure -- of potential exposure; correct? 05:03

MS. FORGIE: Object to the form.

THE WITNESS: That was --

BY MR. LASKER:

Q. The 2008 study? 05:03

A. I have to look. When did they get their cases? 1993? So it's shorter. It's actually shorter because the cases were ascertained in the early '90s and these cases were ascertained after. 05:04

Q. And we're not going to go back. I don't think that's correct, but we'll move on and address that later.

The cumulative exposure in the De Roos study, measured in the De Roos study for glyphosate associated with non-Hodgkin's lymphoma was significantly greater than the cumulative exposure measures in any of the case control studies; correct? 05:04

MS. FORGIE: Object to the form.

THE WITNESS: Again, this is a measure that's cumulative over 22 years, and it is not a measure of intensity.

BY MR. LASKER:

Q. Okay. And the intensity-weighted exposure days that was presented, that is based upon an analysis of intensity in the AHS that looks at mixing status, application method, equipment repair status, and personal protective equipment; correct? 05:04

A. Yes.

Q. And that is a measure that has been looked at and validated through the De Roos -- through the AHS to try and measure the intensity of exposures not only 05:05

to glyphosate but to all the pesticides that they analyzed; correct?

MS. FORGIE: Object to the form.

THE WITNESS: What was that.

BY MR. LASKER: 05:05

Q. The measure of intensity that the Agricultural Health Study uses is a measure that they have validated not only for glyphosate but for all the different pesticides that they're analyzing; correct? 05:05

MS. FORGIE: Object to the form.

THE WITNESS: They actually did not validate that for all the pesticides. They used two or three pesticides for the validation procedure, and I wouldn't 05:05 call that validated because they are only measuring biomarkers over a very short period of time, and they are saying that these short time periods cannot be set to be the same as a 05:05 lifetime exposure.

In fact, we tried in my own studies for occupational exposures to pesticides to reproduce these intensity measures and compared them with very simple 05:06

measures. So we went through all the trouble of weighing in exactly the same way. We asked the same questions, and it made just about no difference whether you used a very simple measure such as 05:06 in Eriksson and Hardell, et cetera, or you used this very complicated measure.

BY MR. LASKER:

Q. When you say the measure that was used in Eriksson and Hardell you're assuming 05:06 the measure they used because they don't report it in those studies; correct?

MS. FORGIE: Object to the form. Mischaracterizes her prior testimony. Asked and answered. 05:06

You can answer it again.

THE WITNESS: No, because what Eriksson describes is very similar to the methods that I know I used. So we had several measures that we tried with 05:06 and without protective equipment, with and without frequency of applications, et cetera. We are using -- we tried to use everything in the same way as the AHS and going back to fairly simple 05:06

measures of, you know, how many times per year did you apply, or how many days per year did you apply made no difference.

BY MR. LASKER: 05:07

Q. In your discussion of the 2005 De Roos dose response analysis in your expert report at page 23, you state that the investigators' decision to conduct their dose response analysis with comparisons only 05:07 between low, mid, and high exposure without an unexposed group reduces the exposure contrast between the three dose groups; correct?

A. Where do I say that? 05:07

Q. Page 23. Right above industry-sponsored studies.

A. Yes.

Q. "This type of approach also reduces any remaining exposure contrast." 05:08

A. Yes.

Q. The exposure contrast, though, in the De Roos study were greater than the contrast between the exposure groups in the McDuffie study and the Eriksson study; 05:08

correct?
MS. FORGIE: Object to the form.
THE WITNESS: That's an assumption, and the assumption is that there's not a major exposure misclassification in the way I described before. 05:08
BY MR. LASKER:
Q. Okay. This exposure misclassification, to the extent that Eriksson analyzed data exposures going into the 1990s, if that's the case, they gathered their data after 1997, would that same issue arise with the Eriksson study? 05:08
A. If they gathered it after 1997, no, because then they would have actually already gotten past the change. 05:09
Q. Well, they --
MS. FORGIE: Wait. Let her finish.
THE WITNESS: The problem is that this study had the change happen in the middle of the enrollment period. 05:09
BY MR. LASKER:
Q. The Eriksson study would be looking back over time so it would be a questionnaire and be asking about prior 05:09

exposures over time; right?
MS. FORGIE: Object to the form.
THE WITNESS: It's a case control study so they would ask cases and controls to remember their lifetime exposure which, by definition, would be prior to the onset of the cancer, yeah. 05:09
BY MR. LASKER:
Q. So if the Eriksson study is asking that question after 1997 for all past exposures and using that data for their analysis, would they have the same misclassification problem that you believe exists for the AHS study? 05:09
A. No, it would not. 05:09
Q. The -- there has been a further analysis of the Agricultural Health Study data, and you address this in your rebuttal report. This is the document we received from Dr. Blair presenting data from 2013. 05:10
Let me ask first at the time that you prepared your initial expert report in this matter, had you seen that 2013AH analysis?
A. First time I was aware of it was in 05:10

that attachment to Dr. Blair's statements.
Q. Okay. But were you -- did you see that attachment -- had you seen that attachment at the time you prepared your initial expert report in this matter? 05:10
MS. FORGIE: Object to the form.
THE WITNESS: I don't believe so or else I would have known because the deposition was after -- when was it? Do we have a date? 05:10
BY MR. LASKER:
Q. We do have a date. I'll represent, and counsel can correct me if I'm wrong, the deposition was taken before your expert report was submitted. That doesn't mean you saw it then? 05:11
A. No, exactly. I don't think I saw any depositions prior to my expert report, so that's fine.
Q. And do you recall whether you saw the AHS2013 data prior to -- you obviously saw it prior to the time you did your rebuttal report. 05:11
A. Yes.
Q. Do you recall if you saw it prior 05:11

to the time you read Dr. Neugut's deposition?
MS. FORGIE: Object to the form.
THE WITNESS: I really don't know.
BY MR. LASKER: 05:11
Q. The 2013 -- why don't we mark that analysis.
(Exhibit Number 19-19 was marked for identification.)
MS. FORGIE: Tell me which version you're using.
MR. LASKER: March, 2013.
MS. FORGIE: So the earlier one.
MR. LASKER: The later one.
MS. FORGIE: Oh, the later one, I'm sorry.
THE WITNESS: Are there more than one.
MR. LASKER: There's February and March. The data doesn't change. 05:12
MS. FORGIE: I object to that comment. It does change. You know it.
MR. LASKER: I don't think it changes actually, but maybe I'm wrong.
///

BY MR. LASKER:
Q. The -- Dr. Blair in his deposition testified that the 2013 data, although for the glyphosate it is reported in a dose-response analysis that includes a never exposure category and then three exposure categories, he calculated that the ever/never risk ratio for glyphosate and NHL in this 2013 data would be about 0.9. Do you recall that? 05:12 05:13
MS. FORGIE: Object to the form. Mischaracterizes the testimony.
THE WITNESS: I don't recall that.
BY MR. LASKER:
Q. Okay. Let's look at Dr. Blair's deposition. I think we marked it as an exhibit. 05:13
MS. SHIMADO: 6.
BY MR. LASKER:
Q. I'm going to hand it to you. It's Exhibit 6 after we find it. 05:13
And Dr. Blair on page -- it's 172. We're looking at the 2013 cohort study data; correct?
MS. FORGIE: Well, she's not there 05:14

yet. She needs some time to read a couple pages before and after, so give her a minute, please.
THE WITNESS: What are we talking about? 05:14
BY MR. LASKER:
Q. On page 172 Dr. Blair is -- I'm asking him some questions about the 2013 data.
Do you see that? 05:14
A. Yes.
Q. I ask him the question at line 11. "This 2013 cohort study finds no association -- no evidence of association between exposure to glyphosate and non-Hodgkin's lymphoma; correct?" 05:14
And Dr. Blair answers, "Correct."
Do you see that?
A. Yes.
Q. And then I ask Dr. Blair, "And based upon the data that's set forth here, if you look at individuals who had no exposure to glyphosate, which is that first row, and you look at the three categories of individuals who did have exposure to 05:14 05:14

glyphosate, if we were to do an ever/never analysis of glyphosate and non-Hodgkin's lymphoma, the relative risk here would be something below 1.0; correct? About 0.9?"
"Answer: That's a reasonable guess I think, yes." 05:15
Do you see that?
A. Yes.
Q. Do you have any reason to disagree that if one were to do an ever/never analysis of the 2013AHS data for glyphosate, the risk ratio that would be reported would be something on the order of 0.9? 05:15
MS. FORGIE: Object to the form.
THE WITNESS: I would have to look at the data, but, in general, I don't believe any of those analyses because I don't believe the exposure assessment. So it doesn't matter. 05:15
BY MR. LASKER: 05:15
Q. I understand that, but let me just make sure I understand and see if you agree with what the numbers would be, and obviously others will decide whether or not those numbers are the -- the significance of 05:15

those numbers. But if we were to look at page 34 in the 2013 study for glyphosate, do you see that data?
A. Yes.
Q. And if we were to calculate from this data an ever/never risk ratio for glyphosate and non-Hodgkin's lymphoma, do you agree with Dr. Blair that the risk ratio would be about 0.9? 05:15
MS. FORGIE: Object to the form. Asked and answered. 05:16
You can answer again.
THE WITNESS: Again, it would be hovering somewhere around the 1. However, I don't think that these categories are sufficiently well established to even make this comparison. 05:16
BY MR. LASKER:
Q. Okay. But just so the record is clear, we have the non- -- the never use is the reference of 1.0; correct? 05:16
A. That's the reference, correct.
Q. And in the exposure groups, we have odds ratios of either below 1 or just at 1; 05:16

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correct?
MS. FORGIE: Object to the form. Asked and answered.
You can answer it again.
THE WITNESS: Well, the relative risks here which they are not odds ratios -- 05:16
BY MR. LASKER:
Q. I'm sorry.
A. -- are actually hovering around the 1. 05:16
Q. So the relative risks are either 0.8, 0.9, or 1.0 for use of glyphosate as compared to non-use of glyphosate as the data is reported here; correct? 05:17
MS. FORGIE: Object to the form. Asked and answered.
You can answer again.
THE WITNESS: Well, the relative risks are rate ratios hover around the 1 and the confidence intervals include the 1, but they go out to 1.4. 05:17
BY MR. LASKER:
Q. The -- in your rebuttal report, you state one of the main concerns you have 05:17

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about the 2013 analysis relates to the imputation method that was used; correct?
A. That's correct.
Q. And the AHS investigators -- and just to be clear, the issue with the imputation method is in their second phase of gathering information on pesticide exposures. They had, I think, 36 percent of individuals who responded to the first survey who didn't respond to the second; correct? 05:17 05:18
MS. FORGIE: Object to the form.
THE WITNESS: So the AHS is a cohort study that has, because there's so many people to be interviewed, a long period of enrollment which is about four or five years. And by the time the last person was enrolled, they pretty much decided they had to update their exposures because they realized that exposures change. 05:18
So in the next phase starting in 1999, I believe, through 2003, they tried to recontact all these farmers who they enrolled in the first phase, and 05:18

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yes, among those that they reached again, that was about 62 percent.
BY MR. LASKER:
Q. And because of that, the AHS investigators used an imputation method to impute what the values would be, the exposure values would be for the individuals who did not respond to the second phase questionnaire based upon the prior information that they had from those individuals and the information they had from the 60 plus percent of subjects who responded to both questionnaires; correct? 05:19 05:19
MS. FORGIE: Object to the form.
THE WITNESS: From what I understand is they basically used the baseline information to impute the follow-up. 05:19
BY MR. LASKER:
Q. So is it your understanding then that they did not use data from the 60 some odd percent who responded to both questionnaires -- 05:19
A. Oh, yes, because they used the baseline for all of them. 05:19

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Q. Used that as well to impute for them?
A. Yes.
Q. And the AHS investigators have used that same imputation method for every pesticide study that they have published that includes data from the phase 2 surveys; correct? 05:19
MS. FORGIE: Object to the form.
THE WITNESS: Yes. They used a general method of imputation for all pesticides, whether or not these pesticides were actually still in use or not, and whether or not the use changed over time specifically between the first and the second survey. 05:20
BY MR. LASKER:
Q. So every publication that has come out of the AHS that looks at pesticides since they've had this phase 2 exposure information, all of the published studies, all the peer-reviewed published studies from the AHS have used this same imputation method that was used in the 2013 analysis included glyphosate; correct? 05:20 05:20

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MS. FORGIE: Object to the form. Asked and answered. It mischaracterizes her prior testimony.
You can answer it again.
THE WITNESS: They used one single imputation method to apply to every single pesticide whether the pesticide has been banned and supposedly not been used since '72 which is DDT and lindane shortly after, or whether it's a pesticide that came on the market and went and was gone by 1993 when they started this study or whether it's a pesticide which is unique such as glyphosate that changed use in the middle of their inrollment period. And they're using the same method for all of these pesticides.
BY MR. LASKER:
Q. Just so I understand, every publication that's come out of the AHS since the second phase data was incorporated into their analysis, every peer-reviewed published study has made use of this general imputation method that was used in the 2013 study; is that correct?
MS. FORGIE: Object to the form. Also asked and answered. She's answered this twice.
You can answer it a third time.
THE WITNESS: Again, this imputation method is one and the same imputation method for every single exposure, and there are big differences between the exposures, the timing of the exposure and, therefore, the validity of this method. So every other paper that comes out has to be judged by how valid this method is, not only for the pesticide but also the outcome.
BY MR. LASKER:
Q. I understand that. But I just want to make sure that I'm clear that every paper that has come out of the AHS and including all the papers that have been peer-reviewed and published from the AHS have used the same imputation method that is used in the 2013 study; is that correct?
MS. FORGIE: Object to the form. Asked and answered. She's answered it four times now.
You can answer it again.
THE WITNESS: So it's a perfectly fine imputation method for something like DDT that supposedly hasn't changed since 1972, and it's a perfectly fine method for any pesticide that was discontinued in use since 1993 because what would change over time since 1993? Nothing. Right? Because supposedly all the exposures you could ever have had for this pesticide would have been recorded at baseline. This is not the case for any exposure that changed and especially not for an exposure that changed dramatically. There's only one I'm aware of in this study, and that was glyphosate for which that changed.
BY MR. LASKER:
Q. Just so I understand this correctly, and I think you'll agree with me on this, but I just need to understand this for the record, am I correct that every study that has been published by the AHS, every peer-reviewed published paper from the AHS looking at pesticides since that second survey was conducted has used the imputation methodology that is used in the 2013 study?
MS. FORGIE: Objection. I object to the form also. You are badgering the witness now. This is the sixth time you've asked the exact same question, the exact same question.
MR. LASKER: And one of these times I'll get an answer.
MS. FORGIE: Wait. Don't do that. You've gotten answers. You're badgering the witness. I object to that. Don't do that.
MR. LASKER: Mark the record here.
MS. FORGIE: Good. Please do.
MR. LASKER: I'm going to ask it again because it's a pretty simple question.
BY MR. LASKER:
Q. Am I correct -- and it's a question that has a yes or no. There may be an explanation you want to give afterwards. But it's a yes or no question. Am I correct that every peer-reviewed publication from

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the AHS that has come out since that phase 2 exposure data was collected has used the same imputation that is used in the 2013 study that included the glyphosate data?
MS. FORGIE: Objection. You are really badgering this witness. This is now like the eighth time. I'm counting. Objection. Asked and answered. 05:24
You can answer it again.
THE WITNESS: There is no yes or no answer to this. And, also, I don't know. Because, for example, if you're assessing lindane and DDT, you don't need an imputation method because you have all the data you want which is the data you collected at baseline. 05:25
However, for any pesticide still in use where you have no updated pesticide information, you would use this imputation method. Whether that's an appropriate method is a totally different question. For glyphosate, I don't believe so. 05:25
BY MR. LASKER:
Q. And the AHS investigators actually 05:25

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did a study in which they tried to test their imputation methodology and to look at how well it performed with respect to the different pesticides; correct?
MS. FORGIE: Object to the form. 05:25
THE WITNESS: It was a very special type of pesticide they looked at. It wasn't glyphosate from what I recall.
BY MR. LASKER:
Q. Let me ask you about the study. 05:25
Maybe we're not talking about the same study. The Heltshe study?
A. Yeah, Heltshe.
(Exhibit Number 19-20 was marked for identification.) 05:26
BY MR. LASKER:
Q. This will be Exhibit 19-20. This Exhibit 19-20 by Heltshe entitled, "Using Multiple Imputation to Assign Pesticide Use for Non-Responders in the Follow-Up Questionnaire in the Agricultural Health Study"; correct? 05:26
A. Yes.
Q. And in this study, they reported that their imputation methodology, and they 05:26

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report this in their abstract, that the distribution of prevalence and days per year of use for specific pesticides were similar across observed and imputated in the holdout sample. 05:26
Do you see that?
MS. FORGIE: Take your time.
BY MR. LASKER:
Q. It's towards the bottom in the abstract. 05:27
A. Oh, in the abstract.
Yes, they're using the data to predict the data.
Q. Right. And what they did in this analysis is they took of the people who had responded to the second phase, they randomly selected 20 percent of them; correct? 05:27
MS. FORGIE: Object to the form.
THE WITNESS: Yes.
BY MR. LASKER: 05:27
Q. And then they used their imputation method to predict what the imputation method would say was the exposure experience of that 20 percent holdout sample; correct?
A. That's correct. 05:27

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Q. And they compared that to the actual data because they had actual data from those individuals; correct?
MS. FORGIE: Object to the form.
THE WITNESS: They have actual data from those individuals that they are putting in the holdout sample, correct. 05:27
BY MR. LASKER:
Q. And they then used that analysis to check on the accuracy of their imputation method. And if you look at figure 2 on page 414, they measure the relative errors on page 414 for -- it's got to be 40 maybe, I didn't count them, but 40 different pesticides starting with methyl bromide on the top down to coumaphos on the bottom; correct? 05:27 05:28
A. Yes.
Q. And for each of those pesticides they checked to see how well their imputation methodology worked; correct? 05:28
A. Correct.
Q. And for glyphosate, they found that their imputation methodology worked about in the middle of the pack for all of these 05:28

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specifically identified pesticides as far as how well their imputation methodology works; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Well, it's not the middle of the pack. It's in relative error on the left of the zero. So they are underestimating. 05:28
BY MR. LASKER:
Q. But there's also one, two, three, four, five at the top. I've done the counting. I think there's maybe 17 that are more relative error, maybe 20 that have less relative error. But if you want to do the counting, you can. 05:29
A. But this is a prevalence, and we are talking about a relative error to predict a ever/never, and 75 percent of all people at baseline already reported use. So you can get, you know, this number very easily just because of the high prevalence. 05:29
Q. But my question to you is: In this published paper from the AHS in which they're checking the validity of their imputation methodology for the individual 05:29

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pesticides that they were analyzing, they found that glyphosate was about in the middle of the pack for prevalence as far as how well the imputation methodology worked; correct? 05:29
MS. FORGIE: Object to the form and asked and answered.
You can answer it again.
THE WITNESS: I don't think this answers to what I've just tried to explain. They can only use to predict from data they actually have; so we don't still know anything about the people for whom they don't have the follow-up data. 05:29 05:30
They are just assuming that those people behaved in the same way as the people they have data for.
BY MR. LASKER:
Q. I understand. 05:30
And the people they have data for would be people who cover this period that you're concerned about where glyphosate exposure increased. The folks who responded to the second survey and the first survey, 05:30

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that's the hold-out sample; correct? The 20 percent?
MS. FORGIE: Objection. Object to the form. And asked and answered.
You can answer it again. 05:30
THE WITNESS: This was done within the 62 percent who answered twice.
BY MR. LASKER:
Q. Right.
A. These 62 percent, as they describe in here, are actually different in many ways from the 30-some percent that did not -- 38 percent that did not answer. So they are using the 62 percent who are very different in many ways, and they actually acknowledging that they're also different in pesticide use to predict what would have happened to 38 percent that they did not have that second answer from. 05:30 05:30
It's easy to predict from people who are answering and are -- and are captured because they want to be captured. They could be younger. They could be more educated. All of that is described in here. So the people, 62 percent is not a 05:31 05:31

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representative sample of the 38 percent.
Q. Okay. I understand that. That's a different question, but I want to get at this issue of changes in glyphosate use over time. 05:31
The individuals who responded to the first survey and the second survey would obviously have gone through that period for glyphosate -- correct? -- where there was expanded use? 05:31
A. Only a small number would have gone through -- no. Okay. We have 1993 through 1997. So the 62 percent supposedly come from that whole time period; correct?
Q. And the second phase because they responded to the second phase as well. 05:32
A. Right.
Q. So '97 to 2001 as well. So for 62 percent, they have exposure data that spans before that first phase period and then also into the 1990s during that period where glyphosate use was impacted by GMOs; correct? 05:32
A. So some of these people, at baseline, would have reported use prior to 05:32

1995, and some would have responded past 1995.

Q. And they had that data?

MS. FORGIE: Wait. Let her finish her answer. 05:32

MR. LASKER: Well, I mean --

MS. FORGIE: No. She gets to finish her answer.

THE WITNESS: So some people changed, and other people didn't. Some 05:32 of this error is because some people changed, and it was a very simple change. So what they're talking about here is a change from yes, no.

There's only 25 percent at baseline 05:32 who did not report glyphosate use. So that's the only group that could have actually reported a change. Everybody else stayed the same if you say yes, no. That tells us nothing about the amount 05:33 of use.

BY MR. LASKER:

Q. Okay. Let me just break this down. First of all, in the original phase 1 study, we are looking at exposures over -- for 05:33

glyphosate, potentially, I think we talked about 20-plus years; correct?

MS. FORGIE: Objection. Mischaracterizes the testimony, and I'll object to the form. 05:33

THE WITNESS: So potential for exposure. We really don't know how far it goes back because none of the Eghal study papers actually describe for glyphosate how much in, you know, the 05:33 past these people reported use.

BY MR. LASKER:

Q. Okay. And what they're trying to measure in the second phase is how much exposure there was from the end of the first 05:33 phase to the second phase -- correct? -- which is a much shorter time period?

MS. FORGIE: Objection. Mischaracterizes the study itself.

THE WITNESS: So what they're 05:33 trying to do is to update the exposure information. Of course, the update is much more drastic in terms of amounts that somebody who reported in 1993 still use glyphosate but increased use in 1995 05:34

enormously and then responds again. Right.

BY MR. LASKER:

Q. And so for the 62 percent that responded to the questionnaire, that would 05:34 be information that you'd get from their second survey response; correct?

MS. FORGIE: Objection. Asked and answered and object to the form as well.

You can answer again. 05:34

THE WITNESS: You get updated information from these people who respond. However, to then use that data to predict how many people would use what who did not respond is a big step. 05:34

BY MR. LASKER:

Q. And I understand that step, and that's a step that we have for all of the pesticides, but for glyphosate, in looking at the individuals who responded at least 05:34 and who had gone through that period of increased use that you're talking about, that introduced whatever error it would introduce into the imputation methodology, and for those people, that error is 05:35

reflected in Table -- or Figure 2 on page 414; correct?

MS. FORGIE: Objection. Object to the form. Also asked and answered. She's answered this question at least 05:35 three times.

You can answer again.

THE WITNESS: And there are at least two wrong statement here. First of all, that's not correct for all the 05:35 pesticides. The pesticides that did not have this extreme change don't have this problem. This problem only has occurred because glyphosate use changed dramatically. 05:35

Second, this imputation method is a method that not only is used for a prevalence of glyphosate yes/no, but to also impute the amount used. And what they're showing you in this little graph 05:35 is just a prevalence yes/no. That's the least you could do and the least piece of information you can have about this method actually working.

Plus it makes the assumption that 05:36

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the 62 percent are representative of the 38 percent, and we have to make that assumption, and it's not right. They're stating that in this paper that it's not correct. 05:36

BY MR. LASKER:

Q. Within the 62 percent that responded when the AHS investigators looked to see for prevalence how well the imputation methodology worked, including the 05:36 fact that for those 62 percent, it spanned over that period when glyphosate use was expanding, the -- they found that the error in that 62 percent through the use of that imputation method when they tested it for 05:36 glyphosate was somewhere in the middle of the pack for all the pesticides that they analyzed, and that's reflected on Figure 2; correct?

MS. FORGIE: Objection. Object to 05:36 the form. You're badgering the witness. This is now about the fifth time you've asked that same exact question.

You can answer it again.

THE WITNESS: I don't believe you 05:37

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can say that because when you have such a high prevalence of use to begin with, 75 percent, then it is like a couple value where you're asking, well, how much agreement is there in a measure 05:37 when 98 percent say no, I never used this pesticide, and 2 percent do use it, and then you're, you know, getting -- okay, now next time around 4 percent say yes, but the 94 percent or the 05:37 75 percent are the overwhelming group that is consistent.

So because they already said yes at the baseline, they will consistently be predicted in the future because a yes is 05:37 a yes.

BY MR. LASKER:

Q. The concern that you are raising now about glyphosate and this imputation methodology is not raised as a concern by 05:37 the investigators, Dr. Heltshe and others, who presented the data for their validation study of the imputation method in which they presented glyphosate data along with the other pesticides; correct? 05:38

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MS. FORGIE: Object to the form.

THE WITNESS: I don't understand this question. Could you repeat?

BY MR. LASKER:

Q. The AHS investigators, including 05:38 Dr. Heltshe, conducted a validation test of their imputation methodology in this publication; correct?

MS. FORGIE: Object to the form.

THE WITNESS: What? A validation 05:38 method? No.

BY MR. LASKER:

Q. The investigators of the AHS study, including Dr. Heltshe, published this paper in 2002 presenting their data on how well 05:38 the imputation methodology worked through the analyses that they conducted in this paper for various pesticides; correct?

MS. FORGIE: No. Object to the form. 05:38

THE WITNESS: This is a 2012 paper.

BY MR. LASKER:

Q. Sorry.

A. And they conducted this method under lots of assumptions. The assumptions 05:39

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they made might be holding for most of these pesticide, but they themselves actually say that certain assumptions might be incorrect, including the missing at random assumption that they're making in this imputation, and 05:39 I'm saying that for glyphosate because of the time -- the exposure period change and the huge increase in glyphosate and that happening in the middle of the first enrollment period, this is not the method to 05:39 test this.

Q. I understand that that's what you're saying.

My question is: Dr. Heltshe and the other investigators who published this 05:39 analysis and presented the data on glyphosate in Figure 2 and also the findings for the other pesticides -- so in glyphosate relative error to be in the middle of the pack, they do not anywhere in this 05:39 publication state that this finding for glyphosate alone is not reliable; correct?

MS. FORGIE: Objection. That's the exact question you just asked twice. She's answered -- 05:40

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MR. LASKER: It's not the exact question. You're coaching the witness. You're coaching witness. I'm asking a different question.
MS. FORGIE: I'm not coaching the witness. I object. I object to the form. Asked and answered. 05:40
You can answer it again.
MR. LASKER: I'll ask the question again because I can't imagine how you're going to remember it at this point. 05:40
BY MR. LASKER:
Q. Dr. Heltshe and her co-investigators who presented this analysis in checking on the validation -- checking on the imputation methodology that they used and reported the relative errors for all of these various pesticides, including glyphosate, showing glyphosate to be in the middle of the pack for the different pesticides looked at in the AHS, nowhere in this publication do they state that there is a different concern about glyphosate that should be taken into account in analyzing the results that they present; correct? 05:40 05:41

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MS. FORGIE: Objection. Object to the form. Asked and answered.
You can answer again.
THE WITNESS: These authors investigated lots of pesticides. They are not making any reference to any single pesticide. They are just treating them as if they are equal in terms of their method. 05:41
BY MR. LASKER: 05:41
Q. They do not state that their method does not work for glyphosate in this analysis; correct?
MS. FORGIE: Objection. Asked and answered. 05:41
You can answer again.
THE WITNESS: In this paper, they are not stating anything specific for any of the pesticides.
BY MR. LASKER: 05:41
Q. Well, that's not true. In Figure 2, they have specific information on each of the pesticides. In Figure 1, they report specific information -- or Table 3, I'm sorry. They present specific 05:41

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information for specific pesticides; correct?
MS. FORGIE: Objection. It's not appropriate to tell the witness one of her answers is not true. 05:41
Objection. Also object to the form. Asked and answered.
You can answer again.
THE WITNESS: I may have misspoken. What I tried to do is answer your questions in terms of whether the authors actually commented on glyphosate being different. They did not comment on these pesticides being one or the other different. They are, of course, producing all of these data for all of the pesticides they imputed. 05:42 05:42
BY MR. LASKER:
Q. And the data that they presented and they decided to present to the world in this peer-reviewed publication so that people could understand their imputation methodology when they're reading these AHS studies that all now use this imputation methodology, the data they presented showed 05:42 05:42

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a relative error for glyphosate that was in the middle of the pack for all the pesticides that they are -- for which they're using the imputation methodology; correct? 05:42
MS. FORGIE: Objection. You're badgering the witness. You've asked her the same question so many times.
You may answer it again.
THE WITNESS: I think you don't understand what I'm getting at, and I'm sorry that I can't express myself in more lay terms or whatever I need to do, but this is not the same as a validation study of the imputation method, and the authors clearly state that this multiple imputation makes lots assumptions and that, you know, for simplicity of modeling, they only used a single set of observed complete data, et cetera, et cetera. 05:42 05:43 05:43
So it is not -- and they also say that some of these assumptions may not be correct and may have to be updated.
///

BY MR. LASKER:
Q. Can you point to anything in the published literature, in the AHS website, anywhere, anyone other than you has stated that the imputation methodology that the AHS 05:43
study is using is uniquely inappropriate for glyphosate?
MS. FORGIE: Object to the form.
THE WITNESS: Well, I haven't looked; so I don't know. 05:43
BY MR. LASKER:
Q. You're not aware of any statement from any of the AHS investigators that the imputation method that they are using for their phase 2 results are not appropriate 05:44
for glyphosate; correct?
MS. FORGIE: Object to the form.
THE WITNESS: I don't understand why they should be doing this if they haven't published on glyphosate. 05:44
BY MR. LASKER:
Q. Are you aware -- and I deposed Dr. Blair. In Dr. Blair's deposition when I deposed him, did he at any point state that the imputation method that was being used in 05:44

the 2013 study was not appropriate for glyphosate?
MS. FORGIE: Object to the form.
THE WITNESS: I can't remember.
BY MR. LASKER: 05:44
Q. In the -- in your role on the executive -- I'm sorry. Not the executive, the external advisory committee for the AHS to the present, have you ever heard anybody say that the imputation method that they're 05:45
using for the phase 2 respondents is not appropriate for glyphosate?
MS. FORGIE: Object to the form.
THE WITNESS: This is a 2012 paper. We have not met since they started doing 05:45
this. So nobody could have objected.
BY MR. LASKER:
Q. And there is nothing in the draft, the 2013 document that you've reviewed, that includes the glyphosate data that says 05:45
anything about the imputation methodology being inappropriate for glyphosate; correct?
MS. FORGIE: Objection to the form. Mischaracterizes the draft manuscript.
THE WITNESS: As far as I know, 05:45

this manuscript actually does refer back to the imputation method, and there was some back and forth between authors about how to present it.
BY MR. LASKER: 05:45
Q. Right.
But in that back and forth, is there any specific discussion that for glyphosate the method is not appropriate?
MS. FORGIE: Objection. Do you 05:46
want her to review to find it?
MR. LASKER: If you want to take a break, we can do that.
MS. FORGIE: No, we're not going to take a break. 05:46
THE WITNESS: So am I supposed to look.
MR. LASKER: Let's take a break.
MS. FORGIE: You're not going to look during the break, though. 05:46
THE VIDEOGRAPHER: We're off the record at 5:46 p.m.
(Recess taken from 5:46 p.m. to 5:54 p.m.)
THE VIDEOGRAPHER: We are back on 05:54

the record at 5:54 p.m.
BY MR. LASKER:
Q. Dr. Ritz, in your role as the chair of the external advisory committee to the AHS, have you spoken with anyone at the AHS 05:54
to share the opinion that you've been offering here today that the imputation method that they're using is inappropriate for glyphosate?
MS. FORGIE: Objection. Asked and 05:54
answered.
You can answer again.
THE WITNESS: I have not talked to them about glyphosate.
BY MR. LASKER: 05:55
Q. In your rebuttal report at page 7, you're talking about -- bottom of page 7, you're talking about the differences between peer-reviewed and unpublished -- a peer-reviewed paper and the unpublished 05:55
manuscript for the Agricultural Health Study 2013 analysis; correct?
A. I think I do. Where is it?
Q. Bottom of page 7, continuing to page 8. 05:55

A. Oh, yes.
Q. All right. One of the things that you state is that there is a footnote in the 2013 AHS analysis that includes glyphosate that states that numbers do not sum to totals due to missing data; correct?
A. Correct.
Q. Now, the manuscript that was the 2013 draft was subsequently published without herbicide data, so without the glyphosate data in 2014; correct?
A. There is a 2014 paper, and I went to that, yes.
MR. LASKER: So let's mark that. This is 19-21.
(Exhibit Number 19-21 was marked for identification.)
BY MR. LASKER:
Q. And 19-21 -- Exhibit 19-21 is the 2014 publication that was the subsequent revisions to the actual -- the 2013 study but without the herbicide data and substituted in fungicide and fumigant data; correct?
MS. FORGIE: Object to the form.

THE WITNESS: This is the insecticide paper. Fungicide and fumigant, right.
BY MR. LASKER:
Q. And if you look at the corresponding tables in the peer-reviewed published literature -- published study in 2014 and you look at the same footnotes that you were looking at in the 2013 study on those same tables, the peer-reviewed published article in 2014 likewise has the footnote that says that the number of cases do not total -- do not equal the total NHL cases because of missing data; correct?
A. Where is that?
Q. If you look at page 6, footnote 2.
A. The subtype, yeah. The subtypes due to missing data.
Q. If you look at page 10 for the dose response analyses of NHL, in general, footnote 2, the same statement, "The number of cases do not sum the total number of NHL cases because of missing data"; correct?
A. Yes.
Q. So that statement which appears

both in the peer-reviewed published 2014 paper and the 2013 draft; correct?
MS. FORGIE: Object to the form.
THE WITNESS: Well, it probably refers to different types of data because missing data are defined by what you're looking at, and this manuscript looked at the subpopulation of pesticides; so the missing data must be different.
BY MR. LASKER:
Q. This study looked at some of the same pesticides -- I know that the herbicides are dropped out, but it looked at some of the same pesticides as the 2013 draft; correct?
A. Yes. It overlaps in terms of all pesticides, but this paper should have less missing data because it dropped out the herbicides. The missing herbicide data should not be affecting this.
Q. So is it your testimony, just so I understand, is that you think that the herbicide, there's more missing data for the glyphosate than there were for other

pesticides that stayed in the analysis?
MS. FORGIE: Object to the form.
THE WITNESS: That's not what I said. I said that it's not exactly referring to the same data or missing data because, by definition, they have to be different.
BY MR. LASKER:
Q. Okay. But the fact that there is missing data noted in the 2013 paper is not something that will prevent that paper from being published in a peer-reviewed literature; correct?
MS. FORGIE: Object to the form.
THE WITNESS: It depends on what missing data does, and obviously here nobody in the peer review community thought that it was an issue.
BY MR. LASKER:
Q. Okay. You also state in your expert report on page 8, you talk about page 19 in the March 15, 2013, draft, and if you can go to that --
A. Well, we --
Q. I'm sorry. In your rebuttal report

on page 8 as another concern that you raise about the unpublished 2013 paper, you point to a comment that appears on page 19 about -- in the section that starts "although this is a large prospective study, there are limitations," and then there is a reference in the 2013 draft that you talk about need to add a paragraph of exposure assessment, discuss the information on our exposure scale in relation to the monitoring work, discuss the likely magnitude of misclassification and its likely impact on the estimates of relative risk"; correct. 06:00

A. Correct.

Q. And you mention this as another indication of why the 2013 analysis was not something that would have withstood peer review; correct? 06:00

MS. FORGIE: Objection.

THE WITNESS: This I cite because I'm asked to review glyphosate. 06:00

BY MR. LASKER:

Q. Okay. You stated that in the next paragraph for the above-stated reasons including the fact that there's this 06:00

question raised in the draft if you would have pointed out the above-mentioned problems -- and let me make sure, let me see if this is one of them. This data I had gotten closer to publication. So let me first ask this. The comment that you're pointing out in the March 15, 2013, draft following "although this is a large prospective study," is that a comment that in your mind will lead you to conclude that this study should not be published in peer-reviewed literature, specifically that comment? 06:01

MS. FORGIE: Object to the form. Asked and answered. 06:01

You can answer it again.

THE WITNESS: This statement was specific to glyphosate, not to anything that's published.

BY MR. LASKER: 06:01

Q. The comment in the draft that you're referring to is not discussing glyphosate; correct?

MS. FORGIE: Object to the form.

THE WITNESS: The comment is 06:01

probably more general, but my idea is that they took glyphosate out because that was the one that had most of the problems.

BY MR. LASKER: 06:02

Q. And if you can look at the 2014 paper again, and you can go to the very end of the paper on page 15 above the section -- above the conclusion, do you see where conclusion is in the same column? 06:02

A. Yeah, uh-huh.

Q. The paragraph above that which starts, "Although this is a large prospective study."

Do you see that? 06:02

A. Yes.

Q. And that is the same language that appeared in the draft in 2013, the same start of that paragraph; correct?

MS. FORGIE: Object to the form. 06:02

THE WITNESS: What was the question?

BY MR. LASKER:

Q. The phrase that starts in the peer-reviewed published study, "Although 06:02

this is a large prospective study" is the same statement that appears in the draft at page 19 where you are mentioning this concern that was being raised -- this comment that was raised in the draft document; correct? 06:02

MS. FORGIE: Object to the form. Misstates the draft.

THE WITNESS: There are two things conflated: One is the statement that was commented on, and the other is the comment. 06:03

BY MR. LASKER:

Q. The comment that you note that appears in the draft of potential limitation in the 2013 study, that is, in fact, discussed in the peer-reviewed published study in 2014; correct? 06:03

MR. BAUM: Object to the form. Again, mischaracterizes the draft. 06:03

THE WITNESS: So, again, the statement I pulled out, I'm referencing this early -- this sentence that starts on this paragraph in order to tell you which comment I'm referring to. The 06:03

comment I'm referring to states, "Need to add a paragraph of exposure assessment, discuss the information on exposure scale in relation to monitoring work, discuss the likely magnitude of misclassification and its likely impact on the estimates of RR." None of that could be done in this publication because they're not publishing on glyphosate. 06:04

BY MR. LASKER:

Q. But the comment that they're saying -- the note they're saying about what needs to be added to the manuscript was, in fact, added to the manuscript as it was published in 2014; correct? That's what the rest of that paragraph does. It responds exactly to that comment. 06:04

MS. FORGIE: Object to the form.

THE WITNESS: I have -- 06:04

MS. FORGIE: Wait. Also asked and answered.

You may answer it again.

THE WITNESS: I can't read it this fast. I would have to read the whole 06:04

paragraph, plus what this statement or this comment requests inserts in the message section, and I haven't reviewed the message section.

BY MR. LASKER: 06:04

Q. In making this criticism in your expert rebuttal report of the 2013 draft, am I correct that you did not compare this comment with what was actually included in the 2014 peer-reviewed published study? 06:04

MS. FORGIE: Object to the form.

THE WITNESS: I would not need to do that because the peer-reviewed study does not address glyphosate, and it is with glyphosate that I have this problem and not with these other pesticides. 06:04

BY MR. LASKER:

Q. Okay. The -- I want to make sure I talked about it. I think there's one study that I did not talk about. I don't think I'm going to have time to go through it in detail, but there was a case control study in France by Dr. Orsi, and that I know you have certain concerns about that I don't think we'll have time to go through in 06:05

detail. But am I correct that that case control population in France, the investigators reported an odds ratio for glyphosate of 1.0 that was not statistically significant? 06:05

MS. FORGIE: Object to the form.

THE WITNESS: They are reporting that for NHL. They also had other outcomes for which the odds ratios were slightly different including multiple myeloma and some sub groups. 06:05

BY MR. LASKER:

Q. But for NHL in the French case control study, they reported an odds ratio of 1.0; is that correct? 06:05

A. With a wide confidence interval and very few exposed subjects.

Q. Okay. And then for the NAPP data which would be the pooled data of all the case control studies in Canada and the U.S. for their ever/never analysis when they adjusted for three pesticides, they reported an odds ratio for glyphosate and non-Hodgkin's lymphoma of 1.13 or for self-respondents only an odds ratio of 0.95; 06:06

correct?

MS. FORGIE: Object to the form.

THE WITNESS: I remember that table, and my problem was that self -- was excluding the proxies is that you're actually excluding the sickest individuals who died before they could be interviewed. So the difference between the two estimates might be that you're actually throwing out the people who are the sickest. 06:06

BY MR. LASKER:

Q. Just so I understand for the NAPP data for pooling together all the case control studies in U.S. and Canada control adjusted for those three other pesticides, the odds ratios and the two ways that they reported it were either 1.13 or 0.95; correct? 06:07

MS. FORGIE: Object to the form. Asked and answered. 06:07

You can answer it again.

A. Those are reported for models that included three pesticides that I am questioning whether or not they should be 06:07

included, and the model that didn't include these pesticides was 1.43 and also for a subgroup analysis with intensity of exposures more than two days per year it actually didn't change at all. 06:07

Q. I understand that you have --

MS. FORGIE: Let me ask a question. How much time do we have left, please?

THE VIDEOGRAPHER: 11 minutes.

MS. FORGIE: Okay, so you'll let us know when seven hours is up, please. 06:07

BY MR. LASKER:

Q. For the De Roos 2005 cohort study, they reported a never/ever use risk ratio adjusted for other exposures of 1.1; correct? 06:07

A. Yes.

Q. And in the 2013 AHS data the never/ever odds ratio, you said, would be somewhere around 1.0. Dr. Blair said it would be around 0.9; correct? 06:08

MS. FORGIE: Objection. Mischaracterizes her testimony.

THE WITNESS: So I would not rely on De Roos, and I would not rely on the 06:08

new data unless somebody can show me that the exposure assessment for glyphosate was not severely misclassified.

BY MR. LASKER: 06:08

Q. I understand that. But the odds ratio reported in that data, and I understand you have reasons why you don't want to rely upon that was, according to Dr. Blair, around 0.9 and you agree it's somewhere around 1.10; correct? 06:08

MS. FORGIE: Object to the form. Also asked and answered.

You can answer it again.

THE WITNESS: That was my answer. I don't think I have to repeat myself. 06:08

BY MR. LASKER:

Q. And for the Swedish study for Eriksson in the multi-regressional analysis, they had an odds ratio of glyphosate non-Hodgkin's lymphoma of 1.5; correct? 06:09

MS. FORGIE: Object to the form.

THE WITNESS: It was about 1.5 in a multi-variated adjusted, yes. 1.53, yes. 06:09

BY MR. LASKER:

Q. We discussed now there was -- the Cocco study very small. The Hardell study was very small. But the four largest study populations then would be that French study, the NAPP study, the Eriksson study, and the De Roos or the AHS cohort. Those are the four largest datasets; correct? 06:09

MS. FORGIE: Object to the form.

THE WITNESS: Orsi is the wrong one to mention. I don't think that Orsi is one we should be looking because the power was very low and it's a case control study that's hospital-based. There are lots of problems with hospital-based controls. 06:09

BY MR. LASKER:

Q. Okay. You would -- and I know you don't agree with -- you have concerns about all of those numbers. But for all of these adjusted odds ratios you have as they're reported by the investigators, you have odds ratios that are bordering around 1.0 when adjusted for other exposures to pesticides; correct? 06:09 06:10

MS. FORGIE: Object to the form. Mischaracterizes the testimony -- the studies.

THE WITNESS: That's not correct. We would have to go study by study. For example, 1.35 is not hovering around 1. 06:10

BY MR. LASKER:

Q. 1.13, 1.0, 1.1 --

A. There was a 2 --

MS. FORGIE: Wait, wait. There's no question. 06:10

BY MR. LASKER:

Q. For ever/never use; correct?

MS. FORGIE: Object to the form. Asked and answered. 06:10

THE WITNESS: Can we go back to De Roos 2003 and check that?

BY MR. LASKER:

Q. Let's -- well, the NAPP includes -- pools all the data that's in De Roos and in McDuffie; correct? 06:10

A. Well, you asked me about all these substudies before.

Q. In your expert report you discuss biological plausibility; correct? 06:10

A. Yes.
Q. And you discuss in there data points for some studies on genotoxicity and oxidative stress; correct?
A. Where's that? 06:11
Q. It's the last page of your expert report, I believe.
A. It's the regular expert?
Q. Yes.
A. The first one. 06:11
MR. WISNER: Second to last page?
MR. LASKER: Yes.
THE WITNESS: Yes.
MR. WISNER: Page 24.
BY MR. LASKER: 06:11
Q. First of all, let me ask you, and I don't know if you've read Dr. Portier's deposition. He goes through the genotox studies in some detail. Dr. Portier testified that in his review of all of the 06:11 glyphosate studies, he did not find evidence from those studies showing that glyphosate is mutagenic. Do you agree with his assessment?
MS. FORGIE: Object to the form, 06:11

and I believe that mischaracterizes the deposition testimony, but you can show her a portion from that.
THE WITNESS: Do you want to show me? 06:11
BY MR. LASKER:
Q. No.
MS. FORGIE: Object to the form.
THE WITNESS: Then I can't comment.
BY MR. LASKER: 06:11
Q. Do you have an independent opinion as to whether or not the glyphosate mutagenicity studies present evidence that glyphosate or glyphosate-based formulations is mutagenic? 06:12
MS. FORGIE: Object to the form.
THE WITNESS: It has never been a point of discussion. It's genotoxicity, not mutagenicity.
BY MR. LASKER: 06:12
Q. So sitting here today, do you have any opinion one way or the other as to whether or not glyphosate is mutagenic?
MS. FORGIE: Object to the form. Asked and answered. 06:12

You can answer it again.
THE WITNESS: It's beside the point because the topic here is genotoxicity and oxidative stress and not mutagenicity. 06:12
BY MR. LASKER:
Q. Do you have an opinion as to whether glyphosate is mutagenic?
MS. FORGIE: Objection. Asked and answered. 06:12
You can answer it again.
THE WITNESS: Mutagenicity is affect in bacteria. Genotoxicity we can assess in human cells and animals, and I believe that the studies that looked at 06:12 genotoxicity showed that there is genotoxicity as I report.
BY MR. LASKER:
Q. Do you have any opinion one way or the other as to whether or not glyphosate is 06:12 mutagenic? Yes or no.
MS. FORGIE: Objection. She doesn't need to give yes or no. You're badgering the witness. You've asked her three times now. 06:13

You can answer it again.
A. I was not evaluating mutagenicity here. I was evaluating genotoxicity, and my statement is about genotoxicity, not mutagenicity. 06:13
Q. Okay. And last document I'll show you -- and we'll have a statement for the record -- is the 2017 slide deck.
MR. LASKER: Has been marked as an exhibit? 06:13
MS. SHIMADO: Yes.
MR. LASKER: This will be my last question. I have a question on one of the slides in there.
MR. WISNER: Exhibit 5. 06:13
MR. LASKER: Yeah, 19-5.
THE WITNESS: My slide deck?
BY MR. LASKER:
Q. Yeah, it's this one.
A. Got it. 06:13
Q. And slide 16 in your slide deck --
MS. FORGIE: You mean page 16?
MR. LASKER: Page 16, slide 16. The number 16 on the slide.
THE WITNESS: Oh, yeah, the Ames 06:14

test.
BY MR. LASKER:
Q. Right.
So you present data here on the Ames test for assessing carcinogens, and you report data that for truly carcinogenic compounds and truly non-carcinogenic compounds and positive and negative on the Ames test; correct? 06:14
A. That's correct. 06:14
Q. My question is: The data in this table, is that data that you made up, or is that data --
A. Not even my data. It's actually Dr. Olson who loves to make these up. 06:14
Q. So this is all made-up data?
A. Yes.
MR. LASKER: Okay. Let's take a break. I've got about four minutes left. I'm going to see if I've got any questions after that point, and I've got a comment for the record. 06:14
THE VIDEOGRAPHER: We're off the record at 6:14 p.m.
(Recess taken from 6:14 p.m. to 06:14

6:32 p.m.)
THE VIDEOGRAPHER: We are back on the record at 6:32 p.m.
BY MR. LASKER:
Q. Dr. Ritz, in your opinion, can scientific studies looking at the issues of genotoxicity and oxidative stress standing alone provide evidence that can establish that a compound causes cancer in humans? 06:32
MS. FORGIE: Object to the form. 06:32
THE WITNESS: These are two criteria that are used by IARC to establish carcinogenicity, but they are just two criteria within the animal study -- within the mechanistic study section. There are several others. 06:32
BY MR. LASKER:
Q. And you would agree that genotoxicity and oxidative stress studies by themselves would not be sufficient for you to be comfortable reaching an opinion of carcinogenicity; correct? 06:32
MS. FORGIE: Object to the form.
THE WITNESS: I cannot subtract from what I know about animal studies, 06:32

mechanism, and human studies, and I would never start with a genotoxicity study. Because I'm an epidemiologist, I always start with human data.
MR. LASKER: I want to make a statement for the record, and then I'll suspend my questioning. There's a couple of issues here. 06:33
One is I mentioned earlier on the record, Dr. Ritz earlier in the deposition suggested, and I don't know whether she does or she does not, that she might have opinions regarding the animal cancer bioassays. 06:33
I have reviewed her expert reports multiple times. I don't see any mention of animal cancer bioassays. To the extent that plaintiff's counsel -- and we don't have to discuss this now -- but if there's going to be the position of plaintiffs that they're reserving the right for Dr. Ritz to offer opinion testimony regarding animal cancer bioassays, we'll move to strike all that testimony. 06:33 06:33

MS. FORGIE: I'm not going to respond to that. I believe her expert report speaks for itself.
MR. LASKER: You just responded.
MS. FORGIE: That's not a response. Just a statement. 06:34
MR. LASKER: Second, we marked a number of points in the transcript where the witness would not respond to a simple yes-or-no question and kept going into soliloquies on issues that were not part of the question. We marked that in the transcript numerous times. 06:34
By doing so, the witness, I think, intentionally was eating into our questioning time. As a result of that, we have not had sufficient time to explore Dr. Ritz's opinions both on the studies that we actually at least mentioned or discussed somewhat in passing or in connection with some of the studies, some of the smaller studies like Hardell and Cocco and also the Orsi study where we did not have time to ask questions pretty much at all, and also 06:34 06:34

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the numerous issues dealing with the Eriksson study in particular and the other studies where because of the witness' refusal to answer questions, we did not have time to go through all those questions.

I will raise an option for plaintiff's counsel that if plaintiff's counsel is agreeing to further questioning at this time for us to ask those questions, we are prepared to stay longer to do so.

If plaintiff's counsel is not prepared to provide us the time necessary to ask those questions and get Dr. Ritz's opinions, we reserve our right, and I'm only going to be suspending my questioning at this point in time to go back to the Court to get additional time because significant portions of time, in our opinion, were taken up because the witness would not answer a simple yes-or-no question, and we've marked those in the record, and the Court can reach its own conclusions 06:35

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about them.

MS. FORGIE: And for the record, how much time is left of his seven hours, or has he used it all? He's out. Could I just have a statement on the record that he's out? 06:35

THE VIDEOGRAPHER: Yeah. He's at seven hours.

MS. FORGIE: Okay. Of course, we don't agree at all with your characterization. In fact, there were multiple times, I would guess hundreds of times where you asked the same question over and over and over again, and that's what ate up into your time. 06:36

I wrote down at least three times where you asked the same question ten times.

Simply because you don't like the answer doesn't give you the right to ask the same question over and over again. That's what ate up your time, and I'm not going to agree to any further time. You can do whatever you want. 06:36

That's outrageous.

MR. LASKER: As I said, the Court 06:36

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will be able to look at the transcript. The witness didn't answer the questions; so of course, I had to ask them again.

MR. WISNER: Just for the record, a large portion of the time during this deposition was eaten up by yourself commenting on the proprietary or responsiveness of the witness' answer, which, quite frankly, is both argumentative, a waste of the testimony because it would never be admissible in court, and a large portion of your commentary was also eaten up. 06:36

So I think at this point -- how much time are you saying you want? Just curious. What's the amount of time you're asking for? 06:36

MR. LASKER: I probably need another two hours or so.

MR. WISNER: Okay. 06:37

MS. FORGIE: All right. So I have a few questions.

MR. LASKER: And further commentary, I'm going to respond to. It's in the transcript. The Court will 06:37

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be able to read that, and the Court will be able to decide whether or not the witness was responsive to questions.

MS. FORGIE: The court certainly will. 06:37

MR. LASKER: Also one more thing I want on the record as well. There was objections to virtually every question, other than what is your name, by plaintiff's counsel which also ate into the time. 06:37

MS. FORGIE: And I'll respond to that. You make incredibly compound, complex questions which are objectionable. I have to object to questions as to form if I want to preserve them, which I do, and you make these declaratory statements beforehand about all kinds of things; so that's why I had to object, and the Court can look at that as well. 06:37

Okay. I have a few questions, Doctor.

EXAMINATION 06:37

BY MS. FORGIE:
Q. Dr. Ritz, can you explain how you went about arriving at your opinions as expressed in your report?
A. Yes. When I'm asked to write a report of a review paper, I use standard methods common to epidemiology which is I go to PubMed, and I put in search terms, multiple search terms to find the biggest amount of literature that I can on PubMed. 06:38
However, I know that certain search terms don't work as well on PubMed; so we also go to Google Scholar which usually gives you a larger number of papers, and a lot of those then have to be weeded out 06:38 because they're not relevant for the question, but it at least allows you to check the literature very thoroughly. So it's a lot of work, but you, you know, go through it. 06:38
Then in addition, you're going to the published literature that is meta-analyses, pooled analyses to cross-reference and make sure you haven't missed anything that's mentioned in one of 06:39

the meta-analyses pooled analyses. You also go to the original literature and check all the references they have because normally every paper refers to papers in this same area prior -- that was published prior. So 06:39 you do that to make sure that you have all the information that you need.
In addition, I, of course, read not only the meta-analyses, the pooled analyses but also previous reports. I also read all 06:39 of the different meta-analyses that kind of keep repeating information about the singular studies. I read the singular studies. I read the IARC report, and I read the EPA CARC report, and all of it together 06:39 I used for my opinion.
Q. And you mentioned that you read the CARC report. How did you decide how much weight, for example, to give the CARC report? 06:39
A. The CARC report was not weighted very heavily because the epidemiology section was rather cursory, and the animal section, that one I actually studied more intensively, seemed to make a lot of use of 06:40

criteria that were contradictory in terms of which studies they were throwing out or throwing in, but it stimulated me to go back to some of the original studies they are citing, but overall, it did not make a big 06:40 impact on my assessment.
Q. You mentioned you reviewed the IARC monograph; is that correct?
A. That's correct.
Q. Did you rely on the IARC monograph, 06:40 or did you form your own opinions?
A. I formed my own opinion. It is very interesting to read the IARC monograph because it summarizes information in an interesting way. However -- and I use it to 06:40 cross check, and I use it to understand their argumentation.
It was published in 2015. There is additional data that came out since.
Q. Are you familiar with something 06:40 known as the Bradford Hill analysis?
A. Of course, yes. We teach that.
Q. Can you explain briefly what it is?
A. Well, Dr. Bradford Hill in the early 1960s, wrote a commentary in which he 06:41

describes viewpoints, he calls them, according to which one can review the scientific literature. It's not just epidemiology. It's all of science more or less. 06:41
Although he meant it for observational studies in order to help us gauge how the data is performing, how the studies are performing in terms of causal assessments because, as you may have 06:41 gathered while I was talking today, there is more to data than just, you know, numbers. We have to put these data into context, and that's what his viewpoints do. They put these data into context of validity, 06:41 biologic plausibility, et cetera.
Q. And with regard to glyphosate-based formulations and non-Hodgkin's lymphoma, did you perform a Bradford Hill analysis?
A. I did, and I talked about it in my 06:42 report.
Q. And what conclusion did you reach after performing your Bradford Hill analysis?
A. After that, I concluded that there 06:42

is reasonable scientific certainty that NHL is associated with glyphosate use in these data.

Q. And did you also -- are you aware as to whether or not IARC also performed a Bradford Hill analysis? 06:42

A. I would presume they did. Actually, they are talking about it; so I think they did.

Q. Okay. And what is your understanding of the conclusion that the IARC reached with regard to their Bradford Hill analysis? 06:42

A. Well, they used their Bradford Hill analysis in the way I just described to put the different pieces together. First, they might have done it work group for work group, but they also do this as a whole group in which they are putting together the human data, the animal data, the mechanistic data and put that in context of these criteria that Bradford Hill suggested. 06:42

Q. Is there a difference between hazard assessment and risk assessment?

A. Absolutely. 06:43

Q. What is the difference?

A. So a hazardous assessment is an assessment in which we are categorizing an agent according to its ability to be toxic including being carcinogenic, but you can also assess reproductive toxicity or other types of toxicity. 06:43

While a risk assessment is something that regulatory agencies use in order to come up with standard setting methods. 06:43

Q. So would it be accurate --

THE VIDEOGRAPHER: I'm going to have to change tapes.

This marks the end of videotape number 4 in the deposition of Dr. Beate Ritz. We're off the record at 6:43 p.m. 06:43

(Recess taken from 6:43 p.m. to 6:45 p.m.)

THE VIDEOGRAPHER: We are back on the record at 6:45 p.m. This marks the beginning of videotape number 5 in the deposition of Dr. Beate Ritz. 06:45

BY MS. FORGIE:

Q. Doctor, we are discussing the 06:46

difference between -- we were discussing what a hazardous assessment is.

Do you recall that before we changed tapes?

A. Yes, I do. 06:46

Q. Would it be fair to say that a hazardous assessment gives you an idea, in general, as to whether or not a particular product is capable of causing a disease?

MR. LASKER: Object to the form. 06:46

THE WITNESS: A hazard assessment is a general evaluation of an agent's potential to be toxic in different ways.

BY MS. FORGIE:

Q. And in this case, would it be accurate to say that a hazard assessment determines whether or not glyphosate is capable of causing non-Hodgkin's lymphoma? 06:46

MR. LASKER: Objection to form.

THE WITNESS: So, in fact, this what IARC is performing is a hazardous assessment. They are making a categorical -- they're taking a categorical approach with a conclusion of carcinogenicity. 06:46 06:47

BY MS. FORGIE:

Q. And did you read the deposition of Dr. John Acquavella in this case?

A. Yes, I did.

Q. From reading that deposition, is it your understanding that Dr. Acquavella is an epidemiologist? 06:47

A. Yes.

Q. Is it also your understanding that Dr. Acquavella was a -- is a former employee of Monsanto? 06:47

A. Yes.

Q. And is it also your understanding that he is a -- that Dr. Acquavella is a current consultant to Monsanto? 06:47

MR. LASKER: Objection to form.

THE WITNESS: I read that in the deposition, I believe, and I met him while he was an employee of Monsanto at some of these meetings. 06:47

BY MS. FORGIE:

Q. Do you recall reading what Dr. Acquavella said about IARC's hazard assessment?

A. Yes. I understood his testimony as 06:47

stating that IARC got the hazard assessment right but that there are questions about the risk assessment.

MR. LASKER: Objection to form.

BY MS. FORGIE: 06:47

Q. So Dr. Acquavella's testimony was that IARC got it right in that in categorizing glyphosate as 2A; is that correct?

MR. LASKER: Objection to form. 06:48

Mischaracterizes the testimony.

THE WITNESS: I did understand from reading his testimony that he actually referred to a correct hazard assessment, and if he meant correct, then he would 06:48 have included the assessment of carcinogenicity in terms of a 2A.

BY MS. FORGIE:

Q. And likewise, it would be correct that in agreeing with IARC's hazard 06:48 assessment, he would have agreed that glyphosate is capable of causing non-Hodgkin's lymphoma; is that correct?

MR. LASKER: Object to the form.

Mischaracterizes testimony. 06:48

THE WITNESS: So since IARC based its evaluation on NHL and quotes a positive association with NHL, I assume that that was what he meant.

BY MS. FORGIE: 06:48

Q. Can you look at Exhibit 16, please.

MR. LASKER: Which one is that?

MS. FORGIE: It's the Brazil slide show, slide deck, PowerPoint, whatever you want to call it. 06:49

THE WITNESS: Yeah.

BY MS. FORGIE:

Q. And on that, can you turn to the Section 26, page 26, "Proxy Versus Self-Respondent," please. 06:49

A. Yes.

MR. LASKER: Page 26?

MS. FORGIE: Yes. This one. "Proxy Versus Self-Respondents."

MR. LASKER: Thanks. 06:49

MS. FORGIE: Do you have it?

MR. LASKER: I do.

BY MS. FORGIE:

Q. Okay. Do you see the section where they're talking about frequency of greater 06:49

than two days per year?

Do you see that?

A. Yes.

Q. And what is the odds ratio there for proxy and self-respondents? 06:49

A. So for proxy and self-respondents, meaning for everyone, it's 1.73 with a confidence interval of 1.02 to 2.94.

Q. And is that odds ratio controlled for use of 2,4-D, dicamba, and malathion? 06:50

A. Yes, it is.

Q. And are those the only three pesticides that you're aware of that are associated as risk factors for non-Hodgkin's lymphoma? 06:50

A. I am aware that 2,4-D is a 2B category according to IARC. Malathion is a 2A. I'm not aware that dicamba is categorized.

Q. Okay. And with the 1.73 odds 06:50 ratio, is that statistically significant?

A. It is.

Q. And is the greater than two days of use per year category there more important than the never/ever use category that was 06:50

discussed earlier by the defense counsel?

MR. LASKER: Objection to form.

THE WITNESS: Absolutely. It's much more important to look at higher intensity because oftentimes that is 06:50 where we see effects when we're evaluating carcinogens.

BY MS. FORGIE:

Q. And with regard to the seven -- the category greater seven lifetime days, years, 06:51 number of years times number of days per year.

Do you see that?

A. Yes.

Q. And it looks like the odds ratio 06:51 has actually gone down in that section.

Do you see that?

A. Yes. The odds ratio hovers around the 1.

Q. Can you explain why the odds ratio 06:51 is lower for that category than for the greater than 2 category where the odds ratio is 1.73?

A. Yeah. These are two different -- very different measures. One is the 06:51

intensity, and the other is duration, and the lifetime days is the product of duration and intensity meaning that, in essence, I am watering out any intensity via duration.

I can get the same numbers with a very low intensity over long duration as with a shorter duration and a higher intensity. So that measure really is more closely related to duration than to intensity. 06:51 06:52

Q. And does that explanation -- how does that tie into whether or not this information tells you -- what information this gives you about glyphosate-based formulations causing non-Hodgkin's lymphoma? 06:52

A. So in terms of occupational epidemiology, we are very interested in high level exposures which we often have a much better way in assessing a much more reliable way in assessing and also believe that high intensity exposures are really what we have to worry about, and we have to protect workers from. 06:52

So I would think that the high intensity more than two days per year is 06:52

what really is an interesting finding in terms of worker health.

Q. And one last question. You see there's two categories here, proxy and self-respondents category A and self-respondents only category B. 06:52

Do you see that?

A. Yes, I see that.

Q. Do you see that under greater than two days of use per year, while the odds ratio goes up from 1.73 for proxy and self-respondents to 1.77 for self-respondents only, it is not statistically significant for self-respondents only. 06:53 06:53

Do you see that?

A. Yes, I see that.

Q. Is there any way to -- what happens when you take out the proxy group?

A. You are pretty much reducing sample size, and when you reduce sample size, you automatically lose statistical power to show a statistically significant effect. So that's what happens here. 06:53

Q. With regard to if you remove 06:53

proxies from the category, is there any reason you would want to include proxies?

A. Well, the one reason I can think of is that proxies are responding because the self-respondent isn't available which means the self-respondent would be too sick to answer or dead. 06:53

So what you're doing is you're pretty much removing the sickest individuals if you're removing the proxy respondents. 06:54

Q. Okay. And then can you turn -- oh, a couple more questions about the NAPP study.

You were shown Exhibit 16. Do you see at the bottom where it says, on the front page, it says Sao Paulo Brazil? 06:54

A. Yes.

Q. Okay. So is it your understanding this is a PowerPoint presentation that accompanied the Brazil presentation? 06:54

A. That's what I understand.

Q. Were you also made -- or have you also seen slide shows with regard to a presentation in Canada?

A. Yes, I was shown that. 06:54

Q. And you also have seen abstracts and posters with regard to a Canadian presentation?

A. Yes.

Q. Have you also seen a slide show, abstracts, or posters related to an IARC presentation? 06:54

A. To the IARC presentation, yes.

Q. And did any of the information -- with regard to your expert report, you, I believe, testified that you only used the Brazil abstract when you were drafting your expert report; is that correct? 06:54

A. That's correct.

Q. So with regard to all of the other materials related to the NAPP study, all these other slide shows, other abstracts, other posters, did any of them affect or change your opinion as stated in your expert report? 06:55 06:55

A. The only way it changed my opinion is that it solidified the opinion that there is, in fact, carcinogenicity to go after.

Q. In assessing the risk of cancer in glyphosate, is there any potential bias in 06:55

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controlling for concurrent pesticide use?
A. Yes. It's always a problem with concurrent exposures. We haven't really found a mathematical way to get around it. There is probably none to get around it. 06:55
If exposures are highly correlated, you have to sit down and ask the question is it more or less likely that these exposures are independent risk factors or indicators or proxies for the actual exposure under 06:56
investigation?
So when you're putting these in the same statistical model, then something occurs that we call co-linearity, and what that means is that there's some technical 06:56
term. These variables split the variants or the explained variants. And in essence, if you put enough highly correlated variables into the same model, none of them will explain anything anymore. All of them will 06:56
go towards the one.
I've seen that multiple, multiple times in air pollution studies where the air pollutants are highly correlated, and this is what you see. Therefore, you are going 06:56

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one by one by one in order to assess their affect on household counts.
Q. Doctor, you were asked many questions about your criticisms of the draft manuscripts of unpublished AHS data. 06:56
Do you recall those questions?
A. Yes.
Q. You've made several criticisms of the draft manuscripts and the unpublished glyphosate data with regard to the AHS 06:57
study; is that correct?
A. That's correct.
Q. With regard to those criticisms of the AHS study, have you ever publicly made those criticisms prior to being retained in 06:57
this litigation?
A. Yes.
Q. And in what format is that?
A. Well, in my teaching. When I teach my students about the cohort design, I warn 06:57
them against the limitations of cohorts because I think I pointed out that this validity slide in the beginning of one of my slide shows is there to actually cause discussion with my students about these 06:57

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blanket validity statements in terms of studies.
So I'm using the AHS study and the loss to follow up as a good example of what to be careful of when you're conducting a 06:57
cohort study.
Q. And, Doctor, I'd like you to turn to Exhibit 17, please.
A. Yes.
Q. And, Doctor, do you see a date on 06:58
this slide presentation?
A. Yeah. That was on my old slides from fall 2012.
Q. So this was approximately four years before you were retained as an expert 06:58
in this litigation; is that correct?
A. That's correct.
Q. And, Doctor, in Exhibit 17, these slide presentations that you use in your teaching at UCLA, do you have criticisms of 06:58
the AHS study incorporated in there?
A. I believe so.
Q. Can you point those out, please?
A. So what I'm doing here is introducing the AHS cohort to the students 06:58

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talking about retro and prospective data collection and what the problems are, and then I'm showing them the composition of the cohort and data collection progress in different phases and specifically on page 6, 06:59
I show them a slide that was given to me during phase 2 data collection in which I point out how many people are actually not completing phase 2 in different parts of phase 2. 06:59
And I'm then directing them to the issue of exposure assessment being incomplete when you have a time varying exposure that you cannot capture at a second time of follow-up. 06:59
Q. So, Doctor, is it accurate to state that approximately four years before being retained as an expert in this litigation, you were teaching -- you were using the AHS problems, exposure assessment problems you 06:59
described with the AHS cohort study as it relates to glyphosate as a teaching tool to your students as to how not to conduct an epidemiological study?
A. Not as not to conduct but what to 06:59

be careful of when you're conducting studies that otherwise seem so perfect.

Q. Doctor, you were asked a lot of questions today, and you were shown a lot of documents. Do any of the documents or questions that you were asked change your opinion as expressed in your expert report that to a reasonable degree of scientific certainty glyphosate causes non-Hodgkin's lymphoma? 07:00

A. I still stand to my conclusions as cited.

Q. And, Doctor, same question, in other words, you were asked a lot of questions and shown a lot of documents today. Do any of them change your opinion to a reasonable degree of scientific certainty glyphosate-based formulations including Roundup cause non-Hodgkin's lymphoma? 07:00

A. Nothing changes my opinion.

MS. FORGIE: That's it.

MR. LASKER: I have one follow-up question. It's not going to take me five seconds. 07:00

MS. FORGIE: I'm not going to allow any time. No more questions. I'm sorry.

MR. WISNER: Let him have one follow-up. 07:01

MS. FORGIE: You guys are a lot nicer than me.

FURTHER EXAMINATION

BY MR. LASKER: 07:01

Q. Dr. Ritz, you provided your slide deck for teaching students in fall of 2012. Do you have any other slide decks of your teaching of your students that mention the AHS study? 07:01

A. Yes. Many. Every year.

Q. Okay. I will for the record object to the fact --

A. It's the same slide deck. It's updated. 07:01

MR. LASKER: I'll ask those slide decks be produced if they refer to the AHS study. Obviously, we understand all slide decks deal with case control studies or cohort studies is over the 07:01

top, but if she has other slide decks that refer to AHS, that seems pretty squarely in line --

MR. WISNER: To the extent they're different than the one you have. 07:01

MS. FORGIE: He just said it's the same.

MR. LASKER: I don't know.

THE WITNESS: It is the same.

MR. LASKER: I don't understand that. I don't know if you've looked at them or not. You can look at them. If they're the exact same slide deck, that's fine. But if they're not the exact same slide deck, we ask they be produced. And you don't have to commit to that. You can look at them. 07:01 07:02

THE WITNESS: Fine.

MS. FORGIE: She said they're the same. I believe her. All right. Done? 07:02

MR. LASKER: I'm sorry. We're off the record.

(Testimony continues on the following page in order to include jurat.) 07:02

THE VIDEOGRAPHER: This concludes today's proceedings in the deposition of Dr. Beate Ritz. The total number of videotapes used today was five, and we're off the record at 7:02 p.m. 07:02

(Time noted: 7:02 p.m.)

_____

Beate Ritz, MD, PhD

Subscribed and sworn to before me this day of , 2017.

_____

(Notary Public)

My Commission expires: _____

C E R T I F I C A T E

STATE OF CALIFORNIA:

I, LISA MOSKOWITZ, CSR, RPR, CRR, CLR, NCRA Realtime Systems Administrator, Certified Shorthand Reporter, do hereby certify:

That the witness whose deposition is hereinbefore set forth was duly sworn, and that such deposition is a true record of the testimony given by such witness.

I further certify that I am not related to any of the parties to this action by blood or marriage, and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 19th day of September, 2017.

_____

LISA MOSKOWITZ, CSR 10816, RPR, CRR, CLR
NCRA Realtime Systems Administrator

NAME OF CASE: In re: Roundup
DATE OF DEPOSITION: September 18, 2017
DEPONENT: BEATE RITZ, MD, PHD

1. To clarify the record.
2. To conform to the facts.
3. To correct transcription error.

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