

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

Before The Honorable Vince Chhabria, Judge

IN RE: ROUNDUP PRODUCTS)
LIABILITY LITIGATION,) NO. M. 16-02741 VC
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San Francisco, California
Wednesday, March 7, 2018

TRANSCRIPT OF PROCEEDINGS

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25 **Also Present: The Honorable Ioana Petrou**
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10:01 a.m.

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

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4 **THE COURT:** Okay. Good morning. A couple quick
5 things.

6 One, I have received an e-mail from some citizen about
7 this case and the issues we're discussing this week, sort of a
8 lengthy e-mail. I have not read it, but I'm going to hand
9 it -- I'm going to let Kristen hand a copy to each side.

10 I have not read it, I'm not planning on reading it; but
11 since somebody tried to communicate with me, I thought I'd give
12 it to you.

13 And then I understand the plaintiffs filed a letter asking
14 for more time. I haven't read the letter, but I have been
15 thinking on my own that it probably would be fair to give the
16 plaintiffs more time. We -- mostly I -- have been interrupting
17 the plaintiffs' experts quite a bit. I anticipate I will have
18 to do that less of the defendants' experts, because I have
19 developing a better understanding of the lay of the land; and
20 the basics of these. And so I think it would be fair to give
21 you some more time.

22 What I'd propose to do now is add 60 minutes to your
23 clock, and with the idea that I -- you asked for 90, right?

24 **MS. WAGSTAFF:** Correct.

25 **THE COURT:** -- with the idea that if you -- you know,

1 if you continue -- you've also been operating in a pretty
2 efficient manner, I think. And so if you -- assuming that
3 continues, and it really is necessary to add another, you know,
4 some a little bit of additional time, I can entertain that, but
5 for now we'll add 60 minutes to your clock.

6 **MS. WAGSTAFF:** Thank you.

7 **THE COURT:** And no, I'm not adding 60 minutes to
8 Monsanto's clock as of now.

9 **MR. LASKER:** That was not my question, actually.

10 **THE COURT:** Okay.

11 **MR. LASKER:** My question's a little about bit
12 different, because we actually have both time on the clock; and
13 actual real time before this hearing is over.

14 And the original chess clock was set up based upon how
15 much time we were going to have in court. If plaintiffs have
16 an extra hour, or what happens at the end is that we have
17 problems getting our witnesses on, even if we have time left,
18 because the time is allocated such that we would end at the end
19 of the day on Friday.

20 **THE COURT:** Yeah, I don't think that will be a
21 problem. I mean, I think even adding another hour to the -- to
22 the plaintiffs' clock, we probably could end easily finish by
23 4:00 o'clock, or before 4:00 o'clock on Friday.

24 But I also would think we may be able to go past -- we'll
25 probably be able to go past 2:00 o'clock tomorrow.

1 I mean, originally we had a hard stop of 2:00 o'clock
2 because we were in the ceremonial courtroom, and there was an
3 induction for a new magistrate judge taking place there, at
4 4:00 o'clock, and so we had to clear out by 2:00.

5 But now that we're here, we don't -- I don't believe we
6 have any reason to have a hard stop at 2:00 o'clock, so that
7 would the way we would probably do it, but I'll get back to you
8 on that.

9 **MR. LASKER:** Thank you, your Honor.

10 **THE COURT:** Sure.

11 **MS. WAGSTAFF:** Okay, and your Honor, plaintiffs are
12 prepared with the witnesses we anticipate being on at
13 4:00 o'clock today to go past 4:00 o'clock, if the Court wants
14 to entertain that, as well.

15 **THE COURT:** Okay, great. So let's -- why don't we go
16 ahead and resume with Dr. Neugut.

17 **MR. MILLER:** Dr. Neugut.

18 **ALFRED I. NEUGUT,**

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

21 **CROSS-EXAMINATION (resumed)**

22 **CROSS-EXAMINATION**

23 **BY MR. LASKER**

24 **Q.** Good morning, Dr. Neugut.

25 **A.** Good morning, Mr. Lasker.

1 Q. I want to focus on issues that were raised by the Court
2 during your testimony earlier today, and see if we can answer
3 some of the Court's questions.

4 I believe you were having conversation with Judge Chhabria
5 about proxy respondents and the potential concern of
6 differential bias, if there were different percentages of
7 proxies among the cases as compared to the controls.

8 Do I have that -- do I understand that correctly?

9 A. Yes.

10 Q. And, in fact, that is what happened in the U.S.-based
11 case-control studies, correct?

12 A. Yes.

13 Q. Okay, if we can put up slide 93, and this is from De Roos
14 2003, which is Defense Exhibit 720.

15 And this is -- there are two columns. One is the overall
16 pooled study, but the second column is the data that was
17 included in the analysis of multiple pesticides; and what we
18 have highlighted here is the fact that there was 40 percent
19 proxy respondents among controls, compared to 31 percent proxy
20 respondents among the cases. Correct?

21 A. Yes.

22 Q. And the concern that you would have, if I understand you
23 correctly, for differential bias, is that if proxies would have
24 less recall of pesticides -- if they just didn't remember,
25 because they were not actually leaving the individuals

1 exposed -- then you would have a lower response rate for a
2 given pesticide for proxies, because of that fact; they just
3 don't know. Correct?

4 **A.** I didn't say lower response for glyphosate, or for
5 herbicides; I said that I would think it would be more
6 erroneous, that there would be -- I would be -- I would have
7 less faith or less confidence in the responses given by -- by
8 proxy respondents.

9 **Q.** I understand. I'm just trying to explore some of the
10 possible biases that coexist.

11 So theoretically, if it were the case that proxies just
12 didn't know, and therefore, did not provide information on
13 pesticides, so that the rate of pesticide usage reported by
14 proxies was lower than the respondents themselves, that would
15 create a potential bias in this situation, correct?

16 **A.** It could.

17 **Q.** And what would happen in that circumstance is that for the
18 controls, the rate of glyphosate usage would be artificially
19 pulled down, right?

20 **A.** I mean, these are things I sit in my office and ponder
21 for -- for a long time, and you're asking me to speculate on
22 the stand and, you know, in a matter of a few moments.

23 It's not an easy question to --

24 **Q.** Well let me --

25 **A.** -- to think through.

1 Q. Let me make it concrete. If proxies were to only remember
2 a pesticide use 5 percent of the time, and the self-respondents
3 would remember it 20 percent of the time, then because you have
4 more proxies than controls, you'll have a lower incidence of
5 pesticide use, because of that proxy bias, correct?

6 A. Theoretically, or possibly. I don't know.

7 Q. Well, mathematically. I mean, that's just a calculation
8 you can make. If you have more proxies than controls, and they
9 are providing you with a lower response rate for pesticide
10 usage, that means you're going to bring down the reported
11 percentage of pesticide use among the controls compared to the
12 cases, correct?

13 A. Again, I don't know that proxies are going to give you a
14 lower rate, and I --

15 Q. I understand that.

16 A. -- erroneous.

17 Q. This is a hypothetical question, I understand that.

18 A. Right.

19 Q. But hypothetically, if the proxies gave a lower number
20 than self-respondents, in this situation, that would create a
21 bias; it would pull the percentage of pesticide use among
22 controls down, and it would result in a bias upward in the odds
23 ratio, correct?

24 A. Ah, what happened to the proxies in the cases?

25 Q. Well, that's the issue with a differential, correct? You

1 have fewer proxies in the cases.

2 So because you have more proxies in the controls, if they
3 have a lower reporting rate, you are going to bias your
4 findings, and the odds ratio will be pushed up, correct?

5 **A.** So theoretically, that would bias the odds ratio up. Is
6 that what you're saying?

7 **Q.** Yes. Is that correct?

8 **A.** If -- if the circumstances that you're describing
9 occurred, that's correct.

10 **Q.** Okay, and, in fact, we know that that is what occurred in
11 this case, don't we?

12 Let's put up slide 97, and this is from plaintiffs'
13 Exhibit 303. Dr. Weisenburger put this data up, or put this
14 study up yesterday; but they pointed to a different part of the
15 study.

16 This was the issue of recall bias, but they had a
17 different calculation in that study that also looked at the
18 issue of proxy or surrogate respondents versus actual
19 respondents, the actual farmers, and what they found and what
20 they reported in that study was that the proxies actually
21 didn't remember this information; and only 1 percent of them
22 identified glyphosate as compared to 13 percent of the actual
23 respondents.

24 So that's exactly the situation we just talked about,
25 given the different response rate in the De Roos 2003 Study,

1 that created a bias that pushed the odds ratio up, correct?

2 **A.** Can I see the paper?

3 **Q.** Sure. It's Plaintiffs' Exhibit 303, and it is at page 59.

4 And there's also -- sorry if your Honors don't have this,
5 but I think we pulled it up.

6 **MR. MILLER:** Excuse me, counsel. May I have a copy?

7 **MR. LASKER:** It's Exhibit 303.

8 **THE WITNESS:** You need to show Table 7 from
9 somewhere. It appears Table 7, which was an entirely different
10 Table 7 --

11 **THE COURT:** Hold on a sec, Dr. Neugut. Let them get
12 straightened out.

13 Can you get a copy to the --

14 **MR. LASKER:** I just handed it to them.

15 **THE COURT:** Great.

16 **BY MR. LASKER**

17 **Q.** We're going to have to wait for this out-of-range to drop
18 out, but the table has a variety of different pesticides, you
19 pull out the glyphosate data, we're pulling it up on the
20 screen, you'll be able to see it as the appears in the paper,
21 and glyphosate is towards the bottom there. There we go.

22 And as we're discussing, the response rate for proxies for
23 glyphosate was 1 percent versus 13 percent for the
24 self-respondents, the farmers. Correct?

25 **A.** Give me a moment. Well, again, I'm working this out,

1 sitting here on a dime, is difficult for me. I mean, this is
2 based on two interviews, in the control group.

3 Q. This is based on all the data presented on respondents
4 from this same case, which is a U.S.-based case-control.

5 This is the data that Dr. Weisenburger presented to
6 explain why there was no recall bias in the study. It's the
7 same paper.

8 A. And the conclusion that you're asking me to draw?

9 Q. Based upon this, and what you just testified about the
10 differential rate of proxies in the De Roos study, because of
11 the fact that the proxies have a much lower response rate for
12 glyphosate, that created a proxy bias that moved the odds ratio
13 in De Roos 2003 for glyphosate and non-Hodgkin's lymphoma
14 upward, correct?

15 A. How many proxies were there in the study?

16 Q. Thirty-nine -- 40 percent versus 31 percent. We just
17 looked at that data.

18 A. Mm-hm.

19 **THE COURT:** And Dr. Neugut, you should feel free to
20 take whatever time you need to review this. This is a study
21 that you are relying on in support of your opinion, and so if
22 you -- to the extent you need to refresh your memory on the
23 details of the study, feel free to do so.

24 And Mr. Lasker, while Dr. Neugut is reviewing it, what --
25 this is -- the document that you are using right now is the

1 actual De Roos 2003?

2 **MR. LASKER:** This is -- no, this is Plaintiff's
3 Exhibit 303. This is an article by Blair that was presented to
4 the Court, and put in to evidence by the plaintiffs on the
5 issue of recall bias.

6 And this was a study that they were explaining showed that
7 there was not recall bias, and they were doing that based on
8 the percentage of respondents both on the case and controls
9 that provided information on pesticide use.

10 **THE COURT:** Okay, and this -- so would this be in
11 Weisenburger's binder?

12 **MR. LASKER:** It should be. It was presented on the
13 second day it came back.

14 **THE COURT:** Oh, I have it. I see it. Yes.

15 **BY MR. LASKER**

16 **Q.** And if it helps you, I believe the page right before the
17 table has a section on surrogate respondents or surrogate
18 interviews; and talks about the fact that I think more than
19 twice of them, as compared to farmers -- that I don't
20 remember -- and said that surrogate farmers in general had less
21 recall, or recalled fewer pesticides, reported fewer
22 pesticides, et cetera.

23 I think -- since you don't have a copy.

24 **A.** Well, I haven't previously read this paper, I don't think,
25 or I don't have a recollection of this particular paper.

1 That's why I'm having more difficulty with it.

2 **THE COURT:** So Dr. Neugut, is the answer basically
3 that you don't know the effect of surrogate responses on the
4 reliability of the De Roos study?

5 **THE WITNESS:** So I'm going to have to pass on that,
6 and I'll have to say that at least -- I don't think it would be
7 fair for me, under these circumstances, to make an assessment
8 just this quickly.

9 **BY MR. LASKER**

10 **Q.** I understand, and we've already discussed the fact --
11 although we did look at this in your deposition, you are aware
12 that in the NAPP, they did an analysis that would have a
13 sensitivity analysis to remove this proxy bias if it exists,
14 and we had prior testimony that that moved the odds ratio from
15 1.13 to .95. We did discuss that during your deposition. Do
16 you recall that?

17 **A.** I don't recall discussing it during the deposition, but
18 again, I've sort of not been discussing the NAPP in general.

19 **Q.** Well, let's move on, then.

20 Dr. Neugut, the -- if we can talk about the 2018 NCI
21 study, and there's been some discussion about the follow-up
22 about how much latency period was available there.

23 You agree that the 2018 study had nearly 40 years of
24 follow-up after the introduction of glyphosate onto the market,
25 correct?

1 A. I'm sorry, I didn't hear what you said.

2 Q. The 2018 *JNCI* study had nearly 40 years of follow-up after
3 the AHS -- of the AHS cohort after glyphosate was introduced to
4 the market, correct?

5 A. Possibly. I don't know for sure, but it had a lot of
6 follow-up.

7 Q. Slide 39. And that was my question, your answer at the
8 deposition. Do you recall that?

9 A. I don't recall it, but if I said it, then I said it.

10 Q. And Judge Petrou asked some questions about how many days
11 of use there were in this study, and there is an analysis in
12 the 2018 *JNCI* study of cumulative days, without any intensity
13 measure. Do you recall that?

14 A. There is an analysis of --

15 Q. The duration, number of cumulative days of exposure of the
16 cohort members in that study.

17 A. I mean, I think that's what the main analyses are based
18 on, aren't they?

19 Q. Well, there are two metrics, but let's just focus on the
20 cumulative days.

21 And if we can, put up slide 94, and this is from Defense
22 Exhibit 544, which is the Andreotti study we've all been
23 looking at.

24 I mean, there's a supplemental table at the back of that
25 study, and it has at the footer at the bottom of the table,

1 quartiles, tertiles and medians, that talk about the number of
2 cumulative days of exposure for the individuals that were
3 placed in the different dose groups.

4 And what this means -- and correct me if I'm -- if I'm
5 wrong -- but am I correct that, for example, the highest
6 quartile of cumulative days exposure in the Andreotti study,
7 those individuals had, on average, over 108 days of exposure to
8 glyphosate?

9 **A.** Yes.

10 **Q.** Now, when we were talking -- when I was talking to
11 Dr. Ritz -- and we can put up slide 98 quickly -- we went
12 through this discussion about hypothetical limitations, and
13 that those should not be sufficient to discount a study
14 findings, and what we really want -- and what we should really
15 demand is data, not opinions.

16 And you agree with that, correct?

17 **A.** I think opinions are sometimes useful, but -- and opinions
18 are -- should be buttressed by data.

19 **Q.** Okay, and while you've raised a number of limitations
20 about the 2018 study, you cannot point to any data that would
21 suggest that if you -- if biases you believe exist did exist,
22 the .85 rate ratio (sic) that's reported in the NCI study would
23 actually be a statistically significant positive association;
24 can you?

25 **A.** .85 was what? I'm sorry if I've having trouble hearing

1 you.

2 **Q.** I'm sorry. The 2018 NCI study, for its overall finding --
3 and we can put up slide 40, because we discussed this in your
4 deposition -- was approximately a 0.85 risk ratio for
5 non-Hodgkin's lymphoma and glyphosate, correct?

6 **A.** Yes.

7 **Q.** And you cannot point to any data that would show that the
8 biases you believe existed did exist; the actual rate ratio
9 (sic) of that study would be statistically significant, above
10 1.

11 And bring up, perhaps, slide 45, if that helps.

12 And I asked you this exact question in your deposition;
13 and you agreed that you could not -- couldn't identify any data
14 to support that opinion, correct?

15 **A.** I mean, in general, when you discuss biases, you're being
16 critical of what's put in front of you. It's rare that one can
17 really have the opportunity to be able to analyze the data and
18 to be able to show that -- that it really has the effect that
19 one suggests.

20 **Q.** And you don't have the data in this case, correct?

21 **A.** No.

22 **Q.** Okay, and you -- you talked about nondifferential
23 misclassification bias, and you talked a little bit about the
24 2005 study, and the questionnaire in that study.

25 In response, you said 90 percent -- or 10 percent error, I

1 think you identified in that first questionnaire?

2 **A.** Mm-hm.

3 **Q.** But you do not believe that the null finding in the
4 dose-response analysis in the 2005 AHS Study was caused by
5 nondifferential misclassification, right?

6 **A.** Hm.

7 **Q.** Let's put slide 46 up.

8 **A.** No, I'm going to be untrue. I would not be certain as to
9 it. I mean, the numbers are small, so it's difficult to know
10 why that was a null finding, but a misclassification of
11 10 percent, again, with a risk ratio of 1.3 or 1.4, could have
12 caused that to be a negative -- a null finding, as well.

13 **Q.** Well, Dr. Neugut, I'm correct that at your deposition,
14 when I asked you this question, you agreed that you do not have
15 a criticism of that finding --

16 (simultaneous colloquy)

17 **A.** Could I see the context of the deposition, and how this
18 was put before and after?

19 **THE COURT:** Two things, Dr. Neugut. One, you
20 absolutely can see the context of the deposition. So if you
21 want to ask them to give you the full deposition transcript,
22 you can. But number two, you can't interrupt.

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

24 **THE COURT:** When he's asking you a question, you have
25 to let him finish his question.

1 **THE WITNESS:** Okay. I'm sorry.

2 **MR. LASKER:** And your Honor, I'm happy to give
3 Dr. Neugut the full transcript.

4 I would ask that if we're going to continue along this
5 way, we get extra time on the defense clock.

6 **THE COURT:** We'll deal with that later.

7 **MR. LASKER:** Okay. Thank you, your Honor.

8 (Discussion off the record.)

9 **THE WITNESS:** That's correct.

10 **MR. LASKER:** Thank you.

11 **Q.** And with respect to the 63 percent of the cohort that
12 responded to the second questionnaire in the 2018 study, you
13 also do not have any concerns about exposure misclassification,
14 correct? We can put that up, if you want. And this is slide
15 47.

16 **A.** You mean, aside from the 10 percent initial
17 misclassification?

18 **Q.** Yeah, aside from the misclassification in the 2005 study
19 we just talked about, you do not have any concerns about
20 exposure misclassification among the 63 percent of the cohort
21 that responded to the second questionnaire on the 2018 study,
22 correct?

23 **A.** Not that I would -- no, we're not -- know whether or not
24 they answered correctly, or how much misclassification error
25 there was on the second questionnaire, as well.

1 Q. But Dr. Neugut, when I asked you this in your deposition,
2 you did agree that, except for that questionnaire that was part
3 of the 2005 study, we just heard your testimony on that, you
4 agreed that you didn't have concern about exposure
5 misclassification with respect to that 63 percent of the cohort
6 in the 2018 study, correct?

7 A. Then I was mistaken then, and I'm correcting my answer
8 now.

9 Q. Okay, and you were aware that in the 2018 study, when they
10 looked at those 63 percent separately and just looked at the
11 association among those individuals, there was no association
12 between glyphosate and non-Hodgkin's lymphoma, correct?

13 A. I'm aware of that, but then again, we're again talking
14 about the same misclassification errors and the same moderate
15 association, and the potential for attenuation towards the
16 null, which we've talked about previously.

17 I mean, in addition, we're talking about a selected --

18 Q. Dr. Neugut, there's no -- the question's been....

19 He's answered the question, your Honor, if I may?

20 **THE COURT:** You can let him finish his response.

21 **THE WITNESS:** So we're talking about an answer in a
22 very selected cohort.

23 **BY MR. LASKER**

24 Q. And Dr. Neugut, outside of this litigation, you were not
25 aware of anyone who's argued in any forum that the use of the

1 imputation methodology makes the findings of the AHS cohort
2 studies unreliable?

3 **THE REPORTER:** Unreliable...?

4 **BY MR. LASKER:**

5 **Q.** Outside of this litigation -- I'm going to put this up,
6 it's slide 57. Outside of this litigation, you were not aware
7 of anyone who's argued in any forum that the use of this
8 imputation methodology makes the findings of these agricultural
9 health cohort studies unreliable; that's correct, right?

10 **A.** I don't understand the question.

11 **Q.** Well, that is what you asked --

12 **A.** Are you talking about in general, or are you talking about
13 with regard to the glyphosate and NHL specifically?

14 **Q.** With respect to glyphosate and NHL specifically, you
15 agreed -- and let me bring up slide 54 -- that glyphosate in
16 the imputation methodology did about as well -- it was sort of
17 in the middle of the pack with respect to all of the different
18 pesticides looked at in the AHS, correct?

19 **A.** Yes.

20 **Q.** And again I'll ask you: And what you've testified in your
21 deposition, you're not aware outside of this litigation of
22 anyone who's argued in any forum that the use of the imputation
23 methodology makes the findings of these AHS studies unreliable.

24 **A.** I don't read the fora where anyone would do it.

25 **Q.** Let's move on, to the Eriksson Study, and you would

1 agree -- and this is Defense Exhibit 877. This is the Swedish
2 study that -- and we talk about this with Dr. Ritz -- that
3 because of the way that they defined "unexposed individuals" as
4 being unexposed to all pesticides, there was a methodological
5 flaw in the Eriksson Study design, correct?

6 **A.** Talking about univariate analysis.

7 **Q.** We're talking about slide 57 for a second. Okay, I'm
8 sorry. I was asking, with respect to the Eriksson Study, you
9 agree that there was a methodological flaw in the study,
10 correct? Because they defined "unexposed" as unexposed to all
11 pesticides, correct?

12 **A.** Which table are you referring to?

13 **Q.** Well, it is the entire study, but I will bring up slide
14 72, and your testimony in response to my question in the
15 deposition,

16 **"QUESTION:** If, in fact the Swedish
17 case-control studies defined "unexposed" so
18 that there was no exposure to any pesticide,
19 and allowed exposures to other pesticides to
20 occur -- that would be a methodological flaw
21 in the study, correct?"

22 **THE REPORTER:** I'm so sorry, Mr. Lasker, I lost you.

23 **"QUESTION:** -- that would be a methodological
24 flaw in the study, correct?

25 And your answer, "Probably, yes."

1 Do you agree with that, Dr. Neugut? Right?

2 A. Yes.

3 Q. And you also agree that this methodological flaw would
4 make it impossible to actually adjust for the potential impact
5 of other confounders, correct?

6 A. So --

7 Q. Slide 73? And I can give you the context, if you want,
8 but it's actually the very next. It's right after the question
9 and answer I just gave you.

10 THE COURT: Mr. Lasker, while he's reviewing the
11 material, let me just make a comment.

12 I think you're pulling up his deposition testimony too
13 quickly.

14 MR. LASKER: Okay.

15 THE COURT: You're asking him a question, and you're
16 giving him half a second to think about the answer, and then
17 you're pulling up his deposition testimony.

18 MR. LASKER: Right.

19 THE COURT: Why don't you let him think about the
20 answer to the question, answer it, and then, if you need to,
21 pull up the deposition testimony.

22 MR. LASKER: I understand, your Honor.

23 THE COURT: Okay.

24 THE WITNESS: So subsequent to our deposition I went
25 back and looked at the paper again; and looked at it again now,

1 and what it says that it excluded control -- it only used
2 controls who were unexposed to herbicides. That was only, as I
3 read the paper, was only in the univariate analyses.

4 **BY MR. LASKER**

5 **Q.** Dr. Neugut, at your deposition, you agreed that given the
6 systematic bias in Eriksson, it was impossible to reach a
7 conclusion with respect to any individual pesticide exposure
8 reported in the study, correct?

9 **A.** I just said that I'm -- at the time I misread the way the
10 study was conducted, and that the exclusion of the herbicides
11 or the exposure to herbicides for controls was only in the
12 univariate analyses.

13 I don't believe that that was the case in the context of
14 the multivariate analyses paragraph.

15 **Q.** And the multivariate analysis is Table 7, where the odds
16 ratio for glyphosate went down to 1.5 and was not statistically
17 significant, correct?

18 **A.** Correct.

19 **Q.** And just, in the final answer to my question, you did
20 testify at your deposition that given the systematic bias in
21 Eriksson, at least as you understood it at that point in time,
22 you believed it was impossible to reach a conclusion with
23 respect --

24 (Simultaneous colloquy.)

25 **THE COURT:** Hold on, you've got to let him finish his

1 question.

2 **BY MR. LASKER:**

3 **Q.** -- with respect -- during your deposition, you testified
4 that given the systematic bias in Eriksson, it was impossible
5 to reach a conclusion with respect to any individual pesticides
6 exposure reported in the Eriksson Study.

7 That was your testimony, correct?

8 **A.** You've got me. That's what I said at the deposition.

9 Thank you.

10 But as I say now, at the time I misread the paper, and
11 understood that the -- when they said in the -- it's a poorly
12 written method section, and when they excluded the herbicide
13 exposures from the controls, they were referring to the
14 univariate analyses, not to the multivariate analyses.

15 So if you want to say that my conclusion and my answer to
16 your deposition question is for the univariate analyses, you're
17 correct, but for the multivariate analyses, that's not true.

18 **MR. LASKER:** Okay. Thank you. No further questions.

19 **THE WITNESS:** Thank you.

20 **THE COURT:** Any redirect?

21 **MR. MILLER:** Very brief, your Honor.

22 **REDIRECT EXAMINATION**

23 **BY MR. MILLER**

24 **Q.** Dr. Neugut, I won't be long. I know you have a flight.

25 I wanted to go first to Exhibit 303, which is a 1993

1 article shown to you by the defense counsel, written by
2 Dr. Blair.

3 Now, you did not put this originally in your reliance
4 materials, right?

5 **A.** I'm sorry, what paper are you referring to?

6 **Q.** It's an article. I believe he handed you a copy.
7 "Patterns of pesticides use among farmers, implications for
8 epidemiologic research."

9 Remember when counsel was talking to you about, he thought
10 there was a bias?

11 **A.** Yes, sir, this paper?

12 **Q.** Yes, sir, because of proxy responders versus... Yeah,
13 yeah, I see the problem. Thank you.

14 So I want to show you the conclusion that they drew in
15 this paper that counsel didn't show you -- I've highlighted
16 it -- and ask you about it. Excuse me, I keep moving around.

17 They conclude, "Comparison of reporting by cases -- "

18 **THE COURT:** Sorry, what page are you on?

19 **MR. MILLER:** I am on page 1, Your Honor --

20 **THE COURT:** Sorry?

21 **MR. MILLER:** -- of Exhibit 303, on the top right-hand
22 column.

23 **THE COURT:** Thank you.

24 **BY MR. MILLER**

25 **Q.** (Reading:)

1 "Comparison of reporting by cases and
2 controls provided no evidence of case
3 response (differential bias), thus
4 inaccurate recall of pesticide use by
5 subjects or surrogates would tend to
6 diminish the risk estimates -- "

7 **A.** Yes.

8 **Q.** " -- and dilute exposure response gradients."

9 Has that been your experience --

10 **A.** In general, yes.

11 **Q.** -- with this? So I think counsel was suggesting it raised
12 the risk, but in fact, these authors concluded it reduced the
13 actual risk odds ratio, right, sir?

14 **A.** Yes.

15 **Q.** All right, sir, we'll move on. You've talked about, when
16 we talked about confounding, how malathion and di- -- I think
17 it's diazinon -- I hope I'm pronouncing that right -- are known
18 by the scientific community to be causes also of non-Hodgkin's
19 lymphoma. Is that a fair understanding of what you said?

20 **A.** I don't know if I said, it but they are.

21 **Q.** Well, and I wanted to you ask you, in the AHS study....

22 Excuse me, is it on?

23 **MR. WISNER:** You want it on?

24 **MR. MILLER:** Yes.

25 **Q.** In the AHS Study -- you prepared this PowerPoint, the

1 slide that I forgot to show you yesterday.

2 **A.** Yes.

3 **Q.** In the AHS study, when they studied malathion and diazinon
4 with their database, were they able to find the positive
5 association with non-Hodgkin's lymphoma, that the scientific
6 community now accepts?

7 **A.** So this is a slide which lists several herbicides and
8 pesticides which IARC has reviewed, and listed as either Type 1
9 or Type 2 -- Type 2A carcinogens. So -- and these were all
10 evaluated in the AHS Study.

11 So I put this slide together. It was in my supplemental
12 report to the court.

13 And just to illustrate that, the AHS Study, when it found
14 no association with glyphosate, which IARC had found to be a
15 probable carcinogen, which I would reiterate from yesterday,
16 again, I think the scientific community would consider that to
17 have a probability of 70 to 90 percent or so of being a
18 carcinogen. So it also missed malathion and diazinon. I have
19 no idea how you say it, but diazinon.

20 It missed two other proven, or -- or two other carcinogens
21 which IARC has also defined as being 2A.

22 So -- so the sensitivity of the AHS Study -- or the
23 ability of the AHS Study to identify potential carcinogens, one
24 really has to have some question, some skepticism about how
25 good a study it is for identifying carcinogens from the

1 herbicide group.

2 And I would suspect it's probably because, as well, they
3 also don't have powerful risk ratios, or there were other
4 reasons why it would have missed it. But the point is,
5 glyphosate is not the only carcinogen which it's missed. It
6 also misses others. So the AHS Study has missed others, as
7 well.

8 **Q.** I only have two other questions. One, I just wanted to
9 mark the exhibit.

10 You said that you initially began your scientific inquiry
11 on this issue of glyphosate and non-Hodgkin's lymphoma by
12 reviewing the monologue (sic) prepared by IARC. If we could
13 turn on the overhead, I just want to confirm we have this.
14 This is the right document? You have a copy of the monologue?

15 **MS. WAGSTAFF:** Monograph.

16 **MR. MILLER:** Monograph, monologue, excuse me. I'm
17 sorry.

18 **Q.** This is a 91-page document prepared by IARC. This is what
19 you initially reviewed?

20 **A.** Yes.

21 **MR. MILLER:** Yeah, and we'll move -- we'll talk about
22 admissibility later.

23 **Q.** And finally, counsel talked with you about the issue of
24 whether Andreotti or the AHS follow-up would change IARC's or
25 the community's -- scientific community's opinion of whether

1 glyphosate causes non-Hodgkin's lymphoma, and I just want to go
2 back to Exhibit 149, which we looked at with you yesterday,
3 which are the briefing notes that IARC scientific and
4 Governing Council members received, prepared by the IARC
5 director.

6 And they talk about this issue. If I can, I'll ask you,
7 on page 4, they state, quote,

8 "The lengthy court testimony given by
9 Dr. Blair does not support any change in
10 the classification of glyphosate consequent
11 to the latest AH publication."

12 Now, is that also your opinion, that the latest paper does
13 not change the classification of --

14 **A.** That's precisely what we've been talking about for the
15 last couple of hours in the testimony, both direct and on
16 cross-exam.

17 **MR. MILLER:** Okay, thank you, Doctor. I have no
18 further questions.

19 **THE COURT:** Anything further, Mr. Lasker?

20 **MR. LASKER:** No, your Honor.

21 **THE COURT:** Okay, thank you.

22 **THE WITNESS:** Nothing further? Thank you for having
23 me.

24 **THE COURT:** Thank you for coming. Better check to
25 make sure your flight didn't get canceled.

1 **THE WITNESS:** Thank you.

2 **THE COURT:** Because I guess the storm is really bad
3 on the East Coast.

4 **THE WITNESS:** It's one of the beauties of California.

5 **THE COURT:** Welcome to stick around, if you like.

6 **MR. MILLER:** Thank you, Doctor.

7 **MS. WAGSTAFF:** Your Honor, we have books for the next
8 witness.

9 **THE COURT:** Thank you.

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

11 **THE COURT:** All right, ready for your next witness?

12 **MS. WAGSTAFF:** Yes. Plaintiffs call Dr. Jameson.

13 And while he is walking up to the stand, I would like to take a
14 moment to thank you for joining us, in this proceeding.

15 **JUDGE PETROU:** My pleasure.

16 **MS. WAGSTAFF:** I know you're very busy in Oakland.

17 **JUDGE PETROU:** I will see some of you next week.

18 **THE CLERK:** Please remain standing, and raise your
19 right hand.

20 **CHARLES W. JAMESON,**

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

23 **THE WITNESS:** I do.

24 **THE CLERK:** Thank you. Please be seated. Go ahead
25 and adjust your microphone so that it's directly in front of

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

3 **THE WITNESS:** My names is Charles W. Jameson.
4 C-h-a-r-l-e-s, J-a-m-e-s-o-n, but I go by "Bill."

5 **DIRECT EXAMINATION**

6 **BY MS. WAGSTAFF**

7 **Q.** All right, thank you. Dr. Jameson, this is your first
8 time appearing as an expert witness in litigation, is it not?

9 **A.** That's correct.

10 **Q.** All right, and this is your first time giving testimony in
11 court?

12 **A.** That's correct.

13 **Q.** All right. So first just take a moment and tell the
14 judges a little bit about yourself.

15 **A.** Okay. Good morning, your Honors.

16 **THE COURT:** Good morning.

17 **THE WITNESS:** I'm Bill Jameson. I have 40-plus years
18 of toxicology experience, working first for the National Cancer
19 Institute, which is part of the National Institutes of Health;
20 and then later at the National Institute of Environmental
21 Health Sciences, which is also part of the National Institutes
22 of Health.

23 In my tenure at the National Institutes of Environmental
24 Health Sciences, I served as the Director of the Report on
25 Carcinogens. Report on Carcinogens is a document required by

1 the Public Health Service Act of 1969 that requires that the
2 Secretary of Health and Human Services submit a report to
3 Congress that lists all the chemicals that are either known or
4 reasonably anticipated to be human carcinogens to which a
5 population of United States are exposed, and my responsibility
6 was to prepare the whole report for the Secretary.

7 I've also been a member of 14 IARC Monograph Working
8 Groups. It was also including IARC Monograph 112 Working
9 Group, where glyphosate was discussed, where I served as
10 Chairman of the Experimental Animal Subgroup for that Working
11 Group.

12 **BY MS. WAGSTAFF**

13 **Q.** Okay, excellent. So to summarize, you've been a
14 governmental toxicologist for about four decades, is that
15 right?

16 **A.** Yes, ma'am.

17 **Q.** Okay, and you're since retired, correct?

18 **A.** Yes, I retired in 2008.

19 **Q.** Okay, and as well as a toxicology opinion, you're also
20 giving an opinion on epidemiology, is that correct?

21 **A.** That's correct.

22 **Q.** Okay, and in support of your epidemiological opinion, you
23 have tendered a list of all of your epidemiological
24 qualifications, training, experience, right?

25 **A.** That's correct. I just put together a list of -- of all

1 the -- all my past dealings with evaluating epidemiology data.

2 Q. Okay, and you've been evaluating epidemiological data for
3 four decades, is that right?

4 A. That's correct.

5 MS. WAGSTAFF: And for your Honors, you have a list
6 that he just discussed in your notebook. It's Exhibit 321.
7 And we'll move it in as an exhibit later.

8 If you could move to the next slide, please.

9 Q. And so Mr., er -- Dr. Jameson, can you please discuss your
10 conclusions in this case?

11 A. Okay. As the slide indicates, in my expert report,
12 I concluded to a reasonable degree of scientific certainty that
13 glyphosate and glyphosate-based formulations are probably human
14 carcinogens; and also concluded to a reasonable degree of
15 scientific certainty that glyphosate and glyphosate-based
16 formulation caused non-Hodgkin's lymphoma.

17 In response to some of the questions that your Honors
18 raised over the past couple of days, I also have on this slide
19 my opinion that exposure to glyphosate not only can cause
20 non-Hodgkin's lymphoma, but it is currently doing so, at
21 current exposure levels today.

22 And I also feel --

23 MR. HOLLINGSWORTH: Your Honor, we object to that
24 opinion. It's not in his expert report. We've never discussed
25 that with him.

1 **THE COURT:** Overruled.

2 **THE WITNESS:** And the epidemiologic data demonstrates
3 credible evidence that exposure to glyphosate and
4 glyphosate-based formulations cause non-Hodgkin's lymphoma in
5 humans.

6 **BY MS. WAGSTAFF:**

7 **Q.** Okay, excellent. And when did you form this opinion?

8 **A.** I first formed this opinion as a result of my
9 participation in the IARC Monograph 112 review of glyphosate,
10 in March of 2015.

11 **Q.** Okay, and so that was before or after you began work in
12 this litigation?

13 **A.** Oh, that was at least a year before I was retained as an
14 expert witness.

15 **Q.** Okay, excellent.

16 If you could, turn to the next slide, please.

17 All right, I'd like to spend a few moments talking about
18 your methodology, how you came to reach those conclusions.

19 **A.** Sure.

20 **Q.** Can you please tell the Court about that?

21 **A.** The methodology I used to reach my conclusions is the same
22 scientific method that I used, using the intellectual rigor
23 that I have been using all my professional life when reviewing
24 data, to determine if this material causes cancer in humans.

25 I performed literature searches. When asked to give my

1 opinion, my first step was to do a thorough literature search
2 of all of the publicly peer-reviewed literature on glyphosate
3 as it relates to its carcinogenic potential.

4 I also was provided with reports from the EPA, during my
5 review at the IARC Monograph; and also I was able to get some
6 of the actual laboratory reports of the animal studies that
7 I reviewed, from counsel.

8 I looked at all of the available epidemiology data that
9 had been published on glyphosate and applied the Bradford Hill
10 criteria, which has been discussed previously, in coming to my
11 conclusions.

12 In toxicology, I evaluated all of the available toxicology
13 data I could find, and it showed that glyphosate is an animal
14 carcinogen, and that is the premise that is widely accepted in
15 the toxicology -- the scientific community, that if something
16 is shown to be an animal carcinogen, then it is probably also a
17 human carcinogen; and it's biologically plausible that it is an
18 animal -- that it is a human carcinogen.

19 And then I also looked at the mechanistic data that is
20 available for glyphosate, and glyphosate-based formulations,
21 and this data shows that glyphosate is gene toxic in humans,
22 and also causes oxidative stress in humans, and oxidative --
23 that's an important observation, because oxidative stress has
24 been linked to the formation of non-Hodgkin's lymphoma in
25 humans.

1 Q. Okay, and that's the same methodology you used while you
2 were a government employee, or -- in government toxicology for
3 four decades, correct?

4 A. That's the same methodology I used for when I participated
5 in the IARC Monograph, and it is also the methodology I used
6 while at the National Institute of Environmental Health
7 Sciences National Toxicology Program for the report on
8 carcinogens.

9 Q. Okay, excellent. And if you could turn to the next slide,
10 please.

11 All right, and I know that Judge Chhabria had some
12 questions about the hazard assessment for Dr. Neugut yesterday,
13 and you heard that testimony; did you not?

14 A. Correct.

15 Q. Okay, so let's just spend a couple of minutes.

16 Why don't you let the Court know if you did hazard
17 assessment, and what that means.

18 A. Well, I performed a hazard assessment in reviewing all of
19 the available data on glyphosate and glyphosate formulations,
20 and the basic question when you do a hazard assessment is, can
21 glyphosate cause cancer in real world exposure levels?

22 **MR. HOLLINGSWORTH:** Objection, your Honor. That's
23 outside the confines of his report, that opinion specifically.

24 **THE COURT:** I understand that, and I'll let him
25 testify about it here today, and we can talk about the validity

1 of the opinion at a later time.

2 **MR. HOLLINGSWORTH:** Thank you.

3 **THE WITNESS:** And the answer to the question is yes,
4 glyphosate can cause cancer in real world exposures levels.

5 And another question that -- that came up in some of the
6 discussions over the past couple of days is, does hazard
7 assessment consider chemicals in the abstract?

8 The purpose of doing a hazard assessment to determine if
9 something is a carcinogen is -- is to get data on a chemical,
10 to see if it could potentially be a human carcinogen.

11 The best way to do that is to do an animal bioassay. You
12 use the animals to test the chemical, to see if it can cause
13 cancer in the animals; and if it does cause cancer in the
14 animals, then it's very -- it's biologically plausible that it
15 very likely will cause cancer in humans.

16 So the chemicals are selected for doing these hazard
17 assessments because there is some concern that there's human
18 exposures to these, and trying to determine if there is a
19 cancer hazard associated with that particular exposure.

20 When doing a risk assessment, you take the information
21 from the hazard assessment that it is a carcinogen, and then
22 apply it to individuals to see if the material -- in this case
23 glyphosate -- if it causes -- if it can cause cancer to an
24 individual, at the -- at the dose levels that they're being
25 exposed to.

1 **BY MS. WAGSTAFF**

2 **Q.** Okay, and I'm not quite sure you answered your own
3 question, but does a hazard assessment consider chemicals in
4 the abstract?

5 **A.** No, it's not in the abstract. Most -- at least in my
6 experience in the National Toxicology Program -- I worked in
7 the rodent bio- -- before I became involved with the Report on
8 Carcinogens I was involved in the NTP rodent bioassay program,
9 and I worked for many years in identifying chemicals to study
10 for that.

11 And basically, there we identified chemicals that have
12 some possibility of human exposure; and if it is human exposure
13 to the chemical, and there's nothing known about the cancer of
14 it, then we would want to investigate it to see if it
15 potentially could be a human carcinogen.

16 **Q.** Okay, excellent. And you conducted hazard assessments at
17 IARC, correct?

18 **A.** That's correct.

19 **Q.** And you conducted hazard assessments when you were at the
20 national NTP, correct?

21 **A.** Correct.

22 **Q.** And is it a generally accepted method for determining
23 whether an agent is an animal carcinogen conducting a hazard
24 assessment?

25 **A.** That is absolutely correct. Not only do we do it in

1 the -- did we do it in the National Toxicology Program and does
2 IARC do it, but regulatory agencies, such as the Environmental
3 Protection Agency, the Food and Drug Administration, require
4 animal bioassays. They -- you know, studies to be submitted to
5 them for registration of pesticide, or a drug, or what have
6 you. And so it is the standard for identifying human
7 carcinogen.

8 **THE COURT:** Before we get too deep into the animal
9 bioassays, could I ask a couple of follow-up questions about
10 hazard assessment?

11 **THE WITNESS:** Yes, sir.

12 **THE COURT:** So I'm looking at your expert report.

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

14 **THE COURT:** Page 5. Do you have your expert report
15 in front of you?

16 **THE WITNESS:** No, sir, I don't.

17 **THE COURT:** Do you want to give him a copy of his
18 report?

19 **THE WITNESS:** Okay.

20 **THE COURT:** Okay, so I'm looking at page 5, and
21 the -- the top paragraph, the carryover paragraph --

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

23 **THE COURT:** -- which discusses the difference between
24 hazard and risk.

25 **THE WITNESS:** Okay.

1 **THE COURT:** Hazard assessment and risk assessment.

2 And this is taken -- this language from your report is taken
3 directly from the IARC preamble, right?

4 **THE WITNESS:** That's correct.

5 **THE COURT:** And so you're describing both the
6 assessment that IARC conducted and the assessment that you are
7 conducting in this report, is that correct?

8 **THE WITNESS:** That's correct.

9 **THE COURT:** And what you've -- the assessment that
10 you conduct in your report is co-extensive with the assessment
11 that IARC conducts?

12 **THE WITNESS:** Correct.

13 **THE COURT:** Okay, and it says in that paragraph the
14 distinction between hazard and risk is important, and the
15 monographs identify cancer hazards even when risks are very
16 low, at current exposure levels, because new uses or unforeseen
17 exposures could engender risks that are significantly higher.
18 In other words, hazard assessment determines whether an agent
19 can cause cancer.

20 So when I read that in your report and in the IARC
21 preamble, I took that to mean that the conclusion reached by
22 IARC that something is a probable carcinogen, or even a known
23 carcinogen, kind of doesn't get you all the way to the
24 conclusion that you identified in the -- in that first slide
25 that was shown about your opinions that a -- a carcinogen is

1 currently causing cancer in human beings at the exposure levels
2 they are currently experiencing.

3 Am I misinterpreting the sentence --

4 **THE WITNESS:** Well --

5 **THE COURT:** -- in your report?

6 **THE WITNESS:** -- yes, in a way, you are.

7 The caveat is put in there because for so many -- for a
8 large number of the chemicals that IARC has reviewed -- and
9 this is also true for a number of the chemicals that are listed
10 in the Report on Carcinogens as carcinogens, and this is
11 usually in the category 2A, and 2B -- when a material is
12 identified as an animal carcinogen, and therefore biologically
13 plausible to be a human carcinogen for a large majority of the
14 cases, there is no human epidemiology data to go along with
15 that.

16 Since there is no human epidemiology data available, you
17 can only say that it is either possibly or probably a human
18 carcinogen because animal data points to it, and there may be
19 some strong mechanistic data that was actually conducted in
20 human cells that show that it was potentially -- you know, that
21 it causes gene mutations, and is a -- a mechanism that one
22 would conclude could lead to cancer in humans. So -- but we
23 have no human data.

24 The human data -- the epidemiology data that you review is
25 the data that -- that shows you in the real world what people

1 are really exposed to when they use it as a farmer, or as -- in
2 a factory, or a drug that an individual is taking for a
3 particular disease. The epidemiology data is what shows you
4 that, under real world exposure situations, this is what
5 happens.

6 And so that description is there for those chemicals,
7 basically, for which there is no epidemiology data. Now, when
8 we did do the hazard assessment reviews --

9 **THE COURT:** Could I ask a quick clarification
10 question about that?

11 **THE WITNESS:** Sure.

12 **THE COURT:** And I want you to finish your thought,
13 but just a quick clarification question.

14 My understanding from reading the IARC Monograph,
15 including the preamble, is that when the Working Group
16 classifies something as a probable carcinogen, it's usually
17 when they do have some epidemiological evidence.

18 **THE WITNESS:** That's correct.

19 **THE COURT:** Okay. So when something is classified as
20 a 2B, it might commonly be because we have no meaningful
21 epidemiological evidence.

22 **THE WITNESS:** Correct.

23 **THE COURT:** And when we classify something as a 2A --
24 that is, a probable carcinogen -- it means we have -- there is
25 some amount of epidemiological evidence, but the IARC describes

1 it as "limited" evidence. Is that right?

2 **THE WITNESS:** Well, that could be the case, but there
3 are also instances --

4 **THE COURT:** But isn't that how IARC describes it, and
5 isn't that how you describe it in your report?

6 **THE WITNESS:** Um, well, if --

7 **THE COURT:** In your report, don't you say that there
8 is limited evidence --

9 **THE WITNESS:** Yes, there's limited evidence --

10 **THE COURT:** Okay.

11 **THE WITNESS:** -- and the reason -- the reason why I
12 say there's limited evidence is because I -- I established
13 criteria for evaluating the data, and I describe in there what
14 is meant by -- by "limited" data.

15 And so I'm sticking to my criteria when I say, you know,
16 based on the -- based on the fact that for the epidemiology, an
17 association is very credible, but confounding factors cannot
18 absolutely be explained away. I mean, it's close, but they --
19 they can't absolutely be explained away. So therefore, by the
20 definition of my criteria, that's limited.

21 That's -- that's my mindset, because for the Report on
22 Carcinogens, I actually wrote the criteria for the Report on
23 Carcinogens, and that's the wording that I used, and it's very
24 similar to what is in IARC, which it's been doing. So that's
25 where that's coming from.

1 To get back to the -- to the previous question -- I've
2 lost my train of thought, sorry.

3 **THE COURT:** Oh, that's okay. Let me ask you the
4 question a different way. It might bring you back to what you
5 were thinking about.

6 **THE WITNESS:** Okay.

7 **THE COURT:** So looking at this Conclusions slide, the
8 one that is up there now --

9 **THE WITNESS:** Okay.

10 **THE COURT:** -- I'm trying to -- the questions I'm
11 asking you are trying to get at, what are the IARC's opinions.

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

13 **THE COURT:** What did the Working Group conclude? And
14 what are you concluding that the Working Group did not
15 conclude? Okay?

16 So looking at the first bullet --

17 **THE WITNESS:** Okay.

18 **THE COURT:** -- you say,

19 "I conclude, to a reasonable degree of
20 scientific certainty, that glyphosate and
21 glyphosate-based formulations are probable
22 human carcinogens."

23 I take it that that is coextensive with the IARC's
24 conclusion, is that right?

25 **THE WITNESS:** That's correct.

1 **THE COURT:** Okay. Now the second sentence:

2 "I also conclude, to a reasonable
3 degree of scientific certainty, that
4 glyphosate and glyphosate-based
5 formulations cause NHL in humans."

6 The IARC did not reach that conclusion; is that correct?

7 **THE WITNESS:** That -- I think if you -- if you read
8 the monograph and look at the epidemiology section, they did
9 say that, that exposure to glyphosate formulations was
10 associated with non-Hodgkin's lymphoma. I think that's the
11 word -- similarly wording to what is in the monograph in the
12 epidemiology section.

13 **THE COURT:** Right, the monograph says that there's
14 limited evidence of carcinogenicity in humans, and identifies
15 the studies that -- where -- that suggest an association.

16 **THE WITNESS:** An association with non-Hodgkin's
17 lymphoma.

18 **THE COURT:** With non-Hodgkin's lymphoma.

19 **THE WITNESS:** Right.

20 **THE COURT:** But there is not a conclusion.

21 **THE WITNESS:** -- that it --

22 **THE COURT:** -- by IARC. If we could go back to the
23 Conclusions slide.

24 There is not a conclusion in the IARC Monograph that
25 glyphosate and glyphosate-based formulations cause NHL in

1 humans, is there?

2 **THE WITNESS:** It is not an absolute statement to that
3 effect, that's correct.

4 **THE COURT:** So to the extent you're providing an
5 opinion on that, that is your opinion.

6 **THE WITNESS:** Absolutely.

7 **THE COURT:** Not the -- not the IARC's opinion.

8 **THE WITNESS:** Not the stated IARC's opinion, but I --
9 but I --

10 **THE COURT:** And you draw from that --

11 **THE WITNESS:** Absolutely.

12 **THE COURT:** -- from the monograph, to reach that
13 conclusion. I understand that.

14 **THE WITNESS:** Yeah.

15 **THE COURT:** But I -- and you'll get to talk more
16 about this with the lawyers, but I just want to have this sort
17 of delineate these basic boundaries --

18 **THE WITNESS:** Sure, sure.

19 **THE COURT:** -- between the IARC's Working Group's
20 conclusions and yours.

21 **THE WITNESS:** Right, right.

22 **THE COURT:** So you draw from their work --

23 **THE WITNESS:** Right.

24 **THE COURT:** -- to reach this conclusion that
25 glyphosate and glyphosate-based formulations cause NHL in

1 humans, but they did not reach that conclusion or articulate
2 that conclusion.

3 **THE WITNESS:** They did not articulate that
4 conclusion.

5 **THE COURT:** Okay.

6 **THE WITNESS:** I had to learn -- being a member of the
7 IARC Working Group, I had the luxury of participating in the
8 discussions during the IARC Working Group meeting, and
9 discussed the data with all of the epidemiologists.

10 **THE COURT:** Can you go back to the Conclusions slide
11 again?

12 **MS. WAGSTAFF:** Sure, sure.

13 **THE WITNESS:** To discuss all the, you know, the data
14 with all of the epidemiologists, the formally trained
15 epidemiologists that were present at the meeting, and -- and I
16 can say there were -- there were a couple of epidemiologists at
17 the meeting, at least when the meeting began, that were saying
18 they really felt that there was sufficient evidence in humans
19 that glyphosate caused non-Hodgkin's lymphoma.

20 But in -- but as the process for the IARC Monograph, the
21 whole Working Group sits down and evaluates all of the data,
22 not only the epi, but the toxicology and mechanistic data, and
23 we have a rather, you know, detailed discussion of what all of
24 the data is saying.

25 And so that's how the -- the ultimate decision of the

1 Working Group is made. And I would point out that the -- that
2 the monograph, and the conclusions in the monograph, are that
3 of that monograph Working Group.

4 And so it's everybody in the Working Group participated in
5 the discussion, and voted on the various -- listings that are
6 in the monograph, so --

7 **THE COURT:** And so you mentioned that were that there
8 were some scientists who believed that there was sort of
9 stronger proof, stronger epidemiological proof, of a link
10 between glyphosate --

11 **THE WITNESS:** And non-Hodgkin's lymphoma.

12 **THE COURT:** -- and non-Hodgkin's lymphoma than what
13 was articulated.

14 **THE WITNESS:** In the --

15 **THE COURT:** In the monograph.

16 **THE WITNESS:** In the monograph.

17 **THE COURT:** Okay, and were there any dissenters?

18 **THE WITNESS:** Yes.

19 **THE COURT:** Were there people who said that it -- you
20 know, there's not -- who argued that there is not enough
21 evidence to support the Working Group's conclusion that there
22 is limited evidence of carcinogenicity in -- of glyphosate.

23 **THE WITNESS:** Yes, sir, I was just about to say that,
24 that, you know, I've always said if you -- if you get three
25 epidemiologists in the room, and you ask them their opinion,

1 you get four opinions. So --

2 (Laughter.)

3 **THE COURT:** So far, it seems like, to me, the
4 epidemiologists have nine opinions.

5 **THE WITNESS:** So there were people that came in there
6 and said, oh, this is a -- excuse me -- a flaming carcinogen,
7 human carcinogen, there's non-Hodgkin's lymphoma everywhere.
8 And then there are others that say, no, no, no, the data's just
9 not there; there's maybe an indication it's not there, it's not
10 statistically significant; even though it is a significant
11 finding it's not statistically significant.

12 And so that's all part of the review process of the IARC
13 and there's a similar thing when I did the Report on
14 Carcinogens, you know, all of the scientists get together, we
15 all discuss the data, and -- and then, you know, after
16 everybody has had an opportunity to discuss their side and
17 their opinion of what the data says, then you come to a general
18 consensus of, okay, we have to look at the criteria that we're
19 given to use when we do an IARC Monograph. We have to use
20 their criteria and use their wording that is in the preamble
21 when we ultimately make a final decision.

22 So that's where a lot of the wording comes from, because
23 we're limited, if you will, by what the preamble says we have
24 to use.

25 **THE COURT:** Okay, and then going to the second bullet

1 on your Conclusions slide, just as --

2 **THE WITNESS:** Okay.

3 **THE COURT:** -- again, just in terms of delineating
4 between what's IARC's conclusion and what's your opinion here.

5 **THE WITNESS:** Right.

6 **THE COURT:** The first part of the bullet, "Exposure
7 to glyphosate can cause NHL."

8 **THE WITNESS:** Right.

9 **THE COURT:** That is coextensive is the IARC's
10 conclusion, yes?

11 **THE WITNESS:** Yes, but it's my --

12 **THE COURT:** And you're -- and then the second part of
13 this bullet is, "is currently doing so at exposures levels of
14 today," that's not an IARC conclusion.

15 **THE WITNESS:** That's mine.

16 **THE COURT:** Okay.

17 **THE WITNESS:** But I mean, it just logically finds --
18 follows that --

19 **THE COURT:** And you'll have time to get into detail
20 about it, but I just -- just for -- in terms of establishing a
21 framework for your testimony --

22 **THE WITNESS:** Sure.

23 **THE COURT:** -- I just wanted to sort of get those
24 basic points out.

25 **THE WITNESS:** Sure.

1 **THE COURT:** And then the third bullet point, I take
2 it, is pretty similar to the IARC's --

3 **THE WITNESS:** Right.

4 **THE COURT:** -- conclusion. The way the IARC puts it
5 is, there's limited evidence of carcinogenicity in humans, and
6 they note that -- that the epidemiological studies have
7 shown -- some of the epidemiological studies have shown an
8 association between glyphosate and NHL, and your conclusion is
9 that there's credible evidence that exposure to glyphosate
10 causes NHL in humans. Correct?

11 **THE WITNESS:** Correct. And if I may, the bullet
12 you're referring to there is basically trying to answer a
13 question you asked the other -- defendant's other experts.

14 Taken alone, what does the epidemiology data tell you? In
15 exclusion to the toxicology or mechanistic data, what does the
16 epidemiology alone tell you?

17 **THE COURT:** Okay. Could I -- just one more follow-up
18 question, at least for now, on this concept of hazard
19 identification --

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

21 **THE COURT:** -- and how it is distinguished from risk
22 assessment.

23 I'm now looking at this briefing note for IARC Scientific
24 and Governing Council members prepared by the IARC Director,
25 January 2018.

1 **THE WITNESS:** Yes.

2 **MS. WAGSTAFF:** (indicating).

3 **THE WITNESS:** Thank you.

4 **THE COURT:** And I'm looking at page 10.

5 **THE WITNESS:** Okay.

6 **THE COURT:** I'm looking at the fourth bullet --

7 **THE WITNESS:** Okay.

8 **THE COURT:** -- on page 10, which reads "In
9 contrast...."

10 **MR. HOLLINGSWORTH:** Your Honor, can we get a copy of
11 that, please?

12 **THE COURT:** Sure. You should have it from yesterday,
13 no?

14 **MR. HOLLINGSWORTH:** Okay.

15 **MS. WAGSTAFF:** It's Exhibit 149. I have a copy, if
16 you'd like me to give you one.

17 **MR. HOLLINGSWORTH:** Sorry, your Honor. Thank you.

18 **THE COURT:** I'm looking at the fourth bullet, which
19 says,

20 "In contrast to hazard identification,
21 the specific exercise of risk assessment
22 typically involves extrapolation beyond the
23 observed data, employs a variety of
24 statistical models, and is based on
25 anticipated levels of exposure and

1 background cancer incidence rates that are
2 often specific to a population or region."

3 So again, when I read that, I took that to mean -- kind of
4 hazard identification is a bit closer to simply inquiring
5 whether the substance is capable of causing cancer; and risk
6 assessment is something closer to inquiring whether people are
7 getting cancer from the substance at current exposure levels.

8 And so again, let me just ask: Am I misinterpreting that
9 bullet point, from IARC?

10 **THE WITNESS:** No, that's absolutely correct. I mean,
11 very simply, hazard identification is asking the question: Can
12 the material cause cancer, yes or no?

13 And so you do the studies. You test the animals at as
14 high a level as they can tolerate, and see if it causes cancer.
15 If it does, then it's biologically plausible and accepted in
16 the scientific community that it's probably a human carcinogen,
17 as well.

18 Then -- then the risk assessment takes that information,
19 hey, this chemical has been shown, let's see what the people
20 are -- are exposed to, and then do the calculations that
21 they're talking about, that, okay, based on the -- you know,
22 some of the -- you could take some of the information gleaned
23 from the toxicology data about dose, what doses cause the
24 cancer, and then try to extrapolate it to the human situation,
25 and do your calculations and what have you and say, okay, the

1 dose is so low that there's not a possibility, or the dose is
2 very close, or when need to do more studies, that type of
3 thing.

4 **THE COURT:** Okay, or, you know, we don't know if the
5 dose is too low or close, and so we are going to -- we're going
6 to proactively impose restrictions --

7 **THE WITNESS:** Sure.

8 **THE COURT:** -- on the use of the chemical --

9 **THE WITNESS:** Right.

10 **THE COURT:** -- to make sure that people aren't
11 getting hurt by it.

12 **THE WITNESS:** To err on the side of safety is
13 absolutely what people -- what I think that the regulatory
14 agencies are trying to do, is to make sure that it's safe,
15 people are safe.

16 **THE COURT:** Okay, great. Thank you. Sorry for the
17 detour.

18 **MS. WAGSTAFF:** Okay, no problem, and I just have a
19 follow-up question from that.

20 **THE WITNESS:** Okay.

21 **BY MS. WAGSTAFF**

22 **Q.** Instead of talking about hazard assessment sort of in the
23 abstract, or -- I think you said that the preamble has a
24 definition of a hazard assessment, that it's supposed to
25 capture whatever data you have on any chemical.

1 Let's talk about what happened actually in the hazard
2 assessment for glyphosate.

3 **A.** Okay.

4 **Q.** The Working Group 112 actually considered the
5 epidemiology, correct?

6 **A.** Correct.

7 **Q.** And that is the real world exposure, right?

8 **A.** Correct.

9 **Q.** Working -- Working Group 112 actually had an exposure
10 group, right?

11 **A.** Absolutely, yes.

12 **Q.** And in that exposure group, they considered real world
13 exposure, right?

14 **A.** That is a very important piece of data that is used by the
15 Working Group especially for the epidemiologists, so they know
16 what people are exposed to, and at what levels.

17 **Q.** Okay, so whether or not hazard assessments are used to
18 determine whether they can cause cancer or at what level, it is
19 true that Monograph Working Group 112 considered real world
20 exposure levels, right?

21 **A.** Absolutely.

22 **Q.** Okay, and did you consider that in your hazard assessment
23 for this case?

24 **A.** Yes, absolutely.

25 **Q.** Okay, and one last thing, if you could pull up Exhibit 57,

1 which is the glyphosate monograph.

2 Do you guys have a copy, or do you need one? Okay.

3 Page 75, if you could blow up that last page, please 75.

4 Yep, on the right, starting with, "In summary."

5 So Judge Chhabria was asking you questions on the position
6 that the Working Group took with respect to NHL, and I didn't
7 know if you wanted to refresh your memory as to what the
8 Working Group determined with respect to NHL.

9 **A.** Right. Well, this is just summarizing the epidemiology
10 data, and basically what the monograph is saying is that in
11 summary, the case-control studies in the U.S., Canada and
12 Sweden reported increased risks of -- for national -- for
13 non-Hodgkin's lymphoma associated with exposure to glyphosate.
14 The increased risk persisted in the studies that adjusted for
15 exposure to other pesticides. However, the AHS -- the AHS
16 cohort did not show an excess of NHL.

17 So the Working Group noted that there was excesses
18 reported for multiple myeloma, in three studies. But that's
19 not part of what we're talking about here.

20 **Q.** Okay, excellent.

21 And then one last question on this. The definition of
22 "limited" as set forth by IARC includes the finding of credible
23 evidence, correct?

24 **A.** Right. It says a credible -- that exposure to the
25 material -- an association between exposure to the material and

1 cancer in humans is credible, but that all the confounders
2 could not absolutely be explained away.

3 **Q.** Okay, excellent. So let's go back to your PowerPoint now,
4 and why don't you tell the Court a little bit about what
5 toxicology is.

6 **A.** Okay. As I've indicated earlier, toxicology is used to
7 determine whether the agent is an animal carcinogen. That's --
8 that is done to see, A, if it causes cancer in laboratory
9 animals, and B, if it does, is it biologically plausible that
10 it is a human carcinogen.

11 This is a premise that is generally accepted in the
12 scientific community, that if an agent causes a cancer in
13 animals, that it's biologically plausible to be a human
14 carcinogen.

15 And while the Bradford Hill has been discussed quite a bit
16 over the past couple of days, and I'll admit that the
17 Bradford Hill initially was published and used in the
18 epidemiology community, but here, the toxicologists have picked
19 up on the Bradford Hill criteria and looked at it, and it was
20 very applicable to the toxicology studies that we do. And so
21 we -- we follow the Bradford-Hill criteria when we do
22 toxicology studies, as well.

23 And so -- the bottom line is, the toxic -- the animal
24 bioassay studies or the animal toxicology data is very
25 applicable to humans, because of a general acceptance that you

1 can apply the results from the animal bioassays to predict that
2 an agent is a human carcinogen.

3 Q. Okay, excellent. You keep talking about a "carcinogen."
4 What does that mean?

5 A. A carcinogen is a material that causes unregulated cell
6 growth in -- in -- in an organ, in animal. It's unregulated
7 cell growth that basically causes the tumor formation in the
8 animal.

9 Q. Okay, so the cancer is -- you're defining the cancer as
10 sort of unregulated cell growth or cell division?

11 A. Correct.

12 Q. Okay, and is it generally accepted by peer-reviewed
13 literature that you can apply what you learned from toxicology
14 to predict cancer in humans?

15 A. Yes.

16 Q. Okay. Can we go to the next slide, please?

17 And Monsanto has made a lot of hay in the papers about
18 your use of rodent models as a predictor of cancer in humans.

19 So in this particular toxicology set, there were four
20 types of rodents used, CD-1 mice, Swiss albino mice, Wistar
21 rats and Sprague-Dawley rats. Are those generally accepted
22 rodent models in toxicology?

23 A. Yes, those are widely accepted and widely used in animal
24 bioassay studies. In fact, those are the species that were
25 used by Monsanto and other industry people in -- in doing the

1 bioassays and submitting the data for registration of their
2 materials.

3 Q. All right. Now, let's focus on your second bullet point.
4 You have a phrase in there called "tumor site"?

5 A. Mm-hm.

6 Q. Can you tell the judges what that means?

7 A. When you refer to a tumor site, basically that just means
8 the organ within the animal where you observe the -- the tumor,
9 the place where the unregulated cell growth happened.

10 Q. Okay, and in this -- in this body of toxicology we have
11 lung tumor sites, right?

12 A. Specifically glyphosate?

13 Q. Yes.

14 A. Yes.

15 Q. We also show renal tumor sites?

16 A. Correct.

17 Q. We also show pancreatic tumor sites?

18 A. Correct.

19 Q. And we show malignant lymphoma, correct?

20 A. Correct.

21 Q. And then NHL is a tumor site, right?

22 A. NHL is a tumor site.

23 Q. So Monsanto has claimed that you have to have matching
24 tumor sites in the animal and in the human. Can you speak to
25 that little bit? And I think you sort of articulate it in your

1 second bullet point.

2 **A.** Yeah. In toxicology, and in at least my vast experience
3 and familiarity with the literature, it's very rare to have an
4 animal model for a specific tumor site in humans. It just --
5 they just don't exist. There may be one or two, but it's very,
6 very rare to have that.

7 The purpose of the toxicology studies is to see, A,
8 does -- is basically just to define if the material causes
9 cancer in animals; therefore, it is probably -- it is very
10 likely to be a human carcinogen.

11 So the animal bioassay data is used to say, yes, it is a
12 carcinogen, and then you use the epidemiology, you go and look
13 at a population that is exposed to a particular material to
14 identify where in humans the tumor site could be.

15 **Q.** All right. So let's just try to make this as simple as
16 possible. You used the animal data to figure out if the
17 chemical causes unregulated cell division in the animal,
18 regardless of where, correct?

19 **A.** Correct.

20 **Q.** Okay, and then once you know that the chemical causes
21 unregulated cell division in the animal, you use the
22 epidemiology to figure out where that will happen in the human,
23 right?

24 **A.** That's correct.

25 **Q.** Okay. So the toxicology gets you so far, and then you

1 couple it with the epidemiology to figure out where
2 specifically it occurs in the human. Is that correct?

3 **A.** Correct.

4 **Q.** Okay, and that's -- that's typically how toxicology is
5 used, correct?

6 **A.** That's very typical of how the -- how it works.

7 **Q.** Okay.

8 **A.** And --

9 **Q.** And is there a specific rodent model for an NHL tumor
10 site?

11 **A.** No, not to my knowledge. There is -- there is no specific
12 tumor or animal model for the NHL, although I will admit
13 that -- that the -- in the mice, the data in mice, we have a
14 number of studies where malignant lymphoma was found in mice;
15 and malignant lymphoma is a tumor of the lymphatic system in
16 mice. NHL is the lymphatic system in the rodents.

17 Humans have B-cells and T-cells, which are the affected
18 cells in non-Hodgkin's lymphoma. The rodents in this case, the
19 mice, also have B-cells and T-cells in the lymphatic system
20 that are effected by glyphosate. And so there you have very
21 good correlation, if you will, which you very -- you don't
22 always have, that glyphosate causes a tumor in a mouse at a
23 similar site to where you see it in humans.

24 **Q.** Okay, excellent. And if you could pull up slide 327,
25 please, you were asked this exact thing -- can you blow up the

1 entire thing? -- in your deposition. You've been deposed three
2 times in there litigation, right? You've had the most
3 depositions. You're very lucky.

4 **A.** That's correct.

5 **Q.** And do you remember being asked by Mr. Hollingsworth,
6 quote,

7 **"QUESTION:** My question is whether the
8 hypotheses that mouse renal tumors are
9 predictive of non-Hodgkin's lymphoma
10 specifically in humans has ever been tested."

11 Do you remember him asking you that?

12 **A.** I remember being asked that many times yes.

13 **Q.** Okay, and throughout the course of your eight-hour
14 deposition, you were asked that about every single tumor site,
15 right?

16 **A.** That's correct.

17 **Q.** And you gave an answer, and what's highlighted is the
18 answer that Monsanto put in your -- in their brief, but I just
19 wanted to show your entire answer, which is consistent with
20 what you've said today, right?

21 **A.** Yes. You know, they -- this -- this is my -- my entire
22 answer to that question.

23 I came back and said, again, this, you know, is the
24 purpose of the bioassays, to see if a chemical causes cancer in
25 animals as a predictive tool for what it causes in humans.

1 Now, I mean, the fact that something causes a kidney tumor
2 in a mouse, I don't know what that means about causing
3 non-Hodgkin's lymphoma in humans --

4 **THE REPORTER:** I'm so sorry, I lost you. I lost you.
5 Something causes kidney tumor in a mouse --

6 **THE WITNESS:** Oh, the fact that something causes a
7 kidney tumor in a mouse, I don't know what that says about
8 causing non-Hodgkin's lymphoma in humans. I don't know that
9 that's been investigated.

10 **BY MS. WAGSTAFF:**

11 **Q.** Okay. So let me just stop you right there, and what you
12 were talking about is what we just discussed --

13 **A.** Correct.

14 **Q.** -- is that the animal data tells you whether or not the
15 chemical causes unregulated cell division, correct?

16 **A.** Right.

17 **Q.** And then what you say at the end of your answer is, the
18 purpose of doing the study in a mouse is to see if it causes
19 cancer, and that's used as a predictive tool to see if it
20 causes cancer in humans.

21 **A.** Correct.

22 **Q.** And that's what we just discussed, is that you use the
23 epidemiology to figure out the human tumor site, correct?

24 **A.** That's correct.

25 **Q.** And Monsanto didn't give the court your full answer,

1 correct?

2 **A.** That's what it appears, yes. I didn't see the briefs, so
3 I don't know.

4 **Q.** Okay, excellent.

5 And this is Exhibit 327, and it's in your -- the judges'
6 notebook, and we'll move that in to evidence as well.

7 If you can go back to the slide, please, Mr. Wisner.

8 Okay, excellent.

9 And the last bullet point, you sort of touched on this a
10 little bit. NHL is a cancer of the lymphatic system, right?

11 **A.** Correct.

12 **Q.** Okay, and is it important in any way that the rodents in
13 the model have a different lymphatic system and/or immune
14 system than humans? Is that important, and how did you put
15 that in your analysis?

16 **A.** Well, I mean, the fact of the matter is, physiologically
17 they're different. The lymphatic system in the mouse is
18 physiologically different than the lymphatic system in humans,
19 but there are also similarities. Like I said, like I indicated
20 before, there are B-cells and T-cells in the mouse, and there
21 are B-cells and T-cells in the human lymphatic system, and
22 those seem to be the cells that are affected by glyphosate that
23 causes cancer.

24 **Q.** Okay. So it would be a more appropriate question that the
25 similarities we should look at that are relevant here rather

1 than the differences, is that correct?

2 **A.** Correct.

3 **Q.** Okay. If we can go to the next slide please?

4 So we're finally getting to the studies you that you
5 reviewed.

6 **A.** Okay.

7 **Q.** Looks like you reviewed 12 rodent studies.

8 **A.** Correct.

9 **Q.** And those were the mice and the rats that we discussed
10 earlier.

11 **A.** Correct.

12 **Q.** Okay, and why don't you tell the Court a little bit about
13 what information you reviewed in making your opinion and
14 whether or not that body of information is generally relied
15 upon in the toxicology community.

16 **A.** Okay, kind of -- and I indicated before, I performed a
17 peer-reviewed literature search to find all peer-reviewed
18 literature available for glyphosate and cancer.

19 I've looked at tumor tables for individual animals. These
20 were tumor tables that were provided in some peer-reviewed
21 publications, or were provided from the actual studies that
22 were performed on the animals.

23 And for some, but not all, I had the actual pathology
24 reports from the study laboratory to review, that I also had
25 narrative summaries from the some of the studies, and in fact,

1 for some of those studies, I also had the entire lab report,
2 testing laboratory report of that bioassay.

3 And this is the type of information that is typically that
4 I -- that I have routinely used in -- in doing cancer hazard
5 identifications throughout my 40-year career. I've been
6 looking at all of the data to -- to come to an evaluation of
7 the potential carcinogenicity of the substance.

8 Q. Okay, excellent.

9 Mr. Wisner, if you could, pull up Exhibit 324. All right,
10 and I have copies. I think these are in your book. If you
11 could, blow it up so that it fits the screen. Yeah, just right
12 there, yep.

13 And so this is a table that you made, correct?

14 A. It's a table, it's a cheat sheet, if you will, that I put
15 together to remind me what -- what I found in the various
16 studies, and --

17 Q. This is tab 8.

18 A. And I use the -- basically, I referred to the Greim
19 publication in this table as a means of just keeping straight
20 which studies I was looking at.

21 The first column is identified -- identifies the study
22 where the study -- who the principal investigators were and the
23 year that the study was done.

24 The second column identifies which strain of animal --

25 Q. Hang on, real quick, when it says Study 10, just so the

1 judges know when they look at this later --

2 **A.** That refers to the Greim paper, study 10 in the
3 Greim paper.

4 **Q.** Okay, and that's really of no significance other than
5 that?

6 **A.** That's the only --

7 **Q.** Okay.

8 **A.** Like I said, I just use that as a means to --

9 **Q.** Organization.

10 **A.** -- to organize my thoughts, if you will.

11 Second column is Strain, which identifies what strain of
12 mouse was used, and if it was a male and females, or females.

13 The third column identifies the dose levels that were used
14 in the studies, and for what duration the study was.

15 The next -- the fourth column identifies the tumors that
16 were observed that were significant, and the incidence.

17 The 1 of 49 means that there was one tumor in 49 animals;
18 and it goes from control low, medium, and high dose. That
19 that's the order of the material -- of the numbers in there.
20 Those are referred to as a tumor incidence.

21 The next column identifies the statistical significance of
22 the tumors that were listed.

23 And then the last column is an evaluation, is basically my
24 comments on the -- on the particular study. And again, I put
25 this together for my -- on my own purpose, just to remind me of

1 what each study -- I found in each study.

2 Q. Okay, excellent, and I'm sure Monsanto's counsel will ask
3 you about these cases in detail on your cross-examination, but
4 I wanted to explain to the Court what these were.

5 If you look on the upper left-hand side of it, you'll see
6 that this is for the mice. This is for the mouse?

7 A. Yes.

8 Q. In your book, you also have one that you made for the
9 rats, right?

10 A. Correct.

11 Q. And also, you have a chart that you made similar to this
12 for the epidemiological case-control, right?

13 A. Correct.

14 Q. You also made one for the meta-analyses, right?

15 A. Correct.

16 Q. Okay, and did you make one for the cohort study?

17 A. I did not make one for the cohort study, the AHS study,
18 because there was only one study, and that found null
19 association, and plus I have some concerns about the study
20 itself. So I didn't make a table for that.

21 Q. But you made an entire expert report, and you submitted
22 for a deposition just on that specific study, correct?

23 A. I did, yes.

24 Q. Okay, and that expert report is also in your book.

25 So those are Exhibits 322, 323, 324, and 325, and we'll

1 move those in to evidence, as well.

2 (Exhibits 322, 323, 324, and 325 entered into evidence.)

3 **MS. WAGSTAFF:** Our next slide, please. Yep, dosing.

4 **Q.** I'd like to talk a little bit about dosing. One of the
5 complaints that Monsanto has about the toxicology is that these
6 animals are just given insane levels of glyphosate that would
7 be irrelevant to any analysis we have to do today.

8 So could you please talk a little bit about dosing within
9 the toxicology field?

10 **A.** Sure, I'd be happy to. As I indicated before, the purpose
11 of an animal bioassay is to determine if, given the dose -- if
12 an animal is given a dose that it can tolerate for its
13 lifetime, does it cause cancer in that animal? So this is done
14 by using what's referred to as a maximum tolerated dose.

15 The maximum tolerated dose is defined as a dose which you
16 can give to the animal over their lifetime which does -- which
17 causes up to -- which causes up to 10 percent decrease, causes
18 no more -- I'm sorry -- that can cause no more than a
19 10 percent decrease in body weight over the study, or a
20 10 percent increase in mortality. In other words, you can have
21 10 percent of -- less than 10 percent of animals die over the
22 course of the lifetime.

23 Now, the animal bioassay was -- was started, if you will,
24 back in the late '50s, early '60s, and rodents or mice and rats
25 were selected as the test species of choice, because of their

1 relatively short lifetime. You can do a lifetime study in a
2 mouse and a rat in two years, or a little over two years.

3 And so the bioassay is -- that was developed to study the
4 animals do a lifetime study in the animals at the maximum
5 tolerated dose, a dose that they can tolerate without seeing
6 these 10 percent decrements in body weight or mortality, and to
7 see if, given as much material as the animals can tolerate,
8 does it cause cancer in the animals.

9 And if it does cause cancer in the animals, then it's
10 biologically plausible that it causes cancer in humans.

11 Q. Okay, thank you. And the MTD is a generally accepted
12 standard for dosing rodents in toxicology, correct?

13 A. That's correct. The National Toxicology Program's rodent
14 bioassay, which is a preëminent study of the government -- the
15 government is part of the National Institutes of Health and so
16 it's a government program -- is the gold standard, if you will,
17 and every protocol for an animal bioassay is performed at the
18 maximum tolerated dose.

19 I can go in to a discussion of, if -- and reaching the
20 maximum tolerated dose is an important concept in evaluating
21 the adequacy of an animal bioassay, because if you test -- do a
22 test at a level that does not reach the maximum tolerated dose,
23 and the study is completed and you don't see a 10 percent
24 decrease in body weight or mortality over the course of the
25 study, then you say the maximum tolerated dose wasn't reached.

1 So if you don't see an effect in that study, then it's an
2 invalid study for carcinogenicity, because the animals could
3 have tolerated more -- more material.

4 But kind of on the flip side of that, you can run a study
5 at less than the maximum tolerated dose; in other words, a
6 study where the animals could have tolerated more material, but
7 if you see an effect in that study, it's a valid study, because
8 you can evaluate that effect.

9 In the animals, even though they could have tolerated
10 more, you just say that, well, if you had given them more, you
11 would have seen more tumors than you did see, but you could see
12 a statistically significant increase in tumors even though you
13 didn't reach the MTD.

14 **Q.** All right, so that's actually two very important concepts
15 I just want to try to summarize in laymen's terms, if I can.

16 So you have this concept of MTD, right?

17 **A.** Yes.

18 **Q.** And toxicologists all over the world use that dosing
19 concept, correct?

20 **A.** Correct.

21 **Q.** Okay, and it's not -- it's not a hard line, right?

22 **A.** It's something you have to determine in the course of
23 doing your studies. You usually do preliminary, what are
24 called prechronic studies for up to 13 weeks, where you test
25 different doses to see what the animals can tolerate over that

1 time period, and then you set your maximum tolerated dose based
2 on those results.

3 Q. Okay, excellent. And you just mentioned two concepts that
4 I'd like to discuss. One is, even if you don't reach the MTD
5 of the animal, if the animal shows some effects, that
6 information is still important and valid and should be
7 considered. Is that --

8 A. Absolutely.

9 Q. -- what I heard you say?

10 A. Absolutely.

11 Q. Okay, and the second one is, even if you reach what you
12 would consider to be MTD, there are other considerations that
13 you take in to account, like body weight or things like that,
14 that would suggest to you, in your experience of 40 years as a
15 toxicologist, that in fact, MTD was not reached, is that
16 correct?

17 A. You look at the data, the body weight data and the
18 survival data, and if you don't see any effect compared to the
19 controls that's more than 10 percent, then it -- they could
20 have tolerated a higher dose.

21 Q. Okay, and those are scientific judgment calls that come
22 with the experience of being a toxicologist, right?

23 A. Correct, that's what comes with toxicologists, and that is
24 the rule, if you will, that is applied by all toxicologists
25 when they review an animal bioassay study. In my experience

1 with IARC and also in reviewing the data for the Report on
2 Carcinogens, that's part of the evaluation. You look at the
3 study, you look at the survival and the body weight, and to see
4 if the animals were tested at the MTD or if they could have
5 tolerated higher levels.

6 Q. Okay, and so you use these principles that you had learned
7 in your 40-plus years as a government toxicologist when you did
8 your analysis on MTD in this toxicology dataset, right?

9 A. Right. That makes me feel like an old man, 40 years.

10 Q. Okay, sorry. It's a good thing.

11 All right. Next slide, please?

12 **THE COURT:** Is now a good time for the old man to
13 take a lunch break?

14 **MS. WAGSTAFF:** I actually only have about 10 more
15 minutes, if we want to finish.

16 **THE COURT:** Okay.

17 **MS. WAGSTAFF:** I could probably be, unless you have a
18 lot of questions for him. Okay.

19 **THE COURT:** Go ahead.

20 **MS. WAGSTAFF:** Unless you want to stop.

21 **THE WITNESS:** No, that's fine.

22 **BY MS. WAGSTAFF**

23 Q. Okay, so next I want to talk about concurrent and
24 historical controls.

25 A. Okay.

1 Q. Please tell the judges what those are, and how you
2 factored them into your analysis today.

3 A. Okay. Concurrent control are the control animals that
4 are -- that you have that are run concurrent with the -- with
5 the study that you're performing. Those are animals that
6 are -- that are the same strain, same genetic background.
7 They're handled exactly the same way as the treated animals.
8 They only difference is, they are not exposed to the material
9 you're studying. In this case, it would be the glyphosate.
10 Those are the concurrent controls.

11 Those are the most appropriate controls to use when you're
12 evaluating the study, to see what the -- what the tumor
13 incidence or the increase in tumor incidence was in the treated
14 animal versus what it was with the animals that got no
15 material.

16 Another piece of information that is -- that toxicologists
17 routinely look at is what we refer to as the historical
18 controls, and a historical control are the control or animals
19 that are not been treated with a chemical, in studies that were
20 performed at -- at other facilities, or in the reported
21 literature, where you get a -- this -- the historical controls
22 are used for people to get a feel for what the spontaneous
23 incidence of a tumor in is an animal. It gives you a larger
24 population, if you will, of animals that were -- (whereupon a
25 document was tendered to the Court) -- were not controlled, but

1 were treated in a similar manner; not the exact manner but, you
2 know, you look at historical controls of all the feeding
3 studies that you can look at, or all of the drinking water
4 studies, or all of inhalation studies that you're looking at.

5 You use that historical data to see, what's the
6 spontaneous incidence of this? And you use that to say, well,
7 the control group in my study has the same number of tumors as
8 has been seen in the past in similar studies; or the control
9 group has fewer tumors than you would see in the past; or has
10 more tumors. But it's just a piece of information that you use
11 in the evaluation.

12 You always use the concurrent controls as the most
13 appropriate control, and you use that -- that gives you more
14 information than looking at the historical controls, but the
15 historical control is a piece of information you need to
16 evaluate the validity of your study.

17 **BY MS. WAGSTAFF:**

18 **Q.** And is your view on the importance of concurrent and
19 historical controls shared by the toxicology community?

20 **A.** Yes. In fact, I've published on this.

21 **Q.** Just handing out your article.

22 **A.** (Laughs.)

23 **Q.** I hope this doesn't make you feel old. It's from 1988.

24 If you could, pull up Exhibit 295, please. The front
25 page, just make it a little bigger. Highlight his name as an

1 author. Well, you guys can probably all see it.

2 Yeah, and then let's move to page 7, and let's look at
3 your views on historical and concurrent controls back from
4 1988.

5 There were some suggestions by Monsanto that your views
6 have changed since you became part of this litigation. So
7 I just -- this was 30 years ago, so I want to make sure that
8 this is still what you believe today.

9 Could you take a moment to look at it?

10 **A.** Sure. What we stated in our paper was that although
11 concurrent control groups are always the first and most
12 appropriate control group used for comparison, the treating
13 control groups, historical control groups, can be helpful in
14 overall assessment of tumor incidence.

15 Consequently control tumor incidence from NTP historical
16 control database are included in -- are included from
17 particular laboratory and from the overall program for the
18 tumors appearing in these associated -- appearing to be
19 associated with chemical exposure.

20 **Q.** Okay. So back in 1988, you were saying the same thing.
21 Both are pieces of information to consider, concurrent is more
22 important.

23 **A.** Absolutely.

24 **Q.** All right, let's just move down to the next paragraph,
25 just because we have this page up, and I just am curious about

1 your opinions on p-values from 1988, and this is the article
2 that you wrote, right?

3 **A.** Right.

4 **Q.** And in this article, you're saying that if you just look
5 at the first....

6 If you could blow up, you know, the first couple
7 sentences -- just keep going down, keep going down, go down.
8 Yeah, that's probably enough. Is there any way to highlight
9 that or something? Yeah, there you go.

10 **A.** Yeah, and in this, we were just saying that p-values are
11 objective facts, but unless a p-value is extreme, proper use of
12 it to decide whether or not the chemical is carcinogenic
13 involves subjective and scientific judgment.

14 Although the p-values may be helpful in deciding whether
15 or not a substance is carcinogenic, they -- but they must not
16 be used inflexibly or given undue weight.

17 **Q.** Okay, excellent. And that was -- that was obviously
18 peer-reviewed -- that opinion was peer-reviewed in 1988.

19 **A.** Yes.

20 **Q.** Okay, and let's -- you can pull that down. Let's turn to
21 the next slide, where we talk a little bit about replication.

22 Please tell the Court what replication is, and how you
23 used it in your opinion.

24 **A.** For the purpose of toxicology, I mean, very simply,
25 replication means you see the same effect in two different

1 studies, or in multiple studies. It's just another piece of
2 evidence that you use in evaluating the overall strength of the
3 data.

4 I would point out that -- that usually you don't have the
5 luxury of having a lot of studies in animal bioassays. For the
6 glyphosate situation, it's -- it's very -- it's extraordinary
7 to have these many animal studies to evaluate, or -- animal
8 carcinogenicity studies to evaluate. Usually, at most, you'll
9 have two, because of the expense, because of the time and
10 expense used that is necessary to do animal bioassay.

11 So it's very rare that you have several studies to compare
12 to see if you have replication, but it's just another piece of
13 the information. And if you do have replication across
14 studies, that just strengthens the evidence that this is an
15 animal carcinogen and this is the tumor site for that
16 particular study.

17 **Q.** All right, excellent. And over your course of your
18 career, did you ever determine that something was an animal
19 carcinogen when there was no replication?

20 **A.** Oh, yes, there was -- that, as I said, you don't usually
21 have the luxury of more than one study, and if the results from
22 a single animal bioassay study is very strong, I mean, then it
23 makes it easy to determine, but you -- you look at the strength
24 of the study, if it's well conducted, done on GOP guidelines,
25 and you look at the data generated from the study and make your

1 evaluation, but you can definitely make an evaluation from one
2 study.

3 Q. Okay, excellent. And you can have replication across
4 species, right?

5 A. You can have replication across species, you can have
6 replication across sex.

7 Q. Strain?

8 A. Strain.

9 Q. And laboratories or authors?

10 A. Right.

11 Q. Okay.

12 A. Right. I mean --

13 Q. So was there replication in this toxicology data set?

14 A. Yes, and for glyphosate, there was replication of several
15 tumor sites.

16 Q. Okay.

17 A. In fact --

18 Q. And in fact, you've made a replication chart.

19 A. I made a chart.

20 Q. And it's number 10 in your notebook. And why don't you
21 tell the Court, while they look for this, explain to them what
22 this is.

23 A. Basically, I've -- I just use this as a method to
24 highlight replication in the studies. We had replication in
25 male CD mice, for angiosarcoma --

1 **THE REPORTER:** I'm sorry, in male CB mice?

2 **THE WITNESS:** In male CD-1 mice. I'll slow down, I'm
3 sorry. We had replication -- we observed liver tumors in
4 Wistar rats and Sprague-Dawley rats, although the incidence of
5 the liver tumors in the Wistar rat were not significant.

6 We had replication of malignant lymphoma in mice. We had
7 replication in males and females in the Swiss mice. We had
8 replication in male CD-1 mice in two separate studies. We had
9 replication of malignant lymphoma in CD-1 mice and in Swiss
10 mice. So we had a lot of replication for the malignant
11 lymphomas in the mouse.

12 Pancreatic islet cell adenoma, we had replication in the
13 rat in males; and for the renal tubular adenomas, we had
14 replication in two studies, in CD-1 mice; and we had
15 replication across species in the CD-1 and the Swiss mouse.

16 **Q.** Okay, excellent. And is it fair to say this is a lot of
17 replication?

18 **A.** Yes, it is. It's -- I mean, that just shows that in the
19 course of the study, there were -- there were similar tumors
20 being -- the same tumor was being observed in different studies
21 done at different laboratories at different times. So that
22 just strengthens the evidence that it's an animal carcinogen.

23 **Q.** Okay, excellent. And was it significant to you -- and you
24 touched on this earlier -- that the malignant lymphoma, which
25 is the closest tumor site to NHL, was replicated four times?

1 A. That's correct.

2 Q. Okay, but you testified in your deposition that there's a
3 high spontaneous rate of malignant lymphoma in Swiss mice.
4 First of all, why don't you tell the Court what a high
5 spontaneous rate means, and secondly, how you that factored
6 that in to your analysis.

7 A. High spontaneous rate just means background rate, just
8 means that -- that the incidence of a particular tumor seen in
9 the historical controls, from a lot of studies, is -- is, you
10 know, relatively high, 10, 20, in fact, some are even up to
11 30 percent of the animals get tumors before they die,
12 spontaneously.

13 And in my deposition, it came up that the malignant
14 lymphoma had a high historical rate, and in fact, that's true
15 in the Swiss mice especially. In the Swiss mice, the
16 background -- historical background rate is very high.

17 But when I did my evaluation and looked at these lymphomas
18 in the CD-1 and the Swiss mice, I went to the literature to see
19 what the background rate was, and the incidence rate in all
20 four of these studies were higher than the historical incidence
21 rate. Even though you have a high incidence rate in the
22 controls, it was the treated animals had a higher rate than
23 seen in the historical controls.

24 Q. Okay, and just for purposes of a hypothetical question, if
25 you remove those four malignant lymphoma brown lines from your

1 chart, that would still be a lot of replication, right?

2 **A.** From the other tumor sites, yes.

3 **Q.** Right, and what's the significance of the -- well, strike
4 that.

5 Is a renal -- a renal tubular adenoma, is that a rare
6 tumor site?

7 **A.** It's a rare tumor in the mouse, yes. The historical rate
8 is very low among those.

9 And again, I looked at the historical rates in the
10 literature and compared to the historical rates that are
11 reported. The incidence of these tumors in these studies were
12 higher, you know, almost by a factor of two in some of the
13 studies, than the historical rates.

14 **Q.** And is it true that when you see rare tumors in these
15 toxicology studies, it just strengthens your opinion that an
16 agent is carcinogenic?

17 **A.** Yes, absolutely. That's one of the criteria, that if you
18 see a significant increase in a rare tumor in the animals, that
19 strengthens the evidence that it's an animal carcinogen.

20 **Q.** Okay, and the renal tubular adenoma was actually
21 replicated three times, twice? Three times --

22 **A.** It was replicated in three different studies --

23 **Q.** Okay.

24 **A.** -- and in two different species.

25 **Q.** Okay. That's what -- you said it more eloquently than me.

1 If we can go to our last slide.

2 All right, and so we've already talked about sort of your
3 IARC experience at the beginning, but why don't you just....

4 You stated that you were the Chairman of the Experimental
5 Animal Subgroup, is that right?

6 **A.** Correct.

7 **Q.** And is that the definition of "sufficient" that IARC
8 found?

9 **A.** Yes. A causal relationship was established between the
10 agent and the increased incidence of the malignant neoplasms.

11 **Q.** Okay, great, and I'll just ask two sort of concluding
12 questions. The first one is the Greim review article. Let's
13 just put this to bed once and for all. Did IARC consider the
14 Greim review article?

15 **A.** Absolutely. We reviewed the Greim article, and as this
16 slide shows, it's discussed in section 313 and section 323 of
17 the IARC Monograph.

18 **Q.** Okay, and the Greim review article came to you in the form
19 of a peer-reviewed review article?

20 **A.** It was a peer-reviewed review article. The article --
21 that article came to us at IARC. It indicated that it had
22 additional information, raw data, which were the individual
23 animal tumor tables. It was available on the website, but we
24 could never get the website to work properly while we were at
25 the IARC meeting, although a hard copy was provided to us

1 during the meeting.

2 Q. Okay, and you have since reviewed those -- that data,
3 correct?

4 A. In reaching my opinion, I had the time to go through and
5 review all of -- all of that data that was a supplement to the
6 Greim Paper, as well as the data from the actual study
7 laboratories.

8 Q. And what effect did that review have on your opinion? Did
9 it strengthen, weaken, or have no --

10 A. Oh, that strengthened my opinion, absolutely, because
11 I determined that there were a number of tumors were present in
12 the studies, as the chart showed before; that there were
13 significant increase in multiple tumors in multiple studies.

14 Q. Okay, and I'm just going to hand to you a chart that you
15 have made that shows the Greim paper.

16 Again, let me hand it to you guys.

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

18 If you could pull up 328.

19 And I will -- I will let Monsanto's counsel ask you
20 specific details about this on their cross, but just for the
21 Court's own edification, this is a chart you made where you,
22 once again, on the left-hand column, identified the studies as
23 they are identified in the Greim Paper, just for organizational
24 purposes, right?

25 A. Right.

1 Q. Okay, and then you list in the middle what -- the findings
2 from IARC, right?

3 A. The -- on -- the middle the middle column says "IARC," and
4 those were the tumor sites that -- the studies and the tumor
5 sites that were identified in the IARC monograph are
6 highlighted in the orange-yellow.

7 And then in the others -- the final column, under the CWJ,
8 those are the additional tumor sites I identified after I went
9 through and evaluated all of the data that was in the
10 supplemental tables and the study reports that I was able to
11 get.

12 Q. Okay, excellent. And you are CWJ, Charles William
13 Jameson?

14 A. I am CWJ.

15 Q. Okay.

16 A. And as I look at this, it looks like I made a mistake.
17 The orange says, tumor sites identified by, it should just say
18 "IARC," and then the blue is the ones that I identified by me
19 and IARC.

20 Q. Oh, so you flipped --

21 A. I'm sorry, that's right. I've identified the same sites
22 as --

23 Q. This is correct isn't it?

24 A. That's correct. I'm sorry.

25 Q. All right, okay. So you can put back up our PowerPoint,

1 please. And that's Exhibit 328, that we will also move in to
2 evidence.

3 (Exhibit 328 entered into evidence.)

4 **BY MS. WAGSTAFF:**

5 **Q.** And one last question. The IARC Working Group 112
6 considered the null finding of the AHS when they made their
7 determin- -- when they made it's determination, is that
8 correct?

9 **A.** Yes. The Agricultural Health Study was discussed
10 extensively during the IARC Monograph, and we had the
11 preliminary De Roos Study that had been published concerning
12 the AHS Study, and it turns out that the -- that we -- during
13 IARC Monograph 112, we looked at, I think, a total of four
14 pesticides that had been evaluated in the AHS.

15 And so it was decided that a very detailed description of
16 the Agricultural Health Study would be included in the
17 malathion monograph from -- from Monograph 112, and all of the
18 other monographs, including glyphosate, would refer the reader
19 to the extensive description of the AHS study in the malathion
20 monograph for more details about the evaluation of the AHS.

21 So this slide just says -- gives the pages in the
22 malathion monograph where the AHS Study is discussed, and just
23 an excerpt taken from the malathion monograph, which also
24 applies to all of the other chemicals, including glyphosate,
25 that,

1 "Nondifferential exposure
2 misclassification bias relative risk
3 estimates towards the null in the AHS, and
4 tends to decrease the study precision.
5 This was something that was observed at the
6 IARC meeting. The Working Group considered
7 the AHS to be a highly informative study."

8 So I mean, it was reviewed, felt to be very informative,
9 but even at that time, they were concerned about
10 misclassification.

11 Q. And at the time, again, just to hammer this point home,
12 the IARC Working Group knew that it was a null finding, is that
13 correct?

14 A. Yes.

15 MS. WAGSTAFF: Okay, and the malathion IARC Monograph
16 is in your book, and it's Exhibit 329. And we can move that
17 in.

18 (Exhibit 329 entered into evidence.)

19 And also, I don't know if I did before, but Exhibit 53 is
20 the -- or 57, I'm sorry, is the IARC Monograph.

21 So unless the Court has any other questions for you, I'll
22 pass the witness?

23 THE COURT: Okay, great. Why don't we take our lunch
24 break and return at 1:00 o'clock. Thank you.

25 (Recess taken from 12:05 p.m. until 1:00 p.m.)

CROSS-EXAMINATION

1
2 BY MR. HOLLINGSWORTH

3 Q. Sir, in your evaluation of the glyphosate rodent bioassay
4 data, you did a hazard assessment, right?

5 A. Yes, sir.

6 Q. And, in fact, at your deposition you told me about 18
7 different times that you had done a hazard assessment, isn't
8 that right?

9 A. I don't know the number of times, but I was asked a lot of
10 times.

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

12 Q. In your report for this court, your expert report, sir,
13 you said that there's an important distinction between the term
14 "hazard" and the term "risk," right?

15 A. That's correct.

16 Q. And you said that, quote,

17 Risk is defined as the probability that
18 exposure to a hazard will lead to a
19 negative consequence."

20 True?

21 A. In general terms, that's accurate.

22 Q. And the IARC preamble also defines the distinction between
23 hazard and risk, doesn't it?

24 A. Yes, sir.

25 Q. The preamble states that a cancer hazard is an agent that

1 can cause cancer under some circumstances, while a cancer risk
2 is an estimate of the carcinogenic effects expected from
3 exposure to a cancer hazard; isn't that right?

4 **A.** That sounds right. I don't know if that's the exact
5 wording. I'd have to look at the preamble to see if that was
6 the exact wording.

7 **Q.** Well, you quoted that at page 5 of your report in this
8 litigation, didn't you?

9 **A.** I did quote the IARC preamble, yes.

10 **Q.** Did I misread that?

11 **A.** I don't know. I'd have to have it in front of me to see
12 if you did or not.

13 **Q.** The preamble to the -- to the IARC methodology also states
14 that a cancer hazard is an agent that can cause cancer under
15 some circumstances. True?

16 **A.** Sorry, could you repeat that?

17 **Q.** The preamble, the IARC preamble states that, quote, "A
18 cancer hazard is an agent that can cause cancer under some
19 circumstances." Isn't that right?

20 **A.** That sounds right.

21 **Q.** That's in your report, isn't it?

22 **A.** But again, I'd have to see the preamble to make sure that
23 was the accurate wording.

24 **Q.** Didn't you state that in your report, sir?

25 **A.** I quoted the preamble in my report, but I'd have to have

1 it in front of me to make sure it was the accurate reading.

2 Q. The IARC preamble also states that a cancer risk is an
3 estimate of the carcinogenic effects expected from exposure to
4 a cancer hazard, right?

5 A. I'm sorry, if I may --

6 THE COURT: Why don't you just put it in front of
7 him.

8 THE WITNESS: Yeah, if you could provide me with the
9 preamble, I could verify what you're saying exactly.

10 THE COURT: Rather than leaving everyone to wonder if
11 you're reading it exactly correctly or not.

12 MR. HOLLINGSWORTH: I'm reading from the same page
13 that I think your Honor was reading from.

14 THE COURT: Why don't you put it in front of him, if
15 you want to pursue this line of questioning.

16 THE WITNESS: Thank you.

17 MR. HOLLINGSWORTH: Sure.

18 THE WITNESS: So where in here is the preamble?

19 BY MR. HOLLINGSWORTH

20 Q. It's -- it's referred to in your report at page 5, which
21 is Exhibit 883.

22 A. But in the documents that you just provided to me, where
23 is it located?

24 MS. KLENICKI: It's under tab 883.

25 MS. WAGSTAFF: It's in Volume 2.

1 **THE WITNESS:** I got that now. And what page is it
2 on, please?

3 **MS. KLENICKI:** Five.

4 **THE WITNESS:** Okay. Now, where exactly in this were
5 you referring? I'm sorry.

6 **BY MR. HOLLINGSWORTH**

7 **Q.** I'm referring to the first incomplete paragraph where you
8 state,

9 "The IARC preamble states that a cancer
10 hazard is an agent that can cause cancer
11 under some circumstances."

12 Do you see that?

13 **A.** "...that can cause cancer under some circumstances," yes.

14 **Q.** Yeah, you wrote that in your report in this case, right?

15 **A.** I was quoting from the preamble, that's correct.

16 **Q.** Okay, and it also says that,

17 "A cancer risk is an estimate of the
18 carcinogenic effects expected from exposure
19 to a cancer hazard."

20 Do you see that? Did I read that correctly?

21 **A.** "A cancer risk is an estimate of the carcinogenic effects
22 expected from exposure to a cancer hazard," yes.

23 **Q.** You wrote that, right?

24 **A.** Yes.

25 **Q.** Okay.

1 **A.** Quoting from the IARC preamble.

2 **Q.** Okay, and then your next sentence is that,

3 "The monographs are an exercise in
4 evaluating cancer hazards, despite the
5 historical presence of the word 'risks' in
6 the title."

7 Did you write that?

8 **A.** I wrote that, and that was -- I was told by the people at
9 IARC that the main reason this is included in the preamble
10 IARC Monograph preamble is because they wanted to make sure
11 that people don't look at the monograph as a risk assessment.
12 It is not a risk assessment document. It is a hazard
13 identification document. And that was the reason this sentence
14 was put in the preamble.

15 **Q.** And your report is an exercise in hazard identification or
16 hazard assessment, isn't it?

17 **A.** That's correct, as I state in my report, it's a hazard
18 assessment.

19 **Q.** Now, if you look at the next sentence, sir, on page 5 of
20 your report, the one that follows from the one that we were
21 just discussing, you state, quote, "The distinction between
22 hazard and risk is important."

23 Do you see that?

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

25 **Q.** See that? And then, your report goes on to state that,

1 "The monographs identify cancer
2 hazards, even when risks are very low at
3 current exposure levels."

4 Do you see that?

5 **A.** Yes. It says that, even though risks are very low, but
6 doesn't say that they don't exist.

7 **Q.** Sir, did you write that?

8 **A.** I wrote -- I wrote that the distinction between the hazard
9 and risk is important, and the monographs identify cancer
10 hazards even when risks are very low, at current exposure
11 levels.

12 **Q.** Did you -- did you tell me in your deposition that hazard
13 assessments do not establish the exposure conditions that would
14 pose cancer risks to individuals in their daily lives?

15 **A.** In my deposition I said what, again? I'm sorry.

16 **Q.** Did you tell me that hazard assessments, quote,

17 "...do not establish the exposure
18 conditions that would pose cancer risks to
19 individuals in their daily lives"?

20 **THE COURT:** Why don't you ask him if he believes that
21 now, what's his opinion now.

22 **MR. HOLLINGSWORTH:** Okay, is it -- is it --

23 **THE COURT:** Hold on a sec. If he says something that
24 you believe is contrary to what he said in his deposition, then
25 you can bring up his deposition testimony. That's normally how

1 we do it.

2 **MR. HOLLINGSWORTH:** Sure.

3 **Q.** Is it fair to state that hazard assessments do not
4 establish the exposure conditions that would pose cancer risks
5 to individuals -- to individuals in their daily lives, sir?

6 **A.** No.

7 **Q.** Is it fair to state that risk assessments are different
8 from hazard assessments?

9 **A.** Yes.

10 **Q.** Isn't it true that the determination of what would pose
11 cancer risks to individuals in their daily lives is a formal
12 risk assessment?

13 **A.** I'm sorry, could you repeat that again?

14 **Q.** Isn't it true that the determination of what would pose
15 cancer risk to individuals in their daily lives is a formal
16 risk assessment, according to your report to Congress?

17 **A.** It -- it could be, but hazard assessment also could be
18 that.

19 **Q.** Do you remember when I asked you a question at your
20 deposition and you gave the following answer to the following
21 question?

22 **"QUESTION:** The determination of what would
23 pose cancer risks to individuals in their
24 daily lives is a formal risk assessment,
25 according to your report to Congress, right?

1 **"ANSWER:** That's correct."

2 Do you remember that?

3 **A.** I'd like to see the deposition -- what the deposition
4 says, I'm sorry.

5 **Q.** No, my question is: Do you recall that?

6 **A.** Sitting here right now, no, I don't.

7 **Q.** Mm-hm.

8 **A.** And just -- I've been -- I've had three depositions, and
9 I've been misquoted and things have been taken out of context
10 about what I said so many times, I'm hesitant to confirm that
11 without seeing what my deposition actually said and the context
12 in which it was said.

13 **Q.** Is it fair to state that the determination of what would
14 pose cancer risks to individuals in their daily lives is a
15 formal risk assessment, according to your report to Congress?

16 **A.** I'd have to look at the -- now are you referring to the
17 Report on Carcinogens?

18 **Q.** Yes.

19 **A.** I'd have to look at the section of the Report on
20 Carcinogens to make sure that's what it actually says.

21 **Q.** Okay.

22 **A.** I don't have it rote to memory.

23 **Q.** Do you recall getting -- do you recall giving me the
24 following answer to this question at your deposition?

25 **"QUESTION:** The determination of what would

1 pose cancer risks to individuals in their
2 daily lives is a formal risk assessment,
3 according to your report to Congress, right?

4 **"ANSWER:** That's correct."

5 **A.** Again, I'd like to see the deposition -- well, you know,
6 see my deposition, to see if that's actually reflecting what I
7 said, and also in what context it was said.

8 **Q.** Sir, EPA performed a risk assessment on glyphosate, didn't
9 they?

10 **A.** They have done it many times, yes.

11 **Q.** And EFSA, which is the European Food Safety Agency -- the
12 European health food -- food health administration called EFSA
13 performed a risk assessment on glyphosate, didn't they?

14 **A.** They have performed a risk assessment, yes.

15 **Q.** And you have not done a risk assessment for glyphosate,
16 have you?

17 **A.** I have not been asked to do one yet, no.

18 **Q.** Okay. Sir, you reviewed a total of five dose feed
19 bioassays of glyphosate in mice, and seven dose feed bioassays
20 on glyphosate in rats, true?

21 **A.** Correct.

22 **Q.** And you agree that the dataset of bioassays in rodent --
23 long-term rodent chronic bioassays is more than you usually see
24 for a particular compound, true?

25 **A.** The dataset for glyphosate is very large, yes.

1 Q. In fact, you said it's an unusually large body of
2 toxicology.

3 A. That's true. You usually don't have the luxury of having
4 that many studies to review for one chemical.

5 Q. You described the amount of toxicology data from animal
6 carcinogenicity bioassays as extraordinary, didn't you, in its
7 size?

8 A. I -- I might have used that term, yes.

9 Q. You didn't cite -- you didn't cite anything in your report
10 that says that the mouse system, experimental mouse system, is
11 a good model for determining or predicting human non-Hodgkin's
12 lymphoma, did you?

13 A. That's because the studies that I reviewed were all animal
14 bioassay studies to determine if glyphosate causes cancer.
15 They weren't designed to see if it was relevant to
16 non-Hodgkin's lymphoma in humans. That wasn't the purpose of
17 any of the studies.

18 Q. You didn't cite anything in your report that states that
19 the experimental mouse system is a valid model for predicting
20 human non-Hodgkin's lymphoma, did you, sir?

21 A. That's really an inappropriate question, because that's
22 not what the bioassay studies are -- are performed for. They
23 are performed to determine if the chemical can cause cancer in
24 laboratory animals.

25 Q. Well, sir, that's not my question. My question is: Do

1 you remember when you were deposed in this case, and you gave
2 this answer to the following question?

3 **"QUESTION:** You didn't cite anything in your
4 report in this case, sir, in which you relied
5 on any publication that states that the
6 experimental mouse system is a valid model
7 for predicting non-Hodgkin's lymphoma in
8 humans, did you?

9 **"ANSWER:** No, I did not use any reference to
10 that effect, no."

11 Do you remember giving that answer to that question?

12 **A.** That is my deposition in here? Can I see what my
13 deposition says?

14 **MR. HOLLINGSWORTH:** Can you pull up slide 2, please?

15 **THE COURT:** If you want to ask questions about prior
16 deposition testimony in the way that you're asking them, then
17 you need to give him the full transcript of the deposition,
18 sir.

19 **MR. HOLLINGSWORTH:** He has the full transcript, sir.

20 **THE COURT:** Okay. So why don't you direct him to the
21 page that you're -- that you're asking him about, so that he
22 can take a look at it in context.

23 **MR. HOLLINGSWORTH:** I have the page on the screen,
24 sir.

25 **THE COURT:** Tell him what the page is in the

1 transcript, so that he can look at it in context.

2 **MR. HOLLINGSWORTH:** I believe it's at page 27, lines
3 18 to 24.

4 **JUDGE PETROU:** And what Exhibit number is it in the
5 binders?

6 **MR. HOLLINGSWORTH:** Exhibit 737.

7 **MS. WAGSTAFF:** And which of the three depositions was
8 that?

9 **MR. HOLLINGSWORTH:** This is the deposition taken on
10 September 21st, which is the deposition about his expert
11 witness report.

12 **JUDGE PETROU:** Okay, I lost the page. It's Exhibit
13 737, what page?

14 **MR. HOLLINGSWORTH:** Page 27, at lines 18 to 24, is
15 what I said. I hope that's right.

16 **Q.** Sir, do you see the question that begins, "You didn't cite
17 anything in your report in this case...?"

18 **A.** Yes. That's line 18?

19 **Q.** Yes.

20 **A.** Okay.

21 **Q.** Did you read that question and answer? Sir, do you have
22 my question in mind?

23 **A.** Yes, I see that, but if you read on in my deposition, you
24 ask again,

25 **"QUESTION:** Isn't it current -- isn't the

1 current literature in the case that the mouse
2 system is not a good -- is not a good
3 predictor of lymphoma in humans?"

4 And then my answer to that is:

5 **"ANSWER:** There may be, or may have -- there
6 may have -- may be some publications in the
7 literature to that effect. But again, the
8 purpose of doing these studies is, most, the
9 studies -- the purpose of doing an animal
10 bioassay study is to determine if the
11 chemical can cause cancer in experimental
12 animals, and it is not -- not looking to
13 investigate, does it form a specific kind of
14 tumor that is the same found in humans. At
15 least routinely, that is not the case.

16 "Now, sometimes I think -- the state of
17 the art is that you can develop genetically
18 modified test species, transplant human genes
19 into the animals or something like that, and
20 do some studies that may give you some more
21 information. As to the formation of a cancer
22 in humans based on the special animals, but
23 I'm not familiar with what the -- that
24 research, and I can't speak to that right
25 now, but I know the type of research is being

1 done. I have no idea if there's anything
2 being done with non-Hodgkin's lymphoma, I
3 haven't looked at that."

4 **Q.** Thank you. Thank you, that's very good. Thank you.

5 You claim that glyphosate causes kidney tumors in male
6 CD-1 mice in the 1983 Knezevich study, which has also been
7 referred to as the 1983 Monsanto mouse study, right?

8 **A.** That's correct.

9 **Q.** And the fact that something causes a kidney tumor in a
10 mouse doesn't really tell you anything about whether it would
11 cause non-Hodgkin's lymphoma in humans, isn't that right?

12 **A.** Again, the design of an animal bioassay is not to look --
13 if it causes -- if it's similar to non-Hodgkin's lymphoma in
14 humans.

15 The purpose of an animal bioassay is to study if the
16 chemical can cause cancer in laboratory animals, and if so,
17 then it's biologically plausible that it's a human carcinogen.
18 That's the purpose of why these studies are run. It's not to
19 investigate non-Hodgkin's lymphoma in humans.

20 So that's really an inappropriate question.

21 **Q.** You told me that the fact that something causes a kidney
22 tumor in a mouse doesn't really tell you anything about whether
23 it cause -- whether that relates up to causing non-Hodgkin's
24 lymphoma in humans, right?

25 **A.** I told you that is really not an appropriate question.

1 Q. Okay. The fact that -- that something causes a kidney
2 tumor in a mouse, and its relationship to non-Hodgkin's
3 lymphoma in humans, really has not been investigated, has it?

4 A. I'm not aware that anybody has done any studies to that
5 effect. And to be honest with you, I don't think anybody
6 would -- would try to do that kind of a study, because it's --
7 it's -- it's -- it's not what the data is telling us from the
8 animal bioassay. Its just telling us that it causes kidney
9 tumors in the CD-1 mouse.

10 Q. You're not aware of any publications or research on
11 whether mouse renal tumors are actually predictive of
12 non-Hodgkin's lymphoma in humans, are you?

13 A. I'm not aware of any studies, no, but....

14 Q. You've never published a paper addressing the issue of the
15 relationship of kidney tumors in mice to non-Hodgkin's lymphoma
16 in humans, right?

17 A. No, I have not.

18 Q. And by the way, before IARC, you had never published a
19 study saying that glyphosate causes non-Hodgkin's lymphoma in
20 humans, yourself, had you?

21 A. Prior to IARC, that is correct.

22 Q. And you don't know of any literature, published medical
23 literature in the entire world literature of medical and
24 science activity, that stated before IARC that glyphosate can
25 cause non-Hodgkin's lymphoma in humans, do you?

1 **A.** I'm sorry, could you repeat that?

2 **Q.** You don't know of any literature in the worldwide science
3 and medical literature that states that glyphosate can cause
4 non-Hodgkin's lymphoma in humans, do you?

5 **A.** There's a lot of literature that says glyphosate causes
6 non-Hodgkin's lymphoma in humans.

7 **Q.** You don't know of any study that's been done on whether
8 there's a mechanism that causes kidney tumors in the mouse that
9 is similar to a known mechanism that leads to non-Hodgkin's
10 lymphoma in humans, do you?

11 **A.** No, but these are all -- you know, they're misleading and
12 irrelevant questions, and they -- they really -- that's not how
13 toxicology works, as far as animal bioassay is concerned.

14 As I indicated before, you do the animal bioassay to see
15 if it causes cancer in animals to say that if it does, then
16 it's biologically plausible that it causes cancer in humans.

17 You then take that data and you look at the humans that
18 are exposed to the chemical, or the formulations, in real world
19 situation and real world doses, and see if you see tumor
20 formation in humans.

21 **Q.** You're not --

22 **A.** So that's how -- that's how it works.

23 **Q.** You're not aware of any data or published studies that
24 record what the error rate would be in predicting non-Hodgkin's
25 lymphoma in humans, based on the finding of kidney tumors in a

1 mouse, are you?

2 **A.** That is really not -- that's not really -- I hate to say,
3 that's a ridiculous question, but it's really not an
4 appropriate question to ask, based on the animal bioassay data
5 that you get. That's not the purpose of an animal bioassay.

6 The animal bioassay is performed just to see if a chemical
7 causes cancer in animals, as it leads to the biological
8 plausibility of a human carcinogen, and that's why those types
9 of studies are required by the EPA, by the FDA. That's why
10 the -- the National Toxicology Program spends tens of millions
11 of dollars a year to do animal bioassay studies.

12 They don't do animal bioassay studies to say, oh, we're
13 studying Compound X in mice and rats, so we can say it causes
14 non-Hodgkin's lymphoma in humans.

15 We've run an animal bioassay study to say Compound X
16 causes cancer in animals, therefore, it's probably a human
17 carcinogen. So we need to get busy and look at the populations
18 out there in the world that are exposed to real world -- real
19 world levels, real world concentrations, of Compound X, in the
20 real world situation, if it causes cancer in humans, and if so,
21 where.

22 **Q.** Sir, you've never attended a lecture where there was a
23 discussion of whether or not mouse renal tumors are predictive
24 of non-Hodgkin's lymphoma in humans, have you?

25 **A.** No.

1 Q. You don't recall that the investigators in the Monsanto
2 1983 study on mice were investigating any type of association
3 between the possible formation of kidney tumors in mice and
4 non-Hodgkin's lymphoma, do you?

5 A. No, the purpose of their study was to determine if
6 glyphosate caused cancer in those animals; only if it caused
7 cancer, not a particular kind of cancer or if it was related to
8 any kind of cancer in humans. The purpose of that study was to
9 determine if it caused cancer in humans.

10 Q. You don't think that --

11 A. I mean, in animals, excuse me, in animals.

12 Q. You don't think that any experimental pathologists have
13 ever looked in to the issue of whether or not mouse renal
14 tumors are associated with non-Hodgkin's lymphoma specifically
15 in humans, do you?

16 A. I don't know -- knowing the veterinary pathologists that
17 reviewed the slides, I don't know that anybody would be
18 interested in trying to do anything like that at all.

19 Q. Now, going back to the Knezevich and Hogan study, which is
20 a 1983 mouse study, again, that's the Monsanto study from 1983,
21 and you have actually read that report, have you, sir?

22 A. Yes.

23 Q. You've read the report of the original investigators who
24 were Knezevich, which I'll spell for you at some point, and
25 Hogan. Right?

1 A. Yes.

2 Q. And if you could, sir, and you've -- I already asked you
3 whether you had read the pathology report. You told me in your
4 deposition that you have. And what I'd like to do is read to
5 you from the conclusion of that report, and ask you if -- if
6 you are familiar with this.

7 And I'll put this on the screen. It should be slide 6.

8 My question is whether you recall reading this in the
9 report, sir, at the conclusion part of the Path report, quote,

10 "Neoplastic findings were of the type
11 commonly encountered in mice. Bronchiolar
12 alveolar tumors of the lungs,
13 hepatocellular neoplasms and tumors of the
14 lymphoreticular system accounted for the
15 majority of those encountered. There were
16 no suspected test substance associated
17 trends in the incidence of these tumors or
18 any of the other spontaneously occurring
19 neoplasms."

20 Did you read that when you did your work in preparing your
21 expert report in this case?

22 MS. WAGSTAFF: Counsel, can I have a copy of that
23 document?

24 MS. KLENICKI: It's in your binder at 1524.

25 MS. WAGSTAFF: Okay, so it's at 1524? Thank you.

1 BY MR. HOLLINGSWORTH

2 Q. You read that, right, sir?

3 A. It sounds familiar; but it's been a while since I read
4 that report. So I don't know if it -- if that is accurate or
5 not.

6 Q. You didn't read that report in preparation for your
7 testimony today?

8 A. I read so many things, I don't know that I went back and
9 read that report.

10 Q. So your -- your sense is that what is written on the
11 screen here, that I represented as the conclusion from the
12 study --

13 A. It sounds -- it sounds familiar, yes.

14 Q. Okay. There's also another conclusion from that slide 7,
15 which addresses the issue of the renal tumors. And I'll put
16 that on the screen.

17 I'd like to ask you if this sounds familiar. It is,

18 "The only other neoplasms that
19 occurred" -- that's neoplasms, excuse me --
20 "with any frequency in treated mice were
21 renal tubular adenomas which occurred in
22 males."

23 Are you looking at the screen, sir?

24 A. I'm sorry?

25 Q. (Reading:)

1 "There were present" -- excuse me --
2 "three were present at the high dose and
3 one at the mid-dose level. The
4 distribution of these benign tumors was
5 considered spurious and unrelated to
6 treatment, due to the absence of other
7 renal lesions suggestive of or supportive
8 of an effect on the urinary system."

9 Do you see that?

10 **A.** Yes, that's what this statement says.

11 **Q.** Did you read that in connection with the preparation of
12 your report, sir?

13 **A.** If it was -- if that is what was in the study report, it
14 is.

15 **Q.** So the original investigation -- investigators in this
16 study, who were Dr. Knezevich and Dr. Hogan, made this
17 conclusion after they reviewed all the -- all the tissue slides
18 in this case, right?

19 **A.** That was their conclusion.

20 **Q.** And they were veterinary pathologists, who actually looked
21 under the microscope at tissues. That's what they did for a
22 living, true?

23 **A.** Being veterinary, I assume. I don't know their
24 background. I don't know them personally, but --

25 **Q.** Did you look at their signatures on this report?

1 A. I did.

2 Q. They're signed "D.V.M." aren't they? Doesn't that stand
3 for Doctor of Veterinary Medicine?

4 A. That doesn't necessarily mean they're pathologists.

5 Q. Okay, but anyway, the people who had reviewed this study
6 and prepared this report are the people who signed the report
7 and made that conclusion, and they had reviewed all of the
8 slides from this report, and you didn't review any of the
9 slides, right?

10 A. I didn't have access to any slides, no.

11 Q. You wouldn't be qualified to review the slides, would you?

12 A. Probably not.

13 Q. You didn't look under the microscope at any slide in
14 connection with preparing your expert report here, did you?

15 A. No, I did not.

16 Q. And you disagree with the conclusion of these authors,
17 true?

18 A. That there were adenomas, three adenomas in the high dose?
19 Could I have the slide up here again, please?

20 Q. Well, you disagree with their conclusion that there were
21 no substance-related lesions or tumors, in the opinion of the
22 authors of this report? You disagree with that, true?

23 A. Well, I disagree with that, and -- and I would say the
24 initial EPA review of this data, that was submitted for
25 registration of glyphosate, also disagreed with that. It was

1 from the EPA report when I --

2 Q. Well, we'll get to it?

3 A. -- when I was reviewing the data that I picked up that
4 these renal tumors were -- were at least in the -- in the first
5 of the review of EPA were -- were significant, and because of
6 the rarity of these tumors in the CD-1 mouse. So that's what
7 raised a red flag for me to take a harder look at the
8 incidences.

9 I don't know if you want me to go into the -- the rest of
10 the history that I know about these, but eventually --

11 Q. I'd like to ask the questions, if you don't mind.

12 A. I wasn't answering a question. I was going to tell you
13 about the study.

14 Q. Well, let me -- let me ask you about -- let me ask -- ask
15 you some additional questions about the study first, and then
16 we'll discuss the history and your understanding of it, okay?

17 A. Okay.

18 Q. You said that these renal tumors that you referred to in
19 your report that were -- you say were caused by glyphosate, in
20 the Knezevich and Hogan study, are rare?

21 A. Yes.

22 Q. And you cited to a report called Chandra & Frith (1994)
23 for that proposition, didn't you?

24 A. Mm-hm.

25 Q. And Chandra & Frith is the same -- is the same study that

1 IARC relied on in its report, true?

2 **A.** It was used in the IARC Monograph, that's correct.

3 **Q.** And that's because Chandra & Frith only had one incidence
4 of renal tumors in their entire historical database, right?

5 **A.** Well, no. Basically, I think it's in the IARC Monograph
6 because it was the only peer-reviewed publication we could find
7 that addressed spontaneous tumors incidence in CD-1 mice.

8 **Q.** You remember seeing in the materials you read, though,
9 that there were three renal tubular lesions that had occurred
10 spontaneously in the Biodynamics database, right?

11 Biodynamics is the laboratory that conducted this study,
12 and that's who Knezevich and Hogan worked for, true?

13 **A.** I don't recall. How many studies were there in there?

14 **Q.** I don't know, but you told me in your deposition that you
15 recalled that from some material that you had read, didn't you?

16 **A.** Yeah, but I'd need to see that information again. I don't
17 recall, and I need to know how many different and how many
18 historical animals I've seen. I mean, three -- just saying
19 three -- that could be three out of 10; three out of 20, three
20 out of 500, three out a thousand. I don't know what that
21 number means.

22 **Q.** Do you remember that there was another laboratory that EPA
23 was reviewing at the time called "Hazelton" that had a 7.1
24 incidence of renal tubular lesions in their spontaneous
25 database?

1 A. That sounds about right.

2 Q. Would you agree that historical control data from the lab
3 that -- lab that actually conducted the study is more reliable
4 as a comparator than historical data from multiple different
5 laboratories?

6 A. It -- it would be if -- if it was the same species -- same
7 strain of mouse, you know; and contemporary time, when this
8 study was done.

9 Q. Now, the high-dose animals -- there were three dose
10 groups, a low dose, mid-dose, and high-dose, and a fourth group
11 was a control dose, which received glyphosate. True?

12 A. I'm sorry, say that again?

13 Q. The study typically has four study groups: A control
14 group at zero glyphosate, a low dose, mid-dose, and high-dose
15 group, and that's how the studies are set up, and there are
16 usually 50 or 60 animals in each of those groups. True?

17 A. That's correct. That's what was in this study.

18 Q. And the high-dose group in this study -- the Knezevich
19 study or the Monsanto '83 study -- received 4,841 mgs per kg
20 per day of glyphosate, true?

21 A. Yeah, 30,000 parts per million into the feed, right.

22 Q. That's milligrams per kilogram per day, right?

23 A. That's, yeah, 30,000 parts per feed based --

24 **THE REPORTER:** I'm sorry, could you kindly repeat
25 your answer? I lost it. 3,000 parts per...?

1 **MR. HOLLINGSWORTH:** No, I think he said, 4,841 mgs
2 per kg per day.

3 **THE REPORTER:** Thank you. Sorry, gentlemen.

4 **BY MR. HOLLINGSWORTH**

5 **Q.** Sir, the high-dose group received 4,000, roughly,
6 4,841 milligrams per kilogram per day, right?

7 **A.** I think that's what it equated to.

8 **Q.** And the EPA guidelines for carcinogenicity testing state
9 that the highest dose tested need not exceed 1,000 milligrams
10 per kilogram per day; isn't that right?

11 **A.** Those are the EPA Guidelines, but that -- that is not what
12 you need for an animal bioassay to determine carcinogenicity.

13 They -- I don't know how they came up with that
14 1,000 milligrams per kilogram body weight per day, value, but
15 as in my experience with doing cancer hazard assessments for
16 the National Toxicology Program Report on Carcinogens and IARC,
17 that's -- that number is rather low.

18 **Q.** So you disagree with EPA, and you also disagree with the
19 OECD, which is the international governing body, which also
20 says that 1,000 milligrams per kilogram per day is the absolute
21 upper-limit dose for animal bioassay; isn't that right?

22 **A.** You have to understand that these agencies are regulatory
23 agencies.

24 **Q.** Well, can you answer my question, sir? My question is:
25 You disagree with EPA, and the international regulatory body

1 OECD on this issue, true?

2 **A.** I'm not disagreeing with what they are recommending. It's
3 the reason why they're recommending it. They are recommending
4 that because their responsibility is risk assessment, and by
5 setting a level of a thousand milligrams per kilogram per day,
6 their saying that in the real world situation for risk
7 assessment, you shouldn't go over that, because that wouldn't
8 mimic what you see in a real world situation of human exposure.

9 What we're doing is trying to do a -- in animal bioassay
10 we're trying to determine, can it cause cancer? We're not
11 doing risk assessment, we're doing hazard assessment. So in
12 order to do hazard assessment, you test it at the maximum
13 tolerated dose, which I've discussed earlier.

14 So that's why I disagree, because it doesn't tell you
15 anything about, is it an animal carcinogen, testing it at a
16 thousand milligrams per kilogram per day, that you need to do
17 it at a higher level, because the animals are able to tolerate
18 a higher dose.

19 That's where the difference comes in. It's two different
20 thing. One is doing a risk assessment, one is doing a hazard
21 assessment, and the hazard assessment is important in this
22 particular issue because we're identifying something as an
23 animal carcinogen, and therefore, biologically plausible to be
24 a human carcinogen.

25 And that's the question: Is it -- could it be a human

1 carcinogen? Yes. All right, go to the real world situation.
2 Look at the real world exposures, the people that use the
3 material and are exposed to it in a real world situation, and
4 look to see if there's any cancers in those individuals.

5 And for the purpose of glyphosate formulations, do you see
6 non-Hodgkin's lymphoma?

7 Q. And when you say you see non-Hodgkin's lymphoma, you're
8 basing your opinion on what IARC said, true?

9 A. I'm basing my opinion on the peer-review literature for
10 the epidemiology studies of glyphosate formulations.

11 Q. Okay. Let's go back to these renal tubules, and the mouse
12 study from 1983. You also looked at another study involving
13 the CD-1 mouse, which was Atkinson, and you talk about Atkinson
14 in your report, don't you?

15 A. Correct.

16 Q. Are you aware that the incidence of renal tubules reported
17 in the male mice in that study was 2200? That is, two in the
18 controls, two in the low-dose group, zero in the mid-dose
19 group, and zero in the high-dose group?

20 A. I recall seeing something to that effect. Yes.

21 Q. Did you consider that in your report?

22 A. I considered that incidence, but that was -- that was a
23 negative finding, because the incidence in the concurrent
24 control animals was the same level as that of the treated
25 animals in the low dose. So that really was not an effective

1 to glyphosate.

2 Q. Why didn't you consider that negative finding overall when
3 you evaluated the effect on renal -- the renal cells that you
4 said you saw in the 1983 study?

5 A. It was negative -- it was a negative effect. So it
6 didn't -- I didn't include it because I -- in my report, I only
7 addressed the positive effects that were seen in the animal
8 bioassay studies.

9 Q. All right. Sticking with Atkinson for a moment, that's a
10 1993 study, right?

11 A. Right.

12 Q. And that's done by a sponsor different than Monsanto; a
13 different sponsor, true?

14 A. Okay.

15 Q. And you have stated in your report that the Atkinson study
16 shows that glyphosate causes hemangiosarcomas in male mice,
17 true?

18 A. That's correct. There was a statistically significant
19 positive trend in the formation of the hemangiosarcomas. The
20 incidence in the high-dose animals was 9 percent versus
21 zero percent in the controls. So it didn't quite reach
22 statistical significance, but that's a pretty good increase.

23 And the historical incidence for this, in this strain of
24 mouse -- of mouse, is around 1 percent.

25 Q. Now you're reading from what you called your cheat sheet

1 at the deposition right?

2 A. Right.

3 Q. Uh-huh. Now, to your knowledge, no one has done an
4 investigation to see if there's a correlation between the
5 formation of hemangiosarcomas in laboratory animals and
6 non-Hodgkin's lymphoma in humans, true?

7 A. Well, again, that's an inappropriate question to ask.
8 That wasn't the purpose of the study. The purpose of the study
9 was to see if it caused cancer in laboratory animals as a
10 predictor of cancer in humans, and it wasn't designed to
11 investigate the correlation of hemangiosarcomas in male CD-1
12 mice to non-Hodgkin's lymphoma in humans. So that's not a --
13 that's not an appropriate question to ask.

14 Q. Now, as a toxicologist, you're familiar with Hayes'
15 Principles of Toxicology, right?

16 A. Okay, yes.

17 Q. Hayes says that the hemangiosarcomas are common background
18 neoplasm in mice, doesn't he?

19 A. I'll take your word for it.

20 Q. Now, EPA concluded that these hemangiosarcomas in the
21 Atkinson study were not treatment-related, didn't they?

22 A. The -- I'm sorry, could you repeat that?

23 Q. EPA concluded that the increase in hemangiosarcomas in the
24 Atkinson study from 1993 in CD-1 mice were not
25 treatment-related; isn't that right?

1 A. That's what they concluded, but they did indicate in their
2 report that it was statistically significant for an increase in
3 the trend of formation of these hemangiosarcomas.

4 Q. And did you -- did you look at the consideration that EPA
5 gave to the background incidence of hemangiosarcomas in
6 CD-1 mice, sir?

7 A. Yeah. I think what they said was the incidence in the
8 high-dose males is above the historical upper limit of
9 8 percent for this tumor at the performing laboratory.

10 Q. And -- and JNPR concluded that the hemangiosarcomas were
11 not considered to be caused by the administration of
12 glyphosate. Isn't that right?

13 A. I -- I recall them saying that. Yes.

14 Q. What is JNPR?

15 A. What is JNPR?

16 Q. Yes.

17 A. It's a European regulatory agency, I believe.

18 Q. The authors of the Atkinson Study also concluded that
19 there were no compound-related neoplastic lesions from that
20 study in CD-1 mice. True?

21 A. That's what their conclusions said, yes.

22 Q. So you disagree with EPA the European JNPR and the
23 original study investigators. Right?

24 A. Well, I was asked to look at the data, evaluate the data,
25 as I -- as I do and have done in the the past, and give my

1 opinion. And in my opinion, based on the data, the increased
2 trend in formation of hemangiosarcomas is statistically
3 significant, and therefore a real effect.

4 And therefore, glyphosate caused these tumors in the CD-1
5 male mice in this study.

6 Q. By the way, there was no increased incidence of
7 hemangiosarcoma in the high-dose males in the Knezevich Study;
8 was there?

9 A. It wasn't any reported, no. There may have been one or
10 two -- I don't know -- but it wasn't significant.

11 Q. Knezevich and Atkinson are both 24-month long-term mouse
12 bioassays. And they're in the same strain of mouse: The
13 CD-1 Mouse. Right?

14 A. That's correct.

15 Q. You know that the high-dose group in Knezevich received
16 over four times the dose of glyphosate as the high-dose group
17 in Atkinson. Right?

18 A. That's correct.

19 Q. Now, you claim that glyphosate caused lung cars no -- I
20 should say lung adeno -- a-d-e-n-o -- carcinoma in male CD-1
21 mice in the Wood Study. Right? Are you looking at your cheat
22 sheet?

23 A. I'm looking at my cheat sheet.

24 Q. Okay. The Wood Study was a 2009 study. Right?

25 A. That's correct.

1 Q. And that was from -- that was by a different sponsor than
2 Monsanto, of course.

3 A. Okay.

4 Q. And, now, you agree that no one has designed a study to
5 determine whether an increased incidence of adenocarcinoma in a
6 mouse has any relationship to the formation of non-Hodgkin's
7 lymphoma in people. Right?

8 A. Again, that's not an appropriate question.

9 Q. There's --

10 A. That's not the purpose of the study.

11 Q. You're not -- you're not aware of any published papers
12 investigating the association between lung adenocarcinoma in
13 the CD-1 Mouse, and non-Hodgkin's lymphoma in people?

14 A. I'm not aware that anybody has ever looked at that. No.

15 Q. You know that EPA concluded that adenocarcinoma --
16 adenocarcinomas in this study were not related to glyphosate
17 treatment. True?

18 A. No, but the EPA report did state that there was a
19 statistically significant increase in the trend of the
20 formation of these tumors in the animal. They made that
21 observation in their report.

22 Q. You've testified before that Dr. Portier is your long-time
23 friend and colleague. Right?

24 A. I -- in my professional career at NIHS, I worked with
25 Chris for almost 30 years, yes.

1 Q. And in your deposition, you told me that you defer to
2 Dr. Portier on his use of statistics and biostatistics. Right?

3 A. I -- I would do that because he is a biostatistician, so
4 he is more adept at doing this statistics than I am.

5 Q. You know that Dr. Portier, in his report, says that he
6 cannot attribute the increase of lung adenocarcinomas in this
7 Wood Study to anything other than chance?

8 A. I skimmed through Chris' report. I really don't know what
9 he did and how he came to that conclusion; but again, I --

10 Q. You know that he came to the conclusion, though. Right?

11 A. Pardon me?

12 Q. You know that he came to that conclusion?

13 A. To which conclusion is that?

14 Q. The one I just stated; that he could not attribute the
15 increased in lung adenocarcinomas in the Wood Study to anything
16 other than chance?

17 A. To be honest with you, I don't know if -- if he -- if
18 that's stated in his report or not. Like I said, I skimmed
19 through the report, and I didn't commit it to memory; but it's
20 not unusual for a toxicologist to come to -- you know, to have
21 different evaluations looking at the data. I was asked to look
22 at the data and come to my conclusion. I'm sure Chris was
23 asked to look at the data and come to his conclusion. And our
24 conclusions are in the report.

25 **MR. HOLLINGSWORTH:** Can you pull up Slide 21, please?

1 Thanks, Scott.

2 MS. WAGSTAFF: What tab is this in, in our book?

3 MR. HOLLINGSWORTH: This is Exhibit 737.

4 MS. KLENICKI: That's incorrect. Hold on.

5 MS. WAGSTAFF: Is it in these books you gave us?

6 MS. KLENICKI: It is in the volumes.

7 MR. HOLLINGSWORTH: I don't know.

8 Q. Do you see this statement from Dr. Portier's report?

9 MS. WAGSTAFF: Hang on. Let us -- can you tell us
10 where?

11 MS. KLENICKI: 885.

12 JUDGE PETROU: You said Exhibit 737. That's the
13 deposition transcript, instead of the report.

14 MS. KLENICKI: It's 885.

15 MR. LASKER: It's 532.

16 JUDGE PETROU: I'm not opening it yet. Are we sure?

17 MS. WAGSTAFF: It's the beginning of Volume 1.

18 JUDGE PETROU: It's good exercise with the binders,
19 but --

20 MR. GRIFFIS: 532.

21 BY MR. HOLLINGSWORTH

22 Q. You're familiar with Dr. Portier's report on this issue of
23 lung adenocarcinomas in male mice in the Wood Study. True?

24 A. Like I said, I skimmed through it. I didn't read it in
25 great detail, and I didn't commit it to memory, no.

1 Q. Have you considered his statement that, *In summary, the*
2 *moderate findings in one 24-month study and the negative*
3 *finding when any studies are pooled suggests that the linkage*
4 *between glyphosate and lung adenocarcinomas in male CD-1 mice*
5 *is due to chance?*

6 A. Well, I mean, that's Chris' opinion, in his looking at the
7 data.

8 Q. Yeah.

9 A. But my opinion in looking at the data, and the fact that
10 it gave a statistically significant positive increase in the
11 trend of the formation of these tumors in the CD-1 mice, led me
12 to believe that that was an effect that was caused by
13 glyphosate.

14 Q. So you disagree with Chris, and you disagree with EPA, and
15 you disagree with the study -- original study investigators,
16 and you disagree with EFSA. True?

17 A. I guess I'm not a very agreeable person.

18 Q. Okay. These lung adenocarcinomas were not replicated in
19 any other mouse or rat study; were they?

20 A. The lung tumors? That's correct.

21 Q. And -- but you agree that it would strengthen the data to
22 replicate the results of a study in other experiments. True?

23 A. It would -- it would add a -- to the -- it would add to
24 the data. And, yes, it would strengthen the data if you could
25 have a replication of this effect in different studies.

1 Q. Now, in your direct testimony here in the court today you
2 talked a lot about malignant lymphoma in mice, and how powerful
3 you thought those findings were. Do you recall that?

4 A. Yes, sir.

5 Q. You claim that glyphosate caused lymphoma in three of the
6 five mouse studies that you reviewed. Right?

7 A. That's correct.

8 Q. And you agreed that there is a high background incidence
9 of lymphoma in experimental mice. True?

10 A. Depends on the strain.

11 Q. In CD-1 mice, you told me that there's a fairly high
12 incidence. True?

13 A. It's fairly high.

14 Q. And I told you that the papers show 50 percent incidence
15 of spontaneous tumors in CD-1 mice that is lymphoma. And you
16 said, yeah, it could go that high.

17 A. Well, subsequent to that, I -- after the deposition, I
18 went back and looked for myself what I could find in the
19 literature. And the incidence of the CD-1 mice is not that
20 high.

21 Q. What is it?

22 A. Not 50 percent.

23 What I could find in the published literature was at
24 4 percent.

25 Q. Was what?

1 A. 4 percent in the CD-1 Mouse.

2 Q. Okay. You know Jerry Ward very well. Right?

3 A. I'm very -- I'm familiar with Jerry Ward.

4 Q. He's an experimental pathologist, and you've published
5 with him?

6 A. Yes.

7 Q. And you believe him to be very well known and respected in
8 his field of experimental pathology, which is the study of mice
9 and rats?

10 A. Yes.

11 Q. And you agree with him that lymphomas are among the most
12 common tumors in many strains and stocks of mice, especially
13 those used in safety assessment. Right?

14 A. Would you repeat that again, please?

15 Q. You would agree with Jerry Ward that lymphomas are among
16 the most common tumors in many strains and stocks of mice,
17 especially those used in safety assessment?

18 A. They -- they are common.

19 Q. You're aware of scientific literature which states that
20 the mouse is not a good model for looking at whether a chemical
21 causes lymphoma, because of the high background incidence or
22 spontaneous incidence of lymphoma in experimental mice. True?

23 A. I don't know that I'm familiar with that literature or
24 not, no.

25 Q. It's true that no increased incidence in lymphoma was

1 observed in any of the seven rat studies that you reviewed.

2 True?

3 **A.** No, there were no lymphomas observed in the rats; but
4 there were lymphomas observed in three different studies in two
5 different strains, and in males and females.

6 **Q.** You reported no increased incidence of lymphoma in the
7 mouse study by Knezevich, which is the 1983 mouse study.

8 Right?

9 **A.** Correct.

10 **Q.** And you reported no increased incidence of lymphoma in the
11 Atkinson Study, which you've referred to in connection with
12 other lesions that you say are caused by glyphosate. Right?

13 **A.** Correct.

14 **Q.** And you agree --

15 **A.** It's not that -- I'm sorry. That's not to say there
16 weren't lymphomas in those studies. They just weren't
17 significant.

18 **Q.** You agree that the CD-1, as a strain, has a high
19 spontaneous incidence of lymphoma. True?

20 **A.** The CD-1?

21 **Q.** Yes.

22 **A.** Yeah. Like I said, my search of the literature found that
23 it was around 4 percent.

24 **Q.** Sir, in fact, lymphoma is one of the highest spontaneous
25 tumors observed in CD-1 mice. Isn't that right?

1 A. Okay, but in what context are you saying that?

2 Q. I'm just quoting you from your deposition, sir, in which
3 you said, *I know it's one of the highest ones.*

4 A. Yeah. It can be one of the highest ones, but that might
5 mean it's only, you know, 2 or 3 percent.

6 Q. Well, that's because --

7 A. That doesn't mean that it's 99 percent or 80 percent. It
8 just means relative to other tumors that are spontaneously
9 formed in the animals, it's high.

10 Q. Well, you're just referring to the known variability of
11 lymphoma in the CD-1 Mouse; aren't you? It varies. It comes
12 across in all kinds of different rates, from -- depending on
13 laboratory and what-have-you. Isn't that right?

14 A. The spontaneous rate?

15 Q. Yeah.

16 A. Well, I mean, you get -- you get different rates from
17 different laboratory --

18 Q. You have variability?

19 A. Oh, you have variability. Yes.

20 Q. Yeah. According to your assessment methodology, sir, just
21 because something occurs because of a spontaneous rate is no
22 reason to discount it from being an effect. Right?

23 A. Just because it has a high background level, there's no
24 reason to discount it? Is that what you're saying?

25 Q. Yeah. Didn't you tell me, quote, "Just because

1 something" --

2 **THE COURT:** Before you get in to his deposition
3 testimony, let him provide his actual testimony.

4 **MR. HOLLINGSWORTH:** Sorry.

5 **THE COURT:** You can wait and see if there is -- if
6 you perceive a conflict between the two, in which case we can
7 get into his deposition testimony.

8 **MR. HOLLINGSWORTH:** Okay. There's a rule on that.
9 I'm trying to follow it, but I slip up sometimes.

10 **THE COURT:** You're not.

11 You want to ask the question again?

12 **MR. HOLLINGSWORTH:** Yes.

13 **Q.** According to your hazard assessment methodology, sir, just
14 because something occurs because of a spontaneous rate is no
15 reason to discount it from being an effect?

16 **A.** That's correct. What I was meaning by that is: Just
17 because an animal has a high spontaneous rate of a particular
18 tumor, if you see a significant increase in that particular
19 type of tumor in your study, and that increase is well above
20 the historical rate, then it's an effect of the chemical, and
21 it's not a reason to discount the study just because there's a
22 high historical incidence of that particular tumor. What the
23 study is telling you is that the chemical is causing an
24 increase in the number of spontaneous tumors that the animal
25 sees, so it is causing additional cancer in the animal.

1 Q. There's no literature anywhere on the planet that says,
2 quote, "Just about because something occurs because of a
3 spontaneous rate is no reason to discount it from being an
4 effect," is there?

5 A. I don't know that I understand the question.

6 Q. Can you point me to any literature anywhere on the planet
7 that says what you say, which is, quote, "Just because
8 something occurs because of a spontaneous rate is no reason to
9 discount it from being an effect"?

10 A. Okay. Well, then, again, I feel like I'm being taken out
11 of context, because what I was saying was just what I said:
12 Just because an animal -- the animals you are looking at have a
13 high spontaneous rate of a particular tumor, and you see a
14 significant increase in the incidence of that tumor when you
15 treat it with a particular chemical, there's no reason to
16 discount it because it has a high -- because it -- because it
17 has a high spontaneous rate, because that significant increase
18 in that tumor is due to the treatment with the chemical.

19 Q. EPA and EFSA and the original investigators in the
20 CD-1 Mouse study had all concluded that there was no effect
21 from treatment with glyphosate in connection with those
22 lymphomas. True?

23 A. That's what they said in their reports.

24 Q. Let me ask you about the Sugimoto -- S-u-g-i-m-o-t-o --
25 Sugimoto 1997 mouse study which was done by Arysta Chemical,

1 A-r-y-s-t-a. That's not Monsanto. That's a different sponsor,
2 called "Arysta." Do you have that in mind, sir?

3 A. Yes.

4 Q. You claim that glyphosate caused lymphoma in the CD-1 mice
5 in that study, too. Right?

6 A. That's correct.

7 Q. And the Sugimoto Study investigators concluded that there
8 were no compound-related neoplastic or oncogenic --
9 o-n-c-o-g-e-n-i-c -- oncogenic affects from the administration
10 of glyphosate in this study. True?

11 A. I'm sorry. I was reading something when you -- could you
12 repeat the question?

13 Q. Sure. The original Sugimoto Study investigator -- those
14 are the guys who are actually experimental pathologists, who
15 look under the microscopes at all of these tissues. Right?
16 Right?

17 A. I --

18 Q. Those are the original investigators?

19 A. I have no idea what their background is or what they do.

20 Q. Did you read their report?

21 A. I read their report.

22 Q. Did you see what the authors said about the report from
23 the path. study in the report?

24 A. Yes.

25 Q. They concluded that there were no compound-related

1 neoplastic or oncogenic affects from the administration of
2 glyphosate in the study. Right?

3 **A.** That's what they said in their report.

4 **Q.** And there were no conclusions from EFSA or EPA that were
5 any different than the original authors' conclusions about the
6 Sugimoto Study. Right?

7 **A.** To the best of my recollection, that's accurate.

8 **Q.** Did you consider the EPA's evaluation of Sugimoto when you
9 did your opinion in this case?

10 **A.** Yes, I read their report.

11 **Q.** So you disagree with EPA, and you also disagree with the
12 European health/safety agency known as "EFSA," E-F-S-A. Right?

13 **A.** Evidently.

14 **Q.** Now, EFSA reported an historical control incidence for
15 this laboratory that conducted the Sugimoto Study as between 4
16 and 19 percent. True?

17 **A.** What tumor are you referring to?

18 **Q.** I'm referring to the Sugimoto Study.

19 **A.** Yeah, but what particular tumor site are you referring to?

20 **Q.** I'm referring to the -- to the lymphomas.

21 **A.** Oh, to the lymphomas. Okay.

22 **Q.** Yeah. Remember, I asked you this in your deposition.

23 **A.** I don't remember being asked that in deposition, but okay.

24 **Q.** Okay. All right.

25 **A.** I had three depositions, so it's hard to remember which --

1 Q. You think that the historical controls from a particular
2 laboratory that has conducted or evaluated a study are very
3 important considerations when you evaluate the results of any
4 bioassay study. True?

5 A. That's correct.

6 Q. Historical controls aid in the evaluation of the data, in
7 your view. True?

8 A. It aids in evaluation, yes, but the concurrent controls
9 are the most appropriate ones to use for comparison.

10 Q. Okay. Let me go back to the Wood Study again. That's the
11 Nufarm Study, N-u-f-a-r-m. Nufarm was the sponsor of this
12 study in 2009. Do you have that in mind, sir?

13 A. Yes, sir.

14 Q. Have you got it on your cheat sheet there?

15 A. Yes, sir.

16 Q. You claim that glyphosate induced lymphoma in the Wood
17 CD-1 Mouse study. Right?

18 A. Yes, sir.

19 Q. But you said that you did not have access to the full
20 study of the investigators?

21 A. For this particular one, I do not believe I have the full
22 study report.

23 Q. And in this study you relied heavily on Greim. True?

24 A. I relied on Greim, and I also relied on EPA evaluation of
25 the study.

1 Q. Now, the original investigators -- the experimental
2 pathologists who put together this report -- said that there
3 was no compound-related effect, whatsoever, in this study with
4 respect to oncogenic or neoplastic affects. True?

5 A. That's -- as I recall, that's what -- the information that
6 I got from the study. That's what they said.

7 Q. And EPA and EFSA specifically considered the lymphoma
8 findings in the male mice in this study, and they did not
9 consider them to be treatment-related because of the high
10 background incidence of lymphoma generally in this mouse
11 strain. True?

12 A. I know they did not -- they discounted these tumors, but I
13 don't remember that they specifically said it was because of
14 the high background incidence, but I just don't recall.

15 Q. Do you disagree with the EPA and EFSA in that case?

16 A. Evidently I do, yes; but if you look at the incidence of
17 the malignant lymphoma, it was zero in the controls. That's
18 the interesting thing about these lymphomas in the CD-1 mice.

19 In the Wood Study, there were -- if it has a high
20 spontaneous incidence, then why, in the control animals, don't
21 we see some lymphomas? And you don't.

22 And then, well, in the Sugimoto Study there were two in
23 the -- in the control --

24 Q. Sir, I discussed with you --

25 A. -- but --

1 Q. -- that the background incidence or spontaneous rate of
2 malignant lymphoma in the CD-1 mice historically at the
3 Wood Laboratory was 12 percent in males. True?

4 A. I don't recall -- no -- seeing that number.

5 Q. Did you -- you don't recall seeing that number?

6 A. No. Like I said, I went to the published, peer-reviewed
7 literature to look at what was reported in there for the
8 spontaneous incidence of malignant lymphoma in CD-1 -- male
9 CD-1 mice, and --

10 Q. You came back with 3 percent? Is that what you said?

11 A. Came back to find was 4.

12 **THE COURT:** Don't interrupt the witness.

13 **MR. HOLLINGSWORTH:** Sorry.

14 **THE WITNESS:** 4 percent is what I saw.

15 **BY MR. HOLLINGSWORTH**

16 Q. 4 percent?

17 Did you read the EFSA report on the Wood Study, in which
18 they said the incidence of spontaneous lymphoma ranged from zero
19 to 30 percent?

20 A. Was that for the CD-1 Mouse.

21 Q. For the CD-1 Mouse?

22 A. I don't know that. I don't remember that, at all. No.
23 That sounds very high for the CD-1 Mouse.

24 Q. Okay.

25 A. If you say Swiss Mouse, I would agree, maybe; but not the

1 CD-1 Mouse.

2 Q. Okay. Is it fair to state that there's a high variability
3 of lymphoma -- spontaneous lymphoma -- in CD-1 mice generally?

4 A. A high variability? I -- I -- that's possible. I don't
5 know that I know that for a fact.

6 And like I said, I relied on what was published in the
7 literature, and that said that the average was around
8 4 percent.

9 Q. Did you read it study by -- or the review article by your
10 friend, Jerry Ward, on the issue of lymphoma -- lymphomas in
11 mice?

12 A. I probably did, but I don't remember. Sorry.

13 Q. Let's turn to rats. You made a claim in your
14 Expert Report that glyphosate causes pancreatic islet cell --
15 that's i-s-l-e-t -- cell tumors in Sprague, S-p-r-a-g-u-e,
16 Dawley, D-a-w-l-e-y, rats. Sprague-Dawley rats. True?

17 A. Which study are you referring to?

18 Q. I'm referring to the -- sorry. I'm referring to the Stout
19 and Ruecker Study.

20 A. Okay.

21 Q. I believe that's 1990. And the sponsor was Monsanto. Do
22 you have in mind?

23 A. Yes, sir.

24 Q. Now, you're not aware of anyone doing any research about
25 the connection between pancreatic islet cell tumors in rats,

1 and non-Hodgkin's lymphoma in humans; are you?

2 **A.** Again, that's really not an appropriate question, because
3 the purpose of the bioassay study is to see if it causes cancer
4 in laboratory animals. And it wasn't designed to investigate
5 if there was a relationship between the tumors you see in
6 animals, and non-Hodgkin's lymphoma in humans. So that's
7 really not an appropriate question to ask.

8 **Q.** You're not aware of anyone who's published papers that a
9 Court could rely on, saying that pancreatic islet cell tumors
10 in rats are predictive of non-Hodgkin's lymphoma in humans?

11 **A.** I'm not aware of any -- anybody investigating that or
12 publishing on that. No.

13 **Q.** Now, you also said that there was no statistically
14 significant trend in the incidence of pancreatic islet tumors
15 in this 1990 rat study by Monsanto. True?

16 **A.** I also reported what? I'm sorry.

17 **Q.** That there was no statistically significant trend in the
18 incidence of these tumors that you claim were caused by
19 glyphosate. True?

20 **A.** No, there was no statistically increase in trends; but
21 there was a statistically significant increase in the incidence
22 in the log₁₀ animals.

23 (Reporter requests clarification.)

24 **THE WITNESS:** Low. Yeah. L-o-w. Low-dose animals.
25 I'm speaking too fast.

1 What I will say: That for the Stout Study in my
2 investigation -- in my review of the EPA report, the EPA
3 performed an additional analysis on the -- on the animals where
4 they excluded the animals that died or were killed before Week
5 54 or 55. And when they did that analysis, the islet-cell
6 adenomas were statistically significant in the low-dose and the
7 high-dose. So when the EPA did their analysis, they found
8 statistically significant increase in both low-dose and
9 high-dose animals.

10 And I'll also point out that the incidence of the
11 islet-cell adenoma in the low-dose and the high-dose was almost
12 twice that seen in historical controls.

13 **Q.** Sir, didn't you report in your report that there's no
14 statistically significant trend in the incidence of these
15 pancreatic islet-cell tumors in rats?

16 **A.** There's no significant increase in the trend; but there's
17 a statistically significant increase in the low-dose and the
18 high-dose animals in here, and that's a very significant
19 finding.

20 **Q.** You also concede that there was no apparent progression to
21 carcinoma, either. Right?

22 **A.** Well, that's -- that's an observation I made, yeah, that
23 they -- they didn't see any carcinomas in there; that they were
24 all adenomas; but it's not --

25 **Q.** Well, that's not --

1 A. Adenomas progress -- you know, eventually could progress
2 to the malignant.

3 Q. That's not even correct, either, sir; is it?

4 A. What's that?

5 Q. There was a carcinoma in the control group.

6 A. But that's what they found; that what -- ha -- but that's
7 in the control group. I was talking about the treated animals.

8 Q. Yeah. Okay.

9 A. I was talking -- I mean, you've got to look at how you
10 evaluate the data. You compare. You're looking at the
11 formation of the adenomas in the treated animals, and if the
12 adenomas in the treated animals then progress to a carcinoma.

13 Q. An adenoma's a benign tumor. Right?

14 A. It's a nonmalignant tumor. That's right.

15 Q. And there was a carcinoma in the control group in this
16 study?

17 JUDGE PETROU: I'm having a very hard time following,
18 because you're constantly speaking over.

19 MR. HOLLINGSWORTH: Okay.

20 THE WITNESS: Sorry.

21 THE COURT: It's his fault.

22 THE WITNESS: Yeah.

23 THE COURT: You want to try to ask your question
24 again?

25

1 BY MR. HOLLINGSWORTH

2 Q. There was a carcinoma in the control group in this -- in
3 this particular study; wasn't there?

4 A. One carcinoma. Yes.

5 Q. Yeah. You didn't report that in your report; did you?

6 A. I'd have to look at my report. I may have. I may not
7 have. I don't know. But it was -- you know, it was -- it was
8 a -- I reported the adenomas that were found. And it could be
9 that since there was only one carcinoma in the control group, I
10 didn't -- I didn't put it down.

11 Q. Okay.

12 A. But that didn't have an effect on my evaluation.

13 Q. You also said in your deposition that there's no
14 dose-response in the incidence of pancreatic-islet-cell tumors
15 in the 1990 study. True?

16 A. No dose-response. Did I say no dose-response, or did I
17 say there was no trend in -- in dose? I'd have to look at my
18 report to see exactly what I said.

19 Q. Do you remember when I asked you this question --

20 *Question:*

21 -- and you gave the following answer?

22 *Question: There was also no -- no dose-response that you*
23 *could observe in these pancreatic-islet-cell adenomas that you*
24 *saw in the treated groups. True?*

25 *Answer: No. It's not a true dose-response.*

1 Do you remember that?

2 **MS. WAGSTAFF:** Counsel, can I get the cite for that?

3 **MR. HOLLINGSWORTH:** Sure 198. 198:3-19.

4 **MS. WAGSTAFF:** Which deposition?

5 **MR. HOLLINGSWORTH:** Sorry. It's the expert
6 deposition. It's the one that was taken in September.

7 **Q.** Sir, let me ask you this. EPA did not consider the
8 pancreatic-islet-cell tumors to be a true carcinogenic effect;
9 did they?

10 **A.** That's -- in their report, that's what they state.

11 **Q.** And neither did EFSA; the European regulatory agency?

12 **A.** That's accurate.

13 **Q.** So you disagree with EFSA, you disagree with EPA, and you
14 disagree with the original authors of this study, who were
15 Stout and Ruecker. Ruecker's spelled R-u-e-c-k-e-r. Right?

16 **A.** I guess that that's accurate.

17 **MR. HOLLINGSWORTH:** Are we okay to keep going, or
18 should we take a break?

19 **JUDGE PETROU:** How much more do you have?

20 **MR. HOLLINGSWORTH:** I have a little more.

21 **JUDGE PETROU:** Sounds like it's time to take a break.

22 **MR. HOLLINGSWORTH:** I'd say a half an hour.

23 **THE COURT:** Okay. Let's take a break. We'll be back
24 at 2:30.

25 **THE CLERK:** Court is in recess.

1 (Recess taken from 2:17 p.m. until 2:33 p.m.)

2 **THE COURT:** All right. Ready to resume?

3 **MR. HOLLINGSWORTH:** Yes, Your Honor.

4 **BY MR. HOLLINGSWORTH**

5 **Q.** Dr. Jameson, do you have in front of you in the tabbed
6 binder Exhibit 873?

7 **A.** Yes, sir.

8 **Q.** This is the revised glyphosate-issue paper, *Valuation of*
9 *Carcinogenic Potential*, by the Office of Pesticide Programs at
10 EPA, December 12th, 2017. And this is about glyphosate. Do
11 you have that? Do you see that?

12 **A.** Yes, sir.

13 **Q.** If you looked at page 88, EPA is discussing the same Wood
14 Study that you and I were discussing before the break. That's
15 the Wood Study that was conducted in 2009 by a different
16 registrant than Monsanto. Do you recall our discussion about
17 Wood?

18 **A.** Yes.

19 **Q.** And our discussion about malignant lymphoma?

20 **A.** Yes.

21 **Q.** And the background incidence of malignant lymphoma?

22 **A.** Yes.

23 **Q.** Do you see the last two sentences -- sorry -- the last
24 three sentences on this page, which is page 88 of 216 in
25 Exhibit 873?

1 A. Okay.

2 Q. Where it states as follows: *Historical control data have*
3 *been submitted from the same testing laboratory for 10 studies*
4 *of similar duration?* Do you see that?

5 A. I'm sorry. Where does it say about --

6 Q. I'm looking at this paragraph titled "malignant Lymphoma."

7 A. Okay.

8 Q. The last three sentences of that paragraph start out with
9 the --

10 THE COURT: I think you're referring to the last four
11 sentences of the paragraph, which is why he's confused.

12 MR. HOLLINGSWORTH: Oh, excuse me. Sorry.

13 THE WITNESS: Okay. I see.

14 MR. HOLLINGSWORTH: Yeah. The last four sentences.

15 Thank you, Your Honor.

16 Q. The first one says, *Historical control data have been*
17 *submitted from the same testing laboratory for 10 studies of*
18 *similar duration.* Do you see that?

19 A. Okay.

20 Q. And it goes on and says, *These data were generated within*
21 *approximately five years of the Wood Study that we're -- we*
22 *have been referring to.*

23 Do you see that sentence?

24 A. Okay. Mm-hm.

25 Q. And the historical control rate -- range EPA reports was

1 zero to 32 percent. Do you see that?

2 **A.** I see that.

3 **Q.** And the last sentence is, *All observed incidences of this*
4 *tumor type were within the historical control range.* Do you
5 see that?

6 **A.** Yes, I see that.

7 **Q.** And that last sentence refers to EPA's evaluation of the
8 Wood 2009 study, in which you claim that lymphomas were caused
9 by glyphosate. True?

10 **A.** Okay.

11 **Q.** So you disagree with EPA?

12 **A.** Well, in this paragraph I'm -- I mean, just a quick read
13 through, but I didn't see. Are they referring to CD-1 Mouse
14 here? Mice here? I don't see that they refer to it
15 specifically for CD-1 mice.

16 **MS. KLENICKI:** It's at the top of the page.

17 **BY MR. HOLLINGSWORTH**

18 **Q.** *In a feeding study, CD-1 mice --*

19 Do you see that; the first sentence?

20 **A.** Well, yeah, they said, *In a feeding study, CD-1 mice*
21 *received glyphosate,* in the first paragraph; but talking about
22 the malignant lymphomas, they were just saying that the data
23 were -- historical and control data were submitted from the
24 same testing laboratory for 10 studies of similar duration, but
25 it doesn't say that it was specific for the CD-1 Mouse, so --

1 Q. Well, the whole study is about CD-1 Mouse. And the whole
2 section of this report is about CD-1 Mouse.

3 Are you suggesting that they're talking about a different
4 strain of mice than CD-1 Mouse?

5 A. They didn't say specifically when they're referring to the
6 historic controls for malignant lymphomas that it's for CD-1
7 mice.

8 Q. The historical control range of malignant lymphoma they
9 refer to is a range of zero to 32 percent. Do you see that?

10 A. For malignant lymphoma in those 10 laboratories.

11 Q. Yeah.

12 A. But it doesn't say that it's for the CD-1 Mouse.

13 Q. No, it doesn't. Okay.

14 A. And all -- and what I'm saying is I went to the
15 peer-reviewed literature; found the peer-reviewed article. And
16 in that article specifically on spontaneous tumors in
17 CD-1 mice, it says 4 percent is the historical rate. So that's
18 where I got my figure from.

19 Q. Sir, do you remember when we were -- when we were deposing
20 you about the Supplemental Report that you had, and I asked you
21 some questions about the malathion section of the IARC
22 Monograph that you worked on involving --

23 **THE COURT:** Why don't you ask him a question? And
24 then if his question is inconsistent with his deposition
25 testimony, you can ask him about his deposition testimony.

1 Okay?

2 **MR. HOLLINGSWORTH:** Sure.

3 **Q.** Dr. Jameson, I'd like to turn to the mouse -- the
4 malathion section of the Monograph 112. Do you have that in
5 mind?

6 **A.** Yes, sir.

7 **Q.** Do you remember when we --

8 **JUDGE PETROU:** I'm sorry. What's the exhibit number?

9 **MS. KLENICKI:** 1030.

10 **THE WITNESS:** It's in this book. Where in this book
11 is it?

12 **JUDGE PETROU:** It's Volume 2, sort of halfway through
13 Tab 1030.

14 **THE WITNESS:** 1030? Thank you.

15 **BY MR. HOLLINGSWORTH**

16 **Q.** Now, this is a discussion of the Agricultural Health
17 Study.

18 **A.** Okay.

19 **Q.** And I believe you referred to this in your direct
20 testimony here today. True?

21 **A.** I'd refer to sections of this. Are you referring to a
22 particular page in this?

23 **Q.** No, I'm not yet.

24 **A.** Okay.

25 **Q.** This -- this is -- this is the malathion subsection of

1 Monograph 112. That included --

2 **A.** Yeah. That's what it looks like. Yes.

3 **Q.** And it included three or four other subsections on
4 different chemicals, one of which was glyphosate. Right?

5 **A.** In Monograph 112 there were --

6 **Q.** Yeah.

7 **A.** -- there was a monograph written on glyphosate. That's
8 correct.

9 **Q.** And the glyphosate section of that monograph refers the
10 readers to the section on malathion for a discussion of the
11 Agricultural Health Study?

12 **A.** That's correct. And I think, as I mentioned earlier
13 today, the reason for doing that is the Monograph Working Group
14 wanted to make sure there was a thorough and complete
15 description of the AHS Study available to the reader; but it
16 was so long, they didn't want to put it in every monograph.

17 And since there were three or four chemicals that we
18 reviewed at that time that were also included in the AHS, they
19 wrote the detailed description in the malathion monograph, and
20 then in the other monographs, including the glyphosate
21 monograph, and the epi section. When they discussed the AHS
22 results, they referred to the malathion monograph for a
23 detailed description of what the AHS Study was.

24 **Q.** And the -- the group -- the IARC group was -- what? -- 16
25 people?

1 A. I -- I don't remember the exact number, but it's usually
2 around 16 or 17 individuals that are actual Working Group
3 members, yes.

4 Q. And you all voted to approve this section on the
5 Agricultural Health Study from the malathion portion of the
6 monograph?

7 A. That's correct. Yes.

8 Q. And you intended that what you said about the Agricultural
9 Health Study in the malathion section of the monograph should
10 apply with equal weight to glyphosate. True?

11 A. In the description of the AHS Study, not -- but I mean,
12 the results for glyphosate and the AHS Study are contained in
13 the glyphosate monograph.

14 Q. So the general comments made about the AHS Study in this
15 portion of the malathion monograph are -- you intended to be
16 applicable equally to glyphosate?

17 A. That's correct.

18 Q. And the group said that it considered the AHS Study to be
19 highly informative. Right?

20 A. Page, please?

21 Q. Page 21 of the malathion monograph?

22 **JUDGE PETROU:** Before you get to that question,
23 Doctor, could you turn to page 9 of this Exhibit?

24 **THE WITNESS:** Okay.

25 **JUDGE PETROU:** And that's where it begins talking

1 about the Agricultural Health Study. Correct?

2 **THE WITNESS:** That's correct.

3 **JUDGE PETROU:** And it keeps going toward page 10, and
4 then to page 11. So my question is: On page 11, about halfway
5 through the column on the left, there's a paragraph in
6 brackets. And also in the right-hand column, halfway through,
7 there's another paragraph in brackets. It says in
8 "Conclusion."

9 My question is simply: What do these bracketed paragraphs
10 mean? Were these part of the Final Report, or --

11 **THE WITNESS:** These -- these are when a bracketed
12 comment is included in an IARC Monograph, those are -- are
13 meant to note that those are the conclusions or the
14 observations of the Working Group, and not part of the
15 publication or the paper that they were describing at the time.

16 So this conclusion that's on the right-hand column of
17 page 11 that's in brackets, "Conclusion of the Working Group,"
18 noted that the exposure assessment methods used in the mouse
19 studies were relatively crude. That is the Working Group
20 saying that.

21 **JUDGE PETROU:** Commenting.

22 **THE WITNESS:** So that's the purpose of bracketed.

23 And if you look at the at all of the monographs, there are
24 bracketed statements all through it. Those are to designate
25 this is what the Working Group was saying, and not what was

1 contained in the actual paper.

2 **JUDGE PETROU:** That's helpful. Thank you.

3 **THE WITNESS:** Okay.

4 **BY MR. HOLLINGSWORTH**

5 **Q.** If you go to -- if you stay on page 9, I'm -- I don't have
6 time to go through this whole thing, but I want to ask you
7 about a couple of things the Working Group said that I'm
8 looking at the first full paragraph in the right column.
9 *Quote, Great efforts were made in the Agricultural Health Study*
10 *to assess exposure among agricultural pesticide applicators and*
11 *their spouses.* Do you see that sentence?

12 **A.** Yes, sir.

13 **Q.** Did I read that correctly?

14 **A.** Yes, sir.

15 **Q.** And in the next sentence says, *These questionnaires and*
16 *algorithms have been extensively described and have undergone*
17 *several tests for reliability and accuracy that have provided*
18 *considerable insight into the quality of this exposure*
19 *assessment.* Do you see that?

20 **A.** Yes, sir.

21 **Q.** And if you look at page 11, there is a section that says,
22 at the bottom on the left-hand column, in which it's stated,
23 *Almost all of the studies --*

24 Do you see that sentence?

25 **A.** Yes.

1 Q. *Almost all of the studies relied on self-reported data,*
2 *which, as discussed above, is reasonably reliable and valid*
3 *when applicators are reporting their own use. Do you see that?*

4 A. Yes.

5 Q. *And then it goes on to say, But may not be suitable for*
6 *spouses or other farmworkers, particularly those exposed by*
7 *re-entry. Do you see that?*

8 A. Yes.

9 Q. *And the next sentence says, Proxy respondents are unlikely*
10 *to know the details of use of specific pesticides by next of*
11 *kin. Do you see that?*

12 A. Yes.

13 Q. Did you vote to approve that?

14 A. To -- to -- yes. I voted for the wording of this section
15 of the monograph. Yes.

16 Q. Do you see the sentence that -- it's -- that -- where IARC
17 says, *Apart from the AHS*, which is the first full paragraph in
18 the right-hand column on page 11?

19 A. Yes.

20 Q. *Apart from the AHS, few of the studies included expert*
21 *review of the data, or performed validity or reliability*
22 *studies. Do you see that?*

23 A. Okay.

24 Q. That paragraph is suggesting that the other studies -- the
25 case-control studies, unlike the AHS -- did not include,

1 necessarily, expert review of the data, nor did they perform
2 validity or reliability studies, such as AHS had done. True?

3 **A.** I don't know that that's what it's referring to, or not.

4 **Q.** Okay.

5 **A.** It doesn't specifically say the case-control studies. It
6 just says few of the studies included expert review.

7 **Q.** Okay.

8 **A.** And to be honest with you, I think it's referring to some
9 of the studies in -- that have been published as a result of
10 the AHS Study.

11 And I would also point out that the bracketed comments
12 that are included here, which are the exact -- are actually the
13 comments from the Working Group. I read it previously, in
14 response to Your Honor's question.

15 *In conclusion -- and this is the statement of the Working*
16 *Group. In conclusion, the Working Group noted that the*
17 *exposure assessment methods used in most studies were*
18 *relatively crude.*

19 So, I mean -- and as I recall, in the discussions we had
20 about the AHS Study at IARC, there was a lot of concern over
21 exposure assessment and misclassification, and the weakness
22 that that imparted to the study. And there were a lot of
23 people that were concerned about that adequacy of the study,
24 even at this point.

25 **Q.** Well, that sentence you just read doesn't refer to the

1 AHS; does it?

2 **A.** Well, it's in the AHS section. I mean, it's in the
3 section discussing the AHS Study.

4 **Q.** It refers to "most studies." It doesn't say it -- that
5 the AHS specifically was crude; does it?

6 **A.** No. I think it's referring to the AHS Study there.

7 **Q.** Okay. If you'll turn to page 15, in the right-hand column
8 at the top of the page, I think it's the second sentence.

9 **THE COURT:** Could I ask a follow-up on your last
10 question before you move on?

11 I'm just looking at page 9, and 10 and 11 --

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

13 **THE COURT:** -- of the monograph; the section on
14 Malathion.

15 On page 9 begins a section in which you discuss the
16 Agricultural Health Study. Page 11 begins a section in which
17 you discuss other epidemiological studies.

18 **THE WITNESS:** Correct.

19 **THE COURT:** So I assume that the --

20 **THE WITNESS:** Oh, oh, oh. I'm so sorry. Yes.

21 **THE COURT:** -- I assume that the "In conclusion"
22 sentence is about the other epidemiological study.

23 **THE WITNESS:** I stand corrected. I'm so sorry. Yes,
24 I misread that. I'm sorry. Absolutely right. Thank you for
25 catching that. I didn't mean to mislead.

1 BY MR. HOLLINGSWORTH

2 Q. If you'll go back to page 15, sir, I was referring to the
3 right-hand column at the top of the page. I was interested in
4 this sentence, which is, *The AHS being a cohort study --*

5 Do you see that?

6 A. Yes.

7 Q. -- *avoids recall bias.*

8 Do you see that?

9 A. Yes.

10 Q. Did I read that correctly?

11 A. Yes.

12 Q. *Since exposure was obtained before the onset of cancer.*

13 Do you see that?

14 A. Yes.

15 Q. And it goes on to say, *Misclassification of pesticide*
16 *exposure in the AHS cannot, however, be excluded, because*
17 *exposure was retrospective and self-reported (as is as is*
18 *typical for most case-control studies).*

19 Do you see that?

20 A. Yes.

21 Q. *But the error would be nondifferential, and in most*
22 *scenarios would not inflate risk estimates.*

23 Do you see that?

24 A. Yes.

25 Q. Did you vote to approve that?

1 **A.** Yes. I think the beginning sentence that you read, *The*
2 *AHS, being the cohort study, avoids recall bias.*

3 Since exposure is obtained before the onset of cancer, it
4 precludes recall bias of -- of people saying that -- people
5 that have a disease when they're first recruited recalling that
6 they were exposed to a pesticide, as opposed to, in a cohort
7 study, you're recruiting people who have no disease; and so
8 therefore, their recall wouldn't be biased by the fact that
9 they already have the disease.

10 **Q.** Now, if you look at page 21, sir, I'm not going to ask
11 many more questions about this, but I want to ask about page
12 21, please, the bottom of the left-hand column.

13 **A.** Okay.

14 **Q.** And if you look at the sentence that starts, *For*
15 *individuals in the AHS who did not complete a Phase 2*
16 *re-interview.*

17 **A.** I'm sorry. On the left-hand column?

18 **Q.** Did I say "right"? Excuse me.

19 **A.** No. I -- I misheard you. I'm sorry. So we're -- talking
20 about the left-hand column?

21 **Q.** Yeah. And sentence that starts, "For individuals." Do
22 you see that?

23 **A.** I'm trying to find it. Where is it in the --

24 **Q.** It's -- it's about nine lines up from the bottom of the
25 left-hand column on page 21, sir.

1 A. Oh, okay. "For individuals." Yes. I'm sorry. Yes.

2 Q. Okay. I'm going to read that. *For individuals in the AHS*
3 *who did not complete a Phase 2 re-interview five years after*
4 *enrollment.* Do you see that?

5 A. Yes.

6 Q. And then it goes on and says, *An imputation method was*
7 *used.* Do you have that?

8 A. Yes.

9 Q. *That permitted inclusion of all participants in Phase 2*
10 *analyses.* Did I read that correctly?

11 A. Yes.

12 Q. And then it says, *The imputation method was based on their*
13 *baseline data, even if portions of subsequent data were*
14 *missing.* Did I read that correctly?

15 A. Yes.

16 Q. *Which led to the observation that neither missing data nor*
17 *imputation had major impacts on the main results for many of*
18 *the pesticides.* Did I read that correctly?

19 A. Yes.

20 Q. Did you vote on that?

21 A. Including parathion, diazinon, and malathion; but it
22 doesn't say anything about glyphosate.

23 Q. Did you read the Heltshe Study?

24 A. Did I read the Heltshe Study?

25 Q. It was a reference to your Supplemental Report, sir.

1 A. Oh, to the -- yeah -- imputation? Describing imputation
2 methods? Yes.

3 Q. Yes. Heltshe includes glyphosate in her list of
4 pesticides that were covered by this methodology; doesn't she?

5 A. Yes.

6 Q. There's a table that includes glyphosate in the Heltshe
7 Report that I asked you about in your deposition. True?

8 A. True, but as I stated in my deposition, the imputation for
9 glyphosate, I feel, is flawed, because they're basing the
10 imputation on data from the original exposure assessments from
11 the questionnaire. And the -- that data was -- was asking for
12 recall of what they were exposed to 10, 20 years before. And
13 so there was a lot of recall bias in that. And so the initial
14 data was flawed.

15 So if you're going to use flawed data and the imputation
16 method, then you're going to get even more flawed data. So
17 that's one of the main criticisms, I understand, of the
18 AHS Study now, because -- because of the method of exposure
19 assessment at the very beginning.

20 And can I make an observation of my own?

21 **THE COURT:** Go ahead.

22 **THE WITNESS:** That as far as the AHS Study, and the
23 most recent Andreotti Paper that came out in 2018, and the
24 question about, *Has there been publications criticizing that*
25 *study or criticizing the results?* -- well, the paper just came

1 out in January of 2018. It's just a month or so ago. And it
2 takes a while for peer-reviewed publications to come through
3 the mill.

4 So the fact that there aren't any doesn't mean that
5 there -- there aren't opinions to that effect, and that there
6 won't be papers coming out in the peer-reviewed literature
7 probably in the very near future. I'm not aware of any that
8 are being done, but that's just my opinion.

9 **BY MR. HOLLINGSWORTH**

10 **Q.** At the time that the -- this IARC Monograph was done in
11 March of 2015, did the Working Group know that the AHS had used
12 imputation methodology for glyphosate?

13 **A.** At the time of the --

14 **Q.** At the time that this IARC Monograph was done, did the --
15 did the Working Group know that -- that AHS had used the
16 imputation method for glyphosate, in particular?

17 **A.** Well, I mean, it's in here. Yes. It's in the monograph.

18 **Q.** Now, Dr. Jameson, you don't consider yourself to be an
19 epidemiologist; do you?

20 **A.** I am not a formally trained epidemiologist, but I have
21 over 30 years of experience in doing assessments -- cancer
22 assessments -- where I have evaluated and reviewed countless
23 epidemiology studies, and given opinions of what the
24 epidemiology data is saying.

25 In my work at the NIHS for Report on Carcinogens, that was

1 part of my everyday work: To evaluate epidemiology data, along
2 with the toxicology and mechanistic data.

3 In my participation in IARC, I'm asked to review the
4 epidemiology data, and vote on the relevancy and the adequacy
5 of the data, and what it means.

6 So, while I'm not formally trained in epidemiology, I feel
7 like I am an expert in epidemiology, because of all of my past
8 experience and work in that area.

9 Q. Do you remember when I asked you the following question,
10 and you gave the following answer at your deposition in
11 September? Or more --

12 **THE COURT:** Hold on a second.

13 What is page number and the line number, so that opposing
14 counsel can look at it, as you are proposing to read it? And,
15 of course, opposing counsel can also propose that you read
16 additional lines, if it's necessary for context.

17 **MR. HOLLINGSWORTH:** Sure. At 44, lines 1-3. This is
18 the supplemental deposition.

19 **JUDGE PETROU:** So what exhibit number is it?

20 **MS. KLENICKI:** 738.

21 **MR. HOLLINGSWORTH:** This is Exhibit 738. It's a
22 supplemental expert deposition. It's at page 44, lines 1-3.

23 And my question is: Do you remember giving the following
24 answer to this question?

25 *Are you an epidemiologist?*

1 *Answer: I am -- I consider myself a toxicologist. I do*
2 *not consider myself an epidemiologist.*

3 **MS. WAGSTAFF:** And I would suggest you read pages
4 starting at 113, please, as well.

5 **THE COURT:** Page 113?

6 **MS. WAGSTAFF:** Yes.

7 **THE WITNESS:** 113?

8 **MS. WAGSTAFF:** Right, but I'm saying to put it in
9 context.

10 **THE WITNESS:** Okay.

11 **MR. HOLLINGSWORTH:** Well, Your Honor, I have a
12 question pending.

13 **THE COURT:** Read it.

14 **THE WITNESS:** Where is it --

15 **THE COURT:** Mr. Hollingsworth, go ahead and read --

16 **MR. HOLLINGSWORTH:** My question: Do you recall
17 giving the following --

18 **THE COURT:** Hold on a second. Page 113. I assume
19 starting at line 13?

20 **MR. HOLLINGSWORTH:** It's page 44. Page 44.

21 **THE COURT:** Mr. Wagstaff, what were you --

22 **MS. WAGSTAFF:** 113, page or line 8.

23 **THE COURT:** Okay. You go ahead and read that.

24 **MR. HOLLINGSWORTH:** This is at page 44, Your Honor.

25 **THE COURT:** You're trying to impeach him with his

1 prior deposition testimony.

2 **MR. HOLLINGSWORTH:** Right.

3 **THE COURT:** And opposing counsel wants to have other
4 deposition testimony read, to put in context your impeachment.
5 I agree that you should have to read that, so can you go ahead
6 and read that?

7 **MR. HOLLINGSWORTH:** Well, I haven't asked -- I
8 haven't gotten an answer to my question yet.

9 **THE COURT:** Read the whole thing, and then ask him
10 the question.

11 **MR. HOLLINGSWORTH:** Okay. My question is, sir --

12 **THE COURT:** No. Read page 113, starting at line 8.

13 **MR. HOLLINGSWORTH:** Okay. That is --

14 (Pause in proceedings.)

15 **MS. KLENICKI:** (Indicating.)

16 **THE COURT:** Why don't you read all the way through
17 page -- about the middle of page 115.

18 **MR. HOLLINGSWORTH:** This is a question by
19 Ms. Wagstaff.

20 **THE COURT:** Yep.

21 **MR. HOLLINGSWORTH:** Okay. (Reading.)

22 "MS. WAGSTAFF: Do you recall when
23 Mr. Hollingsworth asked you if you were an
24 epidemiologist? And I believe you answered that you
25 were a toxicologist. Is that correct?

1 "ANSWER: Correct.

2 "QUESTION: Okay. Can you explain how being a
3 toxicologist relates to you being an epidemiologist,
4 if at all?

5 "ANSWER: Well, I think I stated earlier that in
6 doing your -- the work that I did and continue to do
7 in cancer-hazard identification, the requirement is
8 that you became an expert in a wide variety of
9 different areas, one of which is toxicology, one of
10 which is epidemiology, one of which is genotoxicity
11 and mechanism of action. One is exposure.

12 "And based on the 40 years that I have been doing
13 this work, I have gotten what you considered
14 on-the-job training in all of these areas. My degree
15 is in chemistry, but I have been -- I have done
16 toxicology since I graduated from the University of
17 Maryland. And on-the-job training is as good if not
18 better than a college degree is, just -- in just
19 about all areas.

20 "I have worked closely with the epidemiologists,
21 helping them in their studies. I have been asked to
22 review epidemiology studies and papers as part of my
23 work with IARC, and I give my opinion as to what the
24 epidemiology data is saying, and if it meets their
25 criteria for evaluating epidemiology data, as far as

1 being a sufficient evidence or limited evidence in
2 for causation of cancer.

3 "For the Report on Carcinogens, I also have
4 worked with epidemiologists who help us evaluate the
5 nominations for the Report on Carcinogens. As part
6 of my responsibility, I wrote criteria for evaluating
7 epidemiology data for the Report on Carcinogens. And
8 those criteria are still used today in evaluating the
9 data; the epidemiology data for the Report on
10 Carcinogens.

11 "So while I profess to be a toxicologist, you
12 can't say, *Well, I am a toxicologist, and an*
13 *epidemiologist, and a mechanistic expert, and a*
14 *genotoxicologist, and what-have-you.* I take on the
15 moniker of 'toxicologist,' but you have to understand
16 that in doing hazard identification, you have to
17 become an expert in all of those areas in order to
18 evaluate the data and give an opinion.

19 "And so, while I don't have the degree in
20 epidemiology, I have the experience and training to
21 consider myself an expert in epidemiology to evaluate
22 this data."

23 **THE COURT:** Okay. Can you go ahead and ask him your
24 question now?
25

1 BY MR. HOLLINGSWORTH

2 Q. My question is: Do you recall when I asked you the
3 following question, and you gave the following question?

4 "Are you an epidemiologist?

5 "ANSWER: I am. I consider myself a
6 toxicologist. I do not consider myself an
7 epidemiologist."

8 A. That is what I said at that time; but as you read into the
9 record, I do consider myself an expert in epidemiology because
10 of my past experience and -- and working that I've done over
11 the past 40-plus years.

12 Q. Are you board certified in epidemiology?

13 A. No, sir, I am not board certified in epidemiology.

14 Q. Are you board certified in toxicology?

15 A. No, sir, I am not.

16 Q. Sir, you did the Report on Carcinogens to -- that was made
17 to Congress for about 11 years, you said?

18 A. Well, I was involved with it for about 18 years. I was
19 the director for about 9 or 10 years, yes.

20 Q. During those 18 years, you never reported to Congress that
21 glyphosate was a carcinogen, or that it could cause
22 non-Hodgkin's lymphoma in humans; did you?

23 A. I never reported glyphosate because we never reviewed it.
24 No.

25 MR. HOLLINGSWORTH: No further questions, Your Honor.

1 Thank you.

2 **THE COURT:** Thank you.

3 Do you have any redirect?

4 **MS. WAGSTAFF:** I do. Just a few moments.

5 **REDIRECT EXAMINATION**

6 **BY MS. WAGSTAFF**

7 **Q.** Okay. And the only exhibit I'm going to use is 149, if
8 y'all want to get that handy, which is the briefing note for
9 IARC Scientific and Governing Council members from January of
10 '18.

11 So tell me when you're ready, Dr. Jameson. Do you have
12 that in front of you?

13 **A.** Which is that, now?

14 **Q.** I don't know if yours is marked "149," but it's called,
15 "The Briefing Note for IARC Scientific and Governing Council
16 Members Prepared by the IARC Director." Do you need a copy?

17 **A.** I need a copy. I'm sorry.

18 **THE COURT:** Do you want to grab a copy for
19 Judge Petrou, as well?

20 **JUDGE PETROU:** Sure.

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

22 **THE COURT:** Thank you.

23 **MS. WAGSTAFF:** Mm-hm. All right. This one has
24 little highlights on it, but --

25 **THE WITNESS:** Okay. That's fine.

1 **BY MS. WAGSTAFF**

2 **Q.** All right. The first question I would like to ask you,
3 just -- just to make it clear for the Judges, is: You did
4 review both the negative and positive data for glyphosate being
5 a carcinogen. Correct?

6 **A.** That's correct.

7 **Q.** Okay. And if you could, pull up Exhibit 149. The only
8 thing I really -- I'm afraid maybe I did a bad job of this on
9 my direct, but the hazard assessment -- we're getting hung up
10 on what it means, and the definition of what a hazard
11 assessment is versus a risk assessment. And I think we can
12 simplify this a lot. We can just sort of agree to disagree on
13 what the definition means, by looking at what you guys actually
14 did. Right?

15 **A.** Yes.

16 **Q.** And so whether we agree with Monsanto on what a hazard
17 assessment means, can we agree that you considered in your
18 analysis here the human data?

19 **A.** Yes.

20 **Q.** Okay. And you -- you considered all of the epidemiology.
21 And you considered that in your analysis: The doses that
22 humans receive. Correct?

23 **A.** Yes.

24 **Q.** Okay. And if you can, turn to page -- and I know you
25 can't speak for all 17 or 18 members of IARC 112. Right?

1 A. Right.

2 Q. You can only speak for Dr. Jameson; but luckily the IARC
3 director answered this question for us.

4 A. Right.

5 Q. So it came out in January of 2018, on Exhibit 149. If you
6 turn to page 8, luckily, we know if IARC 112 considered
7 real-world exposures. In fact, it's even in quotes. So can
8 you look at that section, and read into the record those bullet
9 points?

10 A. Okay. The document reads, *Monograph evaluation takes into*
11 *account real-world exposure by evaluation of epidemiological*
12 *studies. A charge leveled at the monographs is that*
13 *evaluations are divorced from the real world; i.e., are named*
14 *without taking into account realistic human exposures.*
15 *However, epidemiological studies are a central part of*
16 *monograph evaluations, and, by definition, deal with people*
17 *exposed in daily life, including at work.*

18 *The study frequently considers the gradient of risk*
19 *observed with different levels of exposure. One part of the*
20 *monograph evaluation is specifically dedicated to describing*
21 *the circumstances under which human exposure occurs, and at*
22 *what levels.*

23 *In addition, when considering scientific evidence of*
24 *carcinogenicity, including by logical mechanisms, the Working*
25 *Groups placed special emphasis on whether the observations are*

1 *relevant to humans.*

2 *In light of occurring 'real-world' human exposures,*
3 *Working Groups synthesize evidence in humans, animals, and*
4 *other model systems in reaching overall conclusions.*

5 Q. All right. So we don't need to get any more on whether
6 IARC considers real-world exposures. Right?

7 A. Correct.

8 Q. And this comes from the director of IARC, which is a
9 pretty high-up person, I would guess, at IARC?

10 A. Within the World Health Organization, yes.

11 Q. And that summarizes sort of the analysis you did, as well,
12 with respect to real-world analysis?

13 A. I absolutely considered real-world exposures in reviewing
14 the epidemiology data.

15 Q. So it's okay if we have different definitions of what
16 "hazard assessment" means. We can all agree that both IARC and
17 Dr. Jameson considered real-world exposures. Right?

18 A. Absolutely.

19 **MS. WAGSTAFF:** All right. No further questions.

20 **THE COURT:** Anything further?

21 **MR. HOLLINGSWORTH:** No, sir.

22 **THE WITNESS:** Thank you for the honor, Your Honor.

23 Thank you.

24 **MS. GREENWALD:** Plaintiffs call

25 Dr. Christopher Portier.

1 **THE CLERK:** Please raise your right hand.

2 CHRISTOPHER PORTIER,

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

5 **THE WITNESS:** I do.

6 **THE CLERK:** Thank you. Please be seated. And for
7 the record, please state your first and last name, and spell
8 both of them.

9 **THE WITNESS:** Christopher Portier.
10 C-h-r-i-s-t-o-p-h-e-r. P-o-r-t-i-e-r.

11 **THE CLERK:** Thank you.

12 DIRECT EXAMINATION

13 **BY MS. GREENWALD**

14 **Q.** Good afternoon, Dr. Portier. You have the slide also in
15 front of you in the book, but you have a screen there, and we
16 can go through it that way.

17 Your Honors, you have notebooks. Great. So the slide
18 deck is Exhibit 1, Tab 1.

19 Dr. Portier, can you please describe your qualifications
20 for the opinions you're providing in this case, focusing in
21 particular on your work-related experience?

22 **A.** Certainly. I have a Ph.D. in biostatistics, with a minor
23 in epidemiology. My thesis topic was the optimal design of --
24 (Reporter requests clarification.)

25 **THE WITNESS:** The optimal design of the two-year

1 rodent carcinogenicity study to assess cancer hazard of
2 chemicals.

3 I spent over 30 years at the NIH; the National Institute
4 of Environmental Health Sciences. During that entire time I
5 was a Principal Investigator there, with my own laboratory,
6 initially starting out in statistics, and ending up in
7 molecular biology and toxicology, with my own wet labs.

8 For part of that time I was the -- I ran the U.S. National
9 Toxicology Program, which is the world's largest tox. program.

10 I was director of CDC's National Center for Environmental
11 Health, and the U.S. Government's Agency for Toxic Substances
12 and Disease Registry. Both of these do risk evaluations.

13 ATS New York advises communities about what to do about
14 toxic dumpsites, and works with EPA so the sites will be
15 cleaned up.

16 I've done a lot of national and international science
17 advisory boards. I was Chair of the President's National
18 Science and Technology Consult Toxics and Risk Subcommittee.
19 I've sat on EPA Science Advisory Panels for pesticides for five
20 years. And I was the Chair of the IARC Advisory Group that
21 rewrote the preamble in 2006.

22 And I will comment on, if you don't mind, a discussion you
23 had regarding the preamble -- I believe it was yesterday -- and
24 the use of the term "quantitative," and under "probably human
25 carcinogen," I think the interpretation that was a little bit

1 wrong.

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

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

9 **BY MS. GREENWALD**

10 **Q.** Dr. Portier, what is biostatistics?

11 **A.** So biostatistics is the discipline of statistics, but
12 applied to assays and experiments in the biological realm.
13 Typically, biostatisticians work in epidemiology or in animal
14 laboratory data.

15 **Q.** And why is biostatistics important to the opinions you're
16 providing in this case?

17 **A.** Well, you have to -- you have to understand statistical
18 significance of each individual experiment in order to move
19 forward, but the -- the important thing about the "bio" in
20 "biostatistics" is that you really want to know your
21 experimental field. You spend time, you spend effort learning
22 how these experiments are done, what are their limitations, et
23 cetera, so that you get the right evaluation to answer the
24 question that is actually being asked.

25 **Q.** Okay. You just mentioned "experimental field." Do you

1 have specialties within biostatistics?

2 **A.** Do I have a specialty?

3 **Q.** No. Are there specialties within biostatistics; and if
4 so, do you have a subspecialty?

5 **A.** Yes. There are specialties in biostatistics. I guess my
6 subspecialty would be environmental laboratory studies.

7 **Q.** Of animal bioassays?

8 **A.** Of animals, and cells, and molecular biology studies, and
9 things along these lines, although I have some valid background
10 in climate change, and other areas.

11 **Q.** Are you a fellow of American Statistical Association?

12 **A.** Yes, I am.

13 **Q.** And when did you become a fellow?

14 **A.** 1992, I believe.

15 **Q.** What does it mean to be a fellow?

16 **A.** It American Statistical Association elevates -- they vote,
17 nominate, bring in one-third -- at the max, one-third of
18 1 percent of the statisticians who belong to the ASA get
19 awarded to be fellows with the American Statistical
20 Association. They do that percent every year, so it's probably
21 about 3 to 4 percent, total. It's an honor.

22 **Q.** Have you read the Expert Report and deposition transcript
23 of Dr. Corcoran?

24 **A.** Yes, I have.

25 **Q.** And is he one of the experts that Monsanto has proffered

1 in this case?

2 **A.** Yes, it is. He is, I believe.

3 **Q.** Based on your review of his report, his CV, and deposition
4 testimony, do you have -- does he have, in your opinion,
5 relevant experience in evaluating bio -- animal bioassays?

6 **A.** No.

7 **Q.** And why is that?

8 **A.** Well, because to evaluate the animal bioassay data to
9 decide whether you're seeing a positive result or a negative
10 result, you have to be able to not only run a statistical test
11 on it, but you really have to understand the biology. A lot of
12 other things go into deciding whether this is a positive
13 finding or not, and I don't think he has experience in that
14 area.

15 **Q.** Dr. Portier, have you ever been an expert witness in a
16 lawsuit before this case?

17 **A.** No, I have not.

18 **Q.** And other than "Science Day," when you presented general
19 science of toxicology to Judge Chhabria in this case, actually,
20 is this the first time you've ever testified -- is this the
21 next time you've ever testified in a courtroom?

22 **A.** Yes, it is.

23 **Q.** Okay. Slide 3, please.

24 **A.** And it makes me nervous.

25 **Q.** No one else. Just you.

1 What are the fields of expertise that underlie your
2 opinions in this case?

3 **A.** I reviewed all of the epidemiology literature, all of the
4 toxicology literature, all of the mechanisms-of-cancer
5 literature, as well as my extensive experience in the field.

6 **Q.** Okay. So before we get into the details of your
7 methodology and your conclusions, please tell the Court what
8 opinions you have reached after reviewing the epidemiology,
9 toxicology, and mechanisms of cancer.

10 **A.** It's right here on the screen. To a reasonable degree of
11 scientific certainty, given the human, animal, and mechanistic
12 evidence, glyphosate probably causes NHL, and the probability
13 that glyphosate causes NHL is high.

14 **Q.** Okay. Now I'd like to get into the methodology that
15 underlies that opinion. Slide 4, please.

16 What epidemiological review did you undertake? I mean,
17 what conclusions did you reach based on that review?

18 **A.** So I reviewed all of the literature. There were six
19 case-control studies showing similar modest increases of
20 associations between glyphosate and NHL.

21 There was one cohort study -- the Agricultural Health
22 Study -- with no apparent effect.

23 I will point out that there were dozens of ancillary
24 studies. Some case-controls studies had special studies
25 looking at their exposure metrics, and how well they were

1 working. The Agriculture Health Study has an extensive
2 collection of ancillary studies, as well.

3 What I concluded is that causality is possible, but
4 there's still the possibility of bias, chance, and confounding
5 in these data. I believe it's not likely that these things
6 would explain the entire association. So my conclusion is that
7 the data supports an association of glyphosate with NHL.

8 Q. So when you say these things would not change your
9 decision, you mean bias, chance, and confounding. Is that
10 right? I just want to make sure that I understand.

11 A. Correct. I don't believe they're strong enough. I don't
12 believe they're strong enough -- bias, chance, and
13 confounding -- to completely explain the entire association.

14 Q. Understood. All right. Slide 5, please.

15 Can you please explain the phases of a two-year animal
16 carcinogenicity study? I'll get that word wrong every time.

17 A. Can he we just say "cancer study"?

18 Q. I want to do "cancer study."

19 A. So a two-year cancer study is intended to cover the major
20 portion of an animal's lifetime. The animals start on this
21 study at six weeks. So six-week-old mouse, a six-week-old
22 rat -- they have reached puberty.

23 These rodents are randomly placed into different dose
24 groups, so that you avoid any chance of bias by putting all of
25 the animals with one weight in one group, or something along

1 those lines. So you're very careful to make it as random as
2 you possibly can. Everything is controlled in these
3 experiments. So the animals get the same diet. The animals
4 get light and dark cycles that are carefully controlled, et
5 cetera.

6 The idea is that the only difference that would -- that
7 would explain the cancers, if you see them increase in the
8 animals in the dose groups, is the dose. So everything else is
9 controlled.

10 So, unlike epidemiology, where you have lots of
11 confounders you have to concern yourself with, there are no
12 confounders in an animal cancer study. It's a completely
13 controlled study.

14 Typically dosing goes for two years.

15 There's generally a control group -- we talked about that
16 earlier -- and three different dose groups.

17 It's generally rats and mice; and males and females.

18 And it's 50 to 75 rodents in each sex-species group in
19 these studies that we're looking at here.

20 Now, I'll say "sex-species group" quite often. That's
21 jargon for toxicologists. It's simply the way you break it up
22 in little, little boxes. There are two sexes, probably two
23 species, and four dose groups. And so when I talk about that,
24 it's looking at each of those.

25 At the end of the study -- again, usually two years -- any

1 rodents that are still alive get sacrificed. Any rodents that
2 died earlier were, of course, kept. Every one of these animals
3 gets a full pathology review on them, which means they
4 typically remove up to 40 organs from the animals. They take
5 slices through those organs; create histopathology slides. You
6 probably saw them in high school. And those are then sent to a
7 pathologist who reads them.

8 In the National Toxicology Program -- I can easily
9 describe that -- that pathologist -- after they read it,
10 another pathologist verifies that the first pathologist got it
11 right.

12 Then any disagreements between those two pathologists --
13 there is a Pathology Working Group that comes in of independent
14 pathologists who look at the disagreements, to make sure
15 everybody gets to the same agreement, in terms of which tumors
16 are which tumors.

17 After that is when the analysis starts. So once you've
18 decided exactly which tumors are there, then come the
19 statisticians and the toxicologists, looking at the results,
20 and finding after about a year after the end of the study, a
21 year and a half, results are reported.

22 Q. Okay. Slide 6, please.

23 Can you please summarize the guidelines that apply to the
24 analysis of animal cancer data among the various agencies that
25 review such data?

1 A. Yes. There's guidelines all over the place. Now, there
2 are guidelines on design of the studies. There's guidelines on
3 how to analyze the studies. And there are guidelines on how to
4 interpret these studies.

5 EPA has Guidelines for the interpretation and analysis of
6 the studies. The NTP has guidelines for all three aspects of
7 it. IARC has guidelines for the evaluation. The European
8 Chemicals Agency doesn't have the independent guidelines for
9 design and analysis -- in fact, they use the guidelines by the
10 OECD; the Organization for Economic Cooperation and
11 Development -- but they do have guidelines for review.

12 We talked about the European Food Safety Authority
13 earlier. The European Food Safety Authority uses EChA's
14 guidelines -- the European Chemicals Agency -- to do their
15 evaluations. And that's because legally, EChA owns the
16 guidance. And EFSA -- they let EFSA do it whenever they feel
17 like it.

18 OECD has guidelines for all three, and the National
19 Academy of Sciences has spoken about all three. I will make
20 one other comment that somebody had discussed earlier, again,
21 if you don't mind. There was a statement that said EFSA did a
22 risk assessment for glyphosate. That is incorrect. By
23 European law, if a compound is a pesticide and it's
24 carcinogenic, it is banned. You don't do a risk assessment.

25 There is a little bit in the law that says if the human

1 exposure is absolutely minuscule, then maybe we'll let you put
2 it in there; but if it's a pesticide -- so they don't do a risk
3 assessment. All they have to do is identify it as a
4 carcinogen, and then it would be banned.

5 The USEPA didn't do a risk assessment, either. The USEPA
6 decided it was not carcinogenic. And once they do that
7 decision, they don't go and calculate risk. So they only do a
8 hazard assessment. So all of these are hazard assessments.

9 Q. So, Dr. Portier, are most of the guidelines of these
10 various agencies that you have on Slide Number 6 -- do they
11 have similar guidelines on how to evaluate, analyze, interpret
12 data of animals; cancer data, studies?

13 (Reporter requests clarification.)

14 **BY MS. GREENWALD**

15 Q. Do they all have similar guidelines for how to evaluate
16 and analyze animal data?

17 A. Yes, they do. I -- I would give you one example. All of
18 them say that if you see a positive Armitage linear trend test
19 or a positive Fisher Exact Test in an animal cancer study,
20 these should be considered positive findings in the statistical
21 means; not the biological means.

22 Q. Did you follow these guidelines in your analysis of this
23 case?

24 A. Yes, I did.

25 Q. Okay. Slide 7, please.

1 Can you please explain the statistical methodology used in
2 toxicology to evaluate rodent cancer studies, and how to
3 interpret those evaluations? I'm staying with "cancer."

4 **A.** Yes. Originally, in the -- in the past, most people did a
5 Fisher Exact Test. Fisher Exact Test compares tumor response
6 in one dose group to the control group. So in a typical study
7 you'd have three Fisher Exact Test p-values.

8 But the correct way to do it is the Cochran-Armitage Trend
9 Test, which is the way most people do it. All of the
10 regulatory reviews have Cochran-Armitage tests in them. The
11 benefit of the Cochran-Armitage Test is that it simultaneously
12 analyzes all of the data, and looks for a trend in the data
13 with increasing dose. Now, these studies are designed to have
14 increase in dose; the idea being that as the dose increases,
15 the probability of getting cancer is increasing. So therefore
16 you want to analyze it that way, which is what the
17 Cochran-Armitage Trend Test does.

18 If you have all of the information -- so if I'm the
19 National Toxicology Program, and I know what happened to every
20 one of my rats and mice in my study, then I'm not going to use
21 either of these two. I would use a survival-adjusted test.
22 That's because of some of the animals die early, and you want
23 to account for that difference in survival between different
24 groups that might make a difference on the p-value.

25 The NTP uses the Poly-3 Test, which is a test I invented.

1 Q. You didn't use the Poly-3 Test here; did you?

2 A. No. That's because I don't have the individual data for
3 the individual animals.

4 Q. Had you -- sorry. Had you --

5 A. And I don't know when an animal died, and whether it had a
6 tumor or not, except for three of the studies that -- I decided
7 that if I'm going to compare studies and look at cancer across
8 all the of the studies, I wanted to use the same methodology,
9 so I stuck to the Cochran-Armitage Trend Test.

10 Q. Can we go to the next slide, please: Slide 8?

11 Can you please describe the Cochran-Armitage Trend Test
12 using this diagram?

13 A. Yeah. This diagram is intended to show you what would
14 typically see an animal bioassay. This is the one you were
15 talking about just now; the Wood Study. Malignant lymphomas in
16 male CD-1 mice.

17 The big black dots -- those are the response signal to
18 each of the doses. The dose is the x-axis. The proportional
19 tumor is the y-axis. The confidence bounds around the dots are
20 just typical 95 percent confidence amounts. Fisher's Exact
21 Test for each one of these. You can see I put the p-value for
22 that.

23 And then the Cochran-Armitage Test, which is making this
24 trend right through the middle, the p-values there -- what the
25 Cochran-Armitage Test really does is calculates the slope of

1 that line, and looks to see if the slope of that line is
2 different from zero.

3 (Reporter requests clarification.)

4 **THE WITNESS:** Different from zero. I'm sorry.

5 If it's significantly different from zero, that's what the
6 p-value is.

7 **BY MS. GREENWALD**

8 **Q.** So, Dr. Portier, I'm going to ask you if we can just slow
9 it down a little bit. The court reporter has been going all
10 day. And you know this material very well, but it's hard to
11 take it all down. So if we can just slow it down a little bit,
12 I know she would appreciate it.

13 **A.** I'll try.

14 **Q.** I know. It's all we can ask.

15 If you can go to the next slide, please.

16 Can you please explain p-values, which have been a lot
17 of -- subject to a lot of discussion and writing in this case,
18 why they are methodologically necessary for scientists, and
19 what p-values inform us about the hazards of glyphosate
20 exposure?

21 **A.** Certainly. I'd be happy to. So statistical tests are
22 built around what's called a "null hypothesis" and an
23 "alternative hypothesis." In animal cancer studies, the null
24 hypothesis is that the chemical does not increase the cancer
25 risk when given to the animals. The alternative hypothesis is

1 they can increase the tumor response, as a function of dose.

2 Now, statistical tests aren't as simple as we sometimes
3 want them to be. Statistical tests depend upon a complex set
4 of assumptions embodied in a statistical model. And so you
5 have to realize when interpreting this statistical test, you're
6 doing it under the assumption that that model is correct. And
7 sometimes that model's not correct, or you're really not
8 certain.

9 Anyway, the p-value is the probability of observing the
10 data that you saw under the null hypothesis that there is no
11 effect. So you're calculating the probability that these data
12 are so different than what the model would say under the null
13 effect, that I think it's -- it's -- it can't possibly be from
14 that null-effect model.

15 Traditionally, p-values less than 0.05 or 0.01 are used to
16 reject a null hypothesis in favor of the alternative
17 hypothesis. So that's how p-values are used in this
18 confection.

19 **Q.** Dr. Portier, you have a reference on your slides here to
20 Greenland. Is that an article about statistical tests?

21 **A.** Yes. The Greenland article talks about the complexity of
22 statistical tests, as well as how to use p-values. We had the
23 discussion yesterday about -- and the day before -- p-values in
24 the epidemiology literature, and whether it's a bold line or
25 not. Greenwald does a good job of discussing that.

1 **MS. GREENWALD:** Your Honor, that's Tab 2 in your
2 book.

3 Okay. If you can, keep it slow.

4 **THE WITNESS:** I'm really trying.

5 **BY MS. GREENWALD**

6 **Q.** Okay. If you can go to the next slide, please.

7 How do scientists determine which tumors to analyze in an
8 animal cancer study, and why?

9 **A.** That's something that's always debated at least a little
10 bit. So you do these 40 organs. You take the slides. You
11 look at all primary tumors -- so a primary tumor's something
12 like a liver carcinoma or a liver adenoma -- but you don't look
13 at every single possible, because there are limitations to what
14 statistics can find for you.

15 And so usually the rule of thumb is if you have three
16 tumors across all of the dose groups -- so maybe one control,
17 one low-dose, one mid-dose. That would be three tumors -- then
18 you include that as a primary tumor that you evaluated for
19 the -- from this study. That's because less than three tumors
20 can't be found statistically significant.

21 You don't do metastatic tumors. So when a tumor forms in
22 the body, as it gets older and older, it begins to bleed off
23 cells. And these cells get picked up in the blood; transported
24 to other parts of the body. And those are metastatic tumors.

25 Common metastatic tumors occur in the lung from -- from a

1 cancer in the liver. These metastatic tumors are interesting,
2 but they're not related to the causing of the cancer. They're
3 consequence of the cancer. So you might analyze them, but
4 they're not included in the discussion of causality in the
5 animals.

6 If there are rare tumors that you think are increased,
7 where you have less than three tumors, then you would use
8 historical controls. I will point out that rare tumors are
9 defined to be less than 1 percent historically in the control
10 animals.

11 You would combine benign and malignant tumors when there
12 are at least three total tumors. So we talked about adenomas
13 and carcinomas before. That's a combination you would do. So
14 you do the primaries, as well as those combinations.

15 And finally you would combine systemic tumors that
16 occurred in multiple organs when there are at least three total
17 tumors. The example here is the malignant lymphomas. They
18 occur in the spleen. They occur in the uterus. They occur in
19 various parts of the body. And you collect all of that
20 information, and do it as one lymphoma or not.

21 **JUDGE PETROU:** So I have a question. The second
22 bullet point was that if it was a rare tumor, less than
23 1 percent historically, then you look at the historic data.
24 Correct?

25 **THE WITNESS:** Correct.

1 **JUDGE PETROU:** So presumably the converse is also
2 true: If it's not a rare tumor, you don't look at the historic
3 data -- or not?

4 **THE WITNESS:** You could -- I like looking at the
5 historic data all of the time, just to see what informs me
6 about the study that I'm looking at; but Rule One in cancer
7 bioassays is that the current control is the best control for
8 doing your analysis. And the reason for that is quite simple.
9 Even though you try to control everything in the laboratory,
10 you can't do it from one study to the next; but the controls
11 that are fed and housed at the same time as the treated animals
12 are most like those treated animals. So you use that.

13 But in a case of a rare tumor, sometimes the historical
14 controls that -- you only see two. Let's take the kidney
15 tumors we're talking about here. You see three at the highest
16 dose. So you can do a p-value on that, but its p-value's .06,
17 so it's not statistically significant at the 5 percent level;
18 but in my historical controls for these kidney tumors, it's
19 very low. The highest we ever saw was 2 in any dose in any
20 control animal. And so that makes this a biologically
21 important finding.

22 And you can actually do an analysis of those historical
23 controls, and see if that's statistically significant.

24 **BY MS. GREENWALD**

25 **Q.** Dr. Portier, what's the basis for requiring three or more

1 tumors?

2 **A.** You require three or more tumors because you can't find a
3 statistically significant finding without three of them. And
4 the question that you would ask that was driven by that point
5 is: Are these tumors arising due to random chance? And I will
6 get to that in a minute.

7 **Q.** Okay. Do you know whether Dr. Corcoran followed the
8 three-or-more-tumor guidance in his analysis?

9 **A.** He mentioned it.

10 **Q.** Did he follow it in his analysis?

11 **A.** Oh, he mentioned it. He talked about it, but when he came
12 to his false discovery rate at the table toward the end of his
13 report, he used all of the -- all of the tumors. So he
14 analyzed somewhere around -- I guess it's about 600 or 650
15 sites that had less than three tumors; one or two tumors.

16 **Q.** Thank you. And did Dr. Corcoran count both primary and
17 secondary tumors in his analysis?

18 **A.** Yes. He did metastatic tumors in the analysis, as well.

19 **Q.** In your opinion, is that methodologically flawed?

20 **A.** It doesn't speak to the question of causing cancer in the
21 animals, if you're talking about metastatic tumors. It doesn't
22 speak to the question at hand.

23 **Q.** So for the issue at hand in this case, would that be
24 methodologically flawed to count both primary and secondary
25 tumors?

1 A. Yes.

2 Q. Next, Slide 11.

3 Can you please state the type of data that was available
4 to you for your review of glyphosate animal studies?

5 A. Sure. A little background. When you do an animal study,
6 you write a full technical report from the animal study. Even
7 though it's not going to be published in the open literature
8 for these type of regulatory studies, they still write an
9 entire report which talks about what materials were used, what
10 methods were used, how the animals were housed, what feed they
11 got. It gives you have the statistical analysis done by the
12 laboratory, and the final conclusions done by the laboratory.

13 I want to make it clear I have none of those full study
14 reports. It's not available to me for almost all of these. I
15 have study reports for three of the studies from Monsanto.
16 They didn't -- to me, to my eyes, having looked at cancer
17 studies for a long number years, they didn't like look like a
18 full study report. I didn't see the statistical analysis I
19 expected to see, and things like that. So I was a little lost.

20 They did have individual animal pathology data, but it was
21 pretty poorly documented. It was hard for me to figure out
22 exactly what was done there.

23 The data by Greim, *et al.*, 2015, they have supplements at
24 the end of their documents. I think you have them there.

25 **MS. GREENWALD:** Yes. It's Tabs 3 and 4, Your Honor.

1 Q. So Tab 3 is the Greim Study, and Tab 4 is the supplements
2 to the Greim study?

3 A. And if you just look at Tab 4, I want to simply show you
4 what this type of data looks like, in terms of what you have to
5 pull to try to figure it -- what to do with it. It's
6 interesting that it isn't electronically available. The
7 National Toxicology Program makes all of their data
8 electronically available to anybody; even the pathology slides.
9 And so none that of that is available in this case.

10 But I did use some of the data from Greim to answer some
11 of the questions I've done. He doesn't provide individual
12 animal data, so I couldn't do survival adjustment. He does not
13 provide combined malignant tumors. That, I had to work on on
14 my own in some other way. Some of the systemic tumors in his
15 table are not available, but the regulatory authorities -- he
16 has done those, so I could use their results to get the same
17 number.

18 So as a result, using only Greim, I wanted to miss [sic]
19 important tumor findings. So you have to use a combination of
20 things.

21 (Reporter requests clarification.)

22 **THE WITNESS:** If you only used Greim's, you would
23 miss important tumor findings.

24 **BY MS. GREENWALD**

25 Q. All right. So we'll slow down again.

1 A. Yes.

2 Q. I'm just going to remind you every other slide to slow
3 down.

4 A. Every slide.

5 Q. Okay. So let's go to Slide 12, please.

6 Did you follow generally accepted methodology when
7 evaluating the animal data for glyphosate?

8 A. Yes. I followed the methodology used by virtually every
9 regulatory agency and IARC --

10 Q. Can you explain that, please?

11 A. -- except for the -- except for the fact that I was not
12 able to really look at the reports from the individual studies.

13 I evaluate the study quality. That's the first thing you
14 have to do.

15 I used a full study reports, where possible. Otherwise, I
16 had no choice but to rely on summaries by the regulatory
17 authorities. EFSA had some fairly decent summaries of these
18 studies that I could work from.

19 Quality issues that I reviewed included survival -- how
20 well did the animals survive? -- weight gain; diet; the
21 substrain used. These were all issues you look at to decide:
22 Is this the right type of study? And is it a quality study?

23 The regulatory agencies did different types of analyses of
24 the data. And I wanted to be very consistent across all the
25 studies, so I re-analyzed all of the tumors, myself, flagged by

1 any regulatory authority, such as EPA, EFSA, IARC, or EChA.
2 And in addition to what was flagged by them, I included seven
3 tumor sites that were identified by Dr. Corcoran, but not
4 identified by regulatory authorities. For each case, I present
5 the Fisher's Test p-values, but that's for informational and
6 discussion purposes. I am using the Cochran-Armitage Trend
7 Test for causality.

8 Once I did that, I analyzed all these same tumor sites in
9 all of the studies, using the same sex-species strain. So if
10 the Wood Study saw malignant lymphomas in CD-1 mice, I went
11 back and looked at malignant lymphomas in all of the other
12 CD-1 Mouse studies, so I could make a direct comparison of what
13 the various studies were telling me.

14 I used historical data for rare tumors. And I used a
15 pooled analysis to evaluate all studies for a particular tumor
16 site. I performed Sensitivity Analysis, in case study designs
17 are different, or in cases where highly different control
18 responses exist. And I also did sensitivity to the pooling
19 exercise. Since they're different statistical methods, I used
20 two different methods for pooling. So that's a sensitivity
21 there, as well, to see if the pooling method makes a
22 difference.

23 Q. Okay. Slide 13, please.

24 You just mentioned historical controls.

25 A. Yeah.

1 Q. What is a historical control range, and how does that fit
2 into your methodology?

3 A. So as identify pointed out before, the concurrent control
4 is the appropriate group to use in almost all analyses of
5 animal cancer data. And everybody agrees on that: All of the
6 regulatory authorities; all of the toxicologists. "Historical
7 controls" refer to multiple controls that unexposed groups from
8 the same sex-species strain, usually from the same laboratory,
9 and usually from the same time period. They are typically used
10 in only two situations. And this is one of my big complaints
11 about the regulatory authorities and how they did glyphosate;
12 but these are the two really acceptable scientific methods --
13 places for using historical controls.

14 Rare tumors -- as I've pointed out before, it's hard to
15 pick up a rare tumor, because you get few of them, and you have
16 to be worried about it. I know an example from the NTP for
17 fluoride where we had two osteosarcomas in a mouse strain from
18 exposure to fluoride. And what was usual was the osteosarcoma
19 wasn't in the bone; it was in the muscle mass, which we've
20 never seen. And so we thought that a positive finding, because
21 having two animals with that was unheard of. We've never seen
22 it since. So clearly, something due to the fluoride.

23 If you see unusual patterns -- and the most obvious one is
24 the one I've drawn here for you: A flat dose-response. Here's
25 a case. If you look on the right, you see where it says

1 "historical controls"? So that's the range of historical
2 controls.

3 Now, what you have here is your three dose groups give the
4 same flat response. It's right in the middle of the historical
5 control range. And the control group is way down toward the
6 bottom of the historical control range. This is probably a
7 statistical -- just occurred by chance. And this would usually
8 be discarded, but here you're looking at historical controls to
9 guide you on whether to discard that finding or not.

10 Q. Dr. Portier, if scientists do not use historical controls
11 with rare tumors, how would that impact their historical
12 analysis?

13 (Reporter requests clarification.)

14 **BY MS. GREENWALD**

15 Q. If scientists do not use historical controls with rare
16 tumors, how would that impact their statistical analyses?

17 A. They would -- they would not see the tumor. The p-value
18 would be less. It would be greater than .05. And they would
19 claim it is not a statistically significant finding. That's
20 why you have to use historical controls.

21 Q. And you say they would not see the tumor. What does that
22 mean: To not see the tumor?

23 A. So if I see a response that is, let's say, 0002 -- so
24 there's two tumors in the highest-exposure group -- that cannot
25 be statistically significant by any test that we're looking at

1 here if it's out of 50, which is the typical size.

2 But if my historical control group, say, is like it is for
3 hemangiosarcomas in CD-1 mice gone for 18 months -- zeroes;
4 completely zeroes on 26 studies -- then those two animals are
5 highly statistically significant when you compare the inside
6 dataset.

7 **Q.** Thank you. Next slide, please.

8 Can you please false-positive rates, and power, and how
9 they apply to the methodology that you employed to your
10 analysis of the data here?

11 **A.** Absolutely. The false-positive rate is the probability of
12 finding a chemical that causes cancer when, in fact, it is not
13 carcinogenic. So this is that 5 percent/1 percent number that
14 you're talking about. You're willing to -- you're willing to
15 take the risk that the 5 percent is strong enough that you're
16 really seeing a cancer finding. That's what the false-positive
17 rate is.

18 Statistical power is the probability of finding a true
19 carcinogenic effect.

20 (Reporter requests clarification.)

21 **THE WITNESS:** Cancer effect. The probability of
22 finding a true cancer effect.

23 This means that if truth is that this is really a
24 carcinogen, then what's my probability of picking it out in
25 this type of study using this test? That's what statistical

1 power is.

2 It's dependent upon the study design. it's dependent upon
3 the magnitude of the effect, so it's going to vary all over the
4 place; but the Cochran-Armitage Trend Test is the most powerful
5 test for linear alternatives when looking at binomial data. So
6 it is the right test, by statistical terms. That's what this
7 means.

8 **BY MS. GREENWALD**

9 **Q.** Can you go to the next slide, please? Can you explain
10 what the --

11 **THE COURT:** Hold on a sec before we do that. Should
12 we -- I'm trying to think how long we should go today, and
13 what -- if we're going to go a little bit longer, whether we
14 should take a little break right now.

15 **MS. GREENWALD:** We'll do whatever works for
16 Your Honor. So shall we -- and we can take a break now, if
17 you'd like. If you're willing to go a little longer, I leave
18 that up to -- where --

19 **THE COURT:** So why don't we take a ten-minute break?
20 And we'll go until around 4:30, 4:40, something like that.

21 **MS. GREENWALD:** Sure. Thank you.

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

23 (Recess taken from 3:52 p.m. until 4:03 p.m.)

24 **THE COURT:** Okay. You can resume.

25 **MR. GRIFFIS:** We need a moment to get our team back,

1 Your Honor.

2 (Discussion off the record.)

3 **THE COURT:** Go ahead.

4 **BY MS. GREENWALD**

5 **Q.** Dr. Portier, right before the break I think you were about
6 to explain how the false-positive rate and power fit into your
7 methodology.

8 **A.** Can I have my slide back?

9 **Q.** Yeah. 15. Slide -- I think they're just putting it up.
10 Yep. There you go.

11 **A.** Ah, yes. Here. So this is one way to evaluate the power
12 of statistical tests using a simple simulation exercise. On
13 the computer here what you've got is just a graphic showing you
14 what a null effect looks like versus what a positive effect
15 looks like.

16 The table below it, the N fold is this thing on the side
17 that says how big the positive effect is. So by using zero the
18 high dose that's at 4 percent, just like a controls -- I can
19 then run 10,000 animal studies on my computer; make them up
20 randomly. And you get a false-positive error rate of
21 4.4 percent, which is right near the target of 5 percent.

22 If it doubles from 4 percent to 8 percent at the high
23 dose, then you have a power of 23 percent. If it triples, you
24 have a power of 52 percent, and it goes up to 16 percent. So
25 it's four times more than a 4 percent. It's 75 percent

1 statistical power.

2 75 percent is a very good statistical power. 23 percent
3 is not so great. So, as I've pointed out, it's a function of
4 how strong the carcinogenic effect can be.

5 **Q.** Next slide, please.

6 You testified before about combining adenomas and
7 carcinomas because of the progression of cells to tumors. Will
8 you please explain that progression, using the chart on page
9 16?

10 **A.** Yes. We seem to have lost a line. Between normal cells
11 and damaged cells, there should be a little arrow there.

12 Cancer is believed to be a multistage process. So you
13 have a bunch of cells that are normal doing the function
14 they're supposed to do. And the thought is you get a damage to
15 DNA in that cell. Cells get damaged. DNA in cells gets
16 damaged all of the time. And there are processes within the
17 cells that take that damage, and repair it.

18 One example is you have two strands of DNA. And one of
19 them is damaged. The other one is not. The machinery reads
20 through, and fixes the one damaged cell. And it has ways of
21 figuring out which way that's supposed to go. And there are
22 lots of different types of DNA repair processes in the cells.

23 But if the damage is still on the cell -- on the DNA --
24 and the cell replicates, then that damage goes with that DNA.
25 It's duplicated. And now you have a cell that doesn't know

1 it's got damage, that's a mutated cell. And that's sort of the
2 start of cancer; but for the most cancers that happens multiple
3 times, different types of mutations, before you really start
4 seeing the adenomas and the carcinomas down the line. So one
5 of the things you look at when you're thinking about a cancer
6 study is this concept of progression of the tumor.

7 I'll take a moment, again, if you don't mind, to address a
8 question you were addressing yesterday or the day before on lag
9 time, because I'm not --

10 **Q.** Do you mean latency? Lag time is latency?

11 **A.** Latency. I'm not sure anyone defined it, and I suspect
12 that everyone had a different idea of what latency is. So
13 there are three definitions that I can think of right away for
14 latency. The first definition is in this model that I have up
15 here. The first mutation of the first cell -- from there to
16 the point where we have an observable tumor, that is a latency
17 period. And so it's clear mechanism of cancer. Latency
18 period. I suspect that's what most people were thinking about.

19 But there's a second latency period, because I get exposed
20 to a chemical like glyphosate. And it has a probability of
21 causing that mutation. And that mutation can be reversed. So
22 it takes a while before you even get that first mutation from
23 exposure to chemical. So the time from first exposure of the
24 chemical to the time of tumor is a different latency period.

25 Okay?

1 Then there's a third latency period. That is I've got
2 5,000 people in my cohort study. And I have to collect enough
3 people with the tumor to be able to detect it in the exposed
4 group. So not only do I have to expose people for long periods
5 of time, but I also have the latency, because I have to wait to
6 find a large enough number of people to see the cancer.

7 When we try to measure latency period in an epi study,
8 it's inexact. It's certainly not measuring the first latency
9 period that I gave you -- the mechanistic one -- but there are
10 some things we can say.

11 Glyphosate. Dr. Weisenburger did a --

12 **THE COURT:** Sorry could I interrupt for a
13 clarification question?

14 **THE WITNESS:** Yeah.

15 **THE COURT:** You said there were three definitions. I
16 got the first two. I didn't quite understand the third. You
17 said there were three potential definitions of latency?

18 **THE WITNESS:** So, yeah. The third one is: In order
19 for me to -- in an epi study, in order for me to be able to --
20 first I have to be able to tell you the causes the cancer. And
21 that requires having enough people with a positive response --
22 so I'm not just looking at one person -- and following them
23 over time. I have a group of people.

24 **JUDGE PETROU:** So just to interrupt, so that I
25 understand, as well -- so, for example, when we were told it

1 took 10 years in that study before we could see, is that the
2 third example that you were giving?

3 **THE WITNESS:** Yes. That's not the latency in terms
4 of the --

5 **JUDGE PETROU:** Right.

6 **THE WITNESS:** It's latency in terms of from start of
7 study, to when we could possibly see something.

8 Now, Dr. Weisenburger did a number of nice papers on lag
9 times for NHL. And in one of the papers -- I believe it was an
10 exposure to radiation -- the lag time from exposure to
11 radiation to the tumor was one year. That's fast. That's a
12 very fast period of time of time; but the radiation probably
13 caused a mutation the minute it was given to them.

14 And so that would argue that the mechanism lag time is
15 probably maybe on the order of a year, or maybe on the order of
16 two years, but it's something in that range. Theoretically it
17 could be in that range.

18 So when we think about lag time, and whether that -- those
19 early case-control studies make any sense, it's very
20 complicated to just be able to say, "No, they don't," or "Yes,
21 they do," because this whole idea of latency is very, very
22 complicated.

23 **BY MS. GREENWALD**

24 **Q.** Okay. Any other questions? Any other comments on latency
25 or this Chart 16?

1 **A.** No.

2 **Q.** Okay. Slide 17, please.

3 What are some of the challenges that scientists face?

4 **JUDGE PETROU:** I'm sorry. Can we go back? Because
5 you put that slide up, and started your conversation about why
6 do you combine adenomas and carcinomas. And so I was hoping
7 you could finish the answer to that. You started to explain.

8 **THE WITNESS:** That's the next slide.

9 **MS. GREENWALD:** That's coming with the next slide.

10 So this is the two slides together. I wish we had a split
11 screen. So Slide 17 talks about --

12 **JUDGE PETROU:** Okay.

13 **MS. GREENWALD:** -- how Dr. Portier and other
14 scientists actually combine the adenomas and carcinomas. And
15 we can go back to the other slide, if you'd like, once he gets
16 to 17.

17 **THE WITNESS:** Next slide.

18 **BY MS. GREENWALD**

19 **Q.** Do we need one more slide? I think --

20 **A.** Right there. So if you see tumor progression in a
21 study -- so I see adenomas, and I see carcinomas -- it
22 strengthens the finding that this chemical is causing that
23 particular set of cancers.

24 Problem is: It's difficult to observe tumor progression.

25 This is -- in statistics, we call this type of experiment

1 "destructive sampling." Once I look at the animal's liver, the
2 animal is dead. So I can't see that the animal had an adenoma,
3 and then later had a carcinoma. All I can do is count adenomas
4 and carcinomas in dead animals. That creates a series of
5 problems for the combined. Benign agents are not always
6 reported. These adenomas are not always reported in some of
7 the studies. Small, benign lesions can be easily missed.

8 Let's take an example. The liver of a mouse is, I
9 believe, about 3 centimeters, 4 centimeters. So I think of it
10 as a ball. Maybe that's too big. That would be too big.
11 Maybe 1 or 2 centimeters -- as a ball.

12 But the NTP takes two slices through those livers for
13 pathology. Each slice of the tissue is .002 inches thick.
14 It's thinner than paper. So the actual sampling of the liver
15 looking for tumors is .1 percent of the liver. It's very
16 little tissue.

17 Now, when you do the liver, you palpate it first. You
18 feel for bumps and lumps. And so you always cut the bumps and
19 lumps; but in terms of trying to find small benign lesions,
20 you're going to miss them, almost certainly.

21 Finally, as you go from these adenomas to carcinomas -- so
22 let's take a theoretical case. A single adenoma is growing.
23 And there's a second mutation which brings in a carcinoma.
24 That carcinoma eats that adenoma, basically. As those cells
25 replicate, they replicate faster than the adenoma. They push

1 the adenoma cells to the edge. And those -- those just get --
2 cytotoxicity takes it away.

3 And so you seldom see a carcinoma with pieces of the
4 adenoma around the edge of it. So it's very hard to observe
5 that progression. As such, then most cancer biologists would
6 say that observing progression from benign to malignant is not
7 required from a cancer bioassay. Seeing it strengthens the
8 finding. Not seeing it should not remove the finding. So if I
9 just see carcinomas and I don't see adenomas, I think that's
10 still a valid finding.

11 **JUDGE PETROU:** So the last bullet point on this slide
12 is missing the word "observing" -- right? -- because it's
13 observing the progression is not required?

14 **THE WITNESS:** Correct.

15 **JUDGE PETROU:** And in regards to going from adenomas
16 to carcinomas -- and I don't know whether you can answer this
17 question -- is the likelihood of an adenoma turning into or
18 evolving into a carcinoma -- does that vary tremendously,
19 depending on the type of tumor we're talking about?

20 **THE WITNESS:** Yeah. Certain tumors don't even have
21 those progressions. Malignant lymphomas -- the only
22 premalignant state is swollen lymph glands. And you get
23 swollen lymph glands with so many different things, that it's
24 unlikely you would look for that or see it --

25 **JUDGE PETROU:** Mm-hm.

1 **THE WITNESS:** -- in these studies, but that's my
2 understanding of the pathology of malignant lymphomas.

3 **BY MS. GREENWALD**

4 **Q.** Can we go to the next slide, please?

5 Now there, we talked earlier about animal studies that you
6 evaluated in this case. You didn't evaluate every single
7 study. Correct?

8 **A.** I evaluated every single study.

9 **Q.** I should say you didn't accept all of the studies.

10 **A.** I evaluated --

11 People have talked about the 12 animal studies. There are
12 actually 21 animal studies available for review for this for
13 glyphosate; 13 in rats. But of those 13 in the rats, 6 of them
14 are really not acceptable for an evaluation of this sort, for a
15 variety of reasons. They don't describe the glyphosate
16 properly, so you don't even know what they've tested. They
17 used 10 animals per group; that's just not big enough. So
18 there are reasons you would discard those.

19 In the mice there were eight studies. Only three were
20 acceptable for use. And that gives you the 12 that you were
21 talking about earlier.

22 **Q.** Now, are the studies that you rejected -- have they been
23 universally rejected by every scientist that's been looking at
24 this data?

25 **A.** Yeah. Of the reviews that include them, they have all

1 excluded those same studies that just were poor quality.

2 **THE COURT:** I just want to correct the record on one
3 thing. You said only -- I think you said only three acceptable
4 mice studies; and your slide says five. I assume you meant to
5 say five?

6 **THE WITNESS:** I meant to say five. I'm sorry. That
7 was a mistake.

8 **MS. GREENWALD:** Thank you, Your Honor. I missed
9 that, also. I appreciate that.

10 **Q.** So Slide 19, please. If you can, use Slide 19 to explain
11 to the Court what evaluation you undertook, using the example
12 in male Wistar rats -- male Wistar rats. And I believe that
13 was talked about, actually, with Dr. Jameson, as well.

14 **A.** Yes. So this is one set of results from a series of three
15 bioassays in male Wistar rats. The first study is -- I think
16 this is about adenomas. This is liver tumors. The first study
17 is the Suresh Study from 1996. You can see the counts there.
18 And the p-value is .374. It's not statistically significant.

19 Brammer is statistically significant, with a p-value of
20 .008.

21 And Wood is not statistically significant.

22 So the question in looking at something like that one asks
23 is, *I've got one positive, two negatives. What does that mean?*
24 How do I turn that into a question of, yes, the positive is
25 real; or no, the positive's not real, and the other two are

1 correct?

2 So scientists face that problem. And there is a way to
3 deal with it.

4 Next slide.

5 **Q.** Next slide, please.

6 **A.** But there's a second problem here that you might have
7 missed on this table, and that is that the Suresh Wistar rats
8 have a control response of almost 50 percent, and the rest of
9 the Wistar rats have a control response of zero.

10 The Wood and the Brammer study -- if you look at the
11 little blue dots and green dots here, they line up very nicely.

12 You look at those red dots. That Suresh Study is way out
13 of line.

14 So there's a second question scientists have to ask. How
15 do you handle the very high control response in the Suresh
16 Study?

17 So the answer to both of these -- next slide --

18 **Q.** Next slide, please.

19 **A.** Thank you.

20 The answer to both of these is to use pooling, and some
21 degree of Sensitivity Analysis. So the first thing you do when
22 you see something like the Suresh Study with the high
23 background is you look for a scientific explanation. So in
24 this case I went back to the original reviews done by EFSA, and
25 I looked at the diets; that they all had three different diets.

1 So then I went and looked at the components of the diets:
2 How much carbohydrates; how much protein from fish; things like
3 this. They're all pretty much identical. So I don't think it
4 was the diet that made the difference.

5 Maybe it's the substrain. They're CD-1 mice, but there
6 are substrains of CD-1 mice. There are all three different
7 substrains, so there's no guarantee that there would be any
8 difference there.

9 So I went to all of that and looked at it. I could not
10 find an explanation.

11 Now, when you take Wistar rats and grow them in one lab
12 through multiple generations, and you grow them in another lab
13 through multiple generations, they drift apart from each other.
14 And so that's a known phenomenon. So this might be some sort
15 of genetic drift in the two different colonies over time; but I
16 can't prove that, because I don't have the genetics of the
17 individual animals checked out.

18 **JUDGE PETROU:** Do you look at all -- I'm just
19 thinking back to what you were saying earlier regarding the
20 historic data. When you have a group like in the Suresh study
21 that has a such a high rate of disease in the control group,
22 are you also then trying to compare it to the historic data, to
23 see if it's in line?

24 **THE WITNESS:** Yeah, but I couldn't find a good
25 historical control dataset for Wistar rats. So -- at least,

1 not one that I believed would allow me to answer that question.

2 So, no, I didn't.

3 So the solution is to analyze the data together, and do
4 some Sensitivity Analysis. Now, in my Expert Report I used
5 simple pooling concept. It's very easy. These studies are
6 supposed to be replicates of each other. And so instead of
7 analyzing them separately, just throw them all together. Treat
8 them as one big bioassay. They keep their doses. Treat them
9 as one big bioassay, and just analyze it using the
10 Cochran-Armitage Trend Test. And then you test the sensitivity
11 to the inclusion of Suresh by doing the analysis with them in
12 there, and with this study out of there; and look to see how
13 much of an impact that has.

14 Dr. Corcoran, in reading my Expert Report, criticized my
15 use of simple pooling, and suggested I use a General Linear
16 Model approach.

17 Cochran-Armitage Trend Test is a General Linear Model. It
18 falls in that class and GLMs can be used to evaluate the
19 impacts of variables beyond dose analysis, so it is a
20 reasonable way to approach the data, as well.

21 I decided to, in addition to simple pooling, use
22 logistical regression for doing the analysis of the pooled
23 data. So you'll see two different poolings, and that allows me
24 to look at the sensitivity of the analysis method to the final
25 result. And sometimes you'll see two p-values, one of which is

1 Suresh in and Suresh out, so that we could look at how
2 sensitive all of these significant findings are to these
3 differences between the studies.

4 Next slide.

5 Q. Well, not yet.

6 A. Oh.

7 Q. Do scientists conduct pooling here to reach a particular
8 result?

9 A. Oh, no.

10 Q. Okay.

11 A. You're doing -- I should have said this earlier.

12 P-values -- I don't believe they're straight lines. I don't
13 believe a 5 percent is: Yes, it's significant/no, it's not. I
14 want to look at these p-values.

15 This pooling is giving me an idea of how sensitive these
16 p-values are to changes in the data, but it's also telling me
17 whether the trend is consistent across the multiple studies.
18 So even though I get a .3 p-value in one study, and a .008 in
19 another, that .3 may be going up ever so slightly, and the .008
20 is going up a lot more. And you put them together, it's still
21 going up, the statistics come back and says, *Yeah, you still*
22 *have a significant finding*. So it allows me to address that
23 question in an objective fashion.

24 Q. So, Dr. Portier, Monsanto, as you know, has criticized you
25 for conducting pooling here to reach a particular result. Do

1 you agree with that?

2 **A.** No, I don't agree with that. I am using pooling for a
3 particular result. I want to understand these data, and find
4 out what they're actually telling us about carcinogenicity for
5 glyphosate.

6 **Q.** So I should have said you didn't do it for a particular
7 outcome. Correct?

8 **A.** That's correct.

9 **Q.** Is the pooling you conducted here the type of analysis you
10 would have performed in the 30-plus years you worked for the
11 federal government designing, evaluating, and investigating
12 animal bioassays?

13 **A.** Yes.

14 **Q.** Interpreting animal bioassays?

15 **A.** Yes.

16 **Q.** I want to make sure the testimony's clear just now,
17 because I think at one point you interspersed CD-1 mice. All
18 of this testimony is about the Wistar rat. Right?

19 **A.** Yes.

20 **Q.** I just want to make sure that's clear.

21 **A.** Up to that point right now, yes.

22 **Q.** Correct. That's what I meant.

23 **A.** Sorry.

24 **Q.** That's okay. And do scientists perform Sensitivity
25 Analyses to reach a particular outcome?

1 A. No. Scientists perform Sensitivity Analyses to -- in
2 these types of situations to see how sensitive the results are
3 to important characteristics in the data that you're looking
4 at.

5 Q. And again, Monsanto has suggested that you conducted a
6 Sensitivity Analysis here to reach a particular outcome. That
7 isn't true; is it?

8 A. That's not true.

9 Q. Okay. And is the Sensitivity Analysis that you conducted
10 here the type of analysis you would have performed in the
11 30-plus years you worked for the federal government, designing
12 and evaluating the animal bioassays?

13 A. Absolutely.

14 Q. Okay. Did Dr. Corcoran conduct a pooling analysis or
15 Sensitivity Analysis of the data, to your knowledge?

16 A. No, he did not. I think he did one example, but I don't
17 think he analyzed all of the data that way.

18 Q. Okay. Next slide, please. Now you get your next slide.

19 A. Okay.

20 Q. Please explain the Sensitivity Analysis that you performed
21 in the studies identified in this slide, and how it works.

22 A. There are three major concerns I see in these studies that
23 I want to look at the sensitivity of. The Lankas 1981 Study is
24 a 26-month study.

25 (Reporter requests clarification.)

1 **THE WITNESS:** Lankas. L-a-n-k-a-s.

2 Lankas is a 26-month study. The other three studies are
3 24 months. That doesn't seem like a lot, but you're going from
4 a moderately old animal to a very aged animal in these two
5 months, and so I want to make sure that Lankas is not driving
6 the results one way or the other.

7 The second is the Wistar rats with Suresh. I showed you
8 the response for adenomas. Suresh's study had a lot of odd
9 control response to it, and so I'm going to check it for
10 everything, and look at it very carefully.

11 And finally the four studies in CD-1 mice. Two of those
12 studies are 18 months; the other two are 24 months. I probably
13 shouldn't combine all four of them, but I will. But I'm going
14 to look at the sensitivity with those combinations to how many
15 months they were evaluated.

16 **BY MS. GREENWALD**

17 **Q.** Okay. So if you'd -- all right. Next slide, please.

18 You have a legend here. What's the purpose of this
19 legend, and how are you go going to use that legend to explain
20 some of the data here?

21 **A.** Yeah. I was trying to figure out how to rapidly,
22 essentially, show you what I see in the data. And so I'm going
23 to show you tables after this point. And the tables will have
24 gray squares, red squares, and different colors of the red
25 squares.

1 Highly significant findings are the dark red. Significant
2 findings have this kind of pinkish color. And largely
3 significant, which is .1 -- .05 to .1 will have this much
4 lighter color.

5 And so what you'll be able to see in the table is where
6 the action is, sort of, and how important some of these tumors
7 are.

8 **Q.** Okay. So if you'll look at Slides 4 and then 25 --

9 **MS. GREENWALD:** Should we maybe -- Your Honors, maybe
10 we can do these -- well, I'll leave it up to you. We have a
11 number of slides to sort of go through the findings, and
12 explain how the Sensitivity Analyses and pooling worked in this
13 case. That would probably take --

14 **THE WITNESS:** Thirty minutes.

15 **MS. GREENWALD:** -- fifteen minutes. You want me to
16 go now and do this?

17 **THE COURT:** It's probably a good time to --

18 **MS. GREENWALD:** It's up to you.

19 **THE COURT:** I don't really care, either. Why don't
20 we -- just to make sure -- maybe just to make sure we're not
21 pinched for time, we should --

22 **MS. GREENWALD:** Okay. We'll do it quickly.

23 **THE COURT:** -- plow ahead now.

24 (Discussion off the record.)

25

1 BY MS. GREENWALD

2 Q. So I think maybe the best thing, since there are 11
3 numbers on this chart, let's not have any testimony too much
4 about numbers. And we can just explain the charts to the
5 Judges, so we don't have a burden on the court reporter with
6 numbers. Okay?

7 A. Okay.

8 Q. So why don't we just go through? I'm going to let you
9 walk through Slides 4 through 30, not talking fast, but move
10 through them quickly so that you can explain basically how you
11 do your pooling and your Sensitivity Analysis with the data you
12 have here in this case.

13 A. Okay.

14 Q. But you'll need to tell Pedram how you want him -- when
15 you want him to move the slides.

16 A. So this is -- this type of data will be appearing from now
17 on. You have the three -- the tumors on the left side. And
18 the middle is the study. And these are the three studies in
19 Wistar rats. Then the -- you have the p-value underneath each
20 of those studies. And then you have the pooled analysis. You
21 have the pooled analysis using a General Linear Model, and the
22 pooled analysis using the simple model.

23 So let's look at the hepatocellular adenomas we were
24 looking at before. The bracketed number under "GLM" is when I
25 excluded from the pooling. And the first number above that is

1 when it's in the pooling.

2 What do we see in this slide here?

3 We see that it doesn't matter how you pool the data. When
4 I pool the data, it's statistically significant.

5 So the pooling on the hepatocellular adenomas suggests
6 that the increase you see in Brammer holds across all of the
7 studies when you put them together.

8 **THE COURT:** Pull your microphone a little tiny bit
9 away from you. It's making a popping noise.

10 **THE WITNESS:** I'm sorry. Thanks.

11 So in this case, it really doesn't matter how you pool.
12 It's a significant finding.

13 **BY MS. GREENWALD**

14 **Q.** When you say it doesn't matter how you pool, you mean
15 whether you use GLM or simple?

16 **A.** Correct, but you have to pool to answer the question.

17 Mammary adenomas and carcinomas is quite different. As
18 you can see here, if I include the Suresh Study, I have a
19 non-significant finding. And if I exclude it, I have a
20 significant finding; again, regardless of which pooled analysis
21 I'm going.

22 So now I have a dilemma. I have to decide which is which.

23 So I went back and looked at the data for mammary gland
24 adenomas and carcinomas. There's actually a statistically
25 significant decrease, as the defenses counsel told me during my

1 debriefing -- my deposition. There is a statistically
2 significant decrease for mammary-gland adenomas and carcinomas
3 in the Suresh Study, and an increase in the Brammer and Wood
4 Study combined; again, pointing to the fact that the Suresh
5 Study appears to be not quite the same Wistar rat as the other
6 two studies.

7 And here, excluding it might -- in fact, in all of these,
8 excluding it might make much -- make more sense.

9 Skin keratoacanthoma. I'm going to spell that for you.
10 K-e-r-a-t-o-c-a-n-t-h-o-m-a. You can see here, again, no
11 matter how I pool it, it doesn't matter. It's pretty much a
12 positive finding.

13 Pituitary adenoma show the opposite. So it's positive in
14 the Wood Study; but no matter how I pool it, it disappears.
15 There is no statistical significance there.

16 And finding pituitary adenomas in females -- pooling does
17 matter in this case. I would argue after looking at these data
18 that we pooled them all together. The simple analysis is not
19 working. The GLM was doing a better job. But when you exclude
20 the Suresh Study, you can see that it becomes highly
21 statistically significant. So again, I have to look at this,
22 and decide what I'm going to say about that one tumor.

23 I think we can -- next slide, please.

24 **Q.** All right. So let's go to Slide 25.

25 **A.** Okay. There. Can we come back to this one tomorrow?

1 This is one we talked about already --

2 Q. Yes, right.

3 A. -- about: Why are tumors other than lymphomas relevant,
4 and how do they fit? And I think --

5 Q. Go through the data first. We'll start with this
6 tomorrow. We'll go back to 25 tomorrow.

7 A. That will work. That will work.

8 Q. So now we're on 26.

9 A. Okay. This is the tumor finding and Sprague-Dawley rats.
10 I'm not going to go through every single one of these. I'll
11 point out a few.

12 This is the case where the Lankas Study is 26 weeks, and
13 the other studies are 24 weeks. So again, the bracketed number
14 is the p-value without Lankas, and the p-value with Lankas.
15 What you see here is pretty much positive findings in
16 everything except for the adrenal cortical carcinomas in the
17 females. And I haven't decided exactly what I'm going to say
18 about that, but it's a weaker finding than some of the others,
19 so I don't really need it. You'll see that at the end, when I
20 address the findings from here.

21 Next slide.

22 Q. Next slide.

23 A. This is the opposite. Here, these are also Sprague-Dawley
24 rats. This is the rest of the tumors in the Sprague-Dawley
25 rats. And these are all effectively negative from the pooling,

1 even though you see some positive findings in the individual
2 studies. The pancreas islet cell tumors here are in here --
3 you talked about that earlier -- but they're in here because
4 the regulatory agencies sought significant values for the
5 Fisher's Test; significant p-values. And so they mentioned it.

6 And since I said I was going to look at all of the tumors
7 that the regulatory agencies looked at, I also included this
8 tumor, even though the trend test is not statistically
9 significant.

10 Q. Next slide, please.

11 A. CD-1 mice. They hemangiosarcomas in male CD-1 mice. You
12 can see that the Atkinson and Knezevich & Hogan study are 24
13 months; Sugimoto and Wood are 18 months. So you actually have
14 three pools of analyses here. You have the 18 months by
15 themselves; the 24 months by themselves. And they're all five
16 of them together.

17 What you see here for the hemangiosarcomas is pretty much
18 a positive finding. The Wood Study had no hemangiosarcomas at
19 all, so it was zero across the board. The Sugimoto Study had a
20 small increase in tumors. But for my historical control
21 database that I used, I had 26 studies for 28 months in
22 CD-1 mice with hemangiosarcomas, and not one of those studies
23 had a hemangiosarcoma in it.

24 And when I compared that to the historical controls, it
25 had a very significant finding. Atkinson and Knezevich & Hogan

1 were, again, the same; but Atkinson was significant by itself.
2 I didn't really have to go to a historical control dataset to
3 get that answer, but I put the historical control number here
4 for you to see, as well. I would call this a positive finding
5 in looking at the pooling, especially in the 18-month studies.

6 Now, you might ask, *Well, why don't the 24-month studies*
7 *have a positive finding, too?*

8 Well, the control response at 24 months is much higher
9 than the control response at 18 months. And so if you see just
10 a small increase with a high control response, statistically
11 you can't pick it up; but if you see it with a very low control
12 response, you can pick it up. And I believe that's what's
13 happening here. The 18-month studies are in a very low
14 control/response place, and you're able to see the trend. The
15 24-month study's not as strong.

16 **Q.** Can you go to the malignant lymphoma? Let's jump down to
17 the malignant lymphoma. And then maybe the rest the slides can
18 be self-explanatory for the Court.

19 **A.** That would be -- that would be great. The malignant
20 lymphomas --

21 **Q.** I saw a big smile.

22 So just let's go to the malignant lymphomas, since
23 obviously that's what we're talking about in this case. And
24 you can tell us whether there's any significance to those,
25 beyond what the numbers are on this piece of paper.

1 A. Sure. The malignant lymphomas -- you see the numbers
2 here. 18-month studies are all highly positive for malignant
3 lymphomas. The 24-month studies are not positive for malignant
4 lymphomas. Overall, it's a marginal finding if I throw them
5 all together.

6 The malignant lymphoma historical control rate from the
7 Ward Paper that you were talking about before is 4 percent,
8 exactly the same as the control rate that Bill was talking
9 about, but that's for 24 months; that's not for 18 months. For
10 18 months, that number's much smaller. I don't have a good --
11 I do have historical database for that, but I don't remember
12 what it was. I can't tell you what it is here.

13 Anyway, I didn't need it, because I saw a statistically
14 significant finding against the concurrent control, which is
15 always the better control. So I don't have to go look at the
16 historical controls. And, in my opinion, this is the positive
17 finding in the 18-month mice.

18 Next slide is all negative, I think.

19 Q. Right. So I think we're going to not go through anymore
20 of the individual data slides. The Court has them.

21 Your Honors, would you want to call it a day now, and then
22 we'll pick up tomorrow morning? And we should only have 15, 20
23 minutes left. And there won't be anymore charts. Well, a
24 couple.

25 **THE COURT:** Use whatever charts you want.

1 You can step down.

2 **THE WITNESS:** Thank you very much.

3 (Witness excused subject to recall.)

4 **THE COURT:** So let's talk about what we have left
5 real quick. What do we have left from plaintiffs?

6 **MS. GREENWALD:** For the plaintiffs we have Dr. Nabhan
7 on Friday. He's not arriving until tomorrow night. And then
8 we have the deposition segments from Dr. Ross and Dr. Blair.
9 And then we rest. That will be the end of our presentation.

10 **THE COURT:** And then what did you say? How much time
11 did you say you planned to take with the deposition excerpts
12 from Blair and --

13 What was the other person's name?

14 **MS. GREENWALD:** Dr. Ross.

15 **THE COURT:** Ross. Sorry.

16 **MS. WAGSTAFF:** Well, actually --

17 **MS. GREENWALD:** I think we're trying to shave it down
18 a little bit.

19 **MS. WAGSTAFF:** Yeah. Plaintiffs didn't make any
20 affirmative designations. They're just in response to
21 Monsanto's. So if they cut theirs down, we would cut ours
22 down. I think we're about --

23 **MS. GREENWALD:** We're at 40 now.

24 **THE COURT:** You mean 40, total; what both sides want
25 to put in from that?

1 **MS. GREENWALD:** No, no. So, as Aimee said -- as
2 Ms. Wagstaff said, we didn't designate anything. And we got a
3 designation from Monsanto. And to make the record full, so we
4 had full responses to what they cut, we need to put in around
5 40 minutes on either side.

6 **THE COURT:** Okay. We're going to hear all of that
7 together.

8 **MS. GREENWALD:** Correct. Correct. I don't know how
9 much. I don't know what their time is.

10 **MR. LASKER:** Ours is a little bit under 30 minutes.
11 I don't know exactly, but I guess we'll be playing something
12 for about an hour and 10 minutes, unless there's further
13 cutting options.

14 **THE COURT:** All right. So that's -- so -- and then
15 Monsanto is just -- are you just calling the four witnesses, or
16 are you calling --

17 **MR. LASKER:** Yes, Your Honor.

18 **THE COURT:** Just those four witnesses. Okay.

19 **MR. LASKER:** Yeah.

20 **THE COURT:** And it looks like the plaintiffs have a
21 little under four hours left. And the defendants have -- what?
22 -- about five and a half hours left?

23 **MR. LASKER:** I think we have more, but I don't --

24 **THE CLERK:** Exact numbers: Plaintiff has 3 hours, 49
25 minutes, 27 seconds.

1 **MR. LASKER:** That's exact.

2 **THE CLERK:** Defense has 5 hours, 48 minutes, 15
3 seconds.

4 **THE COURT:** Shall we round up?

5 **MR. LASKER:** We don't appreciate that. Talk to her.

6 **THE COURT:** I just meant by seconds, not --

7 **JUDGE PETROU:** I have the same question. Would
8 that -- remaining time include the additional 60 minutes that
9 we're going to --

10 **THE CLERK:** Yes.

11 **THE COURT:** Yeah. So it looks like we'll -- you
12 know, we can go past 2:00 o'clock tomorrow if we need to; but I
13 think that we should be finishing well before 4:00 o'clock on
14 Friday, especially if we go --

15 What's that?

16 -- especially if we go past 2:00 o'clock tomorrow.

17 **MS. GREENWALD:** Your Honor, is this a good time?
18 Sometime this week can we talk about when oral argument will
19 be? Doesn't have to be today. I know it's late in the day.
20 We all were wondering when Your Honor might schedule a date.

21 **THE COURT:** Yeah. I was thinking about that. And my
22 guess is that it probably would be useful to have. It wasn't
23 clear to me that it would be useful to have further argument,
24 but I think it probably would be. I would want to do it sooner
25 rather than later, while all of this stuff is fresh. So let's

1 all talk about that a little bit tomorrow.

2 **MS. GREENWALD:** That's very helpful. Thank you very
3 much.

4 **THE COURT:** Thank you.

5 **THE CLERK:** Court is adjourned.

6 (At 4:44 p.m. the proceedings were adjourned.)

7 I certify that the foregoing is a correct transcript from the
8 record of proceedings in the above-entitled matter.

9

10

Lydia Zinn

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March 8, 2018

Signature of Court Reporter/Transcriber Date

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Lydia Zinn

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