SUPERIOR COURT OF THE STATE OF CALIFORNIA

COUNTY OF SAN FRANCISCO

DEWAYNE JOHNSON,

Plaintiff,

vs. Case No. CGC-16-550128

MONSANTO COMPANY, et al.,

Defendants.

_____/

Proceedings held on Friday, July 13, 2018, Volume 9, Morning Session, before the Honorable Suzanne R. Bolanos, at 9:30 a.m.

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| WITNESS | DIRECT | CROSS | REDIRECT | RECROSS |
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| CHRISTOPHER JUDE PORTIER | 1951 | 2024 | | |

EXHIBITS ADMITTED

(None.)

Friday, July 13, 2018

9:30 a.m.

Volume 9

Morning Session

San Francisco, California

Department 504

Judge Suzanne Ramos Bolanos

PROCEEDINGS

THE COURT: Good morning.

Good morning, Ladies and Gentlemen.

Counsel, welcome back.

Mr. Wisner.

09:30:23 MR. WISNER: Thank you, your Honor. We recall Dr. Christopher Portier to the stand.

THE COURT: Very well.

Good morning, Dr. Portier. If you'd please return to the witness stand.

09:30:46 THE WITNESS: Good morning, your Honor. Thank you.

THE COURT: Ladies and Gentlemen, Dr. Portier remains under oath.

And, Mr. Wisner, when you're ready, you may

09:31:00 continue.

MR. WISNER: Thank you, your Honor.

DIRECT EXAMINATION (Continued)

BY MR. WISNER:

09:31:03 Q. Good morning. How are you?

A. Good morning. I'm fine. Thank you.

Q. I have two notes here from the court reporter. It says, "Slow down." So I'm going to try to do that today, Doctor.

09:31:18 Let's start off where you ended off yesterday afternoon. And we were talking about the epidemiological data in this case, and I don't want to go too much farther into the details, but I just want to ask you a few basic questions. This meta-analysis down here, did

09:31:37 it include the Andreotti data?

A. No.

Q. Did it include the AHS data?

A. Yes.

Q. How so?

09:31:46 A. De Roos 2005 data is included in that meta-analysis.

Q. So you're talking about this one up here (indicating)?

A. Correct.

09:31:54 Q. Okay. Now, if you were to redo the

meta-analysis today with the Andreotti data, first of all, would that be possible?

A. It's possible.

Q. Using the data in Andreotti as provided?

09:32:09 A. Not for ever/never use.

Q. Why is that?

A. Andreotti did not provide information on ever/never use.

Q. Is that why on this chart it says, "Not

09:32:18 provided"?

A. Correct.

Q. Now, Andreotti, they did do an intensity analysis; is that right?

A. That is correct.

09:32:26 Q. So they looked at the lowest intensely exposed? How did they divide it?

A. They used the pooling -- the algorithms they had used in the De Roos 2005 study, they look at how many years you've been using glyphosate, they look at how much

09:32:49 you used -- how much use there is per year, and then they look at other characteristics, like your use of protective equipment and other things, and they have a formula that they calculate this thing called intensity.

That formula changed, by the way, from De Roos

09:33:06 2005 to Andreotti 2018. They used different algorithms.

Q. And how was it divided up? How was the intensity weighting divided up?

A. In the De Roos study, they divided it in to -- I believe it was tertiles, which means the bottom third of
09:33:25 exposures, the medium third of exposures and the highest third of exposures. And in the Andreotti, they did quartiles, the -- breaking it up in one-fourth of each, because they had a lot more people.

Q. Okay. And in Andreotti in the fourths, as it
09:33:43 relates to non-Hodgkin's lymphoma, where were the point estimates?

A. For the?

Q. For non-Hodgkin's lymphoma and all the various four different intensity weights for exposure, where were
09:33:56 the point estimates?

A. For Andreotti, they were all below 1.

Q. So Andreotti, they're all actually -- if you were to, sort of, put it on here, because it wouldn't be proper since it's never/ever, they would be on the left
09:34:12 of the line; is that right?

A. That is correct.

Q. Okay. Now, if you were to use -- let's say you had the never/ever data for Andreotti, do you believe it would be appropriate to include that study in a
09:34:28 meta-analysis with the rest of the case control studies?

A. No.

Q. Why is that?

A. The study has some very serious flaws associated with it. They had roughly 40 percent of the people who were in their cohort of almost 55,000 people not respond to the questionnaire, so they couldn't tell whether those people had changed their exposure patterns or not since the last time they asked them, which was in 1993 to 1998.

09:34:39

So they -- they did a thing called an imputation algorithm where they use the people who did respond and their characteristics, and they build a mathematical formula, and then use that formula and the characteristics for the people who didn't respond to estimate what their exposure should have been.

09:35:03

09:35:22

That's an iffy enterprise in most cases, although it is used in epidemiology. But with this big of a proportion not responding, it -- it's questionable. Then they -- they had serious errors. They -- they took a bunch of people who did respond and put them off to the side, and then they built their formula, and then they used that formula to try to predict what the people who did respond, what their exposure really was.

09:35:47

And when they did that, then 7 percent of those people who said they were exposed were estimated by the algorithm to not be exposed, and it could be worse than

09:36:06

that, because they didn't give me the full characteristics.

So the bottom line is that they have serious exposure misclassification. That brings the relevant risk down towards 1, and then they have a bias in the exposure classification, and that can bring it below 1. And so in my opinion, what we're seeing in the Andreotti study is what you would expect to see because of these misclassification problems.

09:36:37 And so it doesn't tell me anything about what it could be, because even if the truth were 1.4 or 1.6, because of these flaws, we'd expect to see it near 1, even possibly below 1.

Q. That's what we saw; is that right?

09:36:53 A. That's what we saw.

Q. Now, this imputation issue, Doctor, isn't it true that epidemiologists use imputation to study pesticides in other cases?

A. Yes, they have.

09:37:08 Q. And so why is glyphosate different?

A. Because of this misclassification that they're getting of putting people who are really exposed in the control group. Typically, with imputation, if it's done right, you're going to get errors on the order of

09:37:27 1 percent, one-half percent, maybe as high as 2 percent.

A 7-percent difference here is a major difference, and in fact, if you look at the data across all the pesticides, because they had to do this -- the AHS study is not just glyphosate. It's lots of pesticides, and when they wrote their studies, they're doing studies on a lot of different endpoints.

09:37:45

And so they -- when you look at their prediction algorithm against the other pesticides, then what you see is as the number of people who are exposed to that pesticide gets bigger, the error gets bigger. So it's a systematic error.

09:38:06

So, for example, for malathion, there were about 50 percent of the people exposed and their exposure error was, I think, about 4 percent. But glyphosate had almost 80 -- they had more than 80 percent of the people exposed at one point or another, and they were off by 7 percent, and so it's a line that's, sort of, dropping.

09:38:24

Q. Now, glyphosate use, we discussed whether or not it changed between 1993 and 2015. Do you recall?

09:38:42

A. Yes.

Q. And I believe you stated that you couldn't really remember. You had to look at your report.

A. Yes.

Q. Did you have a chance to look at your report?

09:38:50

A. Yes. I have the numbers here.

Q. Okay. What was the change in glyphosate use between 1993 and 2015?

A. I didn't have 1993 in my report.

Q. Okay.

09:38:59 A. And I went back to the original reference in my report to make sure I knew what I was looking at. In 1995, by agricultural sector alone, okay, so that's -- that pertains to the people in the agricultural health, it's a health survey, 12.5 million kilograms were applied

09:39:22 in the United States at that time.

Q. Say that again?

A. 12.5 million kilograms.

Q. Okay.

A. In 2014, it's 113.4 million kilograms, so that's

09:39:36 roughly a tenfold increase.

Q. And if you could look at Demonstrative 1030 or Exhibit 1030 in your binder, in the second volume. Yes.

A. I don't have a 1030.

Q. Okay. Well --

09:40:12 THE COURT: Any objection on 1030?

MR. GRIFFIS: No.

THE COURT: Exhibit 1030 may be published.

MR. WISNER: All right.

Q. All right. So, Doctor, we're looking at

09:40:28 Exhibit 1030. This is taken from the EPA's report. And

can you tell the jury what we're seeing here?

A. This is showing the estimated use on agricultural land in pounds per square mile of glyphosate in 1993. The darker colors are where they spray more, and the lighter colors are where they spray less.

09:40:52

Q. And the states -- well, where were the states of the agricultural health study?

A. North Carolina and Iowa.

Q. And you'd agree with me that both of those states are yellow to lightly -- slightly orange?

09:41:06

A. Yes.

Q. Okay. All right. Now, let's look at the same data from 2015. What does this show, Doctor?

A. It's the same basic structure, estimated use on agricultural land in pounds per square mile. The same scale, I think. I don't think that's changed. And again, now you see much more dark brown, much less light yellow in the agricultural parts of the United States.

09:41:22

Q. And so the dark brown says, "Over 88.06 pounds per square mile"; is that right?

09:41:45

A. That's correct.

Q. And so if we go back to 1993, that's less than four pounds; right?

A. For the light yellow, yes.

Q. Yeah. So if it goes from light yellow to dark

09:41:58

or brownish, that could go upwards of a twentyfold increase?

A. In some areas, potentially, yes.

Q. And specifically in Iowa?

09:42:11 A. It certainly has gotten dark brown.

Q. Okay. And is that, Doctor, one of the reasons why there's an issue with regard to exposure misclassification in the AHS?

A. Yes. That is another reason there is an issue. 09:42:28 Since they took five years to ask people about their exposure experience, people at the beginning of the five years may not have been using glyphosate, but the increase was so rapid that by the end of the five years, they might have been using it, but they'd already 09:42:47 answered the question five years earlier, and they didn't get asked again, so their estimate of exposure could be wrong.

Q. All right, Doctor. I want to show you something that was shown to the jury previously.

MR. WISNER: Can you please turn on the Elmo?

Now, your Honor, I already covered this with the defendants. This is a slide from Mr. Lombardi's opening statement.

THE COURT: Very well.

09:43:09 Q. BY MR. WISNER: All right. Doctor, so this is a

slide that was shown to the jury during Mr. -- the Monsanto's opening statement, and here's what was said, I'm going to read it to you. It says, "They started studying pesticides generally, and they did what epidemiologists call exploratory pesticide studies, and what I mean by exploratory pesticide studies, what epidemiologists mean by pesticide studies, is that they're not quite sure what to look at yet. They're exploring to see what to look at. So they did studies that weren't designed to figure out the effect of a particular pesticide or herbicide. They did studies just generally to see if they could pick up any association with pesticides and herbicides generally."

Are you familiar with what an exploratory study is?

A. Yes.

Q. What is an exploratory study?

A. Generally, you have -- in epidemiology -- let's talk about case control studies. You have a population of cases, you have a bunch of controls, and you're looking to see if anything's related to that particular disease. And so you don't have a hypothesis up front. You're trying to generate a hypothesis from looking at the data, and that usually is the first study of its kind.

Q. Now, this is the slide that was shown to the jury, and you can see up here at the top it says, "Exploratory pesticide studies," and it lists a lot of the studies that are on your plot summary.

09:44:44 Do you see that?

A. Yes.

Q. And then it says the second level is glyphosate pooled studies.

Do you see that?

09:44:48 A. Yes.

Q. And there's this reference to NAPP/Pahwa.

Do you see that?

A. Yes.

Q. Then it says, "Glyphosate cohort studies,

09:44:57 De Roos 2005, JNCI 2018."

Do you see that?

A. Yes, I do.

Q. Okay. Let's be very clear. Are any of these studies -- well, let's start off with the AHS ones on the

09:45:08 bottom.

A. Okay.

Q. Did the AHS just look at glyphosate?

A. No. No.

Q. Did it just study NHL?

09:45:16 A. No. The benefit of the cohort study is they can

study any disease arising in the population.

Q. And the study was started back in what year?

A. 1993.

Q. So actually, it started just after the cancer

09:45:29 study; is that right?

A. That is correct.

Q. And it didn't study just glyphosate?

A. That is correct.

Q. So would it be fair or accurate in any way to

09:45:37 characterize the AHS as a glyphosate specific study?

A. No.

Q. What would you call it?

A. An agricultural health study, exactly like they called it. It's about health in the agricultural worker

09:45:52 population.

Q. And would it be fair to say since it was looking at all pesticides and all health outcomes it was an exploratory study?

A. In some aspects, it's an exploratory study. In

09:46:02 other aspect, it's confirmatory, because other things -- there're already things known about certain pesticides that they expect to see in their study.

Q. Okay. Is there -- looking at this chart, is there any other things that you don't think are accurate?

09:46:17 A. Well, Hardell and Eriksson is a food study, so

it obviously is in the wrong category there.

Q. So this one right here (indicating)?

A. Yes.

Q. So you're saying it should be down here (indicating)?

A. Yes. It's a pooled study from two separate studies. De Roos 2003 is a pooled study from three separate studies. That -- that's it.

Q. Okay. And this NAPP study, was that -- was that just about glyphosate?

A. No, no. That -- that's about, again, all pesticide exposures. It's a case control pooled study.

Q. That was my next question. What studies are being pooled into the NAPP study?

A. The three studies that are De Roos and the McDuffie study are being pooled into the NAPP study.

Q. So how can something be a glyphosate specific pooled study when it's pooling from exploratory studies?

A. It can't be.

Q. Okay. Having spent some time talking about epidemiology, Doctor, do you have an opinion about what the epidemiology generally says about whether or not glyphosate can cause -- or strike that -- glyphosate or Roundup can cause cancer or specifically non-Hodgkin's lymphoma?

A. So in -- with the epi, it's all Roundup. It's not -- you can't make a firm statement about glyphosate from the epidemiology data alone. In my looking at this data, I conclude that there's a demonstrated association here. In the meta-analysis, it's statistically significant. When you look at this nice flat summary here, you can see that virtually in all the studies on the right-hand side, that's consistency of the association. So I conclude that there really is an association here.

09:47:59

09:48:17

The next question is, is that association causal or is it just like the pelicans and -- like the storks and the births? I can't conclude it's causal. I can conclude that it -- causality is reasonable here, that it could be causal. There's nothing that says it can't, and there are times when you can know that it's multi-causal, like the pelicans -- like the storks and the births.

09:48:33

Because we have case control studies here where you're asking people about their exposure and they're talking -- they're thinking about what they did in the past, but they already know whether they have the disease or don't have the disease, sometimes that can create a bias. So I can't rule out that bias. Each of the studies looked at it and tried their best to address how bad it could be. But I still can't rule it out.

09:48:53

09:49:07

The effects are small. They're not huge, tenfold relative risks, and so I can't really rule out chance. And whereas most of them did a pretty good job with cofounders, some maybe didn't, but I don't think
09:49:27 confounders are a big problem in this set of data. But even still, I can't rule out that there aren't confounders.

So I come to the exact same conclusion as IARC. There's an association. It's reasonable that it could be
09:49:39 causal, but I can't rule out bias, chance or confounding.

Q. Now, Doctor, would it be scientifically appropriate to just look at the epidemiology and ignore the animals studies and the mechanistic data?

A. If the -- no. Under no condition would it be.
09:49:58 Even if I saw a strong epidemiology across the board, tenfold increased relate risk, I'd still want to look at the animal data to see if -- if there isn't something in the animal data that tells me this -- there's a confounder missing or there's something here that I'm
09:50:16 missing, because this is not realistic based upon what we know about mechanistics and animals, so it would -- it would tailor my judgement a little bit, but, no, it's never good to look at just one set of data.

Q. Is it fair to say that before you can make an
09:50:32 assessment about causality, you have to look at all the

data?

A. It's -- that's common practice. It's good practice.

Q. All right. Let's turn to the last, sort of, pillar of science. Let's talk about the mechanistic data in this case.

A. Okay.

Q. Let me start off with a simple question. Doctor, is there a lot of them?

A. Yeah, there's a -- there's a good bit of data. Here we've looked at 12, 13 animal studies. We looked at the 6 or 7 epi studies. It's somewhere -- between the various mechanisms looked at, I'd guess we're well over 100 studies in the mechanistic arena.

Q. And let's break it down to what those categories of studies are. So we have in human *in vivo*. What is that?

A. You have six categories of mechanistic information of these types of studies. You have studies where, for example, a human population has accidentally been exposed to glyphosate and somebody measures something in them. So that's a human *in vivo* study. That's in the human body.

Then you have studies where people have taken blood from humans or have taken cells from humans and put

them in a petri dish and then exposed those cells to glyphosate or the glyphosate formulations. That's *in vitro*. That's what that means, *in vitro*.

You have the same for animals, and you usually break it out into mammals and non-mammals. So you have six categories: Human *in vivo*, human *in vitro*, mammal *in vivo*, mammal *in vitro*, and then other animals *in vivo* or *in vitro*.

Q. And -- so you looked at all these studies. What mechanisms have you identified that you think are relevant to the issue of causation?

A. Well, I looked at all the data that could have been for any particular mechanisms, but there are only two that have sufficient amount of data to actually make any sort of decision. The first is genotoxicity, so direct damage to genetic material in the cells.

And the other is oxidative stress, which is the cell runs on oxygen. I mean, it's a major component of the chemistry that goes on in the cell, but oxygen's very reactive. It likes to react with everything. That's why it burns so well. In the cell -- but the cell has machinery to control that, okay, control that oxygen.

But when you get too much oxygen, it begins to bind to things in the cell that it shouldn't bind to, and that can cause damage within the cell, which has been

shown in some cases to be associated with cancer.

Q. All right. So let's break those two mechanisms down. Let's talk about genotoxicity.

A. Okay.

09:53:34 Q. How do you determine if something is damaging, you know, genetic material?

A. Oh, there's a lot of different assays for doing that.

Q. Stop right there. What's an assay?

09:53:46 A. Oh, I'm sorry. An assay is an experimental study where you've got -- it's a controlled laboratory-type study where you've got things exposed and not exposed, so the annual cancer studies are cancer bioassays, so scientists talk about assays. That's their

09:54:12 experiment laid out.

Q. So would a -- a really simple way of saying it is it's a test?

A. It's a test.

Q. Okay. And what sort of tests or ways do you

09:54:21 look at to explore whether or not there's genetic damage in the cell?

A. Well, now you're getting really technical. When you -- when you damage DNA, you usually break it in some way, shape or form, and when you break the DNA, when the

09:54:34 cell tries to repair it, sometimes it leaves little

pieces of DNA sitting around, and you can measure those and look at them.

Q. What are those called?

A. Yes?

09:54:47 Q. What are those called?

A. Micronuclei would be one example of that.

Q. Okay.

A. Sometimes when cells get damaged, the DNA can misconnect, so you can look for what's called sister

09:55:02 chromatid exchanges. The DNA flips itself. It's a pair and flipping back and forth. You can look for those. There are other things you can look at, but those are two of the major ones.

Q. And have those tests been done in various forms

09:55:20 of animals and humans and -- cells -- and non-mammal cells?

A. Oh, yes. There are -- they have tests in human cells, tests in animals, tests in animal cells. They have tests in frogs and fish and all kinds of things.

09:55:43 MR. WISNER: Your Honor, at this time, request permission to publish Exhibit 1025. It's a demonstrative --

THE COURT: Any objection?

MR. WISNER: -- from his report, Table 17.

09:56:08 MR. GRIFFIS: No objection.

THE COURT: All right. You may proceed.

Q. BY MR. WISNER: All right, Doctor. I'm looking at a summary of genotoxicity studies. Where is this document from?

09:56:25 A. That's a table from an expert report I wrote.

Q. Okay. An expert report you wrote in this case; right?

A. Correct.

Q. Okay. And what we have here is summary of human

09:56:37 genotoxicity studies, and I just took out the portion related to humans. Okay?

A. Okay.

Q. Is that an appropriate -- what's the most important data to look at when you're looking at

09:56:49 genotoxicity?

A. I don't seem to have that one in my book. I have a different one under 1025.

THE COURT: So, Counsel, the slide that is on the monitor is actually labeled as Plaintiff's 1026.

09:57:03 MR. WISNER: Did I say 1025? I apologize. I miswrote that on my paper.

THE WITNESS: I knew I had notes on this.

MR. WISNER: Thanks, your Honor.

THE WITNESS: Thank you very much, your Honor. Thank you.

MR. WISNER: I got sleep, and now I can't remember anything.

THE WITNESS: So generally speaking, it depends upon the quality of the study. It depends upon how big the sample sizes are, et cetera.

09:57:23

But as a general rule, in my looking at these types of data, I would weigh data in living human beings as -- with the highest weight. I would probably follow that with the animal *in vivo* or maybe the human *in vitro*. It -- again, it depends on the quality in the animal data versus the quality of the human data, mammal, mammals. So they're, sort of, equivalent. Then the mammal *in vitro* non-human, and then the rest.

09:57:44

Q. BY MR. WISNER: All right. Now, maybe I'm missing something here, but on this chart you have an area that would be *in vivo* glyphosate.

09:58:03

Do you see that?

A. Yes.

Q. And so that would be, I guess, exposing human -- living human beings to glyphosate?

09:58:17

A. Correct.

Q. Would that be ethical?

A. If -- if there is a factory that makes glyphosate and people in the factor are exposed, then you would have a study like that. I'm not aware of any study

09:58:33

like that. You wouldn't want to do it in a laboratory, where you actually pull people in and feed them -- give them glyphosate.

Q. I guess that leads me to -- the next question is: Did they do that for glyphosate formulations? You have three studies here. Did they actually bring people into a laboratory and expose them to Roundup?

09:58:44

A. No. All three of these are -- I would call them accidental exposures -- incidental exposures.

Q. All right. Before we move on from the chart, why don't you just briefly explain what the columns are referring to and how to read the chart.

09:59:00

A. So glyphosate means -- that's pure glyphosate. Or at least the purity of the glyphosate in the study is known.

Glyphosate formulations, there were studies that used Roundup or some other formulation in their experiment.

Number positive is the number of studies that I would deem as being positive. Although, that's an over-simplistic way of looking at it. This is just a table for, kind of, keeping track in your head what's there. Because some of these studies are quite complicated and have some positive findings and some negative findings. But I've labeled them positive.

09:59:27

09:59:45

Number of negative is the number of negative studies.

The "2" is the total number of negative studies, and the "1" in parentheses is the number of studies that were submitted to the regulatory authorities from that group that are in that category. So these are studies that are submitted by industry for EPA or others to look at.

09:59:59

So that's how I distinguish between industry studies versus the other studies.

10:00:17

Q. So the number of negative is a total of two, of which one of them was just an industry study?

A. Correct.

Q. All right. And then you have the cell type or tissue. What is that referring to, high level?

10:00:29

A. Well, in the human *in vitro*, you're looking -- oh, I'm sorry. You have them both there. *In vivo* in the humans, they took blood from humans after they were exposed and looked for DNA damage. So that's peripheral blood. That's the tissue they used.

10:00:47

*In vitro*, there are different types of studies. The ones in lymphocytes, they actually took people's blood, separated out the lymphocytes, put the lymphocytes into a petri dish, and then expose it to the chemical and look for changes in those lymphocytes.

10:01:04

HEP 2, GM 38, HT 1080, GM 5757 and TR 146, those are all human cells, but they are derived from some human, and then they are put into the petri dishes and made to be immortal.

10:01:24 And so they grow them up into a colony, and then they take some and they freeze them. Then they take a few of those and they grow a new colony, and they can do a study with it.

The idea would be that if I do a study with

10:01:32 HEP 2 cells, and you do a study with HEP 2 cells, we can get the same answers. So we can verify we're both doing it right. So they have these specific cell lines for that.

Q. Great.

10:01:46 Now, you mentioned the lymphocytes. Is that related in any way to lymphoma or non-Hodgkin's lymphoma?

A. That's -- I don't know.

Q. Okay. We'll ask an oncologist. We have one coming.

10:02:02 A. You should ask an oncologist or a hematologist.

Q. All right. Well, we'll get to what the numbers show generally, but I want to spend a few minutes just talking about the *in vivo* human studies right here, the two possible and one negative.

10:02:21 Do you see that, Doctor?

A. Yes.

Q. All right. What was the context of these studies?

A. Looking at my notes, they're all from South America, Central America, general area. They're -- all involve people who live near areas that are sprayed with glyphosate for various reasons. And they're being compared to people who don't live near those areas, so who aren't sprayed with the glyphosate. And they're looking at genetic markers.

Q. I believe there's three studies possible, Paz-y-Miño, Bolognesi and Paz-y-Miño?

A. That's correct.

Q. Let's talk about the first Paz-y-Miño study. How were the -- I guess, people who were studied in that study, how were they exposed?

A. So that study had 24 people who lived within 3 kilometers of a sprayed area in Ecuador. And they were measured within two months of spraying. And then they had another 21 people who lived 80 kilometers away, and they were measured at some time. And then it compared the two groups.

Q. And the group that was sprayed, were they -- were they sprayed by plane?

A. I don't -- I suspect they were. Yeah, in fact,

in this case, they definitely are. Because if I remember, this was northern Ecuador. And they're being -- they're spraying the fields for illegal drugs. They're trying to kill them.

10:03:58 Q. And then the people who were 80 kilometers away, they weren't being sprayed with glyphosate or Roundup?

A. That's correct. And there were questionnaires given to those people as well, to make sure that they weren't using glyphosate or something else.

10:04:13 Q. And what does the data show in that study, as it relates to the people -- well, strike that.

Did they compare the people who were sprayed versus the people who weren't sprayed?

A. Yes, they did. And they saw significant

10:04:24 increase in DNA damage.

Q. Okay. Then there's another study, Bolognesi. Tell us a little bit about that study.

A. That's a different study. That -- the Bolognesi study was in 2009. That was in five separate small

10:04:39 cities or small settlements within -- what's the country here? I wrote it down. It's not Ecuador.

Q. Columbia.

A. Columbia, I believe. And so what they did was one of those cities lived in -- was next to a farming

10:04:57 region that was all organic farming. So, theoretically,

they have no exposure to any pesticides. But none to glyphosate as well.

Then they had four towns that were close to areas that were sprayed. And I have notes on that.

10:05:15 Let's see. Three were sprayed for drugs and one they sprayed -- the sugarcane fields. In between putting down sugarcane.

Then what they did was before the spraying season began, they took blood in the people in the areas

10:05:30 that were going to be sprayed, and then five -- within five days after spraying occurred, they took blood again in those four areas. And then, again, later on. Let's see. Four months later, in three of the cities -- they didn't do all four -- they took blood again to see if

10:05:48 there was still an increase in DNA damage.

Q. And what did the results show?

A. Statistically significant finding for all four cities at five days after the exposure, I believe. No, three towns showed a significant increase. And these are

10:06:04 binucleated micronuclei. One town did not.

And then four months later, three of the cities showed no change. And in one of the cities, they actually showed a decrease in micronuclei.

Q. Okay. And these are compared to the organic city; right?

A. And they are then compared to the organics, yes.

Q. So when you say statistically significant increased DNA damage, you're referring relative to the people who were not being sprayed?

10:06:30 A. They did a lot of different tests. I'd have to go back and look.

Q. Okay.

A. Because they compared the cities to themselves --

Q. Oh, I see.

A. -- before spraying versus after spraying.

But they also compared -- they compared everything. They compared five -- five days after to four months after. They compared before exposure to the

10:06:50 organic city. So they did all kinds of comparisons.

Q. And in the studies -- so I think I misunderstood. Now I get it.

So these people, they get their blood taken, get a baseline level of DNA damage.

10:07:04 A. Correct.

Q. They get sprayed five days later. They're tested again?

A. After they were sprayed, correct. After spraying occurred.

10:07:12 Q. And then in that period, we see a statistically

significant increase in DNA damage?

A. Yes.

Q. And then for most of them, four months later that damage is gone?

10:07:23 A. Yes.

Q. Okay. What does that tell you as a scientist?

A. Because they have before and after, and this is a significant event, this pretty much tells me this is fairly strong evidence in humans that you can get some

10:07:43 increase in DNA damage in blood -- peripheral blood.

Q. And the fact that it's gone four months later, is that surprising to you?

A. Not as long as there were no further exposures. That's not surprising at all. Blood cells don't stay

10:07:55 around forever. And so even though blood cells can't really repair DNA damage, they're -- they're terminal cells. So they just go away. So it's not surprising that it would disappear.

Q. So in the context of someone who's, say,

10:08:11 spraying every other day or every couple of days, that would constitute repeated insults to their DNA?

A. Yes.

Q. And is there any relationship between that repeated insult to DNA and the development of cancer?

10:08:26 A. For glyphosate, I only have the animal cancer

studies. But they didn't do DNA damage in those studies.

But in other studies for other compounds that do cause DNA damage, you've seen that chronic exposure to DNA damaging agents can lead to cancer.

10:08:47 Q. And then finally there's the last study, the Paz-y-Miño study for 2011; is that right?

A. Correct.

Q. And that one was a negative study?

A. Correct.

10:08:57 Q. What did that show?

A. No effect. They looked at also alterations in a general area. They weren't looking at micronuclei. But they saw no effect. But the time taken after the exposure is much longer.

10:09:12 Q. It's two years; right?

A. Up to two years.

Q. So it doesn't really tell us much more than the Bolognesi study. Because after two years, you wouldn't expect to see DNA damage?

10:09:26 A. It would -- it would agree with the Bolognesi study.

Q. Okay. Putting all this human data -- throwing it all into the mix -- well, actually, before -- let's look at the human data.

10:09:38 What is there, if any, significance to the fact

that there is a lot more positive studies than negative?

A. Well, most of the humans -- yes, there is some significance to that, of course. But you have to look carefully. Let's say this is corroborated -- the

10:09:58 lymphocyte studies are fairly strong corroborating studies to what you saw in the peripheral blood studies.

But the other cell lines, they have two, et cetera. Those are additional information but not as strong information to add to this. Because there's

10:10:18 isolated studies. There's no additional copies of the same study. It's hard to say.

But the fact that they're all positive is positive information on genotoxicity.

Q. So, Doctor, based on your expert opinion, having

10:10:34 reviewed the genotoxicity data, not just in humans but in all other species that you could find -- you said, like, 100 studies or so -- what is your opinion about the genotoxicity of -- let's break it down -- the genotoxicity of glyphosate?

10:10:47 A. Glyphosate is genotoxic.

Q. What about the genotoxicity of glyphosate formulations?

A. The glyphosate formulations that have been looked at are genotoxic.

10:10:58 Q. Are they more genotoxicity than just glyphosate?

A. That varies. There were studies that did both the glyphosate and the glyphosate inflammation. Some of those studies saw an increase, some of those studies saw a decrease. In general, if I were pressed, I would say the formulations are slightly more genotoxic.

10:11:14

Q. Okay. Let's talk about oxidative stress. I understand you've reviewed the oxidative stress studies done related to glyphosate in Roundup?

A. Yes, I have.

10:11:28

Q. And is there as many studies about oxidative stress as there are about -- actually, Doctor, let's not talk about oxidative stress. There's something else I wanted to talk about. I almost forgot.

Let's talk about micronuclei.

10:11:42

A. Okay.

Q. First of all, is there any science or data that you're aware of that suggests that micronuclei are associated with cancer?

A. Yes. That's why they're required in regulatory

10:11:52

submissions. Most regulatory submissions include a micronucleus test in mice.

Q. And was there a meta-analysis done of micronucleus studies as it relates to glyphosate and glyphosate formulations?

10:12:05

A. Yes, there were. Yes, there was a meta-analysis

done. It was done by Ghisi, 2016.

Q. Okay. Let's take a quick look at that. That's Exhibit 766 in your binder. It should be in your second volume.

10:12:33 Is that a fair and accurate copy, when you get to it?

A. Yes, that's --

Q. Is it "Ghisi" or "Ghisi"? Do you know?

A. I don't really know. Yeah, that's -- that's the

10:12:48 study.

MR. WISNER: Permission to publish, your Honor?

THE COURT: Any objection?

MR. GRIFFIS: No objection.

THE COURT: All right. Very well.

10:12:58 You may proceed.

Q. BY MR. WISNER: So this is the study, Doctor; is that right, on the screen?

A. That's correct.

Q. The title is: "Does Exposure to Glyphosate Lead

10:13:08 to an Increase in the Micronuclei Frequency, a Systematic and Meta-Analytic Review"; is that right?

A. That's correct.

Q. It looks like it was done by these three scientists. The lead author is "Ghisi" or "Ghisi." We

10:13:25 haven't decided how to pronounce that. Is that right?

A. Correct.

Q. All right. So I don't want to spend too much time on this. I just want to show you the -- so first of all, starting here at Table 1, this is all the studies they looked at; right?

10:13:41

A. Yes. It's -- it's all the individual doses compared to control in all the studies they looked at.

Q. And do you see Table 1 goes on for a bit? It goes on to another page. It keeps going. It goes on -- okay.

Then all those studies are put into this chart right here.

Do you see this?

A. Yes, I see that.

10:14:05

Q. Walk the jury through what this chart is showing.

A. So this is a forest plot. Just like you saw with the epidemiology data, but much more complicated because there's more data.

10:14:19

The numbers that you see next to each line are the number of the study from that big table we just looked at. They've ordered these from -- the bottom is the ones most to the left in the mean, in the center dot, to the top where they have the ones most to the right with the center mark. And the middle point here is 1.

10:14:40

Because they took log on the bottom axis, the log of 1 is 0. So this is 1, you're looking at here.

And so you can see more than half to the right. And then they did a meta-analysis. But instead of putting the meta-analysis at the bottom like I did with the epidemiology data, here they put this thing called the grand mean in the top area, where it belongs. And you can see it's highly significant. It's clearly above 1. It clearly does not include 1.

10:15:25 Q. So, Doctor, just to be clear, this is actually a confidence interval; is that right?

A. Yeah. That little plus you see right there actually is a confidence interval.

Q. So the 99 and 95 percent confidence interval is 10:15:40 incredibly small; is that right?

A. Yeah. There's a lot of data. It tends to drive that confidence bound small.

Q. And this kind of lends towards what we were talking about yesterday, that the more data you have, the 10:15:48 tighter your confidence interval gets?

A. Correct.

Q. All right. So this is the overall data. I want to show you some other charts in here that I thought were interesting and get your understanding of it.

10:15:59 This first one here is Chart A. What does this

reflect?

A. Here they've broken it down into the studies that were done *in vivo*. So in the live animals. And they broke it down into the types of animals: Mice, crocodiles, amphibians and fish.

Q. All right. If we go to Number B, what have they done there?

A. Again, they're looking at studies within individuals. But now they're looking at mammals versus non-mammals.

Q. And for both of these, all of these data points are above 1; is that right?

A. That is correct.

Q. And it's because 0 on this chart is actually 1, as you see in the plot summary; is that right?

A. Correct.

Q. Okay. Then we have this section. What does this -- oh, actually, before we go on, so we have here mammalian, nonmammalian.

Do you see that, Doctor?

A. Yes, I do.

Q. And it shows that mammalian -- is it just slightly above the grand mean? Is that right?

A. Yes.

Q. And what does that tell you, when you see that,

with regards to micronuclei formation?

A. That there's solid evidence that glyphosate can cause micronuclei in mammals.

Q. Okay. Finally we have another forest plot put together. Walk us through what this is.

10:17:22

A. Here they're looking at the way in which the population was exposed to the glyphosate. Intraperitoneal means they actually ingest it into the peritoneal cavity and the -- the glyphosate, sort of, gets absorbed through the tissues and organs there.

10:17:40

Q. This might not be surprising, but not everyone knows what the intraperitoneal cavity is. What is that?

A. It's -- like, it's here (indicating). They inject it here (indicating).

Q. If I could just --

10:17:53

MR. WISNER: For everybody, he's pointing to his abdominal.

Q. Right?

A. Yes, give or take.

Topical means it's put onto the skin. Spraying is -- I think that's the human population. That the Bolognesi study. Emersion is for fish studies, mostly, but also maybe some of the crocodiles working as well. Oral means it was fed to the animal, whatever the animal was. And not identified is -- oh, that's a different

10:17:59

10:18:22

plot. Never mind.

Q. We'll look at that in a second.

So what does this tell you about exposure and DNA damage?

10:18:32 A. Well, that it matters. The exposure matters in terms of the degree of DNA damage.

Q. And it appears that, for example, spraying is greater than oral; is that right?

A. Yes.

10:18:48 Q. And then, B, they've broken it down into, it looks like -- what is B?

A. Again, looking at *in vivo* studies, I'd have to look at the bottom here. I don't know if that's just mouse -- just mammals or not. Let's see.

10:19:13 Q. It just says "B gender." On the screen, you can see it.

Do you see it?

A. Yes. I don't -- I'd have to read in the text. But it's animals of some sort. And that's males versus

10:19:29 females versus both males and females. And then there's some where the gener is not identified.

Q. Okay. And so based on this, it looks like there's a lot more DNA damage happening in male species than in female?

10:19:43 A. Yeah. But I'd want to look at the data more

carefully to figure out what went on here. But, yeah, as a general statement about the animal kingdom, it looks like males are more sensitive.

Q. And that's a general statement I think we can extrapolate about all meta-analysis; right? They have benefits, and they have -- they have drawbacks; right?

A. Correct.

Q. And the benefit is it gives you, sort of, an overview of the data. The drawback is you kind of have to look at the individual studies as well?

A. Yes. Because -- because you have to agree with the -- that the person who collected this information and put it together has done a good job of -- of including like studies that make sense to be included together.

Q. Thank you, Doctor.

All right. Let's talk about oxidative stress. We started talking about it earlier. I stopped and went back there.

Have there been as many studies on oxidative stress in Roundup or glyphosate as there have been for genotoxicity?

A. No. There have only been a dozen or so oxidative stress studies.

MR. WISNER: Your Honor, permission to publish Exhibit 1027?

THE COURT: Any objection on 1027?

MR. GRIFFIS: No objection.

THE COURT: Very well. You may proceed.

MR. WISNER: This one we have a board.

10:21:06 Q. All right, Doctor. What does 1027 show?

A. These are the various studies that were done. The first set are studies that were done in human cells, and the second set are studies that were done in mammalian cells.

10:21:21 Q. And, Doctor, these are all looking, at least in part, whether or not glyphosate or Roundup induced oxidative stress?

A. Well, let me correct something here.

Q. Sure.

10:21:34 A. The mammal *in vitro*, there are actually *in vivo* studies in there as well.

Q. Okay.

A. So those are mammal studies.

Q. Okay. Fair enough. Thank you.

10:21:51 Does that work (indicating)?

A. Yes, that works.

Q. Let's do this quickly. I don't want to go through each one of these. We will be here all day. But which ones of these showed oxidative stress and which

10:22:06 ones didn't?

A. Mladinic. I'm going to tear up these names.

Q. Read them off.

A. Positive.

Kwiatkowska was positive.

10:22:16 Chaufan did both glyphosate and a formulation. The glyphosate was negative, but the formulation was positive.

Coalova did three different formulations. It was positive.

10:22:25 Q. All three?

A. I'd have to look at my notes.

Q. I'll just do one check.

A. Gehin was positive. Elie-Caille was positive, but I think it was an inadequate study. It was

10:22:41 questionable as to what they did.

And George & Shukla was also positive, but it was questionable so I don't include it. I don't think it's an adequate study.

Q. Terrible question.

10:22:55 A. Bolognesi was -- that was done in mice. It was positive for liver cells in the mouse but negative for kidney.

Cavusoglu looked at a formulation. Liver and kidney were both positive for one of their markers and

10:23:17 negative for the other. No, it made sense. What they

saw made sense.

Jasper did it in mice. It was positive in the liver in both males and females.

Astiz did it in male rats. This was -- this is a study where they used a different chemical -- they used glyphosate to induce oxidative stress, and then they added another chemical to try to get rid of that oxidative stress. Remember I said cells have machinery for cleaning up oxidative stress? Well, you can add that stuff, and they did that and the oxidative stress went away. So that's a positive study.

Q. Are those substances called antioxidants?

A. Yes, they're antioxidants. Some people take them as vitamins.

Cattani exposed pregnant rats and looked at their offspring. It was positive.

And George looked at mice. This was a topical study. They measured proteins and oxidative stress, and I don't have a note here that says whether it was positive or not so I can't --

Q. Okay.

A. I can't be certain.

Q. Okay. We talked about the George study as related to the tumors; right?

A. The initiation promotion study.

Q. That's right.

A. Yes. Initiation promotion. This is the same study in the same animals. They measured oxidative stress using proteins. They did a proteomic evaluation.

10:24:42 Q. All right, Doctor, I'm looking at this chart. Almost everything is positive. What does that tell you?

A. That glyphosate can cause oxidative stress in mammalian systems.

Q. If you were to give an overall weight of the

10:24:57 characterization of the oxidative stress data, what would you say?

A. The evidence is strong in a positive direction.

Q. And would you have said the same about genotoxicity?

10:25:13 A. Yes. Very strong.

Q. All right. So look at that. We got through oxidative stress in like two. A first.

All right. So, Doctor, having looked at all three areas of science: We've looked at the animal data,

10:25:33 we've looked at epidemiology, now we've looked at mechanistic data, which included both oxidative stress and genotoxicity. What is your opinion about whether or not glyphosate, and then separately Roundup, whether or not they can cause cancer?

10:25:53 A. I believe glyphosate is a human carcinogen. I

used some word in my expert report that I'm not going to pull back. I don't remember what the exact wording was. It's not absolute, but in my opinion, 90 percent or higher, I believe glyphosate is a human carcinogen.

10:26:16 Q. What about Roundup?

A. Roundup has glyphosate in it. So by that argument, and you would say immediately that Roundup is also a human carcinogen. The question then becomes is the formulation stronger or not. I can't answer that

10:26:33 because the animal studies only did the glyphosate, and humans are only the formulations. So it's hard to make that decision. And the *in vitro* stuff only gives you some indication.

So they're just both human carcinogens.

10:26:48 Q. So to a reasonable degree of medical certainty, what is your opinion?

A. Well, I'm not a medical doctor.

Q. Scientific.

A. To a reasonable degree of -- a cancer risk

10:26:58 assessment expert, glyphosate is carcinogenic, causing NHL in humans.

Q. All right, Doctor. I want to ask you about a couple other things that I think are going to come up so I'd rather just talk about them now.

10:27:17 Let's start off with a document.

MR. WISNER: Your Honor, permission to publish Exhibit 220. It's already in evidence.

THE COURT: All right. Very well. You may proceed.

10:27:28 Q. BY MR. WISNER: All right, Doctor, I'm showing you a document. It's on the screen. It's already in evidence. As you can see, this is a report, "Evaluation of the Potential Genotoxicity of Glyphosate, Glyphosate Mixtures, and Component Surfactants" by James M. Parry.

10:27:43 Do you see that?

A. Yes, I do.

Q. And this is a report dealing with some of the issues we've talked about today. Have you had a chance to look through this?

10:27:51 A. Yes, I have.

Q. All right. Let's go to -- all right. This is at the end of the report. It says "actions recommended."

Do you see this?

A. Yes, I do see it.

10:28:10 Q. And he lists a bunch of recommended things to do starting at A, B, C, D, E, F, G, H, I.

Do you see that?

A. Correct.

MR. WISNER: I'd like to -- your Honor, first to

10:28:23 publish Exhibit 207. It's already been shown to the

jury. It's a study done by Dr. Heydens.

THE COURT: Is there any objection?

MR. GRIFFIS: I do have an objection to this, your Honor. And may I approach on this line of

10:28:38 questioning?

THE COURT: Yes, yes.

(Discussion off the record.)

THE COURT: Mr. Wisner, you may proceed.

MR. WISNER: Thank you, your Honor.

10:31:51 Q. SO we're looking at the Parry report, and we have all these different action items or recommended actions that were requested.

I'm going to show you Exhibit 207. This is an article. Doctor, I had you read through this the other

10:32:04 day.

Do you recall that?

A. I'm going to look up my notes. Which exhibit?

Q. Exhibit 207.

A. Okay.

10:32:15 Q. Got it?

A. Yes.

Q. All right. And you recall that I had you take a look at this the other day?

A. Yes, I do.

10:32:21 Q. And do you see that it's authored by -- the

first author is William Heydens?

A. Heydens, yes.

Q. And then also you see Dr. Farmer as well as Mark Martens as well as Larry Kier or Kier. I'm not sure how you say his name.

10:32:37

A. Yes.

Q. So having reviewed this paper and looked at those action items or recommendations by Dr. Parry, are the things that Dr. Parry recommended in his report, are all those recommendations done in this paper?

10:32:52

A. So there were recommendations that Dr. Parry had that says do not do this. So they didn't do those in this paper. So we put those aside. I think there was two of the recommendations.

Of the remaining recommendations, the only one I can find in here is they looked at 8 deoxyguanosine, which is a measure of oxidative stress, and I believe that was one of his recommendations.

10:33:10

Q. So of all of Dr. Parry's recommendations asking for affirmative action, only one of them was done in this study?

10:33:27

A. Yes.

Q. All right. All right. Let's talk about another issue. Let's talk about the EPA.

A. Okay.

10:33:44

Q. I understand you've obviously, as someone who's worked in the Federal Government, you have had some experience with the EPA?

A. Yes, I have.

10:33:51 Q. And in fact, we talked earlier about the EPA guidelines.

Do you recall that?

A. Yes.

MR. WISNER: Permission to publish Exhibit 650,

10:34:00 your Honor. It's already in evidence. It was already discussed with the witness.

THE COURT: Any objection?

MR. GRIFFIS: I apologize. I missed the number.

THE COURT: 640.

10:34:10 MR. GRIFFIS: No objection.

THE COURT: Okay. Very well. It may be published.

Q. BY MR. WISNER: All right, Doctor. So we're looking here at the Guidelines For Carcinogen Risk

10:34:21 Assessment US EPA.

Do you see that?

A. Yes.

Q. Has the EPA come to a final conclusion about whether or not glyphosate is carcinogenic?

10:34:28 A. Not that I'm aware of.

Q. What is currently the status of the EPA's assessment?

A. They're going to list glyphosate as not a human carcinogen.

10:34:39 Q. And how do you know that? What's the procedure? What's going on?

A. Well, they drafted a review. That went out for public comment. Then they held a meeting with their science advisory panel. Then they modified the draft

10:34:56 based upon the public comments and the SAP where they thought it was important, and then they put that new draft out for public comment, and I think the comment period maybe ended. I'm not sure. I didn't pay attention to it.

10:35:10 Q. And so the procedure is at some point the EPA will finalize its opinions about its assessment of glyphosate; is that right?

A. That is correct.

Q. And let's talk about the various categories that

10:35:21 the EPA can put a substance, all right? I'm sorry, give me one second. Let me find the page.

Is it your understanding that like IARC, the EPA has different classifications it assigns a substance?

A. They have guidance on wording to use, but

10:35:58 basically it's they have guidance on how to classify into

categories.

Q. And this document -- we discussed this earlier. You have -- you actually helped create this guidance document; is that right?

10:36:13 A. I was part of the team that reviewed it internally.

Q. All right. Let's look at page 254 of this document. I'm going to put it up on the screen. The first and the highest category is carcinogenic to humans.

10:36:24 Do you see that?

A. Yes.

Q. And they describe this as descriptor indicates strong evidence of human carcinogenicity; is that right?

A. Correct.

10:36:35 Q. All right. The next category is -- hold on a second. There we go. The second category is likely to be carcinogenic to humans.

Do you see that?

A. Yes.

10:36:45 Q. And it says: "This descriptor is appropriate when the weight of the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the descriptor carcinogenic to humans."

10:36:59 So what does that mean?

A. Basically it means you haven't reached that top level, which generally requires some very solid epidemiology with clear indication of risk.

Q. Okay. It says: "Adequate evidence consistent
10:37:17 with descriptor covers a broad spectrum. As stated previously, the use of the term 'likely as a weight of evidence descriptor does not correspond to a quantifiable probability."

That's the same thing as IARC; right? They
10:37:34 don't put an actual percentage number on it.

A. That's correct.

Q. Okay. "The tables below are meant to represent the broad range of data combinations that are covered by this descriptor. They are illustrative and provide
10:37:44 either a checklist nor a limitation for the data that might support use of this descriptor. Moreover, additional information, for example, on mode of action, might change the choice of descriptor for the illustrated example. Supporting data for this descriptor may
10:37:59 include," and then it has a bunch of possibilities; right?

A. Correct.

Q. Let's look at the first one. An agent demonstrating a plausible but not definitively causal
10:38:09 association between human exposure and cancer. I'll stop

right there. What does that mean?

A. That's almost word-for-word a description of what I said with the human evidence, human epidemiology evidence, that there's an association. It's not

10:38:23 definitely causal between human exposure and cancer.

Q. Okay. "In most cases, with some supporting biological experimental evidence, though not necessarily carcinogenicity data from animal experiments."

What does that second half of the sentence mean?

10:38:43 A. They want some laboratory evidence to support the positive finding. If it's -- if it's not animal carcinogenicity studies, then you're looking for strong data on genotoxicity or oxidative stress or some of the other potential links between chemicals and the creation

10:39:06 of cancers.

Q. And in your opinion, based on the data we've shown this jury, is there some supporting biological experimental evidence?

A. Yes.

10:39:14 Q. Would you say it's a little bit more than some?

A. I'd say it's strong.

Q. Okay. All right. So based on what you've discussed today with this jury, would you agree that this exact example kind of fits what we're dealing with here

10:39:30 with glyphosate?

A. Well, I think the next example does as well, but --

Q. We'll get to that in a second.

A. -- it doesn't quite fit glyphosate because you have carcinogenicity data from the animal studies for this one.

Q. So the data we have is actually stronger than this hypothetical right here?

A. Yes, it's somewhat stronger than the hypothetical.

Q. Okay. The next hypothetical is the agent has tested positive in animal experiments in more than one species, sex, strain, site, or exposure route, with or without evidence of carcinogenicity in humans.

So what does that mean in simple terms?

A. There's no epidemiology data that's worth bringing into the argument. It's just inadequate. Either there's none there or poor studies. There's lots of reasons that can occur.

And so all you've got is animal carcinogenicity studies, and so you want more than just one study. You want -- you want more than just one finding in one study. You want to see it in different species. That strengthens it. Was it in other species, both sexes, if you see that, it strengthens it, multiple strains, that

strengthens it, et cetera. So that's what they're looking at there.

Q. All right. Doctor, based on what we've seen here, if you were to just take all the epidemiology data in this case and just burn it, throw it away, and we just have the animal data and the mechanism data, would it fall into that category?

A. Yes.

Q. So under the EPA's own definition, even if you got rid of the epi, it would still be likely carcinogenic in humans?

A. Correct.

Q. Okay. Now I understand -- we can go through all these, but I mean, they have a lot of examples here, as you can see. Positive tumor study that is strengthened by other lines of evidence. A rare animal tumor response in a single experiment that is assumed to be relevant to humans. A positive tumor study that raised additional biological concerns beyond that of a statistically significant result. For example, a high degree of malignancy or an early onset.

These are some of the various sort of ideas in which the guidelines contemplate a substance being labeled likely carcinogenic.

A. That's correct.

Q. Okay. So if we go down the scale, the next level is suggested evidence of carcinogenic potential.

Do you see that?

A. Yes.

10:42:00 Q. And then if we keep going, there is the next level, inadequate information to assess carcinogenic potential.

Do you see that?

A. Yes, I do.

10:42:08 Q. Okay. And then the very bottom, not likely to be carcinogenic to humans.

Do you see that?

A. Yes.

Q. And that last one is what the EPA's concluded; 10:42:18 is that right?

A. That is what they proposed.

Q. Do you agree with them?

A. No.

Q. Why?

10:42:22 A. Because it's -- it's -- it's hard for me to say. The evidence to me is so overwhelming. This category is where you have evidence where virtually everything's negative. There's just nothing there that would support a carcinogenic finding, and you have a lot of evidence. 10:42:41 And so you'd say, you know, I'm pretty comfortable with

saying this is not likely to be carcinogenic to humans. That's how -- that's how you would put it into that category.

Q. Well, Doctor, let's talk about how you could get there. Let's say you took all this animal data and you managed to just remove all the tumors from the data. That would be strong evidence that it's not carcinogenic; right?

A. Definitely.

Q. Did the EPA do that?

A. Actually, it would be strong evidence that you did your studies wrong because you should see at least a few things by random chance.

But, yes, that would be strong evidence that there was nothing there.

Q. Now did the EPA essentially do that with the animal data in this case?

A. Yeah, in essence, that's really what they ended up doing because they dismissed each tumor separately and never really talked about the whole pattern of tumors that they were seeing.

Q. Did they use a cutoff for exposure to disregard tumors?

A. Yes, they did.

Q. What was that?

A. 1,000 milligrams per kilogram body weight per day. Basically you weigh the animals and then you give them per kilogram -- they never weigh a kilogram. They weigh a few hundred grams, you give them a dose. That way every animal gets a dose relative to their body size.

10:43:57

Q. And when you remove all the tumors that occurred in exposures greater than 1,000, that number, what happens to the tumors?

A. Well, you're going to lose some of the strong pairwise comparisons, the high dose compared to the controls, and you're going to lose a lot of the statistically significant trends, but not all of them.

10:44:20

Q. Is there any evidence anywhere that for glyphosate 1,000 milligrams per kilograms per body weight is the maximum tolerated dose for a mouse or a rat?

10:44:38

A. No, quite the contrary. There's evidence to suggest it is not.

Q. So by effectively not looking at anything over it, you could create robust data that there is no cancer risk?

10:44:54

A. It still wouldn't be robust -- robust enough because you would still have positives in there that make sense and that link across studies. So it would -- it would not -- for me, even, it wouldn't convince me.

Q. All right. Let's look at the epi then. All

10:45:11

right?

MR. WISNER: Permission to publish the epi chart, your Honor.

THE COURT: You may.

10:45:25 Q. BY MR. WISNER: So looking at this epidemiological plot summary, one way to sort of get rid of all this data, Doctor, is to say, hey, none of it is statistically significant; right?

A. That's one way to do it, yes.

10:45:41 Q. And I mean obviously we have a problem with De Roos 2003 because it is statistically significant even though it's adjusted for pesticides. But let's say you found a way to get rid of that as well, okay? Then you could say, hey, look, there's no epidemiology so now 10:45:58 there's robust evidence that there's no risk of cancer.

Would that be a way of getting there?

A. That would be a very inappropriate way of summarizing the epidemiology data. But I'll give you an example why it's inappropriate. So suppose I have ten 10:46:14 epidemiology data, ten studies, and every one of those studies shows me a relative risk of 1.2. And the lower bound on every one of those studies is .99.

So every one of those studies is not statistically significant, but just barely not 10:46:30 statistically significant. And I've got ten of them in

the same direction. That would be such an inappropriate scientific approach to looking at that data.

Q. Isn't that what the EPA did?

A. Partly. They also gave a lot of weight to the De Roos study and now to the Andreotti study because it's a cohort study, and in their opinion, they think it's a better study. This my opinion, I think it's not a better study.

Q. So if you get rid of the case controls, focus on Andreotti, then you could say, hey, we have some robust epidemiological evidence that it's not carcinogenic in humans?

A. Correct.

MR. GRIFFIS: Object to the continued leading, your Honor.

THE COURT: Please be careful with the leading questions, Mr. Wisner.

MR. WISNER: Yes, your Honor.

Q. What about the mechanistic data? I mean, how do you get rid of that data, Doctor?

A. Well, if I were EPA and I had no epidemiology data that was positive and I had no animal data that was positive, and I had this mechanistic data, even though the mechanistic data is strong, I wouldn't call it carcinogen. But -- so it would fall in one category

higher than this one because there is some evidence that makes you uncomfortable. So you still wouldn't put it in this category.

Q. Okay. Have you expressed your concerns about the EPA's analysis to the EPA?

10:47:53

A. Yes, I have.

Q. How have you done that?

A. During the first request for public comment on the draft proposal they were putting together, I sent them a formal set of comments about what they were doing, went through their document page by page and discussed what I was seeing that they were doing inappropriately.

10:48:11

Q. Did any lawyer ask you to do that?

A. No.

Q. Why did you do it?

10:48:26

A. As I said earlier, my entire career has been about using scientific evidence to make decisions primarily about the carcinogenicity of compounds. And we've worked for years and years to understand how to do that appropriately and how to do it so that you're really presenting good advice that can be used in policy decisions.

10:48:45

And this was just so amazingly wrong in the way they were doing it, not following their own guidelines, I just felt I had to say something about it.

10:49:04

Q. All right. Let's leave the United States. Let's go across the pond to Europe.

THE COURT: Mr. Wisner, before we move into a new topic, I think this is a good time to take the morning recess.

10:49:19

MR. WISNER: Sounds good, your Honor.

THE COURT: All right. So Ladies and Gentlemen, we'll be in recess for 15 minutes, and we'll return again at five after 11:00 on the wall clock. Please remember, do not discuss the case. Thank you.

10:49:31

(Recess.)

THE COURT: Welcome back, Ladies and Gentlemen. Dr. Portier remains under oath.

And Mr. Wisner, you may proceed.

11:06:12

MR. WISNER: Thank you, your Honor.

Q. Dr. Portier, just before the break I wanted to take us out of the United States and across the pond to Europe. I'd like to talk briefly about Europeans assessment of glyphosate and Roundup.

11:06:25

I understand you live in Europe.

A. Yes, I do.

Q. And have you been paying attention or been -- tried to look at the scientific assessments -- strike that.

11:06:35

The assessment of glyphosate that's being done

by the European authorities?

A. Yes, I have.

Q. All right. Briefly explain the process by which the assessments are done in Europe.

11:06:48 A. European Food Safety Agency is the authority in Europe on pesticide registration. The way it goes for renewal, which is what it is with glyphosate -- it was already on the market; they just want to review the literature again -- the industry puts forth a request for

11:07:11 renewal. They provide a document with their -- the science that's there and to some degree their interpretation of that science.

That document is taken by one of the member -- two of the member states, actually. One is the primary

11:07:27 lead. The member states in Europe are Germany, England, not too much longer, France, Belgium. Those are members of the European Union.

So in this case, Germany was the lead member state. They reviewed the document, they edited it, they

11:07:46 made some changes to it. They added some comments. Then that goes to the European Food Safety Agency, EFSA. And then EFSA brings together experts from all of the countries in the EU, who argue, review, decide, send it back to the Germans. They redo it. And then it comes to

11:08:07 them, and then EFSA puts forth a recommendation.

And then the European Commission takes that recommendation and makes a decision. And then that has to be accepted by parliament. That's my understanding.

Q. So at the beginning of the process, then, it looks like the industry actually prepares the first draft of the report; is that right?

A. Not always, but in this case, yes, as far as I understand it.

Q. So then it goes to the German authorities, they make edits; right?

A. Yes.

Q. Then it goes to EFSA. They discuss it?

A. Yes.

Q. And then it goes back to Germany, and then they make edits?

A. Correct.

Q. And at some point they issue a final report, and that goes to the EU, to the government?

A. To government itself, yes. And European Commission, which is the government, not the legislators.

Q. And they decide if they want to follow it?

A. And then they decide to either follow or not follow the recommendation.

Q. Now, the processes in the scientific approach that EFSA uses, is that in any way similar to IARC?

A. Their guideline document for human and animal evidence is identical to -- to that of IARC, with some minor wording differences in terms of who does it. Because at IARC it's the Working Group and at the EU it's someone else.

11:09:37

Q. So they apply the same standards. Did they come to the same conclusions as IARC?

A. EFSA came to the conclusion that the human evidence was very limited, is what they called it, but it's the same limited general area as IARC did. The animal evidence, they said, was suggestive of no effect. They called it completely negative. They called the genotoxicity data negative, and they said the oxidative stress was positive.

11:09:48

11:10:09

Q. Now, the animal data, did they do what the EPA did and exclude tumors over a thousand milligrams, kilograms per body weight?

A. They did almost identical what the EPA did in terms of all the problems they had in their evaluation.

11:10:26

Q. And do you know one way or the other whether or not EFSA or EPA had made a decision to disagree with IARC before they saw the Monograph?

A. I don't know that, no.

Q. All right. I understand that you also, like the EPA, but you expressed criticisms of EFSA's approach; is

11:10:43

that right?

A. Yes, that is correct.

Q. And I believe you said that to EFSA. They responded; is that right?

11:10:54 A. I sent a letter not to EFSA, but to the Commissioner of Health, which is -- EFSA is underneath them. And he instructed them to respond, and they did respond.

Q. And then you prepared a response to the

11:11:10 response; is that right?

A. Me and my co-authors prepared a commentary, a letter to a journal, which included our response.

Q. I'd like to talk about that letter. And was it published in a journal?

11:11:23 A. Yes, it was.

Q. Please turn to Exhibit 293 in your binder. It should be Volume 1.

A. I have it.

Q. Is this a fair and accurate copy of that letter

11:11:54 that was published in the journal?

A. Yes, it is.

MR. WISNER: Permission to publish, your Honor.

THE COURT: Any objection?

MR. GRIFFIS: No, your Honor. Oh, publish, no.

11:12:03 MR. WISNER: We're not putting these into

evidence.

THE COURT: Very well.

Q. BY MR. WISNER: So we're looking here at a copy of that letter; is that right, Doctor?

11:12:21 A. That is correct.

Q. If we look here at the title, "Differences in the Carcinogenic Evaluation of Glyphosate Between the International Agency For Research on Cancer, IARC, and the European Food Safety Authority."

11:12:36 Do you see that?

A. Yes.

Q. And as we can see right here, you are the first author; right?

A. That's correct.

11:12:45 Q. Let's start off with the conclusion that you guys came to. It's in the last page of the document. It says: "The most appropriate and scientifically based evaluation of the cancers reported in humans and laboratory animals as well as supportive mechanistic data

11:13:11 is that glyphosate is a probable human carcinogen. On the basis of this conclusion and in the absence of evidence to the contrary, it is reasonable to conclude that glyphosate formulations should also be considered likely human carcinogens."

11:13:26 And then you go into the CLP criteria. What is

that?

A. That's the criteria that's put forth by the European chemical agency on how to evaluate not just cancer studies, but the entire area.

11:13:44 Q. And their classifications, the highest one is 1A?

A. The highest one is 1A, correct.

Q. And the second one, which is the second highest is 1B?

11:13:54 A. Correct.

Q. It says: "The CLP criteria allows for a similar classification of Category 1B when there are studies showing limited evidence of carcinogenicity in humans together with limited evidence of carcinogenicity in

11:14:07 experimental animals."

So this was the conclusion of this letter; is that right?

A. The first part.

Q. Yeah.

11:14:17 A. The second part -- yes, that's the conclusion.

Q. And that's the sort of conclusion about the issue of carcinogenicity?

A. Correct.

Q. And then after that, you actually have a summary

11:14:28 here. You go through all the different things that the

Working Group did at IARC and then what EFSA did; is that right?

A. Correct.

Q. Raising various concerns point by point about things that you thought were not scientifically valid; is that right?

A. That is correct.

Q. When I say "you," you were not the only author on this paper; is that right?

A. No. There were 94 authors, I believe. I don't remember the name -- number. 96, 94.

Q. All right. So we'll start here. We got Christopher Portier is the first one, and then we have all these different scientists that joined you in this letter regarding the relation that -- well, who's right, IARC or EFSA; is that right?

A. Correct.

Q. And some of these -- some of these scientists I think we've heard of, for example, Dr. De Roos.

Do you see that?

A. Correct. Yes, I see that.

Q. So Dr. De Roos joined you in concluding that glyphosate was a probable human carcinogen?

A. Yes. And that is the same De Roos who did the two studies.

Q. The same De Roos -- okay. Actually, look at that. We have Hardell. Do you see that? He joined you?

A. Yes.

Q. That's the Hardell that we've been hearing about in all these studies?

11:15:49

A. Yes, it is.

Q. There was some discussion that if there really was such a problem, all these authors would have said, hey, this stuff causes cancer. Isn't that what they're doing in this letter?

11:16:03

A. In essence, yes.

Q. I guess my only other question is -- is notwithstanding the fact that you and 94 other scientists have concluded that IARC was right, did EFSA change its position?

11:16:20

A. No.

Q. Did the European Commission agree to fully renew IARC based on EFSA's recommendation?

A. That's what they tried -- that's what they recommended.

11:16:34

Q. And what did the actual government end up doing?

A. They ended up renewing the registration I think for four years, and then I think they intend to phase it out, but I don't know the exact wording. I didn't look at the ruling.

11:16:50

Q. Phase out glyphosate in Europe?

A. Yes.

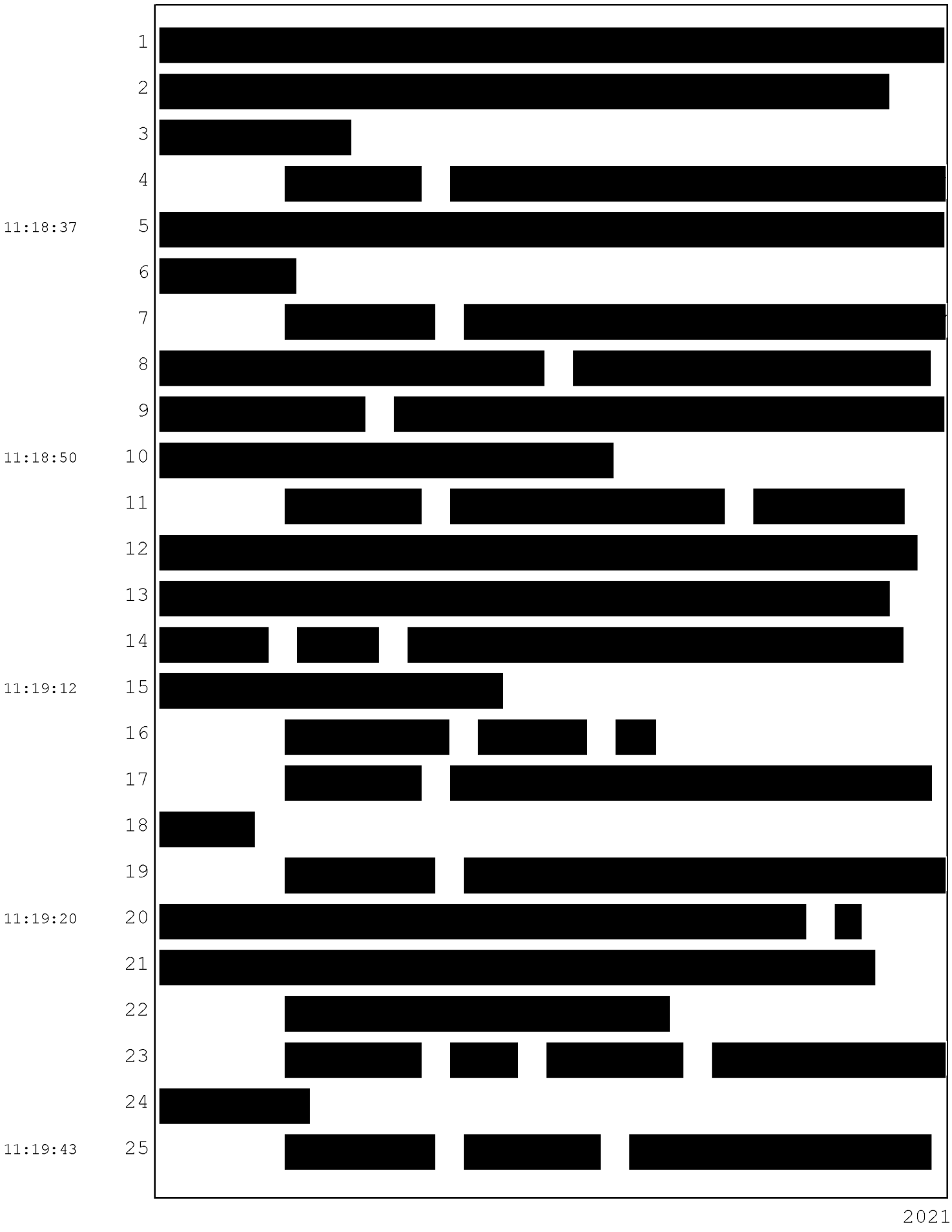
Q. Are you aware of any countries that have banned it in Europe?

11:17:04 MR. GRIFFIS: Objection, your Honor. May we approach?

THE COURT: Yes.

(Sidebar.)

[redacted]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(Sidebar ends.)

Q. BY MR. WISNER: All right. Let's move on from EFSA and IARC. Let's talk finally about something called Bradford Hill. You mentioned that previously in your direct. What are the Bradford Hill factors?

11:19:59

A. So there was a paper published from a speech given by -- I forgot his first name. Sir Bradford Hill, Ph.D. epidemiologist, M.D., in England about how to take epidemiology data and what factors play a role in leading to your decisions that the associations you see are causal and not just associations.

11:20:26

So he developed a set of factors that he felt should be used in thinking through that problem, making it clear that you don't have to have all these factors but -- but that you should look at them as you evaluate and come to a decision based upon seeing how these factors play a role.

11:20:41

Q. And is this process, the Bradford Hill criteria, is it a process that's used at IARC?

A. It's -- it's -- the IARC preamble is partially derived from what Bradford Hill put together. There's a strong linkage between the two.

11:20:57

Q. Is the Bradford Hill criteria also used by the EPA?

A. In fact, in their cancer guidelines, they talk

11:21:13

about the Bradford Hill method. So it's the preamble. So yes, it's part of that as well.

Q. And did you consider the Bradford Hill criteria in arriving at your opinion in this case?

11:21:26 A. Yes, I did.

Q. And based on the totality of the evidence -- the epidemiology, the animal toxicology, the mechanistic data -- what is your reasonable degree of scientific certainty opinion about whether or not glyphosate can

11:21:44 cause cancer and specifically non-Hodgkin's lymphoma?

A. Again, I search for words on how to say it. I -- I believe it's probable -- it's probable -- highly probable that glyphosate causes cancer in humans, and non-Hodgkin's lymphoma is the one cancer we clearly see.

11:22:07 Q. Thank you, Doctor, for your time. I'm now going to turn you over to Monsanto.

MR. WISNER: Thank you, your Honor.

THE COURT: Thank you.

Mr. Griffis.

11:22:15 MR. GRIFFIS: Yes, your Honor. I need a few minutes to get set up.

MR. WISNER: Actually, your Honor, before I finish passing the witness, I'd just like to enter into evidence the demonstratives that were used with the jury.

11:22:40 I'll get the exact exhibit numbers, Exhibit --

THE COURT: Is this something we should do now or take that --

MR. GRIFFIS: I'd say take it up later. I have objections to some that were marked up by Mr. Wisner, for example.

MR. WISNER: Okay.

THE COURT: All right. So let's take it up later.

MR. WISNER: Your Honor, this isn't going to work. I need to be able to see the witness.

11:23:41

THE COURT: Let's see. Mr. Griffis, can you --

(Interruption in proceedings.)

MR. GRIFFIS: May it please the Court.

CROSS-EXAMINATION

BY MR. GRIFFIS:

Q. Good morning, Dr. Portier.

A. Good morning, Counselor.

Q. You have testified, sir, that before --

11:24:40

MR. WISNER: Objection. Hearsay.

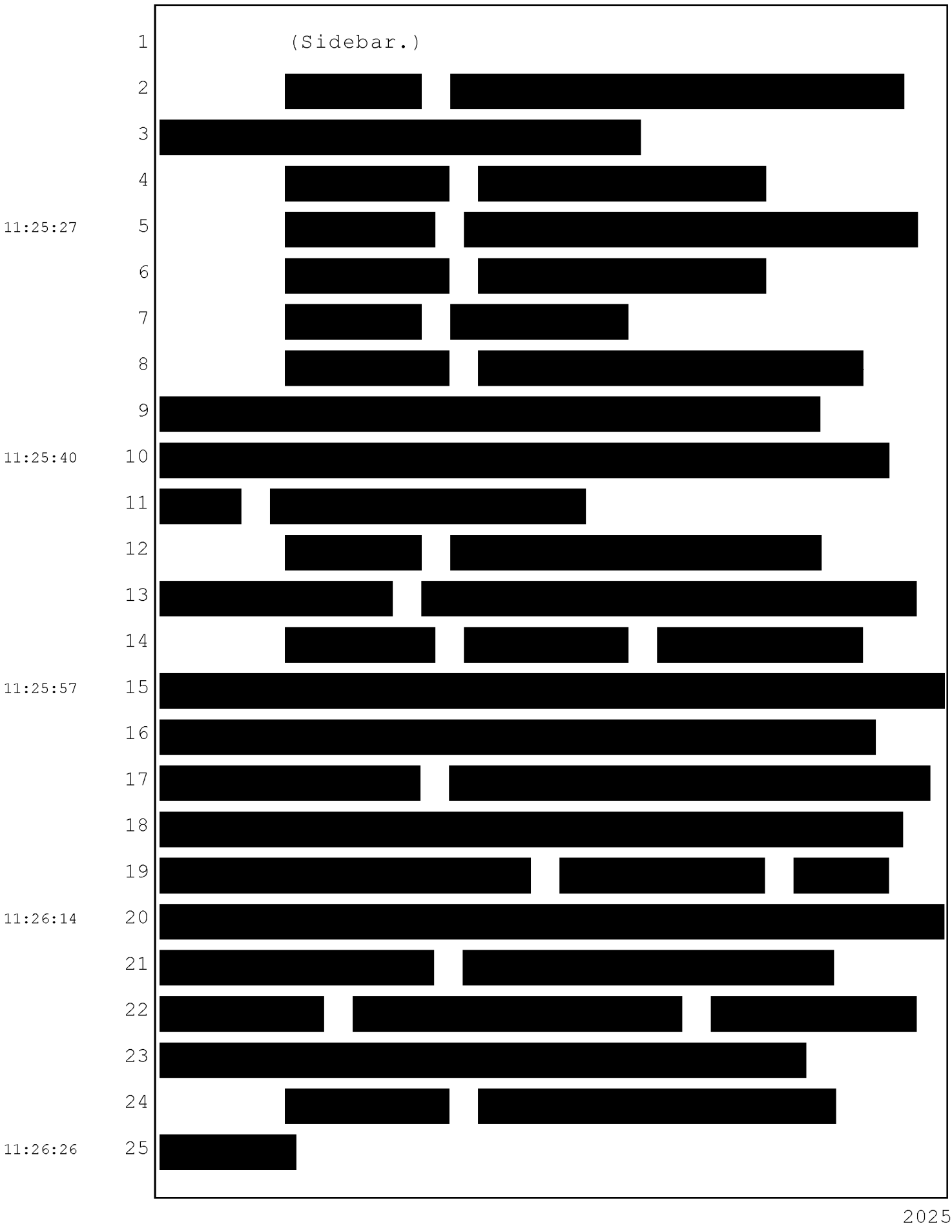
Q. BY MR. GRIFFIS: -- Working Group 112, you'd never thought about glyphosate; is that right?

MR. WISNER: Excuse me. Objection. Hearsay.

THE COURT: All right. Counsel, can you

11:24:49

approach?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[redacted]

(End sidebar.)

Q. BY MR. GRIFFIS: Sir, the first time you'd ever thought about glyphosate was when you were asked to go to Working Group 112; correct?

A. You'd have to show me what I said. I'm sorry.

Q. Is that --

MR. WISNER: Rephrase the question. That's the problem.

MR. GRIFFIS: Thanks.

Q. Sir, I'm asking you a new question.

A. Okay.

Q. The question is this --

A. Go ahead.

Q. -- Before Working Group 112, you'd never thought about glyphosate; right?

A. That wouldn't be technically correct. There was an IARC meeting a year or so before that set up priorities for chemicals for them to review in the future. Glyphosate was one of those chemicals. It

wasn't my responsibility to review the science for it, somebody else's responsibility, but certainly it came up. That's the only other time I would know.

Q. You hadn't done a scientific review of it certainly before Working Group 112?

11:27:52

A. Not that I'm aware of.

Q. The -- you know that from the preamble to the IARC findings, this is in evidence as Plaintiff's Exhibit 166, that the terms "probably carcinogenic" and "possibly carcinogenic" have no quantitative significant; correct?

11:28:13

A. From the preamble?

Q. Yes.

A. Yes.

Q. That's a correct statement?

A. That's a correct statement.

11:28:26

MR. GRIFFIS: Would you put up Slide 387, please, from the preamble, which is in evidence.

Q. So the preamble. And the preamble, this is a 2006 document that -- that binds the Monographs that were generated from 2006 on until the preamble was changed or amended; correct?

11:28:46

A. That's correct.

Q. It's sort of a document that sets forth some standards and criteria that IARC applies; right?

A. That is correct.

11:28:58

Q. And so IARC's own standard on the significance of a finding of probably carcinogenic or possibly carcinogenic is, "These have no quantitative significance and are used simply as descriptors of different levels of
11:29:13 evidence of human carcinogenicity, with probably carcinogenic signifying a higher level of evidence than possibly carcinogenic"; correct?

A. That's an exact quote.

Q. Yes.

11:29:25 And so a particular finding of probably carcinogenic or possibly carcinogenic doesn't mean 75 percent or 80 percent or 40 percent or any other percent, because they're not -- they have no quantitative significance; right?

11:29:36 A. That's IARC's view, correct.

Q. Now, Working Group 112 we know met in Lyon, France, in March 2015; right?

A. That's correct.

Q. And the responsibility -- there was a
11:29:51 three-month lead-in period when people were invited, and people that were in the Working Group -- members of the Working Group -- you weren't one, because you were an invited specialist. Members of the Working Group got assignments to compile information about various
11:30:05 subjects; correct?

A. Three months sounds a little short.

Q. Okay.

A. I would have to look at see. They have a timeline published somewhere. Three months sounds too short.

Q. Okay. Some number of months in the handful range?

A. I think it was a year.

Q. You think it was a year?

A. I think people were nominated and chosen for the Working Group a year in advance, and they could start working immediately.

Q. Okay. Do you recall giving testimony that during a three-month period before the meeting, people had responsibility to assemble data and put it into tables?

A. Review the evidence and begin to draft the reports and put it into tables. The data was already assembled. The scientific papers were assembled before then.

Q. They're assembled in the sense that people are tagging papers for one another so that they're all gathered in one place electronically so that it's easy to access; right?

A. Correct.

Q. Okay. And so some Working Group member might have been assigned to write the malathion -- that was one of the chemicals that was reviewed by Working Group 112; right?

11:31:10 A. Correct.

Q. The malathion animal genotoxicity section by gathering together the information in the data table and doing some summaries, et cetera; right?

A. Correct.

11:31:22 Q. But the evaluation process doesn't start until the beginning of that one-week period when everyone's gathered together; right?

A. That is correct.

Q. And the week that the group was doing the

11:31:33 evaluation had to be divided between glyphosate and diazinon and malathion and parathion and tetrchlorvinphos. Did I pronounce that right?

A. Close enough.

Q. And glyphosate; right?

11:31:47 A. Correct.

Q. So the group spent only about one to two days total, collectively among everyone's individual efforts, analyzing whether glyphosate can cause cancer; right?

A. Not really. The -- the chemicals you're looking

11:32:03 at here -- let's take epi for example. It's almost all

the same epi studies, so the bases are there. They're case-control studies. They looked at this pesticide or that pesticide, and so you end up not having to spend all the time evaluating the quality of the case-control
11:32:25 study, you can look at each of the endpoints separately, so that saves a lot of time.

Any animal data -- there wasn't that much animal data for others, so glyphosate got a little more time and effort in that area. And then in the mechanistic area, I
11:32:40 just couldn't recall how much time, but it's too simple to say, "Well, they got one-fifth."

Q. Do you recall testifying, sir, that you would have had maybe a day or two analysis and evaluation that went into the IARC Working Group's classification of
11:32:58 glyphosate; correct? Answer: Roughly correct.

A. A day or two? Say that again please.

Q. Sure. So you would have had -- you would have maybe a day or two analysis and evaluation that went into the IARC Working Group's calculation of glyphosate;
11:33:16 correct? Roughly correct.

A. Could I see that, please?

Q. And I -- I'm sorry. I apologize. I did not notice -- this is the testimony of Aaron Blair. Do you know who that is?

11:33:25 A. I'm sorry?

Q. This is testimony from Aaron Blair. Do you know who that is?

A. Yes, I do.

Q. Who is --

11:33:30 MR. WISNER: Objection. Hearsay. Move to strike.

Q. BY MR. GRIFFIS: Who is Aaron Blair?

THE COURT: Well, as to the prior objection, that prior objection is sustained, but he may answer this

11:33:43 question: Who is Aaron Blair?

Q. BY MR. GRIFFIS: Who is Aaron Blair?

A. Aaron Blair is an epidemiologist. He's world-renowned. He was head of the National Cancer Institute's epidemiology unit -- or one of their

11:33:59 epidemiology units. He was one of the lead scientists on the agricultural health study.

Q. What was his role in IARC?

A. He was the chair of the IARC Working Group.

Q. Okay. And this is sworn testimony of his that I

11:34:10 just read that has been designated in this case and will be played later by the parties by agreement, sir. And if he said what I said that he said, and he was one of the people that was working on this, who was actually doing the evaluation -- you were a consultant, essentially, to

11:34:26 the Working Group; correct?

MR. WISNER: Objection. Hearsay, attorney's testifying, move to strike.

THE COURT: All right. So without making reference to Mr. Blair's testimony, you may ask -- I think you asked a question whether or not he was a member of this Working Group.

MR. GRIFFIS: Yes.

THE COURT: He may answer that part of the question.

11:34:55 THE WITNESS: I was a consultant to the Working Group. That is a good description.

Q. BY MR. GRIFFIS: And Dr. Blair was the head of it and on the working committee; correct?

A. Dr. Blair was the head of it, and he's a member 11:35:08 of the Working Group.

Q. Right. And if Dr. Blair said that they had maybe a day or two, you would disagree with that?

A. I -- I'd have to read it in the context of what he said. I'm not understanding why Dr. Blair would be 11:35:27 that -- that succinct about it, because I know Dr. Blair knows it's a very complicated process, and it can't be easily summarized like that. So in order to answer your question, I need to see the context of what he said.

Q. Okay. We've been talking about whether it's a 11:35:44 day or two or a little bit longer, but it can't be more

than a week; right?

A. Again, the Working Group has looked at this evidence for months in advance, and they've evaluated it, passed it around amongst each other, so, yes, the actual discussions of the final words that go into the Monograph are during that one week, as well as the overall evaluations in each of the groups and the final evaluation.

11:36:00

Q. Working Group members and invited specialists serve in their individual capacities as scientists and not as representatives of their government or any organization with which they're affiliated; is that right?

11:36:13

A. That is what -- that is in the preamble, I think. That's what it says.

11:36:27

Q. So when an affiliation is provided on a list of members to the Monograph, such as we saw in Plaintiff's Exhibit 295 the other day during your direct testimony yesterday, those affiliations are in no way an endorsement of that agency, nor are the people who are from an agency or from some other organization in any way vouching for their conclusion on behalf of the agency or entity from which they arise; is that correct?

11:36:46

A. That is correct.

Q. Someone who is from EPA isn't saying EPA agrees

11:37:07

with this?

A. That is correct.

Q. Someone from the National Institute of Health isn't saying the National Institute of Health agrees with this and so on?

11:37:19

A. Correct.

Q. Okay. The Monograph, sir, is in evidence, which is Plaintiff's Exhibit 784.

A. I have it.

11:37:41

Q. Okay. It's also 264 in the other binder I've provided you.

MR. GRIFFIS: I'm going to use the Elmo, please.

Q. So I want to go to page 30 of the Monograph, please.

11:38:27

Are you there?

A. Yes, I am.

Q. So this is the section on cancer and experimental animals, and the 3.1 subgroup is for the mouse; right?

11:38:37

A. Yes.

Q. And you testified earlier that there are two major categories of animal data that's relevant to carcinogenicity, that would be mice and rats; right?

A. In this particular case, yes.

11:38:59

Q. Page 31 is just a table, so is 32. Then on 33,

we have the main discussion of the mouse information from this evaluation; correct?

A. Correct.

Q. I'm going to highlight two things and put it back up.

Now, this page, and the preceding page of tests that happens before the table that we skipped over for the time being, is talking about two different mouse studies; correct?

11:39:57 A. I'm going to have to look.

Q. Okay. Go ahead.

A. So starting from the beginning of 3.11?

Q. Yes.

A. Okay. This is talking about one study.

11:40:31 Q. This portion over here on the left is talking about which study, sir? Knezevich?

A. I believe it's Knezevich & Hogan, because of the tumor counts that are looking there.

Q. And then over here is the Atkinson study; correct?

11:40:48 A. The -- the bottom paragraph you have on that side?

Q. Yes.

A. Okay.

11:41:12 No. I don't believe this is Atkinson.

Q. Which study do you think it is?

A. I'd have to go look. Atkinson study had, I believe, five exposure groups. This one only has four.

Q. Sir, we've got two mouse studies here that were considered by IARC in its evaluation; right?

A. Yes.

Q. And the sources that we see are JMPR, EPA, EPA, EPA, EPA. It's mostly agency reviews; correct?

A. That is correct.

Q. They did not look at the original data for these studies; right?

A. That is correct.

Q. And the finding for the Atkinson study here was the significant finding -- the finding that the Working Group considered significant is what, please?

A. So the second study?

Q. The first, the Atkinson.

A. The first one?

Q. I apologize. The Knezevich.

A. Knezevich & Hogan?

Q. Yes.

A. They found an increase in carcino- -- renal tubule carcinomas and renal tubule adenomas.

Q. And what were the renal tubule adenomas in the second study?

A. They don't give it, do they?

Q. No.

A. No. They're not there.

Q. But you know, because you looked, that it's a statistically significant negative trend; right? 2200?

A. I'd have to go look.

Q. Okay. And the second study up here is the significant finding for hemangiosarcoma; right?

A. That's correct.

Q. And in Knezevich, the first study, the hemangiosarcoma score was 0000, totally not significant; correct?

A. I would have to go back and check.

Q. If my numbers are right, sir, then each study would provide evidence against a consistency -- a consistent tumor finding with regard to the other study; right?

A. I disagree. I've shown you my interpretation of the consistency of the studies.

Q. Of all the studies?

A. Of all the studies.

Q. Yes.

A. All at one time, not one against the other. All of the studies.

Q. Well, I'm talking about these two, because these

two are the ones that IARC looked at, and I'm interested right now in the evidence that IARC had available to it. We'll certainly turn to your report later, sir.

A. IARC did not have the zeros. The hemangiosarcoma count in Knezevich & Hogan had not been published, so they didn't have them.

11:43:44

Q. And they didn't have them -- my question right now is: What -- not whether IARC ignored something that they knew they had but that that sort of evidence would tend to demonstrate inconsistency between the two. If you have a tumors that appear in -- if you have two studies and tumor A appears in one, but is negative or a negative trend in the other one, that would be weaker evidence than having it appear in one and some equivocal finding in the other; right?

11:43:59

11:44:16

A. No.

Q. Why not?

A. Well, so let's take the hemangiosarcomas. Okay. A zero response in hemangiosarcomas is not unsurprising since it's a fairly rare tumor, 0 across the board. The other study saw a clear 0004. So I would have to look at the doses that were used, compare the doses. But at the same time -- are these the same mice? Yeah. They're both CD-1, so that's something I'd want to look at, but there are other aspects.

11:44:27

11:44:49

But the point is: If I have two studies -- two animal studies and one's positive and one's negative, all of the guidelines talk about the fact that the current control is the correct control to use, and seeing one positive and one negative, I don't actually know what to do with that, other than to get down into the study, look at the quality of the studies and try to decide from that, because it could be just as wrong that the one with all zeros is the random wrong study. So you really have to get into the body of the evidence.

11:45:04

11:45:23

Q. You have to analyze the evidence, and you have to analyze it not just statistically, but biologically; right?

A. Correct. You have to look across the whole thing. Hemangiosarcomas are a problem, because they don't have precursors. So there's not a lot of -- biologically you can do with that from the pathology we're looking at.

11:45:36

Q. You testified, I believe, yesterday, if it wasn't a little earlier today, that you need pathologists to help with biological evaluation. That's a whole side of this analysis; correct?

11:45:43

A. Well, you need the pathologists to identify the tumors for you. That's quite clear. You need the pathologists to identify any precursor lesions, any other

11:46:00

toxicity in the -- in the data that you're looking at. But you don't necessarily need a pathologist to help you interpret the data once they've done it.

Q. You were asked by the Working Group members who were analyzing these mouse studies, at least the one on the left, Knezevich, for assistance in evaluating from their statistical analyses; right?

A. Yes.

Q. They asked if you could help them find a Cochran-Armitage test. And that was run on the Knezevich study, the one on the left. And they asked you to verify the statistical analysis that was done; right?

A. That's correct.

Q. Okay. And they used something called an approximate trend test to do that analysis; right?

A. They used the approximate estimate of the P value from the Armitage linear trend test.

Q. And there is also an exact test that can be used in that circumstance; correct?

A. There is also an exact -- an exact calculation of the P value that can be used for that same test.

Q. Now, the exact test, when you have rare tumors -- like this is; correct, sir?

A. This isn't -- both of those are rare tumors, yes.

Q. Okay. So the exact test -- when you have a rare tumor, you don't have very many data points. Like, it's not 5, 7, 9, 12, so you have a whole lot. You have 1, 1, 2, 0 or something like that. The exact test gives you
11:47:35 exactly the right value. And the approximate test can give you an erroneous value; right?

A. Well, let's not confuse P values with truth. so for that test, under the assumptions of that test and the statistical model that's derived for it, the
11:47:55 exact P value gives you the exact P value, whereas the approximation is, indeed, an approximation based upon an assumption.

Q. During the course of your back and forth with the EPA, sir, two biostatisticians -- two other
11:48:12 biostatisticians, Dr. Haseman and Dr. Truong, pointed out that right here, this test that was used, that you validated at IARC, should have been an exact test, not an approximate test. And you agreed with them about that; right?

11:48:25 A. I agreed that it would have been better to do the exact test. But the approximate test is a valid test that is used in numerous animal cancer bioassay reports. I just want to be clear on that.

Q. Okay. And, yeah, but with different numbers
11:48:40 than these particular numbers; right?

A. That's right.

Q. The exact test is best for these numbers?

A. It would have been better to use the exact test here.

11:48:47 Q. And the choice of what statistical tool you employ can make a difference as to whether something comes out statistically significant; correct?

A. That's correct.

Q. Now, in this case, this was reported by IARC.

11:48:58 And the animal group understood, in part because you helped them validate that statistical finding, that this was statistically significant. And had the exact test been used, it wouldn't have been; correct?

A. The P value would have been .06 instead of .03

11:49:16 or whatever it was.

Q. It would not have been significant under the .05 standard that you discussed yesterday?

A. It would be marginally significant.

Q. .05 is considered not significant; right?

11:49:24 A. No. Not in my -- not in my opinion. It's marginally significant.

Q. So you don't believe in statistical significance versus not significant?

A. I think that's drawing too tight of a line to

11:49:38 explain a body of evidence.

The P value explains what you see. There's a 6 percent chance that the slope that you're seeing is -- how would we put this? That -- there's a 6 percent chance that the slope you're seeing arose from data that was totally flat.

Q. When you reworked this and you applied the correct test, it came out to be greater than .05; right?

A. That's correct.

Q. And .05 is the number this is used in the 95 percent confidence level that you talked about yesterday as well; right?

A. They use .025 on either end.

Q. And 95 percent confidence interval corresponds to a .05 P value, which corresponds to a 1 in 20 chance of having that happen by chance alone; right?

A. There are numerous publications in the statistical and epidemiological literature telling you not to do this, yes or no. That statistically significant is a guide. But you really need to look at P values or the range of the confidence intervals to make some sense of the data that you're looking at.

So you can call it statistically significant, if you want. I'm going to tell you it's a P value of .06.

Q. How many mouse studies do you consider to have significant information about carcinogenicity in your

testimony here yesterday and today?

A. How many mouse studies?

Q. Yes.

A. Or all studies?

11:51:12 Q. Mouse.

A. Mouse studies, five.

Q. And for IARC, that was two?

A. Yes, I think so.

Q. Immediately after IARC, sir, you published a

11:51:25 little opinion piece in the journal Horizon. And you said that you didn't think the rat studies showed any statistically significant associations; right?

A. I'd have to see it.

Q. Okay. This is -- turn to 2931 in your binder.

11:51:51 It's the last tab.

A. Yes, I remember the article.

Q. Okay. So this is an article in which you were interviewed; correct?

A. No. This is an article I wrote myself.

11:52:04 Q. You co-wrote with Jose Tarazona. Who is he?

A. He's the head of the pesticides unit at the European Food Safety Authority -- or Agency.

Q. And it's a yes/no article. What is that?

A. It's like a debate. I argued the "yes" side,

11:52:20 that glyphosate was carcinogenic. He argued the "no"

side.

Q. And you said, sir, "With the exception of growth and a few nonmalignant tumors" -- I'm in the -- towards the bottom of the large paragraph in the middle column.

11:52:42 "With the exception of growth and a few nonmalignant tumors, none of the rat studies showed any effect"; correct?

A. That's what it says. That's not what I believe now.

11:52:56 Q. Right. Now you believe how many rat studies show a significant effect?

A. I'd have to look at my chart again. They certainly -- there's a lot of significant findings in the rat studies.

11:53:07 Q. And you testified yesterday that you looked at about 5 percent more information than IARC did in reaching your conclusions that you hold today; right?

A. Yes.

Q. Well, as far as the mouse studies go, you must

11:53:24 have looked at a whole lot more than 5 percent; correct?

A. Correct.

Q. As far as the rat studies go, you must have looked at a whole lot more than 5 percent; correct?

A. That is correct.

11:53:35 Q. As far as much epidemiology goes --

A. It's pretty much the same.

Q. -- PHS study is a very large and new piece of information; right?

A. Which study?

11:53:41 Q. The one that was punished in the journal of the National Cancer Institute in 2018.

A. The Andreotti study that I talked about earlier. So what was your question?

Q. That's more than 5 percent of the information

11:53:52 that exists in the epidemiology world; right?

A. No.

Q. It's more than 5 percent of the exposed people that are reported in epidemiology; right?

A. That's correct.

11:54:07 Q. It has the largest number of exposed people of any epidemiology study; right?

A. Yes. I would have to say yes.

Q. I want to talk about the Greim paper, sir.

A. Okay.

11:54:24 Q. The Greim paper was your main source of information about the mice and rats; correct?

A. That's not correct. The main -- so it's -- the Greim paper had an appendix. The appendix has the tumor count data for -- in various formats for the 12 rat and

11:54:51 mouse studies that I focused on. But it doesn't provide

all the other information you need.

So I got information from EFSA, from EPA, from what Greim actually wrote. All of that played a role in -- in my evaluation.

11:55:08 Q. Okay. You have said your main source of information was not the paper itself, but the appendix?

A. That's correct.

Q. Okay. And the Greim paper, we can find it with its appendix at Exhibit 2570; correct?

11:55:30 A. Yes.

MR. GRIFFIS: I move to publish that.

THE COURT: Any objection?

MR. WISNER: No objection to publication.

THE COURT: Very well.

11:55:40 Q. BY MR. GRIFFIS: Now, a little background here. There are six different companies that EPA has approved to sell glyphosate-based herbicides in the US; right?

A. I wouldn't know.

Q. It's about that number?

11:55:53 A. I wouldn't know.

Q. You know that it's more than just Monsanto?

A. Not really.

Q. Do you know that each of the rat and mouse studies that you were talking about the other day in

11:56:03 those boards that were displayed were either -- were

solicited by and done in good laboratory practice labs on behalf of pesticide manufacturers?

A. Absolutely. But I don't know if those were submitted in the United States or submitted somewhere else.

11:56:24

Q. Okay.

A. So I don't know how many people are registered to sell -- to produce and sell glyphosate in the United States.

11:56:31

Q. Okay. You were asked which of those is a Monsanto study. And you said you don't know. You didn't really care about that; is that right?

A. That's correct.

Q. Okay.

11:56:39

A. I'm looking at the data that's in front of me, the science.

Q. You do know this -- when someone wants to sell a -- sell a pesticide or herbicide or pretty much any other chemical substance, they need to get EPA approval first?

11:56:53

A. Not all substances need EPA's approval first. But we'll stick with pesticides. Yes, pesticides. I sat on the science advisory panel for five years. Yes, they absolutely must submit a variety of studies, including 2-year or 18-month chronic carcinogenetic studies.

11:57:10

Q. Okay. And you said a whole variety, including carcinogenicity studies, because there's a whole bunch of other categories, too?

A. Correct.

11:57:20 Q. There are two toxicity tests, dermal tests, eye tests, et cetera, et cetera, et cetera. But what we've been talking about are the carcinogenicity tests, because what we're here about is whether glyphosate in Roundup causes cancer; right?

11:57:35 A. Correct. They have tiered categories at EPA as to which tests had to be done. This one's in Tier 1, from what I understand.

Q. And the standard requirement for carcinogenicity these days is a submission of two animal studies. And

11:57:51 almost every one does rodent studies, rats or mice; right?

A. Correct.

Q. So most substances -- most herbicides and most pesticides, most other substances that are subject to EPA

11:58:05 approval, have been approved on the basis of two rodent studies; right?

A. From what I understand. I'm -- again, I haven't sat at EPA and looked at their work, so I can't answer the question. But, generally, I would guess that that's

11:58:18 the case.

Q. Here we have a very large body with regard to the rodent carcinogenicity studies, at least; correct?

A. It's one of the largest I've seen, yes.

Q. These studies that they do are conducted under the GLP or good laboratory practices standards; correct? That's been around for decades.

A. That is correct.

Q. And that includes audits by a separate -- a separate team of scientists. You have the scientists that are out working in the lab, and then there has to be a completely separate group that does audits of those people. They can't be managed in the same way. They can't report to each other. They have to be independent.

And then all of them can be audited by the EPA; right?

A. You're getting into more detail than I know.

Q. All right.

A. Most GOP studies require an audit. That, I do know. But what -- they're not auditing the science of the study. They're auditing the conduct of the study and the -- the way in which the lab is set up. Such as you -- you have to have one way to come in and one way to go out with animals. When you're sacrificing them, people can only handle animals in certain ways at certain times. It all has to be recorded, et cetera. And that's

what they're looking at.

Q. And between the GLP and the OECD guidelines, there are elaborate regulations about how many animals are -- how many animals are in each dose group, what constitutes a dose group, how to determine what doses to give, how the animals are housed, one door in, one door out, when you're sacrificing the animals, et cetera, et cetera, et cetera, et cetera, an (inaudible) item; right?

11:59:52

12:00:06

A. They're extensive.

Q. It's a very difficult set of standards for a lab to meet; right?

A. It certainly would be difficult for a small lab to meet those standards. Contract labs all meet those standards.

12:00:20

Q. And small academic labs often have a real hard time doing so?

A. They're not required to. There's no guarantee they did it under GLP.

12:00:31

Q. Now, the Greim paper collects data from these registration studies that you were talking about yesterday; right?

A. Correct.

Q. And registration studies is a term referring to the studies that are submitted to EPA, to EFSA, to ECHA,

12:00:52

to other agencies, per their requirements that they be submitted, GLP-certified, carcinogenicity data to review; right?

A. Correct.

12:01:05 Q. It's normally proprietary information; right?

A. That is correct.

Q. It's the property of the company, and if the company gave it out, another company could submit it in support of an application. So the companies keep it as a

12:01:21 trade secret; right?

A. Well, in this case, my understanding is these are no longer the property of the companies. They're the property of another group. But, still, they're the property of someone.

12:01:33 Q. And what the Greim study did -- the Greim article, rather, not a study -- is collect the data tables and reports from a whole bunch of studies, all the ones that you have reported on here. And that enabled you to do many of the statistical analyses that you did;

12:01:51 right?

A. That's correct.

Q. And this was made available at least 30 days before the IARC meeting to IARC; right?

A. I don't know if it was made available to IARC

12:02:00 30 days before the meeting. It was published just before

the meeting. I wouldn't give you exact dates.

Q. Okay. It was published before the meeting, and it may have been made available through some another channel earlier than that, but you don't know?

12:02:13 A. I wouldn't know.

Q. And this is the best publicly available information on the subject of what was done in these studies; correct?

A. No.

12:02:24 Q. What is the best publicly available information?

A. Again, you have to go to EFSA and EPA and their characterizing of the data in order to get an understanding of the studies. These tables don't even have dose in them in some cases. They just -- low, mid, 12:02:45 high. So you certainly couldn't use that to do an evaluation without going to find out what the doses were somewhere else.

Q. Okay, sir. This part is the -- the write-up --

A. Correct.

12:02:54 Q. -- summarizing some of the information in the table.

And this is the important part. This is the important part to you. This is the scientifically valuable part, a bunch of tables from all of these.

12:03:06 A. Correct.

Q. And what was not provided in -- in the Greim review article was the -- for example, the individual animal data. If that had been provided, I'd be stacking up to here and here and here and here and here (indicating). And it would be a very laborious process to bring it into the courtroom, much less to review it; right?

A. Correct.

Q. But that information is available to the EPA, EFSA, ECHA, BfR, et cetera, because it's required to be provided to them?

A. I would assume, but I don't have firsthand knowledge.

Q. It's because they have that, sort of, information that their reviews and analyses of what happened in those studies is valuable to you?

A. Of their review and interpretation -- well, no. They're simply -- summarization of important characteristics of the study is of value to me, yes.

Q. And it's because they have information from those studies that you don't have?

A. Correct. Like survival. Did any of the animals die too early? Did the chemical look like it was killing the animal? Things like this.

MR. GRIFFIS: Would this be a good time to

break, your Honor?

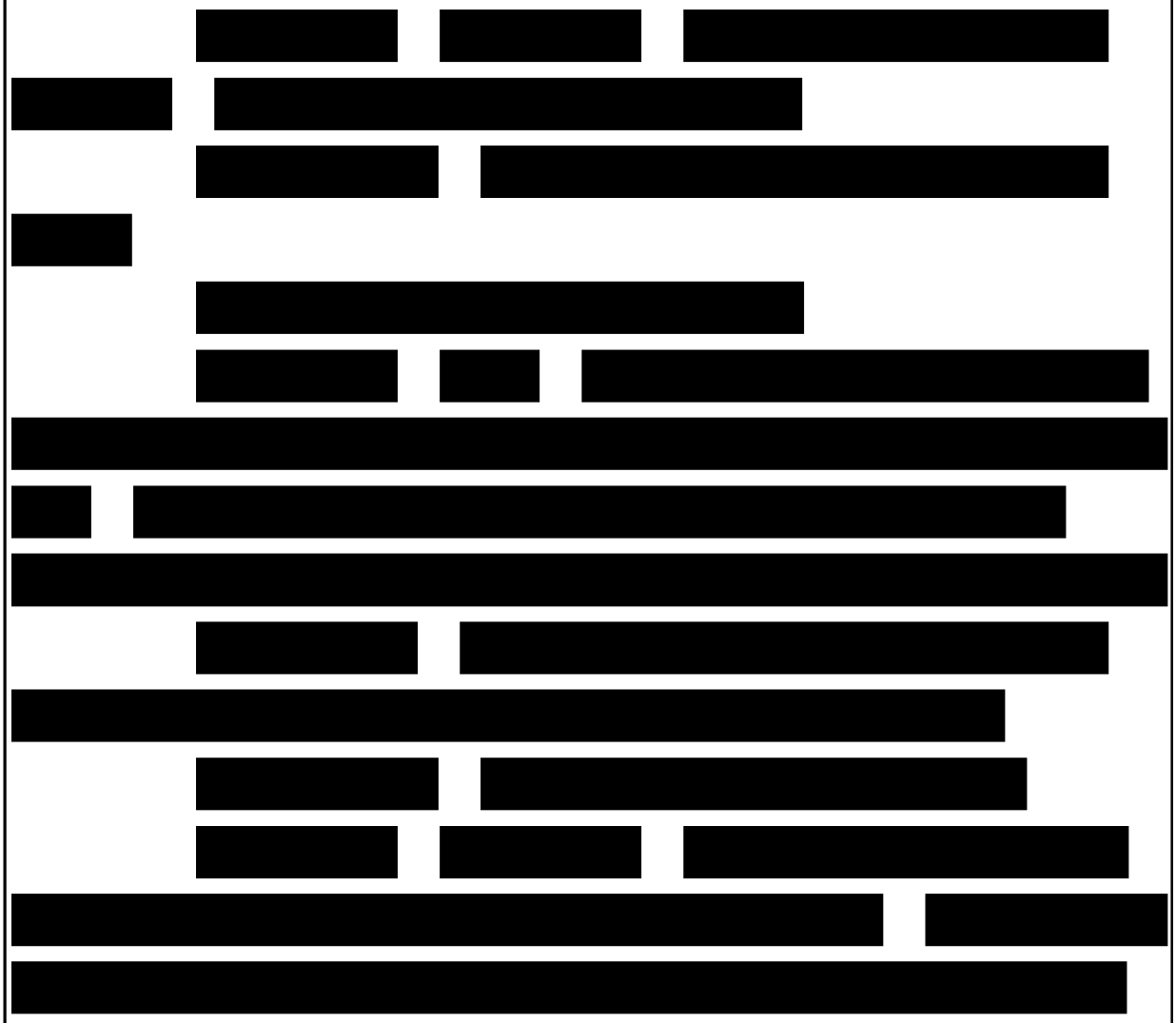
THE COURT: Yes.

All right, Ladies and Gentlemen. We're going to break now for the lunch recess. Please remember: Do not discuss the case with anyone. Please do not do any research. And we will resume again at 1:30. All right? Thank you.

And, Counsel, will you please remain?

MR. GRIFFIS: Yes.

12:06:14 (Jury leaves courtroom.)



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12:07:20 [redacted]

(Time Noted: 12:07 p.m.)

REPORTER'S CERTIFICATE

I certify that the proceedings in the within-titled cause were taken at the time and place herein named; that the proceedings were reported by me, a duly Certified Shorthand Reporter of the State of California authorized to administer oaths and affirmations, and said proceedings were thereafter transcribed into typewriting.

I further certify that I am not of counsel or Attorney for either or any of the parties to said Proceedings, not in any way interested in the outcome of the cause named in said proceedings.

IN WITNESS WHEREOF, I have hereunto set my hand: July 13th, 2018.

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Leslie Rockwood Rosas
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State of California
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