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 Thursday, August 2, 2018, Volume 22, Morning Session, before the Honorable Suzanne R. Bolanos, at 9:38 a.m.

REPORTED BY:

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EXHIBITS

(None.)

Thursday, August 2, 2018

9:38 a.m.

Volume 22

Morning Session

San Francisco, California

Department 504

Judge Suzanne Ramos Bolanos

PROCEEDINGS

08:48:42

THE COURT: Good morning, Ladies and Gentlemen, Counsel. Welcome back, everyone.

MR. DICKENS: Good morning, your Honor.

MR. GRIFFIS: Good morning.

09:38:35

THE COURT: Mr. Griffis, you may call your next witness.

MR. GRIFFIS: Thank you, your Honor.

Monsanto calls Dr. Warren Foster.

THE COURT: Very well, Dr. Foster.

09:38:51

MR. GRIFFIS: May I approach, your Honor?

THE COURT: Yes.

Good morning, Dr. Foster. If you'd please step up here to the witness stand and remain standing while the clerk swears you in.

WARREN FOSTER,

having been first duly sworn, was examined and testified as follows:

09:39:20

MR. GRIFFIS: I'll hand Dr. Foster his binder.

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

THE WITNESS: Warren Foster, W-A-R-R-E-N, F-O-S-T-E-R.

09:39:40

THE CLERK: Thank you.

THE COURT: Thank you.

You may proceed, Mr. Griffis.

MR. GRIFFIS: Thank you, your Honor.

DIRECT EXAMINATION

BY MR. GRIFFIS:

Q. Good morning, sir. Would you please tell the jury about your educational background, Dr. Foster?

09:39:52

A. Sure. I did my undergraduate degree, bachelor of science in human biology, at the University of Guelph, and then I worked for a little while before I decided that I didn't like working, and went back to graduate school and did my master's degree in the Veterinary College at the University of Guelph as well.

09:40:11

Q. Where is the University of Guelph, sir?

A. Guelph is about 30, 45 minutes outside of Toronto. It was at the time the only veterinary school in Canada and one of the premier veterinary schools in the world.

09:40:28

Following graduation with my master's degree, I then went to McMaster University, which is -- it's about 30 miles down the road. It's still in the area of Toronto, near Buffalo, and I did my Ph.D. in medical sciences at the -- at McMaster University.

09:40:48

Q. Thirty minutes down the road from?

A. From Guelph.

Q. From Guelph.

A. Sorry.

Q. Not from here.

09:40:57

So you're a professor at McMaster?

A. Yes, sir.

Q. And what do you do there?

A. I work in the department of obstetrics and gynecology. I design, carry out animal studies, as well as do clinical studies, primarily in the area of endometriosis, but I've also done exposure studies with environmental contaminants. I still do collaborative work with Health Canada, and I do -- have done a fair amount of toxicology work there as well, working with

09:41:08

09:41:29

things like benzo[a]pyrene and cigarette smoke and so forth.

Q. What are the parallels between endometriosis and cancer?

09:41:39 A. Endometriosis is a benign growth of the cells that line the uterine cavity, and they can grow anywhere outside the uterine cavity in the body, and they're a benign growth. They don't become malignant, but they're similar to tumors. And there are a number of

09:41:57 conditions -- you have to understand in women's health, many of the cancers of the reproductive tract are dealt with by obstetricians and gynecologists. They're not generally gone to -- or seen by oncologists. So I've done some work on endometriosis associated with ovarian

09:42:14 cancer and looked at fallopian tube tumors as well in those studies.

Q. How long have you been a professor at McMaster?

A. It's been 12, 13 years now. I've been at Mac for 17 -- I'm in my 18th year there, and it's been

09:42:30 13 years, I guess, I've been a full professor.

Q. And you also have a faculty position at UCSD?

A. Yes, University of California San Diego. I'm a faculty member there in the department of reproductive medicine where I collaborate with the -- with one of my

09:42:47 colleagues there, and we have a number of grants looking

at -- from pharmaceutical companies looking at novel therapeutic interventions for women with endometriosis and to manage pain.

Q. Before you began working at McMaster, where did you work?

09:43:03

A. I worked at Health Canada for close to ten years.

Q. What is Health Canada?

A. Health Canada is roughly equivalent to the US EPA and FDA. In Canada, the Health Canada assumes responsibility for both prescription medications, medical devices, as well as environmental contaminants.

09:43:14

Q. And I'd like to put up a slide, sir, that you helped us prepare. And we've previously gotten approval to show all the slides.

09:43:39

MR. GRIFFIS: Can we have Slide 1?

THE COURT: Very well.

Q. BY MR. GRIFFIS: And this shows EPA requirements for chemical approval, and we said EPA, because we've been talking about the EPA. Are these the same requirements that Health Canada has for chemical approval?

09:43:49

A. Yes, these would be the same group of six studies that Health Canada would require in order to review a compound.

09:44:00

Q. And were you involved with evaluating all of these, particularly including long-term rodent carcinogenicity studies while you were at Health Canada?

A. Yes. While at Health Canada, I provided assistance to the Canadian Environmental Protection Group or -- that worked to regulate chemicals under the -- what we call CEPA, the Canadian Environmental Protection Act, so I was frequently required to assist the regulators in reviewing the documents and providing them with scientific input and helping them interpret what the data said.

Q. Were you involved with research and studies in that role?

A. Yes. From the -- I went to Health Canada in 1990, completed my Ph.D. in 1991, so I was already there just prior to completing my Ph.D., and from the time of arriving there, I was involved in the design and conduct of animal studies in reproductive developmental toxicology, systemic toxicology and also assisting the group in mutagenicity carcinogenicity.

Q. Did you also serve as the OECD representative for the nation of Canada?

A. Yeah.

Q. And what is the OECD? We've heard a little bit about it, but please remind us what it is.

A. The OECD is the Organization For Economic Cooperation and Development. It's headquartered in Paris, and it's an organization that operates under the auspices of the United Nations and World Health Organization, and there's roughly -- I think there's around 32, maybe 35 member countries now.

09:45:39

As the national coordinator for Canada, it was my responsibility to participate in the every nine-month meeting in Paris, where we would review the test guidelines, so that -- the test guideline like those that are developed for neurotoxicology, immunotoxicology and carcinogenicity. So these are the test guidelines that are developed to be used by companies, or anyone else, that are planning on doing studies to develop data for registration of new or existing chemicals.

09:45:57

09:46:25

And the idea behind this is to develop test guidelines that are harmonized across all countries so that wherever the study is done, it's generating reproducible, reliable data, and the data that maybe was -- the study was conducted in the United Kingdom, the company could submit that same data in the United States or in Canada and it would be considered for evaluation purposes.

09:46:46

Q. Would you please tell the jury what you consider to be your area of expertise?

09:46:58

A. My primary area of expertise is in reproductive endocrinology and toxicology. I also do some work in reproductive cancers, and so I've done some studies looking at pesticides like dieldrin and looking at its effect on mammary tumors.

09:47:18

Q. And you have conducted animal studies on numerous occasions; correct?

A. Yes, I have.

Q. And do you consider yourself to be an expert in translational toxicology?

09:47:29

A. It's more that my colleagues consider me to be an expert in translational toxicology. There's things like LinkedIn and other ways that we communicate with our -- amongst ourselves, and my colleagues have pointed that out, that I'm an expert in transitional toxicology.

09:47:49

Q. What is translational toxicology?

A. Translational toxicology is being able to look at animal studies and looking at animal physiology and knowing what's the same and different between humans and being able to translate data to the human situation and recognizing things that might be not translatable because of the differences in the physiology.

09:48:07

Q. We've heard testimony from one or two witnesses that when you work for a university, they don't fund your studies, you need to go get funding from the outside for

09:48:25

the most part. Where does your funding come from?

A. The majority of my funding comes from the Canadian Institutes of Health Research, which is the equivalent to the NIH. It's roughly equivalent in how we review, and our success rates, actually, in Canada are maybe a little better than the NIH, but the majority of my funding comes from there. I have received funding from the Natural Sciences and Engineering Research Council, which is another federal body that provides money for research. At one point I received money from the American Chemical Council for some studies that we did, and then I've also more recently received a number of grants from different pharmaceutical industries to look at different model therapeutic interventions, and in one case, we've just patented a new diagnostic tool for endometriosis.

Q. Sir, obviously you are consulting with us on toxicology issues. Who else do you consult for?

A. I've frequently been approached by Health Canada to provide them with a comment or assistance or even to collect samples for studies. I have been consulted to the Public Health Agency of Canada in helping them lay out a surveillance program for developmental effects. I contract also to nongovernment organizations, like risk assessment services, to provide them with feedback that

they, then, give the other government on other issues. I've consulted to Environmental Health Protection Agency. So quite a broad range of both government and nongovernment groups.

09:50:28 Q. Have you ever served on an IARC Working Group?

A. Yes, I have.

Q. For what chemicals?

A. Chemicals were dieldrin, aldrin, pentachlorophenol, and there was a fourth chemical that I

09:50:42 was not involved with.

Q. Okay. And were you asked when you were -- when you agreed to do that Working Group on aldrin, dieldrin and pentachlorophenol to submit a conflict of interest disclosure?

09:50:58 A. Yes, I was.

Q. And at that time, had you agreed to consult with us about glyphosate?

A. At the time that I was approached by Kate Guyton from IARC, I had not had any contact with Hollingsworth

09:51:16 attorneys, so I did not disclose something that had not happened at that point.

Q. Okay. And then did you go back and retroactively disclose a conflict once you agreed to consult with us?

09:51:28 A. No, I did not.

Q. Would you explain why not?

A. Because the chemicals that we were working with, the aldrin, dieldrin and pentachlorophenol, were three chemicals that is -- to my knowledge and still to my
09:51:41 knowledge, were already banned in North America, and I did not know them to be or -- and still don't believe that they're chemicals that would be manufactured by Monsanto or any other agriculture chemical company that would impact on this.

09:51:58 And I had written previously on dieldrin, and the reason why Dr. Guyton approached me about it acting as a carcinogen, and in my review, I had already determined, in my own view, that it was a carcinogen, so I did not see a real or perceived conflict.

09:52:15 Q. Have you published any medical or scientific articles, sir?

A. Yes, sir.

Q. How many?

A. I have authored or coauthored in excess of 180
09:52:25 peer-reviewed articles, several book chapters and probably 150 or more proceedings for scientific meetings, so roughly 400.

MR. GRIFFIS: Would you put up Slide 2, please, Armando?

09:52:45 Q. And what we have on the screen is an award, sir.

Would you explain what this is?

A. I was nominated by a colleague from the other side of the country, not with my knowledge -- he nominated me for election to the Canadian Academy of Health Sciences, which is the organization within Canada that's the highest organization for people that work in health sciences, in recognition of my contributions to research and teaching and to the community and to the area of health science research.

09:53:22 Q. So you were appointed as a fellow?

A. I was elected, yes.

MR. GRIFFIS: Your Honor, we offer Dr. Foster as an expert in toxicology and the design, evaluation and interpretation of long-term rodent carcinogenicity studies.

09:53:34

THE COURT: Any *voir dire*?

MR. WISNER: Very short, your Honor.

THE COURT: Very well.

VOIR DIRE EXAMINATION

BY MR. WISNER:

Q. How are you doing, Doctor? My name's Brent Wisner. I'm an attorney that represents Dewayne Johnson, the plaintiff in this case. We've never met before;

09:53:48 right?

A. No, sir.

Q. All right. I just want to follow up on a couple things you mentioned, and I just want to clarify some things. You said you had a Ph.D. in medical sciences; is that right?

09:53:58

A. Correct.

Q. Is that like a medical degree in Canada, or is it equivalent to a medical degree?

A. It's in medical sciences with -- in my case, my primary area of focus was women's health, so narrowly focused, yes.

09:54:07

Q. So is it an M.D.?

A. No. It's a Ph.D.

Q. Okay. So you can't prescribe drugs or treat patients?

09:54:18

A. Not in my area of interest. Thank you.

Q. No, no. I wanted to make sure in case there was confusion there.

You said you're also -- translocational toxicology?

09:54:27

A. Translational.

Q. Translational. That makes more sense.

And in translational toxicology, do you consider yourself to be an expert in that or is that just what your friends on LinkedIn said? I wasn't clear.

09:54:38

A. It's what other peers within the field have acknowledged based on the work that I've done, the publications that I've written, and I think it's a fair and accurate portrayal of the work that I do.

09:54:55 Q. Okay. So it's your opinion as well that you're an expert in that?

A. I would agree with that.

Q. Okay. I just wanted to clear that up.

Then you mentioned the IARC Working Group.

09:55:03 A. Yes.

Q. You participated in one of them?

A. Correct.

Q. And at the time that you were asked to participate, you hadn't engaged in any consulting work

09:55:11 with Monsanto; right?

A. When I was approached by Dr. Guyton from IARC, I had not been approached by Hollingsworth at that time.

Q. But you had been approached and agreed to participate or help Monsanto before actually

09:55:26 participating in the program; is that right?

A. I just want to make sure we're clear.

Q. Yeah, that's why I'm asking.

A. I was approached by Dr. Guyton to participate in the IARC program. I agreed to do that and had already

09:55:43 conducted a fair amount of my own background reading for

that meeting.

Q. Okay.

A. Then I was approached by Hollingsworth, asked if I would be willing to assist them, and at that time, I provided them with my CV and asked them to review that and let me know if they thought that was appropriate.

Q. Sure. My question is: Before you actually voted, you had -- by the time you voted, you had already agreed --

A. By the time I voted at IARC --

Q. Yes.

A. -- I had already agreed to work with Hollingsworth, yes.

Q. Okay. And before that, when you did make your disclosures of interest earlier, did you disclose any potential conflicts?

A. At that time, no, I had not, and for the reasons I explained, I didn't believe there to be a conflict.

Q. No, not with Monsanto, but with the pharmaceutical stuff or any other --

A. Oh, sorry. Yes, I did include on the disclosure form that I had contracted with American Chemical Counsel and other groups, yes.

Q. What other groups?

A. At the time, I think it was Ferring

Pharmaceutical and potentially at the time, by that point, ADVI Pharmaceutical.

Q. And what about Exponent?

MR. GRIFFIS: Your Honor, this is beyond the scope of *voir dire* on qualifications.

MR. WISNER: I was just clarifying his testimony.

THE COURT: Overruled.

You may answer.

THE WITNESS: Sorry?

MR. GRIFFIS: You may go ahead.

THE WITNESS: At the time, I don't believe I did, because at the time, the work that I had done with Exponent was on endocrine disruption, and I didn't -- it didn't rise to my attention as being something that needed to be disclosed.

Q. BY MR. WISNER: All right. And then my last few questions and I'll let you go. You're not here -- you're not an epidemiologist; right?

A. That's correct.

Q. So you're not going to offer any opinions about epidemiology?

A. No, sir.

Q. And you're not offering any specific opinions about the mechanism of action; right?

A. Although these are things that I am knowledgeable of and work with in my routine area, I've been asked to focus on the animal studies, so you're correct, I am focusing only on the animal literature.

09:57:50 Q. Great.

MR. WISNER: No further questions, your Honor.

No objections to his certification.

THE COURT: Okay. Thank you. Then I will accept Dr. Foster as an expert in toxicology and the

09:57:58 design, evaluation and interpretation of long-term rodent carcinogenicity studies.

MR. GRIFFIS: Thank you, your Honor.

THE COURT: You may proceed.

DIRECT EXAMINATION (Continued)

BY MR. GRIFFIS:

Q. You're being compensated for your time spent working on this case; is that correct?

A. That's correct.

09:58:13 Q. At what rate?

A. I charge $250 an hour.

Q. And what did you estimate your billing to be on this case, sir?

A. That's very difficult to calculate, but I'm

09:58:25 estimating that it's around 60,000 to $65,000.

Q. Okay. We're going to go over your opinions in some detail as the morning progresses, sir, but so the jury knows what they're going to be, let's get that out right now. And the jury has heard already that there are 12 long-term rodent carcinogenicity studies that everyone agrees to be good enough to be evaluated when you're looking at issues of carcinogenicity of glyphosate. Did you review all of those, sir?

A. I reviewed all 12 studies, yes.

09:59:00 MR. GRIFFIS: Could we have Slide 10, please?

Q. And those are they; correct?

A. They are the studies, yes.

Q. And it's organized by rat studies and mouse studies. We've seen these groupings on Dr. Portier's

09:59:16 charts and in other places, but there are seven rat studies and five mouse studies that everyone agrees are good enough to at least go into the hopper for evaluation; correct?

A. That would be correct, yes.

09:59:30 Q. And did you do an independent evaluation or were you relying on evaluations from other people in reaching your conclusions?

A. I reviewed the -- the studies myself. And where I had the -- some studies I had to rely upon another

09:59:51 published article, the Greim study, where I relied upon

the text that was in the -- not the text, the table that was in Greim and the appended tables that went with it.

Q. Okay.

A. But outside of that, it was a completely independent review.

Q. And we've heard, sir, about Greim. That is a pretty short review article, followed by a pretty long set of appended tables.

A. That's correct.

Q. And the tables, as you understand, come from the original studies that you looked at; is that right?

A. That's correct.

Q. Okay. Based on your review, what was your conclusion as to whether there were compound-related tumors in each of these studies, sir?

A. In reviewing the studies individually and then together, I concluded that there were no compound-related effects in any of these studies.

Q. And how did the approach that you used differ from the approach that you used at Health Canada while you were working there and looking at long-term carcinogenicity studies or in your current life when you do the same?

A. It's the same approach that I've been trained to use, so I did not do anything different here than what I

would do in -- in the course of my normal routine.

Q. And what is your bottom-line conclusion about what the animal studies show about glyphosate and cancer, sir?

10:01:25 A. My bottom-line conclusion is that glyphosate does not introduce compound-related tumors.

Q. And what about whether glyphosate is a rodent carcinogen?

A. In my view, on the basis of this rich data set, 10:01:43 it is not a rodent carcinogen.

Q. And are those views you hold to a reasonable degree of scientific certainty?

A. Yes.

Q. Now, you say not a rodent carcinogen. Why don't 10:01:55 you comment on humans? Why are you saying rodent carcinogen?

A. Because I was asked to review the animal literature, and I focused on what the animal literature is showing me.

10:02:06 Q. Okay. So I'd like to start talking about the analysis that you went through, sir, in reaching these conclusions, and I'd like to start with the sorts of criteria that are applied by you, by the regulators, by the OECD in doing so. So have you helped us prepare 10:02:26 slides on that?

A. Yes.

MR. GRIFFIS: Could we have Slide 3, please.

Q. And we actually have two slides, of which this is the first, on the subject of requirements for regulatory carcinogenicity studies. Would you first explain what a regulatory carcinogenicity study is, what "regulatory" means in that context?

A. The regulatory bodies require that companies, when they carry out a carcinogenicity study or an immunotoxicity study, that they follow test guidelines that have been harmonized under the OECD. And do you want me to go into some detail on the studies? I --

Q. Some detail, yes. I'll jump in if you --

A. Ideally, these studies are -- sorry I'm boring you already.

These studies are conducted in mice or rats. We're looking for -- at Health Canada and elsewhere, we're looking for at least one study in mice and one study in rats. The studies are -- you want them to be statistically robust, so they consist of 50 animals per sex, per each dose group, so that's 200 mice. They're big studies.

Q. Can we pause for one moment, sir? Why is 50 selected rather than 20 or some smaller number that would be more economical?

A. We're looking at, again -- as I indicated, we want statistically robust data, and so we're looking at -- these are screening assays, and you're looking at 40 different tissues from each of the animals, and you
10:04:12 want to make sure that you've got enough animals that it's going to be sensitive enough to pick up rare events if they're present.

Q. So how many amounts are in the whole study?

A. So 100 animals per dose group of four dose
10:04:27 groups. There's 4 -- 200 animals -- sorry.

Q. Okay. Go on.

A. We have a high-dose group, so you use four dose groups. There's a control, a low, a medium and a high-dose group, and OECD instructs us to use a limit
10:04:46 dose of roughly 1,000 milligrams per kilogram as our highest dose.

Q. I'd like to pause about that, because there's been some discussion about that limit dose before, so I'd like to pull up the actual OECD guideline on that.
10:05:00 That's something you're familiar with; correct?

A. Yeah. And I just want to correct one thing. I said 200. It's 400.

Q. Thank you.

So this is in your binder as Defense
10:05:13 Exhibit 2856.

MR. GRIFFIS: And this is OECD Guideline 453.

Permission to publish, your Honor?

THE COURT: Any objection?

MR. WISNER: Can you specify which page you're going to refer to?

MR. GRIFFIS: I want to show the first page so that we can see it and then go to page 6.

MR. WISNER: No objection.

THE COURT: All right. You may proceed.

10:05:48 MR. GRIFFIS: So could we have the first page, first of all.

Q. And this is referred to as OECD 453; right?

A. Correct.

Q. What does that mean?

10:05:56 A. This is the Carcinogenicity Test Guideline Number 453. This is the type of guideline that we would review in the meetings in Paris.

Q. So there are 452 more of these guidelines?

A. There is -- there is a very big binder of the 10:06:13 different guidelines, yes.

Q. Okay. I hope at least some of those are obsolete, so you don't have to know them all.

Could we go to page 6 and look at Guideline 23?

MR. GRIFFIS: Just blow up 23, please.

10:06:29 Q. So would you explain to us what this says about

the 1,000 milligram per kilogram body wait? It's the last sentence.

A. So your question is?

Q. Well, first, just tell us what it says, and then explain the significance of it, please.

10:06:44

A. "A limit of 1,000 milligrams per kilogram body weight per day may apply, except when human exposure indicates the need for a higher dose level to be used."

Q. Okay. So how does this guideline apply a limit of 1,000 milligrams per kilogram per day?

10:06:59

A. What the guideline is instructing those of us that conduct these studies is that we should have a high-dose group that is either 1,000 milligrams per day -- kilograms per day or approaches it.

10:07:17

Because what you're trying to do is get a dose that's going to be sufficiently high enough that you know if there's -- that you're going to get a -- some effect upon the animal, so you know you've got a positive study.

But you don't want it to be too high, going above 1,000 milligrams per day. As it was explained to me at OECD, was that there's really no point going beyond it, because no one is going to ever see that much chemical in an environmental setting.

10:07:36

Q. And when you say "no one," you're talking about no human?

10:07:49

A. No human.

Q. Not no rat or no mouse?

A. Right. So the point is, is that we're at the -- in the age of where we're trying to reduce and limit animal testing to the extent possible. And going above this, we're now starting to use doses that's not providing us with meaningful information that we can interpret.

Q. Okay, sir. Let's go back to Slide 3, then.

Let's go to the next slide, Slide 4.

And these are additional OECD criteria --

A. That's correct.

Q. -- and regulatory criteria for animal studies.

And would you explain these briefly, please?

A. Again, what we're -- we're trying to do in the carcinogenicity world, we're trying to do studies that are going to be sufficiently long enough so it accounts for two-thirds of the normal life span of the mouse or the rat.

So in mice, 18 months is the duration that's preferred. And in rats, 24 months. We chose that time frame because we know that as -- just like people, as animals get older, they get more spontaneous tumors. And if we waited until all the animals were dying naturally, then the problem would be we wouldn't be able to detect

compound-related effects.

Q. So you're trying to find the right time to stop the study in order to maximize the information you get --

A. Correct.

10:09:23 Q. -- in terms of tumors that are -- that are occurring in animals; is that right?

A. Correct.

Q. All right. And you mentioned earlier that Health Canada likes to see one rat study and one mouse

10:09:33 study these days in doing its evaluations; right?

A. That's correct.

Q. How does the glyphosate data set -- and for your purposes, sir, I'm not talking about the data set in the area of epidemiology and mechanism. This is in regard to

10:09:47 animal studies. How does it compare to other studies that you've reviewed on carcinogenicity?

A. In my experience, the glyphosate data is unusual in that it's particularly rich. Normally what we would get in a submission would be one rat, one mouse. And

10:10:03 that would be the sum total of what we would get.

The -- there are other chemicals where you see huge data sets, like Bisphenol A dioxins. So they're very, very large data sets. But for typical pesticides, this is a very rich one.

10:10:23 Q. Did Health Canada standards require that the

animal testing be performed on the active ingredient? Glyphosate, in this case.

A. Yes.

Q. And it's been suggested that Monsanto should have done long-term carcinogenicity testing in formulated product. In your opinion, would that have been scientifically feasible?

A. I've not seen it done with any other pesticide, and I would anticipate that it would be very difficult to carry out. Primarily because as you start increasing the concentration, you're getting into that high dose of 1,000 mgs per kilogram, you're increasing all the -- the surfactants that are present in it. So things like soaps. That would have a very adverse effect upon the GI lining of the gut. The animals just wouldn't be able to survive.

Q. So they'd be having toxic effects from the surfactants before you could tell anything about the carcinogenicity of the compound? Is that your opinion?

A. My opinion is that the data that would be derived from them would not be interpretable.

Q. And we've talked about how there are OECD guidelines and regulatory guidelines governing how long-term carcinogenicity studies are supposed to be done. Are there a coherent set of guidelines for a

mixture study, like people have been saying Monsanto should have done?

A. There has been a huge amount of debate within the scientific community about how to do mixture studies. And there's a study section within the Society of Toxicology that focuses on that.

To my knowledge, there is yet to be a harmonized or agreed upon test guideline for mixtures.

Q. So if you wanted to do a GLP-certified long-term carcinogenicity study of mixture -- I mean, setting aside your doubts about whether it would be feasible to come up with a way to do it, could you look somewhere and find a set of standards that would guide you in doing so?

A. Not to my knowledge, no.

Q. Okay. Now I'd like to focus a little more on how it is a toxicologist determines whether a substance is causing tumors --

A. Okay.

Q. -- in rodents when it looks at a long-term carcinogenicity bioassay.

First, what is the -- what's the purpose of a rodent cancer bioassay? What is it we're trying to do? Obviously we don't -- we might feel compassion for rats and mice, but we're not actually concerned about identifying carcinogens that -- in fact, particularly

ones they wouldn't be exposed to. So why are we doing these studies?

A. These studies are being done in an effort to screen a compound to determine whether or not there is any tumor showing up in any target tissue, from nose to toes.

So from one end of the animal, all the way down to the other end, we're -- we're going in on a fishing expedition. We really don't have any preconceived idea of what we're going to get. We're giving them four doses. We're looking at 40 different tissues from each and every animal, and we're analyzing them. Every single tissue.

And as -- these are nightmare studies. There's 400 animals. You're looking at a week of spending your full day in the necropsy room, as a member of a team, with the animal coming in and being weighed, sacrificed and going all the way through, with one person keeping record, taking notes, all the way through. And you do this all day for about a week, until you make it through all the animals.

And then you're about a year out before you get the results, because there's all the histopathology that has to be done on each of these different tissues.

So 40 tissues, 400 animals, you're looking at

roughly 16,000 determinations. It's a large number. And you have to remember the pathologist is looking at each of those tissue sections, and he's not just looking at a kidney and saying: Is there a tumor? He's looking at
10:14:45 the kidney and saying: Is there any morphological abnormality at all in this tumor -- in this tissue slice, and can I put it into any of my different categories of the types of tumors that I know that appear in kidneys?

So 16,000 determinations probably is even an
10:15:07 underestimate of the number of outcomes we're looking at. So they're horrendous studies.

Q. And by "horrendous" --

A. Time -- time, tedious and complex.

Q. Okay. You're taking about 16,000 different
10:15:21 evaluations and then the necropsy -- say that word again.

A. Sorry?

Q. You say it. The room, the "necropsy" room.

A. The "necropsy" room.

Q. Necropsy room. Too many syllables in there.

10:15:33 No, it does not sound pleasant, sir. Is it -- when the animals are coming in and being evaluated, do the people who are evaluating them know which dose group they belong to, for example?

A. Initially we do, because at Health Canada what
10:15:49 we're doing is we -- we start the control, and we work

our way through low, medium and high, because we're taking blood, and we might be doing residue analysis as well to measure the concentration of the test chemical were metabolizing in different target tissues. And you don't want to be contaminating a low-dose group by sacrificing a higher dose one first.

10:16:08

Q. Okay.

A. So in the room, we know that this is -- from what group they're coming from. But the pathologist does not know.

10:16:26

The pathologist, when they get the slides, they're coded. They're reading them blinded.

Q. Okay. So the people working in the room know. They take slides and then code the slides. And the codes have some barcode or long number on them, and no one knows how to translate that until the end?

10:16:39

A. Right. Each animal has a number assigned to it. And as the tissues are weighed, and they go into the form -- to be fixed -- for histopathology, they go into an appropriately numbered vial that we -- as the study PI, I would have the code. But that's not given to pathology for processing of the samples. It's not given to the biochemistry department for when they run the blood samples. We -- we keep that back, and we break the code at the end.

10:16:56

10:17:17

Q. And these proceedings that you're talking about, that's part of OECD and GLP, the laboratory practices?

A. Yes.

Q. And regulatory standards; right?

10:17:27 A. Yes.

Q. And when the pathologists look at the slides, is it common to find tumors in all the groups, the control group that was not exposed to the substance, low dose, medium and high dose?

10:17:41 A. Yes, it's not uncommon to see spontaneously occurring tumors in mice, in different target tissues. Particularly for some cases where these are commonly occurring tumors in the test species.

MR. GRIFFIS: Can we have Slide 6, please?

10:17:56 Q. So spontaneous tumors in rodents. Why is it that we are seeing spontaneous tumors in these studies? Obviously spontaneous tumors, if it's the control group, you know it's spontaneous. And if it's any other group, it might be spontaneous; right?

10:18:15 A. We all are experiencing mutational events that happen all the time. We are exposed to ambient background radiation. We get sun on our skin, UV radiation. We have chemicals that are in our diet that might interact with the -- the DNA in our cells. But

10:18:36 most of the time, these -- these mutations are being

repaired. There's a DNA repair mechanism that takes care of this.

But every now and then one of those mutations occurs in a tumor suppressor gene or an important area
10:18:52 resulting in a spontaneously occurring tumor. Particularly in a cell type that's rapidly dividing.

Q. You've talked about how it is that -- the general theory of how it is that cancer develops in living organisms. Just now.

10:19:06 A. The general concept?

Q. Yeah, that's what you just laid out.

A. Yeah. Generally we know that there is some kind of mutational event that takes place that is in a gene that's important either for inducing proliferation or new
10:19:24 blood vessel growth or in a tumor suppressor gene or it's knocking out DNA repair enzymes, and the result is that it -- you get an initiation event that takes place.

Now, many of us will have tumors like this in our body all the time. You get initiation in the -- in
10:19:44 the cells. Although an initiation event is -- has taken place, the cells lie dormant. And it's not until later in life when you have a promotional event that the cells then begin to grow, and grow uncontrollably into a tumor.

Q. I'd like to talk about the issue of how
10:20:03 toxicologists tell -- after they've gone through this

process that you've talked about and sent thousands of slides up to the pathologists, and the pathologists have reviewed those, given their evaluation, and the data is unblinded at the end -- I assume that happens at some point -- and you end up with the data --

10:20:17

A. Yes.

Q. -- And then make the evaluation.

MR. GRIFFIS: So could we have Slide 7, please, and talk about the criteria?

10:20:27

Q. Toxicology applies to see if there's a real effect. Would you address this, please, sir?

A. So when I start looking at a study that -- whether I'm helping out the people at Health Canada or when I was looking at the data here, one of the things

10:20:41

that I wanted to look at is to see how well the study complied with the regulatory guidelines. Did they follow an OECD guideline? Some of the studies were done in the 1980s before the OECD test, guidelines for carcinogenicity existed. So in these cases, I'm looking

10:21:01

to see if they followed a study protocol that was in adherence with the intent of what later became the OECD test guideline. Did they have a large enough number of animals? Did they have four dose groups? And so forth.

The other things that I'm looking at are

10:21:19

consistency within the study. Was everything done the

way I expect it to be done for these types of studies? Am I seeing -- am I seeing consistency within the data?

So if I'm seeing a change in a particular area, am I also seeing a change in body wait that would be consistent with what I'm expecting to get?

10:21:38

I want to see a dose response. And I want to be clear on this. When we say "dose response," dose response -- when I was younger, the dose response we would expect to see would be linear. We'd see, as you went from control to medium to low -- sorry, low to medium to high, you would see that linear dose response.

10:21:56

But as my career has become richer, we've seen that we get dose responses that they might go up and curve, because you've already got as many tumors as you can get. So you're not going to get more. So you get that slope that tapers off at the top. Or it might rise a little more slowly. And because we're only looking at four dose groups, you don't get a straight line.

10:22:18

But I'm expecting to see -- as I go from a control to a low, medium and high, I'm going to see a rise.

10:22:31

(Interruption in proceedings.)

THE COURT: All right. Ladies and Gentlemen, let's take a five-minute recess. We'll resume again at 10:30.

10:22:48

(Recess.)

THE COURT: Welcome back, Ladies and Gentlemen.

Dr. Foster remains under oath, and you may continue.

10:30:06 MR. GRIFFIS: Thank you, your Honor.

Q. BY MR. GRIFFIS: Doctor, I've had a request that you sit a little more forward. Closer to the microphone. Thank you very much.

Now, in these rodent carcinogenicity studies 10:30:17 that you've been describing the inner workings of, what is the job of the toxicologist, like you?

A. The toxicologist would be, in my experiences, the person that at Health Canada puts forward the proposal for the study to be conducted in the first 10:30:35 place, takes ownership of getting it funded by the -- by the management, and then organizes the study team and designs the study. So lays out the OECD test guideline criteria for all the -- the people that are going to be involved in the study, making sure you've got enough 10:30:59 bodies on hand to conduct it. And then shepherds the animal healthcare protocol through for approval, and then works with the central animal facility on a day-to-day basis to make sure that the study is run according to plan. And that might involve day-to-day visiting the 10:31:20 animal quarters and checking all 400 of the animals and

making sure that they're doing what they're supposed to be doing.

Q. And you mentioned the team. Does the team usually include a biostatistician?

10:31:32 A. Yes.

Q. What's the role of the biostatistician, normally?

A. The biostatistician, in my experience at Health Canada, is somebody that we go to at the conclusion of

10:31:43 the study with the data and ask them to run the -- the stats for us.

They then generate a report, which they bring back to us, and we sit down and discuss the findings. And it then becomes up to me, as the PI in this case, to

10:32:00 make heads or tails out of all the data that I get.

Q. Now, when is the decision made about which statistical tests are going to be run on the data?

A. Generally, that's already laid out in the OECD test guidelines. It -- it tells us what test it prefers

10:32:20 that we would run, and we would follow that.

Q. And why is it standard practice to specify what statistical tests you are going to be running on the study before you do the study and gather the data?

A. The idea behind it is making sure that we have

10:32:36 enough animals for the test requirements and that the

assumptions of the tests are being met before the study's conducted.

And we decide a priority what we're going to do, so we're not at the end running a study and then shopping for the statistical procedure that gives us the answer we want.

Q. Okay. What do you mean by that, shopping for the statistical procedure that gives you the answer you want?

A. I've not seen it done myself, but I have, in discussions with colleagues, learned of people that will look at the data and apply different statistical methods in order to find one that gives them the statistical significance of less than 0.5 that they're looking for, so they've got a change they can write about.

Q. And by change you can write about, why is it that finding statistical significance in a particular study would give you something to write about and not finding it wouldn't?

A. As an academic, my survival is dependent on being able to publish my work. If I've conducted a study where there's no significant data, I'm not going to be able to publish. And that's certainly going to negatively impact my ability to get my next grant.

Q. Because studies that are negative are less

interesting to publications than studies that are positive, in general?

A. That's the consensus amongst the field. I -- I, on the other hand, take a bit of a different view. I think studies, even when negative, are useful, because they tell me something interesting. It's sometimes in the -- in medicine, it's important to know things that don't work as well as things that do.

10:34:08

Q. Yes, sir. The tendency of journal editors to be interested in wanting to publish positive rather than negative results and associations rather than no associations, that's part of something called publication bias; right?

10:34:27

A. Yes.

Q. Now, let's get back to the statistical analyses that are done on the animal studies.

10:34:39

When you -- let's say there are toxicologists running an animal study. You've gone through this process, and -- I'm not going to try to say that word again.

10:34:51

A. "Necropsy."

Q. Necropsy room and sent off the coded slides for evaluation. The pathologists have done their evaluation, provided the raw numbers. The biostatistician has applied these statistical tests that were agreed on in

10:35:05

advance would be applied and come up with results. And you look at the results, and none of them are statistically significant. Are you done? Would you say: This study is negative, we're done?

10:35:18 A. No. I would still go back and look at the study and evaluate -- sorry. I would still go back and look at the study data and evaluate the study in order to determine whether or not there's a -- there's some irregularities in the data that make me want to question

10:35:36 the statistics.

Q. And do you ignore findings just because they don't reach formal statistical significance?

A. No.

Q. Would you explain?

10:35:45 A. I'm going to look and see how the control group performed in my particular study. If my control is performing outside the range of what I expect for my historical controls, then I have to question: Is there something funny going on in my -- in the study that I

10:36:08 need to evaluate further?

Similarly, if I get an answer where my control didn't produce the effects that I'm anticipating and that I've typically seen in this population of mice, for example, then I'm also going to question how well the

10:36:25 study ran and what might -- what factors might explain

those outcomes.

Q. There are biological considerations that you still need to apply after the numbers have been run; is that right?

10:36:38 A. Yes, absolutely.

Q. Now, if the study is evaluated to be negative, like I just posited you, you run all the tests that were set out in advance, and there aren't any statistically significant findings, is it proper to then go run

10:36:56 additional tests, additional -- new statistical tests, different types of statistical tests, to see if you can find a statistically significant result?

A. Not in my experience. We -- we have not done that. I have not seen that done in Health Canada.

10:37:13 Q. Have you had occasions in your career, sir, where you've encountered findings that are statistically significant but not biologically significant -- not biologically relevant?

A. Absolutely.

10:37:23 Q. Please explain.

A. It's not uncommon. As I think I mentioned earlier, I worked as a reproductive endocrinologist. And it's not uncommon for me to see circulating concentrations of a given hormone that might be

10:37:43 statistically significant and different from the treated

animals. But they're still within the normal range for that hormone. And so I -- it's inconsequential. Particularly if I look at downstream effects.

So if it's in a male animal and I'm seeing a statistically significant increase in LH, but I don't see a corresponding increase in testosterone levels, I would question the value of that data and consider it to be a biologically irrelevant change.

MR. GRIFFIS: Could we have Slide 8 up, please, Armando?

Q. So this is a slide addressing the subject that you were just talking about, the role of statistics in assessing bioassays for long-term animal carcinogenicity studies.

And would you comment on the second bullet point, that a positive statistical test is a starting point?

A. In my experience, we run -- we run the study. We do our assays, the statistics are completed. And to me, that's where the fun starts. That's where the excitement starts. That's where I start to look at whether or not I've got something here that's interesting, and I try and understand what the data are telling me.

I'm a scientist. That's all I am. I'm just a

scientist. And my role is to speak for the data, because it can't speak for itself.

And so I try and look at it to see what it's actually telling me and what I should -- what I should, if anything, do next.

10:39:19

Q. And I'd like to talk just a little bit more about the standards that you're applying before we move on, to get a little more specific, sir.

And we've talked about you being an expert in translational toxicology, which is applying the results of animal studies to humans or not applying them to humans.

10:39:36

A. Correct.

MR. GRIFFIS: And would you put up Slide 9, please, Armando.

10:39:47

Q. Rodents are not tiny people. Would you comment on that, please?

A. It's a criticism that as people that work in toxicology, we -- we're -- we have to use mice. We have to use rats. There's things that we ethically cannot do to people.

10:40:03

And so sometimes we'll get criticisms back from the reviewers that the mice -- the mice -- you can't generalize your results from the mice to humans, because they're not tiny people.

10:40:20

There are physiological differences in mice that are not present in humans. So one -- one quick example that's relevant, the harderian gland is present in mice. We don't have one. Other things, physiologically, mice
10:40:42 have a bursa around their ovary. And as a consequence, they can't -- they go through an estrous cycle, whereas we have a menstrual cycle, women do.

And with menstruation, the women sometimes have backflow of contents that go into the pelvis and cause
10:41:01 endometriosis. Mice can't do that. So using mice for an experimental model is severely limited, and we get criticized occasionally depending upon how you're using your model.

Q. There are some things that mice are pretty poor
10:41:16 models for, and some things that they're better models for?

A. Absolutely.

Q. Endometriosis is one that they are poor models for?

10:41:21 A. They're a poor model for studying cause. They're a great model for studying drug treatment.

Q. Are there known human carcinogens that are not rodent carcinogens?

A. Not that I am aware of.

10:41:34 Q. Are there known rodent carcinogens that are not

human carcinogens?

A. Absolutely.

Q. We're gonna get very specific, sir, about the studies -- about the studies and about the -- particularly about the tumor types that Dr. Portier told the jury were the most important.

But I want to talk first about your look at some conclusions by national and international regulators. And that's something that you did towards the end of your evaluation; is that right?

A. That's correct.

Q. And you looked at the US EPA. You looked at some of the European regulators that we've talked about, the Germany BfR, EFSA and ECHA, and you looked at the UN organization called JMPR; right?

A. Correct.

Q. Why did you do that as part of your evaluation process, sir?

A. I did it for a couple reasons. One, I wanted to be sure that I hadn't missed anything. So I was looking at their reference list as well. I wanted to -- so as I said, I did my own independent search of the literature. I did a PubMed search to make sure that I had everything relevant to look at. You know, it's possible that things got missed. So I wanted to make sure.

And then when I look at the -- the international groups, such as EPA and EFSA and ECHA, my idea there was to look at their reference list, make sure I didn't miss anything there. And then also to see what their conclusions were. And I discovered that they were -- they were compatible.

Q. Okay. And we're not going to go over all of those national and international regulators I just named, sir, but one of the things that they do as part of their periodic review process, when they do a re-review, which the jury heard happens every 10 to 15 years in all those places, is assemble all the references in one place. And that's obviously a valuable resource for people who are looking into the same matters; right?

A. Exactly.

Q. That's one thing that you did.

And you also looked at their conclusions and saw that they were generally in agreement with you about the animal studies?

A. Yes.

Q. So turn to Tab 2482 in your binder, please, and tell us what that is.

MR. GRIFFIS: Exhibit 241, I'm told.

MR. WISNER: Your Honor, can we have a short sidebar?

THE COURT: Yes.

(Sidebar.)

[redacted]

[redacted]

(End sidebar.)

THE COURT: All right. You may proceed.

MR. GRIFFIS: Thank you, your Honor.

10:48:21 Q. So 2481 -- Exhibit 2481 -- which is also at Tab 2481.

Are you there, sir?

A. Yes, sir.

Q. And if you will first go --

10:48:37 MR. GRIFFIS: And we can put this up on the screen. Armando, let's first show the first page.

Q. This is the EPA's Office of Pesticide Program's report from September 12, 2016, and it's one of the documents you read; right, sir?

10:49:06 A. Yes.

Q. And it's a 227-page document including some appendices, and I'd like to direct your attention first to page 96, sir, the bottom paragraph on page 96.

A. Yes.

10:49:33 Q. So, "Based on the weight-of-evidence, the agency

has determined that any tumor findings observed in the rat and mouse carcinogenicity studies for glyphosate are not considered treatment-related."

And this is something that you looked at towards the end of your own analysis; is that right?

10:49:48

A. That's correct.

Q. And when you went through your own review, this is -- when we showed no, no, no, no, no, no for the various studies, that's the same conclusion, that they weren't treatment-related; right?

10:50:00

A. That's correct.

Q. That's the criteria that toxicologists are assessing, treatment-related as to the specific tumors that are seen; right?

10:50:11

A. Exactly.

Q. Okay. "Tumor findings observed at the highest doses tested were also not reproduced in studies in the same animal strain at similar or higher doses."

Was that a conclusion that you came to, sir?

10:50:27

A. Yes.

Q. Would you explain what that means?

A. It means that -- at the highest doses that were tested, so in the cases of the studies that were around a thousand mgs per kilogram in several studies that were --

10:50:39

MR. WISNER: Your Honor, I'm going to have to

object to him interpreting the document. That's exactly what this is not allowed for.

THE COURT: Can you approach, please?

(Sidebar.)

10:51:07 [redacted]

[redacted]

[redacted]

(End sidebar.)

THE COURT: Please, rephrase.

10:51:22 Q. BY MR. GRIFFIS: I don't want you to tell me what EPA meant when they said that, but you said you reached the same conclusion. So would you tell us what you mean by this notion of tumor findings at the highest dose not being reproduced in studies in the same animal

10:51:37 strain at similar or higher dose?

First of all, what principle of toxicology in the assessment of animal carcinogenicity studies is that?

A. The principle is the issue of reproducibility of results, and so I'm not seeing results being reproduced

10:51:54 across studies. More importantly in my own analysis of the data and looking at the high-dose groups, I did not find compound-related effects. So in the absence of compound-related effects, I guess you could argue that the data were reproducible. They were reproduced across

10:52:10 all the studies, failing to show a compound-related

effect, even at the highest dose tested, some of which I think were very high.

Q. Next, "Furthermore, even if the high-dose tumors were considered treatment-related, these findings are not considered relevant for human health risk assessment based on the use pattern and potential exposures for glyphosate."

Again, don't tell me what you think EPA meant, but is that a finding that you also came to before you looked at this?

A. Yes. In my view the high-dose group, when you're looking at -- in excess of 4,000 mgs per kilogram per day is far in excess of any relevant human exposure and wouldn't have had any value, to me, for a risk-assessment purpose.

Q. And why is it you're seeing something in animals -- rats and mice -- exposed to very, very high doses way above OECD guidelines, why is that something that you, kind of, discount for purposes of human health risk assessment and carcinogenicity?

A. Because any of the effects that I saw at that point were not compound-related. And when you're doing a risk assessment, you're looking for the adverse outcome that is the most sensitive outcome in your test animal across all the test guidelines that have been applied.

And in this particular case, carcinogenicity, even if something was present at such a high dose, it wouldn't rise to the level of being used as the point of departure for risk assessment.

10:53:49 Q. And why is that?

A. Because it would be well above anything that was seen in any of the other end points that might have been looked at.

Q. Let's go to page 131. So page 131 of the 2016 10:54:12 OPP report, and I'd like you to look at --

MR. GRIFFIS: Good thing I have a pointer that helps Armando see what I'm pointing at. It doesn't work now that he blew it up.

Q. In seven of these studies -- and we're talking 10:54:27 about the -- they actually looked at more than 12, 15 -- 9 rat and 6 mouse studies, but, "In 7 of the studies, no tumors were identified for detailed evaluation."

And then here's what I'd like to ask you about. "In the remaining studies, tumor incidences were not 10:54:45 increased at doses less than 500 milligrams per kilogram per day except for the testicular tumors observed in one study. The high-dose tumors, as well as the testicular tumors, were not reproduced in other studies, including those testing the same animal strain with similar or 10:55:04 higher dosing."

Is that consistent with your own evaluation?

A. Absolutely.

Q. And is this sort of the same issue of looking for consistency across studies, that if you see something that may be an association in one but you don't see it in others, that's an important thing to take into account?

10:55:15

A. Correct. So in one study, the testicular tumors were observed. They were of questionable relevance, even in that individual study, and in none of the other studies that I looked at did I see testicular tumors appearing. So it's -- it's a statistical artifact.

10:55:31

Q. And then, "Additionally, the tumors typically lacked a monotonic dose response, pairwise significant and/or corroborating preneoplastic lesions."

Did you also draw those conclusions during your independent evaluation?

10:55:56

A. I did, yes.

Q. And so that's going to really need some explanation. Would you explain your findings that the tumors particularly lack a monotonic dose-response?

10:56:08

A. Monotonic dose-response was the classic response I was explaining earlier where you would see, say, one tumor in your control animal, you would see 2 or 3 in your low dose, 4 or 5 in your medium and 12 in your high dose. So you're getting a typical dose-response that you

10:56:28

would expect, and that was not seen.

Q. So you talked about both study increases and then, as your career became richer, there were other versions like going up and leveling off because you're as maxed as you can on the tumor count; is that right?

10:56:47

A. Correct. You have to understand that these are discrete data points. There's discrete doses we're not looking -- as you might in another study where you would see -- say, people taking an antibiotic where they might have different concentrations in their system so we have a continuous line with people at different doses all the way through.

10:57:06

Here, we're looking at discrete units, and so you can't fit a straight line to it. It would be improper to do that. You're looking at the individual bars and seeing what's happening at that dose and asking questions, were there pairwise differences and was there a significant trend.

10:57:18

Q. Let's go to page 140, sir. It's the last page we'll look at now. And the -- I don't believe that's 140. The top paragraph on page 140, "Overall, there is not strong support for the 'suggestive evidence of carcinogenic potential' cancer classification descriptor based on the weight-of-evidence, which includes the fact that even small, non-statistically significant changes

10:57:39

10:58:05

observed in animal carcinogenicity and epidemiological studies were contradicted by studies of equally or higher quality."

So what I'd like to focus on is whether in your evaluation, sir, you found that even small non-statistically significant changes in the animal carcinogenicity studies were contradicted by studies of equally or higher quality?

10:58:19

A. In the animal studies that I looked at -- and I'm only talking about the animal studies -- I did not find any evidence of a compound-related tumor --

10:58:44

And I am aware that there are epidemiological studies out there. I'm not here to talk about those.

Q. Yes.

A. -- but I agree with this comment that they don't generally support the conclusion that glyphosate is a carcinogen.

10:59:01

Q. I'm leaving aside the epidemiologic studies, sir. For the animal carcinogenicity studies, you found contrast in the results between the studies; is that correct?

10:59:16

A. Correct.

Q. And perhaps we'll talk about that more when we look at the studies themselves.

Have you reviewed a 2010 study by Dr. George?

10:59:37

A. Yes.

Q. And that's a study that the jury's heard about in which animals were painted with various substances -- there were multiple groups that were painted with various ways. It was suggested that it was a tumor-promotion study in mice.

You say you're familiar with that, and you've seen that study, sir?

A. Yes.

Q. Not a long-term carcinogenicity study; right?

A. No.

Q. And it's not a -- was that done in a GLP lab; do you know?

A. Not in my interpretation, no. I believe it was done in an academic lab.

Q. This is Plaintiff's Exhibit 765 in your binder, and we can put that up on the screen.

MR. GRIFFIS: Permission to publish?

THE COURT: Any objection?

MR. WISNER: No objection.

THE COURT: Very well.

Q. BY MR. GRIFFIS: The first page, that's the George study that we're talking about; right?

A. That's correct.

Q. And we've heard from Dr. Portier that this study

suggests that glyphosate acts as a tumor promoter.

Do you have a view on that?

A. I agree with -- or I acknowledge that Dr. Portier has made that comment, that it's a tumor-promoter study and it's suggestive evidence of tumor promotion.

11:01:13

Q. And what's your view on that issue?

A. I found this to be a particularly weak study, and I found it to be weak for several important reasons. The reasons why I think it's a weak study are that -- unlike what I would do in my own lab or what I've done at Health Canada, they went to their local market and purchased, I think it was, Roundup, in this particular case, in India, and I'm not -- I have no way of knowing what was in that product.

11:01:31

11:01:53

And typically what we would do in Canada is I would e-mail or phone somebody at a manufacturer and ask them to provide me or sell me a product from the manufacturer, that I would get a certificate of analysis that assured me that what I was testing was indeed the pure Roundup, that it hadn't been modified, alterated in any way. So that's a little bit of a concern to me.

11:02:16

In addition, it's a tumor-promotion study. It's 20 animals per group. It's small, but it's not unheard of in these types of studies. The other issues that

11:02:37

raised concern for me is they lacked a vehicle control.

Q. What does that mean?

A. Sorry?

Q. What does that mean?

11:02:48 A. When you take the glyphosate or you take your test compound, oftentimes you have a vehicle solvent or something that you're using ethanol or acetone as the solvent and you should, when you conduct these studies -- and in my lab we always would use a vehicle control to

11:03:21 know whether or not -- so we can eliminate any potential effects that we see as being due to our test substance versus the vehicle control.

They didn't do that in this particular study, so that would be -- as a reviewer, I would find that as a

11:03:35 criticism that I would have raised with them on it. And then the other concern I had with it that they did not do any histopathological confirmation of the lesions. Now, typically you wouldn't think -- a layperson wouldn't think that would be a big problem, but I can tell you

11:03:52 from my personal experience that surgeons and pathologists often disagree.

It's not uncommon for us in the conduct of our clinical studies to get a specimen that the surgeon has said is this and then we go to the pathology report after

11:04:11 it's actually been examined and the pathologist said no,

it's not that, it does not have the cell types in it that you, the surgeon, thought would be there. So there is disagreement.

And so the lack of having that added step in there, again, gives me some pause for concern for the quality of the study, and so I assigned it low weight and I didn't give it a lot of consideration in my evaluation.

Q. Okay. Sir, this is -- it wasn't just a promotion study, it was an initiation and promotion study; right?

A. Correct. They also looked at initiation.

Q. Which means does the compound -- does Roundup -- the Roundup that they bought at a local market -- and you have a concern about that -- initiate tumors in the animals?

And what was the answer to that?

A. It did not -- there were zero tumors -- skin tumors in that part of the study, so it was not acting as an initiator.

Q. Okay. And please turn back to the OPP 2016 report, sir, and go to page 70.

A. Yes.

MR. GRIFFIS: Can we have page 70 up, Armando. 2481 with a Bates number at the bottom of 2482.

Q. So at the top of the page, it says, "A number of

studies were judged to be inadequate in protocol, conduct or reporting and were not considered in the analysis of glyphosate."

Was it your finding that this was inadequate in protocol, conduct or reporting?

A. In my view, yes.

Q. All three of those or which?

A. Inadequate in the conduct. I think they reported adequately, but it was more the protocol and the conduct.

Q. Okay. Let's go down to Number 3 and below that up.

That's the George study; right?

A. Yes.

Q. And what EPA said is, "Study deficiencies included small number...of animals, tested only males, and lack of histopathological examination."

So, small "number of animals" and "lack of histopathological examination," you just identified those two concerns.

Is "tested only males" a concern that you share?

A. I'm sorry, I didn't get --

Q. Is the fact that the George study tested only males a concern about its conduct and protocol that you share?

A. It's a limitation of the study. It's not uncommon to only look at males because they're not -- the interpretation of the results wouldn't be confounded by the stage of the estrous cycle the animals might be in. So it's not unsurprising that this type of study may have only been done in males, but, again, I would have liked to have seen -- I would have liked to have seen them make the effort to include females as well.

MR. GRIFFIS: You can take that down now, Armando. I'm about to move to a completely new section.

Would you like to take a break now?

THE COURT: Yes.

Ladies and Gentlemen, let's take the morning recess. We'll resume again at 11:20. Please do not discuss the case.

(Recess.)

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

And Mr. Griffis, you may proceed.

MR. GRIFFIS: Thank you, your Honor.

Q. Dr. Foster, I would like to take a look now at Dr. Portier's charts on rat-and-mouse studies and discuss them with you. And you're familiar with those charts; correct?

A. Yes, I am.

Q. So let's put up Slide 13 first, please.

So this is the reproduction -- it's not the original -- of Dr. Portier's rat studies tumor chart, and what we asked Dr. Portier, sir, is to identify the tumor types that drove his finding of sufficiency in the animal studies. He said that the animal studies were sufficient to show carcinogenicity and we asked him to identify the particular tumor types that drove his findings of sufficiency, and he identified for us one in rats, the skin keratoacanthoma.

MR. WISNER: Your Honor, I'm going the object. Mr. Griffis is testifying at this point.

THE COURT: Overruled. He may finish his question.

Q. BY MR. GRIFFIS: And specifically with regard to the skin keratoacanthoma, Dr. Portier testified that -- this is 2153 of the transcript, Counsel -- that the skin keratoacanthoma appear quite a bit and in the rat data, they are probably the strongest finding and the one that would be strongest to him saying glyphosate caused tumors in rats as well.

So my question for you, sir, is: Do you agree that the skin keratoacanthoma finding supports a finding that the animal data is sufficient on the subject of glyphosate causing cancer?

A. The studies themselves were, I think, for the most part -- Lankas was low dose, but for the most part, the studies were sufficient for evaluation. I would agree with his interpretation of the data, that of these
11:29:49 tumors that he noted, the skin keratoacanthoma are the ones that are probably the strongest of the data that's there.

Q. So those are the strongest ones?

A. Yes.

11:30:01 Q. From -- so let's take the others off and talk about those. Another thing he told us is that these are benign tumors; right?

A. They are, yes.

Q. And that if you're looking for a carcinogen,
11:30:18 technically these aren't carcinogenic findings; is that accurate?

A. That is accurate. We wouldn't normally consider them.

Q. Do you have a problem with a skin
11:30:30 keratoacanthoma finding in rats as evidence of human carcinogenicity, sir?

A. I do, yes.

Q. What are they?

A. Primarily that they're benign tumors and that,
11:30:38 in my experience, we would not normally consider these to

be evidence of carcinogenic findings.

Q. Why is that?

A. Because they're benign tumors.

Q. So what is the practice in the field of toxicology among people who conduct animal studies with regard to making carcinogenicity assessments as between benign and malignant tumors?

11:30:50

A. These are common tumors. We expect to see them coming up. They're consistent with the background rate. They're only the males. They're not in females as well. So overall, they're a benign tumor. I'm not seeing consistent findings. It's not something that we would -- typically, in my experience in Health Canada, would consider to be evidence of a carcinogenic finding.

11:31:11

Q. Okay. Now, the fact that it's only seen in males, why is that significant? Can't there be cancers that only appear in males or only appear in females?

11:31:28

A. That can happen or we see a difference incidence rate in one biological sex than another, but I see no -- I'm not aware of any information that would lead me to believe that this is something that I should be seeing only in males. There should be a biological sex difference for this tumor.

11:31:49

Q. There should be a biological reason why it should be seen in one sex and the not the other one?

11:32:03

A. There should be, and I don't see one.

MR. GRIFFIS: Okay. You can take the slide down.

Q. Since Dr. Portier identified four tumor types that drew him towards this conclusion that the evidence in animals was sufficient to support his findings of carcinogenicity overall, I want to spend some time on the Meyer studies.

MR. WISNER: Your Honor, I'm going to object, I believe Mr. Griffis continuously misrepresents Dr. Portier's testimony. That's really not proper. We're here for his opinions, not Dr. Portier's.

THE COURT: Make sure, Mr. Griffis, that you're phrasing your comments in the context of a question.

MR. GRIFFIS: Yes, your Honor. With your permission, can I have Dr. Foster come down and comment on the charts?

THE COURT: Yes.

MR. GRIFFIS: Thank you. And let's try this.

Q. So the first issue -- he's going to stand up so that he can --

THE COURT: Thank you.

Q. BY MR. GRIFFIS: All right. Now, the first issue that we dealt with with the mouse studies chart and the rat studies chart with Dr. Portier was the issue of

multiple testing.

A. Yes.

Q. Would you tell us what the multiple-testing problem is?

11:34:01 A. The problem with multiple testing, as I mentioned earlier this morning, is you're looking at 50 animals per sex per treatment group. You've got 400 animals, 40 tissues that you're looking at in each animal and so you've got 16,000 determinations. And as I

11:34:24 mentioned earlier this morning because the pathologist is looking for any morphological abnormality that might be present in the kidney or the thyroid and there's many different tumor types that can appear in that target tissue, we're looking at many more than comparisons than

11:34:42 16,000.

So just by random chance alone with that many comparisons, you can't help but find some that are going to be statistically significant. The problem is I don't know which ones are truly significant versus false

11:34:55 positives.

Q. So we have a chart up here with 16 different colored boxes, and they're kind of color-coded so that when you look at the same color, you're talking about the same tumor type in general. These -- these are supposed

11:35:09 to be darker blue than those. It didn't come up terribly

clearly, but those are two different colors.

So it's a color-coded box, but if this chart were designed not just to show the ones that Dr. Portier picked out to present to the jury but all the possibilities, how would it look different?

11:35:26

A. Well, the chart would probably extend out past the jury through the next courtroom and probably several streets down.

Q. And how many empty boxes not colored would we need to reflect all of the possible positives that we could have found possibly by chance alone?

11:35:41

A. In excess of 16,000.

Q. Okay. So let's talk about the multiple-testing problem and get real specific. Let's talk about the purple box there, "Spleen Composite Lymphosarcoma." That's in one of the studies the Knezevich & Hogan?

11:35:57

A. Correct.

Q. So -- and we worked together to go into the data from the Knezevich & Hogan study as published in Greim and pull out all of the instances of composite lymphosarcoma in any organ; right?

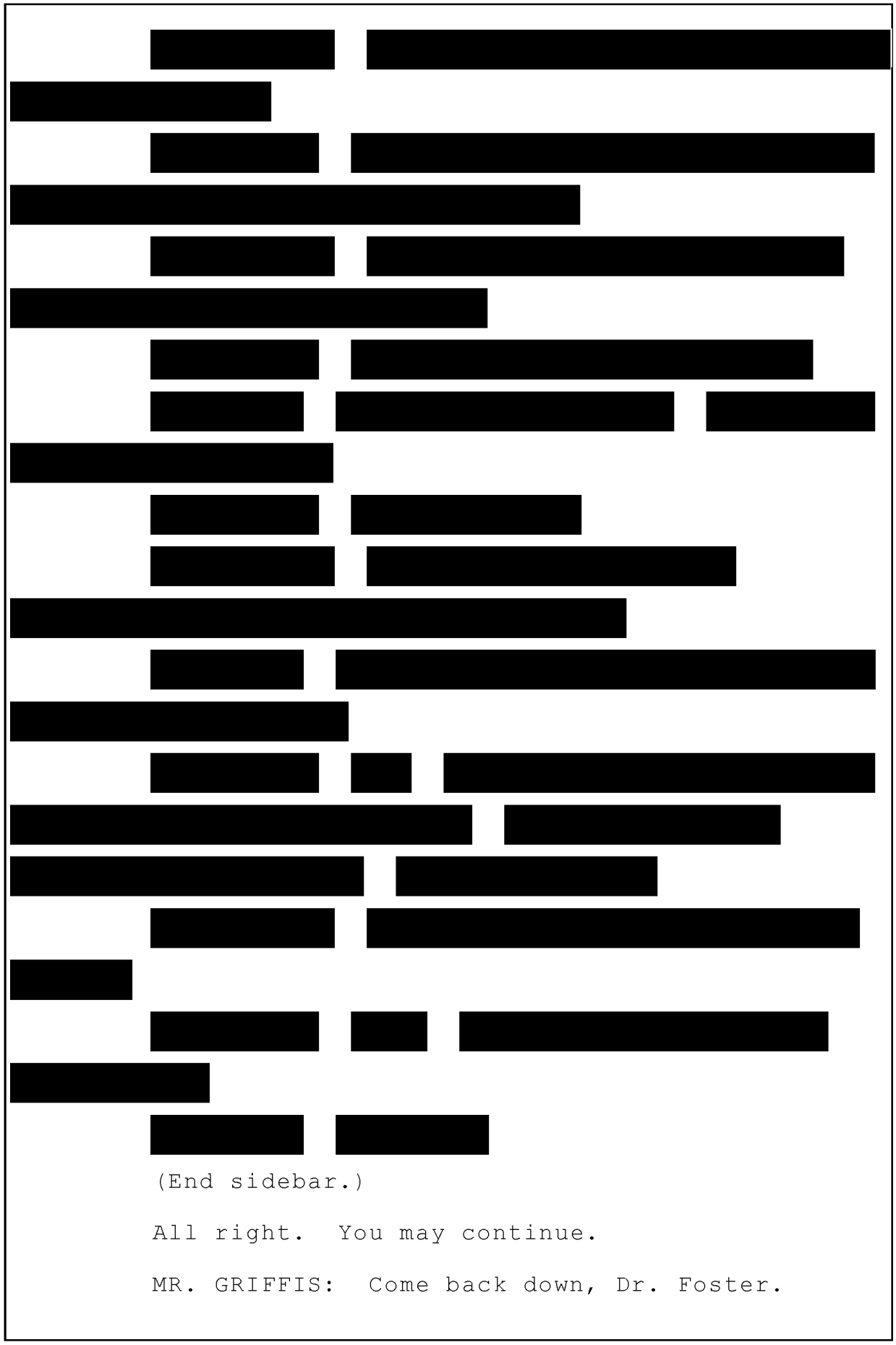
11:36:14

MR. WISNER: I'm going to object, your Honor. Can we have a quick sidebar?

THE COURT: Yes.

11:36:56

(Sidebar.)



(End sidebar.)

All right. You may continue.

MR. GRIFFIS: Come back down, Dr. Foster.

Q. So to get a little context, we are in the Greim tables -- that's Exhibit 2570 -- and the start of the data tables for Knezevich & Hogan which is page 242.

MR. GRIFFIS: Would you just put that up on the
11:38:11 screen so the jury can see it.

With your Honor's permission?

THE COURT: Very well.

Q. BY MR. GRIFFIS: Alternatively, I can show the jury what I have in my hands.

11:38:41 So that's where we went to get this information. And then after page 242, starting on page 243 and continuing to 255, what we did was pull together as an example the composite lymphosarcoma findings for all organs and that is in the males -- the male mice.
11:39:03 And we'll look at the females in a moment.

MR. GRIFFIS: That is Slide 22, I believe. Can you pull that up, please?

Q. Will you explain to the jury what this shows?

MR. WISNER: I'm sorry, what is this?

11:39:24 MR. GRIFFIS: These --

MR. WISNER: I don't understand.

MR. GRIFFIS: It's pulled from -- pulled from the data tables following the introduction to the Knezevich & Hogan. They're tables of raw data on the
11:39:39 animal findings, and we pulled the line entries on

composite lymphosarcoma for each -- yeah, and Armando's putting up some of those examples, so let's go back to 22, because we built it from that. We just pulled together and assembled on one slide all the composite
11:39:57 lymphosarcomas. If you actually looked at the tables, you'd see composite lymphosarcoma and then something else and something else.

MR. WISNER: Got it.

Q. BY MR. GRIFFIS: How does this illustrate, as an
11:40:07 example, the problem of multiple testing with regard to the purple box there?

A. Well, it highlights the point that I was making earlier that pathologists when they go and they're looking at a tissue or across different tissues that
11:40:21 there are many different cancer types that they are able to identify, and this is the composite lymphosarcoma. The composite lymphosarcoma is a tumor type that has multiple different features. And so when pathologists are reviewing slides and debating amongst themselves,
11:40:38 this has become a bit of a catch-all term, because there's some lesions with different features that don't quite fit and they lump sum them all together into this one category and call it composite lymphosarcoma.

So we have, for example, lungs -- composite
11:40:56 lymphosarcoma in the lungs and the numbers, this would be

the tumors down, zero, zero, zero, zero.

Q. Right.

A. That's obviously nothing. Composite lymphosarcoma in the liver in male mice one, zero, zero, zero, that's the control group.

Q. Correct. Another negative finding.

And these are all negative findings on this whole page for all the male mice and organs; correct?

A. Correct.

Q. Let's go to Slide 23. This is the female mice data. And what is significant about that one?

A. We're looking at one, one, one, five.

Q. And that's the one that Dr. Portier pulled out?

A. That's the one that he's pulled out to create this box.

Q. Are any of these others --

A. They're all statistically nonsignificant. So you're looking here -- again, it's a multiple-comparison problem. If you look enough times, you're going to find one that's statistically significant just by chance alone.

Q. As a toxicologist, what does this data mean to you in the collective with regard to with spleen composite lymphosarcoma in female mice in Knezevich & Hogan is a cancer signal?

A. To me, it's not a cancer signal. It's a false positive.

Q. Would you like to take the purple box off, then, sir? I'll hand you a trash can.

11:42:27 Now, was that the only multiple-testing problem that we have?

A. No. It's a problem across all of these studies.

Q. All right. That's the only example we're going to do of it, sir, but you could do the same sort of thing

11:42:42 by digging into the data on all of these issues?

A. Yes.

Q. All right. Before we get into the specific tumor types and what Dr. Portier felt were the most important ones to look at, I'd like to look at this for a

11:42:56 moment study by study, as you said that you did, and talk about the dosages in the Sugimoto and Knezevich & Hogan.

A. The dosages in the Knezevich & Hogan and the Sugimoto study are the two studies that use the exceedingly high dose in excess of 4,000 mgs per kg per

11:43:23 day, so they're four to five times higher than what we would consider to be the limit dose.

Q. And as a toxicologist, what does that tell you about how these results should be interpreted?

A. I give any findings at that level very low

11:43:35 weight. The dose is so high that -- I'm a toxicologist,

and you tell me what you want to see, I can give -- as long as I'm allowed to give as much chemical as I want, I can give you what you want. These are simply at such high doses, no one is ever going to see a concentration
11:43:56 anywhere remotely close to that anywhere on the planet. It's not realistic or relevant.

Q. When you said give me what you want, I think you may have meant exactly the opposite of what that sounds like. You can produce malignant tumors by giving enough
11:44:10 substances; is that right?

A. I can produce malignant tumors. I can give you teratogenics events. I can give you whatever you want.

Q. No, thank you. The Kumar study now -- we've heard some debate about the Kumar study and whether
11:44:24 that's confounded by illness in the animals. Have you found evidence -- written evidence that there was an issue with that in the Kumar study?

A. I found a number of issues with the Kumar study. When I read the Greim paper, he first alerted my
11:44:42 attention to the fact that they might have a viral infection, so that caused me to be a little concerned.

Q. What about a parasitic infection?

A. Then I went back and read further, and I found the pathology report from Weber, who was the pathologist
11:44:58 in the study, and Weber reported that the animals had

endoparasitic and ectoparasitic infections.

MR. WISNER: Objection. Move to strike, not in his report. This is all brand new.

THE COURT: Can you approach?

11:45:20 (Sidebar.)

[redacted]

[redacted]

(End sidebar.)

THE COURT: All right. The objection's sustained. Motion to strike is granted.

You may proceed, Mr. Griffis.

MR. GRIFFIS: Thank you, your Honor.

11:48:08 Q. So setting aside the issue of whether there was a parasitic infection, what were the flaws that you saw in the Kumar study, sir?

A. The main issue, in my view, was that this was a study that was horribly confounded, and I wasn't able to 11:48:25 interpret the results.

Q. Okay. And you did not consider the results in the Kumar study, then, in doing your analysis; is that correct?

A. Correct. I did not.

11:48:32 Q. Because of your evaluation of the inadequacy of

that study?

A. Correct.

Q. Would you take those three boxes off, then, please?

11:48:40 A. (Witness complies.)

Q. And three of those are tumor types that appear elsewhere on the chart, so we will be talking about those tumor types.

So I'd like to start with something simple, I

11:48:56 think, the harderian gland adenoma, and that was one of three that Dr. Portier said he didn't feel the evidence was strong enough to pull him forward. Could you explain why it is that a harderian gland adenoma finding wouldn't be real persuasive to a toxicologist assessing

11:49:19 carcinogenicity?

A. It not very persuasive to me because the harderian gland is a benign tumor and it's in a gland that people don't have.

Q. Okay.

11:49:25 A. It's only something that mice and rats have.

Q. Why don't you take that off, sir.

And as to lung adenocarcinomas, which is green under there under the Wood study.

A. Yes.

11:49:40 Q. Dr. Portier said exactly the same thing, that he

didn't feel it was strong enough to pull him forward.

MR. WISNER: Objection. Please stop paraphrasing Dr. Portier incorrectly. He can say, "Do you think that causes tumors," and it's about --

THE COURT: All right.

MR. WISNER: -- the same.

He keeps putting words in his mouth.

MR. GRIFFIS: I'm actually quoting Dr. Portier from the transcript, your Honor.

11:49:59 THE COURT: All right. So the objection's overruled, and you can address these issues on cross, Mr. Wisner.

You may continue.

Q. BY MR. GRIFFIS: So lung adenocarcinoma, do you 11:50:10 agree that that's not a finding that is sufficient to pull you forward in assessing carcinogenicity?

A. No, I did not. It's combined -- I don't remember it from my report, but I believe it was a nonsignificant finding, and it's not replicated in any of 11:50:24 the other studies.

Q. Okay. And what is the importance of replicability when you have five mouse studies and seven rat studies?

A. These studies are conducted according to 11:50:34 harmonized guidelines, and they're conducted according to

standard operating procedures, and standard operating procedures are -- they're a pain in the rear-end, but they're designed for the main purpose of making sure you've got reproducible results.

11:50:52 So in everything we do in our GLP study with standard operating proceedings, everything in the lab, from where we order our chemical from, there's a standard operating procedure that tells us exactly what supplier to go to, how to get it. There's a standard operating

11:51:11 procedure on how to make up the material. There's a standard operating procedure telling you down to the drawer in the lab where to find the pipettes that you're going to need so that when we go and we do the analyses on the study, we're trying to maximize the probability

11:51:27 that somebody else that does this study somewhere else is going to get the same findings that we do.

The main tenant and hallmark of everything that I do is based on the ability of being able to have it reproduced somewhere else. It really makes no sense at

11:51:44 all to have a scientist working in an institution here or elsewhere that produces results that can't be replicated by anyone else, example cold fusion. It's a waste of taxpayer money, and it's just not something we want.

Q. Sorry to interrupt you.

A. Sorry.

Q. We do need to move a little fast at the moment.

So you rejected lung adenocarcinoma?

A. Yes, I did.

Q. We also heard from Dr. Portier that toxicologists don't really use multiple malignant tumors or neoplasms. It's not really something you do in assessing carcinogenicity. Do you agree with him?

A. I do, yes.

Q. Would you take that down, the yellow.

So we're down to four that he identified as driving his findings of sufficient evidence in animal studies.

MR. GRIFFIS: That's page 2148 of the transcript, Counsel.

Q. And would you comment on why it is that you rejected the hemangioma finding?

A. Hemangioma are a common tumor --

MR. WISNER: I continue my objection, your Honor.

THE COURT: Okay. Overruled.

MR. WISNER: For the record, Kumar was on his.

THE COURT: You may answer.

THE WITNESS: The hemangioma are a benign tumor. They're very common in mice, especially as the mice age, and I saw no reason to -- that this was a

compound-specific effect.

Q. BY MR. GRIFFIS: Okay. Take that off.

Let's talk about hemangiosarcoma. That is a malignant tumor; right?

11:53:13 A. Yes, it is.

Q. Why do you not believe that that shows evidence of carcinogenicity?

A. Only seen in the high dose. It's only in the males, I believe, in this study. And again, I didn't

11:53:25 find it to be a compound-related effect, so when I'm looking at the Sugimoto and the Atkinson study, I'm seeing them confounded at the high dose.

Q. Did any of the tumors that we saw in the chart before you started taking stuff off, did the original

11:53:41 investigators, using the statistical tools they set out *a priori*, identify any of them as indicating a compound-related effect?

A. No, they did not.

Q. Did EPA or EFSA or ECHA or BfR or JMPR find a

11:53:56 compound-related effect for any of those?

A. No, they did not.

Q. And did you, in your independent evaluation before you looked at this chart, find any of them to be a compound-related effect?

11:54:08 A. No, I did not.

Q. Okay. Let's get back to -- take hemangiosarcoma off and do kidney carcinoma and adenoma. Why did you believe that those don't show evidence of carcinogenicity in animals?

11:54:18 A. In the Knezevich & Hogan study, they were nonsignificant. There was an incidence of 1013, and then when you go to the Atkinson study and you look at it and another high-dose study, the prevalence was 2200, so in my view, this is a statistical artifact. It's not -- A,

11:54:37 it's not significant, and the next study over going 2200 tells me this is -- it's not a compound-related --

Q. And the fact that they're both just in males, is that significant to your analysis?

A. Yes.

11:54:51 Q. Why is that?

A. Again, it shouldn't be biologically sex dependent.

Q. The kidney carcinomas and adenomas are not sex-driven tumors?

11:54:56 A. No.

Q. Will you take those off, please?

A. (Witness complies.)

Q. And if you'd let me get by, sir, I'm going to put up a chart.

11:55:15 Stay down, please.

A. Sorry? You said sit down?

Q. No. Stay down.

So let's talk about this chart that you helped us make. So we've been talking, like, 2200, those are the control, low dose, medium dose, high dose --

11:55:34

A. Correct.

Q. -- values, and here it's the lymphoma figures in CD-1 mouse studies?

A. That's correct.

11:55:45

Q. So we have not just Knezevich & Hogan and Sugimoto and Wood, but also -- I mean, Wood, Sugimoto and Atkinson, but also the Knezevich & Hogan data, which you didn't consider to be significant.

A. That's right. It was not significant.

11:56:04

Q. And why did you want to show the jury all of the CD-1 mouse lymphoma data?

A. The reason that I wanted to show it was severalfold. One, these tumors are occurring within the historical range for the tumor, and, moreover, the

11:56:18

average rate -- if you go to Giknis and Clifford, the average prevalence is around 12 tumors -- or 12 percent, sorry, so that's roughly 6 tumors as a mean, an average --

Q. Out of a group of 50, it's 6?

11:56:30

A. Sorry?

Q. Out of a group of 50, it's 6.

A. Out of a group of 50. So 50 is the total number of animals per group. And if you look at the dose response, you've got an impact upside down view. You've got something here that doesn't make any sense. You've got a U shape, and then you've got a linear increase. All of them are below the average historical background, and in none of them the dose response is consistent across studies. All of them, in my opinion, are nothing more than statistical noise.

11:56:42

11:56:59

Q. Now, to a statistician, this sure looks like a linear increase, doesn't it, 0, 1 is bigger than 0, 2 is bigger than 1, 5 is bigger than 2?

A. Correct.

Q. What does it look like to a toxicologist?

11:57:12

A. Well, first off, I know that most lymphomas are a common tumor in mice, so I'm not surprised that I see some in my control group. I'm surprised that I'm not seeing any there. And from a statistical point of view, if you have 0 in your control group, that's going to artificially create the probability that it's statistically significant, because you've got no events in that -- in that dose group, and I know there should be some.

11:57:27

11:57:39

Q. And so the jury can see that you're not just

making up this 12 percent figure, can we put up Defense Exhibit 3114, the Wood analysis on page 3?

MR. GRIFFIS: Any objection?

MR. WISNER: No objection.

11:58:06 Q. BY MR. GRIFFIS: So let's go to the first page of this, so you can tell the jury what it is first? What is this?

A. This is the Safepharm report on their -- their control study looking at the background rates.

11:58:20 Q. So this is a background rate from the same time?

A. Same -- same -- contemporary, same ops, same investigators.

Q. Okay. And then let's go to page 3, top paragraph.

11:58:31 And right there, 6 male mice, 12 percent, and 6, 12 percent female mice developed malignant lymphoma.

Is that the 12 percent figure from contemporaneous data, sir?

A. That's from contemporaneous data, and it also

11:58:58 happens to agrees with Giknis and Clifford.

Q. Okay. And Giknis and Clifford, that's Defense Exhibit 2552.

MR. GRIFFIS: Permission to publish that, your Honor?

11:59:04 THE COURT: Any objection?

MR. WISNER: What is it?

MR. GRIFFIS: 2552. Defense Exhibit 2552, Giknis and Clifford.

MR. WISNER: No objection.

11:59:13 THE COURT: Very well.

Q. BY MR. GRIFFIS: So what is this, sir?

A. This is the Giknis and Clifford report looking at CD-1 mice.

Q. And it's reporting on the control group finding 11:59:23 from a whole bunch of studies from around the right time period from CD-1 mice?

A. And so that actually is 46 studies they looked at.

Q. So let's go to page 21, and flip it sideways, 11:59:38 and go to malignant lymphoma, which is the first thing under whole body, multiple organ systems. And those numbers, those are the control numbers for malignant lymphoma in male mice from a whole bunch of studies from around the same time period; right?

11:59:57 A. Correct. And so you're seeing a range from a low of 0 to as high as 7.

Q. Let's go to the next page where the study -- the study count from here goes from 1 to 23. The next page we go from 24 to 46, call out the same line, and what's 12:00:13 our range of numbers there?

A. Again, this is 0 and a high of 13 in this case.

Q. So we saw a 7. We saw a 6. We see a 13, and you picked 6 as a reasonable top for the historical range; right?

12:00:26 A. I looked at the mean, and that was 6 in this case. 12 percent gives the number 6. 12 percent of 50 is 6.

Q. So when you see a scattering of numbers in the CD-1 mouse studies from malignant lymphoma at or below 6,

12:00:44 how do you interpret that as a toxicologist, sir?

A. As a toxicologist, this tells me it's within the normal range of what would be expected, whether I gave them glyphosate or not.

Q. Okay. So what's your bottom line on malignant

12:00:59 lymphoma?

A. My bottom line on malignant lymphoma is, again, these are not compound-related tumors.

Q. Would you take those down?

A. (Witness complies.)

12:01:13 Q. And take your seat. I just have a couple more questions for you, sir.

So we've been looking at these figures for malignant lymphoma, some figures for -- specific figures on the issue of multiple testing for the one purple box

12:01:48 there. You have seen real rodent carcinogens a number of

times in your work; right?

A. Yes.

Q. And what do those numbers look like?

A. Typically, what I expect to see is that we get reproducible results, so if I'm fortunate enough to have a multiple study, such as this, which is not common, but where we do have replication, we're seeing tumors that are in the ballpark of -- in the low-dose group, they might be around five or six, but as we move into the high-dose group, you're looking at tumors in almost half the animals.

Q. Okay.

A. And you might also be seeing multiple tumors in the same animal. So they -- for instance, in a kidney tumor, I might see a kidney tumor where there are -- sorry -- a section through the kidney with multiple tumors in --

Q. By "multiple tumors," you don't mean a kidney tumor and a hemangiosarcoma?

A. No.

Q. You mean a kidney tumor and a kidney tumor and another kidney tumor?

A. Yes.

Q. Okay. And then when you go to another study, if you have the luxury of having another study, what are you

going to see if you have something that is, in fact, a kidney carcinoma?

A. Similar results.

Q. So you'll see, again, kidney tumors?

12:03:04 A. Yes.

Q. And if you have a dozen studies, what are you going to see for this true carcinogen?

A. You should see it replicated across those studies.

12:03:12 Q. And when you talked about a dose response, you told the jury about two dose response profiles, you know, low, up, up, up and then the up, up, up flattening. You wouldn't normally see up down up or up down as you move to the higher doses; is that right?

12:03:32 A. No. If we're dealing with a true carcinogen, we're going to be seeing that it's going to increase with dose. The pattern of what that looks like, it might be something that reaches a plateau or -- after the medium dose, or it might continue to rise into the high dose, 12:03:51 but it's going to be increasing.

Q. And the size of the effects that you see, how big are the numbers likely to be?

A. How big are the numbers or --

Q. Yes. I mean, we've been talking about 12:04:06 numbers --

A. I mean, it --

Q. -- like 2200, 0125.

What are the numbers for carcinogens --

A. They go beyond the background or the historical spontaneous range.

MR. GRIFFIS: Your Honor, I may have another area to enter into with Dr. Foster. So that I can deliberate about that, this might be a good time to break for lunch.

THE COURT: All right. Very good.

All right. Ladies and Gentlemen, we're going to break now for the lunch recess. Please remember: Do not discuss the case with each other or anyone else. Please don't do any research. We'll resume again at 1:30.

Also, please expect a schedule change next week. Next week by Wednesday I suspect either you'll be in deliberations or, if not, the lawyers will be giving their closing arguments, but in any event, I do need for all of you to be here on Wednesday next week, so next week just, please, assume that you need to be here for the full week.

Thank you very much. I'll see you all at 1:30.

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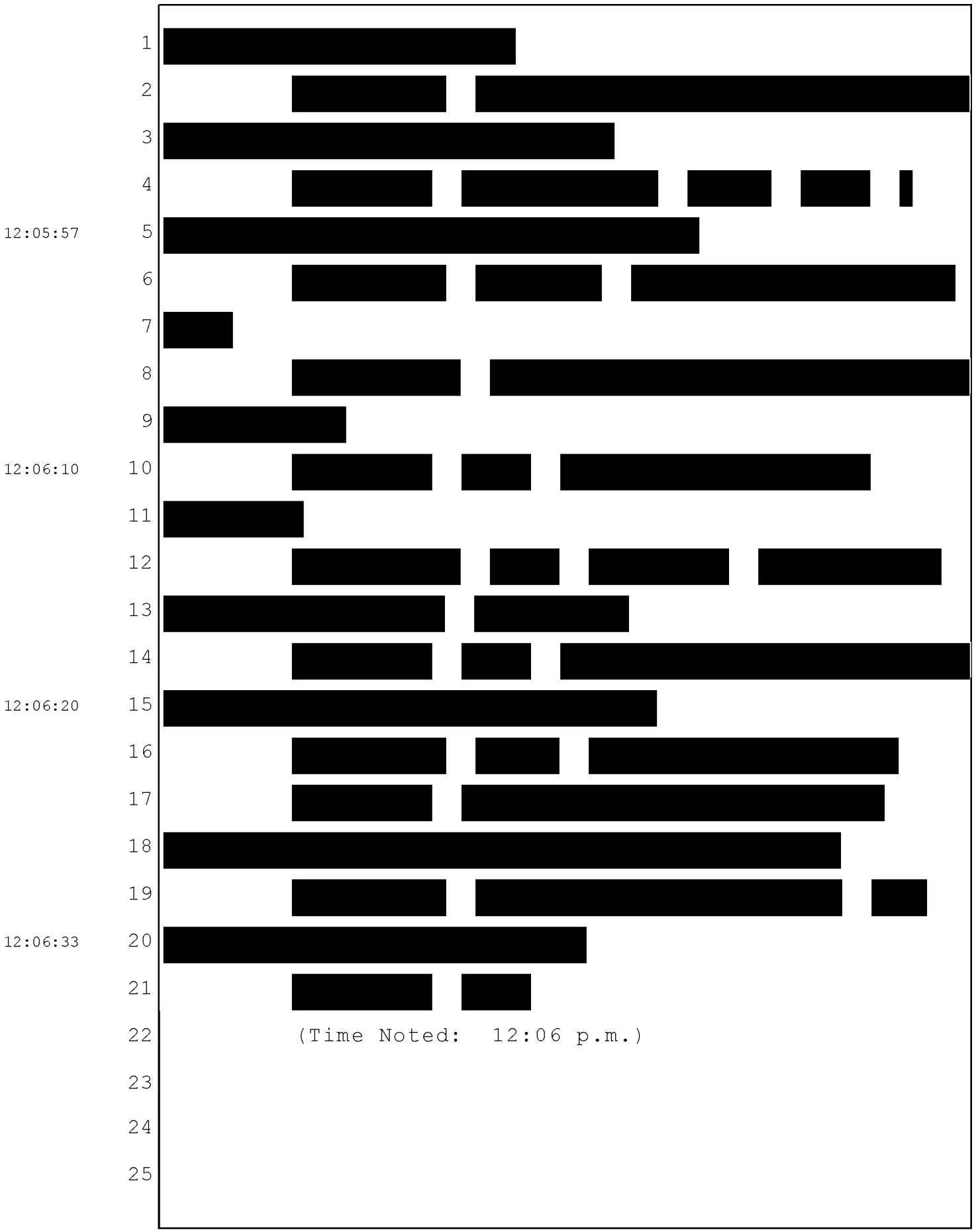
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(Time Noted: 12:06 p.m.)

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: August 1st, 2018.

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Leslie Rockwood Rosas
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State of California
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