EXHIBIT 98

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

MDL No. 2741, Case No. 16-md-02741-VC

VIDEOTAPE DEPOSITION OF:
CHARLES W. JAMESON, Ph.D. - September 21, 2017

IN RE: ROUNDUP PRODUCTS
LIABILITY LITIGATION

This document relates to:
ALL ACTIONS

PURSUANT TO NOTICE, the videotape deposition of CHARLES W. JAMESON, Ph.D., was taken on behalf of the Defendant, Monsanto Company, at 7171 W. Alaska Drive, Lakewood, Colorado 80226, on September 21, 2017 at 9:03 a.m., before Tracy R. Stonehocker, Certified Realtime Reporter, Registered Professional Reporter and Notary Public within Colorado.

JOB NO. 130141

A P P E A R A N C E S.

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Also Present:

John Jensen, Videographer
Robyn Buck, Esq.

I N D E X

(All exhibits were marked by Mr. Hollingsworth.)

WHEREUPON, the following proceedings were taken pursuant to the Federal Rules of Civil Procedure.

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THE VIDEOGRAPHER: This is the start of media labeled number one of the video-recorded deposition of Dr. Charles W. Jameson In Re: Roundup Products Liability Litigation in the United States District Court, Northern District of California, Number 16-md-02741-VC.

This deposition is being held at 7171 West Alaska Drive, Lakewood, Colorado on September 21, 2017 at approximately 9:03 a.m.

My name is John Jensen. I am the legal video specialist for TSG Reporting, Inc. headquartered at 747 Third Avenue, New York, New York. The court reporter is Tracy Stonehocker in association with TSG Reporting. Counsel, please introduce yourselves.

MS. WAGSTAFF: Good morning. Aimee Wagstaff on behalf of the plaintiffs.

MS. ROBERTSON: Pearl Robertson on behalf of plaintiffs.

MR. HOLLINGSWORTH: Joe Hollingsworth, Hollingsworth, LLP on behalf of Monsanto.

MR. HAAKE: Christopher Haake also with Hollingsworth, LLP on behalf of Monsanto.

MS. BUCK: Robyn Buck with Monsanto.

MS. WAGSTAFF: I believe we have some folks on the telephone.

MR. ESFANDIARY: Pedram Esfandiary with plaintiffs.

MS. KLENICKI: Erica Klenicki from Hollingsworth on behalf of Monsanto.

* * * *

CHARLES W. JAMESON, Ph.D., having been first duly sworn to state the whole truth, testified as follows:

(Deponent's reply to oath: I do.)

MS. WAGSTAFF: Mr. Hollingsworth, before we get started, I'd like to correct three typos from Dr. Jameson's expert report and they all three are the same word that was auto-corrected or somehow changed. On page 22, and this is the report dated -- it's not dated, but it's his MDL report. On page 22, about third of the way down, if you want to look over here, like right there.

MR. HOLLINGSWORTH: Yup.

MS. WAGSTAFF: It says,

"Hemangiosarcomas" and it should say "hemangiomas" and the correct line should read, "The EPA also reported," footnote 86, "that hemangiosarcomas in female mice were found to occur with a statistically significant trend in the study," and then it gives a parenthesis with a bunch of numbers, "and the tumor incidence in the high dose female mice was statistically significant with p=0.028 as compared to concurrent controls."

The next one is on page 28. And it's the same correction on the very bottom line of page 28. Once again, it says, "hemangiosarcomas" and it should say "hemangiomas." The correct sentence should read, "There was also a significant positive trend for the formation of adenocarcinomas of the lung in male CD-1 mice in one study," footnote 78, "and hemangiomas in female CD-1 mice in another study."

And the last typo related to this is on page 29 in the second paragraph, the first sentence in the second paragraph, which is really long, right after the footnote 78, it says, and "hemangiosarcomas" and it should say and "hemangiomas" and those are the three. I love that word.

MR. HOLLINGSWORTH: What's the last one?

MS. WAGSTAFF: Okay. Page 29.

MR. HOLLINGSWORTH: Yep.

MS. WAGSTAFF: It's right here.

MR. HOLLINGSWORTH: Right in the middle?

MS. WAGSTAFF: The first --

MR. HOLLINGSWORTH: Okay. I see.

MS. WAGSTAFF: -- sentence right after footnote 78 in parenthesis, "study 74," and it should say "hemangiomas in female in one study period." Got it?

MR. HOLLINGSWORTH: Yep.

EXAMINATION

BY MR. HOLLINGSWORTH:

Q. Good morning, again, Dr. Jameson.

A. Morning.

Q. If you don't understand one of my questions or you want me to repeat it, feel free to do so. If you want to take a break, just let me know.

A. Okay.

Q. As you know, we'll be proceeding in a question and answer format here. I'm going to ask the questions and I hope you'll give me the answers. Listen carefully to what they said -- what I ask you and I'll be happy to repeat a question or clarify it for you if you'd like. Okay?

A. Okay.

Q. The hypothesis that mouse renal tumors are predictive of human NHL has never been tested, has it?

A. Well, in any rodent bioassay, the purpose of doing the study is to see if a material that you're investigating can cause cancer in the experimental animal, and it's been shown that most chemicals that have been shown to be carcinogens in experimental animals are also carcinogens in humans. Not all, but a large majority. If they're positive in animals, it's likely they will cause cancer in humans. That's why you perform the study to see if they cause cancers in the animal as kind of a predictive tool to say, well, there's potential that this chemical will cause cancer in humans.

Q. I'm asking a slightly different thing. I'm talking about a specific kind of cancer in humans, do you understand that, called non-Hodgkin's lymphoma or NHL?

A. Uh-huh.

Q. My question is whether the hypothesis that mouse renal tumors are predictive of non-Hodgkin's lymphoma specifically in humans has ever been tested?

A. Again, this -- you know, the purpose of

a bioassay is to see if the chemical can cause cancer in the animals as a predictive tool for what it -- if it causes cancer in humans. Now, I mean, the fact that something causes a kidney tumor in a mouse, I don't know what that says about causing non-Hodgkin's lymphoma in humans. I don't know that's been investigated. I don't know that anyone has actually done a study to see if you cause a renal tumor in a mouse, if there's some kind of mechanism in the mouse that is similar to a mechanism -- known mechanism in humans that goes on to non-Hodgkin's lymphoma. I don't know if any type of study like that has been done.

So, again, it's really not a relevant question to say, well, you got kidney tumors in a mouse, what does that say about non-Hodgkin's lymphoma. The purpose of doing the study in the mouse is to see if it causes cancer and that's used as a predictive tool to see if it causes cancer in humans.

Q. You understand the proceeding that we're about to embark in in the MDL part of this case has the specific question whether glyphosate can cause non-Hodgkin's lymphoma in humans?

MS. WAGSTAFF: Object to form.

A. I'm sorry, could you ask that again?

Q. (BY MR. HOLLINGSWORTH) Sure. You understand that the procedure -- the legal proceeding that we're about to embark on in the multidistrict litigation case that your report has been submitted in states that the purpose of the proceeding is to determine whether glyphosate can cause non-Hodgkin's lymphoma in humans.

MS. WAGSTAFF: Object to the form.

Q. (BY MR. HOLLINGSWORTH) Do you understand that?

A. Well, the litigation, yeah, I -- that's my understanding that the litigation is over -- -- that exposure to glyphosate caused non-Hodgkin's lymphoma in an exposed population or exposed individual.

Q. And your testimony is that the question of whether renal tumors are predictive of non-Hodgkin's lymphoma, that is, mouse renal tumors is predictive of non-Hodgkin's lymphoma has not been studied as far as you know?

A. I'm not aware of any publications or any research that has been done. That's not to say that it hadn't, but I haven't come across it yet.

Q. You didn't cite any publication or study in your report in this case which says that renal

tumors in mice are predictive of non-Hodgkin's lymphoma in humans, did you?

A. No. I did not have any citations in my report to that effect, no.

Q. Sir, I have your report here, what I think is your report and I've marked it as 22-1 and it's titled "Expert Report of Dr. Charles Jameson, Ph.D. in Support of General Causation on Behalf of Plaintiffs." Do you see this?

A. Uh-huh.

Q. And I hand -- in my handwritten notes in that version of your report, which you have before you, I marked in the corrections that were made in three or four different places from the term "hemangiosarcoma" to "hemangioma," which is what you wanted to do, right?

A. Right.

Q. That's the correction you wanted to correct, you wanted to change the "hemangiosarcomas" that you referred to in those four places to the word "hemangiomas"?

MS. WAGSTAFF: Three.

A. In three places in the study in female CD-1 mice.

Q. (BY MR. HOLLINGSWORTH) Yes.

A. The typo was -- originally said "hemangiosarcoma" and it should have read "hemangioma."

Q. Is there any data that you've cited in your report that records what the error rate would be in predicting non-Hodgkin's lymphoma based on renal tumors in mice?

A. Could you please define what you mean by "error rate."

Q. What I mean by error rate is the rate of error in a test -- in a study that's been done involving renal tumors in mice that are predictive for non-Hodgkin's lymphoma. And I take it since you said it hadn't been published in your prior answer that there is no such study involving what the rate of error is in such a situation?

MS. WAGSTAFF: Object to form.

A. I do not know of any published studies that have looked at that. That's not to say there isn't, but I haven't found any. But, again, I would say the purpose of the study in the mouse was to see if the glyphosate would cause cancer. That was the purpose of the study.

Q. (BY MR. HOLLINGSWORTH) Yes.

A. The purpose of the study wasn't to see

if -- if -- if you got a -- cancer in the kidneys of the mouse it was related to non-Hodgkin's lymphoma.

Q. Yes.

A. So that wasn't the purpose of the study.

Q. I understand that. But the purpose of this hearing is to determine whether glyphosate causes non-Hodgkin's lymphoma in humans and that's why I'm asking you these questions. Do you understand that, Dr. Jameson?

MS. WAGSTAFF: Object to form. By the way, plaintiffs are alleging that glyphosate formulations is what is causing NHL, as well as just glyphosate.

Q. (BY MR. HOLLINGSWORTH) Can you answer my question?

A. I'm sorry, could you repeat it?

MR. HOLLINGSWORTH: Can you read it back, please, Tracy?

(The question was read back as follows: "I understand that. But the purpose of this hearing is to determine whether glyphosate causes non-Hodgkin's lymphoma in humans and that's why I'm asking you these questions. Do you understand that, Dr. Jameson?")

MS. WAGSTAFF: Object to form.

A. I'm sorry, are you saying the purpose of -- of today of this deposition is to do that?
Q. (BY MR. HOLLINGSWORTH) I'm referring to the legal proceeding, the hearing that we're having eventually in which your report is going to be introduced and I assume you're going to testify.
MS. WAGSTAFF: Objection, calls for a legal conclusion.
Q. (BY MR. HOLLINGSWORTH) The purpose of that hearing is to determine whether glyphosate can cause non-Hodgkin's lymphoma in humans and you understand that, right?
MS. WAGSTAFF: Objection, calls for a legal conclusion.
A. I understand that I've been asked my expert opinion about if -- if glyphosate and glyphosate formulations cause non-Hodgkin's lymphoma in humans.
Q. (BY MR. HOLLINGSWORTH) Your report says in the last sentence, if you look at it, that your opinion is based on a reasonable degree of scientific certainty is that glyphosate can cause non-Hodgkin's lymphoma in humans, doesn't it? Can't you remember that without looking at your report?
MS. WAGSTAFF: Objection. Don't get

aggressive.
A. You're asking what my report says, so. . .
Q. (BY MR. HOLLINGSWORTH) The last sentence. The last sentence --
MS. WAGSTAFF: Go to the last page.
A. The last page, last sentence of my conclusion?
Q. (BY MR. HOLLINGSWORTH) Yes.
A. The last page of my conclusion says, "I also conclude to a reasonable degree of scientific certainty that glyphosate and glyphosate-based formulations cause non-Hodgkin's lymphoma in humans."
Q. Okay. Have you ever published a study that says mouse renal tumors are predictive of non-Hodgkin's lymphoma in humans?
A. Okay. Me, personally, I have not published a paper that addresses the issue of the relationship of kidney tumors in mice to non-Hodgkin's lymphoma in humans.
Q. Have you ever attended a lecture where there was a discussion of whether or not mouse renal tumors are predictive of non-Hodgkin's lymphoma in humans?
A. Not that I recall. I've attended many

lectures and seminars about the results of animal bioassay studies where the material being investigated had caused kidney tumors in mice, but to the best of my knowledge, I don't recall that any of the investigators that were -- that -- that were performing this study were investigating the -- any type of an association between the possible formation of kidney tumors in mice and non-Hodgkin's lymphoma in humans. I just don't think anybody has looked into that.
Q. Okay. Thank you. When IARC's committee on monograph 112 met, it wasn't your purpose to sit down and decide whether glyphosate caused non-Hodgkin's lymphoma in humans, was it?
A. Well --
MS. WAGSTAFF: I'm going to allow this question, but I will note for the record that you guys have already deposed him on the deliberations and the purpose of the IARC 112 meeting. That is not what he is being presented for today. So if you go too far into it, I'm going to instruct him not to answer. You can answer.
A. Okay. So -- I'm sorry, could you repeat the question?
Q. (BY MR. HOLLINGSWORTH) When the IARC

monograph committee on -- monograph 112 sat down to deliberate, it was not your purpose to determine whether glyphosate can cause NHL in humans, was it?
A. Well, the IARC monograph or the International Agency for Research on Cancer holds these working group meetings to evaluate the potential carcinogenesis or the potential cancer-causing ability of particular materials that they had identified for review. Now, the reviews are based on publicly available information and the peer-reviewed literature and it's also made -- also from government publications. And also publicly available information that -- that other -- any individual could submit for review by the working group.
Now, the working group is instructed to review all the data, and then in the preamble of the IARC monograph, there is a set of criteria that the individuals are instructed to evaluate the data based on the criteria that is outlined in the preamble. The preamble -- and the data that is looked at for a monograph includes human data, animal data and mechanistic data.
So in investigating the human data for a chemical, the epidemiology is investigated. All the epidemiology data that's available is evaluated and

it's determined if there is evidence that the particular material causes cancer in exposed human populations, and it is also part of this evaluation that they identify the tumor sites where the chemical caused the increase in tumors in the human population.

So following that line of logic, if you will, it was the purpose of the IARC monograph to evaluate the human epidemiology data and to determine if it did cause cancer in humans and at what particular sites in humans or what particular type of tumors in humans the cancer is -- is formed.

Q. Okay. The IARC committee was not able to determine that there was sufficient epidemiologic evidence to say that glyphosate causes non-Hodgkin's Lymphoma in humans, was it?

MS. WAGSTAFF: Object to form.

A. Well --

Q. (BY MR. HOLLINGSWORTH) Can you answer my question yes or no?

MS. WAGSTAFF: Objection. Can you let him answer before --

MR. HOLLINGSWORTH: Sorry.

A. The --

Q. (BY MR. HOLLINGSWORTH) My question is --

A. The criteria --

Q. My question arises not from -- I'm not -- I don't want to go into your prior deposition. I really didn't intend to. But I'm referring back to the last sentence of your report, which you read into the record.

And my question is, whether the IARC committee determined that there was sufficient evidence to say that glyphosate causes non-Hodgkin's Lymphoma in humans?

A. Okay. Well, that was --

MS. WAGSTAFF: Hang on. I object to that because you are suggesting that his expert report is based on what the IARC determined and this is an expert report from Dr. Jameson. It's not a regurgitation of the IARC and he wasn't constrained by the IARC rules, definitions and preamble in his expert report, but answer if you can.

A. Okay. Well, that's what I was basically going to say. The opinion in my report is my opinion.

Q. (BY MR. HOLLINGSWORTH) Okay.

A. It has nothing to do with the -- with what IARC did or with what IARC said. Now, as far as the IARC not finding -- I'm sorry, what did he say, sufficient evidence?

Q. Sufficient evidence.

A. Okay. The criteria, as I indicated previously, that is -- that is listed in the preamble of the IARC monograph has definitions of what is meant for sufficient evidence, for limited evidence, for inadequate evidence and what have you. And so if you look at the different definitions, sufficient evidence means that their causation is credible and there are no confounders.

I'm paraphrasing, but basically it -- the data is positive and confounders and what have you have been accounted for and do not affect that observation.

The second one, which is limited says a -- an association between the material and cancer is a very credible -- means that there's evidence that it causes -- that the material causes cancer in humans. The evidence is there. But there are some issues of, you know, bias or confounding or chance that just haven't been adequate -- just can't be adequately addressed, so that's why they say that the evidence is limited. So that's why IARC came up with -- had to say limited because of the restrictions of the criteria.

Q. IARC was not able to say that there was

sufficient evidence that glyphosate causes NHL in humans, correct?

MS. WAGSTAFF: Objection, asked and answered.

A. Again, if you look at the preamble, the IARC has criteria and the criteria that you are required to evaluate the data against is listed -- is in there and the working group members are told you have to use -- apply this criteria in your overall evaluation.

So -- and the overall evaluation, the IARC working group -- now, this is a whole working group, it's not just the human subgroup. The whole working group came to the conclusion that causation of -- between glyphosate, glyphosate formulations and non-Hodgkin's lymphoma is a credible evaluation that the data says that glyphosate and glyphosate formulations cause non-Hodgkin's lymphoma in the exposed population.

But there were some -- some other issues like bias or chance or what have you that came into play that they could not explain away, so it met the limited criteria.

Q. (BY MR. HOLLINGSWORTH) And the IARC committee, therefore, was not able to say that there

was sufficient evidence that glyphosate can cause NHL in humans?

MS. WAGSTAFF: Objection, this is the third time that you've asked that question.

MR. HOLLINGSWORTH: Well, he's not answering my question.

MS. WAGSTAFF: He is answering. If you don't like --

MR. HOLLINGSWORTH: Despite your coaching.

MS. WAGSTAFF: If you don't like his response, I'm sorry, but he's answered very sufficiently.

A. I'm going to give you the same answer.

Q. (BY MR. HOLLINGSWORTH) Can you show me from the IARC report where they say that glyphosate can cause non-Hodgkin's Lymphoma in humans?

A. I can show you where it says it is evidence -- yeah, that there is evidence -- the evidence is credible that glyphosate and glyphosate formulations cause non-Hodgkin's lymphoma.

Q. You're saying that the IARC committee said that?

A. In the monograph.

Q. That there was sufficient evidence

to --

A. No.

MS. WAGSTAFF: Objection.

A. I did not say that.

Q. (BY MR. HOLLINGSWORTH) Okay. So there wasn't sufficient evidence to say that, but they said it never -- nevertheless, is that what you're testifying to here today?

A. I did not say that either.

MS. WAGSTAFF: Objection, asked and answered five times.

Q. (BY MR. HOLLINGSWORTH) Sir, is the -- has the hypothesis that mouse hemangiosarcomas are predictive of non-Hodgkin's lymphoma been tested?

A. Again, you have a similar situation to what you have with the kidney tumors in mice. The studies were conducted to see if particular material would cause cancer in animals. The study indicated that hemangiosarcomas were caused in this particular study. And there was a significant increase in these tumors in the animals, so there's -- it can be said that glyphosate caused the hemangiosarcomas in that particular study.

But to my knowledge, I don't know that anybody has done an investigation to see -- to see if

there is a correlation between the formation of hemangiosarcomas in laboratory animals and non-Hodgkin's lymphoma in humans, but the study does say that glyphosate causes hemangiosarcomas in experimental animals, so it's an animal carcinogen and, therefore, it could possibly cause cancer in humans.

Q. Has anybody done an investigation of whether or not findings of mouse hemangiomas are predictive of non-Hodgkin's lymphoma in humans?

A. Again, the study was conducted to see if glyphosate could cause hemangiomas or any cancers, in this case, I believe it was in female mice. The results of the study indicated that exposure to glyphosate did cause hemangiomas to be formed in the female mice, so, therefore, it -- glyphosate caused hemangiomas in mice, so it's an animal carcinogen and a potential carcinogen in humans.

To the best of my knowledge, I don't know that anybody has done an investigation where they exposed animals to glyphosate and to investigate if there was an association between formation of hemangiomas in female mice and non-Hodgkin's lymphoma in humans. I don't think it -- I'm not aware that anybody has done and/or published any research in that

particular area.

Q. Are you aware whether anybody has done or published research in the area of an investigation of lung adenocarcinomas and their predict -- their predictability of non-Hodgkin's lymphoma in humans? I'm talking about lung adenocarcinomas.

A. Lung adenocarcinomas?

Q. Yes.

A. The study was conducted to see if glyphosate caused cancer in the experimental animals. The result of the study was lung adenocarcinomas were formed, so therefore glyphosate caused lung adenocarcinomas in the experimental animals. It is therefore an animal carcinogen and a potential human carcinogen.

I do not know if anybody has done an experiment to investigate any type of association of the formation of hemangiomas -- I'm sorry, lung adenocarcinomas in the experimental animals and non-Hodgkin's lymphoma in humans.

Q. Has anybody done an investigation of the relationship between rat testicular interstitial cell tumors and non-Hodgkin's lymphoma in humans to your knowledge?

A. I'm -- I'm going to give you a similar

answer to what I've given to all of them. The study was conducted on experimental animals to see if glyphosate caused cancer in the experiment. In this particular study, I believe it's in male rats, the glyphosate was found to cause an increased incidence of interstitial tumors of the testes in the male rats. Therefore, exposure to glyphosate caused interstitial tumors in the male rats.

It is positive animal carcinogen for male rats because of the tumors and is, therefore, a potential human carcinogen.

Again, I'm not aware of anyone doing any research or publishing any papers that did an investigation of the formation of interstitial cell tumors of the testes in male rats and non-Hodgkin's lymphoma in humans.

Q. Would you give the same answer for rat hepatocellular adenomas?

A. I would.

Q. Would you give the same answer for rat pancreatic -- pancreatic islet cell tumors?

A. I would.

Q. And would you give the same answer for rat thyroid follicular tumors?

A. I would.

Q. Would you give the same answer for rat -- excuse me, for mouse -- mouse lymphoma?

A. I would give the same answer for mouse lymphoma, but I might give a little side comment that the lymphomas are a particular tumor type that is similar to the lymphoma -- non-Hodgkin's lymphoma that is humans.

In other words, you're forming a lymphoma in the animals and what you're talking about is non-Hodgkin's lymphoma in humans, so that's a little more closely associated with the actual human tumor site and -- but, again, I'm not aware of anybody doing any research or publishing any paper where they -- they investigated the formation of the mouse lymphomas and its association to non-Hodgkin's lymphoma in humans, but there may be, but I'm not aware of any.

Q. You didn't cite anything in your report in this case, sir, in which you relied on any publication that states that the experimental mouse system is a valid model for predicting non-Hodgkin's lymphoma in humans, did you?

A. No, I did not use any reference to that effect, no.

Q. Isn't it true that the current

literature indicates that the mouse system is not a good -- not a good predictor of lymphoma in humans?

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) For a number of reasons?

MS. WAGSTAFF: Object to form.

A. There may have -- may be some publications in the literature to that effect, but, again, the purpose of doing these studies is -- most -- the studies -- the purpose of doing an animal bioassay study is to determine if the chemical can cause cancer in the experimental animals. And it's not -- not looking to investigate does it form a specific kind of tumor that is the same as found in humans. At least routinely that's not the case.

Now, sometimes -- I think the state of the art is that you can develop genetically modified test species, transplant human genes into an animal or something like that and do some studies that may give you some more information as to the formation of the cancer in humans based on the special -- special animals, but I'm not familiar with that research, and I can't speak to that right now, but I know that type of research is being done.

I have no idea if there's anything being

done with non-Hodgkin's lymphoma. I haven't looked into that, to be honest.

Q. Your paper doesn't cite any study involving genetically modified mice who've been injected with human genes to determine whether or not there's a relationship between mouse lymphoma and non-Hodgkin's lymphoma in humans?

A. I'm not aware of any, and I don't have any. I did not cite any in my report.

Q. So the answer to my question is no?

MS. WAGSTAFF: Objection, argumentative.

A. I don't have any in my report.

Q. (BY MR. HOLLINGSWORTH) Okay. In fact, doesn't the current literature say that the mouse system -- the mouse system is not a good model for predicting non-Hodgkin's lymphoma or any lymphoma in humans because malignant lymphoma in mice has such a high background incidence in control animals that have not been fed any substance?

MS. WAGSTAFF: Objection, asked and answered.

A. I'm -- I'm not aware of the arguments that it's not a good model. I mean, of -- I'm not aware of the arguments that it's a not a good model for non-Hodgkin's lymphoma because of the high

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background incidence of lymphomas in mice. It's an argument that the mouse isn't a good model for looking for lymphomas for the cause -- for a chemical to cause lymphomas in mice because of the high background level in mice.

Q. (BY MR. HOLLINGSWORTH) Thank you. You have -- you have written papers on -- when you were at the NTP down at research triangle park about the interpretation of experimental animal studies in order to decide whether or not a substance is a carcinogen or not, haven't you?

A. True.

Q. And you've written those papers with people like Joe Haseman?

A. I've -- I am co-author of a couple of papers with Joe Haseman, yes.

Q. And Dr. Huff?

A. And James Huff.

Q. Is Dr. Huff still living?

A. Yes. I believe he is.

Q. In -- in those papers, you and your colleagues at NTP said that to determine whether an experimental animal results in truth supports a finding of carcinogenesis, the -- the result in a study should be represented or replicated in other

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experiments similarly situated and designed by different laboratories, true?

A. If possible, that would -- would strengthen the data.

Q. Yep. And you and your colleagues at NTP also wrote that to determine the truth about the carcinogenicity about a study -- additional studies of other strains of the same animal species should be done if the same finding has been made in the same strain in a different strain of the same species, right?

MS. WAGSTAFF: Object, I would ask if you're reading from something he wrote that you afford him the pleasure of being able to see what he wrote.

Q. (BY MR. HOLLINGSWORTH) Do you understand my question?

A. I think I understand -- would you repeat it? I'm sorry.

Q. Sure. You and your colleagues at NTP have also suggested that in order to determine the truth of whether a substance under test is carcinogenic from an experimental animal that the same test should show carcinogenicity in other strains of the same animal species like a different strain of mouse, for example?

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MS. WAGSTAFF: Objection.

Q. (BY MR. HOLLINGSWORTH) You've written that, haven't you?

MS. WAGSTAFF: Objection to your colleagues at NTP and the same objection from before.

A. That was written quite awhile ago. In a perfect world, that would be a -- a -- a preferred situation, I guess. If you had unlimited resources and unlimited funds and what have you to repeat it -- to repeat these million-dollar animal bioassay studies, that data would strengthen the observation of a chemical causing cancer in that particular strain of -- of a particular species of animal. But it's not necessary to -- for the interpretation of does the -- does the chemical cause cancer in experimental animals and is it an animal carcinogenic carcinogen.

Q. Well, you have -- you've referred to 12 different studies in your report, I think, five mice and seven rats, true?

A. Uh-huh.

Q. That's an immense amount of data, isn't it, on glyphosate?

A. That's more than you usually see for a particular compound.

Q. There's a --

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A. I'll agree to that.

Q. It's two different species of animals and various strains of rats and mice involved?

A. I think it's two strains of rats and two strains of mice --

Q. Right.

A. -- we have data for.

Q. Right. You and your colleagues at NTP said that results in a carcinogen study in order to determine the truth of the carcinogenicity of the test compound should be replicated in different species like in the mouse and in the rat, true?

MS. WAGSTAFF: Object to form of the question.

A. To be honest with you, I'd prefer to see -- see the publication and let me read through it to see -- to refresh my memory. Like I said, this was published some time ago. I don't recall the exact wording.

Q. (BY MR. HOLLINGSWORTH) Well, doesn't it seem reasonable to you that you and your colleagues said in the same paper that the replication of a result in a mouse study in a different study in the rat would be powerful evidence of whether or not the carcinogen -- the substance is truly a carcinogen in

truth, isn't that what you said in the paper?
MS. WAGSTAFF: Objection, you're asking him about a publication that you clearly have a copy of and you're refusing to give it to him. I've asked you to give it to him now and he requested it. If you're going to keep asking him about it, I would ask that you give him a copy of the publication.
MR. HOLLINGSWORTH: I'm just here to test his expertise and his opinion.
MS. WAGSTAFF: You're testing his memory on something he wrote probably decades ago.
MR. HOLLINGSWORTH: My question went to whether or not it was reasonable to say among scientists that are your peers to determine the truth if a compound was a carcinogen, it would be very valuable to have results that are replicated in different species both in the mouse and the rat?
MS. WAGSTAFF: Hang on. I repeat my request to give him a copy of the publication that you're apparently trying to trip him up on.
A. It -- if you could get results in two species of animals, that strengthens the observation that the chemical causes cancer in experimental animals, but under the current criteria that people use for hazard identification, be it the IARC or the

NTP for the reported carcinogens, it's not necessary to have a positive response in two species.
Q. (BY MR. HOLLINGSWORTH) So the paper I was referring to was published in 1988, you and Huff and Joe Haseman.
A. Haseman and about 10 other people.
Q. Are you saying that the criteria at NTP has changed since 1988?
MS. WAGSTAFF: Object to form.
A. You're referring to a publication, you're not referring to criteria that was used at the time for -- for either IARC or the report on carcinogens, so I mean, it's apples and oranges.
Q. (BY MR. HOLLINGSWORTH) Would your opinion today be different than it was in 1988?
MS. WAGSTAFF: Objection, please let him see the publication if you're asking if his opinion is the same so he can read the publication. That's 19 (sic) years ago.
A. I'd have to read everything that was said in the publication to really give you a good answer to that.
Q. (BY MR. HOLLINGSWORTH) You and your colleagues at NTP also wrote that it would -- it would -- it would strengthen the opinion to determine

whether in truth a substance was carcinogenic if the results of a finding of cancer in a laboratory animal were repeated in a different or in the opposite sex as well in the same study or in different studies, isn't that what you -- isn't that what you guys thought?
MS. WAGSTAFF: Objection, once again.
A. I'd have to read the paper to see if that's what was actually said.
Q. (BY MR. HOLLINGSWORTH) You don't remember stating that?
A. Like I said, this was 1988. I don't remember what we said in the publication. I'd really like to see it so I could refresh my memory.
Q. You said previously that whether animal study results with the same chemical are repeated in animals of a different sex should be considered in an attempt to assess the truth of whether or not the substance is carcinogenic, haven't you?
A. Again, without looking at the paper, I can't recall exactly what the wording that was said in the paper -- what we said. Sorry.
Q. Does that sound wrong to you, what I just said, is that something you wouldn't subscribe to you?
A. Like I said, I really would like to see

the paper, please.
Q. Okay.
A. So I can refresh my memory.
Q. Now, you claim in your report that there is evidence of lymphoma in three studies in mice that is sufficient to support your opinion, right?
A. I believe that's what I said.
Q. Yep.
MS. WAGSTAFF: Is there a question on the table?
MR. HOLLINGSWORTH: Yes. Yeah, that is.
Q. (BY MR. HOLLINGSWORTH) I said you state in your report that there is evidence of lymphoma in three studies in mice that supports your opinion; isn't that right?
A. This is in -- what's the tumor site, please?
Q. Lymphoma --
A. Lymphoma.
Q. -- in mice.
A. I say that glyphosate caused a --
THE REPORTER: I'm sorry.
A. I'm sorry. Glyphosate caused a significant increase in the incidence of malignant lymphoma in male CD-1 mice in two studies and I give

references to the two studies. And in male and female Swiss albino mice in another study.

Q. (BY MR. HOLLINGSWORTH) What page is that, sir?

A. 28.

Q. You cite to no evidence anywhere in your report that glyphosate causes lymphoma in rats, do you?

MS. WAGSTAFF: Object to form.

A. No, I don't believe I did, but if I may, it caused lymphoma in two different studies in CD-1 mice and it also caused lymphoma in male and female Swiss mice, so that's very strong evidence that it caused lymphoma in mice, so --

Q. (BY MR. HOLLINGSWORTH) I'm going to talk to you in detail about the Swiss albino mice study and the other two studies, but my question is whether that evidence of lymphoma that you cite in your case in mice involving mice was replicated in rats -- in the rat studies that you cite involving seven different rat studies?

A. I don't believe -- I'd have to go back and read in more detail. There may have been lymphomas caused, but it may not have been significant increase in lymphomas in the rats, so I have to -- I'd

have to go back and look to say specifically that no lymphomas were caused in the rats.

Q. You don't cite to findings of lymphoma in any of the rat studies that you reviewed, do you?

A. I did not mention it. If I did not mention it, it doesn't mean that they weren't formed. It just means that they weren't significantly increased in that -- in the rats.

Q. So you don't recall finding any significant increases of lymphoma in rats?

A. I -- based on what the -- my summary here, I do not, but I need to go back and look at the studies in a little more detail to say absolutely that no lymphomas were caused. They may -- again, like I said, there may have been some, but it may not have reached the level of significance for me to include it in my writeup.

Q. Well, you agree with me that you don't say anything about lymphomas being found anywhere in any of the 11 rat studies that you reviewed, true?

A. I don't say anything in the summary that I look at right now, no.

Q. Okay. So your report does not say that the findings of malignant lymphoma in mice have been replicated across species that is to include rats?

MS. WAGSTAFF: Object to form.

A. No, I did not say that it -- that -- that lymphomas were found -- were a significant increase in lymphomas were found in rats. I did not state that. That's correct.

Q. You also claim in your report that there is evidence of kidney tumors in male mice in three different studies, right? I believe you already testified to that this morning, sir.

A. To the same three studies?

Q. The same three studies. I'm referring to the same three studies now that you've already talked about. So my question is, whether you claim in your report that there is evidence of kidney tumors in males in three studies, three mouse studies and your answer is yes, right?

MS. WAGSTAFF: You can read your report if you need to.

A. Repeat the question, please.

Q. (BY MR. HOLLINGSWORTH) Sure. You claim in your report that there is evidence of malignant lymphoma in three different studies involving the mouse?

A. Three different studies in mice. Okay. Yes. I thought you were talking about kidney tumors.

I'm sorry.

Q. Yeah.

MS. WAGSTAFF: I think you originally said kidney tumors.

Q. (BY MR. HOLLINGSWORTH) Sorry. I said the wrong thing. My apologies.

A. So we were talking about the lymphomas?

Q. No, I've changed to kidney tumors.

MS. WAGSTAFF: Start the question over.

MR. HOLLINGSWORTH: My apologies.

A. Okay. Repeat the question just so we're clear.

Q. (BY MR. HOLLINGSWORTH) You claim in your report that there is evidence of kidney tumors in three different mouse studies?

A. I don't believe so, no. Oh, I apologize. I apologize.

Q. Yeah.

A. It is three. I apologize.

Q. Yeah. You've got renal tubule lesions that you say were caused by glyphosate in the Monsanto 1983 study and you have renal cell adenomas in males in the Feinchemie Swiss albino mouse study?

A. Right.

Q. And then you have said you have claimed

that there are malignant renal or -- I'm sorry, not malignant, but renal adenomas in the Arysta, that's A-r-y-s-t-a, true?

A. Okay. Yes, I'm sorry.

Q. Okay. You cite to no evidence anywhere in your report involving renal tumors in rats, do you?

MS. WAGSTAFF: Object to form.

A. I know there was one study in rats where they did see some renal tumors. I'd have to go back and find that. I don't know -- again, I don't know if there were -- if it reached the level of statistical significance, but I know there was one study in rats where there was an increase in renal tumors observed, which is a pretty rare finding in rats.

Q. (BY MR. HOLLINGSWORTH) Sir, that's not my question. My question is whether your report cites to a finding anywhere in your report of renal tumors in rats and it doesn't, does it?

A. I need to look through the report in a little more detail to see that because I remember seeing renal tumors in rats -- in one rat study at least.

Q. Well, your -- your report does not indicate that there are renal tumors in rats and that you found and that you rely on as a basis of a

conclusion in your report?

MS. WAGSTAFF: Do you want him to take the time to look through it?

MR. HOLLINGSWORTH: I thought he would know his report better than this.

MS. WAGSTAFF: He knows his report fine, but you're asking him minutia and you guys disagree and he said let me look at something.

MR. HOLLINGSWORTH: Well, it's not minutia, it's serious evidence.

MS. WAGSTAFF: It's very serious evidence, I agree with that, and he disagreed with something you said and he said, if I can look through my report and I can tell you better, and if you want him to take the time to do that, he will. Do you want him to take the time to do that?

Q. (BY MR. HOLLINGSWORTH) Sir, as you sit here today, you don't recall citing any evidence of renal tumors in the rat out of the seven studies that you looked at, do you?

MS. WAGSTAFF: Object to form. He just said he recalled that there was one.

A. I -- I recall that in one study there were renal tumors seen in rats. Again, I don't recall if it reached the level of statistical significance,

and in skimming through this, I don't see where I refer to that, so in my report, I don't know that I referred to it.

Q. (BY MR. HOLLINGSWORTH) Okay. Thank you. My question was whether you cited to that in your report, and your answer is no, right?

MS. WAGSTAFF: Objection, misstates his testimony.

A. After -- with just a quick skimming through it, I can't -- I don't see it right now.

Q. (BY MR. HOLLINGSWORTH) Okay. Based on that review of your report, in which we found no mention of a kidney tumor in rats --

MS. WAGSTAFF: Objection, you have not given him the opportunity to look through his report in detail. He says that he remembers citing to it. I asked if you want him to look through and you said no and now you've making a record that we scoured the report to look for it. If you want him to look for it, you can.

Q. (BY MR. HOLLINGSWORTH) Can you find any reference in your report, sir, to the existence of renal tumors in the rat that you've relied on in your report?

A. Okay. Give me a minute to read through

this and I'll let you know. Okay. I don't see any reference to a kidney tumor in the rats in my report. I do remember in reading -- in looking -- in reading the study, the actual studies that I did see an IARC study that reported increases in kidney tumors, but it wasn't statistically significant, so that's probably why I didn't include it in the report. But that's -- also I would state that it is not that unusual when you do a study in mice and rats that you see a tumor at one site in one species and you don't see the corresponding tumor site in the other species.

I think if you go through and look at the incidences of tumors in, take for example, the NCP bioassay program and the technical report series, I think it's usually the case. I won't say that it's -- that it's always the case, but I think it's usually the case that if you see a tumor in one species, you don't see the same tumor in the same corresponding tumors in the other species all the time, so the fact that you see kidney tumors in mice and you didn't see it in rats is -- is not all that surprising.

Q. Sir, you didn't -- your answer is that you didn't cite to any evidence of kidney tumors in rats in your report?

MS. WAGSTAFF: Object to form.

A. In my report, I did not.
Q. (BY MR. HOLLINGSWORTH) So you haven't cited to any evidence that the findings of kidney tumors in three -- three mouse studies that you referred to were replicated in the rat?
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Did you?
A. Again, I will state that that is not that unusual that you see corresponding tumor sites in two different species when you do a study. A lot of times you get certain types of tumors in the mouse and you'll get a completely different set of tumors in the rats in the study conducted at the same laboratory at the same time with the same chemical, so that's not a surprising finding to me, but that's correct.
Q. (BY MR. HOLLINGSWORTH) So the answer is that there's no evidence in your report that the findings that you refer to involving kidney tumors in male mice were replicated in the rat species, true?
MS. WAGSTAFF: Objection, asked and answered.
A. That is correct.
Q. (BY MR. HOLLINGSWORTH) Thank you.
A. But the incidence of kidney tumors was replicated in two different strains of mice.

Q. I understand that.
A. CD-1 mice and the Swiss mouse.
Q. But that wasn't my question. My question went to whether or not it was replicated in the rat, do you understand that?
A. Right. But that's not a surprising finding.
Q. Okay. You cite no evidence in your report that the kidney tumors that you refer to in male mice were replicated in female mice, do you?
A. I say that there were kidney tumors observed in the female Swiss mice, I believe.
Q. Sir, would you look at page 28 of your report which says "Summary for Experimental Animal Data."
A. Okay.
Q. Now, this is an accurate summary of your report, right, on experimental animals?
MS. WAGSTAFF: You can read it if you need to. Are you talking about all of page 29 as well?
MR. HOLLINGSWORTH: Yes.
MS. WAGSTAFF: Okay.
A. I'm sorry. I misspoke again. I was thinking of the lymphomas. It's the -- yeah, it's the

lymphomas. I'm sorry.
Q. (BY MR. HOLLINGSWORTH) My question is whether this summary at 28 and 29 is an accurate summary?
A. Is an accurate summary?
Q. Of your opinion.
A. To the best of my knowledge, it is.
Q. Did you write this?
A. Yes.
Q. Okay. Now, you say that there is evidence of kidney tumors in female mice and that's where from the Swiss albino mouse study, because I don't find anything in your study that says that -- I mean in your report that says that.
A. Like I said, I was mistaking -- I was confusing that with the lymphomas.
Q. That's understandable. But there -- you cite to no evidence in your study, sir, that says that there are kidney tumors in the female mice studies that you reviewed, true?
A. I don't think we found any, no.
Q. So, therefore, the evidence that you rely on involving kidney tumors in male mice was not replicated across sexes, was it?
MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) You were wrong when you indicated that earlier in your testimony?
A. When I stated --
MS. WAGSTAFF: He wasn't wrong. He already admitted that he was confusing it with lymphomas.
A. I was confusing it with the lymphoma data. Again, it's a situation where there -- I believe, there were kidney tumors observed in females, but it didn't reach a significant level, so, therefore, I didn't include it in the report.
Q. (BY MR. HOLLINGSWORTH) Okay. So you didn't state in your report that the evidence of kidney tumors in mice had been replicated in the female mice specifically, true?
A. I did not say that, that's correct.
Q. Now, you claim that there is evidence of hemangiosarcoma in males in two studies in mice, correct?
A. I believe that's right.
Q. And you cite to no evidence in your report of any hemangiosarcoma in rats, do you?
A. Correct.
Q. And, therefore, you cite no evidence that hemangiosarcomas have been replicated across

species, do you?
MS. WAGSTAFF: Object to form.
A. Again, that's what I said, but as I stated before, I wouldn't consider that all that unusual. You don't always see the same tumor in one animal species that you observe in a different animal species, even in studies conducted under -- at the same time with the same chemical.
Q. (BY MR. HOLLINGSWORTH) I understand that, but in this specific report, you don't refer to -- you didn't refer the Court to any evidence that the hemangiosarcomas that you claim existed in two male mouse studies have been replicated in rats, true?
MS. WAGSTAFF: Object to form. Asked and answered.
A. Like I said, I -- I don't -- I did not report any hemangiosarcomas in rats in my report.
Q. (BY MR. HOLLINGSWORTH) Okay. You cite no evidence of hemangiosarcomas in female mice either, do you?
A. That's correct, I corrected my report to say -- initially the report submitted said hemangiosarcomas, but I corrected that. It was hemangiomas.
Q. So you haven't cited the Court to any

evidence that hemangiosarcomas in male mice have been replicated across sexes in the same species, true?
A. That is correct.
Q. You claim that there is evidence of pancreatic cell tumors in males in two different rat studies, true?
A. Pancreatic?
Q. The Monsanto 1990 rat, do you see that?
MS. WAGSTAFF: What page are you looking at?
MR. HOLLINGSWORTH: I've memorized it.
MS. WAGSTAFF: I wouldn't be surprised.
A. Are we talking about pancreatic tumors?
Q. (BY MR. HOLLINGSWORTH) I'm talking about pancreatic cell tumors. They're referred to in your report sometimes as pancreatic islet cell adenomas.
A. Okay.
Q. And you referred to two studies. The 1990 Sprague-Dawley study and the 1981 Sprague-Dawley study, correct?
A. To be honest, I thought I only referred to one study where there were pancreatic islet tumors.
MS. WAGSTAFF: If you have a specific page or a reference for him, that may speed it up.

Q. (BY MR. HOLLINGSWORTH) Sir, are you looking at your report regarding the Monsanto 1990 Sprague-Dawley rat study? You refer to pancreatic islet cell adenomas in there.
A. For one study?
Q. The 1990 study and then there's the 1981 study. Also in Sprague-Dawley rats. That's one of the seven rat studies you referred to also and you mentioned pancreatic islet cell evidence in that study as well, true?
A. Which page is that on? Oh, you don't have that?
Q. I don't have a page.
A. I didn't refer to the studies by their date. I referred to them basically by their Greim study number.
Q. Okay. The 1981 rat study is referred to by you at page 24, I think.
A. Okay.
Q. Isn't that the 1981 study?
MS. WAGSTAFF: Are you talking about this last paragraph on page 24?
MR. HOLLINGSWORTH: Yeah, and it proceeds over to page 25 and it mentions that he believed there was a -- the author of the report

Dr. Jameson believes there was a significant increase in the incidence of pancreatic islet cell adenoma from this study.
A. Okay.
Q. (BY MR. HOLLINGSWORTH) Okay. And then if you look at the study involving the 1990 Sprague-Dawley rat study, which --
A. Okay.
Q. -- that's the study you report as by the author called Dr. Stout?
A. Stout, uh-huh.
Q. And you refer to pancreatic islet cell adenomas there as well, right?
A. Correct.
Q. Okay. So there's two --
A. Two studies.
Q. -- two studies involving what you claim are pancreatic cell tumors in rats?
A. Uh-huh.
Q. Right?
A. Correct.
Q. Those two studies, one in 1981 and one in 1990, both in the Sprague-Dawley rat, true?
A. True.
Q. Those pancreatic cell tumors weren't

replicated in any other rat studies, were they?
A. I don't believe so, no.
Q. And they weren't replicated in any mouse studies?
A. I believe that's correct.
Q. So there's no evidence of pancreatic cell tumors in mice that you have reported in your report, true?
A. There -- there were no statistically significant increases in pancreatic islet cell tumors in mice, so, therefore, I didn't include it in my report.
Q. And, therefore, have you -- you haven't cited in your report any evidence that these pancreatic cell tumors were replicated across species, true?
MS. WAGSTAFF: Object to form.
A. That's correct, but, again, I'll say as I said before, that's not a surprising finding because you don't always see the same tumor sites in animals tested at the same time by the same -- in the same laboratory under the same conditions.
Q. (BY MR. HOLLINGSWORTH) There's -- there's no evidence anywhere in your report that you've cited that the pancreatic tumors that were seen

in the male rat studies were replicated across sexes into female rats or female mice, are there?
A. I did not report any -- I'm sorry. There were probably no -- there were no statistically significant increased incidences in those tumors in the female rats or mice reported, so I did not include that in my report.
Q. Sir, you claim that there is evidence of hepatocellular adenomas and you claim that those occurred in statistically significant numbers in male rats, two different studies, true?
A. Yes, in two studies. Male rats.
Q. Did you cite us to any published literature that says hepatocellular carcinomas in male rats are predictive of non-Hodgkin's lymphoma in humans?
A. Again, the studies were conducted to see if glyphosate caused cancer in experimental animals.
Q. Okay.
A. The studies showed that there were hepatocellular carcinomas formed in the studies, in this case, in the rats, and significantly increased and so, therefore, it was positive in the male rats as an animal carcinogen. Being an animal carcinogen is -- is -- indicates that it is -- could be -- it

could be a human carcinogen.
I'm not aware of any studies that have been conducted that were investigating any association between the formation of hepatocellular adenomas in rats -- in male rats and non-Hodgkin's lymphoma. I don't know if anybody has done any research in that area or published in that particular.
Q. All right. Thank you.
MS. WAGSTAFF: We've been going a little over an hour. Whenever you find a good stopping point, if we can take a break.
MR. HOLLINGSWORTH: Any time is fine with me.
MS. WAGSTAFF: It's your depo.
MR. HOLLINGSWORTH: All right. Let me ask a couple more questions about these hepatocellular adenomas in rats. I won't be long.
Q. (BY MR. HOLLINGSWORTH) There's no evidence of hepatocellular carcinoma in mice that you have reported in your report to the -- to the Court in this case, is there, Dr. Jameson?
A. No. I didn't report any, which would indicate to me that there were no statistically significant increases in those tumors reported in the studies, so I did not include it in my report. It's

not to say there weren't some I've seen, but they were probably not statistically significant.
Q. So there's no evidence in your report that these results you have cited to involving male rats have been replicated across species?
MS. WAGSTAFF: Object to form.
A. That -- that is correct. But, again, I would state that's not unusual to see a tumor in one species and not in another -- the same tumor in another species in the studies done with the same chemical at the same laboratory at the same time.
Q. (BY MR. HOLLINGSWORTH) You don't cite to any study or evidence in your report that states that the hepatocellular adenomotis effect that you say exists in male rats has been replicated across sexes in any study anywhere, do you?
A. None of the data that I reviewed indicated that, no.
MR. HOLLINGSWORTH: All right. We can stop now. Thank you, sir.
THE VIDEOGRAPHER: Going off the record. The time is 10:17 a.m.
(Recess taken, 10:17 a.m. to 10:34 a.m.)
THE VIDEOGRAPHER: We are back on the record. The time is 10:34 a.m.

Q. (BY MR. HOLLINGSWORTH) Sir, you claim in your report that there is evidence of lung adenocarcinoma in male mice in one study, true?
A. Yes.
Q. And you rely on that in support of your -- your opinion that glyphosate can cause non-Hodgkin's lymphoma, right?
A. I use that to -- in my opinion that glyphosate causes cancer in laboratory animals because it causes significant increase in that particular tumor there.
Q. You -- in the last sentence of your report, you state that it's your opinion to a reasonable degree of scientific certainty that glyphosate can cause non-Hodgkin's lymphoma in humans, right?
A. That's what I state, yes.
Q. And does this study -- this single mouse study finding adenocarcinoma or adenomas in male mice is supportive of that opinion that last sentence in your report?
A. That particular opinion that I made in my report is based on an evaluation of all the available data on glyphosate and glyphosate formulations that -- that the data -- all the data

taken together state in -- it's my opinion that all the data indicates that glyphosate and glyphosate formulations cause non-Hodgkin's lymphoma.
Q. Okay. But you understand my question here is -- my question here goes to the evidence that you cite in your report of adenocarcinoma in male mice in a single study?
A. That's one piece of the data. One piece of the information that I used in my overall evaluation.
Q. Did you cite to any evidence or investigation that's been published anywhere on the planet that discusses whether lung adenocarcinoma in male mice is predictive of human cancer involving non-Hodgkin's lymphoma?
A. Well, the study that I evaluated was conducted to see if glyphosate would cause cancer in experimental animals, and in this particular study, it caused lung adenocarcinomas, and so, therefore, since it caused a significant increase of lung adenocarcinomas, in this particular study, it's an animal carcinogen, and being an animal carcinogen, it could -- it indicates that it potentially could be a human carcinogen, so -- but I am not aware of anybody that has designed or conducted a study to investigate

the association of lung adenocarcinoma with non-Hodgkin's lymphoma or published any -- any papers on that.
Q. Sir, thank you. You cite to no evidence in your report of lung adenocarcinoma in any other rat or mouse study in your report and there are 11 other rodent studies that you rely on in your report.
A. I don't cite to any significant increases in lung adenocarcinomas in any of the studies. If I think -- in reviewing all the data, there were several studies where lung tumors were observed, but they weren't significant enough to include in my particular report.
Q. In your report, you only included findings that were statistically significant in the 12 rodent studies that you looked at, true?
A. The -- the only ones that I included in my report were the -- were the -- were the tumor sites where there was an increase in the incidence over the -- over the controls, so, yes, it was -- it was those where you saw a significant increase over the controls.
Q. You claim that there is evidence of testicular interstitial cell tumor in -- of course, that's in male rats in one study, right?

A. Correct.
Q. And did you consider whether the existence of interstitial cell tumors in the testes of rats has ever been studied to determine whether it is predictive of non-Hodgkin's lymphoma in humans?
A. Well, the -- the -- for this particular study, glyphosate was tested to see if it caused cancer in the male rats. It caused these interstitial testicular cell tumors in the male rats. It was increased significantly increased and therefore, glyphosate caused cancer in laboratory -- in -- in these male rats, so, therefore, it's an animal carcinogen. Being an animal carcinogen is -- it's a potential human carcinogen.
I'm not aware that anybody has designed or conducted a study to investigate any association between male testicular tumors in rats and non-Hodgkin's lymphoma in humans or published any -- any papers on that.
Q. You cite to no evidence that the testicular interstitial cell tumors that you refer to in the single rat study was replicated in any of the five mice studies, do you?
MS. WAGSTAFF: Object to form.
A. That's correct. There -- there were not

testicular tumors reported in any of the mice studies, but, again, I'll point out that that's not an unusual finding to find one tumor site in one strain of animals or one species and not find the same tumor site in another species, studies conducted with the same chemical at the same laboratory at the same time.

Q. (BY MR. HOLLINGSWORTH) But you cite to no evidence that that interstitial testicular cell tumor in single rat study was replicated in any of the other four rat studies, do you?

A. No. It wasn't observed in any of the other rat studies.

Q. And it wasn't replicated in any of the five mouse studies in male mice?

MS. WAGSTAFF: Object, asked and answered.

Q. (BY MR. HOLLINGSWORTH) True?

A. It wasn't seen in mice, no.

Q. (BY MR. HOLLINGSWORTH) You claim that there's evidence of thyroid follicular cell tumors in female rats, true?

A. True.

Q. And that was in one study. Do you cite any evidence that the finding of follicular cell tumors in female rats is predictive of non-Hodgkin's

lymphoma in humans?

A. Well, in this particular study, glyphosate was -- was exposed -- tested in the rats to see if it would cause cancer. The glyphosate caused these follicular cell tumors in the female rats to a significant -- there was a significant effect, therefore, glyphosate caused cancer, caused these tumors in the female rats. It, therefore, is an animal carcinogen and a potential -- therefore, and also, therefore, a human -- potential human carcinogen.

And I'm not aware of anybody who has designed or conducted a study to investigate any association between these follicular cell tumors in female rats and non-Hodgkin's lymphoma or published any studies for that or published any papers to that effect.

Q. Sir, you haven't cited anything in your report of the other 11 rodent studies that you refer to in your report in which female follicular cell tumors were replicated, true?

A. I did not see any -- in any of the other studies that there was a significant increase in follicular cell tumors in the female animals --

Q. So there's --

A. -- so I didn't include it in my report.

Q. So there's no replication across species that you've cited in your report?

MS. WAGSTAFF: Object to form. He's already indicated that a tumor site does not have to be the same to equal replication.

A. True. And just -- just to point out, I mean, when you're talking about replication, you don't necessarily have to have replication between sexes or between species. If you have replication in a number of the tumor sites that we've discussed earlier, the -- the tumor was -- the tumor was replicated in different studies. It may have been in the same species, but they were in different studies conducted at different times, at different laboratories, so that is a replication of an experiment and gives extremely strong evidence that this particular compound causes that tumor in that -- in experimental animals, and that's something we have done in my 30 plus years' experience as a toxicologist has always been if you can replicate the study in the same sex -- in the same sex or same species, if you replicate it at a different laboratory, it's very strong evidence that it is an animal carcinogen at that tumor site in that sex and species of animal.

Q. (BY MR. HOLLINGSWORTH) Sir, the follicular cell tumors in female rats that you were referring to weren't replicated in any study you've reported anywhere in your report to this case, true?

MS. WAGSTAFF: Object to form.

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

Q. (BY MR. HOLLINGSWORTH) I said the female follicular cell tumors that you're referring to in your report and in your prior recent answers involving follicular cell tumors in female rats aren't reported anywhere in your report to have been seen in any study involving rats or mice of either sex anywhere else in your report, true?

A. In any other study?

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) Yes.

A. In the other studies I reviewed, that particular tumor was not increased significantly over controls and so while they may have been -- those tumors may have been induced in those studies, if it wasn't significantly increased over the control incidence, I didn't include it in any report.

Q. You've previously said that historical control data should be considered in an attempt to assess the truth whether or not there is an actual

carcinogenic effect in a mouse or a rat species, true?
A. Did I say that in my report? I don't remember.
Q. No, I said that you have -- you have published that, you've said that before that historical control data should be considered in an attempt to assess the truth whether or not an agent is actually carcinogenic?
MS. WAGSTAFF: I would request that you allow Dr. Jameson to review the publication in total before asking him questions about piecemeal.
A. I was -- yeah, where -- I was going to --
Q. (BY MR. HOLLINGSWORTH) Do you recall stating that?
A. Do I recall stating that?
Q. Yes. That historical control data should be considered in an attempt to assess the truth about the frequency of a tumor type among control animals in a particular strain of animal?
MS. WAGSTAFF: Same objection.
A. It may have been in a publication sometime ago. I just don't remember.
Q. (BY MR. HOLLINGSWORTH) Do you disagree with that proposition as you sit here today?

A. Historical control -- consideration of historical controls is an important consideration in any toxicology or bioassay study, but the most appropriate controls to use in any study is the concurrent controls that you have for that particular study. Historical controls can help you evaluate the data, but they are not as important as the concurrent controls.
Q. You've referred to historical controls in your report and you've relied on historical controls in the report that you've given to the Court in this case, haven't you?
A. That's correct. I'm not saying -- again, like I said, the historical controls are important and they aid in the evaluation of the data.
Q. You've also said before, haven't you, Dr. Jameson, that the presence or absence of preneoplastic lesions is a key factor when determining what conclusion can be drawn from a long-term animal bioassay?
MS. WAGSTAFF: I would repeat my same request, if you are quoting from a publication that Dr. Jameson be afforded the opportunity to read the entire publication.
A. I -- it may appear in some of my earlier

publications. I don't remember how it -- how I worded it or what I said, but. . .
Q. (BY MR. HOLLINGSWORTH) So do you disagree today that the presence or absence of preneoplastic lesions involving an agent under test is a key factor in determining whether or not there's a carcinogenic effect?
A. It's a factor. I mean, the fact that you see preneoplastic lesions are, again, a helpful indication that you're going to see a carcinogenic effect, but it is not absolutely required that you see preneoplastic lesions to say that something is or is not a carcinogen.
There are instances in the literature where tumors are seen in the absence of preneoplastic lesions, so preneoplastic lesions are an important part of any study if you see them, but if you don't see them, you may say, wow, that's surprising, I didn't see preneoplastic lesions, but that's no reason to discount the finding of tumors being formed because you didn't see any preneoplastic lesions.
Q. Let me ask you specifically about the 1983 mouse study that you refer to. Do you have that in mind?
A. Okay.

Q. Did you read that study by Knezevich and Hogan? Knezevich is K-n-e-z-e-v-i-c-h.
A. Did I read the study? I looked at the data from that study, yes.
Q. But you didn't read the actual study?
A. The study report that was submitted by the lab? For that particular one, I don't know if I had access to the entire report or not, but I did have access to a lot of it, a lot of the actual report from the laboratory.
Q. But you don't think you read the actual report?
MS. WAGSTAFF: Objection.
A. I saw excerpts of the actual report, yes.
Q. (BY MR. HOLLINGSWORTH) Did plaintiffs' counsel show you that report?
A. It was provided to me by plaintiffs' counsel, yes.
Q. The entire report?
A. Again, I'd have to go back and look in my files and see if I have the entire report, but I had a very large portion of it.
Q. Did you read the author's statement that, quote, there were no suspected test substance

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associated trends in the incidence of bronchioalveolar, hepatocellular neoplasms and tumors of the lymphoreticular symptoms or any of the other spontaneous occurring neoplasms, unquote, did you read that statement in their report?

A. I -- I think I remember that statement. Yeah. This is the -- excuse me. This is the mouse study, the CD-1 mouse study.

Q. Yes. 1983?

A. '83.

Q. Knezevich and Hogan were the investigators --

A. Investigators.

Q. -- on that report, right?

A. Uh-huh.

Q. They're doctors of veterinary medicine, aren't they?

A. I'm sorry, I don't know their background.

Q. Okay.

MS. WAGSTAFF: I'd request that you allow him to look at the report if you're questioning if he saw the entire thing and you're quoting from it.

MR. HOLLINGSWORTH: Well, I'm just asking if he recalls because I'm going to investigate

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the extent of his knowledge about this report.

A. Okay.

Q. (BY MR. HOLLINGSWORTH) Do you recall that the conclusion of the report was regarding the renal tubule lesions that were observed in that report, that, quote, the distribution of these benign tumors was considered spurious and unrelated to treatment, unquote?

MS. WAGSTAFF: And hang on a second. This is not supposed to be a memory test. If you would like to know his knowledge of it, why don't you give him a copy of the report and let him follow along with you as you read from it.

Q. (BY MR. HOLLINGSWORTH) I'd just like to know, sir, whether you remember whether that was the conclusion of the people who did the original report and conducted the original study.

MS. WAGSTAFF: So why don't you let him see the report.

MR. HOLLINGSWORTH: You've given him the report, he says I'm asking for his knowledge about the report and I'm entitled to do that.

A. I remember that was the bottom -- that that was their conclusion, yes.

Q. (BY MR. HOLLINGSWORTH) Okay. Thank you.

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Would it -- would it be fair in your report to this Court, this MDL Court, for you to have included the original reports of the original authors of that study so that the judge could see them?

A. For me to include them in my report?

Q. Yeah. Wouldn't it have been fair for you to include the conclusions of the original authors of the study in the report that you made to the Court in this case?

MS. WAGSTAFF: Objection, that calls for a legal conclusion. How is he supposed to know what's fair to the MDL judge?

A. Plus the -- well, you know, I don't know. I don't know if -- I mean, I'm sure if the judge would want to see that, we could make that available to him. I would point out that this study is included in the Greim publication, and all the relevant data supposedly from this study is included in the Greim paper and it -- the EPA refers to the Greim paper when they made their recent evaluation, so -- and I reference the Greim paper in this report.

Q. (BY MR. HOLLINGSWORTH) Sir, I'm not asking about the Greim paper. I'll talk about Greim later.

My question is whether it would be fair

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in your opinion, as a scientist, to have included the conclusions of the original investigators of this 1983 study on CD-1 mice in your report to the judge of the Court in this multidistrict litigation?

MS. WAGSTAFF: Objection, asked and answered and this is becoming argumentative, and he already has stated if the judge would like this report, then he can give it to him and I'm sure your experts have included it in their report.

Q. (BY MR. HOLLINGSWORTH) No, my question is whether it would be fair as a scientist in your opinion to have included the conclusions of the original authors.

MS. WAGSTAFF: Objection, asked and answered. That's a legal conclusion.

A. I was asked to provide my opinion of the data as it relates to glyphosate and glyphosate formulations and non-Hodgkin's lymphoma. And as part of evaluate -- as a part of doing my evaluation and -- and reviewing all the available information pertaining to that, I looked at the study and I summarize it in my report and I put the -- what I felt were the appropriate references in my report for this particular study, so --

Q. (BY MR. HOLLINGSWORTH) But you did not

in your report include these two conclusions of the original authors of the study that you were reporting about, did you?

A. Again, I was asked to give my opinion, not somebody else's opinion, so I looked at the data, formulated my opinion and put it in my report.

Q. Well, your opinion is different than the original investigators, isn't it?

MS. WAGSTAFF: Objection argumentative.

Q. (BY MR. HOLLINGSWORTH) Isn't it?

A. Yes.

Q. But you didn't tell the Court what the original authors had concluded after reviewing the data that they reviewed, did you?

A. I was not asked to put everybody's opinion in my report. I was asked to review the data and give my opinion and that's what I did.

Q. Did you review in connection with your report any of the morphologic slides, any morphology at all?

A. I -- first of all, I'm not a pathologist. I don't read slides. So I -- I couldn't. I would not be able to look at the slides and evaluate them. That's not my background, so it wouldn't -- it would not be appropriate for me to do

that.

Q. Dr. Knezevich and Hogan were veterinary medical doctors who looked at the actual slides from this study themselves, didn't they?

MS. WAGSTAFF: Objection, already testified he didn't know their background.

A. I -- I assume that's what they did, but I don't know.

Q. (BY MR. HOLLINGSWORTH) How long does it take a veterinary pathologist to review slides from a long-term bioassay?

MS. WAGSTAFF: Objection, speculation.

A. I can only -- I can only speak to my past experience from the NTP bioassay where -- you know, it would depend on the design of the study. It depends on how many -- how many dose groups you have, how many animals per dose group, how many interim sacrifices you have, if it's in both rats and mice, I mean, you could -- you could be looking at upwards of 10,000 or more slides. So in my past experience, it's taken them six to nine months to evaluate a rodent bioassay, so it's a very involved process.

Q. (BY MR. HOLLINGSWORTH) In the -- in the -- with respect to the 1983 mouse study, did you look at their individual animal reviews of any -- any

of the slides or any single animal from the 1983 mouse study?

A. Did I look at any of the slides?

Q. Did you look at any slides or reports on the review of slides?

A. I looked at the tumor tables and the tables in the report of individual animals evaluation. I looked at all that data, yes.

Q. Where did you find the individual animal evaluations?

A. They have tables -- in the report they have tumor tables or individual animal tumor tables where they list the animals by their animal number and it has a -- in tabular form, it gives you the organ site and what they found.

Q. In this case, did you do that from the materials that plaintiffs' counsel gave you?

A. From the report of the -- of the -- of the Knezevich report.

Q. Okay. You know that the 1983 report was submitted to the EPA, right?

A. That's correct.

Q. And you talked in your report about some of the regulatory history of that 1983 mouse study, true?

A. True, where the EPA did their initial evaluation and came up with a category C as a carcinogen for glyphosate initially.

Q. Initially?

A. Yes.

Q. Did they change that -- that regulatory finding later?

A. Over the years -- over the years, they appeared to have changed it.

Q. "They" meaning EPA has changed it?

A. EPA. Sorry.

Q. This was a 24-month typical long-term chronic bioassay of mice that we're referring to, right?

A. Yes.

Q. And your report -- in your report, you say that the renal tubule was found in among the four treatment groups in the -- in the -- in the order as follows zero, zero, zero, one, three, right?

A. Okay. That was -- that was the initial evaluation --

Q. Yes.

A. -- from the lab, yes.

Q. Yes. And then -- and you said that the finding of renal tubules adenomas or carcinomas is a

rare event; is that right?
A. Yes, for the CD-1 mouse.
Q. And for the CD-1 mouse, you rely on the publication Chandra and Firth for your conclusion that it is a rare lesion?
MS. WAGSTAFF: Object to form.
A. That's a reference I used, yes.
Q. (BY MR. HOLLINGSWORTH) In your report?
A. In the report.
Q. That's the same reference that IARC used in the monograph 112, true?
A. I believe it is.
Q. Did you read in the materials that you reviewed that the Biodynamic's lab itself had three incidents of renal tubule adenomas or adenocarcinomas in control animals prior to this study?
A. I remember seeing that they did have a historical incidence in their lab, but I don't remember to be honest the specific numbers or, you know, how many studies that included.
Q. Did you read also that the Hazleton laboratory, which is a big laboratory in the United States -- you're familiar with that, right?
A. Correct.
Q. They had an incidence of 7.1 percent in

control animals involving renal tubule lesions at the time, true?
MS. WAGSTAFF: Object to form, foundation.
A. I think I remember seeing something to that effect in the report, yes.
Q. (BY MR. HOLLINGSWORTH) And the -- you also saw a reference to IRDC, which was also a big contract laboratory in the 1970's and '80's and '90's, I think that stands for International Research --
A. And Development --
Q. -- Development Corporation, you're familiar with that group?
A. Yes.
Q. They also had a much higher incidence of renal tubule adenomas or carcinomas in control animals that Chandra and Firth reported; isn't that right?
MS. WAGSTAFF: Object to form of the phraseology of "much higher."
A. Well, they did have a higher incidence, but to be honest, I wouldn't put a whole lot of faith in any of the data that came out of IRDC because of their history and the litigations brought against them and what have you. I -- in my experience with IRDC, they're a very unreliable lab, so I just can't take

any of that data with any confidence. I'm sorry.
Q. (BY MR. HOLLINGSWORTH) Are you saying that Biodynamics and Hazleton are not reliable?
MS. WAGSTAFF: Objection, misstates testimony.
A. I don't have -- I don't have experience with them. I do have some past experience with IRDC, so that's where my opinion is going from.
Q. (BY MR. HOLLINGSWORTH) Do you have experience with the data that Chandra and Firth relied on, personal experience?
A. I don't have any personal experience but that's in a peer-reviewed publication, so I -- I put a lot of confidence in that since it's --
Q. Okay. There was no consistent finding for renal tubule adenomas or carcinomas in the female mice at all, was there?
MS. WAGSTAFF: Object to form.
A. I think there was -- I think they might have found one tumor in the female mice, but I'd have to go back and look at the report to confirm that.
Q. (BY MR. HOLLINGSWORTH) Well, you don't have to do that. The incidence in female mice was actually, zero, zero, zero, wasn't it?
A. Again, I'd have to go back and look at

the report. Like I said, I don't recall -- I don't remember.
Q. Did you rely on what plaintiffs' counsel had given you about this report or the Greim study and the Greim tables about this 1983 mouse study?
A. I used both.
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Is Greim reliable?
A. From the standpoint that it is -- comes from a peer-reviewed source, I would say it is fairly reliable. Although, in my review of the information from the Greim report, I was able to find additional tumor incidences that were not emphasized in his report that I included in mine. But coming from a peer-reviewed source, you have to accept that it is fairly reliable.
Q. Sir, you've cited Greim in your report over 10 times, haven't you?
A. Yeah, I use that as a method of identifying the studies. I -- I use that as -- as a manner of convenience more than anything else to keep straight which studies I was looking at.
Q. So you cited Greim, but you don't think it's -- you don't think it's necessarily reliable; is

that right?
A. I didn't say that. I said it comes from a peer-reviewed source, so it should be considered a reliable source. The data should be in there -- at least should be accurate.
Q. So you haven't knowingly cited an unreliable source in your report to the judge in this case, right?
MS. WAGSTAFF: Objection, argumentative.
A. I hope not. Not that I'm aware of.
Q. (BY MR. HOLLINGSWORTH) Well, I just understood you to say that you had reservations about Greim, but then I counted up about 11 references to Greim from your report just sitting here and I was wondering why you were citing --
A. I'm sorry.
MS. WAGSTAFF: Objection, misstates the testimony.
A. I don't remember saying that.
Q. (BY MR. HOLLINGSWORTH) Okay. Now, the renal tubule adenomas in this case were -- after this report was completed, were the subject of some controversy, weren't they?
A. Correct.
Q. And Monsanto sent all the male kidney

slides off to a guy by the name of Dr. Marvin Kuschner, right?
A. That's my understanding.
Q. And that was in around 1983 or '84, true?
A. The time frame sounds about right.
Q. Okay. And you know who Marvin Kuschner was, right?
A. No. Sorry.
Q. He was preeminent in the field of veterinary pathology and experimental pathology testing in the United States. You didn't know that?
A. No, sir.
Q. Okay. All right. You know he was at Stoneybrook?
A. I didn't know where he was from. Sorry.
Q. Okay. And Dr. Kuschner, when he went through all of these mouse kidney slides, including the controls, the low dose, the mid dose and the high dose, found a renal tubule adenoma in a control animal that hadn't been reported before; isn't that right?
MS. WAGSTAFF: Objection, misstates the evidence.
A. That's what the information indicated that I got, yes.

Q. (BY MR. HOLLINGSWORTH) Yeah. And he also did a statistical analysis on the data and he concluded in his report at the time that there was no statistically significant increase in renal tubule adenomas from the 1983 mouse study, right?
A. The report that I saw indicated that, yes.
Q. Yes. And -- sorry. And, yes -- and then the EPA wanted to have six additional sections cut from each -- I'm sorry. Let me start over. Sorry about that, Tracy.
The EPA wanted to have three additional sections cut from each kidney of each male mouse in the entire study, and that was carried out at some point after Kuschner did his review, true?
A. Was it additional step sections of every kidney from every dose level?
Q. It was from every dose level -- it was -- it was three sections from each kidney of each male mouse for each dose level. And the control.
A. Okay. I --
Q. You refer to some of this history in your report, don't you?
A. Uh-huh.
Q. Okay. And those were reviewed by

pathologists and no further -- including the original pathologist, Dr. Knezevich or whatever the pronunciation is and his colleague, and they found no lesions whatsoever out of the additional study slides from that?
A. The report that came back indicated they found no additional tumors, correct.
Q. And to come up with three additional sections of each kidney in each male mouse involving 60 animals and four different groups comes out to about 1,500 additional slides, right?
A. Do the math, yes.
Q. 1,500 additional sections on those kidneys, and they found no cancer, no adenomas, no lesion of any -- of any kind that they reported, true?
A. That's what the report says.
Q. Yes. And -- and do you know who Dr. Klaus Stemmer was?
A. No, sorry.
Q. You never heard of him?
A. Klaus.
Q. Klaus Stemmer, S-t-e-m-m-e-r.
A. (Deponent shook head from side to side.)
Q. He was the head of medical pathology at the University of Cincinnati Medical School and you

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know from reading what you've read, I think, that he reviewed these slides in the control animals and in the high dose animals, and he said -- and also -- also the other two treatment groups, low and mid dose, and he said that he agreed with Dr. Kuschner that the lesions that he saw, if you took them in the order of treatment were one in the control, zero in the low dose, one in the mid dose and three in the high dose and that that was not statistically significant either in his opinion?

MS. WAGSTAFF: Objection to counsel testifying. There's no question on the table and you're just reading into the record your version of events.

Q. (BY MR. HOLLINGSWORTH) True?

A. I don't recall reading a report from --

Q. Stemmer, Klaus Stemmer.

A. I don't remember.

Q. Do you recall reading a report from Dr. Robert Squire, Bob Squire?

A. Yeah, I did see something from Dr. Squire.

Q. You probably knew Bob Squire?

A. Yes, I do.

Q. He was a famous guy in Washington,

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wasn't he?

A. Famous, infamous, yes.

Q. He was the head of the NCI carcinogenesis program?

A. That's correct.

Q. For a long time?

A. That's correct.

Q. And he looked at these slides himself, he was an experimental pathologist, right?

A. Correct.

Q. And he agreed with Dr. Stemmer and Dr. Kuschner, right?

A. The report I read from him, he did, yes.

Q. Yes. His conclusion was that the renal tumors were not treatment related and there was no statistical significance, right?

A. That's what he wrote in his report.

Q. Did you read the report of Dr. Robert Olson and Dr. Andre Varma?

A. I'd have to go back to my files and see. I mean, I read as many of the reports that I could find.

Q. All those reports are on the internet, aren't they?

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MS. WAGSTAFF: Objection, form.

A. On the internet?

Q. (BY MR. HOLLINGSWORTH) They're online through EPA's website.

A. Through EPA?

Q. Excuse me.

A. I'm sorry. My -- I've always had difficulty with the EPA websites. It's very difficult to find information from their website, at least in my experience. So --

Q. Okay.

A. -- I get very frustrated when I go there and try to find something. But anyway, they're probably available on the website.

Q. (BY MR. HOLLINGSWORTH) Okay.

A. Are they submitted as part of the submission for registration?

Q. Yes, they were.

MS. WAGSTAFF: If you don't know, don't speculate on whether or not they're available.

Q. (BY MR. HOLLINGSWORTH) That's okay. We can go on.

I want to ask you because you mentioned it in your report about the pathology working group that was convened. Do you recall that?

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A. I do.

Q. Okay. And I don't want to go back through stuff that was already a part of your first deposition, but since you --

A. May I --

Q. Sure.

A. May I ask a question?

Q. Sure.

A. Are you going to ask about the report from the EPA pathologist?

Q. Yes, I am.

A. Okay.

Q. Okay.

A. Okay.

Q. The EPA pathologist looked at that control lesion, right?

A. That's correct.

Q. And he didn't make a diagnosis of it, did he?

A. He said he could not confirm that there was a tumor there or not, and he had other pathologists look at it and they could not confirm that was a tumor.

Q. Well, the other pathologists aren't mentioned in Dr. -- you're referring to Dr. Kosza,

right, the EPA pathologist?
A. Oh, yeah.
Q. Dr. Kosza, K-o-s-z-a; is that right?
A. Yes.
Q. He doesn't refer to other pathologists in that report?
A. Again, I -- I remember him referring to a Dr. McConnell, I believe. Looking at it.
Q. Wasn't Dr. McConnell his boss?
A. I don't know.
Q. Okay. You're not suggesting that Kosza formed a pathology working group?
A. No, no, no, no, no. All I'm saying is he was -- he -- my understanding of the information I got pertaining to this particular activity is EPA wanted one of their pathologists to look at the slides to -- to get their own opinion, to give their own opinion of what the tumor incidence was in the kidneys of these male CD-1 mice.
Q. Yep.
A. And the EPA pathologist looked at -- got the slides, looked at them and confirmed that there was three adenomas in the high dose, one in the mid dose, none in the low dose and none -- well, and he said he could not confirm that there was an additional

tumor in the control animals.
Q. Well, he saw something that he said --
A. He said something that may or may not be preneoplastic.
Q. Yeah.
A. But he could not confirm that there was an adenoma in the controls.
Q. Yeah.
A. And I believe in his report he also says that he asked another pathologist or maybe two to look at the slides and they concurred with what he said that they couldn't confirm that there was a tumor in the control group.
Q. Well, I'll come back to that, but did you read the report about that control adenoma which said that it was as wide as five renal tubules?
A. I don't recall reading that, no.
Q. I mean, something that is as wide as five renal tubules is a pretty significant lesion, isn't it?
A. It is.
MS. WAGSTAFF: Object to form.
A. So why was it missed in the initial review?
Q. (BY MR. HOLLINGSWORTH) Well, I -- you

know, nobody knows. But --
MS. WAGSTAFF: Objection. If you haven't seen it and you have it, maybe it would be helpful if you saw it.
THE DEPONENT: Yeah.
Q. (BY MR. HOLLINGSWORTH) Sir, so this pathology working group was convened, right, and you mentioned that in your report to the judge in this case?
A. Correct.
Q. And the pathology working group is something you're familiar with because you've actually written about what pathology working groups are and how they should proceed and what their procedure should be, haven't you?
A. Written about what pathology working groups should do?
Q. Yes.
A. I -- sorry, I don't recall that.
Q. Okay. This pathology working group was made up of five veterinary pathologists, right?
A. I believe that's right, and I believe -- now, this was a pathology working group convened by Monsanto, correct?
Q. Well, EPA required Monsanto to convene

this pathology working group, didn't it?
A. Yes.
Q. And, of course, Monsanto -- nothing happens for free and Monsanto had to convene it, right? Nothing happens for free and Monsanto convened this group --
MS. WAGSTAFF: Object to form. Some things happen for free.
Q. (BY MR. HOLLINGSWORTH) -- in response to EPA's requirement, is that a fair statement?
A. Okay. Yes.
Q. And this group included five doctors. I think, some of them you may know. Doctor, did you know Dr. R.M. Sauer?
A. Sauer?
Q. Yeah, S-a-u-e-r?
A. No, sir.
Q. He had been the pathologist for the National Zoo in Washington for years and was a professor at George Washington University.
A. I'm not familiar with him.
Q. Another one was Dr. Marion Anver (phonetic), did you see her name in those notes?
A. I believe I saw her name, yes.
Q. Do you know her?

A. No.
Q. She was at NCI, National Cancer Institute, for many years. You were there, too, right?
A. Yes.
Q. But it's a big place and you didn't encounter --
A. Right. No, I didn't.
Q. Another member of the PWG was Dr. Strandberg?
A. Strandberg, Strandberg. I saw his name there, too, but I'm not familiar with him.
Q. You don't know Dr. Strandberg?
A. Not that I recall.
Q. Okay. He was at Johns Hopkins experimental laboratory for 30 years, very well known in Washington.
MS. WAGSTAFF: Object to form testifying.
Q. (BY MR. HOLLINGSWORTH) You don't remember him?
A. I don't personally know him, no.
Q. Another guy on this pathology working group that looked at the 1983 mouse renal kidney slides was Dr. Jerry Ward. You know him, right?

A. I know Jerry Ward, yes.
Q. You've published with him before, haven't you?
A. Yes.
Q. You don't have any question -- any reason to question his ability as a --
A. Oh, Jerry Ward?
Q. -- experimental pathologist?
A. No.
Q. He's very well known and very well respected, correct?
A. Correct.
Q. He's still living?
A. I believe so.
Q. The fifth person was Dr. Dawn Goodman, did you know her?
A. Yes, I knew -- I knew Dawn Goodman. Not -- I mean, I knew of her, I guess I should say. I didn't know her personally.
Q. Now, the chairman Dr. Sauer read all these slides again, the same ones that Dr. -- that Dr. Kuschner reviewed and then Dr. Stemmer reviewed and these guys are all looking at these slides through a microscope?
A. I'm sorry, when you say all the slides,

what do you mean?
Q. All the mouse male kidney slides.
MS. WAGSTAFF: Objection to counsel testifying and making a declaratory statement as if they are evidence or true.
A. Okay. I'm -- in my -- all I can state in my experience with the PWGs --
Q. (BY MR. HOLLINGSWORTH) Okay.
A. -- they don't necessarily look at all slides.
Q. I'm going to get to that. Because in the -- in the literature about how PWGs are set up, it's stated -- and I won't remind you that you're an author of this -- it's stated that the chairman of the PWG should look at all the slides and then with respect to the disputed or controversial lesions, he gives those out in a blinded format to the other four members. That's the way PWGs are set up?
A. Right.
Q. True?
A. Right.
Q. And that's what happened here, isn't it?
A. Okay. That's why with when you said all the slides it didn't ring a bell.
Q. Yeah. Sorry. That was my fault.

Dr. Sauer looked at them all and then he gave out to the other four people, including Jerry Ward and Dawn Goodman and the others, the slides that he thought that they should look at and he asked them to look at all the four lesions, the one -- the five lesions, one, zero, one, three and some other things within those mouse -- mouse kidney slides. And they wrote a report about it, didn't they?
MS. WAGSTAFF: Objection to counsel testifying.
A. They wrote a report of their findings, correct.
Q. (BY MR. HOLLINGSWORTH) Okay. And their conclusion was that there was no oncogenic effect that they saw based on their review because they confirmed that there was an adenoma in the control animal, true?
A. They confirmed -- they -- their report indicated that there was an adenoma in the controls, but they also reported that there were two carcinomas in the high dose and one carcinoma in the mid dose, so they diagnosed malignant tumors in the kidney as opposed to the adenomas, which are non-malignant tumors, so what they did was they confirmed the number of tumors, but they upgrade the tumors from adenomas -- three of the five tumors, they upgraded

from adenomas to carcinomas.
Q. Yeah. Okay. Well, I don't think that's quite right but I'm not going to dispute that with you. The conclusion of the five people was unanimous that there was no oncogenic effect from glyphosate that they saw based on their review of the slides, isn't that true?
A. That was their conclusion, I believe, yes.
Q. Now, there was a science advisory panel that was convened by the United States EPA thereafter, an SAP to look at the question of the -- of whether or not glyphosate was carcinogenic in this mouse study in 1983, true?
A. Correct.
Q. And you saw in what you read that there were two members of that scientific advisory panel who looked at these mouse lesions from the male mice kidneys that were part of the controversy, true?
A. I'm sorry, could you repeat that?
Q. There were two members of the science advisory panel at EPA who looked at the same male mouse slides from the 1983 studies as part of the Fifro (phonetic) science advisory science review in 1986, true?

MS. WAGSTAFF: Object to the suggestion that it was the same slides.
A. I -- I -- I don't recall that. I don't know.
Q. (BY MR. HOLLINGSWORTH) I thought that you already testified that the -- you were aware that EPA convened a scientific advisory panel to evaluate the 1983 mouse study data in 1986?
A. I read -- yeah, I read the report.
Q. Yes. And there were two members of that committee who were veterinary pathologists who actually got the microscopes out and looked at those mouse kidney tumors that the EPA had asked them to evaluate in 1986 as part of the scientific advisory panel, right?
A. Is that in their report?
Q. Yes, it is.
A. I'd have to --
Q. You didn't see that?
A. I'd have to look at the report again to refresh my memory.
Q. Okay. You knew a guy who sat on that panel who was an experimental pathologist, a DVM by the name of Swenberg (phonetic), right?
A. Oh, Jim Swenberg, yes.

Q. And you published with him, too, didn't you?
A. I think maybe one or two papers.
Q. Jim Swenberg looked at one of those -- was one of the two pathologists on the science advisory panel to EPA in 1986 that looked at those mouse kidney lesions under the microscope, right, you've read that?
A. I -- again, I'd need to look at the report to refresh my memory. I'm sorry.
Q. Okay. There's another mouse study that you looked at and the author is Dr. Atkinson from 1993 and the sponsor of that study was a company called Cheminova.
A. Okay.
Q. And the authors, Atkinson and others, concluded that there were no compound related neoplastic lesions in that mouse study, true?
A. Okay.
Q. Did you report that to the judge in this case in your expert witness report?
A. I -- again, I was asked to give my opinion of what the data was and my report contains my independent opinion of what the data says, and so I did not put that in the report. It's -- what

I -- I'll just leave it at that.
MS. WAGSTAFF: No. If you have more to say, go ahead.
A. What I was going to say it -- in doing that is not unlike what is done in a number of -- in my past experience as a toxicologist over the past 30 plus years, it's not unusual to convene a -- either a panel or ask somebody to give their opinion of what a data or a set of data says, and when the people, either the group or the individual puts together their report, it is accepted and anticipated that they will put in the report their opinion because that's what's being asked and they will not include other people's -- other author's interpretation of the data because that's not what they're asked to do. They're asked to give their opinion, so the report contains their opinion.
Q. (BY MR. HOLLINGSWORTH) Well, the -- Dr. Atkinson wasn't just an author, he was the original investigator who actually looked at all the slides, wasn't he?
A. I believe he was the pathologist that looked at the slides in this study, yes.
Q. Yeah. But you didn't think that it was necessary, as a scientist, to tell the judge that his

conclusion was that there were no compound-related lesions, neoplastic or otherwise in the study?

A. Again, I wasn't asked to give other people's opinion of what the data said. I was asked to give my opinion.

Q. Okay. You didn't review the full study report for the -- this 1993 Atkinson mouse study that was sponsored by Cheminova, did you?

A. I reviewed all of the study reports and information that was provided to me.

Q. What was provided to you on this study, sir?

A. There were parts of the actual report. Again, I'd have to go back to my files and see exactly all the pieces that I had, but there were -- there were portions of the report, there were -- and usual -- and tables, tumor tables.

Q. Okay. Were these materials provided to you by plaintiffs' counsel?

A. Yes, sir.

Q. Did you rely on Dr. Greim's published review article as a basis for your opinions on the Atkinson --

A. What I would do is I would take the materials provided to me by plaintiff, the reports I

got from this particular study. I would review those and then I would also look at the Greim paper and any additional supporting information from the Greim paper and compare, and then put the information -- and usually -- and I would -- I would say in just about every case, there was correspondence between what was in the Greim and what I was able to glean from the study reports and I used that to prepare my report.

Q. So Greim was reliable in that respect?

A. I told you before, Greim -- I consider Greim reliable because it's a published -- a peer-reviewed paper.

Q. Okay. So you were aware of Dr. Atkinson's and his collaborator's conclusion that this study did not show any neoplastic effect based on administration of glyphosate?

A. I read their opinion, yes.

Q. How did you go -- and you rejected that opinion?

A. I -- I looked at the data, and looking at the results of this particular study, I concluded that there was a significant increase in the particular tumors, in this case, I believe it was hemangiosarcomas. There was a significant increase in the treated animals versus the controlled and it was

due to the exposure to glyphosate and there may have been other cites too.

Q. Did you read -- do you know what JMPR is?

A. That is a -- another regulatory agency of -- I'm not --

Q. It's called the Joint Meeting of Pesticide Residues and it's a part of EFSA?

A. EFSA.

Q. Are you aware that they evaluated the 1993 Atkinson study?

A. Yes, I had seen their report as part of my review and when I participated in the IARC working group.

Q. And you knew that the European regulators at JMPR concluded that this study was not considered to be -- excuse me. You knew that the JMPR regulators reviewed these hemangiosarcomas that you're referring to in the Atkinson report, and they concluded that they -- that those lesions were not considered to be caused by administration of glyphosate, true?

A. I saw that they had done their review, they did a risk assessment for -- for that, and based on their risk assessment of the data, they said it

wasn't -- they did not consider it a carcinogen. However, I did a hazard assessment for glyphosate in my report, and in the hazard assessment you look at the results of the particular study, you evaluate the incidence of the tumors caused by exposure to the compound, and so there was a significant increase in the hemangiosarcomas from this study, and so in my opinion, glyphosate caused those hemangiosarcomas and, therefore, it's carcinogenic in animals.

Q. The -- this same JMPR review that you're referring to or that I referred to in my prior question concluded that glyphosate produced, quote, no signs of carcinogenic potential at any dose, unquote, didn't they?

A. That was in their report, correct.

Q. How did you discount that?

A. I didn't agree with them discounting the hemangiosarcomas as not being compound related. My interpretation was they were compound related, so for the purpose of this hazard identification that I did --

Q. Okay. Did you notice that in the Atkinson report, the incidence of renal tubule adenomas in mice, male mice was two, two, zero, zero?

A. Yeah, I believe I remember that, yeah.

Q. Yeah. So -- so that is another study that finds additional renal tubule lesions in control animals, right?

MS. WAGSTAFF: Object to form.

A. They reported additional -- they had reported tumors in the control animals, that's correct.

Q. (BY MR. HOLLINGSWORTH) When you did your report and made the conclusions that you made about the 1983 mouse study and renal tubule adenomas and carcinomas, did you take into consideration the Cheminova 1993 mouse study authored by Atkinson where they found two renal tubule adenomas in the control animals?

A. For the purpose of my hazard identification, I look at each study individually and I didn't compare them, and, you know, the Atkinson study was done 10 years after the Knezevich or whatever study, so they're not contemporary studies, so. . .

Q. But -- but they would be included in the category of control -- of -- of historic controls, wouldn't they?

A. They would be, but as I indicated before, the most appropriate controls for any study is

the concurrent controls. First, you look at the results of the exposure to the treated animals versus the concurrent controls, and see if there is an increase in tumor formation in the treated animals, that is the most appropriate control to use in any study. Then after you've done that evaluation, you go and look at the historical control data to see if well, maybe this was a spurious result or something, so -- but, you still have to look at the -- the study that, as it was performed, and the concurrent controls, that is the most important thing to do in your evaluation of a particular study.

Q. Haven't you published that using the historic controls is a piece of quote, key data --

MS. WAGSTAFF: Objection, asked and answered already.

Q. (BY MR. HOLLINGSWORTH) -- in doing that evaluation?

A. I don't recall that. I'd have to see the publication.

Q. All right. Now, on -- regarding your opinion on the hemangiosarcomas in these male mice in the Atkinson study, the data that you were looking at going from control to low dose to mid dose to high, was zero in the controls, zero in the low dose, zero

in the mid dose and four hemangiosarcomas in the high dose animals, right?

A. Correct.

Q. And you're talking about male mice here, true?

A. Correct.

Q. And you refer this -- to this in your report as a dose-related increase, right?

A. Well, it was a positive trend test. It was positive in the trend test, so. . . There was a positive increase in trend of the tumor as you increased dose.

Q. Isn't -- isn't it true that the incidence in the high dose group was not statistically significant when it was done in comparison to the control animals?

A. In a pair-wise comparison, it did not reach statistical significance that's controlled, that's correct, but in a pair-wise comparison for trend, it was positive. So there was an increase in the trend in the formation of these hemangiosarcomas in these animals, so, therefore, it's a positive effect, a positive response to the glyphosate causing an increase in the trend in the formation of these tumors in these animals.

Q. You didn't do that trend test yourself, did you?

A. No, I didn't.

Q. You relied on someone else?

A. Yes.

Q. Who did you rely on?

A. I think it was -- I think it was the EPA. I don't know. I don't remember. I'd have to -- I really actually need my other sheet to -- I put on there where I got the trend test from.

Q. Are you talking about one of your cheat sheets?

A. The sheet that I prepared where I just summarized all of the information as a quick reference so I wouldn't have to go leafing through this.

MS. WAGSTAFF: If it's important to you to get an answer to that, he can reference it if you want.

MR. HOLLINGSWORTH: No, you know, I can understand why you might need a cheat sheet to get through this kind of stuff.

MS. WAGSTAFF: Sort of a dense deposition.

A. A lot of information to remember.

Q. (BY MR. HOLLINGSWORTH) I've got a few of

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them myself.
Now, you didn't find any consistent -- any finding consistent with males with hemangiosarcomas when you looked at female animals, did you?
A. For the females, there was an increase, but it was -- it was only zero, zero, one, so one tumor was found in the high dose females. Just seeing one tumor in the females was not enough to infer any -- anything, really, but the fact of the matter is there was one seen in the female mice.
Q. But there was no replication of the finding of hemangiosarcomas in males that you report on in this report that you gave to the judge in the MDL when you looked at the female mice, true?
MS. WAGSTAFF: Object to form --
A. In this study --
MS. WAGSTAFF: -- with the word "replication."
A. Sorry. In this study, I didn't see, no.
Q. (BY MR. HOLLINGSWORTH) You didn't see replication in it -- in the other sex?
A. In the female.
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Okay. And you

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know that this Atkinson study that we're talking about now was submitted to EPA?
A. Yes, sir.
Q. And you know that EPA didn't consider the increase in hemangiosarcomas to be treatment related, that is related to the administration of the test compound glyphosate?
MS. WAGSTAFF: Object to form.
A. When the EPA did their risk assessment of this particular study, for glyphosate, that was their conclusion for the purposes of their risk assessment. Again, what I performed was a hazard identification for this particular study evaluation, and I felt that the -- the increased incidences and trend of the hemangiosarcomas in the male mice was due to the treatment of glyphosate. So for my interpretation is that it was compound related or related to glyphosate exposure and a positive response.
Q. (BY MR. HOLLINGSWORTH) Did you have the impression when you were reviewing the materials that you reviewed on the Atkinson Cheminova -- Cheminova is C-h-e-m-i-n-o-v-a, mouse study that the EPA had more data available to it than what you reviewed?
MS. WAGSTAFF: Object to form.

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A. I don't know that they had more data than I did or not. I wasn't at the EPA reviews, so I -- I really am not, I guess, privy to all the -- to all the data -- knowing all the data that they had, so I really can't say.
Q. (BY MR. HOLLINGSWORTH) Has your opinion that these hemangiosarcomas in the male mice in the Atkinson study is related to glyphosate been published and peer reviewed?
A. Has my opinion?
Q. Yes.
A. No. My opinion has just been, I guess, quote, published in this report.
Q. Do you know of anywhere in the peer-reviewed literature where the finding of hemangiosarcomas in male mice has been published and peer reviewed?
A. I'm sorry, could you repeat?
Q. Sure. Do you know of any published peer-reviewed report in the medical literature anywhere that the findings of hemangiosarcoma that you describe in your report, which you claim are attributable to glyphosate has been published and peer reviewed?
A. I'm not aware of any report published in

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the peer-reviewed literature to that effect, no.
Q. Okay. I'd like to ask you about the third mouse study which is by Arysta as the sponsor. A-r-y-s-t-a. And Dr. Sugimoto was the lead veterinary pathologist on that study. Are you familiar with that study?
A. Yes.
Q. And are you aware that the study authors and investigators concluded that there was no compound-related neoplastic or oncogenic or carcinogenic effect from glyphosate in the administration to mice in this study?
A. Of the -- I'm sorry. Could you repeat?
Q. Sure. Are you aware that the original authors and investigators on this study wrote a conclusion stating that there were no compound-related neoplastic or oncogenic effects from the administration of glyphosate to these mice?
A. I did read that in their report, yes.
Q. Did you report that to the judge in this case in your expert report?
A. Again, I was asked to give my opinion of the data and so that is what I put in my report and not the opinion of anybody else.
Q. Now, the Arysta or Sugimoto report was

submitted to the United States Environmental Protection Agency, right?

A. Correct.

Q. What data did you rely on specifically in making your evaluation of this?

A. Similar to the other report, I looked at the study report or the study reports or the portions of the study reports that were provided to me by plaintiffs' attorney. That included portions of the -- of the actual report and/or tumor tables. I looked at that, and then I went and looked at the Greim publication. Looked at the data that was provided in that. I would compare, and like I said before, they usually matched pretty well. And then I would take that information and wrote my report accordingly.

Q. Okay. Did you read the actual pathology report from this study?

A. Again, I'd have to go back to my files and see if -- if I had the actual pathology report. I know I had -- I know I had the tumor tables from the report. I don't recall for this particular study if I had the pathology report or not. I'd have to go back to my files to look at it. If I had it, I definitely read it, but I -- to be honest, I just -- for this

study, I just don't recall.

Q. Isn't it always important to read the original pathology report from an author like -- or investigator like Dr. Sugimoto?

MS. WAGSTAFF: Objection to form.

A. If -- if I -- if the pathology report is available, yes, you should read the pathology report to see what the original pathologist said. And like I said, if the report was there, I read it, but I just don't remember for this study.

Q. (BY MR. HOLLINGSWORTH) Did you ask counsel for the plaintiffs to provide you with the original pathology reports in each of these 12 written studies that you looked at?

A. I asked them to provide me all the data -- all the information they had and I relied on them to provide me that -- what information they had available to them. And I'm confident if they had anything on any of these studies, they forwarded it on to me for my review.

Q. What piece of information informed you that you were -- and that made you aware that the original investigator, Dr. Sugimoto and his collaborators, concluded that there were no compound-related neoplastic or oncogenic effects from

administration of glyphosate to these rats, I mean, excuse me, these mice in 1997?

A. I -- I'm sorry, I missed the first part of that question. Could you repeat? I'm sorry.

Q. All right.

MR. HOLLINGSWORTH: Tracy, here is a test for you.

MS. WAGSTAFF: This is not nice.

(The question was read back as follows: "What piece of information informed you that you were -- and that made you aware that the original investigator, Dr. Sugimoto and his collaborators, concluded that there were no compound-related neoplastic or oncogenic effects from administration of glyphosate to these rats, I mean, excuse me, these mice in 1997?")

A. So for that it -- it would have been in the -- in the report that I got from -- from plaintiffs' attorneys. It would have been in the -- in -- in the -- probably in the summary of the report or what have you. I -- you know --

Q. Okay.

A. -- I can't remember.

MS. WAGSTAFF: Can I ask just an administrative question? It's 11:45, so I don't know

if you want to -- if you want to take a late lunch, we should probably break now, but if you want to eat earlier, I don't know. You guys are on East Coast time, so what do you want to do?

MR. HOLLINGSWORTH: We're -- we're-- we're good.

MS. WAGSTAFF: Okay. So do you want to take a small break and eat lunch at 1:00 or do you want to go --

MR. HOLLINGSWORTH: You want to take another break now?

MS. WAGSTAFF: If we're going to go another hour and something. I'm saying it's 11:50, so we can either take a short break and -- do you want to take a little break right now? Let's take a little break.

THE DEPONENT: Okay. We can take a little break right now if --

MR. HOLLINGSWORTH: Okay.

MS. WAGSTAFF: Yeah.

THE VIDEOGRAPHER: Going off the record. The time is 11:50 a.m.

(Recess taken, 11:50 a.m. to 12:02 p.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 12:02 p.m.

MR. HOLLINGSWORTH: All right. Counsel, when did you want to adjourn for lunch?
MS. WAGSTAFF: Well, what do you think? I would leave it most up to Dr. Jameson, who --
MR. HOLLINGSWORTH: Sure.
THE DEPONENT: I mean, I'm good. We could adjourn at 1:00 if that's okay with everybody or --
MR. HOLLINGSWORTH: Is that all right with everybody?
THE DEPONENT: Or sooner if they need it.
MS. WAGSTAFF: I'm the only one that lives on mountain here.
MR. HOLLINGSWORTH: If I need to stop before lunch, I'll let you know that, but I'll probably be all right.
Q. (BY MR. HOLLINGSWORTH) Sir, we were talking about the Sugimoto 1997 mouse study?
A. Uh-huh.
Q. Sponsor was Arysta. Did you say that you had reviewed the pathology study for this? Sorry if you already testified.
A. The pathology study?
Q. I'm sorry, the pathology report within

the study.
A. Again, specific to this particular study, I don't remember if I had the pathology report. If I did, I'm -- I did review it.
Q. Do you have in mind your review of the hemangiosarcomas in this study?
A. Yeah, the incidences, yes.
Q. The incidence was zero in the control, zero in low dose and zero in mid dose and two in high dose males? Zero, zero, zero, two.
A. Four.
Q. Not four, two.
A. 4 percent. I'm sorry.
Q. When you said 4 percent, you're referring to the high dose percentage right?
A. Right.
Q. And you said that this results in a significant P value using the Chi-Square test?
A. Yes.
Q. Why did you use the Chi-Square test here, sir?
A. Again, I'd have to go back and look. I did not perform the statistics myself, I don't believe. I'd have to go back and see the source of this. It -- I just don't recall where -- where --

where I got it from.
Q. Who performed the statistics using the Chi-Square test?
A. Again, I'm going to need my other sheet.
MS. WAGSTAFF: All right. Counsel, I'd like to -- I'm going to give him a copy of his cheat sheet and I'll give you a copy as well if you'd like one.
MR. HOLLINGSWORTH: Okay. I've been dying to get that.
MS. WAGSTAFF: You have been, I know.
MR. HOLLINGSWORTH: You notice I specifically did not ask for it.
MS. WAGSTAFF: Okay. So I'm looking for ones that don't have handwriting on it.
THE DEPONENT: I have --
MS. WAGSTAFF: Okay. Here is yours. Here is one for rat and for mouse.
MR. HOLLINGSWORTH: Thank you.
MS. WAGSTAFF: If you want to mark those as an exhibit or whatever you'd like to do.
A. I got the numbers from -- from something I got from Chris Portier.
Q. (BY MR. HOLLINGSWORTH) Okay. Thank you. Let's mark this --

MS. WAGSTAFF: There's two separate ones.
Q. (BY MR. HOLLINGSWORTH) Okay. We'll mark the first one of these two page documents as two Exhibit 22-2 and you referred to this earlier this morning euphemistically as a cheat sheet. I haven't looked at it yet and I believe and then I'll mark the next one as --
MS. WAGSTAFF: You can see one is labeled rat and one is mouse up on the left.
Q. (BY MR. HOLLINGSWORTH) Okay. Good. 22-3 is the --
A. The upper left-hand corner.
MR. HOLLINGSWORTH: 22-3.
MS. WAGSTAFF: Is rat. It's upper left. 22-2 is mouse and I'm just making sure this is the same one before I hand it over. Which one did I give you before, the rat or the mouse?
MR. HAAKE: Rat.
MR. HOLLINGSWORTH: Thank you.
Q. (BY MR. HOLLINGSWORTH) So you think the Chi-Square test came from Dr. Portier?
A. Yes, sir.
Q. Did you rely on Chi-Square test for renal tubule tumors as well? Or renal tumors as

well?
A. Are you talking about for the Knezevich?
Q. No, I'm talking about the Sugimoto on 1997 Arysta. I'm still talking about the hemangiosarcomas.
A. Hemangiosarcomas?
Q. In the male mice, and then I was wondering whether you had also run a Chi-Squared P value case for renal tumors?
A. I believe that's the case, yes.
Q. Okay. Now, are you -- are you aware that Dr. Portier submitted an amended report in this case?
MS. WAGSTAFF: Object to form.
A. I'm not sure what report you're referring to.
Q. (BY MR. HOLLINGSWORTH) Okay. He has two reports. He has a report -- an opening report like yours and then he submitted an amended report in addition. Have you read both of his reports?
MS. WAGSTAFF: Object to form.
A. I'm sorry, are you referring to his expert report?
Q. (BY MR. HOLLINGSWORTH) Yes. In this case.

A. I'm sorry.
Q. Sorry.
A. That's okay. Yes.
Q. Okay. And are you aware that for the incidence of hemangiosarcomas in male mice in this study, the Arysta 1997 study by Sugimoto, Dr. Portier reported a non-statistically significant trend with a P value of .06?
A. I'm trying to remember if I saw that in his report or not. The value that I have here is based on some -- how shall I -- I don't know if it's communication or what. After -- let me back up. As you know, or are aware, I've known Chris Portier for a long time. In fact, we worked together for a very long time and Chris was also a special -- I forget what his title was, but at the monograph 12, he was also a special invitee who attended the meeting. And after the meeting, he and I and a number of other people also published some -- some -- some work in response to the -- the findings that we made at the IARC meeting.
And he and I kept in contact about glyphosate because of that and this -- this particular number came from some -- some of the conversations we had when we were putting together that publication,

and prior to his expert report. So if he has a number in his expert report that is different than this, it's probably due to the fact that he did additional analysis or subsequent analysis of the data because being a statistician, they always evaluate and reevaluate the data, so that --
MS. WAGSTAFF: If you don't know, don't speculate.
A. But I don't know.
Q. (BY MR. HOLLINGSWORTH) Would you defer to Dr. Portier and his opinion based on the issues of statistics and biostatistics?
A. Okay. Since Chris is a well-known biostatistician, I would have to defer to him, correct.
Q. And would you agree that the Chi-Squared test is not a traditional method that's used to evaluate the incidence of tumors in long-term chronic bioassays in rodents?
MS. WAGSTAFF: Object to form.
A. There are a number of different statistical methods used in the evaluation of data for animal toxicity and chronic carcinogenicity studies and they all are used frequently in all the publications that I see, so. . .

Q. (BY MR. HOLLINGSWORTH) Okay. You can do the Chi-Squared test yourself, can't you?
A. I could.
Q. I mean, I can do it on the back of an envelope, right, it's an easy thing to do?
MS. WAGSTAFF: Object to form.
A. If you say you can, I guess, I don't know.
Q. (BY MR. HOLLINGSWORTH) Okay. You can do one?
A. If I had to, I could do one.
Q. And were you also aware -- we were just referring to the hemangiosarcomas and your opinion that they were statistically significant and Dr. Portier's opinion that they were not statistically significant. Do you understand that?
A. Yeah, that's what we were talking about.
MS. WAGSTAFF: Form.
Q. (BY MR. HOLLINGSWORTH) Okay. He also -- he, Dr. Portier, also ran statistics on the renal adenomas, and, of course, you concluded that using the Chi-Squared test that the renal adenomas that were found in the male mice in 1997 study were statistically significant. Did you know that?
MS. WAGSTAFF: I'm going to object

to -- to quoting or paraphrasing Dr. Portier's expert testimony and/or report. I think that you are cherry picking pieces of his report out of context and not giving the full context of his report. If you'd like him to opine on Dr. Portier's report, let's pull out Dr. Portier's report and let him read the whole thing.

Q. (BY MR. HOLLINGSWORTH) I'm not asking that. My question is whether he's aware that Dr. Portier also ran statistics on the renal adenomas and other renal lesions seen in the 1997 Arysta study.

MS. WAGSTAFF: Same objection.

A. I -- I don't know if he did or didn't.

Q. (BY MR. HOLLINGSWORTH) Okay. You don't know that he found a P value of 0.62 also for the renal adenomas which was not statistically significant?

MS. WAGSTAFF: Same objection and throughout this deposition, we've asked for documents that you've been citing to and every time you have refused to provide a document, so if you want him to opine on Dr. Portier's testimony, I would request that you allow him to read the deposition transcript right now or the expert report of which you cite.

MR. HOLLINGSWORTH: Well, when he's at lunch he can look at page 42 -- 41 and 42 of Portier's

report because that's where I got that information from. So if I'm wrong, you can tell me after lunch.

MS. WAGSTAFF: No, that's not how it's going to happen. If you want him to look at something, it will be on the record and will go against your time as your lawyers have made in our depositions, specifically including the Mark Martinez deposition when I asked him to read something off the record, and it was counted against my time, so if you want him to read something, he will for sure do it, but it's going to be on the record.

MR. HOLLINGSWORTH: Okay.

Q. (BY MR. HOLLINGSWORTH) My question, though, is are you aware that your friend Chris Portier, your long-time friend, had run statistics on the renal adenomas that were recorded in male mice in the Arysta study?

MS. WAGSTAFF: Object to the form of the question.

A. I -- I'd like to see his report before I respond to that.

Q. (BY MR. HOLLINGSWORTH) Okay. It's at 41 and 42 if you want to look at it over the lunch period.

MS. WAGSTAFF: Objection. I just told

you if you want him to read something and to respond to one of your questions, provide him with the document and he'll do it on the record.

Q. (BY MR. HOLLINGSWORTH) Sir, you also considered this Arysta 1997 study by Dr. Sugimoto and others to show an increased incidence of what you say is malignant lymphoma, true?

A. Correct.

Q. And the incidence that you report in your report to the judge is two, two, zero, six, right?

A. Correct.

Q. 12 percent in the high dose animals?

A. (Deponent nodded head up and down.)

Q. 12 percent incidences is what you report, right?

A. Correct.

Q. And the incidence of six in the high dose animals was not statistically significant when compared with the concurrent controls, was it?

A. The incidence in the high dose was not statistically significantly different from the controls.

Q. Correct.

A. Correct.

Q. Do you report that?

MS. WAGSTAFF: Object to form.

A. Do I report that?

Q. (BY MR. HOLLINGSWORTH) Yes. At 22 and 23.

A. Are you talking about the hemangiomas -- lymphomas?

Q. Yes. You report that, don't you?

A. I'm looking.

MS. WAGSTAFF: Object to the phraseology of "report that."

A. Okay. Could you repeat the sentence again, please?

Q. (BY MR. HOLLINGSWORTH) I said do you report that the incidence of six in the high dose group regarding malignant lymphoma was not statistically significant when compared with current controls?

MS. WAGSTAFF: Object to form.

A. That's what I report, yes.

Q. (BY MR. HOLLINGSWORTH) Are you aware that the European regulators did an analysis of the Arysta 1997 report, including statistical analyses?

MS. WAGSTAFF: Object to the form.

A. Okay. I'm sorry. I was looking at

something.
Q. (BY MR. HOLLINGSWORTH) Okay.
A. I'd like to add something to the -- to my last response, but I'll answer this first.
Q. Okay.
A. So if you could repeat the question.
Q. The question was this, you are aware that the European regulators reviewed this report and did a statistical analysis of the Arysta study -- I shouldn't say report. It's a study.
A. Yes.
Q. Okay. And let me just finish my question --
A. Sure.
Q. -- and you can go back and correct. And you're aware that the historical control rate that they report for malignant lymphoma is 4 to 19 percent in control animals as a range?
A. For historical control?
Q. Yes.
A. In the -- I'm sorry -- in the -- in their report?
Q. Yes.
A. Yes. Okay.
Q. You've read their report, right?

A. Yes.
Q. You responded to their report partially, you and Chris Portier did, didn't you?
A. Yes.
Q. So you're familiar with that control range that they reported and -- and you would agree that the 12 percent rate that was found in the high dose males is within that control rate --
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) -- that the European regulators reported?
A. It's within that -- that report, indicated in the report. As I indicated before, the most appropriate controls for this study and any study is the concurrent controls. So -- and based on the concurrent controls is an increase in trend with this incidence.
Q. Well, the -- you -- you determined that the incidence was not statistically significant, didn't you?
A. In the high dose?
Q. Yeah.
A. That's what -- in this particular case, yes.
Q. Okay.

A. But if I can continue on with that, I also state in my report --
Q. Where are you now?
A. On page 22.
Q. Yep.
A. Towards the end of the paragraph.
Q. Yep.
A. I also state in my report that I also reviewed the Tier II summary for glyphosate carcinogenicity --
THE REPORTER: I'm sorry, I didn't understand that. --
Q. (BY MR. HOLLINGSWORTH) Where are you now on page 22?
A. Page 22.
Q. I see. Okay. Thank you.
A. I also reviewed the Tier II summaries --
Q. Yes.
A. -- for glyphosate carcinogenicity studies from Greim, et al., for study 12, which is Sugimoto.
Q. Sugimoto.
A. Sugimoto, excuse me. Which showed a reported statistically significant increase in malignant lymphoma in high dose male mice.

Q. I understand that. I was getting ready to ask you about that, but I haven't asked you about that.
A. Okay.
MS. WAGSTAFF: Do you want to correct your previous answer before we get too far down the road? You put a note on the record that --
THE DEPONENT: This is the --
MR. HOLLINGSWORTH: That's the correction --
A. This is what I wanted to add that I found additional information from the Greim that actually had a different tumor incidence and that particular tumor incidence was statistically significant in the high dose. That was the point I wanted to make.
Q. (BY MR. HOLLINGSWORTH) Yeah. You're aware of literature and you've already testified to it this morning, I think, that there is a -- that malignant lymphoma is among the most commonly occurring spontaneous neoplasm in mice?
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Isn't that right?
A. It depends on the strain.

Q. In CD-1 mice.
A. In CD-1 mice, there's a fairly high incidence.
Q. Yeah. I mean, it goes up to 50 percent, doesn't it?
A. I don't know. I don't know what -- how high it goes up to off the top of my head. But I know it has a high spontaneous incidence.
Q. We had figured out that your report was wrong where it referred to hemangiosarcoma --
A. Oh, hemangiosarcoma --
THE REPORTER: Please don't speak at the same time.
THE DEPONENT: I'm so sorry.
MS. WAGSTAFF: Object, it wasn't wrong. We told you that there was a typo that changed it in three places, and I object to you calling it wrong.
MR. HOLLINGSWORTH: I said we thought it was wrong based on the way his report was written and the way that we received it and we went back to all the data and we could see that the numbers were completely wrong, so thanks for making that correction.
Q. (BY MR. HOLLINGSWORTH) Now, as to Nufarm, which is the next study I'd like to ask you

about, Dr. Jameson. I think that's the fourth of five mouse studies which you have referred to in your report.
A. Uh-huh.
Q. And the investigator was Dr. Wood and others. Did you know Dr. Wood?
A. No.
Q. Okay. Did you know anyone at that laboratory?
A. Which laboratory was this?
Q. No. I don't have that information.
A. Okay.
Q. Now, the study authors, the original study authors of the Nufarm 2009 study, Nufarm was the sponsor, right?
MS. WAGSTAFF: Object to form.
A. That's what it said in the Greim publication. They identified it as that, yes.
Q. (BY MR. HOLLINGSWORTH) Was Nufarm a company that wanted to manufacture glyphosate and get a registration for it?
A. I know nothing about that company.
Q. Okay. Now, the original authors, Dr. Wood and others, concluded that there was no compound-related effect whatsoever in this study with

respect to oncogenic or neoplastic effects, true?
A. I recall reading that in the report that I reviewed.
Q. Okay. Did you review all of the data from this study, including the pathology report?
MS. WAGSTAFF: Object to form.
A. For this particular study, I think I did not have -- I know I did not have the full study report. I know I had some tumor tables to look at. And some other documents from the -- from the report, but I -- I did not have the pathology report for this one, I'm sure.
Q. (BY MR. HOLLINGSWORTH) Where did you get the information that you did have about the Nufarm study by Dr. Wood?
A. Well, again, I got -- I got some information from plaintiffs' lawyers and -- but probably for this particular one, I think I relied heavily on the information in the Greim publication.
Q. And you know that the Nufarm study in 2009 by Dr. Wood was submitted to EPA, right?
A. Yes.
Q. And you -- you say in your report at page 23, that the formation of malignant lymphomas and the formation of adenocarcinomas of the lung -- do you

see that?
A. Yes.
Q. -- in this study was due to treatment with glyphosate in male mice. Do you see that?
A. Yes.
Q. And then you make a reference to malignant lymphoma and high dose -- in the high dose male treatment group, right?
A. Yes.
Q. And an increase in the trend of formation of adenocarcinomas of the lung and -- sorry -- malignant lymphomas as your third point, right?
A. I'm sorry, I didn't hear that last part.
Q. You make a reference to an increase in the trend of formation of the adenocarcinomas of the lung -- lung -- lung?
A. Yes.
Q. I have a question about, and then you say and malignant lymphomas in males, true?
A. Yes.
Q. Now -- now, the incidence of lung adenomas or I should say adenocarcinoma that you refer to in the high dose males was not statistically significant when compared to controls, was it?

A. When compared to the concurrent controls, it was not statistically significant, that's correct. It was positive -- it was statistically significant increase in trend for the formation of these tumors in the male mice.

Q. Have you read the EPA's Office of Pesticide Programs' report on glyphosate and the re-registration of glyphosate in 2016?

A. Yes.

Q. They -- they do an analysis and state that that -- that those lung adenocarcinomas in high dose males are not statistically significant, don't they?

A. That the incidence of tumors is not statistically significant?

Q. Yes.

A. Yes. They say the -- the incidence is not statistically significant.

Q. And they say that there were no treatment-related preneoplastic lesions that were observed in that study?

A. I have to look at the -- at that report again to say definitely that they -- that they said no -- no preneoplastic lesions, but I -- I -- I think that's correct.

Q. You didn't comment on that in your report to the judge, did you?

A. No.

Q. Now, did you tell me that you -- that you don't think that the existence and progression of and incidence of preneoplastic lesions is as important today as you thought it was years ago?

MS. WAGSTAFF: Object to form.

A. I don't recall saying I didn't think it's as important today as it was before. I don't remember saying that particular thing.

Q. (BY MR. HOLLINGSWORTH) Is it fair to state that the interpretation of tumor responses in two-year assays is an art?

A. The interpretation of --

MS. WAGSTAFF: Object to form.

A. I'm sorry, could you rephrase that question?

Q. (BY MR. HOLLINGSWORTH) Is it fair to state that the interpretation of tumor responses in two-year assays is an art?

MS. WAGSTAFF: Same objection.

A. I -- well, some individuals might think it's an art.

Q. (BY MR. HOLLINGSWORTH) Okay.

A. Are you -- I don't know where you're getting that quote from. You're probably getting it from a publication.

Q. John Booker was a long-time friend of yours, right?

A. John is, yes.

Q. Yep. And he was -- was he your boss?

A. Yes.

Q. Okay. These -- going back to the adenocarcinomas in high dose males, they weren't repeated or seen in any other mouse studies, were they?

MS. WAGSTAFF: Object to form.

A. I'd have to go back and check and see. Are you talking about in the mice?

Q. (BY MR. HOLLINGSWORTH) Yes.

A. No. I don't believe it was seen in any other studies in a significant manner. That's not to say that there weren't some lung tumors seen, some adenocarcinomas seen in some of the other studies, but they -- they were not at a significant -- they weren't significant compared to controls and I didn't include them in my report.

Q. Okay. So there was no replication of the adenocarcinomas in other mouse studies that you

reviewed, the four other mouse studies I'm referring to, of course?

A. Like I said, there -- I don't recall the specifics, but I -- I -- I vaguely remember seeing lung tumors reported in some of these other studies, but they weren't significantly different than what was found in the control, so I didn't include them in my report. So -- but no -- no other study had a statistically significant increase in lung adenocarcinomas.

Q. That's including rats, too, isn't it?

A. Yes, I think that's probably correct for rats, but, again, it may have been tumors, lung tumors seen in some of the studies, but they weren't significantly different than what was observed in the controls --

Q. I'm --

A. -- so I didn't include them in my report.

Q. So you didn't report the replication of findings of adenocarcinoma in the lung in any other mouse or rat study besides the Nufarm 2009 study that we're referring to now?

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) True?

A. The -- that was the only study that I reviewed where there was a significant increase in lung adenocarcinomas reported.
Q. Are you aware that Dr. Portier has determined on his own statistical evaluation that the incidence of lung adenocarcinomas in this study that you reported about in your report to the judge is due to chance?
MS. WAGSTAFF: Objection.
A. I'd have to see Chris' report to comment on that. I don't know.
Q. (BY MR. HOLLINGSWORTH) No one has -- no one has pointed that out to you?
A. No one has pointed that out to me, no.
Q. Okay. And you're aware that the United States EPA's Office of Pesticide Programs report in 2016 concluded that the lung adenocarcinomas in this study was not treatment related?
MS. WAGSTAFF: Objection.
Q. (BY MR. HOLLINGSWORTH) Excuse me.
A. I'm sorry, could you repeat that?
Q. The United States Office of Pesticide Programs determined in 2016 in their report, which you said you had read, right?
A. Yes.

Q. That the lung adenocarcinoma that you state -- you stated in your report is statistically significant in the Nufarm 2009 study was not a positive finding based on -- based on administration of glyphosate to these male mice?
MS. WAGSTAFF: Objection, misstates the report.
A. Well, that finding by the EPA was based on their risk assessment that they were doing for glyphosate. And I -- and evidently based on the criteria that they used for doing a risk assessment, it did not meet that criteria to be called a carcinogen.
What I have done is a hazard identification assessment of this particular study, and based on my evaluation of the data for the adenocarcinomas, there was a positive trend in the formation of the lung adenocarcinomas in the male mice, and it is that increased -- that trend is attributed to the glyphosate, so, therefore, glyphosate caused those tumors or caused cancer in the experimental animals, so it's an animal carcinogen and therefore a potential human carcinogen.
Q. (BY MR. HOLLINGSWORTH) So you disagree with the EPA when they stated that the incidence of

lung adenocarcinomas in this study, the Nufarm study in 2009, is not due to treatment with glyphosate?
MS. WAGSTAFF: Objection, misstates the report.
A. Again, the EPA did a risk assessment, and based on their risk assessment, evidently, they did not feel that the adenocarcinomas could be called a carcinogen for their risk assessment. But for the push of the hazard identification that I did, I determined that the adenocarcinomas seen in the male mice in this study were caused by glyphosate, so glyphosate caused an increase in the trend of these tumors, therefore it's an animal carcinogen and a potential human carcinogen.
Q. (BY MR. HOLLINGSWORTH) So you disagree with EPA's report by the Office of Pesticide Programs in 2016?
MS. WAGSTAFF: Objection, asked and answered.
A. They -- they are -- you're asking me to compare apples and oranges.
Q. (BY MR. HOLLINGSWORTH) Okay.
A. They did -- they did a risk assessment, I did a hazard assessment. For the purpose of my hazard assessment, I don't agree with the way they

threw out that particular study.
Q. (BY MR. HOLLINGSWORTH) Okay. Now, again in this study you refer to malignant lymphoma. Do you have that in mind?
A. Yes.
Q. Have you read Jerry Ward's publication on the incidence of malignant lymphoma in aging mice?
A. I don't think I've read that particular paper, no.
Q. Okay. How would you rate, in -- given your experience, your vast experience, how would you rate the incidence of malignant or the ranking of malignant lymphoma in mice from most common to least common lesion or tumor?
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) In other words, would you say it is the first, most common tumor seen in mice, it meaning malignant lymphoma or the second or third or the 15th or what?
A. Well, there, again, it depends on what strain of mouse you're talking about.
Q. We're talking about CD-1.
A. And male or female.
Q. Talking about CD-1 males and females.
A. Males and females?

Q. Yes.
A. I know that malignant lymphomas are found in -- let me rephrase that. I know that spontaneous incidence of malignant lymphomas in CD-1 mice is -- is relatively high, but I don't know how it ranks amongst all of the various different types of spontaneous tumors seen in that strain of mouse. I'd have to go look it up, but I know -- I know it's one of the high -- highest ones, but I don't know how it ranks compared to the rest of the spontaneous tumors seen in those animals.

But just because something occurs because of a spontaneous rate is no reason to discount it from being an effect in a carcinogenicity study.

Q. (BY MR. HOLLINGSWORTH) Well, would -- if you were doing a risk assessment instead of a hazard assessment, would you have reason to discount the high level of -- the extremely high background incidence of malignant lymphoma in mice?

MS. WAGSTAFF: Object to form. It's outside the scope of his expert testimony.

A. I haven't done a risk assessment on that, so I can't comment on that until I've done one.

Q. (BY MR. HOLLINGSWORTH) Is there something in the hazard assessment protocol that

allows you to discount a high background incidence of tumors that occurs spontaneously in mice like malignant lymphomas?

A. Well, if -- if you will -- if you look in my report, I think there was a -- a study in rats where there was a -- an increase in the incidence of -- is it liver tumors? I think it was liver tumors in rats. That was -- that was a positive increase in the incidence of liver tumors in rats, but I discounted it because of the high background -- high historical incidence.

So I have discounted studies because of high historical rates, but for this particular case, and for this mouse study, I didn't think it was appropriate to do.

Q. Why?

A. Because the -- the -- the incidence -- are you talking about the lymphomas?

Q. Yes.

A. Because first of all, for the malignant lymphomas, there was a statistically significant increase in the incidence of malignant lymphomas in the high dose animals compared to control. So that was a statistically significant increase in the high dose animals. Then in addition to that, there was

also a statistically significant increase for trend for formation of this tumor in malignant lymphomas in the mice in this study.

So because you had a significant increase in the incidence in the high dose and you also had a significant increase in the trend for the formation of this tumor in the animals, I felt it wasn't appropriate to discount this particular study.

I mean, I'll grant you that zero out of 51 in the controls is a low -- is -- is -- is low for this -- for CD-1 mouse in the study, but that's what the concurrent controls are. They found no malignant lymphomas in the controls, so, therefore, this is -- this is a very -- in my mind, this is a very strong finding and I really am surprised to the point of shock that the EPA would throw out something like that, so, but -- enough said.

Q. Okay.

A. And just -- I'm sorry. I don't mean to interrupt, but just for your reference, that study that I was referring to or I threw out -- where I discounted the study because of the incidence was within the historical rate, it is the Bramer (phonetic) study in rats. 2001. This was in the Wistar rat. It's the Greim study seven.

And they had a significantly -- a significant increase in -- in the liver tumors in this one, but the -- it was within the historical control, so I discounted it.

Q. Well, your -- are you aware that the German or EFSA, European regulators, show an incidence of lymphoma ranging from zero to 32 on a spontaneous basis, that is 32 percent at the high, in CD-1 mice?

A. I'd have to look at the report to refresh my memory on that, but I'm -- okay.

Q. They found a study that had zero in the controls in Europe, too.

A. Okay.

Q. And they -- but they saw a range of zero to 32.

A. I'm sorry. I didn't mean to interrupt.

Q. No, go ahead.

A. In this particular study, you're talking about?

Q. No, I'm talking about when they did their -- the European assessment of the IARC report to which you responded. They made the observation that their own historical control from nine studies involving the CD-1 mice, all from the same period by sister laboratories, included a range of malignant

lymphomas from zero to 32, which tells me that it's not so surprising that you might have a study out there, an outlier, that has zero lymphomas in one of the either control or treatment groups.

A. Okay.

MS. WAGSTAFF: Wait. Objection, I move to strike that testimony from counsel about what he finds surprising and doesn't find surprising.

MR. HOLLINGSWORTH: Well, that's in reference to the witness's answer to a prior question indicating that he was shocked at what EPA did with respect to this data.

MS. WAGSTAFF: But, Dr. Jameson is a witness in this case and Joe Hollingsworth is not. So what Joe Hollingsworth finds is surprising or not is really irrelevant. And what Dr. Jameson finds is surprising is relevant. So I move to strike your testimony, Counsel.

Q. (BY MR. HOLLINGSWORTH) Can you answer my question?

MS. WAGSTAFF: I'm not sure there's a question pending.

A. Yeah, could you repeat it, please?

Q. (BY MR. HOLLINGSWORTH) Well, my question is that the fact that the European regulators found a

background incidence and a range involving lymphoma in CD-1 mice to be zero to 32 percent in 2016 means that your statement that you're shocked that EPA would not take into consideration a zero finding in concurrent controls is really not so shocking?

MS. WAGSTAFF: Objection to form. Background incidence does not equal background range, so object to the form of the question.

A. What I was -- what I was trying to convey my surprise, rather than shock, I guess, is the fact that not only was there a low -- a low incidence in the controls, but the fact that my -- my surprise is the fact that you got a positive -- a statistically significant positive response in the high dose animals.

There was a high -- there was a statistically significant increase in the tumors, in malignant lymphomas in the high dose animals in this study, so that's a positive response. And you have a positive trend in the formation of these tumors in the mice. So two positive findings in this study in male mice for malignant lymphomas, and I'm just surprised the EPA would throw that out because you have two positive responses for malignant lymphomas in the male mice. Positive -- significant increase in the high

dose animals and a significant increase in the trend for the formation of this tumor in the animals. That's what I was saying.

Q. (BY MR. HOLLINGSWORTH) Well, you know that EPA, in addition to what you did statistically, did an adjustment for multiple comparisons, right, you read about that?

A. Uh-huh.

Q. And when they adjusted that finding for multiple comparisons in the high dose animal, the increased incidence in the high dose animal was not statistically significant, and that was the basis of what EPA did, and you knew that, didn't you?

MS. WAGSTAFF: Objection, argumentative.

A. I guess I knew that.

Q. (BY MR. HOLLINGSWORTH) Yeah. You didn't report that to the judge in this case, though?

A. No. Again, EPA did their risk assessment, and I was asked to do a hazard assessment and to give my opinion and that's what's in my report.

Q. Do you know how to adjust for multiple comparisons when you're doing studies involving long-term bioassays?

A. Do I know how -- I'm sorry, could you repeat?

Q. Do you know how to do an adjustment for multiple comparisons when you're doing a statistical significance analysis involving long-term bioassays?

MS. WAGSTAFF: Object to form.

A. I couldn't do it for you right here and now, no, but given the data, I could -- I could find a program to calculate that.

Q. (BY MR. HOLLINGSWORTH) Were you aware that the German regulators and the European regulators at EFSA reported a range of malignant lymphomas in female CD-1 mouse of between 4 and 32 percent?

A. I have to look at the -- their report to refresh my memory, but that sounds possible, yes.

Q. The fact that they -- the European regulators found a range for malignant lymphomas in control animals, that is, control CD-1 mice, in females of between 4 and 32 percent would not surprise you based on your overall experience in the field, right?

MS. WAGSTAFF: Objection, outside the scope of Dr. Jameson's testimony. He's not a statistician, he's testifying as a toxicologist.

A. Based on -- based on my experience, I think I've seen studies that have fairly high incidences in their controls. I don't know if it is

up to 32 percent, but I -- I could accept that level.
Q. (BY MR. HOLLINGSWORTH) You're referring to incidence of malignant lymphoma in mice?
A. Lymphoma in mice.
Q. Okay. Is it fair to state that there's a high variability of lymphoma, spontaneous lymphoma in CD-1 mice generally?
A. Well, based on the range that you gave me there, I would -- I would think that that's possible.
Q. EFSA considered this -- that is the European regulators, the European Food Safety Agent considered this same study you're opining about as showing no carcinogenic effect, true?
MS. WAGSTAFF: Objection, misstates the report.
A. I think for the purpose of their risk assessment, that's what they concluded, but, again, they were doing risk assessment and I was -- I was asked to do, and I did a hazard assessment for glyphosate, and so it's apples and oranges.
Q. (BY MR. HOLLINGSWORTH) Well, EFSA's statement that there was no carcinogenic effect comes from its conclusion on pesticide peer review, right?
MS. WAGSTAFF: Object to form.

A. But they were doing their risk assessment. My understanding is they were performing a risk assessment.
Q. (BY MR. HOLLINGSWORTH) Okay. The fifth mouse study is the Swiss albino mice study that I said I was going to ask you about, Dr. Jameson. Do you remember that?
A. Yes, sir.
Q. This was a company sponsored study by a company called Feinchemie, F-e-i-n-c-h-e-m-i-e in 2001?
A. Uh-huh.
Q. And I think the lead or one of the lead investigators was Kumar, right?
A. Yes.
Q. Do you have that study in mind?
A. Yes, sir.
Q. Have you read the conclusions of the authors of that study, I mean, the investigators of that study?
MS. WAGSTAFF: Object to form.
A. As I recall, I think this is -- I can't remember if I did or not. This is one of the studies where there wasn't a whole lot of original data from the lab available to me for -- to review. So I don't

know that I had a copy of their final report, to be honest. I know I did have tumor tables to look at and I looked at the tumor tables, and then I went to the Greim paper and compared the information in there and got a lot of information from the Greim paper.
Q. (BY MR. HOLLINGSWORTH) Did you -- are you sure you read anything other than Greim?
A. For the Kumar?
Q. Yeah.
A. Yeah, I had some of the -- some of the tumor tables from Kumar.
Q. Okay. Did you read the pathology report?
A. I don't believe I had access to the pathology report.
Q. Did you read the author's -- I shouldn't say author's -- the veterinarian pathologists' conclusions about the Feinchemie study?
A. Well, I don't have the pathology report, so. . .
Q. Okay. Did you know that the authors concluded that there were no compound-related neoplastic lesions in this study on mice, Swiss albino mice?
A. Like I said, I didn't have -- I didn't

have excerpts -- I didn't have the study reports, so I -- I did not read that -- could not read that.
Q. Did you ask plaintiffs' counsel to give you a copy of the study report?
A. I -- like I said before, I asked the plaintiffs' counsel to provide me with all the information that they had available to them and that is -- I'm sure that's what they did. So any of the information that was made available to me, I reviewed.
Q. So you didn't read the full data from this study by Kumar, Dr. Jameson?
MS. WAGSTAFF: Object to form.
A. I said I had the tumor tables that I could refer to and the Greim -- and the Greim paper that had a description of the -- of the study and the details of the study in that.
Q. (BY MR. HOLLINGSWORTH) Does your report refer to anything more than just Greim?
A. It refers to the --
MS. WAGSTAFF: Object to form.
A. I think Greim is the only -- only reference I have for this.
Q. (BY MR. HOLLINGSWORTH) And you're looking at page 24, right?
A. Wait a minute. Hold on.

Q. Greim is the only source you refer to; isn't that right, Doctor?
A. No. I also refer to some Tier II summaries from the Greim --
Q. Where is that, sir?
A. Okay. In the -- on page 24.
Q. Okay.
A. In about the fifth or sixth line down talking about the --
Q. Okay.
A. -- incidence as well as above the historical rate, and that particular reference is 87, which is the Tier II summaries for glyphosate carcinogenicity studies from Greim. And then a little bit further down, I think I say it is referring to the claim of a viral infection in the colony of these animals. I refer to the Kumar summary table 20 and 21.
Q. Okay. The Kumar summary table that you just mentioned, who gave you that?
A. That had to be provided to me by counsel.
Q. Okay. But counsel didn't provide you with the pathology report that Dr. Kumar prepared?
MS. WAGSTAFF: Object to form.

A. I do not -- no, I don't believe they did.
Q. (BY MR. HOLLINGSWORTH) Okay. Now, have you read recently the reevaluation of the Swiss albino mouse study?
A. I'm not -- I don't know what you're referring to.
Q. I'm referring to a report by -- I think his name is Dr. Klaus Weber, W-e-b-e-r. It's called reanalysis of the Kumar study and it's dated January 23, 2017.
A. I'm not familiar with that, no.
Q. Okay.
MS. WAGSTAFF: Counsel, it's 1 o'clock. What do you want to do?
MR. HOLLINGSWORTH: Okay.
MS. WAGSTAFF: I mean, if you want to finish the Kumar study, if you have a few more minutes, or do you want to break?
MR. HOLLINGSWORTH: Doesn't matter to me. We can break now.
MS. WAGSTAFF: Okay.
THE VIDEOGRAPHER: Going off the record. The time is 1:00 p.m.
(Recess taken, 1:00 p.m. to 2:06 p.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 2:06 p.m.
Q. (BY MR. HOLLINGSWORTH) Okay. Dr. Jameson, we were talking before lunch about the Kumar study, do you recall that?
A. Yes, sir.
Q. That's the 2001 mouse study and it's the fifth of five mouse studies that you considered?
A. Uh-huh.
Q. And the sponsor was Feinchemie Schwebda, who I hope someone spelled for Tracy, because I can't spell that. But this was the study -- this was the study on Swiss albino mice; is that right?
A. Yes.
Q. And I had already asked you about the study investigator's conclusion in that study. Excuse me.
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) And I was going to ask you if you knew whether this study was submitted to EPA, U.S. EPA?
A. Yes, it was.
Q. And are you aware that EPA did not evaluate the study because of the confounding factor of the presence of the viral infection and -- and

other infections?
MS. WAGSTAFF: Objection.
Q. (BY MR. HOLLINGSWORTH) In the -- in the study animals.
A. I -- I read the EPA report that said that based on information they received, and I think it was based on information that they had been provided in the Greim report that because they assumed that there was a viral infection in the colony, that they thought the study was invalid, however, I think I've indicated in my report that in my review of the particular study, it's not clear whether or not a viral component may have contributed to the incident value reported in the lower survival seen in the high dose in the study.
I had access to an internal Monsanto e-mail, among the authors of Greim, that would indicate there was no viral infection in the mouse colony during the study.
Further, if you look at the Greim publication, Greim reports that this study is GLP and OECD compliant, so I thought this was a very acceptable study to consider, so that's why I included it in my evaluation.
Q. Now, you were reading from a document

that you have in your hands in front of you. What is that?

A. This is my report.

Q. Okay. In fact, you agree that there's a possibility of contamination of this or confounding of the results of this study by viral infection; isn't that right?

A. From the materials that I had to review this study and the documents that I reviewed from this study, I have no reason to think that there was a viral infection in the colony and that -- in my opinion, this is a -- is a sufficient study and not compromised in any way by a viral infection.

Q. Okay. So you don't agree with me that you agree that there's a possibility of a viral infection that confounded this study?

A. I'm sorry, you're going to have to make that question a little more clearer. I think I heard a couple of double negatives in there or something.

Q. Okay. So you -- you -- you've stated that you did not agree in your expert report that there was a possibility of confounding of this report by viral infections?

A. Well, in any given situation, there's always a possibility of something happening.

Q. But that's not what I asked you.

A. Based on my evaluation of the information I had that from the -- from the data that was obtained from the testing laboratory itself in the Monsanto document that I looked at, that was made available to me, there was no indication of a viral infection in this particular colony.

In addition, Greim published in his paper that he felt that the study was GLP and OECD compliant. So from that standpoint, I felt this was -- this study was sufficient to consider for my evaluation and it was not compromised by a viral infection.

Q. Well, the Office of Pesticide Programs disagrees with you, right?

A. In their report, they discounted it and it was mainly because of a statement in -- I believe a statement in the Greim publication that implied that there may be a viral infection, but my evaluation of the available information does not point to a viral infection at all, so I feel it's an adequate study to consider.

Q. Do you agree with the statement that Murine leukemia viruses are also a common cause of lymphoma --

MS. WAGSTAFF: I will object.

Q. (BY MR. HOLLINGSWORTH) -- in many strains of mice?

MS. WAGSTAFF: Sorry. I will object to the counsel is reading from a 300-page document and if you'd like Dr. Jameson to opine, I would request the document be given to him.

Q. (BY MR. HOLLINGSWORTH) Can you answer my question?

A. I mean, you're reading that from an EPA document, but --

Q. Yeah.

A. I'd really like to see in what context that statement is being made before I comment on it.

Q. Okay. You know that EPA excluded from consideration this Kumar albino mice study due to the presence of a viral infection in the colony?

MS. WAGSTAFF: Object to form.

A. What I can state is in their report, that's what they said -- that's the reason they gave for not evaluating it. In my evaluation of the study, I found no evidence that there was a viral infection in this particular colony, and this was based on documents that I saw coming from the principal investigator at the laboratory who said he was not --

he did not feel there was a viral infection in the colony. So I thought there was no reason to discount this study, so I included it in my evaluation.

Q. (BY MR. HOLLINGSWORTH) Did you read the individual animal reports from the pathology report?

A. I did not have the pathology report for this study, but I did have animal tumor tables.

Q. Did you ask anyone for the pathology report?

A. I asked for all of the -- as much -- for all the information that plaintiffs' counsel had available for this particular study, and I'm confident they provided me with all the information they had.

Q. Have you seen a reference to the existence of skin lesions and bacterial infections in individual animals in this study?

A. I don't recall seeing that, no.

Q. You'd agree that if there was a viral infection or some kind of other infection in this colony, that it might confound the results of the -- and the statistical analysis of this study, true?

A. My evaluation of all the documents I could find relating to the study indicated that there was no viral infection in the colony, so in my opinion, and my past experience in evaluating animal

bioassays, I saw no reason to discount the study. There was no evidence that there was a viral infection, so I think it's perfectly -- this is a good study and that's why I considered it in my evaluation.

Q. Have you read what the U.S. EPA's Office of Pesticide Program says about this study?

A. The document you have in your hand, I have read, yes.

Q. Okay. Have you read what EFSA said about this study, the European regulatory agency?

A. I remember reading the EFSA report. I can't recall exactly what it said. I'd have to look at the report to -- to tell you what -- what exactly is said about that study.

Q. Do you recall that EFSA said that this animal study by Kumar was not acceptable due to viral infections that could influence the survival as well as tumor incidence, especially lymphomas?

A. I -- I -- as I said, I -- I don't absolute -- I'm not absolutely certain, but that sounds like what I remember reading from the EFSA study. I -- you know, I have no idea other than perhaps what they read in the Greim report for their rationale for discounting the study. My evaluation of the data and the documents available to me from this

report shows that there was no viral infection in the colony. The principal investigator of the study said in a memo or a document that I read that in his opinion, his colony had no viral infection, and so I saw no reason not to accept this study. It's a perfectly acceptable study.

Q. Aren't there publications in the general background literature on long-term animal bioassays and their interpretation that state that the incidence of lymphoma due to the effect of viral contamination of a colony can increase the amount of malignant lymphoma found in the animals?

A. There is publications to that effect. In fact, in my experience, my long experience with the National Toxicology Program and its animal bioassay studies, we have conducted studies where -- where really -- we could not ultimately evaluate because of infections in the colony, because of poor animal husbandry. It happens. It happens not frequently, but it does happen, and it's just part of doing toxicology, part of doing toxicology studies, so there are studies that have been done that are compromised because of different viral infections and it's been documented in the literature. Sorry.

Q. Right. Thanks. Are you done?

A. Yes.

MS. WAGSTAFF: Just answer the question he asks.

THE DEPONENT: Sorry.

Q. (BY MR. HOLLINGSWORTH) Is it fair to state that the higher incidence of lymphoma that other -- that other authors have seen from the effect of virus in a colony is due to the effect of the virus on the animal's immune system, which leads to more lymphoma?

A. Sorry. Would you repeat that? Sorry.

Q. Would you agree that the background literature states that the higher incidence of lymphoma that is seen in experimental animal colonies that have been infected by viral infections is due to the adverse effect on the animal's immune system?

MS. WAGSTAFF: Object to form.

A. I -- I don't -- the question is not clear to me, so I -- I can't comment. I don't know --

Q. (BY MR. HOLLINGSWORTH) What's unclear about the question?

A. You're saying about something -- did you mention something about historical data or control incidence? I'm sorry.

Q. No, I was just saying the background

publicly available information.

A. Oh, the information that's available?

Q. Yes.

A. Okay. Would indicate? I'm sorry.

Q. Would indicate that where virus has infected an animal colony, the increased findings of lymphoma, malignant lymphomas in those colonies is caused by the effect on the animal's immune systems?

MS. WAGSTAFF: Object to the form.

A. That could be one of the effects.

Q. (BY MR. HOLLINGSWORTH) Okay. In the mouse, the malignant lymphoma findings are mediated by the immune system of the mouse in part, aren't they?

A. It plays a role in the formation of the lymphoma.

Q. Did the mouse have the same kind of immune system, the CD-1 mice or the Swiss albino mouse, as humans?

A. I would not say yes to that, no.

Q. Okay. So you accepted this study as proper and appropriate for evaluation even though EFSA and EPA did not, right?

A. That's correct.

Q. And you state that the formation of malignant lymphoma in male and female mice occurred in

the Kumar study, right?
A. Yes.
Q. Okay. And you say that there was an increased incidence of renal cell adenomas in male mice in this study, correct?
A. That's correct.
Q. Are you aware of any literature that says that renal cell adenomas are affected by -- by -- by the infection of a mouse colony by viruses?
A. Sitting here today, I don't -- I don't recall any, but that's not to say there isn't any.
Q. You didn't consider the historical control rate in both males and females in Swiss albino mice, did you?
A. For this particular study, I didn't indicate that, no, I -- I did not.
Q. Were you aware that the range of malignant lymphoma observed by the same laboratory during the same time frame was 6 to 30 percent for males?
A. I don't remember that, no.
Q. Do you recall that the range of malignant lymphoma observed by this same laboratory during the same time frame was 14 to 58 percent for females?

A. No, my -- the data that I had, as I indicated in my report, that the incidence of malignant lymphoma in the high dose male was double the historic rate reported to be 18 percent from males and for high dose female mice was well above the historical rate of 41 percent, and the reference I used for that was the Tier II summaries for glyphosate carcinogenicity studies from Greim, 2015.
Q. That's Greim, Greim at page 201?
A. I didn't put the page number.
Q. Doesn't Greim state that the -- that the malignant lymphoma observed by this same laboratory involving other studies in the same Swiss albino mice was between 6 and 30 percent for males?
A. This was taken from the Greim Tier II tables that I -- that I had access to. That's the reference that I used. I wasn't using the Greim paper itself.
Q. Okay. You're aware that Dr. Portier found no statistically significant trend from this data involving malignant lymphoma, aren't you?
MS. WAGSTAFF: Objection, misstates testimony.
A. I wasn't -- I'm not familiar with -- with what Chris reported.

Q. (BY MR. HOLLINGSWORTH) You still haven't looked at his amended report?
A. This is from his expert report?
Q. Yes.
MS. WAGSTAFF: Objection.
A. To be honest with you, I skimmed through it, but I didn't read it in detail.
Q. (BY MR. HOLLINGSWORTH) Okay. It's always good to be honest.
MS. WAGSTAFF: Objection, argumentative. Have you not been honest today, Dr. Jameson?
THE DEPONENT: I hope I've been.
MR. HOLLINGSWORTH: You can ask him that when you have your chance.
MS. WAGSTAFF: You just suggested he hasn't been honest.
MR. HOLLINGSWORTH: He said, well, "to be honest with you." I thought that indicated to me he wasn't being honest with me previously.
MS. WAGSTAFF: Are you kidding?
MR. HOLLINGSWORTH: That's what I thought.
MS. WAGSTAFF: I'm glad I corrected the record.
THE DEPONENT: I've got to remember not

to editorialize, I guess.
MS. WAGSTAFF: Have you been honest today?
THE DEPONENT: I have been honest to the best of my ability.
MS. WAGSTAFF: Okay.
Q. (BY MR. HOLLINGSWORTH) So has your disagreement with EPA and EFSA about this Swiss albino mouse study by Kumar and the conclusions you've reached been published and peer reviewed anywhere?
MS. WAGSTAFF: Object to form.
A. They've only been published in my report, my expert report, that I submitted for this litigation.
Q. (BY MR. HOLLINGSWORTH) Did you talk to Dr. Portier about this Kumar study?
A. No, I did not.
Q. Okay. Okay. Sir, you -- you also reviewed and include in your report as a basis for your opinion the Lankas, L-a-n-k-a-s, Dr. Lankas' 1981 rat study.
A. Okay.
Q. And you concluded that the incidences of testicular interstitial cell tumors was within the -- I'm sorry. Let me -- let me -- let me rephrase

that.

Did you read the authors of the Lankas study or the investigator's report of what their conclusions were from this study? Do you understand my question?

A. Yes, I'm just trying to find where I am. Bear with me. Sorry. So you asked if I could -- if I read the report?

Q. Yes. We're on 1981 Sprague-Dawley rat study that was sponsored by Monsanto.

A. For this particular report, I think I did have the report to review -- to to read.

Q. Did you read the pathology report within the study?

A. If it was in the report that I had, I did read it.

Q. The report was four or 5,000 pages?

A. Four or 5,000?

Q. Yeah. The report by the laboratory.

A. I know it was long, but the report -- the document I had wasn't that long. It was probably about six or 700 pages.

Q. Who gave you the document that you read?

A. It was provided by counsel.

Q. Okay. Were you familiar with that study

before you read it in preparation for this litigation?

A. I'd have to go back and check. I believe -- I believe this was one of the studies that was reviewed as part of the IARC monographs. But that review was based on the EPA reports for their review of that study.

Q. But your review was based on a different -- different dataset than what IARC had?

A. I had more data to look at than what was available. As I indicated for the IARC review, as I recall, it was EPA documents that were made available to -- to the IARC that we used in our review.

Q. Since you read the report, you're aware that the investigators, including Dr. Lankas and others, wrote a conclusion which was that the interstitial cell tumors, that you refer to in your expert report, were within the normal biological variation observed for tumors at this site in this strain of rat, and, therefore, they said that the testicular tumors were not compound related, true?

MS. WAGSTAFF: Objection to counsel testifying again.

A. Oops, looking at the wrong thing. Sorry. Okay. In my report --

Q. (BY MR. HOLLINGSWORTH) What page are

you looking at, sir?

A. This is -- okay. I'm looking on page 25.

Q. Okay.

A. Okay. What I'm reading -- at the top of page 25, I state in my report, that the incidence of interstitial cell tumors in the testes in the high dose animals in this study is almost twice that seen in the range of tumors, 3.4 percent to 6.7 percent in control animals, historical controls in five contemporary studies, and I reference the Greim Tier II tables.

Q. You didn't answer my question. My question was whether you were aware of the conclusion of the original investigators of this study that the interstitial cell tumors of the testes, which you were talking about were, quote, within the normal biologic variations for tumors at this site in this strain of rat, unquote?

MS. WAGSTAFF: Again, I would request that you give the document to Dr. Jameson if you're quoting from something so he can see the context of the document. And without that, it's hard to opine.

A. I'd like to see the report, but I don't remember seeing -- reading that.

Q. (BY MR. HOLLINGSWORTH) You don't remember reading that the authors of the report looked at the interstitial testicular tumors in particular and said that they were within the normal biologic variation observed for tumors at this site in this strain of rat?

MS. WAGSTAFF: Hang on. We all know that everyone has looked at dozens and dozens, if not hundreds, of reports. You mentioned earlier this one was 4,000 pages. You have something in your hand that you're reading from. Why don't you just let Dr. Jameson look at it.

MR. HOLLINGSWORTH: I would just like to know if he can answer my question whether if he was aware of that original conclusion by the authors or not when he started preparing his opinion in this case.

MS. WAGSTAFF: This is not a memory test.

A. I -- I -- like I said, I don't recall reading that. In looking at the documents I had.

Q. (BY MR. HOLLINGSWORTH) Do you recall that the authors, the actual investigators of this report from 1981, the veterinary pathologist who did the report said that the gross and microscopic changes

that otherwise occurred besides the interstitial cell tumors occurred sporadically in the control and/or treated rats and were considered unrelated to administration of glyphosate?

MS. WAGSTAFF: Same objection.

A. I remember reading something to that effect.

Q. (BY MR. HOLLINGSWORTH) Did you tell the judge about the conclusions of the original investigators of this report in 1981 that you're -- opining about?

MS. WAGSTAFF: Objection, he wasn't retained to tell the judge about other people's conclusions.

A. I -- I -- as I've indicated in previous questions about this same issue, I was asked to give my opinion of the data and do a hazard identification exercise on the data for the exposure of glyphosate and glyphosate formulations and its association with non-Hodgkin's lymphoma.

As part of that evaluation, I looked at these animal studies. So what I did was gave my opinion as to what the adequacy of the studies and the results of the studies, so what I was asked to do was give my opinion, and that's what I did in this report.

Q. (BY MR. HOLLINGSWORTH) You had in -- in this case you had the entire report, you said, you had seven or 800 pages?

A. I had a large document to look at, yes.

Q. Did you look at what the authors' conclusions were about the carcinogenicity of the --

A. I'm sure I did if I -- from the full report. I would read what the authors or investigators would have said.

Q. Do you think that a fair scientist should have reported to the judge in this case what the original investigators said about the conclusions they got from their own study?

MS. WAGSTAFF: Objection, calls for a legal conclusion and asking him what's fair to report in a legal context is just inappropriate.

MR. HOLLINGSWORTH: I'm asking in a scientific context.

A. Again, as I --

MS. WAGSTAFF: He's not -- it's a legal conclusion.

A. Sorry. As I stated before, this is not unlike what I had done in the past and what other scientists, toxicologists, pathologists, epidemiologists, what have you, it's not unlike what

they are asked to do is to be given a dataset and gave their opinion of what the dataset says. That's what I was retained to do. That's what I did when I reviewed these studies and that's what I wrote in my report was my opinion.

Q. (BY MR. HOLLINGSWORTH) Did you know that EPA had reviewed this study?

A. Yes, sir.

Q. And did you know that EPA considered it to not show a carcinogenic effect in any of the treated groups of animals?

MS. WAGSTAFF: Object to form.

A. Again, the EPA did their risk assessment of this particular -- of glyphosate from this particular study, and based on that their criteria for risk assessments, evidently, they decided that these interstitial cell tumors were -- were not relevant to their exercise of doing a risk assessment.

I am doing or I did a hazard identification. For the purpose of the hazard identification, it's appropriate to consider these tumors, these tumors caused -- the glyphosate caused the formation of these tumors in the rats, and, so, therefore, it's an animal carcinogen and a potential human carcinogen.

Q. (BY MR. HOLLINGSWORTH) Didn't you say that this study was not valid for reviewing purposes because the high dose in these rats was only 300 parts per million?

A. No.

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) Did you review summary animal data and individual animal data in this report or I should say this study report?

A. Did my report?

Q. Did your review --

A. Did my review?

Q. -- include summary animal data and individual animal data?

A. You're going to need to define "summary" versus "individual" for me, please.

Q. Well, I just -- I think summary animal data and individual animal data as it relates to a pathology report from a long-term bioassay is standard terminology. You don't know what that means?

A. That's not what you asked me. You didn't say anything about a pathology table.

Q. I said, did you review -- did your review include summary animal data and individual animal data from this report --

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MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) -- by these investigators.
A. In my report, no, not specifically my report.
Q. (BY MR. HOLLINGSWORTH) You're aware that these interstitial cell tumors in the testes are known to be age related, right?
A. There are a number of different tumors in experimental animals as in humans that the incidence of the tumors increase as the animal ages.
Q. I'm --
A. So --
Q. I'm talking about testicular tumors in particular.
A. Well, I mean, just like -- just like you and I will get prostate cancer if we live long enough, it is the case in rats that the older they are, the more likely it is that you may see testicular tumors in the aging male rats.
Q. Did you observe when you reviewed the data that you reserved about the Lankas 1981 rat study that the survival in the control group was significantly decreased from survival in the high dose group?

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A. In this study?
Q. Yeah.
A. According to my report, there was no treatment-related effect on body rate or survival at any dose level in this study, so I --
Q. So you disagree with that?
A. Based on what I have written in my report, I -- I can't agree with that.
Q. Okay. You don't remember that for the 18-month-old males eight control animals had died and only one high dose animal had died?
MS. WAGSTAFF: Objection, again if you want to show him the study, that would help refresh his memory.
A. Again, I don't -- I don't -- I can't speak to that because I -- I didn't memorize the interim death rates in this particular study. I need to see the tables and what the -- and what the final survival data looked like as well.
Q. (BY MR. HOLLINGSWORTH) Is the -- is the survival at 18 months not significant to you in connection with a 24-month chronic bioassay in rats?
A. Again --
MS. WAGSTAFF: Object to form.
A. -- I can't comment without looking at

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the data and looking at all of the data.
Q. (BY MR. HOLLINGSWORTH) You don't remember that the long-term -- the high dose animals had -- had one-eighth the number of deaths that the control animals who weren't fed any glyphosate had?
MS. WAGSTAFF: Object to form.
A. Again, that is contrary to what I have written in my report.
Q. (BY MR. HOLLINGSWORTH) Okay.
A. I'd have to look at the full report, again, to see what you're talking about.
Q. Okay. Well, if the high dose males out-survive the control males and you're considering a tumor like testicular tumor in rats, it wouldn't be surprising that there would be a higher rate of testicular cancer in the high dose group, would there -- would it?
A. All I can say is what I have stated in my report was there was no significant difference in survival in any of the dose groups, so. . .
Q. Okay. Now, you also say that in this study that there was an increased incidence of pancreatic islet cell adenomas, correct?
A. Right.
Q. Pancreatic islet cell adenomas, and the

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incidence was zero, five, two, two, according to your report, correct?
A. Correct.
Q. And that doesn't demonstrate a dose response, does it?
A. No, it doesn't demonstrate a dose response, but it demonstrates a statistically significant increase in the low dose animals, so that's a positive response caused by glyphosate in this study.
Q. Zero, five, two, two is not a statistically significant difference, is it?
MS. WAGSTAFF: Object to form.
A. It is not a trend, but it's a significant increase in the low dose animals compared to the controls by a pair-wise comparison. And that comparison is statistically significant.
Q. (BY MR. HOLLINGSWORTH) Now, the IARC monograph reported that there was no evidence in this study of progression from adenomas to carcinomas for the pancreatic islet tumors, true?
A. That's what was reported.
Q. And you have written in the past that the evidence of progression from benign to malignant to neoplasia is an important factor to be considered

in rodent bioassay evaluations; isn't that right?
A. That sounds like something I would have written awhile ago.
Q. So as you sit here today, do you disagree with that?
A. Disagree with again? I'm sorry.
Q. Have you changed your view on that issue now?
MS. WAGSTAFF: Object to form.
A. On the issue?
MR. HOLLINGSWORTH: Yeah.
A. Would you repeat?
Q. (BY MR. HOLLINGSWORTH) You said in answer to the question I asked you just previously, you said it sounded like something that I would have written long ago. And my question -- follow-up question on that is are you suggesting that you've changed your opinion on that issue now?
A. And the issue is?
Q. That the evidence of progression from benign to malignant neoplasia is a factor that should be considered in evaluating rodent bioassay data?
A. I agree it is a factor that is as it should be considered in rodent bioassay studies, but it is not necessary to have that progression in order

to say that there's a positive effect of tumor formation.
Q. Did you tell the Court that you had published before the fact that it's important to consider evidence of progression for benign to malignant neoplasia in evaluating rodent bioassay data?
A. Did I tell the Court?
Q. Did you tell the Court in your report that?
A. I don't -- I don't recall putting that in my report, no.
Q. You know that the original investigators who were the pathologist, the experimental pathologists that evaluated the histopathology from the study determined that this study did not produce any compound-related changes due to glyphosate administration, true?
MS. WAGSTAFF: Object to form.
A. That sounds like what they may have written in the report.
Q. (BY MR. HOLLINGSWORTH) I've asked you about this before, but the high dose here was 300 parts per million, right?
A. 300, that's correct.

Q. And other studies in rats involving glyphosate that you reviewed had high dose administrations of 10,000 parts per million or 30,000 parts per million or up to 3 percent of the rat's total diet, right?
A. That's correct.
Q. And none of those studies had any evidence of interstitial testicular -- interstitial cell testicular carcinoma, did they?
A. Not that I recall.
Q. You didn't report a single one?
A. That's not to say that there wasn't some of those tumors found in one or two of those studies, but it wasn't significantly different than the controls, so I didn't include it in the report.
Q. With given those high doses of 10,000 or up to 30,000 or 3 percent of the animal's total diet and no interstitial cell testicular tumors from any of those studies, don't you think that's biologically significant in the evaluation of the overall carcinogenic effect of glyphosate on rats?
MS. WAGSTAFF: Object to form, misstates evidence.
A. What -- again, what I've been doing or do in this report is a hazard identification, so I

take the studies and evaluate them individually as to their adequacy and if they showed a positive response. In this particular study, glyphosate was given to rats and the male rats got interstitial cell tumors, so for this particular study, there was a significant increase in interstitial tumors in the male rats, so therefore, glyphosate caused these tumors in male rats and from that, it is an animal carcinogen and a potential human carcinogen.
Q. (BY MR. HOLLINGSWORTH) That's not exactly my question, Dr. Jameson. My question is whether the fact that the later rat studies in which rats in the high dose groups were fed up to actually 40,000 parts per million in their diet, but who, when evaluated, had no testicular carcinoma caused you to rethink your conclusion about testicular cancer in a study where the high dose animals only received 300 parts per million in their diet?
MS. WAGSTAFF: Object to form and asked and answered.
A. I've already answered what my thought is on that.
Q. (BY MR. HOLLINGSWORTH) Okay. That didn't cause you to change your -- to go back and question your opinion --

A. No.
Q. -- about the Lankas cell -- Lankas rat study when you saw that rats in all the other rat studies had been fed in the high doses 10 to 40,000 parts per million, whereas Lankas only -- the Lankas study only fed the high dose rats at 300 parts per million?
A. Right. But not knowing the mechanism of action or how the high doses affected the metabolism or absorption or the immune system of the animals, it's -- you know, all these different variables have to be taken into consideration. But, no, it didn't.
Q. Is there any evidence from the rat studies that the immune systems of these rats in these nine studies that you looked at -- I'm sorry, seven studies that you looked at were affected?
A. I don't recall. I'd have to go back and look at the studies. I don't -- I don't know if they did any studies to investigate the effect on the immune system.
Q. Have you --
MS. WAGSTAFF: Can you guys put it on mute, please.
Q. (BY MR. HOLLINGSWORTH) Do you recall your review of the 1990 rat study? It's another

Sprague-Dawley rat study, I believe, by Dr. Stout and others.
A. Dr. Stout?
Q. Yes, S-t-o-u-t.
A. Uh-huh. Okay.
Q. The original investigators in that study, which included Dr. Stout and others, concluded that an oncogenic effect or carcinogenic effect was not seen or observed in that study at all; isn't that right?
A. I remember -- I recall that that's what they said in their report.
Q. And that full study report, including the pathology report, was provided to you by plaintiffs' counsel, right?
A. I did get a study report for this. And I know the report also included tumor tables. So I reviewed all the information that was in the report and tumor tables.
Q. The -- there was a pathology report in this overall study report as well, too, true?
A. Okay. I believe there was.
Q. Yeah. And there were individual animal data and lots of summaries on various tumors that were found when these animals died or were sacrificed,

right?
A. Correct.
Q. And you read all that stuff?
A. I looked through all of that, yes.
Q. Did you tell the Court in your report what the individual authors or investigators actually reported about the tumors that were observed in this study on serial sacrifice or at the time of mortality before sacrifice or at final sacrifice at 24 months?
MS. WAGSTAFF: Object to the form of the question.
A. I concentrated on the final sacrifice data, the terminal sacrifice data and any data that any -- any pathology that had been conducted on the animals that had died earlier as included in the tumor tables.
Q. (BY MR. HOLLINGSWORTH) You know that this report was submitted to EPA, true?
A. That's correct.
Q. And you know that EPA published a report about this rat study in 1990 in connection with the registration of glyphosate, right?
A. Correct.
Q. And the EPA concluded that there were no treatment-related neoplastic or carcinogenic or cancer

changes in these animals in any dose group, true?
A. That's what they reported as a result of their risk assessment, but, again, I did not do a risk assessment, I did a hazard identification.
Q. Now, the high dose group in this study received 20,000 parts per million?
A. Correct.
Q. Or 2 percent of their total diet of glyphosate?
A. Correct.
Q. And Lankas and the other authors reported that out in the reports that you read about this study, true?
A. I'm sorry, who?
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) I'm sorry, excuse me. We're talking about Dr. Stout now. I apologize.
A. Right.
Q. Dr. Stout reported in various places in this report that the top -- the high dose group had received 20,000 parts per million of glyphosate in their diet and that compares to the 300 parts per million high dose group that -- that we talked about from the Lankas study in 1981, right?
A. Correct.

Q. And are you aware that the incidence of testicular interstitial cell tumors from Dr. Stout's study in 1991 on the same strain of mouse, Sprague-Dawley. Sprague, S-p-r-a-g-u-e dash Dawley, D-a-w-l-e-y, rats was two, zero, three, two?

A. Two --

Q. Two, zero, three, two.

A. Okay.

Q. You're aware of that, right?

A. That was in the report.

Q. So this study didn't repeat the testicular interstitial cell tumors or replicate the study done by Lankas in 1981, did it?

MS. WAGSTAFF: Object to form.

A. Well, no, I mean, the -- the Lankas study was done at much lower doses.

Q. (BY MR. HOLLINGSWORTH) Isn't it biologically sound to expect the higher dose animals to have more testicular tumors than the lower dosed animals? Isn't that what biologic significance means to an experimental pathologist?

MS. WAGSTAFF: Object to form.

A. Well, I mean, you would -- you would -- you would expect to see more tumors at higher doses, but that doesn't preclude the fact that at lower

doses, you may be seeing different biological events happening in the animals at lower doses than -- than what happens in the higher doses. The higher doses could be blocking a particular type of activity, so the fact that you see something in lower doses that you don't see something in higher doses is -- is seen in -- in toxicology and carcinogenicity studies.

Q. (BY MR. HOLLINGSWORTH) Has anyone published a study, a peer-reviewed study anywhere on the planet that says the effects of glyphosate at lower doses may be more virulent in terms of cancer than the effects of -- at higher doses in rats?

A. I'm not aware of any, no.

Q. None of the other six rat studies besides the 1981 Lankas study had any increased incidence of testicular interstitial cell tumors, did they?

A. No. No significant increase in those tumors, correct.

Q. In this -- in this 1990 study by Dr. Stout and others, you report in your expert witness report an increased incidence of pancreatic cell adenomas, true?

A. Correct.

Q. And that's in the low dose males, right?

A. In the low dose males, correct.

Q. And you can see that there's no apparent progression to carcinoma in these lesions?

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) True?

A. I'm sorry, say again. I was reading something.

Q. You can see that there's no apparent progression to carcinoma from your review of the information on these lesions?

A. In these studies there was no apparent progression to the carcinoma, correct.

Q. So the adenoma did not progress to carcinoma?

MS. WAGSTAFF: Object to form.

A. I'm sorry, say again.

Q. (BY MR. HOLLINGSWORTH) The adenoma in these pancreatic islet cell lesions, the adenomas, did not progress to cancer in any of these animals?

A. It appears that way, yes.

Q. And you have written that that is a significant effect to be reviewed in connection with evaluating rodent bioassay data, true?

MS. WAGSTAFF: Object to form. He testified moments ago differently, but. . .

A. That progression is important?

Q. (BY MR. HOLLINGSWORTH) Yes.

A. Well, if you see progression, that's an important observation. But it's not necessary to -- to indicate that a particular material causes a tumor.

Q. So there was no progression from adenoma to something more virulent like carcinoma in the animals that were treated with glyphosate and who developed pancreatic islet cell adenomas, true?

A. That's correct in this.

Q. Are you aware that there was, in fact, a carcinoma found in the control group?

A. In this control group?

Q. Yes.

MS. WAGSTAFF: Object to form.

A. There was one carcinoma found.

Q. (BY MR. HOLLINGSWORTH) In fact, the only pancreatic carcinoma occurred in the control group in this study; is that right?

A. I'd have to go back and look. I don't have that information in my report, so I'd have to go back and look at the reports.

MS. WAGSTAFF: Once again, I mean, if you're asking him these sort of details, we would

request that you give him a copy of the report as this is not a memory test.

Q. (BY MR. HOLLINGSWORTH) There was also no -- no dose response that you could observe in these pancreatic islet cell adenomas that you saw in the treated groups, true? 8, 5, 7 is not a dose response, is it?

A. No, it's not a true dose response, but then, again, if you -- if you look at the incidence here, originally as reported, there was a statistically significant increase in the low dose animals, but if you read the EPA's evaluation of this particular study, the EPA performed additional analyses which they included the animals that were killed or died before 54 or 55 weeks, and during that particular evaluation, they found an incidence of one in 43 for -- these are for the pancreatic cell -- islet cell adenomas. They found one in 43 for the controls, eight in 45 for the low dose, which is also -- which is significant. Five of 49 in the mid dose and seven of 48 in the high dose, which now becomes significant.

So when the EPA reevaluated the studies, excluding the early deaths, you found a significant increase in tumors in both the low and the high dose

animals from this particular study for the pancreatic islet cell tumors.

Q. Assuming the control animal had a carcinoma, it's not surprising that that male died early, is it?

MS. WAGSTAFF: Object to form.

A. Well, you -- you can't argue one way or the other for that.

Q. (BY MR. HOLLINGSWORTH) Does that have biologic significance to you that the only animal in this study that had actual carcinoma was a control animal?

MS. WAGSTAFF: Objection. The doctor has asked to see the data and you're prefacing an entire line of questioning on an assumption that he would like to look at the report and determine the significance of it.

Q. (BY MR. HOLLINGSWORTH) Do you want to hear my question again?

A. Please.

Q. Would it have biologic significance to you that in a case where the control animal is the only animal that has actual cancer?

MS. WAGSTAFF: Object to form.

A. I'd have to look at the -- at the data

little more closely to give you an adequate answer to that. I'd have to see, you know, what time the animal -- what time, when the animal died, if it was an early death. If it was an early death, then there may have been something genetically wrong with the animal to cause it to be -- to have an early onset of a tumor like that.

Q. (BY MR. HOLLINGSWORTH) This --

A. I'm sorry.

Q. This result that you talk about in the male animals with respect to pancreatic islet cell adenomas was not replicated in the female animals, was it?

A. In this study, no.

MS. WAGSTAFF: Object to form.

Q. (BY MR. HOLLINGSWORTH) Yes. The pancreatic islet cell adenomas in the females was six, one, four, zero, right?

A. I'd have to look at the report to see what the incidence was.

Q. Well, if the -- if the incidence, in fact, was six, one, four, zero, that indicates there's no replication between the sexes in terms of pancreatic islet cell adenoma findings from the study, true?

A. Between the --

MS. WAGSTAFF: Object to form.

A. Between the males and the females?

Q. (BY MR. HOLLINGSWORTH) Yes.

A. Correct, as I indicated earlier, it's not unusual to see a different incidence or a significant incidence of a tumor in one sex and not in the other sex. That's -- that's found in a lot of different studies.

Q. (BY MR. HOLLINGSWORTH) If the pancreatic islet cell adenomas in the female rats is six, one, four, zero, it's true that the control animals had more pancreatic islet cell carcinomas in toto than any of the three control groups, true?

MS. WAGSTAFF: Object to form.

A. Okay. Well, the females had more carcinomas in them than the males, but then, again, that -- that is an instance where you might want to bring in historical control incidences to see what the historical incidence of pancreatic cell carcinomas in male and female rats are, so that you can make an evaluation of that.

Q. (BY MR. HOLLINGSWORTH) Okay. In the female rats, there were the -- the pancreatic islet cell adenomas were one, four, zero. And if there --

A. Pancreatic islet cell adenomas?
Q. Yes.
A. In the female rats?
Q. Yes. Control was six.
A. I don't have the data in front of me, so I'm just trying to keep up.
MS. WAGSTAFF: What -- I'll make about my 25th request today to please show him the data. You're asking him if he's memorized these random string of numbers that --
MR. HOLLINGSWORTH: Well, he's relied on Greim.
MS. WAGSTAFF: Of course he relied on Greim, but --
MR. HOLLINGSWORTH: It's right out of Greim. I'm asking if he remembers.
MS. WAGSTAFF: Do you think he's memorized it? You've got it right in front of him. It wouldn't be that hard to give him the data instead of trying to trip him up on numbers.
MR. HOLLINGSWORTH: I'm not tripping him up.
MS. WAGSTAFF: Just saying, I'd like the record to reflect that we've asked for the data to look at it about 25 times and you've refused every

time.
Q. (BY MR. HOLLINGSWORTH) You also note significant trends in three additional tumor types in this study, don't you, Doctor?
A. Significant trends?
Q. Yes.
A. In -- okay -- in which particular tumor sites?
Q. Hepatocellular adenoma.
A. Okay.
Q. Do you know of any study that says hepatocellular rates that are increased in treated animals in a long-term bioassay has a relationship to non-Hodgkin's lymphoma in humans?
A. The purpose of this study was to see if glyphosate caused cancer in the Sprague-Dawley rats. When glyphosate was given to the animals, it caused liver -- an increase in the trend in liver hepatocellular adenomas in the male rats. So, therefore, the exposure or treatment with glyphosate caused liver tumors in rats and, therefore, it's an animal carcinogen and a potential human carcinogen.
I am not aware of any -- anybody who has designed or conducted a study to investigate the association between hepatocellular adenomas in rats

and non-Hodgkin's lymphoma in humans or I'm not aware of anybody publishing any data or articles on that.
Q. Are you aware that -- are you aware that Dr. Portier has concluded that the increase in hepatocellular adenomas that you report in your expert report could be due to chance?
MS. WAGSTAFF: Object to form.
A. I -- I -- I don't recall that.
Q. (BY MR. HOLLINGSWORTH) Now, do you recall what the incidences were of follicular cell adenomas, which you say in your report based on this 1990 rat study by Stout were caused by administration of glyphosate?
MS. WAGSTAFF: Once again, another request to please provide the witness with the data.
A. Follicular cell?
MS. WAGSTAFF: It's not surprising you haven't memorized them.
A. Okay. Yes.
Q. (BY MR. HOLLINGSWORTH) Do you report what the incidences were of follicular cell adenoma?
A. No, when I was reading through my report, I noticed that I neglected to put the incidences in and that's a deficiency in the report that I need to correct.

Q. Did you look at what the -- in preparation for your testimony, did you look at what the incidence of thyroid follicular cell adenoma is as you report it to be in -- in your report?
A. Did I -- I'm sorry, did I do what?
Q. Did you look at the incidence of follicular cell adenoma? I'm sorry, did you look at the incidence of thyroid follicular cell adenomas in the four groups within this rat study?
A. In preparation for this?
Q. Yes.
A. I did not. No.
Q. Did you state in your report that the incidence of thyroid cell follicular cell adenoma is significant by pair-wise comparison?
MS. WAGSTAFF: Object to form.
A. I did. And the reference for that is there's an EPA report is where I got that information from. It's a glyphosate issue paper, evaluation of --
THE REPORTER: I'm sorry.
A. I'm sorry I read too fast. I'm so sorry. Glyphosate, it's EPA 2016, glyphosate issue paper. Evaluation of carcinogenic potential. And it's EPA's Office of Pesticide Program, September 2016. That's the reference I used in my paper. I

apologize, like I said, I noticed when I was reading through it last night, that I forgot to put the incidences in and that was my oversight and I will correct it.

Q. (BY MR. HOLLINGSWORTH) Okay. Sir, you're well aware that EPA after considering all the data within the Office of Pesticides Program actually did not consider the increases in pancreatic islet cell adenomas or carcinomas to be significant, aren't you?

MS. WAGSTAFF: Object to form.

A. Again, the EPA in performing their risk assessment and looking at these particular tumors in this study, evidently it did not meet their criteria for inclusion for the purposes of risk assessment.

I did a hazard identification, and in my evaluation for a hazard identification, this observation is significant. And so that's why I included it in my report.

Q. (BY MR. HOLLINGSWORTH) Did the EPA use a different statistical different method of analysis than what you used?

A. No, the statistics that I report here in my report come from EPA.

Q. And didn't the EPA also conclude that

that hepatocellular tumors that you refer to in your expert witness reports were not compound related?

A. Again, the EPA was doing their risk assessment, and evidently for the risk assessment, the -- these particular tumors did not meet their criteria for inclusion in their risk assessment or however, for the purpose of the hazard identification I did, these liver tumors -- I consider these liver tumors to be associated with exposure to glyphosate and, therefore, I included them in my report.

Q. You also said in your report that in this 1990 rat study by Dr. Stout, thyroid C cell tumors that you observed were related to treatment with glyphosate; isn't that right?

A. That's correct.

Q. And EPA -- EPA's Office of Pesticide Programs, after considering all the study data, concluded that the thyroid C cell tumors were not treatment related, that is not related to glyphosate, didn't they?

MS. WAGSTAFF: Object to form.

A. This is the same argument. The EPA were conducting a risk assessment. Evidently, the results for the thyroid C cell adenomas in the females did not meet their criteria for inclusion in their risk

assessment, that's why they did not consider them.

For the purpose of my hazard identification, I evaluated the increase in trends of these thyroid C cell adenomas in the females. It was sufficient and, therefore, I included it in my report.

Q. (BY MR. HOLLINGSWORTH) That increase that you talk about in thyroid C cell tumors, was not statistically significant by pair-wise comparison, was it?

A. It was significant for trend, but not pair-wise.

Q. Yes. EFSA looked at this data, too, didn't they?

A. I believe they did.

Q. And EFSA concluded that there was no evidence that the pancreatic islet cell tumors in this study were compound related or related to treatment by glyphosate, right?

MS. WAGSTAFF: Object to form.

A. Again, EFSA was doing a risk assessment, so evidently the data there did not meet their criteria for doing a risk assessment. That's why they discounted these tumors.

For my hazard identification, I felt it was showing that this trend was due to exposure to

glyphosate, so therefore, I included it in my report.

Q. (BY MR. HOLLINGSWORTH) Do you think that you had as much data about this report as EPA and EFSA had?

MS. WAGSTAFF: Objection.

A. I -- to be honest, I don't know what data EFSA and EPA had, so I can't comment.

Q. (BY MR. HOLLINGSWORTH) There's no published peer review anywhere on this planet that says any one of the findings you refer to individually or all the findings you refer to jointly about tumors in the rats studied by Dr. Stout and others are compound related or caused by glyphosate, true?

A. There -- other than the Greim paper, which lists the Stout study, which is a peer-reviewed published -- publication, no other study refers to this -- no other publication refers to this Stout study.

Q. Does Greim make a conclusion about the carcinogenicity of glyphosate in connection with he and his authors, his co-authors' review of the 1990 Monsanto sponsored study by Dr. Stout?

A. I believe his conclusion was there was no effect of glyphosate.

Q. And the conclusion that you have, which

is the opposite, that there is an effect of glyphosate that's shown by this study has not been subjected to any kind of peer review, has it?

MS. WAGSTAFF: Object to form.

A. Not that I'm aware of.

Q. (BY MR. HOLLINGSWORTH) Do you remember reviewing a rat study that was reported out in 1996 by Feinchemie, F-e-i-n-c-h-e-m-i-e?

A. What was the date?

Q. 1996, sir.

A. Is that the Suresh study on Wistar rats?

Q. Yes.

A. Okay.

Q. We're going from Sprague-Dawley rats to Wistar rats.

A. Correct.

Q. Did that make a difference to you in the way that you interpreted the Feinchemie study?

MS. WAGSTAFF: Object to form.

A. I'm sorry, would you repeat that?

Q. (BY MR. HOLLINGSWORTH) Did the fact that the Feinchemie study involved Wistar rats rather than Sprague-Dawley rats make a difference to you in the way that you interpreted the results of the Feinchemie study?

A. The fact that one used Sprague-Dawley as on opposed to Wistar?

Q. Yes.

A. That wouldn't make a -- no. Should not.

Q. The different strains of rats would not make a difference to you?

A. As to the way I evaluate it?

Q. Yeah.

A. Not necessarily. The only consideration would be, you know, historical background rates for the Wistar would be different than the Sprague-Dawley rats, but both of those strains of rats are very widely used in toxicology carcinogenicity studies, so there's a large database for both of them.

Q. You know that the authors of Feinchemie study concluded there are no compound-related neoplastic lesions anywhere in this study?

A. Correct.

Q. Did you have the full study report from the Feinchemie 1996 rat bioassay?

A. Again, I'd have to go back and look at my files to see just what exactly all I had. I don't recall that I had a full report for this particular study.

Q. Did you tell the Court in your expert

witness report that the original investigators of the Feinchemie 1996 rat study concluded that there were no compound-related neoplastic lesions in any of the treated animals in this study?

MS. WAGSTAFF: Object to the form of the question.

A. I was asked to give my opinion, do a hazard assessment and give my opinion for glyphosate and glyphosate formulations, and so I reviewed the data and my report reflects my opinion.

Q. (BY MR. HOLLINGSWORTH) You didn't tell the judge what the original authors had concluded, did you?

A. No.

MS. WAGSTAFF: Objection, asked and answered.

A. I -- like I said, I -- I was asked to give my opinion and I gave my opinion.

Q. (BY MR. HOLLINGSWORTH) Now, this was -- this study was submitted to the U.S. EPA, correct?

A. Correct.

Q. And have you looked on the EPA online database to see what's there about this study?

A. I looked on the online database for a number of these studies, I don't recall that this was

one -- this one in particular I looked for or not.

Q. Okay. You relied totally on -- you relied totally on Greim's published data in your evaluation of the 1996 Feinchemie rat study, didn't you?

MS. WAGSTAFF: Object to form on the use of "totally."

A. The Suresh study? No. I had some additional documents to look at from that study.

Q. (BY MR. HOLLINGSWORTH) Did the plaintiffs' counsel give you those documents?

A. They provided me with all the information they had on this particular study.

Q. Now, isn't it true that this study stated there were no treatment-related deaths or clinical signs in any of the dose groups and there were no treatment-related effects on body weight gain or food consumption?

A. Correct.

Q. Did you look at the original pathology report from the overall study?

A. I'd have to go back and look at my files to see if we had -- if I had the original pathology report. If I had, I did look at it, but I can't remember.

Q. Now, these animals were treated with -- in the high dose group with over 1,000 milligrams per kilogram per day doses of glyphosate; isn't that right?
A. In the high dose?
Q. Yes.
A. Much higher than the 1,000, yes.
Q. But you concluded that the -- that the maximum tolerated dose was not reached, right?
A. Based on my observations or the reported survival and body weight gains for these animals, it would appear that an MTD was not reached.
Q. I didn't say that -- in my prior question about 1,000 milligrams per kilograms per day, I'm talking about mgs per kgs, you understand that right?
A. I'm sorry.
Q. Mgs per kgs is something different?
A. Right. I -- I heard parts per million. I apologize.
Q. And the acceptable OECD and EPA standard regimen for treating -- for the high doses in experimental mouse studies is to reach 1,000 mgs per kgs per day; is that right?
A. That is their criteria, per day.

Q. In this study, Feinchemie -- Feinchemie that we're talking about now, the 1996 rat study reached 1,000 mgs per kgs per day in the high dose animals; isn't that right?
A. That's what was reported.
Q. Mgs per kgs is m-g slash k-g slash day, right?
A. Yes, sir.
Q. Has your conclusion that the MTD, maximum tolerated dose, was not reached in this study been subject to peer review and publication?
A. My opinion?
Q. Yes.
A. Not that I'm aware of, but this -- this 1,000 milligrams per kilogram body weight that is the upper limit for, is this -- what agency is this for EFSA? No.
Q. It's for EPA.
A. EPA. That's for their purposes of doing risk assessment. If you look at chronic bioassay studies, at least in my long experience with the National Toxicology Program, Animal Bioassay Program, there's not an upper limit. The only upper limit in a chronic two-year animal bioassay in the NTP is -- for feed would be 50,000 parts per million. 5 percent of

the diet is the maximum dose that do for a study.
Now, I'm giving you too much information. But the dose of -- that is limited at 5 percent because once you go over 5 percent in the diet, you're going to start impacting nutritional content of the food that the animals are eating, so the effects you see may be due to nutritional effect as opposed to just to the chemical, so it is not uncommon to go up to 50,000 parts per million if the animals will tolerate it for chronic bioassay study.
So this 1,000 mgs per kgs that the EPA has is their value in assessing risk assessment, but for chronic animal bioassays and for hazard identification, much higher levels are tolerated for those studies.
Q. Excuse me. The OECD guidelines of reaching at least a 1,000 mgs per kgs per day in the high dose animals is worldwide standard, isn't it?
MS. WAGSTAFF: Object to form. Standard for what?
A. I can't talk --
Q. (BY MR. HOLLINGSWORTH) It's a standard that EFSA, the European regulatory authorities also adhere to, isn't it?
A. That may very well be. And, again,

that's for their purposes of risk assessment. But we -- what I have done is hazard identification.
Q. You didn't find any evidence of an increased incidence of adenoma or carcinoma in any organ in any of these rats, did you, in the Feinchemie study?
A. In the Feinchemie study, no, I found no evidence of that, but I also determined that the tolerated dose was not reached, and so in my opinion, this was an inadequate study to evaluate the carcinogenicity of glyphosate.
Q. It's not a negative study?
A. It's an inadequate study.
Q. And that is based on a standard that's imposed by the National Tox Program project?
A. Based on my many years of experience within the National Toxicology Program and also that would be a -- something that would also be considered by the IARC monograph program as an indication that the study is inadequate because the doses were too low to see an effect.
Q. Is the National Tox Program standard published?
A. Absolutely.
Q. So where do you find that?

A. You can go online to the NTP.com or dot gov, excuse me.
Q. And then what you do you do?
A. Just look from their site you go to study reports.
Q. And you'll find there that the maximum tolerated dose that NTP wants to see is 50,000 parts per million?
A. I didn't say that that's what they want to see. I mean, sometimes -- you have to do your dose setting to see what doses the animals will tolerate and you do a series of studies to evaluate what doses the animals will study -- will tolerate. And based on that, you set your doses. But if the animals appear to be able to tolerate acutely a dose greater than 5 percent, the NTP will not do a study above 5 percent because once you add more than 5 percent to the feed, you're going to start affecting the nutritional value and, therefore, the effects you see may be due to the restriction of the feed or restriction on nutritional intake as opposed to solely the chemical that you're studying.
Q. What was the high dose group in the Feinchemie rat study receiving in parts per million in the diet?

A. 40,000 parts per million is what I have in my report.
Q. So they were receiving 40,000 parts per million?
A. Right.
Q. And you're telling us that the NTP program would go to 50,000 parts per million?
A. If the animals would tolerate.
MS. WAGSTAFF: Objection, misstates testimony.
Q. (BY MR. HOLLINGSWORTH) Okay. Okay. So you don't think 40,000 parts per million is a sufficiently high dose to test glyphosate with in Wistar rats?
A. Based on the results of this study after two years, you saw no effect on body weight or survival of the controls versus the high dose treated animals, so, therefore, it appears the animals could have tolerated a higher dose. So, therefore, you did not dose the animals at a high enough level to see an effect if an effect -- if, you know, if it was present. So. . .
Q. Are you aware of the conclusion reached by the original authors, that is, the investigators, the veterinary pathologists who conducted the -- the

2009 rat study by Dr. Wood, the sponsor was Nufarm.
A. Okay. Now we're going on to Wood. Okay. Okay.
Q. Now, is this another study where you say that the maximum tolerated dose or MTD was not reached and therefore it is inadequate for evaluation?
A. That's what I said in my report, correct.
Q. Did you think that the 300 parts per million high dose level for the Monsanto 1981 rat study by Dr. Lankas was at a high enough level to be adequate for review?
A. The Lankas study?
Q. Yes.
A. It's adequate for review because you saw an effect. So, therefore, you can -- you can make an evaluation. The fact that you saw an effect in the Lankas study indicates that you can make an evaluation of the study because an effect was observed and it was a significant effect in the testes, interstitial cell tissues of the rats. So even though an MTD wasn't reached, it's still an adequate study for evaluation because you saw an effect.
But in these other studies, you saw no effect. You saw no effect on body weight. You saw no

effect on survival. You saw no increased incidences of any type of tumors, so you got -- essentially you got no effect. So since you saw no effect, and you didn't test them at the -- at a top dose that they could tolerate, it's an inadequate study for the evaluation of the carcinogenic potential in this particular study.
Q. Are you aware that the Wood 2009 rat study was submitted to EPA?
A. Yes.
Q. And EPA did not consider there to be any treatment-related incidence of cancer in any organ in any animal, true?
A. That was their conclusion, because in my opinion --
MS. WAGSTAFF: Object to form.
A. -- it was their opinion because it was an inadequate study. My opinion that it's an inadequate study, therefore --
Q. (BY MR. HOLLINGSWORTH) Okay. What was the high dose group receiving by way of parts per million glyphosate in the diet?
A. In --
MS. WAGSTAFF: In which case?
A. In the Wood study?

Q. (BY MR. HOLLINGSWORTH) Yes.
A. Parts per million was 15 parts per million for 24 months.
MS. WAGSTAFF: Did you say 15 or 50?
THE DEPONENT: 15, 1-5.
Q. (BY MR. HOLLINGSWORTH) Okay. The EPA did not conclude that the motion -- that the maximum -- motion -- maximum tolerated dose was reached, did they?
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Was not reached, did they?
A. I didn't see anything in the EPA report addressing maximum tolerated dose, no.
Q. They didn't say -- they didn't make the observation that this study is invalid because the maximum tolerated dose was not reached, did they?
MS. WAGSTAFF: Object to form.
A. No, but there again, you have to consider that the EPA was doing a risk assessment, so for the purposes of their risk assessment, the fact that the MTD was not reached may not be a part of their criteria or part of their evaluation. So that's why they would not address that issue.
But for the purpose of a hazard

identification, if you're going to do a carcinogenicity study, you need to treat the animals at a level that they can tolerate without showing overt toxicity, and that is to find a maximum tolerated dose. And my evaluation of the Wood study is the MTD was not reached, so, therefore, it's not a valid study for determining carcinogenicity because you saw no effect.
Q. That report has been submitted to EFSA also, hasn't it?
A. I believe it has.
Q. And EFSA concluded there was no carcinogenic effect of that study due to the administration of glyphosate, didn't they?
A. Again --
MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) Is that right?
A. Again, the EFSA are doing risk assessment and their criteria for risk assessment evidently say that this study is -- is negative.
Q. Didn't EFSA say that the study showed no carcinogenic effect?
A. No carcinogenic effect, that's what they said for the purpose of their risk assessment.
Q. Now, you looked at three additional rat

studies, didn't you?
A. Okay.
Q. Cheminova, 1993; Syngenta, 2001 and Arysta, A-r-y-s-t-a, 1997.
A. Okay.
Q. And you concede that those three studies are negative for the carcinogenicity of glyphosate, true?
A. Which ones are they again? I'm sorry.
Q. I believe they're Cheminova, 1993.
A. Okay.
Q. You concluded with respect to that study, which was a two-year rat study in Sprague-Dawley rats, right?
A. Correct.
Q. That there was no evidence of carcinogenic activity that you could see based on your review of that study?
A. Right, no statistically significant increase versus control.
Q. And you said the same thing for the Syngenta -- the sponsor is Syngenta in 2001, right? And the Syngenta study is in a slightly different strain of rat, isn't it?
A. This is a 2001?

Q. I believe so.
A. It's in the Wistar rat.
Q. Okay. No, wait a minute.
A. Yes, and I said that was negative.
Q. Yup. And that's in the Wistar rat?
A. Correct.
Q. Okay. And so you said that the Syngenta 2001 study is negative?
A. Correct.
Q. And the Arysta 1997 study, do you have that in mind?
A. Syngenta 1997?
Q. Arysta.
A. Arysta, okay.
Q. Arysta is a Japanese -- no.
A. Okay. Yes.
Q. Is Arysta a Japanese company or an Israeli company?
A. I do not know.
Q. Anyway, the Arysta study in 1997 was conducted in Sprague-Dawley rats, true?
A. Correct.
Q. And you concluded that there was no evidence of carcinogenic activity in that study at all, correct?

A. That's correct.
Q. Greim and his co-authors reviewed all the studies that you have reviewed, true?
A. Yes. Yes. I think the only one that I'm -- yes. That's correct.
Q. Do you know how much time Dr. Greim and his co-authors spent reviewing the studies that they reference in their paper?
MS. WAGSTAFF: Objection, calls for speculation.
A. I have no idea.
Q. (BY MR. HOLLINGSWORTH) You didn't inquire into that?
A. No, sir.
Q. Isn't that something that you'd like to know as a scientist?
A. How much time they spent going through the data?
Q. Yes. How much time did the authors spend evaluating the data?
A. I mean, I'm sure they took as much time as they needed to get the data together and put in the publication.
Q. Do you know how Dr. Greim and his co-authors selected the specific tumor data that they

chose to report for their study?
A. No.
Q. Isn't that something that you'd like to know before you rely on their opinions?
A. Well, they --
MS. WAGSTAFF: Object to form.
A. They -- they did explain in the -- in the beginning of their paper how they went about gathering the data and putting the data together. So that type of information was available in the publication. I assume since it's a peer-reviewed publication that the people who peer reviewed the paper were satisfied that the methods that were outlined in the Greim paper as to how they put together the tables and chose the studies and what have you were acceptable.
Q. (BY MR. HOLLINGSWORTH) Do you know whether Dr. Greim and his co-authors conducted their own statistical evaluation of the tumor data from the nine rat studies and five mouse studies that they reviewed -- I'm sorry, from the seven rat studies and the five mouse studies that they reviewed, excuse me?
A. I'd have to go back and look at the data to refresh my memory. I can't recall if they did the statistics or where they got the statistics from.

Q. Do you know where or why they chose the particular statistic methods that they chose?
A. Again, I'd have to look at the paper and see the rationale that they would have used -- that they would have stated. I don't recall. I'd have to look at the paper again.
Q. Wouldn't you want to know that as a scientific evaluator?
A. Well, sure.
Q. Doing the kind of report you were doing?
A. Sure. But that's what I said. You look at the paper, you read the Greim paper and when you read the paper, they should have outlined in there their method for selecting the studies, for putting together the table and their selection of the statistics that they used in the paper if they did the statistics, so I would have read that when I read the Greim paper.
Q. And you relied on that?
A. Well, I -- I relied on that or I relied on EPA or I relied on information I had obtained from Chris Portier, and I referenced that in my report where the source of the statistics that I used in my report.
Q. Did you know that Dr. Portier also

relied on data from Dr. Greim's publication?
A. Well, of course. I mean, that was -- that was the only publicly available source of -- for a lot of these studies. So of course he would use that. Now --
MS. WAGSTAFF: We've been going almost two hours. When you get a chance, can we take a break?
MR. HOLLINGSWORTH: Sure, we can break now.
MS. WAGSTAFF: Okay.
THE VIDEOGRAPHER: Going off the record. The time is 3:46 p.m.
(Recess taken, 3:46 p.m. to 4:08 p.m.)
THE VIDEOGRAPHER: We are back on the record. The time is 4:08 p.m.
Q. (BY MR. HOLLINGSWORTH) Can we assume that Dr. Greim and his co-authors had the summary tables for tumors in each of the 12 long-term bioassays that they evaluated in their published paper?
MS. WAGSTAFF: Objection, calls for speculation and assumption.
A. I -- I'd -- I really need to take a look at the Greim paper to make sure that it was true for

all the studies. I know they had summary tables for a number of the studies, but I can't say that they had them for all of them.

And while we're on the Greim, if I may, first I want to make it -- make it clear that -- that I did not rely totally on the Greim for my report. I use the Greim to get some information on tumor incidences and that type of thing, but I did not rely on that exclusively or totally.

And while we're on the subject of the Greim paper, I hate to express my unhappiness or my anger about something, but Monsanto has been making it sound like when the review of glyphosate took place at IARC that they totally ignored the Greim paper and that is absolutely not true.

The Greim paper was provided to us, it was provided to me, kind of, as I testified, at the last minute. But we did review the paper as best we could with the time we had and we also addressed it in the monograph, so the Greim paper is addressed in the monograph. So to say that IARC ignored all of the data that Greim provided is absolutely not true and you need to stop it. You need to stop telling the media that IARC didn't look at it. They did.

In fact, it's in the monograph. If you

look at the monograph, it addresses the Greim paper in several of the studies in the Greim paper, so I just wanted to express my displeasure with the way my testimony was given to the press and then misrepresented, so stop with the fake news.

Q. (BY MR. HOLLINGSWORTH) Well, thanks for your advice, Dr. Jameson, I read your deposition, the so-called fact deposition, and I know what you said there and I know you expressed tremendous surprise when you saw that the Greim paper had been provided to the other members of the IARC committee but not to you and I'll leave the record at that unless you want to argue about it.

A. No, no, no, it's -- it is what it is.

Q. It is what it is.

A. I -- and I was -- as I -- as you can tell and the expression I made is going to haunt me forever because that's what got in the media, of course. But I was just surprised that IARC had access to it, little bit further -- little bit earlier than I was made aware of it. That's all.

Q. Okay. I'll move to strike everything that you said because it wasn't in response to any question I had.

A. That's up to you.

Q. Sir, we can assume -- you can fairly assume as --

MS. WAGSTAFF: Before we move on, I will say that that is absolutely in response to your questions about asking about Greim all day long, but go ahead.

MR. HOLLINGSWORTH: Okay. That's okay.

Q. (BY MR. HOLLINGSWORTH) Sir, you know from your reading of the Greim materials that they -- those authors had at least the summary -- tumor summary table for every single study that they talked about, didn't they?

A. To the best of my recollection, they -- that's what they stated.

Q. And didn't you say that you relied on Greim totally for the tumor incidences?

A. No. I did not say that.

MS. WAGSTAFF: Objection, misstates testimony.

A. No, I absolutely did not say that.

Q. (BY MR. HOLLINGSWORTH) Okay.

A. I relied -- to be honest, I relied on the study reports that I received from the individual studies from the laboratories, the laboratory reports. That would be my first source of getting the tumor

data. I would take that information and I would compare it to what was in Greim. I think that's what I said. I would look at the tumor data, tumor tables, get the information and then take the opportunity to compare it to Greim to make sure they -- they were the same and -- and that would be my first source.

To be honest, my second source would be if the EPA had written a report or published a document on their review of a particular study, I would also go to that and use that as a source for tumor incidences if it was included in their report.

Again, I would take that information, compare it to Greim, but, no, Greim was definitely not my primary source for the information.

Q. Isn't it true that in your report, you referred -- you referred to 14 rodent studies and 11 times you referred to Greim?

A. True. But I think as I indicated before, I used that more as -- for convenience to keep straight all the different studies than -- than anything else.

Q. When you were comparing the studies -- excuse me, when you were comparing the tumor tables from the actual studies themselves to what Greim said about them, did you find any material differences

between what Greim said was a tumor incidence and what the actual original studies themselves said?

A. Sitting here today, I don't recall that I did see any -- any differences. Although, I think I mentioned in my -- in one place in my report that I looked at the Greim Tier II report and got some incidences from that, and that was a little bit -- that was different than what was listed in the actual study tumor tables that I got, but that -- and I indicated I couldn't resolve why one was different from the other, but that -- that's the only one I addressed in my report.

Q. Which study was that?

A. I'm going to have to go through my report to find it, but it is listed in my report. That's for the Sugimoto study, study 12 in Greim. Talking about the -- it started midway, do you want me to read it --

Q. Just tell me what you're referring to, what page.

A. This is on page 22.

Q. Yep.

A. The Sugimoto, it's the second paragraph, and about midway down it starts talking about review of nine tumor tables shows that there was significant

trend in development of hemangiosarcomas.

Q. Yep.

A. And then about a third -- seven or eight lines, I'd say I also reviewed the Tier II summaries for glyphosate from Greim, which showed a reported statistically significant increase in lymphoma.

Q. Yep.

A. In mice. However, I could not resolve the difference in the tumor incidence between the Greim summary and the published Greim, et al. and the Sugimoto tumor tables that's the discrepancy that I found.

Q. That wasn't a significant discrepancy even if it was a discrepancy, was it?

A. A significant discrepancy?

Q. Yeah.

A. Well, it depends on what you -- I mean, it affected --

Q. It wasn't a material discrepancy, was it?

A. Well, it was a discrepancy in the incidence, reported incidence.

Q. Okay. How did you get ahold of the Sugimoto study report?

A. That was provided to me by counsel.

And, again -- well, by counsel.

Q. Okay. So you had reports on these pathology studies, these long-term bioassays on more than just the three Monsanto studies?

MS. WAGSTAFF: Object to form.

A. Okay. I had -- I had some information on all of the studies. The amount of information I had depended on who the -- who the study was performed for. And if memory serves me correctly, if it was a Monsanto study, I had a lot more -- a lot more documents to look at than from the other -- from the studies that were performed in support of other organizations.

Q. (BY MR. HOLLINGSWORTH) Well, the Sugimoto study and all the other studies other than the Monsanto study are not publicly available, so I'm wondering how you got those study reports, the actual study reports.

A. Like I said, I -- I -- for -- other than the Monsanto studies, the information I had was a lot less, so -- and I think as I indicated earlier in my testimony, some of them I didn't have much information. I may not have even had the report or much more than some tumor tables.

Q. You just told us that you had the actual

study report for Sugimoto?

A. Did I say that?

Q. Yeah.

A. Then I misspoke. I apologize.

Q. Because you said you had the study from which you compared the Sugimoto actual report data to the Sugimoto data reported out by the Greim publication.

A. But that was the data from the tumor tables that I had.

Q. What were -- do those tumor tables come from Greim too?

A. There were tumor tables in Greim.

Q. Yeah. There were online -- they were tables of actual animal by animal data?

A. Right.

Q. In the Greim online supplement?

A. Correct.

Q. Is that what you're referring to?

A. Usually I refer -- I would -- like I said, I would look at the tumor tables from the actual study lab because I think I had tumor tables for every study. And then I would take that and I -- actually, I compared it to what Greim had in his publication and usually they compared very well and I didn't go any

further.
Q. Okay. Do you know whether Dr. Greim and his co-authors actually reviewed the underlying study reports for each of the studies they report in their publication?
A. I don't recall if they indicated they did that in their publication or not.
Q. Wouldn't you want to know that information before you made an opinion about it?
A. Well, like I said, the Greim paper is published in a peer-reviewed journal. The fact that it was peer reviewed and accepted for publication indicates that the methodology that they explained in their -- in their paper was adequate for the peer reviewers to accept the publication, so -- and like I said, sitting here today, I don't remember exactly what -- what they said in the Greim paper, but I -- so I'd have to look at the Greim paper to say if they indicated in there they looked at all the study reports.
Q. Do you know whether the authors with Dr. Greim and his co-authors reinterpreted the 12 studies that they included in the Greim published report or did they recount exactly what the pathologist who originally investigated those reports

had concluded?
A. I know that they -- in the Greim paper, they made comment on the adequacy of each study. In other words, they had some criteria based on some -- I don't know if it's from a publication or from an industry source or a government source, but they did have some criteria by which they measured the validity and what have you of each study and so indicated in their reports, so they did do an evaluation of the study from that standpoint.
As far as reinterpreting the actual data, the tumor data or what have you, I -- I -- again, I'd have to look at the paper to say definitely what they did because I'm sure they describe in the paper what they did. I'm under the impression they didn't change anything or try to change anything.
MS. WAGSTAFF: I'll make an additional request to please provide the study to Dr. Jameson if you're going to be asking this level of detail. It's not a memory test.
Q. (BY MR. HOLLINGSWORTH) The Greim authors did not reject the original investigators' conclusions in any single one of the 14 studies that they reviewed in their peer-reviewed publication, did they?
A. I'd have to get the paper out and look

at what they said about each one to answer that.
Q. Wouldn't you like to know that?
A. Well, I'm -- I assume they addressed that in the -- they addressed that issue in their report, so I'm sure it's in -- I would assume that it is -- what they did is in the report, so, again, I need to look at the report to adequately respond to that question.
Q. Do you agree with Dr. Greim and his co-authors that there is no evidence of a carcinogenic effect related to glyphosate treatment in any of the 14 long-term bioassays which they reviewed in their paper? Instead of 14, I should have said 12. Sorry.
MS. WAGSTAFF: Object to form.
A. Obviously in my report I indicated a number of the studies showed a positive response to glyphosate in both rats and mice. So obviously I do not agree.
Q. (BY MR. HOLLINGSWORTH) How many peer-reviewed studies have you authored in the published literature which state that glyphosate can cause non-Hodgkin's lymphoma in humans?
A. Peer-reviewed articles in the literature, I have authored none.
Q. Is this issue of whether glyphosate can

cause non-Hodgkin's lymphoma in humans something that you had studied before your work on monograph 112?
A. No, monograph 112 was the first time I addressed the issue of the potential carcinogenicity of glyphosate.
Q. And there's nothing in your curriculum vitae that indicates anywhere that you studied the issue of whether glyphosate can cause non-Hodgkin's lymphoma in humans prior to your work in -- starting in 2015 or late 2014 in connection with monograph 112 by IARC?
A. Specific to glyphosate, that would be an accurate statement. However, in my career with the National Toxicology Program, I spent many years evaluating many different chemicals for listing in the report carcinogens where I evaluated the same type of data that is available for glyphosate to decide if sufficient evidence or inadequate evidence in mice or in laboratory animals, and also if there was limited or sufficient evidence in humans based on review of epidemiology data and made recommendations for listing that in the report on carcinogens and/or the IARC monographs.
Q. You worked on the National Tox Program for many years, true?

A. That's correct.
Q. And you were in charge for eight years of the reports to Congress about what carcinogens the National Tox Program had studied, true?
A. Well, that's not quite accurate. I -- for the eight years I was director of the program, I was director of report on carcinogens. For about five years prior to that, I worked on the report on carcinogens at the -- at the National -- for the National Toxicology Program. But -- so what was the question? I'm sorry.
Q. That's -- I'll take that as an answer.
A. Okay.
Q. Here is my next question, during the time that you worked on the National Program, National Tox Program, is that NIEHS?
A. NIEHS, yes.
Q. Did the NTP ever report that glyphosate can cause non-Hodgkin's lymphoma in humans?
A. To the best of my recollection, they never addressed that issue, no.
Q. Has anyone in the United States government, Department of Health or FDA or EPA or any health agency reported to Congress that glyphosate can cause non-Hodgkin's lymphoma --

MS. WAGSTAFF: Object to form.
Q. (BY MR. HOLLINGSWORTH) -- in humans?
A. I am -- I don't know that I can answer that. That nobody has said nothing to Congress. To my knowledge, I don't know of anyone that has.
Q. When you were at the National Tox Program, you did not -- as far as you know, the National Tox Program did not report to Congress that glyphosate can cause non-Hodgkin's lymphoma in humans, true?
A. They did not while I was there, that's correct.
Q. Does the IARC preamble allow the monograph collaborators to consider potential human exposures when they do their hazard assessment?
A. Do they allow them to consider potential human?
Q. Yes. Does the -- do you understand my question?
A. Yes, sir. I think I do. It's part of the review process for the working group at IARC. When they're evaluating a chemical to address the issue of exposure and that is a section that is in each monograph. That is an important part of the review.

Q. So the IARC preamble does not permit IARC committee participants to fail to consider potential human exposure in the real world environment, true?
MS. WAGSTAFF: I'm just going to say that we're starting to get into testimony that related to his fact witness deposition that's already taken place. I think if we go much further, I'm going to have to instruct him not to answer.
A. Could you repeat the question, I didn't quite understand what you were driving at.
Q. (BY MR. HOLLINGSWORTH) Just listen to my question, please, and see if you can answer it.
A. Does the IARC monograph standards or the IARC preamble permit IARC committee participants to refuse to consider real world potential exposure to the substance under review?
MS. WAGSTAFF: Object to the form of the question.
A. So does it prevent them from not considering, is that what you're saying?
Q. (BY MR. HOLLINGSWORTH) Yes.
A. So it's like a double negative. I mean, it's in the preamble and the process that exposure is a major part of the review of a chemical by the IARC

monograph program, and so exposure data is -- is investigated, they -- there is a section in each monograph on exposure. Turns out that exposure is an extremely important area for the epidemiologists. They need to know how people are exposed, where they're exposed, what the -- the levels that are being processed so they get an idea of the levels that people are exposed to. So exposure is a very important part of the IARC monograph.
So, yes, they are asked to review the exposure information for each chemical that they review for the monograph. So -- but, you know, they don't twist people's arm and say you have to -- have to look at this. But they ask for their opinion and they ask -- ask to make sure that they agree with what's written in the monograph because the monograph is a product of the whole working group, not just an individual or not just a subgroup.
It's the whole working group is -- is responsible for producing that monograph, so the monograph is a product of every person on that monograph, so every person on the monograph votes on the acceptability of each section, so I'm not aware of that a monograph review has ever taken place where exposure wasn't an important aspect of the review.

Q. You recall my questions about the three negative rat studies that you reviewed in connection with the report, the expert report that you prepared?
A. The ones that -- that I indicated that were --
Q. Yes, were negative?
A. No effect. Were negative.
Q. Yes.
A. Yes.
Q. Did the IARC preamble preclude IARC committee members from looking and considering -- looking at and considering negative data --
A. No.
Q. -- such as those three studies?
A. No.
Q. Does the IARC report itself provide a sufficient scientific basis for your opinion in this case that glyphosate can cause non-Hodgkin's lymphoma in humans?
A. What I can say is my participation on the IARC working group -- I formed my initial opinion of glyphosate based on my work with the IARC monograph and the IARC -- we, as the IARC monograph working group, agreed that it met the criteria for a two-way human carcinogen -- I'm sorry, possible -- probable

human carcinogen, and that there was an association of exposure to glyphosate in glyphosate formulations to non-Hodgkin's lymphoma in humans based on the epidemiology studies, so that's where I formed my initial opinion.
But after asking to review all of the available data, I was -- I had the opportunity to delve into it into more detail, look at new data. It gave me the opportunity to take the Greim -- the studies in the Greim paper and the Greim paper itself and the tables in the Greim paper, and I had the time to sit down, look at the data and evaluate it and the Greim paper just strengthened my opinion that it -- that glyphosate is an animal carcinogen because we found more tumors from that -- from those studies that are -- were identified in the Greim paper.
And so that's how I formed my opinion that glyphosate -- on glyphosate in non-Hodgkin's lymphoma.
Q. Do the hazard assessments that the IARC monograph committees may take into account whether any effects seen from studies that are reviewed by the IARC committees regarding carcinogenicity are conducted at human relevant doses?
A. Are you implying -- the animal studies?

Q. Yes.
A. No. I'm sorry, I guess maybe it's getting late in the day.
Q. Let me reask the question.
A. Yes, please.
Q. Does the hazard assessment that you made based on animal studies in your expert witness report take into account that effects on animals are seen or not seen at doses that are relevant to the human environment?
MS. WAGSTAFF: Object to form.
A. Well, doing a hazard assessment, the purpose of the hazard assessment is to evaluate the material to see if it can cause cancer in animals. Let's just address the animal part, because that's what you -- the question was about in animals. So the hazard identification is performed to identify if a chemical under the most extreme conditions can cause cancer in experimental animals, it does not worry about the levels that are -- humans are exposed to.
The first question is can it cause cancer, is it an animal carcinogen, so under standard process of doing a hazard identification, you look at animal bioassays, and bioassays, as I identified before, are done trying to use the maximum tolerated

dose. So the maximum tolerated dose is the dose the animals can tolerate without showing overt toxicity, so that is the purpose of the bioassay and that is what the hazard identification uses to establish if something is an animal carcinogen or not.
So I mean, that is -- that argument about human relevant doses is -- is -- goes on -- has been going on for years and years and years in toxicology, but the state of the science is first we have to establish is it an animal carcinogen and then you do additional studies. You do the risk analysis to see what happens at the human relevant doses.
Q. (BY MR. HOLLINGSWORTH) When you do your hazard assessment, I think you say that the -- you said that the hazard assessment does not worry about levels that a human is exposed to; is that right?
A. Well, maybe I -- maybe I -- I used the wrong term about not worry about. When you do a hazard assessment, first you have to determine, you know, is it an animal carcinogen, is it a human carcinogen. And since your question spoke directly about animals, to -- the best way to identify if it's an animal carcinogen is to look at the bioassay data. And by definition, when you do a carcinogenesis bioassay, you try to expose the animals to the MTD.

You have to do things in steps and so that's why the doses are high for the -- initially for the animal studies, but it's based on the animal studies that limits are set and risk assessments are done.

Q. Does a hazard assessment based on animals consider whether the substance being studied by the review committee is -- is a carcinogen at levels that humans are exposed to?

MS. WAGSTAFF: Object to form.

A. I'm trying to formulate the question in my mind. I'm sorry, what was it again?

Q. (BY MR. HOLLINGSWORTH) Does the hazard assessment that the IARC committee members look at when they're evaluating animal data consider whether the substance, the test substance, is a carcinogen at levels which humans are exposed to?

A. As part of the evaluation of all of the data that is done, they always -- the working group, the people of the working group are always -- try to make themselves, at least in my experience with the working group, you try to make yourself familiar with what the human exposure levels are.

That's why there's a whole section in IARC monograph on exposure. That gives you an idea of

what the potential exposure could be, and so that's always in the back -- they always know, if you will, based on the exposure assessment what human levels are -- what levels are that humans are exposed to. So they're aware of that. But, again, like I said, for the purpose of hazard identification, the question asked is, is it an animal carcinogen, and the best -- and the data that is used for that is from an animal bioassay study, so for animal bioassay studies, they use high levels.

Now, a lot of times the lower levels that are used in a bioassay are, you know, may be an order or two of magnitude of the high dose and sometimes the low dose approaches a human exposure level, but that just depends on the design of the study.

MS. WAGSTAFF: For the reasons I set forth on the break, can we take another break here in a few minutes?

MR. HOLLINGSWORTH: Sure, when this is done. Tracy, can you read back my question, please, because he didn't answer my question.

(The question was read back as follows: "Does the hazard assessment that the IARC committee members look at when they're evaluating animal data

consider whether the substance, the test substance, is a carcinogen at levels which humans are exposed to?")

MS. WAGSTAFF: I'm going to object to the fact that this is related to questions already asked at his fact witness deposition and he just asked and answered it.

Q. (BY MR. HOLLINGSWORTH) Can you give me a yes or no answer to that?

MS. WAGSTAFF: He's answered the question.

A. I gave you an answer before. I stick to that answer. Sorry.

Q. (BY MR. HOLLINGSWORTH) What did you mean when you said that the hazard assessment group that you worked with does not worry about what levels humans are exposed to when they make their hazard assessment?

MS. WAGSTAFF: Objection. He already testified that he misspoke when he said does not worry.

Q. (BY MR. HOLLINGSWORTH) What did you mean does not worry?

A. What I --

Q. It seems to me like you mean does not take into consideration what actual human exposures

are, that's what it seems like to me?

MS. WAGSTAFF: Misstates testimony. Argumentative.

A. That's not what I meant. I shouldn't have said don't worry about. The purpose is to -- the first step in a hazard identification, one of the first steps, as far as animals are concerned, is to determine if it causes -- if it's an animal carcinogen, and an animal bioassay is the main study that addresses the issue of can a chemical cause cancer in animals.

And the standard protocol for an animal bioassay study is to do it at the maximum tolerated dose and increments below the maximum tolerated dose to see if it does -- if it can cause cancer under any circumstances. That's the question that's being addressed. So the working group will consider all the doses that are -- that are studied in a particular bioassay and they will make an observation of, oh, look at the low dose level, it's within an order of magnitude of what the humans are exposed to, so they take that -- they are cognizant of that and they take that into consideration.

And, in fact, sometimes -- I can't quote to a particular place, but sometimes, in -- in the

monograph, if it is -- if it is the case, they will say, you know, exposure at dose such and such parenthesis or brackets, if it's a comment from the work group, a level that's less than order of magnitude greater than what humans -- the EPA standard or the OSHA standard for it is, those particular types of comments are made in the study, so they do take into account -- they do consider the human exposure.

It's just that the design of the study for animal carcinogenicity is to find out if the study -- if the chemical can cause cancer in the animals.

Q. Did you cite any evidence in your report, your expert report to the judge in the MDL, that says that any one of the feeding levels in any of the 12 studies you reviewed in your report was close to the human doses in the real world environment?

A. I did not address that in my report, no.

Q. Do you know of anybody who has published such a report in the peer-reviewed medical literature?

A. I'm not aware of any, but to be honest with you, I haven't searched for that.

Q. Are you aware of any published case report from a medical doctor or a scientist that says that he or she had seen a patient whom he or she

thought had non-Hodgkin's lymphoma that was caused by exposure to glyphosate?

A. A report -- a clinical report -- a report from a clinician?

Q. A case report from a clinician, yes. Have you seen that?

A. I -- I'd have to go back and look at some of the epidemiology studies to see what they had in those reports, where they got some of the information for the case control studies. But sitting here today, I can't recall, but I'd have to go back and look at the literature again.

Q. You don't cite any study in the published peer-reviewed literature or any material that you have considered that states there is a case report that has been published by a clinician that says that glyphosate caused non-Hodgkin's lymphoma in a patient anywhere on the planet, do you?

MS. WAGSTAFF: Object to the form of the question.

A. I don't have it in my report, no, but that's because I haven't done a search for that. It's not to say that there isn't some reports out there in the literature.

Q. (BY MR. HOLLINGSWORTH) My question --

A. But I haven't searched for one.

Q. My question went to whether there was such a report in your materials considered list that's attached to your expert report.

A. And I said no, there isn't.

MS. WAGSTAFF: Can we take that break now?

MR. HOLLINGSWORTH: Sure.

THE VIDEOGRAPHER: Going off the record. The time is 4:47 p.m.

(Recess taken, 4:47 p.m. to 5:01 p.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 5:01 p.m.

Q. (BY MR. HOLLINGSWORTH) Sir, when you and your colleagues at the National Tox Program made the reports you made to Congress for the -- regarding the list of carcinogens, you were reporting on what you had determined based on a hazard assessment, right?

A. What we were -- what we reported on was our review of the available data based on the criteria that had been established and approved by the Secretary of Health and Human Services for listing substances in the report as either known or reasonably anticipated to be human carcinogens.

Q. The hazard assessment that the National

Tox Program did and reported to Congress did not take into account whether any effect seen that support carcinogenicity from the studies, the animal studies are at human real relevant doses, true?

A. In the animal studies?

Q. Yes.

A. Again, the criteria for listing in the report on carcinogens, as far as the animals are concerned, is sufficient evidence in animals from studies in -- in -- in animals by multiple rounds of exposure, I could go -- I'd have to look at the thing to remember all of the criteria -- exactly what the criteria said, but they did the hazard assessment based on data in animals, and data in -- in humans and the data in animals was based on the carcinogenicity studies that are conducted in animals.

And as I indicated before, the carcinogenicity studies standard in toxicology for the 35 plus years I've been doing this type of work, the standard is to do an animal bioassay carcinogenicity study at the maximum tolerated dose.

Q. Isn't --

A. The purpose is to identify if under whatever the -- you know, if you want the most extreme circumstance, but can the chemical cause cancer in

experimental animals.
Q. Isn't it true that the listing of a substance within the report to Congress by the National Tox Program only indicates a potential hazard and does not establish the exposure conditions that would pose cancer risks to individuals in their daily lives?
A. That is what you're reading from the -- probably the introduction to the report on carcinogens.
Q. Correct.
A. I remember writing that.
Q. Yes. I'm reading from the one in 2004.
A. Uh-huh.
Q. That's the one that you wrote, right?
A. Uh-huh.
Q. So you wrote that "thus listing of the substances in the report on carcinogens only indicates a potential hazard," right?
A. That's what it says, yes.
Q. And it does not establish the exposure conditions that would pose cancer risks from that substance to individuals in their daily lives, true?
A. That is -- that is saying that we -- what was performed was a hazard identification and

that the report on carcinogens is not a risk assessment document.
Q. The -- the determination of what would pose cancer risks to individuals in their daily lives is a formal risk assessment according to your report to Congress, right?
A. That's correct.
MS. WAGSTAFF: I would request that you provide him with a copy of the 2004 document.
MR. HOLLINGSWORTH: Sure. I'll mark this as Exhibit 22-4 and this appears to be the 11th report on carcinogens which Dr. Jameson just testified that he wrote dated 2004.
THE DEPONENT: Do you need to stamp this or anything?
MS. WAGSTAFF: He put the sticker on it.
THE DEPONENT: I'm sorry.
Q. (BY MR. HOLLINGSWORTH) You're correct when you testified that I'm reading from the introduction at the bottom of the left-hand column.
A. First page of the introduction?
Q. Yes.
A. Okay.
Q. And I was reading from the next to last -- the penultimate sentence in the last full

paragraph on the left-hand column, do you see that?
A. Yes.
Q. And you wrote this, right?
A. Correct.
Q. And you also wrote the sentence which says, "Such formal risk assessments, referring to cancer risks to individuals in their daily lives, are the responsibility of the appropriate federal, state and local regulatory and research agencies," correct, did I read that correctly?
A. That is what was -- is written in the introduction. And as I indicated before, the reason for that being in there is to -- to let the reader know that what was -- what the reported carcinogens is all about is a hazard identification of the material -- of the substance that are listed in there as either known or reasonably anticipated to be a human carcinogen, and that it is not a risk assessment and the risk assessments are routinely done by the state, federal and local regulatory authorities.
Q. And what you have done in your report, your expert witness report, in this case is a hazard assessment?
A. That's as I indicated in my report, that's what I did.

Q. And that's the same type of hazard assessment that's identified in the report to Congress that you just read?
MS. WAGSTAFF: Object to the form.
A. The report on carcinogen is a hazard assessment document, correct.
Q. (BY MR. HOLLINGSWORTH) All right. Thank you. Would you agree that hazard assessments err on the side of caution in designating a compound a probable carcinogen?
A. What do you mean by "err on the side of caution"?
Q. Err on the side of protection.
A. "Err on the side of protection" of -- of what?
Q. Of the public.
A. Of the public?
Q. Yes.
A. I don't know I would say that it errs on the side of protection of the public. The purpose of this hazard identification document is to get the information to the public that these materials have been found to be, based on the available data, have been found to be either known or reasonably anticipated to be human carcinogens.

This is information that the general public needs to know so that they can make an assessment as to if are, A, are they in danger by being exposed to these materials or are these materials something they see in their daily lives or is this material something that you use either in your work or at home that you can't avoid, but now that I know -- now they know it's a possibility or reasonably anticipated or known human carcinogen, they can then take steps to protect themselves.

So the document is to get the information out to the public that, hey, this has been shown to be a known human carcinogen or a reasonably anticipated to be a human carcinogen, you need to know this information so that you can make your own -- can make an assessment of the -- your particular risk and take steps to protect yourself. And that's my interpretation of why -- of what the report is supposed to be doing.

Q. Are -- so you don't agree that hazard assessments err on the side of caution?

MS. WAGSTAFF: Objection, asked and answered.

A. I don't know how to respond to that.

Q. (BY MR. HOLLINGSWORTH) Okay.

A. It's getting the information out to the public that they need to know in order to assess their risk and make judgments as to what they want to do about it.

Q. Would you agree with the statement that a cancer hazard is an agent that is capable of causing cancer under some circumstances, while a cancer risk is an estimate of the carcinogenic effects expected from exposure to a cancer hazard?

A. May I ask where you're reading that from?

Q. It's from your report.

A. From my report?

Q. Yep.

A. Okay. Can you tell me where in the report -- is it in the introduction?

MS. WAGSTAFF: Are you talking about his expert report?

Q. (BY MR. HOLLINGSWORTH) That's not from your expert witness report, that statement?

A. That's why I'm asking. I don't -- I don't recall.

Q. Don't you state in your expert witness report exactly what I asked, which is that a cancer hazard is an agent that can cause cancer under certain

circumstances, while a cancer risk is the estimate of the carcinogenic effects expected from exposure to a cancer hazard?

MS. WAGSTAFF: Can you state what page you're reading from?

MR. HOLLINGSWORTH: Page 5 of his expert witness report.

MS. WAGSTAFF: Okay.

Q. (BY MR. HOLLINGSWORTH) Do you remember making that statement in your report, sir?

MS. WAGSTAFF: Are you talking about where he's quoting IARC right there?

MR. HOLLINGSWORTH: Yes.

A. Okay. That's what IARC says.

Q. (BY MR. HOLLINGSWORTH) It's in your report, right?

A. It's in my report, but as I said in reference to IARC preamble, that's what they state in defining a cancer hazard and a cancer risk.

Q. Do you subscribe to that definition?

A. That's -- that's pretty accurate, but, again, it's in the IARC preamble and continuing they're using that to -- to explain what it is that the -- that the -- what the IARC monographs are i.e. they are a hazard identification document. And, also,

I think it is an attempt of them -- I think if you look at the title of the IARC monographs, it's -- it -- the title -- the actual title of the IARC monographs includes the word "risk." And they wanted to make it clear to the reader that -- that while the title, which is something they're stuck with, if you will, has the word "risk" in it.

The documents that they prepare are not risk assessments, they're hazard identifications and this is what they are presenting in their preamble, but it's an accurate statement.

Q. Is your report based on a hazard assessment as defined by the National Tox Program to Congress or is it based on a hazard identification as defined by IARC?

MS. WAGSTAFF: Object to form.

A. It's based -- my assessment is based on the criteria that I outlined in my report.

Q. (BY MR. HOLLINGSWORTH) Is that based on the National Tox Program's identification of hazard assessment?

MS. WAGSTAFF: Object to form.

A. I can read the exact wording, but basically I said I developed the criteria for this particular report based on the criteria that I

developed for the report on carcinogen and similar to that as outlined by IARC.

Q. (BY MR. HOLLINGSWORTH) Okay. Is it a better definition of what your report defines hazard assessment as to refer to IARC or to refer to the report to Congress by the National Tox Program?

A. It's best to refer --

MS. WAGSTAFF: Objection.

A. -- to the criteria that I have in my document.

Q. (BY MR. HOLLINGSWORTH) Okay. And that's your criteria, that doesn't really belong to the National Tox Program or to IARC, is that fair?

A. It's very similar to it, but I came -- I developed those specifically for this -- for my expert report.

Q. Okay. Thank you. Now, Dr. Jameson, I'd like to show you an e-mail which we received in response to the subpoena that we issued to you in connection with this deposition, and I've marked this as Exhibit 22-5. I'm handing a copy to you, a copy to counsel. And this is an e-mail from Chris Portier who you described as your long-time friend and colleague, right?

A. Yes.

Q. Dated Tuesday, November 10, 2015. Do you see that?

A. Okay.

Q. And it refers to IARC monograph volume 112.

A. Well, IARC monograph 112 EFSA review of glyphosate.

Q. Yes. I see. Monograph 112 and EFSA review of glyphosate, both?

A. Right.

Q. That's important. And you cc'd Kate Guyton, right, and she's someone at IARC?

A. Correct. That's correct.

Q. And you're letting Chris Portier know in response to his invitation that you'd like to have the opportunity to participate in this IARC monograph process, right?

A. Well, that's what I told him then.

MS. WAGSTAFF: Object to form. Misstates the evidence.

Q. (BY MR. HOLLINGSWORTH) Okay. And then the -- the rest of this e-mail that's attached here is an e-mail from Chris Portier to a bunch of people including you and Aaron Blair and Matt Martin and other people that were on the IARC monograph

committee, right?

A. Right.

Q. But not all members of the IARC monograph committee, true?

A. I -- I'd have to read through all the list and see, but I can't say for sure.

MS. WAGSTAFF: Are our exhibits 21 or 22?

Q. (BY MR. HOLLINGSWORTH) Do you recall receiving this e-mail?

A. Yes.

Q. When was the last time you read it?

A. When was the last time I read it?

Q. Yes. The most recent time.

A. This particular e-mail?

Q. Yes.

A. Let's see, I got it on November -- I sent it to Chris on November 10 of 2015. I don't know. Maybe a week or two later after that would have been the last time I saw it.

Q. Chris' e-mail to you is dated November 9, 2015, right?

A. That's what it says.

Q. And in his e-mail he's discussing developments within EFSA, the European Food Safety

Agency, right?

A. Yes, that's what it says.

Q. And the developments that he's discussing are in connection with -- in connection with the assessment for regulatory purposes of the safety of glyphosate?

A. That's what EFSA is doing, trying to do.

Q. And he notes in the second paragraph of this e-mail that the German Federation Institute for Risk Assessment had taken the lead in drafting the reassessment of glyphosate and that its report had been drafted prior to the IARC review or prior to what was going to be the IARC review, true?

A. That's what it says.

Q. And he says that following the IARC review, the German regulators went back and analyzed glyphosate again, right?

A. That's what it says.

Q. And this time taking into account the IARC assessment specifically, right?

A. That's what it says.

Q. So this was -- this e-mail was something that was received by you after you had concluded your meeting of monograph 112?

A. After the IARC meeting in.

MS. WAGSTAFF: Object to form.
A. Based on the date.
Q. (BY MR. HOLLINGSWORTH) Yes.
A. Yes.
Q. And Dr. Portier reports in this e-mail that the German regulators confirmed their original conclusion and had, again, found that glyphosate does not have any carcinogenic potential, right?
MS. WAGSTAFF: Where are you reading that from?
A. I don't see that, but --
Q. (BY MR. HOLLINGSWORTH) I'm reading that from this e-mail.
A. Where in this e-mail?
MS. WAGSTAFF: I'm going to object to that question because that's not what the e-mail states.
A. I don't see that in this e-mail.
Q. (BY MR. HOLLINGSWORTH) This e-mail says that the European Food Agency -- Safety Agency was about to release its reassessment of glyphosate concluding that glyphosate had no carcinogenic potential, right?
A. That's EFSA, yes.
Q. Yes. I said the European Food Safety

Agency?
A. Before you said BfR.
Q. Sorry.
MS. WAGSTAFF: Before you said BfR before IARC.
Q. (BY MR. HOLLINGSWORTH) Excuse me. Sorry. I meant EFSA.
A. Okay. That's what it says.
Q. And then Dr. Portier, if you go back to the first paragraph of this e-mail, says that his opinion is that the EFSA conclusion creates two problems, do you see that?
A. Uh-huh.
Q. One, that it weakens the strength of the IARC assessment. Do you see that?
A. It --
MS. WAGSTAFF: That's not the full --
A. No.
MS. WAGSTAFF: Object to -- you need to read the whole sentence.
Q. (BY MR. HOLLINGSWORTH) The -- the EFSA re-assessment of glyphosate creates two problems, he says, as he sees it, right?
A. Okay.
Q. And the first is that this -- that this

re-assessment by EFSA will weaken the strength of the IARC monograph program?
MS. WAGSTAFF: To stimulate change.
A. To stimulate change --
Q. (BY MR. HOLLINGSWORTH) Yeah.
A. -- in how some of these agents are reviewed and addressed.
Q. That's what he says.
MS. WAGSTAFF: You're reading half the sentence.
A. That's what he said.
Q. (BY MR. HOLLINGSWORTH) And the second problem that he says exists due to EFSA's report is that it suggests is that IARC did not do our assessment adequately. Do you see that?
A. Correct.
Q. And that had we seen all of the data they saw, we would have gotten a different answer, is that what he says?
A. That's what he says, and, again, this is relating to something I brought up before of my anger over the way Monsanto is expressing the -- in the press how IARC did not look at the Greim papers and the information in the Greim papers, which is not true. The Greim paper was looked at by IARC and we

evaluated it to the best of our ability with the time we had and we addressed the Greim paper in the monograph, so the monograph addresses the Greim paper, so that's another indication of where this -- this false information that got out into the media has affected what other people think we did, that IARC did.
Q. Your testimony is that the IARC committee relied on the Greim paper?
A. They looked at the Greim paper.
Q. Did they rely on it?
A. They said -- if you look at the monograph and read what's in the monograph as it relates to the Greim paper, we summarize several of the studies in the Greim paper indicating what was reported in the Greim paper, but indicate that because we did not have enough time to adequately evaluate it, we can't really -- can't really include it as a study in the evaluation.
Q. Well, the IARC monograph says that it looked at the Greim paper refers to the Greim paper, excuse me. The IARC monograph refers to the Greim paper several times, doesn't it?
A. Yes, it does.
Q. Did you ask Chris Portier what he meant

when he said, "I do not intend to let this happen"?
A. Well, he was -- he was concerned that, you know.
MS. WAGSTAFF: Objection, calls for speculation.
Q. (BY MR. HOLLINGSWORTH) Did you talk to him about it?
A. I had a -- to be very honest with you, to the best of my recollection, this is my response to him that I -- hey, I'd like to see what you write and maybe I'd like to contribute to it, maybe I wouldn't, but I told him I was busy until, what, the 12th and the time frame that I had was not good for Chris.
He needed -- he wanted to get something out sooner than that so basically this is -- this was the end of it for this, for me.
Q. So you didn't participate any further in this?
A. I don't recall that I participated in this, no.
Q. Didn't you sign the letter that --
A. Was this the one with the letter that went out?
Q. Yes. Didn't you sign that?
A. There was so many, I can't remember.

Q. Well, you signed the letter that he's talking about here, didn't you?
A. If -- if this is to EFSA --
Q. Yes.
A. -- that might be -- that must be the one that I signed.
Q. I mean, Chris Portier drafted up a letter that he proposed to send to EFSA and that he wanted the people on this e-mail chain and others to sign?
A. And that was an open letter to EFSA?
Q. Yes.
A. Okay. I'd like to see that before I say anything else that I signed it or not. Like I said, there were a number of things coming out around this time and Chris was throwing things -- Chris was spearheading a number of issues, a number of things related to this, and I know there was one that I was able to comment on and then there was another one that I just didn't have time to work with. So before I comment any further, I'd like to see this open letter to EFSA.
Q. What -- what other things was Chris doing that you did not participate in that you're referring to?

MS. WAGSTAFF: Object to form. Calls for speculation.
A. I can't remember.
Q. (BY MR. HOLLINGSWORTH) You can't remember?
A. I know there were a number of things. These mostly had to do with the regulatory agencies in Europe.
Q. Did you understand that IARC and EFSA had conducted different kinds of analyses of glyphosate?
A. Well, my understanding is EFSA was doing a risk analysis and IARC did a hazard identification.
Q. Do the risk assessments like EFSA conducted on glyphosate consider exposure in real world scenarios?
A. I am not familiar with what protocol they use when they're doing their risk assessment, so I really can't address that.
Q. Okay. After Chris and you and others sent the letter regarding EFSA's evaluation or reevaluation of glyphosate which disagreed with IARC, did you and Dr. Portier send a reply to that letter?
MS. WAGSTAFF: Object to the form of the question. Dr. Jameson has asked to see the open

letter before he comments more.
A. I can't respond to that until I see the first letter and the response you're referring to.
Q. (BY MR. HOLLINGSWORTH) You don't remember -- you didn't remember sending a response?
A. I can't address that --
MS. WAGSTAFF: Object to the form of the question.
A. -- until I see the documents. I'm sorry.
Q. (BY MR. HOLLINGSWORTH) Okay. Now, before you started participating in -- with Dr. Portier in these responses to EFSA in November of 2015, did you ask Dr. Portier if he had any personal interest in that effort to respond to EFSA that went beyond just being a scientist, an interested scientist?
A. No, Chris contacted me because I was a member of the working group at IARC. As you can see, he contacted most everybody that was on IARC and it was based on his concern that what EFSA was doing would -- would reflect badly on IARC and he was trying to protect IARC, basically.
Q. Did you know that as of March 29, 2015 or about nine days after the monograph was issued on

about March 15 or March 20 or somewhere thereabouts in 2015 that Dr. Portier had started working for plaintiffs' lawyers who were intending to bring suit against Monsanto?

A. No. I wasn't aware of that.

Q. I've marked for the record as 22-6 a letter from a lawyer named Hunter Lundy to Dr. Portier which lays out an agreement that they had for Dr. Portier to consult the law firm in connection with glyphosate.

MS. WAGSTAFF: Can I have a copy?

Q. (BY MR. HOLLINGSWORTH) Have you ever seen that before?

MS. WAGSTAFF: Wait. Can I have a copy?

MR. HOLLINGSWORTH: Sure.

MS. WAGSTAFF: I'm going to object to asking him questions on a contractual agreement that he's not a party to.

MR. HOLLINGSWORTH: I'm just asking him if he's aware of this.

MS. WAGSTAFF: We've asked for documents that you've been questioning him on all day and this is the one that you decide to give him?

MR. HOLLINGSWORTH: That's right. It's my deposition.

Q. (BY MR. HOLLINGSWORTH) So my question is were you aware that Dr. Portier was working as a consultant to a law firm that represents plaintiffs in this MDL as of March 29, 2015?

A. No, I wasn't.

MS. WAGSTAFF: I'll object to the fact that this is an unsigned contract.

Q. (BY MR. HOLLINGSWORTH) Did you know that as of June of 2015 Dr. Portier was billing these lawyers to represent plaintiffs in this MDL in connection with issues involving glyphosate? And I'm handing you a document that I've identified for the record as 22-7.

MS. WAGSTAFF: Can I have one, please?

MR. HOLLINGSWORTH: Oh, sure.

Q. (BY MR. HOLLINGSWORTH) Were you aware of that, sir?

A. Was I aware that he got paid?

Q. Yes.

A. No, sir, I was not aware.

Q. I'm going to mark for the record as 22-8 a copy of an e-mail that Mr. Portier originated to a list of folks that includes you, Dr. Jameson, Bill Jameson is the name that's dated November 9, 2015.

A. November 9, 2015.

Q. Yes.

MS. WAGSTAFF: Can I please have a copy?

MR. HOLLINGSWORTH: Yes.

A. Okay. So this is the original e-mail that is on the first -- on document 22-5 --

Q. (BY MR. HOLLINGSWORTH) Yes, that's right.

MS. WAGSTAFF: There's no question on the table.

THE DEPONENT: I'm sorry.

Q. (BY MR. HOLLINGSWORTH) What is that e-mail, sir?

A. This was the original e-mail from Chris to the -- all or most of the participants of the IARC monograph 112 about this EFSA and the BfR activities.

Q. And that was in connection with the letter that you were signing on to criticizing EFSA because of its --

A. Yeah, that was the original letter from Chris saying what he wanted to do.

Q. Now, did you know that when Chris wrote -- Chris Portier wrote that letter in November of 2015 that he was working for plaintiffs' lawyers here in the United States who were representing plaintiffs suing Monsanto in connection with

glyphosate?

MS. WAGSTAFF: Objection, in Chris Portier's testimony he clearly testified that his work on this was unrelated and was not paid by plaintiffs' counsel, so it's a misrepresentation of the evidence and of the testimony.

Q. (BY MR. HOLLINGSWORTH) Can you answer my question?

A. I really have no idea what relevance this has to this deposition, but I didn't know he was being paid or that he was -- had been retained by this law firm.

Q. Okay. I'm attaching a -- I have marked as 22-9 an e-mail exchange between you and Chris Portier around Thanksgiving of 2015 in which he says he attaches the -- his version of the final glyphosate letter. Does that --

MS. WAGSTAFF: Can I have one, please?

Q. (BY MR. HOLLINGSWORTH) Is that something that you recall?

MS. WAGSTAFF: You just -- I think this is -- you just gave me 22-8 again.

MR. HOLLINGSWORTH: Oh, sorry.

MS. WAGSTAFF: I wrote 22-9 on it.

MR. HOLLINGSWORTH: Sorry.

MS. WAGSTAFF: That's okay.
MR. HOLLINGSWORTH: Here you go.
A. Okay. The question again?
Q. (BY MR. HOLLINGSWORTH) This is an e-mail exchange between you and Chris Portier around November 26, 2015, do you recall this?
A. I see this, yes.
Q. And in it he says he has attached the final version of the glyphosate letter. Do you see that?
A. I see that. That's what it says.
Q. And in that paragraph he's referring to a letter that he drafted and he was asking his group to sign on to, that is a response to EFSA's critique to IARC, true?
A. That's what it says.
Q. Does this help refresh your recollection as to whether you actually signed onto that letter or not?
A. No. Because the final paragraph reads, "For those of you who will be co-authors on the commentary, I plan to submit to JCEH, I hope to have it available to you." He was sending this to everybody because the original message is from Chris Portier to Chris Portier, so I don't know who he sent

the original message to and until I see the -- the -- the letters that you are referring to, I can't comment.
Q. Were you aware at the time this e-mail was -- e-mail exchange was had between you and Dr. Portier that Dr. Portier was working for plaintiffs' lawyers in the United States in lawsuits that were being brought against Monsanto involving glyphosate?
MS. WAGSTAFF: I have the same objection. This is misstating Chris Portier's testimony.
MR. HOLLINGSWORTH: I'm not referring to Chris Portier's testimony. I'm just asking you --
MS. WAGSTAFF: The suggestion you're leaving in the air is that -- is misstating his testimony, so. . .
MR. HOLLINGSWORTH: Okay.
A. I have no idea who Chris Portier was working for at this time.
Q. (BY MR. HOLLINGSWORTH) When -- did you ever learn that he was working on a consulting arrangement with a plaintiffs' law firm in the United States in connection with lawsuits against Monsanto?
A. With this -- with this law firm?

Q. Yes.
A. I never learned that he was a consultant to this law firm, no.
Q. Did you ever learn that he was a consultant to any law firm representing plaintiffs in the United States against Monsanto?
A. Are you asking me -- say -- was I --
Q. Did you ever learn that he was a consultant?
A. I did learn, yes.
Q. When did you learn that?
A. I think I learned that sometime within the last six months.
Q. Okay.
A. To the best of my recollection. It might have been sooner than that. It might have been later than that. It wasn't much more than about six months ago.
Q. Okay. I'm going to mark as Exhibit 22-10 another e-mail from Chris Portier. It's a one-page, one-paragraph, seven-line e-mail, do you see that?
A. Uh-huh.
Q. Have you seen that before?
A. Have I seen this before?

MS. WAGSTAFF: Can I have one, please?
MR. HOLLINGSWORTH: Sure.
MS. WAGSTAFF: This is 22-10?
MR. HOLLINGSWORTH: Yes.
A. Okay. This is an e-mail from Chris Portier to C Portier. So I may have gotten this. I -- but to be honest, it was so long ago, I don't remember.
Q. (BY MR. HOLLINGSWORTH) Okay.
MS. WAGSTAFF: Counsel, there's no Bates on this. I'm just wondering if that's -- it's probably an oversight or it got cut off on the printing. Is there supposed to be Bates on this. There is on all your other e-mails. Just so we know where it came from. Like, for example, 22-5 has Portier, so does 7. 8 has Mississippi State and 9 has Jameson.
MR. HOLLINGSWORTH: I don't know.
MS. WAGSTAFF: I would request a Bates number for that one.
MR. HOLLINGSWORTH: Okay.
Q. (BY MR. HOLLINGSWORTH) All right.
MR. HOLLINGSWORTH: All right. How much -- are you going to be asking questions?
MS. WAGSTAFF: Uh-huh.

MR. HOLLINGSWORTH: How long do you think it'll take?
MS. WAGSTAFF: Well, if you stop right now, probably 20, 25 minutes. Maybe not.
MR. HOLLINGSWORTH: Okay. I'll stop.
MS. WAGSTAFF: Okay.
THE DEPONENT: Can I take a break first?
MR. HOLLINGSWORTH: Sure.
THE VIDEOGRAPHER: Going off the record the time is 5:41 p.m.
(Recess taken, 5:41 p.m. to 6:02 p.m.)
THE VIDEOGRAPHER: We are back on the record. The time is 6:02 p.m.
EXAMINATION
BY MS. WAGSTAFF:
Q. Good evening, Dr. Jameson. You've had quite a long day, I know we've been going for about nine hours on a very dense subject, so I'll try to make this quick for you.
In relation to MDL 2741, which is the federal litigation in the Roundup litigation, you produced an expert report which has been labeled 22-1, Exhibit 22-1 to this deposition, correct?
A. Correct.
Q. And my reading of that testimony is that

it -- or that expert report is that it is typed, single-spaced typed and it goes on to the 32nd page, correct?
A. Correct.
Q. And it has on there my brief review is it had about 101 citations to different medical literature; is that correct?
A. Toxicology literature.
Q. Toxicology?
A. And cancer literature.
Q. Okay. And it had, I think, somewhere around five medical pieces of information or literature that you considered, but didn't -- but you discounted for one reason or another; is that correct?
A. You're referring to some of the animal studies that I discounted?
Q. Yes.
A. Yes, that's correct.
Q. When you were reading this report, this 32-page typed report, you actually read each of those 101 studies, correct?
A. All the references that I have in there, I've read, yes.
Q. And when you were writing your report, you had access to those documents and you would

reference those documents as you were writing the report in real time, correct?
A. Yes.
MR. HOLLINGSWORTH: Leading. Objection, leading.
Q. (BY MS. WAGSTAFF) Did you have access to those medical records -- I mean, I'm sorry -- strike that.
Did you have access to that medical literature when you were writing your report?
A. Can I -- just for clarification, you're referring to them as medical.
Q. I'm sorry. Scientific literature.
A. Right.
Q. Let me --
A. Not specifically medical.
Q. Let me rephrase that.
A. Okay.
Q. This pharma lawyer is --
A. I just want to be clear.
Q. Did you have access to the scientific literature cited in your expert report while you were writing your expert report?
A. Yes.
Q. Okay. And today, for the past six and a

half hours, Monsanto's lawyers have asked you about that medical -- that scientific literature, correct?
A. Yes.
MR. HOLLINGSWORTH: Objection, leading.
Q. (BY MS. WAGSTAFF) And during those questions you were -- you were often asked about specific details of the scientific literature; is that right?
MR. HOLLINGSWORTH: Objection leading.
A. Yes.
Q. (BY MS. WAGSTAFF) Okay. And did you -- have you memorized those -- that scientific literature?
A. No. I have not memorized it.
Q. Okay. And did you ask Monsanto's lawyers to provide you with that scientific literature to refresh your recollection?
A. Yes.
Q. Okay. And did Monsanto's lawyers refuse?
MR. HOLLINGSWORTH: Objection, leading.
A. Yes.
Q. (BY MS. WAGSTAFF) So Monsanto's lawyers refused to provide the medical literature -- or the scientific literature that you cited in your expert

report despite asking you specific questions about it, correct?

MR. HOLLINGSWORTH: Objection, leading.

A. Yes.

Q. (BY MS. WAGSTAFF) Would it have been helpful to have that scientific literature to refresh your recollection and provide better or more comprehensive answers?

MR. HOLLINGSWORTH: Objection, leading.

A. Yes.

Q. (BY MS. WAGSTAFF) Excellent. And in fact, there were 101 scientific literature cited in your expert report; is that correct?

A. Yes.

Q. And only one of those was the Greim study; is that correct?

MR. HOLLINGSWORTH: Objection, leading.

A. Yes, only one was -- had Greim as the primary author.

Q. (BY MS. WAGSTAFF) Okay. I'm going to take you back to the beginning of the deposition, about eight or nine hours ago when this started. And do you remember Mr. Hollingsworth, Monsanto's lawyers, asking you questions about whether -- whether there have been studies to specifically test or investigate

whether a particular tumor in a rat or a mice is a good predicate for NHL in humans? Do you remember those questions?

A. Yes.

Q. And do you remember I wrote down the list of about eight or nine of them and then I quit -- I quit writing them down because the questions were throughout the entire day, but some of them were do you remember if there have been studies designed to test whether rat testicular interstitial tumors is a good predicate to cause NHL in tumors? Do you remember that question?

MR. HOLLINGSWORTH: Objection, leading.

A. Yes.

Q. (BY MS. WAGSTAFF) Do you remember the question on whether anyone has studied whether lung adenocarcinoma is a good predicate for NHL in humans?

A. Yes.

Q. And there was about four or five other ones, and what was your response to those questions?

A. Well, it was pretty much the same answer, the -- the studies that I reviewed were designed to see if glyphosate would cause cancer in the experimental animals, so the animals were exposed to glyphosate, there was an increased incidence of the

particular tumor that the question was about in -- in that animal, so therefore, glyphosate in that study glyphosate caused that cancer in experimental animals, so it's an experimental animal carcinogen, and as a -- as an animal carcinogen, it is a potential human carcinogen, so -- and to the best of my knowledge, I'm not aware of anybody that has designed studies to investigate the association of those particular tumors in the rats or the mice in non-Hodgkin's lymphoma, nor am I aware that anybody has published an article addressing that issue.

Q. Okay. So even though no -- even though to the best of your knowledge, no one has specifically tested whether those particular rodent tumors are a good predicate for NHL in humans, is this the type of information that toxicologists rely on to make a determination of whether a chemical is a human carcinogen?

MR. HOLLINGSWORTH: Objection, leading.

A. Absolutely. That is the premise of doing the bioassay that if it is shown to be a carcinogen in experimental animals, then it is potential a human carcinogen.

Q. (BY MS. WAGSTAFF) All right. Isn't it true, Dr. Jameson, that we conduct testing on

experimental animals because tumors in rodents may indicate carcinogenesis of a test chemical?

A. That's correct.

Q. And isn't it true that rodent carcinogenesis is applied to the potential for an agent to cause cancer in humans?

A. Yes.

Q. And isn't it true we test carcinogenicity of an agent in this way because it's unethical to test on humans?

A. Yes.

MR. HOLLINGSWORTH: Leading.

Q. (BY MS. WAGSTAFF) So it's accurate to say that animal bioassay general screening tests are best way for us as human to test to carcinogenicity of a chemical, correct?

MR. HOLLINGSWORTH: Objection, leading.

A. That's correct.

Q. (BY MS. WAGSTAFF) And this is very common -- is this very common in the toxicology world?

A. Yes.

MR. HOLLINGSWORTH: Objection, leading.

A. This is -- this is kind of the standard in the toxicology world used by government, academia, industry, that that is the process by which they test

a chemical to see if it causes cancer in -- cancer causes in experimental animals as a predictor of cancer in humans.

Q. (BY MS. WAGSTAFF) Okay. Isn't it true that males and females have different organs?

A. Yes, that's true. Thank goodness.

Q. And that's true in rodents and in humans?

A. Yes.

Q. Isn't it true that replication across studies doesn't look to compare males and females for tumor incidence?

A. Yes.

Q. All right. Let's talk a little bit about statistical significance --

A. Okay.

Q. -- for a moment. That phrase was tossed around a lot today by Monsanto's counsel and by yourself. Will you tell me or tell the jury and the judge sort of what your idea of statistical significance means?

A. Statistical significance is when you see a -- for example, when you're comparing tumor incidences. Statistical significance means that the incidence that you observe in the control animals --

let me turn that around.

Statistical significance is when the incidence that you see in the treated animals is higher than what you observe in the control animals, and if the incidence in the treated animals is much larger based on the mathematical calculation, much larger than in the controlled animals, then it is said to reach the statistical significance.

But what we are seeing now in the state of the science in both toxicology and epidemiology statistical significance is not playing as crucial a role in the evaluation of the data as it has in the past because people have learned to look at the -- at increased incidence as a real effect, even though it may not reach statistical significance, but it is a significant finding because it demonstrates that an increase is more than what you get when you are not exposed to the particular chemical.

Q. Okay. Now, you testified earlier today and it's in your CV that you spent a lot of time working at the NTP, right?

A. Correct.

Q. Okay. What does the NTP stand for?

A. NTP stands for the National Toxicology Program.

Q. Okay. I believe you testified earlier that while you were working for the NTP, you didn't look at glyphosate and human data; is that correct?

A. I did not look at glyphosate in human data because it was not nominated for consideration and it never came up for consideration while I was there.

Q. Okay. And how long were you at NTP roughly?

A. I was a member of the NTP from its inception in I believe it was 197 -- '77 or '78, I may be wrong, but any way, from the early '70s until I retired from the government in 2008.

Q. Okay. So that's like 35 --

A. 35, 40 years.

Q. So between 35 and 40 years you were at NTP?

A. Yes.

Q. During those 35 to 40 years at NTP, did you look at chemicals other than glyphosate and human data?

A. Absolutely. We -- as part of the review for the report on carcinogens, we routinely looked at all the available carcinogenicity data, the animal and the human epidemiology data. And as I indicated in my

report, we have criteria for sufficient -- for the human data, and for the animal data, so when we were reviewing chemicals for the report on carcinogens, we would have to evaluate the human epidemiology data to see if there was an increased incidence in tumors in humans, if it was increased, and also the same for the animals, so I -- I've looked at the epidemiology data for -- I can't estimate a number -- between 75 and 100 chemicals for the report on carcinogens.

Q. As part of your job?

A. At part of any job at the NTP, right.

Q. Do you remember numerous times today when Monsanto's lawyer would ask you whether or not you had the full study data or the pathology report when talking about a particular study?

A. Yes.

Q. And sometimes I believe you testified that you had that data and sometimes you testified that it wasn't available to you; is that correct?

A. The full data -- the full study report, yes.

Q. And in the instances when you did not have the full study data because it was not available to you or the pathology report, does that make your reliance on that study or that material unreliable?

MR. HOLLINGSWORTH: Objection, leading.
A. Does it make my -- if I didn't have the report?
Q. (BY MS. WAGSTAFF) Uh-huh.
A. If I didn't have the full report -- if I had the tumor data, tumor tables and what have you and could -- could -- could verify the -- the incidences in either the EPA or the Greim publication, the data was reliable. In no case did I feel the data wasn't reliable.
Q. I think I wrote down a quote that you said earlier which was that you had a, quote, deficiency in your report because you didn't include incidence rates -- incident -- incidence rates. Do you remember that testimony?
A. Yes.
Q. Okay. Can you tell the Court what an incidence rate is?
A. That -- the incidence rate would be listing of the incidence of the tumors in the controls and the treated animals indicating the number of tumors observed in each -- in each dose group.
Q. Okay. And even though that wasn't in your report, did you rely on that information?
A. Oh, I -- I looked at that information.

Q. Okay.
A. And maybe I used the wrong word in describing that, but, no, the numbers that I put in my report are based on the incidence rates that I reviewed in the reports. I just didn't include it in the report for some reason. But I should have.
Q. Sorry. So the incidence rates that you relied on in drafting your expert reports are in the studies themselves, correct?
A. Absolutely.
Q. Okay. Does IARC -- isn't it true that IARC does not heavily consider or weigh expert review summaries?
A. They -- well, that is true. They -- they will review or use expert summaries or review papers. That's what you're referring to are review papers. They will use review papers or look at review papers, but if they have the opportunity to go back to the original papers that the reviews were written from, they will definitely get the original papers and place more weight on the original papers than on the review of them.
Q. Is the Greim paper an expert review summary paper?
A. Yes.

Q. All right. You testified also at some point today that you developed criteria specifically for your expert report in this MDL, correct?
A. Correct.
Q. But the method -- the methodology that you created and that you used is widely recognized in the toxicology field, correct?
MR. HOLLINGSWORTH: Objection, leading.
A. That's correct.
Q. (BY MS. WAGSTAFF) Let me reask the question.
A. Okay.
Q. Does the toxicology field recognize the methodology that you used as a sound method?
A. I would --
MR. HOLLINGSWORTH: Objection.
A. I would say yes.
MR. HOLLINGSWORTH: Calls for speculation.
A. When I was writing my expert report, I wanted to make it clear within the report the criteria that I was using in evaluating the data and making -- and giving my opinion, so I -- I said I developed this criteria, but basically this criteria is based on the criteria I developed for the report on carcinogens

that was approved by the Secretary of Health and Human Services for preparing the report on carcinogens and listing materials in there as known or reasonably anticipated to be human carcinogens and also to let people know that the criteria that I developed are quite similar to also what IARC uses in their evaluation of materials and both NTP, ROC report on carcinogens criteria and IARC criteria are both widely recognized and accepted throughout the world.
Q. (BY MS. WAGSTAFF) All right. And during those IARC deliberations, the panelists knew that the AHS study did not show a statistically significant increase odds ratio, although it did show a slight increase of 1.1, was that known?
MR. HOLLINGSWORTH: Objection, leading and beyond the scope.
A. In the IARC review, AHS study was -- was discussed. It was pointed out that while there was an increase in the incidence of non-Hodgkin's lymphoma observed in that study, it was not -- not statistically significant, and so all of that information was from that study that was available at the time was considered and reviewed and is so referenced in the monograph.
Q. (BY MS. WAGSTAFF) So that information

wasn't withheld from the IARC?
A. No, it was -- no.
Q. All right. I may be -- okay.
Isn't it true that the -- let's talk about Exhibit 22-4 which Monsanto's counsel has identified as an exhibit. 22-4. Isn't it true the NTP updates its reports on carcinogens?
A. Yeah, the report is updated -- it's supposed to be updated every two years now.
Q. Okay. So if this one was dated 2004, and here we sit in the end of 2017, that means roughly at least six more versions of this have come out, give or take?
A. Well, I said it's supposed to be published every two years. I think the latest version of the report on carcinogens was the 14th, so they haven't quite made the two year cut off but that's not unusual.
Q. So at least there's three more updated versions?
A. Yes.
Q. Than this 11th version?
A. Correct.
Q. So this 11th version that we have as Exhibit 22-4 is not the most current version?

A. Not the most current, that's correct.
MS. WAGSTAFF: No more questions. I reserve some -- any if you have something new.
MR. HOLLINGSWORTH: Okay.
EXAMINATION
BY MR. HOLLINGSWORTH:
Q. Sir, you said that as an animal carcinogen as determined by the National Tox Program or IARC, then that means that it is a potential human carcinogen, true?
A. Right.
Q. What is the -- what does the term "potential" mean?
A. Means that the -- the chemical has the -- has the potential of causing cancer in humans.
Q. Does it mean that it's more probable than not that the chemical will cause cancer in humans?
A. That's the implication, yes.
Q. That's what "potential" means?
A. That's what "potential" means.
Q. Does the IARC monograph or the National Tox Program define the word "potential" in that way?
A. I'm not sure. I'd have to look at the IARC preamble to see if they define potential.

Q. You said that if a substance is shown to be a carcinogen in a experimental animal, it is a potential human carcinogen, right?
A. Correct.
Q. And that's based on the IARC and the National Tox Program evaluation?
A. Well --
Q. Excuse me.
A. I'm sorry.
Q. That's based on the IARC and National Tox Program evaluation standards; is that right?
A. I think that's pretty much an accepted premises of toxicology, that if you -- if something is found to cause cancer in experimental animals, then it's -- potentially could cause cancer in humans and should be investigated.
Q. And the word "potential" means that that if an -- if a -- if a -- excuse me. Let me start over.
By the use of the term "potential," you mean that if an experimental animal study shows cancer, it has a more than 50 percent likelihood of being a human carcinogen, true?
A. I don't know that you can put a percentage on it.

Q. When you say in your report that you've used the -- you have cited to incidence rates when you have referred in your expert witness reports to various studies, do you have that in mind?
A. Yes.
Q. Did you mean to state in your examination by Ms. Wagstaff that incidence rates are equivalent to statistical significance as used in your report?
A. No.
Q. Okay. Just wanted to make sure.
MR. HOLLINGSWORTH: Okay. That's all I have.
MS. WAGSTAFF: Really?
MR. HOLLINGSWORTH: Yeah.
MS. WAGSTAFF: Let's go off the record before I say how excited I am that we're done with this.
THE DEPONENT: Not as excited as me.
MS. WAGSTAFF: Oh, dang it, you got that on the record.
THE VIDEOGRAPHER: Going off the record. This concludes the videotape deposition of Charles W. Jameson. The time is 6:25 p.m. We are off the record.

WHEREUPON, the within proceedings were concluded at the approximate hour of 6:25 p.m. on the 21st day of September, 2017.

* * * * *

REPORTER'S CERTIFICATE

STATE OF COLORADO)
) ss.
CITY AND COUNTY OF DENVER)

I, TRACY R. STONEHOCKER, Certified Realtime Reporter, Registered Professional Reporter and Notary Public ID 19924009337, State of Colorado, do hereby certify that previous to the commencement of the examination, the said CHARLES W. JAMESON, Ph.D., was duly sworn by me to testify to the truth in relation to the matters in controversy between the parties hereto; that the said deposition was taken in machine shorthand by me at the time and place aforesaid and was thereafter reduced to typewritten form; that the foregoing is a true transcript of the questions asked, testimony given, and proceedings had.

I further certify that I am not employed by, related to, nor of counsel for any of the parties herein, nor otherwise interested in the outcome of this litigation.

IN WITNESS WHEREOF, I have affixed my signature this 22nd day of September, 2017.

____
TRACY R. STONEHOCKER
My commission expires June 12, 2020.

_____ Reading and Signing was requested.

_____ Reading and Signing was waived.

__X__ Reading and Signing is not required.

ERRATA SHEET

Case Name:
Deposition Date:
Deponent:

Pg.	No.	Now Reads	Should Read	Reason
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Signature of Deponent

SUBSCRIBED AND SWORN BEFORE ME
THIS ____ DAY OF __________, 2017.

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(Notary Public) MY COMMISSION EXPIRES:__________

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UNITED STATES DISTRICT COURT

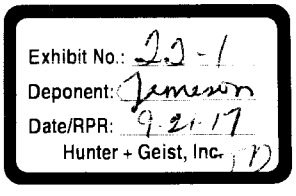
NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	MDL No. 2741 Case No. 16-md-02741-VC
This document relates to: ALL ACTIONS	

EXPERT REPORT OF DR. CHARLES W. JAMESON, Ph.D.

IN SUPPORT OF GENERAL CAUSATION

ON BEHALF OF PLAINTIFFS



Charles William Jameson, Ph.D.
Independent Consultant
May 12, 2017

Statement of Purpose

I have been asked to provide my expert opinions regarding the carcinogenic potential of glyphosate and glyphosate-based formulations. As a chemist and toxicologist, I evaluated the association of cancer, including non-Hodgkin's lymphoma ("NHL"), with exposure to glyphosate and/or glyphosate-based formulations. In performing my analysis, I relied on standard methods used in toxicology. I reviewed published, peer-reviewed scientific literature, publically available Government and Industry documents, and internal company documents and studies provided to me. All my opinions expressed in this report are based on a reasonable degree of scientific certainty. I reserve the right to supplement this report if additional information becomes available that are relevant to my opinions.

Qualifications

I am a private consultant in environmental toxicology specializing in carcinogenesis. I received my undergraduate degree in chemistry in 1970 from Mount Saint Mary's College, Emmitsburg, Maryland, and my Ph.D. in Organic Chemistry in 1975 from the University of Maryland, College Park. I started my career in 1965 where, as a rising high school senior, I spent the summer at a bioassay research laboratory first as a mouse room tech cleaning cages and later as an assistant in the chemistry lab mixing pesticides in rodent feed for the bioassay studies. Upon completion of my Ph.D. and a brief post-doc at the University of Maryland, I began working in 1976 as a contractor to the National Institutes of Health's (NIH) National Cancer Institute (NCI), serving as a senior chemist in support of NCI's Rodent Bioassay Program. In this capacity I was responsible for helping to monitor and evaluate the chemistry performed at the NCI's contract bioassay laboratories. In addition, I also provided support to the NCI staff for the identification of new substances to be studied in the NCI Bioassay Program. This

support included preparing and providing the background data from the scientific literature concerning exposure and the carcinogenic potential of the substance of interest. I was recruited by, and joined, the NCI in 1979 to serve as the chief chemist for their Rodent Bioassay Program and was responsible for directing and monitoring all chemistry activities, participating in the development of experimental protocols for the 2 year rodent bioassays conducted at the contract laboratories, and doing on-site inspections of all bioassay contract labs to insure they were following our protocols. In addition, I took over the responsibility as secretary for the NCI's Chemical Selection Working Group (CSWG) where I coordinated all activities for the identification of new substances to be studied in the Bioassay Program, including the oversight of the scientific literature searching, gathering and summarization process, documentation of the CSWG's review of the data and recommendations for study by the NCI, and the forwarding of the recommendation to the Director of the NCI Bioassay Program.

Following the formation of the National Toxicology Program (NTP) in 1978, the NCI Rodent Bioassay Program was transferred to the NIH's National Institute of Environmental Health Sciences (NIEHS) in 1980 where I transferred to and assumed the responsibility for all chemistry aspects of the NIEHS Division of Toxicology Research and Testing. I served as the program leader for chemistry in the National Toxicology Program (NTP) from 1978 until 1990. While chemistry program leader, I developed chemistry standards for bioassay studies that were widely accepted as an integral part of many toxicology-testing programs. I am listed as a contributor for the evaluation, interpretation and reporting of results for more than 100 chemicals studied in chronic two-year bioassay studies by the National Toxicology Program as published in the Technical Report Series (1980-1990). These bioassay studies were peer reviewed by the NTP Board of Scientific Counselors.

In 1990, I transferred to the NIEHS Director's Office and became involved with the NTP's Report on Carcinogens (RoC), working on it for more than 18 years, serving as its Director for 13 years before retiring from the NIEHS in February of 2008. The RoC is prepared in response to Section 301(b)(4) of the Public Health Service Act, which stipulates that the Secretary of the Department of Health and Human Services (DHHS) shall publish a report which contains a list of all substances which either are known or may reasonably be anticipated to be human carcinogens; and to which a significant

number of persons residing in the United States are exposed. This responsibility has been delegated by the Secretary to the Director, NTP. As Director of the RoC, I was responsible for the report's overall preparation, review and approval for the Director, NIEHS/NTP. In this capacity, I coordinated all review activities related to the RoC, which is one of the most visible and highly scrutinized activities of the NTP and the DHHS. I oversaw the identification and review of all new nominations for listing and delisting in upcoming editions of the RoC. I served as Chairman of the NIEHS RoC Review Committee, Chairman of the NTP Executive Committee's Interagency Working Group for the RoC, and Advisor to the NTP's Board of Scientific Counselors' Subcommittee for the RoC. I supervised the review of each nomination to the RoC, insuring all relevant information and data for each nomination was available for the review committees and managed the reviews by the three scientific review committees. Shortly after I became Director of the RoC in 1995, the Director, NTP, ordered that a review of the RoC be done to broaden input into its preparation, broaden the scope of scientific review associated with the Report, and provide review of the criteria used for inclusion of substances in the RoC. I coordinated this activity, which lead to revised criteria for the RoC being approved by the Secretary, DHHS in July of 1996. I served as Project Officer for the resource support contract for the preparation of the RoC, which included providing technical direction and coordination of the preparation of the documents prepared for each new nomination to the RoC as well as the preparation of 4 editions of the RoC for submission to the DHHS Secretary for approval.

I am the Senior Author for 69 NTP Report on Carcinogens Background Documents, which contained all available data concerning the exposure and potential carcinogenic activity of the substance being reviewed for possible listing in the RoC. I maintained a continuing liaison with other government agencies, private industries, other non-government research organizations and international organizations to keep abreast of work being done in chemical carcinogenesis, priorities for the listing of substances in the RoC, and resources available for the review of substances nominated for listing in the RoC. I served as the point of contact and focus for all RoC activities which included interacting with stakeholders from national and international government, industry, legal, consumer advocate, and other private concerns. I responded to requests for information from both the national and international press and private individuals on a

routine basis. Upon my retirement in 2008, I established CWJ Consulting LLC as a vehicle for providing expert consulting services in environmental toxicology specializing in carcinogenesis.

During my career, I participated as a Working Group Member for the United Nations' World Health Organization (WHO) International Agency for Research on Cancer (IARC). On several occasions, I served as either overall Chair of the Working Group or Chair of the Subgroup for Cancer in Experimental Animals evaluating cancer data and publishing monographs of the evaluation. I served as a consultant to the WHO, serving as a Task Group member to develop Environmental Health Criteria documents for partially halogenated chlorofluorocarbons (freons).

I am the author or co-author of over 80 peer reviewed scientific publications and nine book chapters. The vast majority of these publications relate to studies conducted in support of animal carcinogenesis bioassay programs. As mentioned above, I was the editor of four editions of the RoC, senior author for 69 NTP RoC Background Documents for substances reviewed for listing in the Report and listed as a contributor for the evaluation, interpretation and reporting of results for more than 100 chemicals studied in chronic two-year bioassay studies by the NTP as published in the Technical Report Series (1980-1990). I co-edited two books: "Chemistry for Toxicity Testing" and "Health and Safety for Toxicity Testing." A copy of my current curriculum vitae is attached as Exhibit A.

International Agency for Research on Cancer (IARC)

As an introduction, I would like to explain the International Agency for Research on Cancer's (IARC) review of glyphosate to assess its potential carcinogenicity, and the development of Monograph 112. The Working Group classified glyphosate as "probably carcinogenic to humans" (Group 2A) at their meeting in March of 2015. Following this meeting, there have been a number of publications (including, but not limited to, Williams et al.[1, 2]; Chang and Delzell[3], Solomon[4]) criticizing the IARC review process and conclusions.

The purpose of the *Monographs* is to render critical reviews and evaluations of carcinogenicity evidence of a wide range of human exposures.[5] The *Monographs*

represent a hazard identification that involves examination of all relevant information to assess the strength of the available evidence that an agent can cause human cancer. Identifying carcinogens is a key step in cancer prevention, and this activity represents an important international activity towards improving public health. The IARC Preamble[5] states that a "cancer 'hazard' is an agent that can cause cancer under some circumstances, while a cancer 'risk' is an estimate of the carcinogenic effects expected from exposure to a cancer hazard. The *Monographs* are an exercise in evaluating cancer hazards, despite the historical presence of the word 'risks' in the title. The distinction between hazard and risk is important, and the *Monographs* identify cancer hazards even when risks are very low at current exposure levels, because new uses or unforeseen exposures could engender risks that are significantly higher." In other words, hazard assessment determines whether an agent can cause cancer.

For the review of glyphosate as it relates to Monograph 112, IARC perfomed a search for all relevant biological and epidemiological data from publically available sources and sent copies of the materials found to the Working Group participants approximately six months prior to the start of the meeting. In addition to the materials sent from IARC, Working Group participants perform their own independent search of the scientific literature. As the IARC Preamble notes, "with regard to epidemiological studies, cancer bioassays, and mechanistic and other relevant data, only reports that have been published or accepted for publication in the openly available scientific literature were reviewed."[5] IARC also considers relevant and publically available material from US Environmental Protection Agency ("EPA"). Studies determined to be irrelevant, inadequate, or published too late to be adequately evaluated were cited but were not summarized. This process of data collection is typical of all IARC *Monographs* and is the body of literature used by the Working Group participants during each Monograph anaylsis.

The IARC Working Group meeting takes places at its headquarters in Lyon, France and lasts for approximately seven to eight days, where the Working Group will then finalize the texts and formulate its final evaluations. Participants are assigned to one of four subgroups covering either exposure data, cancer in humans, cancer in experimental animals, or mechanistic and other relevant data. Working Group participants are also assigned individual chemicals or agents being evaluated and asked to prepare preliminary

working papers for their specific subgroup that are then distributed prior to the meeting. The subgroups prepare joint drafts and summaries in breakout sessions during the first few days. The entire Working Group meets in brief plenary sessions every day to get updates on the progress of each individual subgroup and to discuss any issues the subgroups may have identified. The final days of the meeting consists of plenary session meetings to discuss all relevant data, review the subgroup drafts and develop the final evaluations. The entire Monograph volume is considered the joint product of the Working Group, and there are no individually authored sections.[5]

For Monograph 112, I served as Chairman of the subgroup for Cancer in Experimental Animals to assess the carcinogenicity of several organophosphate pesticides that included glyphosate, the active ingredient in Roundup®. This meeting was held March 3-10, 2015 and the Working Group classified glyphosate as "probably carcinogenic to humans" (Group 2A). This classification was based on limited evidence in humans for the carcinogenicity of glyphosate where a positive association has been observed for NHL, sufficient evidence in experimental animals for the carcinogenicity of glyphosate and that mechanistic and other relevant data support the classification of glyphosate in Group 2A. To provide a better understanding of this, I will: discuss the process used by the Working Group to arrive at this classification, define terms, explain the types of evidence considered, explain the scientific criteria that guide the evaluations, and explain how conclusions were reached throughout the process.

The following summary of the Working Group's evaluation of the available literature is offered here, but also found in the IARC's Preamble[5]:

•Exposure Data: The Working Group concluded there is wide spread exposure to glyphosate based on its use as the active ingredient in Roundup® which is a broad-spectrum herbicide. Glyphosate is the most heavily used herbicide in the world[6] and can be found in soil, air, surface water, groundwater, and food. According to several studies, glyphosate has also been detected in urine from persons around the world.[7-10] The general population is mainly exposed to glyphosate through diet and from use as a household weed control.

•Cancer in Humans: The Working Group identified seven reports from the Agricultural Health Study (AHS) cohort and numerous reports from case-control studies

in the evaluation of the epidemiological studies reporting on cancer risks associated with exposure to glyphosate. This Working Group applied the Bradford Hill criteria in its analyses and determined that in several case–control studies there was an increased risks for NHL due to glyphosate exposure.[11-18] The Working Group further noted that the increased risk for NHL persisted in the studies that adjusted for exposure to other pesticides. The Working Group concluded a positive association has been observed for exposure to glyphosate and NHL and that there is "limited evidence" in humans for the carcinogenicity of glyphosate. IARC determines limited evidence of carcinogenicity for an agent when "a positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence."[5]

•Cancer in Experimental Animals: The Working Group reviewed scientific literature and reports including two studies in which glyphosate was reported to be tested for carcinogenicity in male and female mice by dietary administration, five studies that tested glyphosate in male and female rats by dietary administration and in drinking-water in one study. Studies of a glyphosate-based formulation tested in drinking-water in one study in male and female rats and by skin application in one initiation–promotion study in male mice were also reviewed. They observed that in one feeding study in male CD-1 mice,[19-22] glyphosate induced a positive trend in the incidence of kidney renal tubule carcinoma, a rare tumor in this strain of mice. A second feeding study[23] reported a positive trend for hemangiosarcoma (a blood vessel tumor) in male mice. Glyphosate also increased pancreatic islet-cell adenoma in male rats in two feeding studies.[24-26] The Working Group concluded there is "sufficient evidence" in experimental animals for the carcinogenicity of glyphosate. IARC defines "sufficient evidence" in experimental animals is as "a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms in (a) two or more species of animals or (b) two or more independent studies in one species carried out at different times or in different laboratories or under different protocols."[5]

•Mechanistic and Other Relevant Data: The Working Group reported the mechanistic data literature contained strong evidence that glyphosate causes genotoxicity

and oxidative stress. The strong evidence of genotoxicity came from studies conducted in human cells in vitro,[27-32] in mammalian model systems in vivo[27,32] and in vitro,[33,34] and from studies in other non-mammalian organisms[29,35,36,37], all of which yielded largely positive results. The Working Group also found strong evidence for genotoxicity caused by glyphosate-based formulations. There were three studies of genotoxicity end-points in community residents exposed to glyphosate-based formulations, two of which reported positive associations.[38,39] Strong evidence for oxidative stress was determined by studies conducted in human cells in vitro[28,40,41] and in many rodent tissues in vivo.[32,42,43] The Working Group found weak evidence that glyphosate or glyphosate-based formulations induce receptor-mediated effects,[44,45] may affect cell proliferation or death,[44,46] and may also affect the immune system in rodents[47] and fish.[48,49] The Working Group considered the body of evidence described above as a whole and reached an overall evaluation of Group 2A: glyphosate is probably carcinogenic to humans. IARC uses this category when evidence of carcinogenicity in humans is limited and evidence of carcinogenicity in experimental animals is sufficient.[5]

IARC uses the hazard identification process for its review, and this was done for Monograph 112. Hazard identification reflects the toxicological "law" of specificity of effects[50]. Hazard identification uses a strength of the evidence approach. As applied, the Working Groups for Monograph 112 rigorously assessed the toxicological, mechanistic, and epidemiological data to form a judgment regarding the likelihood that the agent produces cancer.

Information Reviewed

During the course of work on this case, I reviewed the following materials:

- scientific literature relating to the carcinogenicity of glyphosate and/or glyphosate-based formulations;
- government documents relevant to assessing the carcinogenic hazard and risks associated with glyphosate and/or glyphosate-based formulations; and,
- various studies and documents produced in the litigation.

For a list of additional materials I reviewed, please see Exhibit B.

Description of the Methodology Used to Assess Carcinogenic Potential Associated with Exposure to Glyphosate and/or Glyphosate-Based Formulations.

Toxicologists routinely assess the hazards to human health related to exposure to chemicals in the everyday environment using a process called hazard identification. A hazard is any agent that can cause harm or damage to humans, property, or the environment.[51] In other words, a hazard is any agent that can cause a specific damage. In this case, the hazard being examined is glyphosate and/or glyphosate-based formulations, the specific damage is NHL, and the hazard assessment I am making is to determine whether or not glyphosate and/or glyphosate-based formulations can cause NHL. The terms hazard and risk are often used interchangeably; however, these are two distinct terms. Risk is defined as the probability that exposure to a hazard will lead to a negative consequence, or more simply, risk = hazard x dose (exposure).[52]

Toxicology is the basis on which hazard identification is established. Hazard assessment has been used for over four decades by a wide variety of governmental and nongovernmental organizations to evaluate the potential adverse health effects from chemical exposures. Hazard identification is a standard tool used by toxicologists when they are trying to determine if exposure to a chemical(s) can cause an adverse health effect in humans and is the first step in risk analysis. Hazard identification is performed by identifying the chemical someone has been exposed to and then reviewing the available toxicity data to outline the spectrum of adverse effects that would be associated with exposure to that particular chemical.[53] The toxicity data could be from studies in humans, in whole animals, or in cells, or could be data collected on chemically-similar substances when data on the chemical of interest are limited.

I used the following criteria for my hazard based assessment of glyphosate and/or glyphosate-based formulations, that is based on the criteria I developed for the Report on Carcinogens[54] and is the same as defined and characterized by IARC[5]:

• Cancer in Humans – Numerous case-control studies and the Agricultural Health Study (AHS) cohort reporting on possible associations of cancer and exposure to glyphosate were evaluated for any evidence of a causal relationship between glyphosate and human cancer.

- "Sufficient" evidence is defined as when a causal relationship was established between exposure to glyphosate and cancer and that chance, bias and confounding could be ruled out.[5]
- "Limited" evidence is defined as a positive association has been observed between exposure to glyphosate and cancer and a causal interpretation is credible but alternative explanations such as chance, bias or confounding could not be ruled out.[5]
- "Inadequate" evidence is defined as available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding a causal association between glyphosate exposure and cancer.[5]

• Cancer in Experimental Animals – the experimental animal studies reporting on possible associations of cancer and exposure to glyphosate were evaluated for any evidence of a causal relationship between glyphosate and cancer.

- "Sufficient" evidence is defined as a causal relationship between exposure to glyphosate and an increased incidence of malignant and/or a combination of malignant and benign tumors, in multiple species or at multiple tissue sites or from multiple studies, or by multiple routes of exposure, or to an unusual degree with regard to incidence, site, or type of tumor, or age at onset.[5]
- "Limited" evidence is defined as the data suggest a carcinogenic effect but are limited for making a definitive evaluation because, e.g. the evidence of carcinogenicity is restricted to a single experiment; there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the studies; or the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potentials.[5]
- "Inadequate" evidence is defined as studies that cannot be interpreted to show either the presence or absence of a positive carcinogenic effect because of major qualitative or quantitative limitations such as inadequate numbers of animals, lack of adequate pathology, poor survival, major impurities in the test agent, too low a dose to see an effect, etc. It should be noted that although animal testing is routinely used to identify cancer hazard, the sites

> of cancer observed in animals do not always correlate directly with the sites of cancer that would be observed in humans[55]. This can be due to the differences in metabolism in laboratory animals and humans, differences in pharmacokinetics, or differences in tissue reactivity (pharmacodynamics) between species. Animal studies, instead, are used to identify a threat of cancer that is applied to human health hazard assessment[55]. All chemicals known to induce cancer in humans, that have been studied under adequate experimental conditions, also cause cancer in laboratory animals[55] and underscores the concept that chemicals found to cause cancer in laboratory animals must be considered capable of causing cancer in humans.[5]

•Mechanistic and other data – studies containing data relevant to the possible mechanim(s) of glyphosate carcinogenesis (genetic toxicity, epigenetic effects, etc.) were also evaluated. Mechanistic data may provide evidence of carcinogenicity and help in assessing the relevance and importance of findings of cancer in animals and humans.[5]

Hazard Assessment of the Human Data for Glyphosate and/or Glyphosate-Based Formulations

Before discussing the human data for glyphosate and/or glyphosate-based formulations, I will define the type of epidemiology studies that were reviewed:

• Case-Control Study - In a case-control study, investigators start by enrolling a group of people with disease. As a comparison group, the investigator then enrolls a group of people without disease (controls). Investigators then compare previous exposures between the two groups. The control group provides an estimate of the baseline or expected amount of exposure in that population. If the amount of exposure among the case group is substantially higher than the amount you would expect based on the control group, then illness is said to be associated with that exposure. The key in a case-control study is to identify an appropriate control group, comparable to the case group in most respects, to provide a reasonable estimate of the baseline or expected exposure.[56]

• Cohort Study - According to Centers for Disease Control and Prevention (CDC),[57] in a cohort study the epidemiologist records whether each study participant is exposed or not, and then tracks the participants to see if they develop the disease of interest. After a

period, the investigator compares the disease rate in the exposed group with the disease rate in the unexposed group. The unexposed group serves as the comparison group or control, providing an estimate of the baseline or expected amount of disease occurrence in the community. If the disease rate is substantively different in the exposed group compared to the unexposed group, the exposure is said to be associated with illness.

• Meta-Analysis – A meta-analysis is an important component of systematic review procedure that combines and analyzes quantitative and qualitative data from several separate but similar experiments or studies to test the pooled data for statistical significance. Combining the results of multiple studies produces a weighted average of the included study results and leads to a conclusion with greater statistical power and point estimate than would be possible from any individual study.

Case Control Studies

• Cantor et al. (1992)[14] evaluated the incidence of NHL among males located in Iowa and Minnesota. A total of 622 men and 1245 population-based controls were included in the study. The association with farming occupation and specific agricultural exposures were evaluated. When compared with non-farmers, the positive associations (odds ratios) for NHL were significant at 1.2 (95% CI, 1.0–1.5) for men who had ever farmed, and not significant at 1.1 (95% CI, 0.7–1.9) for 26 exposed cases for ever handling glyphosate and adjusted for confounders (vital status, age, state, cigarette smoking status, family history of lymphohaematopoietic cancer, high-risk occupations, and high-risk exposures).

•DeRoos et al. (2003)[11] pooled the data from three case–control studies[12-14] to study pesticide exposures as risk factors for NHL in men. Of a total study population of 870 cases and 2569 controls, there were 650 cases and 1933 controls included for the analysis of 47 pesticides that also controlled for potential confounding by other pesticides. A positive association (odds ratios) for the association between exposure to glyphosate and NHL in the 36 cases exposed was reported to be significant at 2.1 (95% CI, 1.1–4.0) in the logistic regression analyses but not in the hierarchical regression analysis (which uses a more conservative adjustment estimate) at 1.6 (95% CI, 0.9–2.8).

•The effect of asthma as a modifier of the association between pesticide exposure and NHL was reported on by Lee et al. (2004)[58]. The study contained 872 cases diagnosed

with NHL, 45 of which had been told they also had asthma and 2381 matched controls, 132 reporting to have asthma. Individuals in the study group with a history of asthma had a non-significantly lower risk of NHL than non-asthmatics and no effect was seen with pesticide exposure. A positive associations (odds ratio) for NHL associated with glyphosate use were reported but were not significant at 1.4 (95% CI, 0.98–2.1; 53 exposed cases) among non-asthmatics and 1.2 (95% CI, 0.4–3.3; 6 exposed cases) for asthmatics, when compared with non-asthmatic non-exposed farmers.

•The associations between exposure to pesticides and NHL was studied by McDuffie et al. (2001)[15] in a multicenter population-based study that included 517 cases and 1506 controls among men of six Canadian provinces. A non-significant positive association (odds ratios) of 1.26 (95% CI, 0.87–1.80; 51 exposed cases; adjusted for age and province) and 1.20 (95% CI, 0.83–1.74, adjusted for age, province, high-risk exposures) were observed for exposure to glyphosate. In an analysis by frequency of exposure to glyphosate, participants with more than 2 days of exposure per year had a statistically significant positive association (odds ratio) of 2.12 (95% CI, 1.20–3.73, 23 exposed cases) compared with those with some, but less than 2 days of exposure.

•Nordstrom et al (1998)[59] conducted a study in Sweden on hairy cell leukemia (considered to be a subtype of NHL). There were 121 cases in men and 484 controls matched for age and sex. A non-significant age-adjusted positive association (odds ratio) of 3.1 (95% CI, 0.8–12; 4 exposed cases) was reported for exposure to glyphosate.

•Hardell and Eriksson (1999)[60] reported on the results of the incidence of NHL in men associated with pesticide exposure in four northern counties in Sweden and included 404 cases and 741 controls. The authors reported a non-significant positive association (odds ratio) for ever-use of glyphosate of 2.3 (95% CI, 0.4–13; 4 exposed cases) in an analysis of glyphosate only, and 5.8 (95% CI, 0.6–54) in a multivariable analysis.

•Hardell et al. (2002)[17] performed a pooled analysis of two case–control studies, one on NHL[60] and another on hairy cell leukemia.[59] These pooled analyses were based on 515 cases and 1141 controls. A significant positive association was found for exposure to glyphosate compared to controls (odds ratio, 3.04; 95% CI, 1.08–8.52; 8 exposed cases), but the positive association (odds ratio) decreased to a non-significant 1.85 (95% CI, 0.55–6.20) when study area, and vital status were considered.

•A large population based case–control study of exposure to pesticides as a risk factor for NHL in Sweden was conducted by Eriksson et al. (2008)[18]. There were 910 cases and 1016 controls included in the study. The association (odds ratio) for exposure to glyphosate to NHL was positive and significant at 2.02 (95% CI, 1.10–3.71) compared to controls, but positive and non-significant at 1.51 (95% CI, 0.77–2.94) when confounders that included exposure to other pesticides, age, sex, and year of diagnosis or enrolment were included in the analysis. When exposure to glyphosate for more than 10 days per year was considered, the positive association (odds ratio) was significant at 2.36 (95% CI, 1.04–5.37). Considering a latency period of greater than 10 years gave a positive association (odds ratio) that was also significant at 2.26 (95% CI, 1.16–4.40). The authors also reported an association with exposure to glyphosate and lymphoma subtypes. Positive associations were reported for most of the cancer forms, including B-cell lymphoma (odds ratio of 1.87; 95% CI, 0.998–3.51, non-significant) and the subcategory of small lymphocytic lymphoma/chronic lymphocytic leukemia (odds ratio of 3.35; 95% CI, 1.42–7.89, significant). These odds ratios were not adjusted for other pesticides.

•Orsi et al. (2009)[61] reported the results of a case–control study conducted in France. The study included 491 cases (244 cases of NHL, 87 cases of Hodgkin lymphoma, 104 of lymphoproliferative syndrome, and 56 cases of multiple myeloma), and 456 age- and sex-matched controls. Positive, non-significant associations (odds ratios) for any exposure to glyphosate were reported: 1.2 (95% CI, 0.6–2.1; 27 exposed cases) for all lymphoid neoplasms combined, 1.0 (95% CI, 0.5–2.2; 12 exposed cases) for NHL, 0.6 (95% CI, 0.2–2.1; 4 exposed cases) for lymphoproliferative syndrome, 2.4 (95% CI, 0.8–7.3) for multiple myeloma, and 1.7 (95% CI, 0.6–5.0; 6 exposed cases) for Hodgkin lymphoma, after adjusting for age, and socioeconomic category.

•Cocco et al. (2013)[62] performed a pooled analysis of case–control studies from six European countries to investigate the role of occupational exposure to specific groups of chemicals in the causation of lymphoma overall, B-cell lymphoma, and its most prevalent subtypes. A total of 2348 incident cases of lymphoma and 2462 controls were included in the study. Analyses were conducted for lymphoma and the most prevalent lymphoma subtypes and adjusted for age, sex, and education. A positive, non-significant association (odds ratio) of 3.1 (95% CI, 0.6–17.1) was reported for exposure to glyphosate and B-cell lymphoma.

I would note that the findings in the McDuffie et al. (2001)[15]; and Eriksson et al.[18] studies is significant because their results are supported by the results reported for micronucleus formation studies in the bone marrow of mice by Rank et al. (1993)[63] where a single dose caused no effect while Bolognesi et al. (1997)[32] and Manas et al. (2009)[27] reported that two daily doses of glyphosate did cause micronucleus formation in the bone marrow of mice in their studies. This implies that level of exposure is an important consideration in the formation of NHL from exposure to glyphosate.

Cohort Studies

The Agricultural Health Study (AHS)[64] is a large prospective study of cancer and other health outcomes in a cohort of licensed pesticide applicators and their spouses from Iowa and North Carolina. The AHS began in 1993 with the goal of answering important questions about how agricultural, lifestyle and genetic factors affect the health of farming populations. More than 89,000 farmers and their spouses in Iowa and North Carolina have participated in the study. It is the only cohort study to date to have published findings on exposure to glyphosate and the risk of cancer at many different sites. My summary of the 7 papers available evaluating cancer incidence associated with pesticide use in the AHS cohort follows:

•No risk estimates and no significant exposure-response associations with cancer of the prostate and exposure to glyphosate were reported by Alavania et al (1996).[65]

•DeRoos et al. (2005)[66,67] evaluated associations between glyphosate exposure and the incidence of cancer at multiple sites in this cohort including lung, melanoma, multiple myeloma, and NHL, oral cavity, colon, rectum, pancreas, kidney, bladder, prostate, and leukemia. No significant exposure–response association with cancer at any of these sites was found.

•Flower et al.,[68] reported the results of the analyses of risk of childhood cancer associated with pesticide application by the parents of 17,357 children of Iowa pesticide applicators from the AHS cohort. For all the children of the pesticide applicators, the risk of cancer was increased for all childhood cancers combined, for all lymphomas combined, and for Hodgkin lymphoma, compared with the general population. A non-significant association (odds ratio) for use of glyphosate and risk of childhood cancer was reported to be 0.61 (95% CI, 0.32–1.16; 13 exposed cases) for maternal use and 0.84 (95% CI, 0.35–

2.34; 6 exposed cases) for paternal use.

• The incidence of cancer of the breast among farmers' wives in the AHS cohort, which included 30,454 women with no history of cancer of the breast before enrolment was reported by Engel et al.,[69]. There was no difference in incidence of breast cancer for women who reported ever applying pesticides compared with the general population. A non-significant association (relative risk) for cancer of the breast was reported to be 0.9 (95% CI, 0.7–1.1; 82 cases) among women who had personally used glyphosate and a non-significant positive association (relative risk) of 1.3 (95% CI, 0.8–1.9; 109 cases) among women who never used pesticides but whose husband had used glyphosate.

•Lee et al.,[70] studied the relationship between exposure to agricultural pesticides and incidence of cancer of the colorectum in the AHS cohort. Non-significant positive associations (relative risks) with exposure to glyphosate was reported to be 1.2 (95% CI, 0.9–1.6) for cancers of the colorectum, and 1.6 (95% CI, 0.9–2.9) for cancers of the rectum. A non-positive association of 1.0 (95% CI, 0.7–1.5) was reported for cancers of the colon.

•Andreotti et al.,[71] used a case–control analysis nested in the AHS cohort to study associations between the use of pesticides and cancer of the pancreas. For pancreatic cancer, a positive association (odds ratio) for ever- versus never-exposure to glyphosate was found but not significant at 1.1 (95% CI, 0.6–1.7; 55 exposed cases) and for highest category of level of intensity-weighted lifetime days was also found but not significant at 1.2 (95% CI, 0.6–2.6; 19 exposed cases).

•Dennis et al.,[72] reported that exposure to glyphosate was not associated with cutaneous melanoma within the AHS cohort but did not report a risk estimate.

Meta-Analyses

•Schinasi & Leon[73] conducted a systematic review and meta-analysis of NHL and occupational exposure to agricultural pesticides, including glyphosate. The meta-analysis for glyphosate included six studies (McDuffie et al.,[15] Hardell et al.,[17] DeRoos et al.,[67,11] Eriksson et al.,[18] and Orsi et al.[61]) and yielded a significant positive asso ciation (meta risk-ratio) of 1.5 (95% CI, 1.1–2.0) for exposure to glyphosate and NHL.

•IARC[74] conducted an additional meta-analysis of NHL and occupational exposure to agricultural pesticides, including glyphosate using data from Schinasi & Leon[73] and

included the fully adjusted risk estimates from the studies published by Hardell et al.,[17] and Eriksson et al.[18] After considering the adjusted estimates of the two Swedish studies in the meta-analysis, the positive association (meta risk-ratio) was still significant at 1.3 (95% CI, 1.03–1.65).

•Chang and Delzell[3] also conducted a systematic review and meta-analysis to examine the relationship between glyphosate exposure and risk of lymphohematopoietic cancer including NHL, Hodgkin lymphoma, multiple myeloma, and leukemia. Their analysis showed a positive association (meta-relative risks or meta-RRs) and was statistically significant for the association between any versus no use of glyphosate and risk of NHL (meta-RR=1.3, 95% confidence interval (CI)=1.0–1.6, based on six studies) and multiple myeloma (meta-RR =1.4, 95% CI=1.0–1.9; four studies). The authors conducted four meta-analyses for NHL, all reporting to have a significant positive association (meta-RR) of 1.3 or 1.4 with 95% CIs ranging from (1.0-1.6) to (1.0-1.8). The authors concluded "we found marginally significant positive meta-RRs for the association between glyphosate use and risk of NHL."

Summary for Human Data

I have evaluated available epidemiology data. Based on my experience doing hazard assessments, I learned that epidemiologists consider case–control studies particularly valuable for determining the carcinogenicity of an agent because their design facilitates exposure assessment and reduces the potential for certain biases. My review of the literature finds that the two case-control studies from the United States and Canada, and the two case–control studies from Sweden indicated statistically significant positive associations between exposure to glyphosate and NHL. The Canadian study, McDuffie (2001)[15], reported a positive association between glyphosate exposure and NHL for those case subjects with more than two days/year of exposure (odds ratio of 2.12 (95% CI, 1.20–3.73) when compared to those with less than two days exposure. Three studies reported excesses for NHL associated with exposure to glyphosate, after adjustment for other pesticides, De Roos (2003) reported a significant positive association (odds ratio) for a pooled US study[11] at 2.1 (95% CI, 1.1–4.0).; and the two Swedish studies (Hardell (2002)[17], Eriksson (2008)[18]) reported significant positive associations of 3.04; 95% CI, 1.08–8.52

and 2.36 (95% CI, 1.04–5.37). The positive association from Hardell (2002)[17] decreased to non-significance (1.85 (95% CI, 0.55–6.2)) when study area, and vital status were considered. Subtype-specific analyses in a Eriksson (2008)[18] indicated positive associations for total NHL, as well as all subtypes, but this association was statistically significant only for the subgroup of lymphocytic lymphoma/chronic lymphocytic leukemia (odds ratio, 3.35; 95% CI, 1.42–7.89). A European study[62] based on few cases also indicated an elevated risk (OR, 3.1; 95% CI, 0.6–17.1) for B-cell lymphoma. A French hospital-based case–control study[61] did not find an association between exposure to glyphosate and NHL (OR, 1.0; 95% CI, 0.5–2.2) based on few exposed cases. For the evaluation of glyphosate, the Agricultural Health Study (AHS) is currently the only cohort study available providing information on its potential carcinogenicity and did not show an excess of NHL. There were three groups that did meta-analyses of the human data for an association between glyphosate use and NHL. Schinasi and Leon[73] reported a significant positive association (meta-RR) of 1.5 (95% CI, 1.1–2.0). The IARC study[74] showed a positive association (meta-RR) of 1.3 (95% CI, 1.03-1.65). Chang and Delzel[3] provided four separate meta-analyses, all of which are reported as having a significant association (meta-RR) of either 1.3 or 1.4 with CIs ranging from (1.0-1.6) to (1.0-1.8). When the data across all epidemiological studies are combined, results indicate a positive association between glyphosate exposure and NHL in humans.

Interpreting the epidemiology findings requires one to properly weight studies according to quality rather than simply count the number of positives and negatives. The pooled case–control analysis from the USA[11] contained 650 cases of NHL. It follows that the case-control studies provide a stronger assessment of the potential carcinogenicity of glyphosate. The case-control studies in the US[11], Canada[15] and Sweden[17,18] indicate a significant positive association for NHL with exposure to glyphosate. This positive association was also observed in the studies that adjusted for other pesticides. The AHS cohort did not show an excess of NHL; however it reports on only 92 NHL cases in the unadjusted analysis.[64] The three meta-analyses I reviewed are good examples of objective evaluations and show a consistent positive association between glyphosate and NHL. Drawing on the Bradford-Hill criteria[75] for causality, I would state that the observations are consistent (relative risks and meta analyses are positive for the case control studies), significant, not specific, temporally observed, shows a biological gradient, and is coherent

with the animal evidence (discussed below). Using my stated criteria, I conclude there is "Limited" evidence for the carcinogenicity of glyphosate in humans, because a positive association has been observed between exposure to glyphosate and NHL, and a causal interpretation is creditable but alternative explanations such as chance, bias or confounding could not be completely ruled out.

Hazard Assessment of the Experimental Animal Data for Glyphosate and/or Glyphosate-Based Formulations

Before discussing the experimental animal data for glyphosate and/or glyphosate-based formulations, I will define what is involved in a cancer bioassay in experimental animals. The basic cancer bioassay design has remained relatively constant for more than 40 years and consists of groups of 50 male and female mice and rats in each dose and control group. Treatment traditionally lasts for 24 months and commences when the animals are 6–8 weeks of age. Early bioassay studies involved two treatment groups plus a control group. The first treatment group was a high dose, referred to as a maximally tolerated dose (MTD), and the second treatment group was half that dose. More recent studies typically include three (and sometimes up to five) treatment groups plus the control group.

In the bioassays, I reviewed the nature and extent of impurities or contaminants, the animal species, strain, sex, numbers per group, age at start of treatment, route of exposure, dose levels, duration of exposure, survival and information on tumors. With regard to the tumors, I evaluated the incidence, latency, severity or multiplicity of neoplasms or preneoplastic lesions. Studies in experimental animals that I determined to be inadequate for evaluation (e.g. too short a duration, too few animals, poor survival) can be found at the end of my reference list.

Cancer Bioassays in Mice

•Knezevich and Hogan[76] (1983) were the authors of a report submitted to the Environmental Protection Agency (EPA)[77] by Monsanto in support of the registration of glyphosate as an herbicide. This report was also discussed in the paper by Greim[78] (referred to as Study 10). For 24 months, groups of 50 male and 50 female CD-1 mice received diets containing glyphosate (purity, 99.7%) at a concentration of 0, 1000, 5000,

or 30,000 ppm, ad libitum. The study observed no treatment-related effect on body weight in male and female mice at the lowest or intermediate dose, but a slight reduction in body weight in the male and female mice at the highest dose compared with controls. Survival in all dose groups was similar to controls. (It does not appear that a MTD was reached). There was a positive trend[79] (p = 0.016, trend test) in the incidence of renal tubule adenoma in dosed male mice: 0/49, 0/49, 1/50 (2%), 3/50 (6%). Renal tubule adenoma is a rare tumor in CD-1 mice. Historical control data from 14 studies conducted between 1977 and 1981 at the testing laboratory indicated that the mouse renal tumors ranged from 0 to 3% and the incidence in the current study (3/50; 6%) exceeded the upper limit of the historical control range by a factor of two. The rarity of this tumor in CD-1 mice is documented in a publication by Chandra and Frith[80] that reports only 1 out of 725 [0.14%] CD-1 male mice in their large historical database had developed renal cell tumors (one carcinoma). No tumors of the kidney were observed in the female mice. No other tumor sites were identified.

A re-evaluation of the original renal section was conducted by a Monsanto consulting pathologist who reported a small renal tubule adenoma in one control male mouse, which was not diagnosed as such in the original pathology report.[81] This finding was contrary to the initial findings of Bio/dynamics lab, the lab commissioned to complete this report. Following Monsanto's submission of the consulting pathologist's report, the EPA reported there was no difference in diagnoses between his and other pathologists' diagnoses with respect to kidney tumors in mid- and high-dose groups (i.e. 0/49, 0/49, 1/50 (2%), 3/50 (6%)). The EPA pathologist also indicated in his report[79] this data also shows a positive trend (p = 0.016, trend test) in the incidence of renal tubule adenoma in the dosed male mice. Regarding the questionable male control kidney, it was his opinion that the presence of a tumor cannot definitely be established. Nonetheless, the EPA requested additional renal sections be cut and evaluated from all male mice in the control and treated groups; this additional review found no additional tumors.[81] The EPA also requested that a pathology working group (PWG) be convened to evaluate the tumors of the kidney observed in male mice treated with glyphosate, including the additional renal sections.[82] Monsanto sponsored a PWG that reported the incidence of adenoma of the renal tubule was 1/49 (2%), 0/49, 0/50, 1/50 (2%)(not statistically significant); the incidence of carcinoma of the renal tubule was 0/49, 0/49, 1/50 (2%),

2/50 (4%) (which gives a significant p = 0.037, trend test for carcinoma); and the incidence of adenoma or carcinoma (combined) of the renal tubule was 1/49 (2%), 0/49, 1/50 (2%), 3/50 (6%) (which gives a significant p = 0.034, trend test for combined). The PWG did not discuss their finding of an adenoma in the control male mice or address the previous opinion that the presence of a tumor in the control male mice cannot definitely be established and concluded the kidney tumors were not compound related.[83] It is important to note that the renal tumor identified in the controls by the PWG after re-evaluation of the original slides was not seen in the re-sectioned kidney slides. My conclusion of the results discussed above is that there was a significant increase in the incidence of these rare kidney tumors in the CD-1 mouse, with a dose-related trend, which is caused by glyphosate. For the purpose of this hazard identification the increase the incidence of carcinoma of the renal tubule and the incidence of adenoma or carcinoma (combined) of the renal tubule in male mice is due to treatment with glyphosate that caused a significant, dose related increase of these rare tumors in male CD-1 mice.

•Atkinson et al.[84] (1993) were the authors of a report submitted to the EPA in support of the re-registration of glyphosate as an herbicide. This study was also discussed in the paper by Greim[78] (Study 11). Groups of 50 male and 50 female CD-1 mice were given diets containing glyphosate (purity, 98.6%) at a concentration that was adjusted weekly for the first 13 weeks and every 4 weeks thereafter to give doses of 0, 100, 300, or 1000 mg/kg bw, ad libitum, for 104 weeks. There was no treatment-related effect on body weight or survival in any of the dosed groups indicating a maximum tolerated dose was not achieved. The EPA reported[77] a statistically significant increase in the incidence of hemangiosarcoma (blood vessel tumor) in males – 0/47, 0/45, 0/50, 4/45 (9%) ($p < 0.01$, trend test), and non-significant increase in females – 0/50, 2/50 (4%), 0/50, 1/50 (2%). The EPA pointed out that the incidence in the high dose males was near the upper limit (0-8%) for the performing laboratory. However, if one looks at excerpts from the full report,[84] Table 15 (page 97) indicates that as few as 2 animals per dose group were examined histologically for this tumor. This would lead one to consider that the incidence of this tumor could have been higher in this study as more of these tumors could have been found if all 50 animals per dose group were examined. There was also reported a non-significant increase in the incidence of histiocytic sarcoma in the lymphoreticular/haemopoietic tissue in males – 0/50, 2/50 (4%), 0/50, 2/50 (4%), and

in females – 0/50, 3/50 (6%), 3/50 (6%), 1/50 (2%). The EPA stated[77] that for their risk analysis, the increase in hemangiosarcomas in male mice was not considered to be treatment-related. For the purpose of this hazard identification, I determined the increased incidence of hemangiosarcomas in male mice is due to the treatment with glyphosate that caused a significant dose related increase in the incidence of hemangiosarcoma in male CD-1 mice. This association may have been stronger if all the animals in this study had been examined histologically for this tumor.

•Greim[78] (Study 12, Sugimoto, K.) reported on a study submitted by Arysta Life Sciences to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of ICR-CD-1 mice (50/sex/group received diets containing glyphosate (94.6–97.6% pure) at 0, 1600, 8000 or 40,000 ppm for 18 months. Parameters evaluated included clinical signs, body weight, food consumption, hematology, clinical chemistry, and urinalysis, organ weights, gross necropsy and histopathological examination. The EPA reported[77] no adverse effects on survival were observed in either sex across the doses tested and there were no statistically significant increases in any tumor type in this study based on details provided by Greim[78]. A review of the tumor tables for this study (Sugimoto[85]) shows that there was a significant trend for the development of hemangiosarcomas in male mice (0/50; 0/50; 0/50; 2/50 (4%)) with a p-value for trend of 0.008, Chi-Square test; a significant trend for the development of malignant lymphomas in male mice (2/50 (4%); 2/50 (4%); 0/50; 6/50 (12%)) with a p-value for trend of 0.008, Chi-Square test; and a significant trend for the development of renal adenomas (0/50; 0/50; 0/50; 2/50 (4%)) with a p-value for trend of 0.008, Chi-Square test seen in male mice. The EPA also reported[86] that hemangiosarcomas in female mice were found to occur with a statistically significant trend in this study (0/50; 0/50; 2/50, (4%); 5/50, (10%) p=0.002, Trend test), and the tumor incidence in the high-dose female mice was statistically significant with p=0.028 as compared to concurrent controls. I also reviewed the Tier II Summaries for Glyphosate Carcinogenicity Studies from Greim, et al.[87] for Study 12, Sugimoto, which showed a reported statistically significant increase in malignant lymphoma in high dose male mice – 0/26, 0/34, 1/27(4%), 5/29(17%) ($p<0.05$ Fisher's exact test); however I could not resolve the difference in the tumor incidence between the Greim Tier II Summary[87], the published Greim et al, 2015[78] and the Sugimoto[85] tumor tables. These appear to be low response rates but this is only an 18-month study where low rates of

tumors are not unusual. For the purpose of this hazard identification there was an increased incidence of malignant and/or a combination of malignant and benign tumors, at multiple tissue sites in male and female CD-1 mice in this study. The significant increase in malignant lymphoma in high dose male mice, and the significant trend in the development of hemangiosarcomas, malignant lymphomas, and renal adenomas in male mice is due to treatment with glyphosate that caused these cancers in male CD-1 mice. The significant trend in the development of hemangiosarcomas in female mice is also related to treatment with glyphosate that caused this cancer in female CD-1 mice.

•Greim[78] (Study 14, Wood, et al. 2009b) reported on a study submitted by Nufarm to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 51 male and 51 female CD-1 mice were given diets containing glyphosate (purity, 94.6–97.6%) at a concentration of 0, 500, 1500, or 5000 ppm for 18 months. Parameters evaluated included clinical signs, body weight, food consumption, organ weights, gross necropsy and histopathological examination. There was no treatment-related effect on survival. In male mice at the high dose there was a significant increase in the incidence of malignant lymphomas (0/51, 1/50(10%), 2/51(4%), 5/51(10%) $p<0.05$, pair-wise comparison, $p<0.01$ for trend) and a significant increase in the trend of formation of adenocarcinomas of the lung (5/51(10%), 5/51(10%), 7/51(14%), 11/51(22%) $p<0.01$ for trend[86]). For the purpose of this hazard identification, I determined the formation of malignant lymphomas and the formation of adenocarcinomas of the lung in male mice in this study is due to treatment with glyphosate that caused a significant increase in the incidence of malignant lymphoma in high dose male CD-1 mice and an increase in the trend of formation of the adenocarcinomas of the lung and malignant lymphomas in male CD-1 mice.

•Greim[78] (Study 13, Kumar) reported on a study submitted by Feinchemie Schwebda to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 50 male and 50 female Swiss albino mice [age at start not reported] were given diets containing glyphosate (purity >95%) at a concentration of 0, 100, 1000, or 10,000 ppm for 18 months. There were no treatment-related effects on clinical signs, behavior, body weight, body weight gain, food consumption, and differential white blood cell counts in both sexes. There was a slightly higher mortality rate observed in the high dose groups. There was a significant increase in malignant lymphoma reported in high dose male mice

(10/50, 20%; 15/50, 30%; 16/50, 32%; 19/50, 38%, p<0.05 pair wise) and female mice (18/50, 36%; 20/50,40%; 19/50, 38%; 25/50, 50%, p<0.05 pair wise). There was also a significant increased trend (one-sided p-value for trend=0.05) for the formation of this tumor in males. The incidence of malignant lymphoma in the high dose male was double the historical rate, reported to be 18%[87] for males, and for high dose female mice the incidence was well above the historical rate of 41%[87]. There was also a significant increased trend in the incidence of kidney renal cell adenomas reported[88] in males (0/50; 0/26; 1/26 (4%); 2/50 (4%); one-sided p-value for trend p=0.04). I would note that the EPA stated[77] this study was not included in their review due to the report by Greim (2015)[78] that there was possibly a viral infection within the colony, which confounded the interpretation of the study findings. EPA also stated although the incidences in this study were within or near the normal variation of background occurrence. It is not clear whether or not ther viral component may have contributed to incidence value reported or the lower survival seen at the high dose in the study.[89] An internal Monsanto email among the authors of Greim would indicate there was no viral infection in the mouse colony during this study. Further, Greim[78] (table 18, p. 201) considers this study GLP and OECD compliant. For the purpose of this hazard identification, I determined formation of malignant lymphoma in the male and female mice and the renal cell adenomas in males in this study is due to treatment with glyphosate that caused a significant increase in the incidence of malignant lymphoma in high dose male and female Swiss albino mice and renal cell adenomas in male Swiss albino mice.

Cancer Bioassays in Rats

•Greim[78] reported on a Bio/dynamics study (Study 1, Lankas, et al.) submitted by Monsanto to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats were fed diets containing glyphosate (98.7%, pure) at concentrations of 0, 30, 100 or 300 ppm for 26 months. These concentrations were adjusted during the course of the study so that actual doses of 0, 3, 10, and 31 mg/kg/day in males and 0, 3, 11, and 34 mg/kg/day in females were maintained. There were no treatment-related effects on body weight or survival at any dose level. An MTD was not achieved. There was a significant increase reported in the incidences of interstitial cell tumors in the testes of male rats: controls 0/50, 0%; low dose

3/5, 6%; mid dose 1/50, 2%; high dose 6/50; 12%; p=0.013 by pairwise comparison[77]. The incidence of interstitial cell tumors in the testes in the high dose animals in this study is almost twice that seen in the range of this tumor (3.4% to 6.7%) in control animals (historical controls) from 5 contemporary studies[87]. There was also a significant increase in the incidence of pancreatic islet cell adenoma reported in males at the low dose: controls, 0/50; low dose 5/49, 10% ($p < 0.05$ Fisher exact test); mid dose 2/50, 4%; high dose 2/50, 4%. For the purpose of this hazard identification, I determined the increase in the incidence of interstitial cell tumors in the testes and pancreatic cell tumors in male rats is due to the treatment with glyphosate that caused a significant increase in the incidence of interstitial cell tumors in the testes and pancreatic islet cell tumors in male Sprague-Dawley rats.

•Greim[78] reported on a study (Study 2, Stout, et al.) submitted by Monsanto to the EPA in support of the registration of glyphosate as an herbicide. Groups of 60 male and 60 female Sprague-Dawley rats were given diets containing glyphosate (technical grade; purity, 96.5%) at a concentration of 0 ppm, 2000 ppm, 8000 ppm, or 20,000 ppm, ad libitum, for 24 months. No compound-related effect on survival was observed. There was no statistically significant decreases in body-weight gain in male rats. The study reported significant decreases in body-weight gain in females at the highest dose, beginning on day 51. There was a statistically significant increase in the incidence of pancreatic islet cell adenoma in males at the lowest dose compared with controls: control 1/58, 2%; low dose 8/57, 14% ($p \leq 0.05$ Fisher exact test); mid dose 5/60, 8%; high dose 7/59, 12%. The EPA[77] did additional analysis of this data for pancreatic islet cell adenoma by excluding rats that died or were killed before week 55 and then using statically analyses (Cochran–Armitage trend test and Fisher exact test) that gave a statistically significant higher incidence of these tumors in males at the lowest and highest doses compared with controls: control 1/43, 2%; low dose 8/45, 18% ($p = 0.018$; pairwise test); mid dose 5/49, 10%; high dose 7/48, 15% ($p = 0.042$; pairwise test). The incidence of these adenomas in the low (18%) and high (15%) dose males was almost twice that seen in historical controls. The range for historical controls for pancreatic islet cell adenoma reported in males at this laboratory was 1.8–8.5%[77]. One should note that there was no statistically significant positive trend in the incidence of these tumors, and no apparent progression to carcinoma. There was also a statistically significant positive trend ($p = 0.016$) in the

incidence of hepatocellular adenoma observed in male rats[86] and a statistically significant positive trend of thyroid follicular cell adenomas (p = 0.031) and thyroid follicular cell adenomas and carcinomas combined (p=0.033) observed in female rats[86] reported in this study. For the purpose of this hazard identification, I determined that the increase in the incidence of pancreatic islet cell adenoma in male rats is due to the treatment with glyphosate that caused a significant positive increase in the incidence of pancreatic islet cell adenomas of male Sprague-Dawley rats. Glyphosate also caused a significant increase in the trend for formation of hepatocellular adenomas in male Sprague-Dawley rats and of thyroid follicular cell adenomas and follicular cell adenomas and carcinomas combined in female Sprague-Dawley rats.

•Greim[78] reported on a study (Study 3, Atkinson, et al.) submitted by Cheminova to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats were given diets containing glyphosate, purity, 98.7–98.9%, at a concentration that were adjusted to provide doses of 0, 10, 100, 300, or 1,000 mg/kg bw/day, ad libitum, for 104 weeks. Decreased body-weight gain was observed in males and females at the highest dose. There was no significant decrease in survival reported at any dose level. Neoplasms were noted in control and treated groups, but dose-responses were not evident, and no statistically significant increases versus controls were noted for any tumor type. Additionally, EPA's evaluation[86] of this study indicated there were no treatment-related increases in the occurrence of any tumor type in this study.

•Greim[78] reported on a study (Study 7, Brammer) submitted by Syngenta to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 52 male and 52 female Wistar rats received diets containing 0, 2,000, 6,000, and 20,000 ppm glyphosate (97.6% pure), ad libitum, for 24 months. Survival in the high dose group males was significantly better than the other dose groups throughout the study while survival in the females was similar across all dose groups. The bodyweights of the high dose males and females were statistically significantly lower than controls throughout the study. The study's author reported no significant increase in turmor incidence in any of the treated groups. The EPA's evaluation[77] of this study indicated there was a significant increase in the incidence of hepatocellular adenomas in male rats at the high dose when compared to controls (control 0/52, 0%; low dose 2/52, 4%; mid dose 0/52, 0%; high dose 5/52, 10%, p=0.03). There was also a significant trend (p=0.008) in the formation of this tumor in

male rats. The EPA goes on to state the incidences observed were within the range (0–11.5%) of historical controls for this strain of rats in 26 studies conducted during the relevant time period (1984–2003) at the testing laboratory indicating this increase was not considered to be related to treatment with glyphosate. For the purpose of this hazard identification, I determined the increase in the formation of hepatocellular adenomas in male Wistar rats could not be attributed to exposure to glyphosate in this study despite the fact that there was an observation of increased incidence of hepatocellular adenomas in male rats.

•Greim[78] reported on a study (Study 4, Suresh) submitted by Feinchemie Schwebda to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Wistar rats received diets containing 0, 100, 1,000, and 10,000 ppm glyphosate (97.6% pure), ad libitum, for 24 months. There were no treatment-related deaths or clinical signs in any of the dose-groups and there were no treatment related effects on body weight gain or food consumption noted. This suggests that the MTD was not reached, and this study is inadequate for the evaluation of the carcinogenicity of glyphosate.

•Greim[78] reported on a study (Study 6, Enomoto) submitted by Arista Life Sciences to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats received diets containing 0, 3,000, 10,000, or 30,000 ppm glyphosate (94.6–97.6% pure) for 24 months. Decreases in body weight were observed in both sexes in the mid and high dose group along with a lower food consumption. Survival in the high dose males was lower than controls while there was no compound-related effect on survival in any other dose group. There were no statistically significant increases in any tumor type reported for this study.

•Greim[82] reported on a study (Study 8, Wood 2009a) submitted by Nufarm to the EPA in support of the registration of glyphosate as an herbicide. Groups of 51 male and 51 female Wistar rats received diets containing 0, 3,000, 10,000, or 15,000 ppm glyphosate (95.7% pure) for 24 months, the highest dose level was progressively increased to 24000 ppm by week 40. There were no treatment-related deaths or clinical signs in any of the dose-groups. No significant treatment-related effects on mortality were observed during the study. This suggests that the MTD was not reached, and this study is inadequate for the evaluation of the carcinogenicity of glyphosate.

•Chruscielska et al.[90] gave groups of 55 male and 55 female Wistar rats drinking-water containing an ammonium salt of glyphosate (purity not given) that was used to make drinking water solutions of 0, 300, 900, and 2700 mg/L, for 24 months. The authors reported that survival and body-weight gain were similar in treated and control animals and that no significant increase in tumor incidence was observed in any of the treated groups. There was limited information provided on dosing regimen, histopathological examination method, and tumor incidences that makes this study inadequate for the purpose of this hazard assessment.

Summary for Experimental Animal Data

I reviewed a total of five dose feed bioassays of glyphosate in mice. Four of these studies (Study 12 and Study 14 in Greim[78], Knezevich and Hogan (1983)[76], and Atkinson[84]) were in male and female CD-1 mice, and one study[78(Study13)] was in male and female Swiss albino mice. Glyphosate caused a significant increase in the incidence of adenoma or carcinoma (combined) and a significant positive trend for the formation of adenoma or carcinoma (combined) of the renal tubule in male CD-1 mice in one study[76], and a significant positive trend for the formation of adenomas of the renal tubule in male CD-1 mice in another study[78(Study 12)]. Glyphosate also caused a significant increase in the incidence of renal cell adenomas in male Swiss albino mice[78(Study13)]. Adenoma and carcinoma of the renal tubule constitutes a morphological continuum in the development and progression of renal neoplasia in mice[91,92]. It is important to note that renal tubule carcinoma is a very rare tumor in CD1 mice[80] and that this tumor was caused by exposure to glyphosate in two different strains of mice (CD-1 and Swiss). Glyphosate caused a significant increase in the incidence of malignant lymphoma in male CD-1 mice in two studies[78(Study 12, Study 14)] and in male and female Swiss albino mice in another study[78 (Study 12)]. Glyphosate also caused a significant positive trend for the formation of malignant lymphoma in one of these studies in male CD-1 mice[78(Study 12)] and caused a significant positive trend for the formation of hemangiosarcomas in 2 separate studies in male CD-1 mice[78(Study 12),84]. There was also a significant positive trend for the formation of adenocarcinomas of the lung in male CD-1 mice in one study[78(Study 14)] and hemangiosarcomas in female CD-1 mice in another study[82(Study 12)].

I reviewed a total of 7 dosed feed and 2 drinking water bioassays of glyphosate in rats. Four of the feed studies and one drinking water study were in male and female Sprague-Dawley rats and three feed studies and one drinking water study were in male and female Wistar rats. Glyphosate caused a significant increase in the incidence of pancreatic islet cell adenoma in two feeding studies in male Sprague-Dawley rats[78(Study 1 and Study 2)]. Glyphosate caused a significant increase in the incidence of thyroid tumors in male Sprague-Dawley rats in one feeding study[78(Study 1)] and a significant positive trend for the formation of thyroid tumors in female Sprague-Dawley rats in another feeding study[78(Study 2)]. Glyphosate caused a significant increase in the incidence of interstitial cell tumors in the testes of male Sprague-Dawley rats in one feeding study and a significant positive trend for the formation of hepatocellular adenomas in male Sprague-Dawley rats in another feeding study[78(Study 1)].

To state my findings more concisely, I determined that in CD-1 mice, glyphosate expsoure causes kidney tumors in males in two separate studies[76,78(Study 12)], hemangiosarcomas in males in two separate studies,[78(Study 12),84] malignant lymphoma in males in two separate studies[78(Study 12, Study 14)], adenocarcinomas of the lung in males in one study[78(Study 14)], and hemangio~~sarcomas~~mas in females in one study[78(Study 12)]. In one study[78(Study 13)] in Swiss albino mice, exposure to glyphosate causes malignant lymphoma in males and females and kidney tumors in males.

I also determined that in Sprague-Dawley rats, glyphosate exposure causes pancreatic cell tumors in males in one study[78(Study 2)], interstitial cell tumors in the testes in males in one study[78(Study 1)], hepatocellular adenomas in males in two studies[78(Study 2, Study 7)], and thyroid follicular cell tumors in females in one study[78(Study 2)].

Considering all data from the mice and rat studies I reviewed, there is "Sufficient" evidence that shows glyphosate is carcinogenic in experimental animals causing kidney tumors, hemangiosarcomas, malignant lymphoma, adenocarcinomas of the lung, and hemangiomas in mice and pancreatic cell tumors, interstitial cell tumors in the testes, hepatocellular adenomas, and thyroid follicular cell tumors in rats. This statement is based on my stated criteria of a causal relationship between exposure to glyphosate and an increased incidence of malignant and/or a combination of malignant and benign tumors, in multiple species, at multiple tissue sites, from multiple studies, and to an unusual degree with regard to incidence, site, or type of tumor.

Hazard Assessment of the Mechanistic and Other Data for Glyphosate and Glyphosate-Based Formulations

Data on the absorption of glyphosate via intake of food and water in humans could not be found in the published literature. Glyphosate has been found in the urine of agricultural workers. In a study by Acquavella[7], 60% of farmers had detectable levels of glyphosate in 24-hour composite urine samples taken on the day they had applied a glyphosate-based formulation. Wearing protective gear such as rubber gloves reduced the concentrations of glyphosate in the urine. This implies that dermal absorption is a relevant route of exposure. Curwin[8] demonstrated that glyphosate is also present in the urine of non-farm families. No data in humans on the distribution of glyphosate in systemic tissues other than blood were found in the available published literature. In cases of accidental or deliberate intoxication involving ingestion of glyphosate-based formulations, glyphosate was measured in blood.

Strong evidence indicates that glyphosate is genotoxic. As noted in Monograph 112, studies in human cells[27,31,32], mammalian model systems[27,32,33], and in non-mammalian organisms[35,37] have given positive results . The end-points evaluated in these studies included biomarkers of DNA adducts and various types of chromosomal damage. Tests in bacterial assays gave consistently negative results.

The evidence for genotoxicity caused by glyphosate-based formulations is also strong. As noted in Monograph 112, three studies[39,93,94] reported examining genotoxic end-points in community residents exposed to glyphosate-based formulations and two of these studies reported positive associations. One study[39] looked at micronucleus formation in circulating blood cells before and after aerial spraying with glyphosate-based formulations to determined chromosomal damage in exposed individuals. This study revealed a significant increase in micronucleus formation after exposure in three out of four different geographical areas. Additional positive evidence came from in vitro studies with positive results in human cells[32,45], in vivo[27,32] and in vitro[95] studies in mammalian systems, and studies in non-mammalian organisms[35,96] such as fish. Biomarkers of DNA adducts and different types of chromosomal damage were examined in these studies. The pattern of tissue specificity of genotoxicity end-points observed with glyphosate-based

formulations is similar to that observed with glyphosate. Tests of glyphosate-based formulations in bacterial assays gave generally negative results.

There is strong evidence that glyphosate and glyphosate-based formulations induce oxidative stress. As noted in Monograph 112, vidence of oxidative stress comes from in vitro studies in human cells[97,98] and in many in vivo studies[32,42], examining rodent tissues. Studies of oxidative stress and glyphosate in non-human mammalian experimental systems were conducted in rats and mice, and examined a range of exposure durations, doses, preparations (glyphosate and glyphosate-based formulations), administration routes and tissues. In these studies glyphosate caused free radicals and oxidative stress in mouse and rat tissues through alteration of antioxidant enzyme activity, depletion of glutathione, and increases in lipid peroxidation. In at least one of the studies in human cells the oxidative stress caused by glyphosate was ameliorated by co-administration of antioxidants[40]. Similar findings of oxidative stress have been reported in fish and other aquatic species providing additional evidence for glyphosate-induced oxidative stress[99]. Molecular epidemiology studies[100,101] have documented that oxidative stress is a pathway to the formation of NHL in humans. Further, the in vitro studies in humans cells and in vivo and in vitro studies in rodents provides evidence that exposure to glyphosate causes oxidative stress. Logically it follows that there is a positive association between oxidative stress caused by glyphosate and glyphosate-based formulations and NHL observed in humans exposed to glyphosate-based formulations and that a causal interpretation is credible.

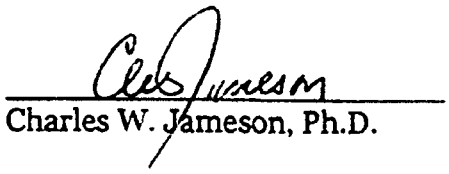
Hazard Assessment Conclusion

Based on the significant positive association observed in the studies discussed above, I conclude that there is evidence that glyphosate and glyphosate-based formulations are carcinogenic in humans. First, the human study data supports a positive association between exposure to glyphosate and glyphosate-based formulations and the development of NHL. Second, all the data from the animal bioassay studies provide evidence that glyphosate is carcinogenic in experimental animals. Third, the mechanistic data show that glyphosate and glyphosate-based formulations cause genotoxicity and oxidative stress in humans and animals. Therefore, I conclude to a reasonable degree of

scientific certainty that glyphosate and glyphosate-based formulations are probable human carcinogens. I also conclude to a reasonable degree of scientific certainty that glyphosate and glyphosate-based formulations cause NHL in humans.

Compensation and Testimony

My billing rate is $400/hr plus travel fees and expenses. I have not testified in any case in the last four years.



Charles W. Jameson, Ph.D.

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Greim[78] reported on a study (Study 4, Suresh): Greim H, Saltmiras D, Mostert V, Strupp C (2015). Evaluation of carcinogenic potential of the herbicide glyphosate, drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies. Crit Rev Toxicol, 45(3):185–208.

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Chruscielska K, Brzezinski J, Kita K, Kalhorn D, Kita I, Graffstein B et al. (2000). Glyphosate - Evaluation of chronic activity and possible far-reaching effects. Part 1. Studies on chronic toxicity. Pestycydy (Warsaw), 3–4:11–20.

Seralini GE, Clair E, Mesnage R, Gress S, Defarge N, Manuela Malatesta M et al. (2014). Republished study: long-term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize. Environmental Sciences Europe, 26(1):1–14

George J, Prasad S, Mahmood Z, Shukla Y (2010). Studies on glyphosate-induced carcinogenicity in mouse skin: a proteomic approach. J Proteomics, 73(5):951–64.

EXHIBIT A

Name: Charles William Jameson

Mailing Address: [redacted]

Date And Place Of Birth:

Citizenship:

Marital Status:

Education:

B.S. 1970
Chemistry,
Mount Saint Mary's College
Emmitsburg, Maryland

Ph.D. 1975
Organic Chemistry, Physical Chemistry minor
University of Maryland
College Park, Maryland

Brief Chronology of Employment:

1965	Chemistry Laboratory Technician, Bionetics Research Laboratories, Falls Church, Virginia
1968 – 1969:	Organic Chemistry Laboratory Assistant, Mount Saint Mary's College, Emmitsburg, Maryland
1969 – 1970:	Organic Chemistry Laboratory Instructor, Mount Saint Mary's College, Emmitsburg, Maryland
1970 – 1973:	Graduate Teaching Assistant, Chemistry Dept., University of Maryland College Park, Maryland
1973 – 1975:	Graduate Research Assistant, Center of Materials Research, University of Maryland, College Park, Maryland
1975 – 1976	Faculty Graduate Assistant, Chemistry Dept., University of Maryland, College Park, Maryland
1976 – 1979:	Senior Chemist, Tracor Jitco, Inc., Rockville, Maryland
1979 – 1980:	Chemist, Carcinogenesis Testing Program, National Cancer Institute, National Institutes of Health (NIH), Bethesda, Maryland

1980 – 1983:	Head, Chemistry Section, Program Resources Branch, National Toxicology Program (NTP), National Institute of Environmental Health Sciences (NIEHS), NIH, Research Triangle Park, North Carolina
1983 – 1985:	Acting Chief, Program Resources Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1985 – 1989:	Head, Program Resources Group, Carcinogenesis and Toxicologic Evaluation Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1989 – 1990:	Supervisory Chemist, Experimental Toxicology Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1990 – 1995:	Senior Chemist, Office of the Senior Scientific Advisor to the Director NIEHS, NIH, Research Triangle Park, North Carolina
1995 – 2008	Director, Report on Carcinogens, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
2008 – present	Principal, CWJ Consulting, LLC, Cape Coral, Florida

Department of Health and Human Services Activities

Chairman, National Toxicology Program's Executive Committee's Interagency Working Group for the Report on Carcinogens, 1995 to 2005

National Institutes of Health Activities

NIEHS Representative to the Deafness and Other Communication Disorders Interagency Coordination Committee, 1990 - 1996.

NIEHS Representative on the Task Force on Aging Research, 1990-1994.

National Institutes of Environmental Health Sciences Activities

Chairman, NIEHS/NTP Review Committee for the Report on Carcinogens, 1995 to 2005

Chairman, Search Committee for NIEHS Tenure / Tenure Track Staff Epidemiologist 1998

Peer-Review Panel Member for Draft Report on Carcinogens Monograph on Cobalt and Certain Cobalt Compounds. July, 2015

Member and Chairman for the Special Emphasis Panel to review proposals responding to RFP ES2015038, "Scientific Information Management and Literature-Based Evaluations for the National

Toxicology Program (NTP)." The objective of this contract is to provide scientific and technical expertise and support for the NTP to compile, review, and analyze information and data from the scientific literature and other sources regarding the effects of environmental substances and other issues that may impact public health. October, 2015

International Activities

Member, WHO Task Group on Environmental Health Criteria for Fully Halogenated Chlorofluorocarbons, Neuherberg, Federal Republic of Germany, November 21 – 25, 1988.

Member, WHO Task Group on Environmental Health Criteria for Partially Halogenated Chlorofluorocarbons (Ethane Derivatives), Carshalton, Surrey, United Kingdom, September 30 – October 5, 1991.

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 82 on the Carcinogenic Risks To Humans Of Some Traditional Herbal Medicines, Some Mycotoxins, Naphthalene And Styrene, Lyon, France, February 11 – 20, 2002

Member, IARC *Monographs* Advisory Group for Five Year Plan, Lyon, France, 18-21 February 2003

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 87 on The Carcinogenic Risks To Humans Of Lead And Lead Compounds, Lyon, France, February 8 – 18, 2004

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 91 on The Carcinogenic Risks To Humans Of Combined Oral Contraceptives And Estrogen-Progestogen Replacement Therapy, Lyon, France, June 4-15, 2005.

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 93 on The Carcinogenic Risks To Humans Of Carbon Black, Titanium Dioxide And Non-Asbestiform Talc, Lyon, France, February 4 – 15, 2006

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 97 on The Carcinogenic Risks To Humans Of 1,3 –Butadiene, Ethylene Oxide, And Vinyl Halides (Vinyl Fluoride, Vinyl Chloride And Vinyl Bromide), Lyon, France, June 6-15, 2007.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 99 on The Carcinogenic Risks To Humans Of Some Industrial And Cosmetic Dyes And Related Exposures, Lyon, France, February 4-13, 2008.

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph 100A on A Review Of Human Carcinogens - Pharmaceuticals (Anti-Cancer Drugs – Hormonal Drugs & Therapies – Others), Lyon, France, October 14 – 21, 2008.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 100F on A Review Of Human Carcinogens - Chemical Agents And Related Occupations, Lyon, France, October 20 – 27, 2009.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 103 on Bitumen And Bitumen Fumes, And Some Heterocyclic Aromatic Hydrocarbons, Lyon, France, October 11 - 18, 2011.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 105 on Diesel And Gasoline Exhausts And Some Nitroarenes, Lyon, France, June 5 - 12, 2012.

Member WHO's International Agency for Research on Cancer (IARC) Workshop on Tumour Concordance And Mechanisms Of Carcinogenesis: Lessons Learned From Volume 100 of the IARC Monographs, Lyon, France: April 16-18, 2012 and November 28-30, 2012

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 108 On Some Drugs And Herbal Medicines, Lyon, France, June 4 - 11, 2013.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 112 on Some Organophosphate Insecticides And Herbicides, Lyon, France, March 3-10, 2015.

Member and overall Chair, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 115 on Some Industrial Chemicals, Lyon, France, February 2-9, 2016.

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph 116 on Coffee, Mate And Very Hot Beverages, Lyon, France, May 24 – 311, 2016.

Honors and Awards

President, Student Affiliate Chapter of the American Chemical Society, Mount Saint Mary's College, 1969; Vice President, 1968.

National Toxicology Program Representative to American Chemical Society's Committee on Regulatory Affairs 1982 – 1992.

National Institutes of Health Special Achievement Cash Award (Spy Dust Project): 1986.

Merit Pay Cash Award for Sustained High Quality Work Performance, NIEHS: 1982, 1989

Performance Award for Sustained High Quality Work Performance, NIEHS: 1991, 1992, 1993, 1995, 1996, 2001, 2002, 2003, 2004, 2006, 2007.

Special Act or Service Award, NIEHS: 1996 (Review of Report on Carcinogens criteria); 1997 (Publication of 8th Report on Carcinogens); 1998 (Recruitment of NTP Staff Epidemiologist), 1998 (Restructuring of lead biokinetics contract and establishment of new Report on Carcinogens support contract)

Staff Recognition Award, NIEHS: 1999 (Preparation of final draft of 9th Report on Carcinogens)

NIEHS Director's Award, NIEHS: 2000 (Review of nominations for the 9th Report on Carcinogens)

Special Training

American Chemical Society, Short Course: "Chemical Carcinogenesis," 1978.

National Institutes of Health (NIH) Training Course: "Project Officers Civil Rights Contract Compliance," 1979.

Department of Health and Human Services Training (DHHS) Course: "Program Officials Guide to Contracting," 1980.

U. S. Office of Personnel Management (OPM) Training Course: "EEO - Its Place in the Federal Government," 1983.

U. S. OPM Training Course: "Introduction to Supervision," 1984.

NIH Training Course: "Employee Performance Management System Training," 1984.

DHHS Training Course: "Advanced Project Officer Training," 1985.

National Institute of Environmental Health Sciences Training Course: "Care and Handling of Laboratory Animals," 1986.

Rockhurst College Continuing Education Center: "How to Manage Projects, Priorities and Deadlines," 1992.

NIH Training Course: "PHS Animal Welfare Policy for HSA's," 1993.

Fred Pryor Seminars: "Total Quality Management," 1994.

Fred Pryor Seminars: "How to Manage Priorities and Meet Deadlines," 1994.

NIH Training Course: "Workplace Violence," 1994.

NIH Training Course: "NIH Guidelines on the Inclusion of Women and Minorities as Subjects in Clinical Research," 1994.

NIH Training Course: "Workplace Issues Associated with HIV/AIDS," 1994.

The Bookings Institution Course: "Issues in Science and Technology Policy", 1996

Professional Society Memberships and Activities

American Chemical Society

- Division of Analytical Chemistry
- Division of Chemical Health and Safety
- National Toxicology Program Representative to American Chemical Society's Committee on Regulatory Affairs 1982 – 1992
- Overall Co-Organizer and Co-Chairman of a symposium entitled "Chemistry and Safety for Toxicity Testing of Environmental Chemicals," sponsored by the Divisions of Chemical Health and Safety, Analytical Chemistry and Environmental Chemistry at the 183rd National American Chemical Society Meeting, Las Vegas, NV, March 1982.

Society of Toxicology

Research interests:

Chemical Carcinogenesis
Analytical chemistry methods development to support toxicology studies.

Reviewer for Scientific Journals

Analytical Chemistry
Bulletin of Environmental Contamination & Toxicology (Member of Editorial Board)
Environmental Health Perspectives (Contributing Editor)
Fundamental and Applied Toxicology
Journal of the National Cancer Institute
Science

Invited Papers

Invited to be Session Chairman and to present paper entitled "Analytical Chemistry Requirements for Toxicity Testing of Environmental Chemicals" at the Symposium on Chemistry and Safety for Toxicity Testing of Environmental Chemicals, at the 183rd National American Chemical Society Meeting, Las Vegas, NV, March 1982.

Invited to serve as a panelist on the NBC nationally televised series "Health Field" with Dr. Frank Field. A two-day series was filmed on Environmental Chemistry and Chemical Health Concerns, 1982.

Invited to give a seminar entitled "Analytical Chemistry Requirements for Toxicity Testing." Duke University, Durham, NC, July 1982.

Invited to present a paper entitled "Practical Aspects of Analytical Chemistry Support for Toxicity Testing" at the Symposium on the Role of the Analytical Chemist in Animal and Molecular Toxicology, at the Federation of Analytical Chemistry and Spectroscopy Societies Meeting XI, Philadelphia, PA. September 16-21, 1984.

Invited to present a paper entitled "Application of Microencapsulation in Toxicity Testing" at the NIEHS Center Directors Meeting, Research Triangle Park, North Carolina, November 1984.

Invited to be Session Chairman and to present paper entitled "Chemical Quality Assurance Techniques for Toxicity Testing of Environmental Chemicals" at the Symposium on Accurate Measurements of Environmental Pollutants, at the 1984 International Chemical Congress of Pacific Basin Societies, Honolulu, Hawaii, December 16-21, 1984.

Invited to present a paper entitled "Lack of Evidence for Involvement of Cyanide in Methyl Isocyanate (MIC) Toxicity" at the Society of Toxicology Meeting, New Orleans, LA, March 3-7, 1986.

Invited to present a paper entitled "Toxicology From A Chemist's Viewpoint" at the Mount Saint Mary's College Science Alumni Homecoming, Emmitsburg, Maryland, October 23-26, 1986.

Invited to be Session Chairman and to present paper entitled "Application of Microencapsulation for Toxicity Studies" at the Symposium on Techniques for Microencapsulation of Chemicals at the 198th National Meeting of the American Chemical Society, Dallas, Texas, April 10-14, 1989.

Invited to be Session Chairman and to present paper entitled "Application of a Fischer Rat Leukemia Transplant Model as a Screen for the Leukemogenic Potential of Chemicals" at the International Symposium on Toxicology, Beijing, P. R. China, October 16-19, 1990.

Invited to present a paper entitled "Investigation of Alternative Vehicles for Use in Toxicology Research: Use of Microencapsulated and Molecular Encapsulated Chemicals in Toxicity Studies" at the Institute of Pharmacology and Toxicology, Academy of Military Medical Sciences, Beijing, P. R. China, October 20, 1990.

Invited to present a paper entitled "Toxicology and Carcinogenicity Studies of d- Limonene in Male and Female F344 Rats and B6C3F1 Mice" at the Symposium on Food Phytochemicals for Cancer Chemoprevention at the 204th National Meeting of the American Chemical Society, Washington, D.C., August 23-28, 1992.

Invited to be a Faculty Member and to present talk entitled " The National Toxicology Program's Report on Carcinogens " at the Toxicology Forum, Washington, DC, February 1995.

Invited to be a Faculty Member and to present talk entitled " The Report On Carcinogens (RoC): Status Of The Review Of The Criteria For Listing Substances In The RoC " at the Toxicology Forum, Washington, DC, February 1996.

Invited to be a Faculty Member and to present talk entitled " Update of 1997 review of Nominations for the 9th Report on Carcinogens " at the Toxicology Forum, Washington, DC, February 1998.

Invited to be a Faculty Member and to present talk entitled " NTP Report on Carcinogens: History and the Process " at the Toxicology Forum, Aspen, CO, July 1999.

BIBLIOGRAPHY

Publications

1. Mazzocchi PH, Ammon HL, **Jameson CW**. Lanthanide Shift Reagents III: Errors Resulting from the Neglect of Angle Dependence, Tetrahedron Letters, 573, 1973.

2. **Jameson CW**. I. Study of Lanthanide shift Reagent - Substrate Interaction in Solution. II. Competitive Photochemical Type I and Type II Reactions of Amides and Imides. Dissertation Abstracts, 1975.

3. Ennis DM, Kramer A, Mazzocchi PH, **Jameson CW**, Bailey WJ. Synthetic N-Releasing Biodegradable Soil Conditioners I, Hort Science, 10, 505, 1975.

4. Ammon HL, Mazzocchi PH, Colicelli E, **Jameson CW**, Liu L. A Convenient Method for Mixing ^{2}H and ^{13}C Lanthanide Induced Shift (LIS) Calculations, A Technique for Facilitating ^{13}C Assignments, Tetrahedron Letters, 1745, 1976.

5. Ennis DM, Kramer A, **Jameson CW**, Mazzocchi PH, Bailey WJ. Structural Factors Influencing the Biodegradation of Imides, Appl Environ Microbiology, 35, 51, 1978.

6. Murrill EA, Woodhouse EJ, Olin SS, **Jameson CW**. Carcinogenesis Testing and Analytical Chemistry, Analytical Chemistry, 52, 1188A, 1980.

7. Douglas JF, Hamm TE, **Jameson CW**, Mahar H, Stinson S, Whitmire CE. Monitoring Guidelines for the Conduct of Carcinogen Bioassays. US Department of Health and Human Services. DHHS Publication No. (NIH) 81-1774. Washington, DC, US Government Printing Office, 80 pp., 1981.

8. Dieter MP, Luster MI, Boorman GA, **Jameson CW**, Dean JH, Cox JW. Immunological and Biochemical Responses in Mice Treated with Mercuric Chloride, Toxicol Appl Pharmachol, 68, 218, 1983.

9. **Jameson CW**, Dunnick JK, Brown RD, Murrill EA. Chemical Characterization of Psoralens Used in the National Toxicology Program Research Projects, National Cancer Institute Monograph, 66, 103, 1984.

10. Timmons L, Cannon M, Grese D, Brown R, Haile C, Murrill E, **Jameson CW**. Identification of Chlorinated Phenyl and Phenoxy Substituted Dibenzodioxin, Dibenzofuran and Diphenyl Ether Homologs in Commercial Grade Pentachlorophenol, Analytical Letters, 17(A4), 277-296, 1984.

11. Timmons L, Steel D, Cannon M, Grese D, Brown R, Murrill E, **Jameson CW**. Identification of Bromotertrachlorophenol in Commercial Pentachlorophenol Samples, Journal of Chromatography, V 314, 476-481, 1984.

12. Dunnick JK, **Jameson CW**, Benson JM. Toxicology and Carcinogenesis Studies of Nickel Oxide, Nickel Subsulfide and Nickel Sulfate. Annals of Clinical and Laboratory Science. V14.N5. 400-401, 1984.

13. Lamb JC, IV, **Jameson CW**, Choudury H, Gulati D K. Fertility Assessment by Continuous Breeding: Evaluation of Diethylstilbestrol and a Comparison of Results from Two Laboratories. J Amer Coll Toxicol 4, 173, 1985.

14. Thigpen JE, Liu LA, Richter CB, Lebetkin EH, Haseman JK, **Jameson CW**. The Comparative Estrogenic Activity of Semipurified, Certified, Standard and Open Formula Rodent Diets. Laboratory Animal Science, V35, N5, 526-527, 1985.

15. Kline DA, Hanna GR, Kuhn GO, Honaker CB, **Jameson CW**. Preparation and Stability of Animal Feed Mixtures Dosed with Rotenone, J Asso Off Anal Chem, Vol. 69, #4, 660-663, 1986.

16. **Jameson CW**, Moseman RF, Collins BJ, Hooper ND. Spy Dust: Methods for the Detection and Cleanup of a Chemical Tracking Agent. Analytical Chemistry, 58, 915A, 1986.

17. Agarwal DK, Eustis S, Lamb JC, **Jameson CW**, Kluwe WM. Influence of Dietary Zinc on Di(2-ethylhexyl)phthalate-Induced Testicular Atrophy and Zinc Depletion in Adult-Rats. Toxicology and Applied Pharmacology, V84, N1, 12-24, 1986.

18. Boorman GA, Hong HL, **Jameson CW**, Yoshitomi K, Maronpot, RP. Regression of Methyl Bromide Induced Forestomach Lesions in the Rat. Toxicology and Applied Pharmacology, 86, 131-139, 1986.

19. Collins B, Goehl TJ, **Jameson CW**, Kuhn G, Dux T. Analytical Methods for the Analysis of Microencapsulated Trichloroethylene in Corn Oil, Feed Dosage Formulations and Rat Whole Blood. J. of Analytical Toxicology, 10, 236, 1986.

20. **Jameson CW**, NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrakis(hydroxymethyl)phosphonium sulfate (THPS) and Tetrakis(hydroxymethyl)phosphonium Chloride (THPC) in F344/N Rats and B6C3F1 Mice (Gavage Studies). NIH Publication No. 296, 1987.

21. Dunnick J K, **Jameson CW**, Montgomery CA. Subchronic Toxicity of Propantheline Bromide Administered in the Feed to Fischer 344/N Rats and B6C3F1 Mice. Fundamental and Applied Toxicology, V9, N3, 496-503, 1987.

22. Germolec DR, Burleson GR, **Jameson CW**, Ackermann MF, Lamm KR, Hayes HT, Luster MI. Depression of Natural-Killer Cell-Activity by Ochratoxin-A. Environmental Health Perspectives, V75, No. 5, 145-145, 1987.

23. **Jameson CW**, Moseman RF, Hooper ND, Collins BJ. Spy Dust - Detecting a Chemical Tracking Agent. Environmental Health Perspectives, V75, No. 5, 143-143, 1987.

24. Melnick RL, **Jameson CW**, Goehl TJ. Application of Microencapsulation for Toxicology Studies - Stability, Bioavailability, and Toxicity of Microencapsulated Trichloroethylene. Environmental Health Perspectives, V75, No. 5, 142-142, 1987.

25. Melnick RL, **Jameson CW**, Goehl TJ, Kuhn GO. Application of Microencapsulation for Toxicology Studies. 1. Principles and Stabilization of Trichloroethylene In Gelatin-Sorbitol Microcapsules. Fundamental and Applied Toxicology, V8, N4, 425-431, 1987.

26. Melnick RL, **Jameson CW**, Goehl TJ, Maronpot RR, Collins BJ, Greenwell A, Harrington FW, Wilson RE, Tomaszewski KE, Agarwal DW. Application of Microencapsulation for Toxicology Studies. 2. Toxicity of Microencapsulated Trichloroethylene in Fischer 344 Rats. Fundamental and Applied Toxicology, V8, N4, 432-442, 1987.

27. Thigpen JE, Lung-An L, Richter CB, Lebetkin, EH, Haseman, JK, **Jameson CW**. The Mouse Bioassay Test for the Detection of Estrogenic Activity in Feeds and Foodstuffs. Part I: A Standardized Method for Conducting the Mouse Bioassay using the CD-1 Mouse. Laboratory Animal Science, V37, N5, 596-601, 1987.

28. Thigpen JE, Lung-An L, Richter CB, Lebetkin EH, **Jameson CW**. The Mouse Bioassay Test for the Detection of Estrogenic Activity in Feeds and Foodstuffs. Part II: The Comparative Estrogenic Activity of Purified, Certified Standard, Open and Closed Formula Rodent Diets. Laboratory Animal Science, V37, N5, 602-605, 1987.

29. Bucher JR, Gupta BN, Adkins B, Thompson M, **Jameson CW**, Thigpen J E, Schwetz BA. The Toxicity of Inhaled Methyl Isocyanate in F344/N Rats and B6C3F1 Mice. I: Acute Exposure and Recovery Studies. Environmental Health Perspectives, V72, 53-61, 1987.

30. Luster MI, Germolec DR, Burleson GR, **Jameson CW**, Ackermann MF, Lamm KR, Hayes HT. Selective Immunosuppression in Mice of Natural Killer Cell Activity by Ochratoxin A. Cancer Research, Vol. 47, 2259-2263, 1987.

31. Dieter MP, **Jameson CW**, Tucker AN, Luster MI, French JE, Hong, HL, Boorman, GA. Evaluation of Tissue Disposition, Myelopoietic and Immunologic Responses in Mice After Long-term Exposure to Nickel Sulfate in the Drinking Water. Journal of Toxicology and Environmental Health, V24, 357-372, 1988.

32. Huff JE, McConnell EE, Haseman JK, Boorman GA, Eustis SL, Schwetz BA, Rao GN, **Jameson CW**, Hart LG, Rall DP. Carcinogenesis Studies Results of 398 Experiments on 104 Chemicals from the U. S. National Toxicology Program. Annuals of the New York Academy of Sciences V534, 1-30, 1988.

33. Shan A, Harben D, **Jameson CW**. Analyses of Two Azo Dyes by High Performance Liquid Chromatography. Journal of Chromatographic Science, V26, 439-442, 1988.

34. Hong HL, Canipe J, **Jameson CW**, Boorman GA: Comparative Effects of Ethylene Glycol and Ethylene Glycol Monomethyl Ether Exposure on Hematopoiesis and Histopathology in B6C3F1 Mice. Journal of Environmental Pathology, Toxicology, and Oncology, V8, N7, 27-38, 1988.

35. Hong HL, **Jameson CW**, Boorman GA. Residual Hematopoietic Effect of Ochratoxin A in Mice Exposed to Irradiation. Toxicology, V53, 57-67, 1988.

36. Dieter MP, **Jameson CW**, French JE, Gangjee S, Stefanski SA, Chan, PC. Development and Validation of a Cellular Transplant Model for Leukemia in Fischer Rats: A Short-term Assay for Potential Anti-leukemic Chemicals. Leukemia Research, V13, 841-849, 1989.

37. Timmons L, Brown R, Arneson DW, **Jameson CW**. Rapid Determination of Low pg/mg Amounts of N-Nitrosodiethylamine in Rodent Body Fluid and Tissue Samples by Isotope-Dilution High Resolution Mass Spectrometry. J. Anal. Tox., V13, N6, 333-336, 1989.

38. Heindel JJ, Lamb JC, Chapin RE, Gulati DK, Hope E, George J, **Jameson CW**, Teague J, Schwetz BA. Reproductive Toxicity Testing by Continuous Breeding Test Protocol in CD-1 Mice. DHHS Publication No. (NIH) 89 Washington, DC, US Government Printing Office, 1989.

39. Cannon JM, Brown D, Murrill EM, **Jameson CW**. Identification of Components in Iodinated Glycerol. Journal of Pharmaceutical Sciences, V78, N1, 48-51, 1989.

40. Morgan DL, **Jameson CW**, Mennear JH, Prejean JD. 14-Day and 90-Day Toxicity Studies of C.I. Pigment Red 3 in Fischer 344 Rats and B6C3F1 Mice. Fd. Chem. Toxic., V27, N12, 793-800, 1989.

41. Morgan DL, **Jameson CW**, Mennear JH, Ulland BM. Thirteen-Week Toxicity Studies of C1 Direct Blue 15 and 3,3'-Dimethoxybenzidine in the Fischer 344 Rat. Toxicology, V59, 297-309, 1989.

42. Dieter MP, **Jameson CW**, Maronpot RR, Langenbach RJ, Braun AG. The Chemotherapeutic Potential of Glycol Alkyl Ethers: Structure-Activity Studies of Nine Compounds in a Fischer Rat Leukemia Transplant Model. Cancer Chemother. Pharmacol., 26, 173-180, 1990.

43. Gorski T, Goehl TJ, **Jameson CW**, Collins BJ. Sources of Error in the Determination of Trichloroethylene in Blood. Bull. Environ. Contam. Toxicol., V45, 1-5, 1990.

44. Dieter MP, Boorman GA, **Jameson CW**, Matthews HB, Huff JE. The Carcinogenic Activity of Commercial Grade Toluene Diisocyanate in Rats and Mice in Relation to the Metabolism of the 2,4- and 2,6-TDI Isomers. Toxicology and Industrial Health, V6, No. 6, 599-621, 1990.

45. Morrissey RE, Fowler BA, Harris MA, Moorman MP, **Jameson CW**, Schwetz BA. Arsine: Absence of Developmental Toxicity in Rats and Mice. Fundamental and Applied Toxicology 15, 350-356, 1990.

46. **Jameson CW**, NTP Technical Report on the Toxicology and Carcinogenesis Studies of d-Limonene in F344/N Rats and B6C3F1 Mice (Gavage Studies). NIH Publication No. 347, 1990.

47. Gorski T, Goehl TJ, **Jameson CW**, Collins BJ, Bursey J, Moseman R. Gas Chromatic Determination of 2-Ethylhexanol and 2-Ethylhexanoic Acid as Derivatives suitable for Electron Capture and Nitrogen-

Phosphorus Detection After Single Reaction with Heptafluorobutyrlimidazole. Journal of Chromatography, 509, 383-389, 1990

48. Yuan J, **Jameson CW**, Goehl TJ, Collins BJ, Corniffee G, Kuhn G, Castro C. Effects of Physical Binding of o-Nitroanisole with Feed Upon its Systemic Availability in Male F344 Rats. Bulletin of Environmental Contamination and Toxicology, 47: 152-159, 1991.

49. Yuan J, **Jameson CW**, Goehl TJ, Collins BJ, Purde W, Judd L. Application of Molecular Encapsulation for Toxicity Studies: Toxicokinetics of p-Chloro-α,α,α-trifluorotoluene in β-Cyclodextrin or Corn Oil Vehicles in Male F344 Rats. Toxicology and Applied Pharmacology, 111, 107-115, 1991.

50. Dieter MP, **Jameson CW**, Elwell M, Lodge JW, Hejtmancik M, Grumbein SL, Ryan M, Peters AC. Comparative Toxicity and Tissue Distribution of Antimony Potassium Tartrate in Rats and Mice Dosed by Drinking Water and Intraperitoneal Injection. Journal of Toxicology and Environmental Health, 34, 51-82, 1991.

51. Yuan J, Bucher JR, Goehl TJ, Dieter MP, **Jameson CW**. Quantitation of Cinnamaldehyde and Cinnamic Acid in Blood by HPLC. Journal of Analytical Toxicology, 16, N6: 359-362, 1992.

52. Yuan J, **Jameson CW**, Goehl TJ, Elwell MR, Leininger JR, Thompson MB, Corniffe G, Carleton T. Application of Molecular Encapsulation for Toxicology Studies: Comparative Toxicity of p-Chloro-α,α,α-trifluorotoluene in β-Cyclodextrin Vehicle versus Corn Oil Vehicle in Male and Female Fischer 344 Rats and B6C3F1 Mice. Fundamental and Applied Toxicology, 18, 460-470, 1992.

53. Dieter MP, Maronpot RR, **Jameson CW**, Ward SM. The Effects of Iodinated Glycerol, Trichlorfon, Acetaminophen on Tumor Progression in a Fischer Rat Leukemia Transplant Model. Cancer Detection and Prevention, V16, No. 3, 173-183, 1992.

54. Yuan J, Dieter MP, Bucher JR, **Jameson CW**. Toxicokinetics of Cinnamaldehyde in F344 Rats. Food and Chemical Toxicology, 30, N12: 997-1004, 1992.

55. Yuan J, **Jameson CW**, Goehl TJ, Collins BJ. Molecular Encapsulator: A Novel Vehicle for Toxicology Studies. Toxicology Methods, V1, No.4, 231-241, 1992.

56. Dieter MP, Boorman GA, **Jameson CW**, Eustis SL. Development of Renal Toxicity in F344 Rats gavaged with Mercuric Chloride for 2 Weeks, or 2, 4, 6, 15, and 24 Months. Journal of Toxicology and Environmental Health, 36, 319-340, 1992.

57. Yuan J, Dieter MP, Bucher JR, **Jameson CW**. Application of Microencapsulation for Toxicology Studies III, Bioavailability of Microencapsulated Cinnamaldehyde. Fundamental and Applied Toxicology, 20, N1: 83-87, 1993.

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5. 2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) - 2002
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9. 2,2-bis-(bromomethyl)-1,3-propanediol (BBMP) (Technical Grade) - 2000
10. Boot & Shoe Manufacturing - 1998
11. 1,3- Butadiene - 1997
12. Cadmium and Cadmium Compounds - 1997
13. Chloramphenicol - 2000
14. Chloroprene - 1997
15. Chlorozotocin - 1996
16. p -Chloro-o-toluidine and its Hydrochloride Salt - 1996
17. Cobalt Sulfate - 2002
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19. Danthron (1,8-Dihydroxyanthraquinone) - 1996
20. Diazoaminobenzene - 2002
21. 2,3-Dibromo-1-propanol - 2000
22. Diesel Exhaust Particulates - 1998
23. Diethanolamine - 2002
24. 1,6-Dinitropyrene & 1,8-Dinitropyrene - 1996
25. Disperse Blue 1 - 1996
26. Dyes Metabolized to Benzidine (Benzidine Dyes as a Class) - 1997
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29. Environmental Tobacco Smoke - 1998
30. Estrogens, Steriodal - 2000
31. Ethyl Acrylate - 1998
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33. Furan - 1996
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37. Isoprene - 1998
38. Lead and Lead Compound - 2003
39. Methyleugenol - 2000
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41. Naphthalene - 2002
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45. Nitrobenzene - 2002
46. 6-Nitrochrysene - 1996
47. Nitromethane - 2002
48. 1-Nitropyrene - 1996
49. 4-Nitropyrene - 1996
50. Phenolphthalein - 1997
51. Saccharin - 1997
52. Silica, Crystalline (Respirable Size) - 1998
53. Smokeless Tobacco - 1997
54. Solar Radiation & Exposure to Sunlamps or Sunbeds - 1997
55. Strong Inorganic Acid Mists Containing Sulfuric Acid - 1997
56. Styrene-7,8-oxide - 2000

57. Tamoxifen - 1997
58. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) - 1997
59. Tetrafluoroethylene - 1997
60. 4,4'-Thiodianiline - 2002
61. Thiotepa - 1996
62. Tobacco Smoking - 1997
63. 1,2,3-Trichloropropane - 1996
64. Trichloroethylene - 1997, 2000
65. Ultraviolet (UV) Radiation, Broad Spectrum and UVA, UVB, and UVC - 2000
66. Vinyl Bromide - 2000
67. Vinyl Fluoride - 2000
68. Wood Dust - 2000
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CWJ/Greim Experimental Animal Summary

Mouse

Study	Strain	Dose	Tumors	Significance	Evaluation
Greim: Knezevich and Hogan (1983) (Study 10)	Mouse,CD-1 (males)	0, 1,000, 5,000, or 30,000 ppm in feed for 24 months	Renal tubule adenoma: 1/49 (2%), 0/49, 0/50, 1/50 (2%) Renal tubule carcinoma: 0/49, 0/49, 1/50 (2%), 2/50 (4%) Renal tubule adenoma or carcinoma (combined): 1/49 (2%), 0/49, 1/50 (2%), 3/50 (6%)	*P* for trend = 0.037 (EPA) *P* for trend = 0.034 (EPA)	Historical control data from 14 studies conducted between 1977 and 1981 at the testing laboratory indicated that the mouse renal tumors ranged from 0 to 3% and the incidence in the current study (3/50; 6%) exceeded the upper limit of the historical control range by a factor of two. For the purpose of this hazard identification the increase the incidence of carcinoma of the renal tubule and the incidence of adenoma or carcinoma (combined) of the renal tubule in male mice is due to treatment with glyphosate
Greim: Atkinson *et al.* (1993) (Study 11)	Mouse,CD-1 (males)	0, 100, 300, 1000 mg/kg bw in feed for 104wk	Males: Haemangiosarcoma: 0/50, 0/50, 0/50, 4/50 (8%)	*P* for trend <0.01 (EPA)	The EPA pointed out that the incidence in the high dose males was near the upper limit (0-8%) for the performing laboratory. For the purpose of this hazard identification the increased incidence of hemangiosarcomas in male mice is due to the treatment with glyphosate
Greim: Sugimoto, (1997) (Study 12)	Mouse,CD-1 (M&F)	0, 1600, 8000, or 40 000 ppm in feed for 18 months	Males: Hemangiosarcomas: 0/50, 0/50, 0/50, 2/50 (4%) Kidney: renal cell adenomas 0/50; 0/50; 0/50; 2/50 (4%) Malignant lymphoma 2/50 (4%), 2/50 (4%), 0/50, 6/50 (12%) [0/26, 0/34, 1/27 (4%), 5/29* (17%) – Greim Tier II] Females: Hemangiomas: (0/50; 0/50; 2/50, (4%); 5/50*, (10%)	*P* for trend=0.008 (Portier) *P* for trend=0.008 (Portier) *P* for trend=0.008 (Portier) [**P* <0.05, Greim Tier II] **P* = 0.028, (EPA) *P* for trend=0.002	The significant increase in malignant lymphoma in high dose male mice, and the significant trend in the development of hemangiosarcomas, malignant lymphomas, and renal adenomas in male mice is due to treatment with glyphosate that caused these cancers in male CD-1 mice. The significant trend in the development of hemangiosarcomas in female mice is also related to treatment with glyphosate that caused this cancer in female CD-1 mice.
Greim: Kumar (2001) (Study 13)	Mouse-Swiss (M&F)	0, 100, 1000, or 10000 ppm in feed for 18 months.	Males: Malignant lymphoma: 10/50 (20%), 15/50 (30%), 16/50 (32%), 19/50* (38%) Kidney: renal cell adenomas: 0/50, 0/26, 1/26 (4%), 2/50 (4%)	**P*<0.05, *P* for trend = 0.05 (Portier) *P* for trend=0.04 (Portier)	The incidence of malignant lymphoma in the high dose male was double the historical rate, reported to be 18%[87] for males, and for high dose female mice the incidence was well above the historical rate of 41%[87].

			Females: Malignant lymphoma: 18/50 (36%), 20/50 (40%), 19/50 (38%), 25/50* (50%)	*P<0.05, P for trend=0.05 (Portier)	For the purpose of this hazard identification the formation of malignant lymphoma in the male and female mice and the renal cell adenomas in males in this study is due to treatment with glyphosate
Greim: Wood (2009) (Study14)	Mouse,CD-1(males)	0, 500, 1500, or 5000 ppm in feed for 18 months.	Malignant lymphomas: 0/51, 1/50(10%), 2/51(4%), 5/51*(10%) Lung: Adenocarcinomas: 5/51(10%), 5/51(10%), 7/51(14%), 11/51(22%)	*P<0.05, P for trend<0.01 (EPA) P for trend<0.01 (EPA)	For the purpose of this hazard identification the formation of malignant lymphomas and the formation of adenocarcinomas of the lung in this study is due to treatment with glyphosate

CWJ/Greim Experimental Animal Summary

Rat

Study	Strain	Dose	Tumors	Significance	Evaluation
Greim: Lankas, *et al.* (1981) (Study1)	Rat, Sprague-Dawley (Males & Females)	0, 30, 100, 300 ppm in feed for up to 26 months	Males: Testes: Interstitial cell tumors 0/50, 3/5 (6%), 1/50 (2), 6/50* (12%) Pancreas (isletcell): Adenoma: 0/50, 5/49** (10%), 2/50 (4%), 2/50 (4%)	*P =0.013 (EPA) **P < 0.05 (EPA)	The incidence of interstitial cell tumors in the testes in the high dose animals in this study is almost twice that seen in the range of this tumor (3.4% to 6.7%) in control animals (historical controls) from 5 contemporary studies[87] For the purpose of this hazard identification the increase in incidence of testes interstitial cell tumors and pancreatic cell tumors in male rats are due to the treatment with glyphosate
Greim: Stout, *et al.* (1990) (Study 2)	Rat, Sprague-Dawley (Males & Females)	0, 2000, 8000, or 20,000 ppm in feed for 24 months	Males: Pancreas (islet cell): Adenoma: 1/58 (2%), 8/57 (14%)*, 5/60 (8%), 7/59 (12%) Liver: Hepatocellular adenoma: 2/60 (3%), 2/60 (3%), 3/60 (6%), 7/60 (12%) Females: Thyroid: C-cell adenoma: 2/60 (3%), 2/60 (3%), 6/60 (10%), 6/60 (10%)	*P <0.05 (EPA performed additional analyses excluding animals that died or were killed before wk 54–55: Adenoma: 1/43 (2%), 8/45 (18%; P = 0.018), 5/49 (10%), 7/48 (15%; P=0.042) P for trend = 0.016 (EPA) P for trend = 0.033 (EPA)	The incidence of these adenomas in the low (18%) and high (15%) dose males was almost twice that seen in historical controls. The range for historical controls for pancreatic islet cell adenoma reported in males at this laboratory was 1.8–8.5%[77] For the purpose of this hazard identification glyphosate caused an increase in incidence of pancreatic islet cell adenoma in male rats. Glyphosate also caused a significant increase in the trend for formation of hepatocellular adenomas in male Sprague-Dawley rats and of thyroid follicular cell adenomas and adenomas and carcinomas combined in female Sprague-Dawley rats.
Greim: Atkinson *et al.* (1993)(Study 3)	Rat, Sprague-Dawley (Males & Females)	0, 10, 100, 300, or 1,000 mg/kg bw/day in feed for 104 weeks			Neoplasms were noted in control and treated groups, but dose responses were not evident, and no statistically significant increase versus controls were noted for any tumor type.
Greim: Suresh (1996) (Study 4)	Rat-Wistar (Males &Females)	0, 1600, 8000, or 40 000 ppm in feed for 18 months			There were no treatment related deaths or clinical signs in any of the dose-groups and no treatment related effects on body weight gain or food consumption noted. This suggests that the MTD was not reached, and this study is inadequate for the

					evaluation of the carcinogenicity of glyphosate.
Greim: Excel (1997) (Study 5)	Rat, Sprague-Dawley (Males & Females)	0, 3000, 15 000, and 25 000 ppm in feed for 24 months			Concur with Greim that study is unreliable for carcinogenicity evaluation
Greim: Enomoto (1997) (Study 6)	Rat, Sprague-Dawley (Males & Females)	0, 3,000, 10,000, or 30,000 ppm in feed for 24 months			There were no statistically significant increases in any tumor type reported for this study.
Greim: Brammer (2001) (Study 7)	Rat, Wistar (Males &Females)	0, 2,000, 6,000, and 20,000 ppm in feed for 24 months	Males: Liver: hepatocellular adenomas 0/52, 2/52, (4%), 0/52, 5/52* (10%)	*P=0.03 (EPA) P for trend =0.008 (EPA)	The incidences of liver tumors observed were within the historical range (0–11.5%) for this strain of rats in 26 studies conducted during the relevant time period (1984–2003) at the testing laboratory. For the purpose of this hazard identification, the increase in hepatocellular adenomas in male Wistar rats could not be attributed to exposure to glyphosate despite the fact that there was an observation of increased incidence of hepatocellular adenomas in male rats.
Greim: Wood (2009) (Study 8)	Rat, Wistar (Males &Females)	0, 3,000, 10,000, or 15,000 ppm in feed for 24 months			There were no treatment-related deaths or clinical signs in any of the dose-groups. No significant treatment-related effects on mortality were observed during the study. This suggests that the MTD was not reached, and this study is inadequate for the evaluation of the carcinogenicity of glyphosate.
Greim: Chruscielska *et al.* (2000) (Study 9)	Rat, Wistar (Males &Females)	0, 300, 900, and 2700 mg/L in drinking water for 24 months			There was limited information provided on dosing regimen, histopathological examination method, and tumor incidences that makes this study inadequate for the purpose of this hazard assessment

11th Report on Carcinogens

2004

U.S. Department of
Health and Human Services
Public Health Service
National Toxicology Program

Pursuant to Section 301(b) (4)
of the Public Health Service Act
as Amended by Section 262, PL 95-622

Report on Carcinogens, Eleventh Edition

Carcinogen Profiles
2004

U.S. Department of Health and Human Services
Public Health Service
National Toxicology Program

Prepared for the
National Institute of Environmental Health Sciences
Research Triangle Park, North Carolina

Prepared by
Constella Group, Incorporated
Durham, North Carolina
Under Contract Number NO1- ES-35505

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III. Substance Profiles *(Continued)*

Introduction

The probability that a resident of the United States will develop cancer at some point in his or her lifetime is 1 in 2 for men and 1 in 3 for women (ACS 2004). Nearly everyone's life has been directly or indirectly affected by cancer. Most scientists involved in cancer research believe that the environment in which we live and work may be a major contributor to the development of cancer (Lichtenstein *et al.* 2000). In this context, the "environment" is anything that people interact with, including exposures resulting from lifestyle choices, such as what we eat, drink, or smoke; natural and medical radiation, including exposure to sunlight; workplace exposures; drugs; socioeconomic factors that affect exposures and susceptibility; and substances in air, water, and soil (OTA 1981, IOM 2001). Other factors that play a major role in cancer development are infectious diseases, aging, and individual susceptibility, such as genetic predisposition (Montesano 2001). We rarely know what environmental factors and conditions are responsible for the onset and development of cancers; however, we have some understanding of how some types of cancer develop, especially cancers related to certain occupational exposures or the use of specific drugs. Many experts firmly believe that much of the cancer associated with the environment may be avoided (Tomatis *et al.* 1997).

The people of the United States, concerned about the relationship between their environment and cancer, have asked, through the U.S. Congress, for information about substances that are known or appear likely to cause cancer (i.e., to be carcinogenic). Section 301(b)(4) of the Public Health Service Act, as amended, provides that the Secretary of the Department of Health and Human Services (DHHS) shall publish a biennial report that contains the following information:

A) A list of all substances (1) which either are known to be human carcinogens or may reasonably be anticipated to be human carcinogens and (2) to which a significant number of persons residing in the United States are exposed.
B) Information concerning the nature of such exposure and the estimated number of persons exposed to such substances.
C) A statement identifying (1) each substance contained in this list for which no effluent, ambient, or exposure standard has been established by a Federal agency and (2) for each effluent, ambient, or exposure standard established by a Federal agency with respect to a substance contained in this list, the extent to which such standard decreases the risk to public health from exposure to the substance.
D) A description of (1) each request received during the year to conduct research into, or testing for, the carcinogenicity of a substance and (2) how the Secretary and other responsible entities responded to each request.

The Report on Carcinogens (RoC) is an informational scientific and public health document that identifies and discusses agents, substances, mixtures, or exposure circumstances that may pose a hazard to human health by virtue of their carcinogenicity. It serves as a meaningful and useful compilation of data on (1) the carcinogenicity (ability to cause cancer), genotoxicity (ability to damage genes), and biologic mechanisms (modes of action in the body) of the listed substances in humans and/or in animals, (2) the potential for human exposure to these substances, and (3) Federal regulations to limit exposures. The RoC does not present quantitative assessments of the risks of cancer associated with these substances. Thus listing of substances in the RoC only indicates a potential hazard and does not establish the exposure conditions that would pose cancer risks to individuals in their daily lives. Such formal risk assessments are the responsibility of the appropriate federal, state, and local health regulatory and research agencies.

The substances listed in the RoC are either known or reasonably anticipated to cause cancer in humans in certain situations. With many listed substances, cancer may develop only after prolonged exposure. For example, smoking tobacco is known to cause cancer in humans, but not all people who smoke develop smoking-related cancer. With some substances or exposure circumstances, however, cancer may develop after even brief exposure. Examples include certain occupational exposures to asbestos or bis(chloromethyl) ether. The cancer hazard that listed substances pose to any one person depends on many factors. Among these are the intrinsic carcinogenicity of the substance, the amount and duration of exposure, and an individual's susceptibility to the carcinogenic action of the substance. Because of these considerations, the RoC does not attempt to rank substances according to the relative cancer hazards they pose.

Potential Beneficial Effects of Listed Carcinogens

As stated above, the purpose of the RoC is to identify hazards to human health posed by carcinogenic substances; therefore, it is not within the scope of this report to address potential *benefits* of exposure to certain carcinogenic substances in special situations. For example, numerous drugs typically used to treat cancer or other medical conditions have been shown to increase the frequency of primary or secondary cancers in patients undergoing treatment for specific diseases. In these cases, the benefits of using the drug to treat or prevent a specific disease outweigh the added cancer risks associated with its use. Personal decisions concerning voluntary exposure to carcinogenic substances should be based on information that is beyond the scope of the RoC. Individuals should not make decisions concerning the use of a given drug, or any other listed substance, based solely on the information contained in the RoC. Such decisions should be made only after consultation with a physician or other appropriate specialist.

Identification of Carcinogens

For many years, government research agencies (including the National Toxicology Program), industries, academia, and other research organizations have studied various substances to identify those that may cause cancer. Much of this information on specific chemicals or occupational exposures has been published in the scientific literature or in publicly available and peer-reviewed technical reports. This literature is a primary source of information for identifying and evaluating substances for listing in the RoC. Many of the listed substances also have been reviewed and evaluated by other organizations, including the International Agency for Research on Cancer (IARC) in Lyon, France, the Environmental Protection Agency of the State of California, and other U.S. Federal and international agencies.

Both human and laboratory animal studies are used to evaluate whether substances are possible human carcinogens. The strongest evidence for establishing a relationship between exposure to any given substance and cancer in humans comes from epidemiological studies—studies of the occurrence of a disease in a defined population and the factors that affect its occurrence (Bradford 1971). Epidemiological studies of human exposure and cancer are difficult (Rothman 1986). They must rely on natural, not experimental, human exposures and must therefore consider many factors that may affect cancer prevalence besides the exposure under study. One such factor is the latency period for cancer development. The exposure to a carcinogen often occurs many years (sometimes 20 to 30 years or more) before the first sign of cancer appears. Another valuable method for identifying substances as potential human carcinogens is the long-term animal bioassay. These studies provide accurate information about dose and duration of exposure and they are less affected than epidemiology studies by possible interaction of the test substance with other chemicals or modifying factors (Huff 1999). In these studies, the substance is given to one or (usually) two species of laboratory rodents over a range of doses for nearly the animals' entire lives.

Experimental cancer research is based on the scientific assumption that substances causing cancer in animals will have similar effects in humans. It is not possible to predict with complete certainty from

animal studies alone which substances will be carcinogenic in humans. However, known human carcinogens that have been tested adequately in laboratory animals also cause cancer in laboratory animals (Fung *et al.* 1995). In many cases, a substance first was found to cause cancer in animals and later confirmed to cause cancer in humans (Huff 1993). How laboratory animals respond to substances, including developing cancer and other illnesses, does not always strictly correspond to how people will respond. Nevertheless, laboratory animal studies remain the best tool for detecting potential human health hazards of all kinds, including cancer (OTA 1981, Tomatis *et al.* 1997).

Listing Criteria

The criteria for listing an agent, substance, mixture, or exposure circumstance in the RoC are as follows:

Known To Be Human Carcinogen:

There is sufficient evidence of carcinogenicity from studies in humans*, which indicates a causal relationship between exposure to the agent, substance, or mixture, and human cancer.

Reasonably Anticipated To Be Human Carcinogen:

There is limited evidence of carcinogenicity from studies in humans*, which indicates that causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding factors, could not adequately be excluded,

or

there is sufficient evidence of carcinogenicity from studies in experimental animals, which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors (1) in multiple species or at multiple tissue sites, or (2) by multiple routes of exposure, or (3) to an unusual degree with regard to incidence, site, or type of tumor, or age at onset,

or

there is less than sufficient evidence of carcinogenicity in humans or laboratory animals; however, the agent, substance, or mixture belongs to a well-defined, structurally related class of substances whose members are listed in a previous Report on Carcinogens as either known to be a human carcinogen or reasonably anticipated to be a human carcinogen, or there is convincing relevant information that the agent acts through mechanisms indicating it would likely cause cancer in humans.

Conclusions regarding carcinogenicity in humans or experimental animals are based on scientific judgment, with consideration given to all relevant information. Relevant information includes, but is not limited to, dose response, route of exposure, chemical structure, metabolism, pharmacokinetics, sensitive sub-populations, genetic effects, or other data relating to mechanism of action or factors that may be unique to a given substance. For example, there may be substances for which there is evidence of carcinogenicity in laboratory animals, but there are compelling data indicating that the agent acts through mechanisms which do not operate in humans and would therefore not reasonably be anticipated to cause cancer in humans.

*This evidence can include traditional cancer epidemiology studies, data from clinical studies, and/or data derived from the study of tissues or cells from humans exposed to the substance in question that can be useful for evaluating whether a relevant cancer mechanism is operating in people.

The listing criteria presented here were first adopted for use in the *Eighth Report on Carcinogens*, which was published in 1998. The clarification noted above was issued in a *Federal Register* notice dated April 2, 1999 (see 64FR15983-15984, see also *Federal Register* notice dated April 19, 1999: 64FR 19188-19189). Listing criteria for substances listed in earlier editions of the RoC are outlined in the introductions to those editions.

Preparation of the RoC

Within the DHHS, the Secretary has delegated the responsibility for preparing the RoC to the National Toxicology Program (NTP). The process used to prepare the RoC involves several levels of review of the nominations considered for listing in or delisting (removal) from the report. Opportunities for public comment and participation are an integral part of the review process.

Nominations for listing in or delisting from the RoC are received from a number of sources. Periodic requests for nominations from the public are published in the *Federal Register*, the NTP Update newsletter, and other appropriate publications. The NTP actively solicits nominations from member agencies of the NTP Executive Committee.[1] Nominations for the RoC also come from reviews of the literature performed by the NTP. Potential nominations are identified from such sources as the NTP Technical Reports, the IARC *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, the California Environmental Protection Agency's Carcinogen List, and other similar sources.

Two Federal scientific review groups and one non-governmental scientific peer-review body (a standing subcommittee of the NTP Board of Scientific Counselors) evaluate the nominations for listing in or delisting from the RoC. Each group reviews the relevant data on the carcinogenicity of the substances nominated and the exposure of U.S. residents to the substances. The members of these three review groups may be found in Appendix D, List of Participants.

The nominations for listing in the *Eleventh Report on Carcinogens* initially were evaluated by a Report on Carcinogens Review Committee (RG1), composed of scientists from the National Institute of Environmental Health Sciences. For each nomination, the RG1 determined whether the information available was sufficient for applying the criteria for listing and whether the nomination warranted formal consideration by the NTP. This committee received the information submitted with each nomination and any relevant supplemental materials identified by RoC staff. For each nomination the committee reviewed this information and made a formal recommendation to the Director, NTP, either to continue with the formal review for listing or delisting or not to pursue the nomination at that time. The criterion for not pursuing a nomination was the lack of sufficient information for applying the listing criteria. Those nominations not accepted for review were returned to the original nominator who was invited to resubmit the nomination with additional justification, such as new cancer data or exposure information. The NTP Executive Committee and the NTP Board of Scientific Counselors were informed of all nominations not accepted for review.

Upon approval of the nominations by the Director, the NTP announced its intent to review the nominations for the *Eleventh Report on Carcinogens* and solicited public comment on all nominations through announcements in the *Federal Register* and NTP publications. The NTP then initiated an independent search and

[1]Agencies represented on the NTP Executive Committee include: Agency for Toxic Substances and Disease Registry (ATSDR), Consumer Product Safety Commission (CPSC), Environmental Protection Agency (EPA), Food and Drug Administration (FDA), National Center for Environmental Health (NCEH/CDC), National Institute for Occupational Safety and Health (NIOSH), Occupational Safety and Health Administration (OSHA), Department of Health and Human Services (DHHS). National Institutes of Health (NIH), National Cancer Institute (NCI), and National Institute of Environmental Health Sciences/NTP (NIEHS/NTP).

review of the scientific literature and prepared a background document for each nomination under consideration. The comments received in response to the public announcement were used to help identify issues that should be addressed in the background documents. Whenever possible, the background documents were prepared with the assistance of a consultant or a panel of consultants with recognized expertise on the nomination.

The RG1 then conducted the initial scientific review of a nomination for listing in the *Eleventh Report on Carcinogens*. The RG1 first reviewed the background document prepared for each nomination and determined whether it was adequate for use in reviewing the nomination and applying the criteria for listing in the RoC. After acceptance of the background document the RG1 then proceeded with scientific review of the nomination. It considered the information in the background document and all public comments received in response to the announcement of the nomination, and made a formal recommendation to the NTP Director for its listing in the RoC. Upon acceptance of the background document by the RG1, it was considered the final document of record and was placed on the NTP RoC web site with a notice published on the NTP list-serv and the NTP home web site announcing its availability.

The NTP Executive Committee's Interagency Working Group for the Report on Carcinogens (RG2), a governmental interagency scientific review group, conducted a second review of the nominations. For each nomination, the RG2 assessed whether relevant information was available and sufficient for its listing in the RoC. The RG2 considered the original nomination, the background document, and all public comments received in response to announcements of the nominations. Upon completion of its review, the RG2 made its formal recommendations to the NTP Director for listing the nominations in the RoC.

The third review of the nominations was an independent external scientific peer review by a standing subcommittee of the NTP Board of Scientific Counselors (the RoC Subcommittee). The RoC Subcommittee assessed whether the relevant information available for each nomination was sufficient for its listing in the RoC. This review was conducted in an open public meeting. A notice of the review announcing the meeting and the availability of the background documents, and soliciting public comment on the nominations was published in the *Federal Register* and NTP publications. The notice invited interested groups or individuals to submit written comments and/or address the RoC Subcommittee during the public meeting. Upon completion of its review, the RoC Subcommittee made its formal recommendations to the NTP Director for listing the nominations in the RoC.

Following completion of the reviews by the RG1, RG2 and RoC Subcommittee, the NTP published the nominations and the review groups' recommendations for each nomination in the *Federal Register,* and solicited the third and final round of public comment and input on the nominations.

The recommendations of the RG1, RG2, and RoC Subcommittee and all public comments received were presented to the NTP Executive Committee for review and comment. The NTP Executive Committee reviewed the information on each nomination and provided to the NTP Director a recommendation on its listing in the RoC.

The NTP Director received the independent recommendations of the RG1, RG2 and RoC Subcommittee, the opinion of the NTP Executive Committee, and all public comments concerning the nominations. The NTP Director evaluated this input and any other relevant information on the nominations and developed recommendations to the Secretary, DHHS regarding whether to list or not to list the nominations in the RoC.

The NTP prepared the final draft of the RoC based on the NTP Director's recommendations and submitted it to the Secretary, DHHS, for review and approval. Upon approval of the RoC, the Secretary submitted it to the U. S. Congress as a final document. Submittal of the RoC to Congress constituted publication of the report, and it became available to the public at that time. The NTP published a notice of the publication and availability of the Eleventh Edition of the RoC, indicating all newly listed agents, substances, mixtures or exposure circumstances in the *Federal Register* and NTP publications.

Estimation of Exposure

The RoC is required to list only substances to which a significant number of people living in the United States are exposed; therefore, substances to which very few people are exposed are generally not listed. Some substances that have been banned or restricted in use (e.g., safrole, arsenical pesticides, and mirex) are listed either because people who were previously exposed remain potentially at risk or because these substances still are present in the environment.

The RoC also is required to provide information about the nature of exposures and the estimated numbers of people exposed to listed substances. Four of the agencies participating with the NTP in preparation of the *Eleventh Report on Carcinogens*—the Consumer Product Safety Commission (CPSC), U.S. Environmental Protection Agency (EPA), Food and Drug Administration (FDA), and Occupational Safety and Health Administration (OSHA)—are responsible for regulating hazardous substances and limiting the exposure to and use of such substances. Information on use, production, and exposure in each entry of the RoC was reviewed by staff members from these four regulatory agencies. Because little information typically is available, estimating the number of people who could be exposed, and the route, intensity, and duration of exposure for each substance is a very difficult task. This RoC attempts to respond to these questions, and adequate answers that could be obtained are included in the individual profiles for each listing.

The National Institute for Occupational Safety and Health (NIOSH) has conducted two occupational exposure surveys: the National Occupational Hazard Survey (NOHS), conducted from 1972 to 1974, and the National Occupational Exposure Survey (NOES), conducted from 1981 to 1983. These surveys yielded data on potential exposure to many listed substances. Although dated, NOES estimates are provided in the profiles of the listings when available, and NOHS figures are given in some profiles if no other exposure data were available.

Regulations and Guidelines

The RoC is required to identify each listed substance for which no standard for exposure or release into the environment has been established by a Federal Agency. The *Eleventh Report on Carcinogens* addresses this requirement by providing in each profile a summary of the regulations and guidelines that are likely to decrease exposure to that substance. Some of these regulations and guidelines have been enacted for reasons other than the substance's carcinogenicity (*for example,* to prevent adverse health effects other than cancer or to prevent accidental poisoning of children). These regulations are included in the profiles, because reduction of exposure to a carcinogen will likely reduce the risk for cancer. In earlier editions of the RoC, each profile contained a summary of relevant regulations with a cumulative list of the *Code of Federal Regulations* and *Federal Register* citations for each listing published in a separate volume. All regulations have been researched and presented in the *Eleventh Report on Carcinogens* using a new format. Starting with this edition, the regulations for a listing are organized by regulatory agencies and major acts, and are provided at the end of the profile rather than in a separate volume.

The majority of the regulations cited in the RoC were enacted by the following federal agencies: CPSC, the U.S. Department of Transportation, the EPA, the FDA, and OSHA. The guidelines cited

in the RoC are primarily those published by NIOSH and the American Conference of Governmental Industrial Hygienists. Additionally, regulations and guidelines enacted by other governmental agencies not listed above are cited if their likely outcome is to reduce exposure to the substance. It is beyond the scope of this report to provide detailed information or interpretation concerning the implementation of each regulatory act, and no attempt is made to do so. Some commonly used regulatory terms are defined in the glossary (Appendix F), and links to the websites for the *Code of Federal Regulations* and for each of the major regulatory agencies are provided in the reference section of this Introduction for those wishing to obtain additional information on these agencies and their regulations.

Two regulations were identified that apply to all substances listed in the RoC:

1. OSHA's Hazard Communication Standard
   This regulation is intended to communicate the hazards of chemicals and appropriate protective measures to protect employees. The program includes maintenance of a list of hazardous chemicals, labeling of containers in the workplace, and preparation and distribution of material safety data sheets to employees. The rule states that chemicals shall be considered "hazardous" if they have been listed as a carcinogen or potential carcinogen in (1) the NTP's RoC (latest edition) or (2) the IARC Monographs (latest editions) or (3) OSHA's Occupational Safety and Health Standards, Subpart Z – Toxic and Hazardous Substances.
2. EPA's Criteria for the Evaluation of Permit Applications for Ocean Dumping of Materials under the Toxic Substances Control Act (TSCA)
   This regulation prohibits ocean dumping of materials containing "known carcinogens, mutagens, or teratogens or materials suspected to be carcinogens, mutagens, or teratogens by responsible scientific opinion" as other than trace contaminants.

Because both of these regulations apply to all substances listed in the RoC, they are not identified individually in the listing profiles. However, the reader should be aware that these regulations pertain to all substances listed in the RoC, and that their likely outcome is to reduce exposure to listed substances.

Two OSHA regulations identified in some of the listing profiles require clarification:

1. Specific substances are listed as having "comprehensive standards" if, in addition to the permissible exposure limit (PEL), OSHA has regulations for the substance that include provisions for: exposure monitoring, engineering and work practice controls, use of respirators and protective garments and equipment, hygiene facilities, information and training, labeling of substance containers and worker areas in which the substance is used, and health screening programs.
2. The OSHA PEL identified in the profiles for glass wool (respirable size), ceramic fibers (respirable size), and wood dust are based on the standard for Particulates Not Otherwise Regulated (PNOR). This standard sets limits applicable to all inert or nuisance dusts, whether mineral, inorganic, or organic, not identified specifically by substance name. OSHA recommended that the profiles for these three substances include the PEL established by the PNOR standard.

Estimation of Risk Reduction

For each effluent, ambient, or exposure standard established by a Federal agency for a listed substance, the RoC is required to state the extent to which, on the basis of available medical, scientific, or other data, the implementation of that standard decreases the public's risk for cancer. This statement requires quantitative information on how much protection from cancer the public is afforded by established Federal standards.

Estimating the extent to which listing a substance in the RoC protects public health is perhaps the most difficult task in preparing the RoC. The carcinogenic risk (i.e., the probability of developing cancer) depends on many things, including the intensity, route, and duration of exposure to a carcinogen. People may respond differently to similar exposures, depending on their age, sex, nutritional status, overall health, genetics, and many other factors. Only in a few instances can risk for cancer be estimated with complete confidence, and these estimations require studies of long-term human exposures and cancer incidence in restricted environments, which rarely are available.

One possible way to provide quantitative estimates of risk reduction might be to assume that the cancer risk is directly proportional to exposure. This approach also presumes that data exists on past and present exposure levels, or that all workplace conditions comply with regulations. It is rare that one has information supporting these assumptions. Despite these limitations, it is reasonable and prudent to accept that reducing exposure, for any reason, particularly to substances shown to be carcinogenic in experimental animals, will decrease the incidence of cancer in people (Tomatis *et al.* 1997, Montesano *et al.* 2001). This relationship is the basis of current regulatory policies that aim to lower human exposure to cancer-causing substances, and thereby, improve public health.

Major environmental pollution prevention acts, such as the EPA's Resource Conservation and Recovery Act, Clean Water Act and Clean Air Act, were passed in the early 1970s. These laws have lead to the reduction in exposure to a number of substances listed in the RoC. Although one can not draw a direct cause and effect relationship between pollution reduction and cancer incidence, recent data from the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute show decreasing cancer trends for many cancers, although others are increasing (SEER 2003). The "Annual Report to the Nation on the Status of Cancer, 1975-2000" (Wier *et al.* 2003) is based in part on the most recent SEER data and provides an update on cancer mortality (death rates), incidence rates (new cases), and trends in the United States. The report is issued annually by the Centers for Disease Control and Prevention (CDC), the American Cancer Society (ACS), the National Cancer Institute (NCI) of the National Institutes of Health, and the North American Association of Central Cancer Registries (NAACCR). This report indicates that overall, cancer death rates (for men and women combined) were stable from 1998 through 2000 - that is, rates neither increased nor decreased. Before this time, death rates increased through 1990, stabilized through 1994, and declined from 1994 through 1998. Throughout the late 1990s, trends for women stabilized, while death rates for men continued to decline. Lung, colorectal, breast and prostate cancers have the highest prevalence in the United States and account for more than half of all cancer cases:

- Lung cancer is the leading cause of death from cancer in men and women in the United States. Lung cancer death rates among white and black men declined throughout the 1990s, while the rate of increase in deaths among women slowed during the same period, reflecting reductions in tobacco smoking. It is interesting to note that recently published studies have shown a rise in lung cancer and cardiopulmonary disease due to air pollution (Montesano *et al.* 2001).
- Colorectal cancer death rates have been declining for both white and black men and women beginning in the 1970s, with steeper declines beginning in the mid-1980s. This decline is attributed to better screening and treatment methods for this cancer.

- Breast cancer death rates continue to fall despite a gradual, long-term increase in incidence rates. Decreasing rates in deaths from breast cancer and increasing incidence rates during the 1990s have been attributed, in part, to increased use of mammography screening and the availability of improved therapies.
- Prostrate cancer death rates have been declining since 1994, while incidence rates have been rising since 1995, with a 3.0 percent per year increase in incidence in white men and a 2.3 percent per year increase in black men. No currently recognized risk factors account for the decline in prostate cancer mortality, although the decrease might reflect improvements in treatment combined with improved detection using a blood test for prostate specific antigen (PSA).

Cancer sites without significant improvement in survival rates in the past 25 years include the uterine corpus, cervix, larynx, liver, lung, pancreas, stomach, and esophagus (Jemal *et al.* 2004).

Cancer incidence rates for all types of cancer combined increased from the mid-1970s through 1992, declined from 1992 through 1995, and then stabilized (a non-significant increase) from 1995 through 2000. Increases in incidence rates in breast cancer and prostate cancer offset long-term decreases in lung cancer in men (Wier *et al.* 2003). The SEER data also indicate that the incidences of liver, thyroid, melonoma of the skin and kidney cancers increased over the time interval between 1992 ad 2000 (SEER 2003).

Listing Substances in the *Eleventh Report on Carcinogens*

The *Eleventh Report on Carcinogens* contains 246 entries, 17 of which have not appeared in earlier editions of the RoC.

The *Eleventh Report on Carcinogens* lists lead and lead compounds as *reasonably anticipated to be human carcinogens.* This listing of lead and lead compounds supersedes the listings of individual lead compounds (including lead acetate and lead phosphate) in previous editions of the RoC and applies to lead and all lead compounds.

The heterocyclic amines 2-amino-3,4-dimethylimidazo[4,5-*f*]quinoline (MeIQ), 2-amino-3,8-dimethylimazo[4,5-*f*]quinoxaline (MeIQx), and 2-amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine (PhIP), are listed for the first time in the *Eleventh Report on Carcinogens* as *reasonably anticipated to be human carcinogens.* Another heterocyclic amine, 2-amino-3-methylimidazo [4,5-*f*]quinoline (IQ) was listed in the *Tenth Report on Carcinogens,* also as *reasonably anticipated to be a human carcinogen.* These four listings have been grouped together as a family under the title "Selected Heterocyclic Amines." The listing first gives evidence for the carcinogenicity for each heterocyclic amine separately, and then presents a combined section that discusses other information relevant to carcinogenicity, properties, use, production, exposure and regulations.

Three types of ionizing radiation (X-radiation, gamma radiation, and neutrons) are listed as *known to be human carcinogens* for the first time in the *Eleventh Report on Carcinogens.* The radioactive compound thorium dioxide, which decays by emission of alpha particles, was first listed in the *Second Annual Report on Carcinogens* (1981). Radon and its most common isotopic forms (radon-220 and radon-222), which also emit primarily alpha particles, were first listed in the *Seventh Annual Report on Carcinogens* (1994). The profiles for these sources of ionizing radiation have been placed together as a family of profiles under the title "Ionizing Radiation."

Diethanolamine was nominated for possible listing in the *Eleventh Report on Carcinogens,* but after a formal scientific review of all relevant information pertaining to its possible carcinogenicity, was not recommended for listing. The basis for the recommendation not to list diethanolamine is summarized in Appendix C of the *Eleventh Report on Carcinogens.*

Section II lists the names of all the agents, substances, mixtures, or exposure circumstances listed in the *Eleventh Report on Carcinogens.* It has two parts: Section II.A identifies 58 substances as *known to be human carcinogens,* and Section II.B identifies 188 substances as *reasonably anticipated to be human carcinogens.*

Section III, Substance Profiles, contains a brief description of each substance with a summary of the evidence for its carcinogenicity; relevant information on properties, use, production and exposure; and a summary of the regulations and guidelines that are likely to decrease the exposure to the substance. These profiles are in alphabetical order and include references to scientific literature used to support the listings.

The substances listed in the *Eleventh Report on Carcinogens* may constitute only a fraction of actual human carcinogens. The RoC lists only those nominated agents, substances, mixtures or exposure circumstances for which relevant data exist and have been reviewed and found to meet the listing criteria defined above. As additional substances are nominated, they will be considered and reviewed for possible listing in future editions of the RoC.

Certain manufacturing processes, occupations, and exposure circumstances have been considered by IARC and are classified by that agency as known to be carcinogenic to humans because of associated increased incidences of cancer among workers in these settings. However, certain aspects of occupational exposures may differ in different parts of the world or may have changed over time; therefore, the manufacturing processes and occupations reviewed by IARC may not be applicable to past or current occupational exposures in the United States. The NTP has not yet reviewed the data supporting the listing of these occupational situations as posing a cancer hazard. In the interest of public health and for completeness, these occupational exposures are identified in Appendix A of the RoC with the corresponding IARC references.

Other Information Provided in this RoC

Section IV provides tables listing requests to the DHHS for research, testing, and other information relating to carcinogenicity, either from other Federal agencies or from within the DHHS, and how the DHHS responded to the requests. Section V details the listing and delisting procedures for the RoC.

The *Eleventh Report on Carcinogens* also includes seven appendices and an index:

- Appendix A lists manufacturing processes, occupations, and exposure circumstances classified by IARC as known to be carcinogenic to humans.
- Appendix B lists the agents, substances, mixtures, or exposure circumstances that have been delisted from the RoC.
- Appendix C lists the agents, substances, mixtures, or exposure circumstances that have been reviewed but not recommended for listing in the RoC.
- Appendix D lists participants who collaborated in preparing the *Eleventh Report on Carcinogens.*
- Appendices E, F, and G are, respectively, a glossary of terms, a list of acronyms and abbreviations, and a list of units of measurement used frequently in the RoC.
- The index (a feature introduced in the *Eleventh Report on Carcinogens*) allows the user to search for listings by commonly used synonyms or abbreviations included in the profiles or by CAS Registry Numbers of chemical substances discussed in the profiles.

The eleventh edition of the RoC was prepared following procedures that maximized the quality, objectivity, utility and integrity of the information contained in the report. Although not anticipated, factual errors or omissions in this report may be identified after its distribution. If this should happen, these errors or omissions will be addressed by the NTP. Where appropriate, corrections will initially be posted on the RoC web site at http://ntp-server.niehs.nih.gov/NewHomeRoc/AboutRoC.html and then made in the next edition of

the RoC. For more information on the Eleventh Edition of the RoC, including how to order a printed copy or access it on the Internet, visit the NTP RoC web site at the address above or contact Dr. C. W. Jameson, Head, Report on Carcinogens, National Toxicology Program, MD EC-14, P.O. Box 12233, Research Triangle Park, NC 27709; telephone (919) 541-4096; fax (919) 541-0144; e-mail jameson@niehs.nih.gov.

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Websites

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Department of Transportation http://www.dot.gov/

Environmental Protection Agency http://www.epa.gov/

Food and Drug Administration http://www.fda.gov/

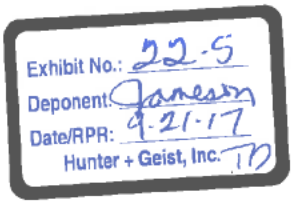
Occupational Safety and Health Administration http://www.osha.gov/

American Conference of Governmental Industrial Hygienists http://www.acgih.org/home.htm

National Institute for Occupational Safety and Health: Pocket Guide to Chemical Hazards http://www.cdc.gov/niosh/homepage.html

Code of Federal Regulations (CFR): http://www.gpoaccess.gov/cfr/index.html

Subject: Re: IARC Monograph vol 112- EFSA Review of Glyphosate
Date: Tuesday, November 10, 2015 at 7:38:53 AM Eastern Standard Time
From: drjameson
To: Chris Portier
CC: [redacted]
Priority: High



Chris,

I would like the opportunity to review and participate in this but am pretty much tied up until Thursday (11/12). I'll try to get something to you before Friday.

Please give Mikie our regards.

Bill

-----Original Message-----
From: Chris Portier <[redacted]>
Date: Monday, November 9, 2015 at 6:05 AM
To: Isabelle Baldi <[redacted]>, Aaron Blair <[redacted]>, "Egeghy, Peter" <[redacted]>, "Forastiere, Francesco" <[redacted]>, Lin Fritschi <[redacted]>, Gloria Jahnke <[redacted]>, Bill Jameson <[redacted]>, "Kromhout, J. (Hans)" <[redacted]>, frank lecurieux <[redacted]>, Matt Martin <[redacted]>, John McLaughlin <[redacted]>, Teresa Rodriguez <[redacted]>, Matthew Ross <[redacted]>, "Rusyn, Ivan" <[redacted]>, Consolato Sergi <[redacted]>, "Mannetje, Andrea" <[redacted]>, Lauren Zeise <[redacted]>
Cc: Kate Guyton <[redacted]>
Subject: IARC Monograph vol 112- EFSA Review of Glyphosate

Dear all,

This week, the European Food Safety Agency (EFSA) will release their reassessment of glyphosate. In this review, they will conclude that glyphosate has no carcinogenic potential. This creates two problems as I see it. The first is that this wekens the strength of the IARC Monograph Program to stimulate change in how some of these agents are reviewed and addressed. The second is that it suggests we did not do our assessment adequately and that, had we seen all of the data they saw, we would have gotten a different answer. I do not intend to let this happen.

The German Federal Institute for Risk Assessment (BfR) was the lead

country agency in drafting the reassessment report. This report was drafted prior to the IARC review. In August of this year, following the release of the full Monograph on glyphosate, the BfR drafted an Addendum to their report that specifically addresses the Monograph review. I have decided to draft a letter that I intend to try to get published in Carcinogenesis that addresses the points made by the BfR in their review. Failing my ability to get this into Carcinogenesis, EHP or some other Journal, I intend to send it as an open letter to the European Commission. I am enclosing both the BfR Addendum and my response for you to look over. I would like as many members of the Working Group to be co-authors on this as possible. If you wish to see changes made to the letter I can certainly work on that. If you are uncomfortable signing on to such a letter, I can appreciate that as in my previous job this would have been impossible. Please let me know by Friday November 13 if you can or cannot join me in this endeavor.

Sincerely,

Christopher Portier

Privileged and Confidential
Attorney Client Privilege
Attorney Work Product

In re Glyphosate/Roundup Litigation

March 29, 2015

Hunter W. Lundy
LUNDY, LUNDY SOILEAU & SOUTH, LLP
501 Broad Street
Lake Charles, LA 70601
Email: hlundy@lundylawllp.com
Telephone: 337 439-0707 / Fax: 337 439-1029

Expert Name
Christopher J. Portier, Ph.D.
Email [redacted]

Dear Dr. Portier:

This will confirm that Hunter W. Lundy, acting on behalf of the law firms of Lundy, Lundy, Soileau and South, LLP and Weitz & Luxenberg, PC ("Attorneys" or "Firms"), has retained you for the sole purpose of consulting with these Attorneys in connection with anticipated litigation involving claims arising from injury or damage caused, or potentially caused, by exposure to Roundup and/or other herbicides containing Glyphosate (the "Engagement"). The terms of the Engagement are as follows:

1. You are hereby engaged to provide expert consultation and analysis in connection with the cases to be filed (the "Roundup Cases"), relating to, without limitation, any area of expertise that you have or possess pertaining to the question of whether Roundup and/or Glyphosate-containing herbicides can cause adverse biological/physiological health effects in humans; relevant mechanisms of injury; any research or scientific studies that you have conducted or participated in conducting; and any other related issues.

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Attorney Work Product

2. All work conducted in connection with this Engagement as a consulting expert and/or a testifying expert witness pursuant to the direction, authority, and/or funding of the referenced Attorneys, including any reports, drafts, data, notes, work papers, correspondence, or other work documents you may generate or receive in connection with the Roundup Cases shall be considered and treated as confidential work product. All such documents and materials (and any information they contain that is not publicly available data or previously available to you) may be used only for purposes of this Engagement and may not be disclosed to anyone without our written consent in advance. This Engagement does not pertain to nor shall it affect your research and/or scientific studies, and it is expressly understood and acknowledged that we have not, nor will we fund, participate, sponsor or be involved in any of your past, present or future research or scientific studies.

3. In recognition of the confidential nature of this Engagement and subject to the terms of paragraph 2, you agree to not discuss or share any of this work, work product, analysis and/or opinions developed or prepared in connection with this Engagement with anyone else including, but not limited to, media organizations, trade journals, professional publications, members of the public, other purported experts, etc., and to notify us promptly if you receive:

   a. Any request to reveal information related to this Engagement or to examine, inspect or copy any documents you generate or receive; or

   b. Any actual or attempted service of a subpoena, summons or order purporting to require the disclosure of any such information or documents; and

   c. In consequence of such requests, subpoena(s), summons or order to require disclosure, the above-named law firm shall provide whatever legal services that are required to Christopher J. Portier without fee, any resultant out-of-pocket expenses, and payment of hourly rate.

Privileged and Confidential
Attorney Client Privilege
Attorney Work Product

4. You have assured us that you do not have any conflict of interest which might interfere with your performance of services contemplated by this Engagement, and you agree to avoid any such conflict during the term of this Engagement. More specifically, it is understood that until this matter is resolved (including any appeals), you will not accept any Roundup and/or Glyphosate-related engagement with any law firm that is a party to Roundup and/or Glyphosate-related litigation without our written consent in advance. However, if written consent is requested by Christopher J. Portier regarding another matter outside the specifics of this litigation, such consent shall not be unreasonably withheld. The request shall list the reasons why consent is requested. Should requested consent be withheld by Firms, they shall supply specific written reasons referencing the specific reasons listed in the written consent request. If Expert and Firms cannot agree, a single arbiter agreed upon by both parties shall decide.

5. Your fee for specific consultation, analysis and any requested report(s) shall be $450.00 (US Dollars) per hour in addition to reimbursement for any out-of-pocket expenses. You shall receive a retainer of $5,000.00 from which charges shall be drawn. You will send a monthly invoice as necessitated by the requested work which identifies the time spent and services rendered. Upon the depletion of the $5,000.00 retainer, payment will be made within 30 days from receipt of your invoice. Bills should be issued to the attention of Hunter W. Lundy at Lundy, Lundy, Soileau & South, LLP, 501 Broad Street, Lake Charles, LA 70601.

6. You will be working under the exclusive direction of Hunter W. Lundy, Matthew E. Lundy and Kristie M. Hightower with the law firm of Lundy, Lundy, Soileau & South, LLP, and Robin L. Greenwald with the law firm of Weitz and Luxenberg, PC.

7. Any and all work product created by you or on your behalf in whole or in part during the course of this Engagement, authorized by the Committee, shall be considered a work for hire and the property of the Firms.

8. You or we may terminate this agreement in writing at any time, in which event

you must stop work and bill only for the work performed up until receipt of the written termination. However, in the event of such termination, the restrictions described in paragraphs 2, 3 and 4 (related to work product generated) above will remain in effect absent a mutual agreement to the contrary. Such mutual agreement shall not be unreasonably withheld.

9. Any controversy, dispute or claim arising out of or relating to this Engagement or breach of this Agreement, shall be decided by a single arbitrator to be mutually selected in a privately administered arbitration to be held in _____, using the rules of the American Arbitration Association. The Firms and you expressly consent to personal jurisdiction in the courts of _____, and waive any objection thereto.

Please acknowledge that you accept these terms by signing the enclosed copy of this letter and returning it to us.

Sincerely,

LUNDY, LUNDY, SOILEAU & SOUTH, LLP

By:_____
Hunter W. Lundy

Agreed to by:

_____
Christopher J. Portier, Ph.D.

Dated: _____

INVOICE

Christopher Portier

Regarding:

Bill to:
Glyphosate/Roundup Litigation
Attn: Hunter W. Lundy
LUNDY, LUNDY SOILEAU & SOUTH, LLP
501 Broad Street
Lake Charles, LA 70601
Email: hlundy@lundylawllp.com
Telephone: 337 439-0707 / Fax: 337 439-1029

Invoice Date: 10/19/2015
Invoice #: 15002

Quantity	Date	Unit	Description	Rate	Amount Due
0.5	6/17/15	hr	Meet with H. Lundy at BIOEM meeting, general issues regarding Glyphosate	$450.00	$225.00
1	6/19/15	hr	Meet with H. Lundy and Robin Greenwald in Davis, CA, general issues regarding Glyphosate	$450.00	$450.00
2	7/9/15	hr	Background research on glyphosate and AML, cancers in the Ag. Health Study and onset time for NHL	$450.00	$900.00
3.5	10/19/15	hr	Reduce value of retainer (balance $5000.00) by cost this invoice (new balance $3425.00)	-$450.00	-$1575.00
				Total	$0.00

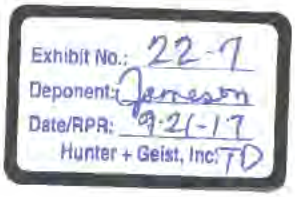
Reimbursement Information:

Name: Christopher Portier

[redacted]

[redacted]

Signature:



005071

From:	Consolato Sergi
To:	Chris Portier
Cc:	Kate Guyton; Ross, Matthew; Egeghy, Peter; Teresa Rodriguez; frank lecuriieux; Kromhout, J. (Hans); Rusyn, Ivan; John McLaughlin; Aaron Blair; Lauren Zeise; Matt Martin; Jahnke, Gloria (NIH/NIEHS) [E]; Isabelle Baldi; Bill Jameson; Mannetje, Andrea; [redacted] Lin Fritschi; Forastiere, Francesco
Subject:	Re: IARC Monograph vol 112- EFSA Review of Glyphosate
Date:	Monday, November 9, 2015 6:24:56 AM

Dear Chris,

Thank you for your email and your wise counteroffensive policy. I will sign the letter, but I would like to read the letter probably today and I will send you my comments by the end of the day.

Thank you again!

Best regards

Consolato

On Nov 9, 2015 4:05 AM, "Chris Portier" <[redacted]> wrote:

Dear all,

This week, the European Food Safety Agency (EFSA) will release their reassessment of glyphosate. In this review, they will conclude that glyphosate has no carcinogenic potential. This creates two problems as I see it. The first is that this wekens the strength of the IARC Monograph Program to stimulate change in how some of these agents are reviewed and addressed. The second is that it suggests we did not do our assessment adequately and that, had we seen all of the data they saw, we would have gotten a different answer. I do not intend to let this happen.

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Sincerely,

Christopher Portier

Subject: Re: Final Glyphosate Letter
Date: Thursday, November 26, 2015 at 6:57:38 AM Eastern Standard Time
From: drjameson
To: Chris Portier

Thanks Chris and Happy Thanksgiving!

Bill

-----Original Message-----
From: Chris Portier <[redacted]>
Date: Thursday, November 26, 2015 at 1:30 AM
To: Chris Portier <[redacted]>
Subject: Final Glyphosate Letter

Dear Colleagues,

Attached is the final version of the Glyphosate letter. I plan to mail it out tomorrow morning. If you have concerns or need something changed, please write back and I will try, but I must have these before 8:00 am CET on Friday, November 25. I want to thank you all for your efforts in drafting this letter.

I will cc all of you when I release the document. It will be going to everyone on the cc line as well as Mr. Andriukaitis. In addition, it will also be circulated to several other groups with an embargo of Monday so that the recipients actually have time to read the letter before being blasted with media inquiries. There is a meeting in Brussels on Tuesday morning that I will attend, but not be speaking. Kurt Straif and Kate Guyton from IARC will be there and will testify. Following this will be a lunchtime debate that I will be participating in where I hope to raise many of the issues that are contained in this letter. I will also let you know of any response I receive from Mr. Andriukaitis or the other recipients, although I doubt we will see a formal response. If any press on this comes my way, I will share that as well.

For those of you who will be co-authors on the Commentary I plan to submit to JCEH, I hope to have that available to you sometime on monday for your review and editing.

Thanks.

C.

Exhibit No.: 22-9
Deponent: Jameson
Date/RPR: 9-21-17
Hunter + Geist, Inc.

003136

From: Chris Portier
To: [redacted]
Subject: Glyphosate
Date: Sunday, December 6, 2015 8:21:23 AM
Attachments: s_2014_2019_plmrep_COMMITTEES_ENVI_DV_2015_12-01_Glyphosate_1_Dec_2015_EFSA_presentation_EN.pdf
ATT00001.htm
s_2014_2019_plmrep_COMMITTEES_ENVI_DV_2015_12-01_IARC_20151201_EN.pdf
ATT00002.htm

I promised to keep you updated on the press etc. These are below. During the EU Parliament discussion of glyphosate, the letter got a lot of attention. The Executive Director of EFSA got quite upset and referred to us as "Facebook" Scientists. He was implying we sign onto a letter just to see how many responses we can get. The debate following the hearing is given below. I mentioned the Facebook comment since the EFSA ED was in the audience. I have received correspondence from the Commissioner asking for a meeting. Nothing is set yet.

C.

Link to the lunch debate in Brussels.

http://www.greens-efa-service.org/medialib/mcinfo/pub/en/scc/4289

Media

http://www.sueddeutsche.de/wirtschaft/streit-um-glyphosat-brisanter-brief-nach-bruessel-1.2759599

http://www.farminguk.com/news/Over-90-scientists-challenge-EFSA-claim-of-glyphosate-safety_37926.html

http://gmwatch.org/news/latest-news/16568-scientists-challenge-efsa-claim-of-glyphosate-safety

http://www.amisdelaterre.org/Glyphosate-et-cancer-la-decision.html

https://news.google.com/news/story?cf=all&hl=de&pz=1&ned=de&q=glyphosat&scoring=d&cf=all&ncl=duZQ_tq1z42TItMUQj7BwnxwIBj_M&start=0

http://www.zeit.de/wissen/umwelt/2015-11/glyphosat-pflanzenschutzmittel-krebs-risiko

http://www.keine-gentechnik.de/nachricht/31426/

http://www.sueddeutsche.de/wirtschaft/streit-um-unkrautvernichtungsmittel-wissenschaftler-protestieren-gegen-glyphosat-bewertung-1.2759599

http://www.dw.com/en/independent-scientists-warn-over-monsanto-pesticide/a-

18886833

http://switchboard.nrdc.org/blogs/jsass/glyphosate_-_iarc_got_it_right.html

BfR

Wie schätzt das BfR den „Offenen Brief" einiger Wissenschaftler an den EU-Kommissar für Gesundheit und Lebensmittelsicherheit ein?

Besagter „Offener Brief" richtet sich an den zuständigen EU-Kommissar, nachdem nunmehr die Risikobewertung durch die in der EU zuständigen wissenschaftlichen Institutionen abgeschlossen und publiziert ist. Eine erste Überprüfung des Schreibens zeigt, dass dort keine neuen wissenschaftlichen Erkenntnisse aufgeführt werden, die nicht bereits von der EFSA und den europäischen Mitgliedstaaten im Rahmen der EU-Wirkstoffprüfung bewertet wurden. Die in dem Brief getroffenen Aussagen zur Kanzerogenität von Glyphosat kann das Bundesinstitut für Risikobewertung (BfR) wissenschaftlich nicht nachvollziehen. Diese Aussagen kontrastieren, wie auch die Schlussfolgerungen des IARC, sämtliche Bewertungen der zuständigen nationalen und internationalen Institutionen einschließlich des WHO/FAO Joint Meeting on Pesticide Residues (JMPR). Die gesundheitliche Bewertung des Pflanzenschutzmittelwirkstoffes Glyphosat ergab nach Prüfung aller vorliegender Studien durch diese Institutionen, dass sich nach der derzeitigen Datenlage bei bestimmungsgemäßer Anwendung von Glyphosat kein krebserzeugendes Risiko für den Menschen ableiten lässt. Zu der Einschätzung kommen auch die amerikanische Umweltbehörde (US-EPA) und die kanadische Behörde (Canada Health). Unterzeichner des offenen Briefes ist nicht die IARC selbst. Der Initiator und Verfasser des Briefes ist nach eigenen Angaben aktives Mitglied des Environmental Defense Fund, einer US- amerikanischen Nichtregierungsorganisation.

Das BfR empfiehlt grundsätzlich, Diskussionen über wissenschaftliche Studien auf wissenschaftlicher Ebene, selbstverständlich auch wenn nötig kontrovers, zu führen. Ein integraler Bestandteil der Wissenschaft ist dabei der wissenschaftliche Publikationsprozess. Thesen oder Kommentare zu Studien können dem wissenschaftlichen Diskurs nur zugeführt werden, wenn diese publiziert wurden und die entsprechenden Schlussfolgerungen transparent nachvollziehbar sind. Da die wissenschaftliche Bewertung des Wirkstoffes Glyphosat durch die zuständige EU-Behörde und die Risikobewertungsbehörden der Mitgliedstaaten abgeschlossen ist, können die zuständigen politischen Gremien in der EU nun auf Basis der wissenschaftlichen Bewertung entscheiden.