SUPERIOR COURT OF CALIFORNIA

COUNTY OF ALAMEDA

BEFORE THE HONORABLE WINIFRED Y. SMITH, JUDGE PRESIDING

DEPARTMENT NUMBER 21

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COORDINATION PROCEEDING SPECIAL TITLE (RULE 3.550) ROUNDUP PRODUCTS CASE THIS TRANSCRIPT RELATES TO: Pilliod, et al. vs. Monsanto Company, et al.	**JCCP No. 4953** **Case No. RG17862702** **Pages 5055 - 5312** **Volume 30**

Reporter's Transcript of Proceedings

Monday, May 6, 2019

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Stenographic Court Reporter

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I N D E X

Monday, May 6, 2019

Monday, May 6, 2019 8:50 a.m.

(The following proceedings were heard out of the presence of the jury:)

MR. MILLER: Just a few small matters. I've been handed a PowerPoint for the next witness, the last witness for the defendants, Dr. Levine.

THE COURT: Uh-huh.

MR. MILLER: Well, we got a detailed report from Dr. Levine. We took Dr. Levine's deposition. Dr. Levine made it clear as a bell in her report and her deposition she's not a -- she's not a geneticist and wasn't going to give any opinions about that. But the PowerPoint that I was provided is very detailed genetic information about how this kind of cell runs into that kind of cell, cells that are not mentioned in her report, not mentioned in her deposition.

So I'm pretty good with her PowerPoint except for that. It's right out of left field. It's nothing that we've ever been able to prepare for, or we would have.

And the other objection is this EPA report, I guess you would call it, that came out last week by -- signed by Billy Mitchell. Apparently, Billy has a bachelor's degree and --

THE COURT: I'm sorry. Who is Billy Mitchell?

MR. MILLER: He works at the EPA.

THE COURT: Is that the subject of the motion that was filed at the end of the week?

MR. MILLER: It is.

THE COURT: Okay.

MR. MILLER: And the report is rifled with violations to the motions in limine. They talk about the EPA standard dose and how --

THE COURT: Well, let me stop you. Because the reason I didn't even address that was because I was waiting for plaintiffs to file an opposition. There's really no way for me to review it and give it any thought.

So are you going to file something, or is this just going to be the subject of an oral motion, and/or is this something that Dr. Levine is going to testify to?

MR. MILLER: We have filed an opposition.

THE COURT: Okay. I just haven't seen it yet.

MR. MILLER: It was filed this morning.

THE COURT: Is Dr. Levine addressing --

MR. ISMAIL: Yes. We did intend to have Dr. Levine address that issue. She has reviewed regulatory documents up until this last filing by EPA, and this obviously updates what she's already reviewed,

consistent with what other witnesses have done as things have come out during trial and commented on it.

THE COURT: Let me just stop you there. I did take a look at it briefly before I left. And my first question was what's the relevance? Because the time period that the plaintiffs used the product was 2017. I sort of get that the science is current, so whatever comes out with respect to what the state of the science is would be considered.

But with respect to the state of what EPA thinks or doesn't think or more importantly -- I'm sorry -- what drove any corporate behavior on Monsanto's part with respect to the plaintiffs, I don't understand why anything coming out yesterday or last week would be relevant to that.

MR. ISMAIL: Your Honor, the relevance is because the plaintiffs, both in opening and through their witnesses, have suggested that the EPA determination is hanging in the balance, so to speak, that it can change at any moment. And, therefore, they should consider the evidence that has come in as being equivocal or just preliminary and not final.

They had Dr. Benbrook make that assertion. Mr. Wisner made that assertion in opening statements. In fact, he even suggested that the outcome here may

have some sort of influence on the EPA's determination.

This here is a response to -- specifically with Dr. Benbrook. Dr. Portier mentioned it is the OPP report that's in evidence. It should be downgraded, I guess, in significance because -- their characterization that it's not final.

We have here reaffirmation of that position by the EPA in 2019 considering public comment that has been made. And, because of that, it is directly responsive to questioning in evidence that the plaintiffs have put in about the earlier report.

MR. MILLER: It's not a final. If it was a final, then maybe we could consider it at some other argument or level. But it's no more than one fella at the EPA saying, "Here are my responses to some of the things that have been raised in the public, and here's what I think about them." But the registration process will continue; the public comment process will continue.

It's nothing final in this document; it's simply something that mysteriously appeared last week. And it says the same stuff that the other two say. So I think the dating issue is correct.

THE COURT: I'm going to just take a break and take a look at it. Tentatively, I would say no. If it's not a final decision by the EPA, then I don't see

the relevance of yet another -- I mean, I understand that the EPA's decision is sort of ongoing. I don't know. It seems to morph. I'm not sure what triggers deciding -- issuing a particular report or making a particular statement. My recollection about the testimony, plaintiffs' witnesses were essentially -- you know, whatever -- garbage in, garbage out kind of thing with respect to the EPA.

So I don't know if it's hanging in the balance so much as, whatever they're doing, is painted -- long story, whatever, the witnesses have said. But there's sort of a litany of that opinion as opposed to let's just wait and see what the EPA says.

MR. EVANS: Your Honor, we've discussed this with you before. But the official act requirement of 452 does not require a final decision. And Your Honor, in addressing the earlier EPA documents, had allowed those in --

THE COURT: I have done that. And, technically, that would be true. But that's -- first of all, it's discretionary. And, second of all, it has to have some relevance. So there are a combination of things, most of which is just exercising my discretion over what I think is appropriate to bring in beyond what may technically meet the requirements of 1280. I would

say it probably does.

MR. EVANS: So, in addition to addressing the -- not only implications but actual statements from plaintiffs' counsel and witnesses regarding that this is somehow the EPA is going to revisit this whole issue. And this is the next affirmation of the prior assessments. And the scientific assessment that's contained within this is evidence of causation, just as much as, for example, the IARC assessment is.

So it's not just responding to them; it's actually affirmative evidence --

THE COURT: No, I disagree with that. I think that a statement of the EPA with regard to glyphosate -- and, as you recall, when he made that argument in the first instance, it was, well, we're entitled to demonstrate what we knew and when we knew it and independent influences of -- among other things, potentially, punitive damages. And it drove perhaps corporate behavior.

I specifically only allowed the statements in because the underlying science was hearsay and not admitted for just that reason, which is it is not analysis of the science. At least I didn't think that the underlying science is admissible because it is hearsay.

So the statement regarding what EPA thinks is, I think, potentially fine under 1280. Whether or not it's otherwise admissible or relevant is another story. But, no, I don't think that the underlying -- put it this way: It's based on all the same science. I mean, all the same studies have been shown. They've been reviewed and rereviewed by a number of bodies.

But, with respect to the underlying science of these other foreign documents and the EPA documents specifically, I eliminated all of that. Because the statement of the EPA and the extent to which Monsanto relied on it, I do think is relevant. But looking at that to assess causation, I disagree that it's admissible for that purpose, which is why I wouldn't admit it.

MR. EVANS: Again, Your Honor, we've had several long discussions about this. If you want to take a look at the briefs again, we can talk about it.

THE COURT: I'll take a peek at it. I haven't seen plaintiffs' brief. The reason I wanted to know if it related to Dr. Levine is because she'll be on this morning talking about this, and I haven't really had a chance to look at the briefs. So let me take a peek before we get the jury in here.

MR. ISMAIL: Mr. Miller said he had additional

objections, and I just wanted to make sure we had a chance to address them before the witness takes the stand.

MR. MILLER: They are PowerPoint Slides 16 through 20 --

THE COURT: I don't have a copy of it.

MR. ISMAIL: I gave mine to Chris.

MR. MILLER: 23.

16 to 23, Your Honor.

THE COURT: Okay.

MR. MILLER: Nowhere in Dr. Levine's report does she talk about macrophages killing TB germs, nowhere in her deposition. Nowhere in her deposition did she talk about natural killer cell recognizing and killing cancer cells. It's simply not in her report.

18, "Dendritic Cell Gives Messages to T Cells." If I had seen that in her report or she had mentioned it in her deposition, I would have researched it, and I would know what a dendritic cell is. But I stand here now two minutes before she takes the stand clueless about what they're talking about. And that was the plan, I'm afraid.

"Cytoxic T8 Cells," not in her report, not in her deposition.

"Genetically Engineered CAR-T Cells Attacking

Cancer Cells," did not talk about it.

"Rituximab Immunotherapy" attacking or attaching to a cancer cell through CD20, that -- just wasn't in her report. And it's not in her deposition.

And that ends up with 23 -- at page 23, these are just all new subjects that she didn't put in her report, didn't put in her deposition.

MR. ISMAIL: She produced these slides at her deposition. They had ample notice. We produced these at her deposition, same pictures.

THE COURT: Maybe you guys need to talk first.

MR. MILLER: Yeah.

THE COURT: Why don't you have that conversation.

MR. MILLER: We'll have that conversation.

Your Honor, one last thing, unrelated minor request. I've had permission from defense counsel. My wife, who is an attorney who has not been admitted in this case, would like permission to sit at counsel table for one witness. She's organized the binder. She won't speak, just hand me documents on cross-examination.

MR. ISMAIL: No objection.

THE COURT: Okay.

MR. MILLER: We'll talk to counsel. If they were there, then I owe them an apology. But I wasn't

there; Curtis was. But we'll talk.

THE COURT: Okay.

(Recess taken from 9:01 a.m. to 9:11 a.m.)

MR. MILLER: I've spoken to Mr. Ismail. We were not provided those. However, apparently, she brought a big pile of documents with her. They were in that pile of documents. They never got looked at or reviewed. That's okay. I accept her at her word.

What we've agreed to do to keep this moving is those exhibits that we referenced, they don't magically relate somehow to Mr. Pilliod; he's using them in the general sense and not specific as to Mr. Pilliod, and that's fine.

THE COURT: Okay.

MR. ISMAIL: The pictures, Your Honor, are how the immune system interacts with cancer surveillance and suppression. She's going to describe it in the general sense. Obviously, her opinion is Mr. Pilliod does have a compromised immune system. She's not going to put up a picture and say "This is his T-cell" or "I think this is what his T-cell looked like." It's demonstrative of the system. And I think we have an accord. And they were provided.

THE COURT: Thank you. I have had a chance to take a look at this. And I am going to exclude it for a

couple of reasons.

First of all, this isn't really a decision; this is really a comment on comments. And, to the extent there's a conclusion, it's, well, the comments haven't really changed our mind yet, so the process is going to keep going.

Correct me if I'm wrong.

So, to the extent that it doesn't really represent a decision of EPA in the sense of what I think requires compliance with 1280, that's one reason. The other is the nature of this really does focus on comments which comment on the analysis, the scientific analysis.

To introduce this now, I would have to allow the plaintiffs to come back and rebut this in some fashion. It's actually more open-ended than the documents I did allow in because they were sort of closed-end decisions based on a specific analysis.

This goes through and says, well, human incidence in epidemiology, and then they talk about the literature. They go through. And, at the end of the day, they don't really draw any conclusions. They have lots of subsections, which they're sort of regurgitating a lot of science.

But, at the end of the day, it doesn't really

come to a conclusion about anything other than we're going to still continue to look at this. So I don't believe this really even qualifies under 1280.

I also think exercising my discretion at this point, after the close of plaintiffs' case, allowing this document to come in, which I don't think represents a decision that falls within 1280 of the evidence code, would be unduly prejudicial.

And then I have to let the plaintiffs come in and rebut this in some fashion. I just don't think, at this stage in this litigation, it's going to add to the body of evidence that the jury is going to consider.

I mean, the current decision -- official decision is glyphosate doesn't cause cancer. The decision is glyphosate doesn't cause cancer.

So -- I mean, to the extent that that's the EPA's position, it's still the EPA's position. So, anyway, the request by defendant is denied.

So let me get the jury in.

(Recess taken from 9:14 a.m. to 9:20 a.m.)

(The following proceedings were heard in the presence of the jury:)

THE COURT: Good morning, ladies and gentlemen. We're ready to keep going this morning, and Mr. Ismail will present the defendants' next witness.

MR. ISMAIL: Thank you, Your Honor.

Good morning, everyone.

Defense calls Dr. Alexandra Levine to the stand as the final witness in the trial.

ALEXANDRA LEVINE,

called as a witness for the Defendant, having been duly sworn, testified as follows:

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

THE WITNESS: My name is Alexandra Levine; A-L-E-X-A-N-D-R-A, last name L-E-V-I-N-E.

THE COURT: So, Dr. Levine, you're going to have to speak way up for this crowd.

THE WITNESS: Will do.

THE COURT: Into the mic, because the reporter has to take down everything, and we want the jurors to hear everything you're saying.

THE WITNESS: Will do. Thank you.

MR. ISMAIL: May I proceed, Your Honor?

THE COURT: Yes, you may.

MR. ISMAIL: May I approach with a binder for the witness?

THE COURT: Yes.

DIRECT EXAMINATION

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BY MR. ISMAIL:

Q. I am handing you a binder of some exhibits we're going to go through today.

I've provided a copy to counsel, and Your Honor has a copy as well.

Good morning, Doctor.

A. Good morning.

Q. Can you please introduce yourself to the ladies and gentlemen of the jury and tell the folks what you do for a living.

A. My name is Alexandra Levine. I am an oncologist. That means that I take care of patients who have cancer. I work at the City of Hope National Cancer Center. That is in Duarte outside of Los Angeles. It is a comprehensive cancer center that is supported by the National Cancer Institute.

Q. And, Dr. Levine, do you have a particular area of focus within the field of oncology?

A. Yes. My specialty is non-Hodgkin's lymphoma and Hodgkin's lymphoma as well.

Q. And, Doctor, have we asked you to look at the medical records with respect to Mr. Pilliod and some scientific information regarding Roundup to form an opinion as to whether or not Roundup played any role in his development of non-Hodgkin's lymphoma?

A. Yes, you did.

Q. And are you prepared today to discuss with the jury your opinions and your findings on those questions?

A. I am.

Q. And before we get to your opinions, Doctor, I would like to give folks a little bit better sense of your professional experience and background.

And I notice, Doctor, you have a CV that is over a hundred pages long. And rather than walking through the CV, have you helped us put together some slides to summarize your professional experience?

A. Yes, I did.

Q. So they're in the binder there in the front, but I'll also show them on the screen here for you.

Can you please tell us -- let's start with your education and early professional medical training.

A. First, undergraduate, I went to UC Berkeley, right close to here.

After that, I went to University of Southern California, USC, for medical school.

After medical school, you are an intern. You're still being trained as an intern and then what's called a resident. So I was an intern and a resident in internal medicine at the big Los Angeles County USC Medical Center, the big county hospital for the County

of Los Angeles.

After that, if you want to specialize more within the field of medicine, you take a fellowship. And that allows, again, more sophisticated training.

I became a fellow in hematology and oncology at Emory University in Atlanta, in Georgia. And, after that, I came back to Los Angeles, came back to the big county hospital. And I was a fellow in clinical research in hematology, specifically cancers of the blood system. And that was at the county hospital again.

Q. So, Doctor, what sparked your interest in oncology?

A. It's a long question -- a long answer, I guess. Basically, I've always been challenged by people, patients who have complicated illnesses who are a challenge. It's something that is interesting to me, the challenge of significant illness.

The second thing I learned earlier in my training was that, if somebody was really ill, somebody with cancer, as an example, and you had the time or wanted to take the time just to sit at the bedside and hear, you could hear all of life. You could hear what was important, what isn't important.

And so the human aspect was very moving to me,

and the challenge of difficult disease was moving to me. And I think those were the two main things that got me into oncology.

Q. And did you begin -- at that point in your fellowship back at Emory and probably even your earlier training, beginning caring for patients with cancer in the manner that you just described?

A. Yes. So I -- the county hospital is big. It was 2,000 beds at that time. I had a tremendous amount of experience with cancer patients even before I took the fellowship. That's what told me that I wanted to go in that direction.

Q. So let's talk about your clinical experience, Doctor.

After your fellowship, did you continue your practice in caring for patients with cancer?

A. Yes, I did.

Q. Can you walk us through the clinical positions you've held and your experience at those hospitals?

A. First of all, at the county hospital, I became the clinical director of the adult hematologic neoplasia service. What that means is I was in charge of the care of the patients, all of the patients who had cancers of the blood system: leukemias, lymphoma, multiple myeloma, diseases such as that.

And that was many years. That was from 1997 until I left USC at the end of 2006.

In time, I became the interim chief of the division of medical oncology. That really was not my field. They were recruiting for a director, and I agreed to be the interim for that year.

What I really did, from 1991 through 2006, I became chief of the division of hematology. I was in charge of the whole area of hematology at USC.

Then -- in 1983, USC opened their first private hospital, a cancer hospital, USC Norris Cancer Hospital. And at first I was the deputy clinical director of the cancer center in general, and then I became the medical director of USC Norris Cancer Hospital. And I did that from '96 to 2006, when I left USC.

I left at the end of 2006 and I went to City of Hope. I was asked to be the chief medical officer at City of Hope. And at the end of 2016, I -- at 12/31/16, I retired from the administrative positions, could never really retire from being a doctor. So I continue to take care of my patients, even though I'm no longer the medical director of the cancer center there at the hospital there.

Q. So as you've described your clinical

experience here on this slide from 1977 to the present, throughout that entire time, did you continue your clinical practice caring for patients with cancer?

A. I really couldn't do what I do if I wasn't a doctor. And, as an example, when I was chief of the division of hematology, I was in charge of making the schedules for all the doctors. The hardest schedule would be to be an attending physician at the county hospital. And I specifically made myself the attending for six months out of the year. I love taking care of patients. I've always taken care of patients. And I still take care of patients.

Q. That was going to be my next question. Even though you have stepped down as the chief medical officer at City of Hope, do you continue to this day being a clinical physician in oncology?

A. Absolutely. The last time I got a call from a patient was last night.

Q. And when are you next going to be in clinic or in the hospital taking care of patients?

A. Tomorrow, my first appointment at 7:00 a.m.

Q. And throughout this time, Doctor, this 40-plus-year career you've had as an oncologist, what has been your area of clinical specialty?

A. My clinical specialty is non-Hodgkin's

lymphoma. That's what I do.

Q. And this is probably a difficult question to answer, but, as you look back over your four-plus decades as a clinician, can you give us a sense of the number of parents with non-Hodgkin's lymphoma that you have seen and cared for?

A. I don't know exactly. It is thousands. It is literally thousands, starting from the county hospital, being ultimately in charge of all of those patients who had non-Hodgkin's lymphoma. The interns, the residents, the fellows, the other attendings would come to me. I knew those patients over time.

So it is literally tens of thousands of patients.

Q. Now, the jury has had an opportunity to meet another physician at City of Hope named Dennis Weisenburger.

Do you know Dr. Weisenburger?

A. I certainly do.

Q. How do you know Dr. Weisenburger?

A. I know him as a colleague, as a pathologist. I know him also because I was in charge of recruiting all the physicians, the quality of their work and so forth, and I recruited Dr. Weisenburger to be chief of pathology at City of Hope.

Q. So you, in essence, hired Dr. Weisenburger?

A. I certainly did.

Q. And as chief medical officer at City of Hope, were you responsible for all the physicians at that hospital?

A. Yes. The physicians reported to me. I was responsible for the clinical care that they delivered. I was responsible for the quality of their care. I was responsible for the research that they did. I was responsible for assuring that they were abiding by all the rules and regulation and so forth.

Q. Do you respect Dr. Weisenburger as a pathologist?

A. I absolutely respect Dr. Weisenburger as a pathologist.

Q. Now, at City of Hope do the pathologists become involved in diagnosing the cause -- assessing the clinical risk factors of a patient and diagnosing the cause of those patients' non-Hodgkin's lymphoma?

A. No. It is not the role of the pathologist to diagnose a cause of an illness. The role of the pathologist is to diagnose the illness in the first place by looking under the microscope, by looking at laboratory tests and so forth, and diagnosing what that patient has. That's what a pathologist is supposed to

do and does.

Q. Is that true for Dr. Weisenburger as well in your experience --

A. Yes, it is. And that's why he was hired.

Q. And at City of Hope, what is the group of physicians who do became involved in assessing a patient's clinical presentation and trying to understand what's going on with their cancer?

A. That would be the oncologist. That would be the clinician who talks to the patient, who understands what the patient's history has been, medical history and so forth, and tries to put it all together in a way that will be helpful.

Q. And is that an area in which you've worked in for the nearly last 50 years?

A. Yes.

Q. Do you also have roles as a teacher?

A. Yes, absolutely.

Q. Can you walk us through your teaching positions and the areas in which you were instructing young medical students and doctors?

A. Yes. First of all, I love to teach. I always teach. As it turns out, the word "doctor" comes from the Latin which means teacher. So that's part of my job, and it's always been part of my job to me,

important to me.

At USC, first I was an assistant processor and then an associate and then a professor. And in 1977 I was designated as a distinguished professor of medicine. That's an honorary kind of degree.

Right now, since I've left USC, I'm called a distinguished professor emeritus. In 2010 I also became an adjunct processor at Claremont Graduate University. That means I'm not fully employed there, but I teach there and work in some sense there.

In 2007, when I started at City of Hope, I became a professor of hematology, oncology, and hematopoietic cell transplantation. That means like bone marrow transplant.

Then in 2012 I was given another honorary degree or honorary title, Melinda and Norman Payson Professor of Medicine.

So those are the titles that speak to the fact that I have been a teacher over those many years.

Q. And who is it that you teach?

A. I teach everybody. I teach the community. I teach patients. I teach the families of patients. I teach undergrad students. I teach medical students extensively. I teach interns, residents, fellows. I teach other physicians quite regularly, actually, in

community settings or in big national or international conferences. I teach a lot.

Q. And has your teaching experience also focused in the area of lymphomas and blood-related cancers?

A. Yes, it has.

Q. Tomorrow morning, when you are seeing your patients, will you have residents or fellows with you?

A. Usually I'll have a fellow with me. I don't know exactly who's assigned tomorrow.

Q. In addition to your clinical experience you've described, your teaching experience you've just described, have you also been involved in research?

A. Yes, I have.

Q. Can you tell us what has been your focus in your areas of research?

A. My general focus in research is non-Hodgkin's lymphoma. As time went on, I became involved in the area of what causes lymphoma in terms of various germs, various viruses and organisms.

Q. Have you published your work in the peer-reviewed medical literature?

A. Yes, I have.

Q. Let's just break this down.

How many peer-reviewed articles have you published in the literature?

A. About 325, 326.

Q. And have you also published -- contributed book chapters to textbooks in medicine?

A. Yes, I have.

Q. Can you give us a sense of that order of magnitude?

A. 70 of those. I get requests to do that. It takes a long time to write a chapter, and at a certain point in my career, I decided that I would only write one chapter a year. So that was limited to 70.

Q. Have you also published abstracts in medical journals?

A. Multiple, multiple abstracts in medical journals, yes.

Q. Can you give us a sense of the order of magnitude of that contribution?

A. At a certain point, I didn't even put them on my CV. There were too many of them. Every paper I wrote would have started with abstracts, beginning of the information, beginning to understand, publishing those preliminary data and then eventually the whole publication. I will say it's probably in the order of 900 or 1,000, perhaps, abstracts.

Q. So, in total, looking at how published is Dr. Levine in the medical literature, can you give us a

sense of peer-reviewed journal articles, abstracts, book chapters?

A. Well over a thousand publications, peer-reviewed.

Q. Have you -- has your work publications appeared in some well-known medical journals like the New England Journal of Medicine and Journal of American Medical Association?

A. Yes, they have.

Q. Have you served as an editor for any of the journals the jury may have heard about during this trial?

A. Yes, I have.

Q. For example?

A. Oncology.

Q. The jury has heard about the peer-reviewed process and how articles are sent out for comment by experts in the field.

Have you had an opportunity to do that as well?

A. Yes. I do that very, very regularly. The last paper I was asked to review was last week. So many, many, many. Hundreds of thousands, I would think -- not hundreds of thousands, but perhaps a thousand of those reviews over 40 years.

Q. Have you -- I think you mentioned as well that you have presented on your areas of expertise to other physicians?

A. That's correct.

Q. And you've been asked to speak at conferences about non-Hodgkin's lymphoma?

A. Yes, conferences both nationally and internationally.

Q. In your speaking experience, has the topic of pesticides come up? Speaking generally.

A. No.

Q. Have you ever told a group of physicians that Roundup causes non-Hodgkin's lymphoma?

A. No, I have not.

Q. Why not?

A. I believe that the majority of the data are clear in terms of the fact that Roundup does not cause lymphoma.

Q. Now, Dr. Levine, your CV has many awards listed on it.

I'd like to ask you, of all the awards that are listed on your CV, which are the most meaningful and significant to you?

A. I guess, first related to teaching, the Outstanding Clinical Professor Award. So there aren't

nominations; the students just pick whatever name they want to pick and vote. And at a certain point, the school made a decision that I was not allowed to receive this more than every five years. But then, when I was going to leave, they allowed that to occur. And that was important to me because teaching is important to me.

I was chosen by President Clinton to be a member of his advisory committee on HIV/AIDS and was chosen to be the chairman of the research committee, of that AIDS advisory committee. That was important to me.

I was elected a master of the American College of Physicians. And that was important to me because it spoke to my care of patients and the full -- what I did in my career.

And I guess the most recent one was in March of this year, the Margaret Kripke. It was called the Legend Award for Promotion of Women in Cancer Medicine and Cancer Science. And that was important for me because I really do think it's important for women to be involved in these fields.

In a certain kind of a way, I guess everyone believes that nurses should be women because they are kind and compassionate. Well, women should be doctors too, and women should be scientists too. So that was important to me that I was recognized for doing that.

Q. In addition to your work and your speaking, have you also been asked to consult with health ministries outside the United States?

A. Yes, I have.

Q. And we're showing here some of that work that you have done?

A. Yes.

Q. Can you describe in general what that work is and what you did in these points?

A. Early in the AIDS epidemic, I began to see young men with very, very unusual non-Hodgkin's lymphomas. And that was the beginning of my interest in AIDS-related lymphomas. I eventually defined that these lymphomas were going to be part of the AIDS epidemic, and they were.

And as I got involved in the malignancies and the cancer related to AIDS, you couldn't just get involved in that; you had to look at the whole big epidemic. And I became involved in the big epidemic. And that meant that it was important to me to do whatever else I could do to try to help any other country, any other group to prevent that disease from getting into that country in the first place.

So I very much was wanting to do these consultantships in the various countries when I was

asked to do so. I went to Mexico. I went to Chile. I was asked to speak to the health minister in India. I went to Russia. I went several times to China. That was very interesting to me because I believed that, if there was any one country in the world that might have had the capability of stopping that epidemic from occurring, it would have been China. But I failed. It didn't work. I tried.

Q. And, Doctor, how is it or why is it that a lymphoma specialist and cancer researcher, such as you, became so involved in understanding the AIDS epidemic and working on that as a public health issue?

A. Well, it started with my patient care. I saw patients, and it just took one or two to realize that I was seeing lymphomas that were very, very different than any of the cases I had seen before.

That led me, after two or three cases, to look at population-based data. Was that just me at the county hospital seeing something funny, or was this really happening on a larger scale?

And I worked with some of the epidemiologists at USC. Leslie Bernstein, for example, was in charge of the SEER registry, which is a population-based cancer registry. If any doctor diagnoses cancer in my practice, it turns out that's a reportable disease to

the government. The government needs to track cases of cancer. And you can see in a quick look whether there are changes. Is something happening to the incidence of one kind of cancer or another?

So I looked at the SEER data, I looked at what the CDC was also discussing, and realized that it wasn't just my hospital; this was happening.

I started -- that was my beginning. So starting with my own patients, realizing something different, going to a larger dataset to see it wasn't just me, and at that point trying to figure out what was this? What was the emerging epidemic of a very, very unusual lymphoma?

And, again, as I said, it was very difficult. It was such a difficult epidemic, such a difficult time in the history of these people and in my life also. You couldn't just be involved in a little section of lymphoma and not get involved in the entire epidemic, what was causing all of these infections and so forth.

Q. Did this emerging issue, as you saw the first -- the leading edge of it with respect to lymphoma, did that even have a name at the time?

A. No. At first it had no name at all. Eventually it was called GRID, gay-related immune deficiency. And it turned out that it had nothing

really to do with gay; it was just a sexually transmitted disease or by blood to blood.

Eventually the name was changed to AIDS or became AIDS, acquired immune deficiency disease. In time it was found to be caused by a virus, and that virus was called the human immunodeficiency virus, HIV.

I actually wrote a letter to several people at the NIH and CDC suggesting that the name of this virus really should be HIV because, first of all, human, the only animal, if you will, that is infected by this virus is people, is humans. Immunodeficiency. Immune is our immune system, our defense system. This is a virus that causes deficiency of the immune system in humans. And I thought that HIV was probably a pretty good name for it.

I wrote the letter, and subsequent to that, I got a note from the Library of Congress asking if they could put that letter into the Library of Congress, and I said yes.

Q. Doctor, we're going to talk today with the jury about the role of the immune system in cancer surveillance and cancer protection; is that correct?

A. Yes.

Q. Is that issue of the immune system how the HIV and AIDS conditions affect the risk of a patient developing lymphoma?

A. Yes. It turns out that our defense system, our immune system, is responsible for recognizing things that are foreign to us.

So, first of all, germs. It's your immune system that will recognize that you have an infection, see that it's foreign to you, and try to kill it for you. And usually it will work in one way or another.

But it turns out that a cancer cell is also foreign to you. There's been a change in that cell so that it doesn't look like self anymore, doesn't look like me anymore. The immune system should be able to recognize that, that that cell is foreign, and destroy it.

And that's why people who have deficiencies or abnormalities of the immune system are so at risk for these various cancers.

Q. Doctor, is it fair to say that you have over 40 years of experience assessing patients and recognizing whether or not their immune system puts them at risk of developing non-Hodgkin's lymphoma?

A. Yes, I have.

Q. Doctor, are you being compensated for your time?

A. Yes, I am.

Q. At what rate?

A. $500 an hour.

Q. In terms of the materials that you reviewed to arrive at your opinions you're going to talk about today, first with respect to Mr. Pilliod, did you receive and review the medical records that were provided?

MR. MILLER: Excuse me, Counselor. Are you done with voir dire?

MR. ISMAIL: No. I'm establishing her materials that she reviewed. I will tender the witness.

MR. MILLER: Okay.

MR. ISMAIL: May I continue, Your Honor?

THE COURT: Yes.

BY MR. ISMAIL:

Q. So, Doctor, my question, just so you have it in mind, as part of your review in this case, were you provided and did you review Mr. Pilliod's medical records?

A. Yes, I did. They came at various time intervals. And I looked at them at various time intervals when I received them.

Q. Did you also look at his testing results as they were contained in his medical records?

A. Yes, I did.

Q. Did you review the depositions of Mr. Pilliod

and that of Mrs. Pilliod?

A. Yes, I did.

Q. Did you review the depositions of physicians for Mr. Pilliod?

A. I'm not sure I got all of them, but I think I did, yes.

Q. And did you review the reports and depositions of the experts that the plaintiffs have --

A. Yes, I did.

Q. -- tendered?

A. Yes, I did.

Q. Did you also review the scientific literature and other information regarding glyphosate-based products like Roundup?

A. I did.

Q. Did you also review some of the regulatory organization reviews around the issue of whether products like Roundup are related in any way to non-Hodgkin's lymphoma?

A. Yes, I did.

Q. Did you review the IARC monograph as part of your review?

A. Yes, I did.

Q. Did you also, Doctor, rely on your education, training, and experience that you described for the jury

this morning in arriving at your opinions?

A. I did.

Q. Did you have an opportunity to speak with Mr. Pilliod directly?

A. No, I didn't.

Q. And do you believe that in any way limits your ability to comment about his medical history and his non-Hodgkin's lymphoma?

A. No, I don't think that at all. I was able to go through his deposition carefully, and I certainly take him at his word with everything he said there.

Q. And did his medical records provide you the information you needed about his diagnoses and other related conditions over the years to form your opinions in this case?

A. It certainly did.

Q. Doctor, all the opinions you're going to offer today will be to a reasonable degree of medical certainty?

A. Yes.

Q. And with respect to the opinions you have formed and that you will share with us today, did you apply the same standards as you would with your own patients, your own students, your own colleagues when you are a practicing oncologist and researcher?

A. Yes, I did.

Q. Doctor, are you familiar with the term "evidence-based medicine"?

A. Yes.

Q. What is that term?

A. It means that, when we are making decisions related to people, related to patients -- treatment decisions, prognosis decisions -- we can't just think that we -- I think I want to do this; I think I want to do that. It has to be based on evidence. Somebody's life is going to depend on you. And you need real data, scientifically valid data.

And that's what "evidence-based" means. It's not something I just think; it's something I know because the science is there behind it.

Q. And do you teach and practice the principles of evidence-based medicine in your roles that you described for us?

A. Without question. That is the basis of medical education. It is based upon scientific fact. Has to be.

Q. And have you followed those concepts of evidence-based medicine in your review and forming your opinions here?

A. Yes, I have.

MR. ISMAIL: Your Honor, at this time I would tender Dr. Levine in as an expert in lymphoma, its diagnosis, treatment, causes generally, and Mr. Pilliod specifically.

THE COURT: Voir dire.

MR. MILLER: Yes, Your Honor. Thank you.

VOIR DIRE EXAMINATION

BY MR. MILLER:

Q. Thank you. Good morning, Doctor.

A. Good morning.

Q. I'm Michael Miller. We haven't met.

A. Hello.

Q. Hi. This is my wife and law partner, Nancy Miller. She's going to help me a little bit. Okay?

A. Sure.

Q. You're the last witness, and we're going to be done. And I just want to ask you a few follow-up.

Now, first of all, I want to thank you for everybody who has HIV or AIDS for your remarkable work in that area.

A. Thank you for saying that.

Q. That's serious.

You agree that Al Pilliod does not have HIV or AIDS, right?

A. Mr. Pilliod does not have HIV and does not

have AIDS. You're right.

Q. So you and I agree that the patients you see generally have a known cause for their non-Hodgkin's lymphoma. Isn't that true?

A. No, I would disagree with that.

Q. Do you remember us taking your deposition in March, ma'am?

MR. ISMAIL: Your Honor, is this going beyond the qualifications at this point?

THE COURT: Let's talk about qualifications first. You may want to do that portion on cross-examination.

MR. MILLER: All right.

BY MR. MILLER:

Q. Have you testified for Monsanto before?

A. Yes, I have.

Q. And when you testified for Monsanto, I believe March 11th in San Francisco, right?

A. I'd have to look at the document to assure you of the date.

Q. But in March, whatever the exact date, do you remember saying that most of your patients had a known cause of NHL, either HIV, Hepatitis B, or Hepatitis C?

A. If you'll show me the document, I can confirm what I said.

Q. That's fair. And I think the Court wants me to do that after, when I start cross-examination.

THE COURT: Just voir dire on qualifications.

MR. MILLER: I will move on.

BY MR. MILLER:

Q. Now, there are scientists in the world of non-Hodgkin's lymphoma who research the issue of pesticides and non-Hodgkin's lymphoma. That's fair, isn't it?

A. Yes, it is.

Q. And in your 48 years, you have never researched the relationship between pesticides and non-Hodgkin's lymphoma.

A. That's --

Q. Right, ma'am?

A. That is correct.

Q. I didn't want to interrupt you.

So in 48 years, you've never published anything on the relationship between pesticides and non-Hodgkin's lymphoma. That's also true.

A. That is correct.

Q. All right. So you've never done any bench work on the issue of Roundup and non-Hodgkin's lymphoma?

A. That is correct.

Q. And you never published anything on the issue

of Roundup and non-Hodgkin's lymphoma?

A. That's correct.

Q. Okay. You mentioned Dr. Weisenburger is an excellent physician there at the same hospital, City of Hope.

A. I mentioned he was an excellent pathologist -- and he is -- at the City of Hope.

Q. And I think he thinks highly of you as well. It's interesting. We have two very good physicians, both at City of Hope hospital, right?

A. Correct.

Q. Who have come to, frankly, different conclusions on whether or not Roundup was a substantial contributing factor in causing Mr. Pilliod's non-Hodgkin's lymphoma?

A. We have different opinions, correct.

Q. Yes. And I think it's good for the whole country. We can disagree without being disagreeable, can't we?

A. That's nice. Yes.

Q. In fairness, between the two of you, if the jury was like, hmm, which one has more experience with pesticides and its relationship to non-Hodgkin's lymphoma, the truth is it would be Dr. Weisenburger?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Counsel, let's just stick with qualifications to render any opinion, and then we'll talk later about whether or not you want to challenge the opinion and on what basis.

MR. MILLER: All right. Very well.

BY MR. MILLER:

Q. But just to close up that topic, then, you do not consider yourself an expert in the relationship of pesticides and non-Hodgkin's lymphoma, having not researched it for the last 48 years?

A. I do not consider myself an expert on pesticides. On the other hand, I've read a great deal of scientific data about pesticides and the relationship to lymphoma.

Q. Since Monsanto hired you?

A. Since Monsanto hired me and a bit before as well.

Q. Can you name one article that you read before?

A. I would have to think and go back into my list of articles -- or my files. I can't list you an article right now.

Q. All right. You're not a toxicologist, right?

A. I'm not a toxicologist.

Q. And there is, within the field of medicine, something called infectious disease specialists, right?

A. That is true.

Q. And they can get boarded in that specialty, right?

A. That's true.

Q. And you're not a boarded specialist in infectious disease, right?

A. I'm not a boarded specialist in infectious disease, but I am deeply experienced in the care of patients with all kinds of infections. Patients with cancer have an increased risk of infections, all kinds of regular infections, all kinds of what is called opportunistic infections, such as those that occur in HIV.

So I am not board-certified in infectious disease; I know a good deal and have had a massive experience in dealing with patients who have all kinds of infectious diseases.

Q. And that's fair. HIV and AIDS is an infectious process, right?

A. Yes, it is.

Q. And you've spent a lot of your life studying those areas, right?

A. I did. And one of the things that the AIDS patients get are all of these various infections, which come to my attention because I have to take care of

patients who have them.

Q. And you're not an epidemiologist, right?

A. No, I'm not an epidemiologist, but I have spent a tremendous amount of my scientific time looking at epidemiology. My colleagues have been epidemiologists. My papers have dealt with epidemiology of lymphoma, what causes lymphoma, based initially on data, on population base, what is happening epidemiologically.

So I'm not an expert, but I have spent 40 years in dealing with epidemiology and how it relates to non-Hodgkin's lymphoma.

Q. The last time you testified, you didn't want to testify about epidemiology for Monsanto, right?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Sustained.

MR. MILLER: All right. We'll go back to that later.

BY MR. MILLER:

Q. Now, you have, you told us, about 325 publications on your CV?

A. Correct.

Q. About 200 of them deal with AIDS, right?

A. I'd have to count them. Many of them do.

Q. And both of your books deal with AIDS-related

cancers?

A. They do.

Q. Okay. Of your 12 internet publications on your CV, we counted 11 of them dealt with AIDS and HIV.

A. I'd have to count them to assure that that is true, but I will accept that.

Q. And I think we can probably agree, Doctor, skin cancer, for the skin, there is a specialty called dermatology?

A. That is correct.

Q. And you don't hold yourself out as a dermatologist?

A. No, I don't, but patients with cancer, patients with HIV as well, develop all kinds of problems with their skin. I am well versed in dealing with problems with the skin and cancers of the skin.

Q. And I looked at your CV last night. Of your 325 articles in your CV, none of them dealt with skin cancer, right?

A. That is correct.

Q. Not an area of specialty of yours?

A. It is not my specialty; it is non-Hodgkin's lymphoma.

Q. You had 249 book chapters, which is, again, very impressive. 170 of them dealt with AIDS and HIV,

right?

A. I have 70 book chapters.

Q. Oh, excuse me. All right. And how many of them deal with AIDS and HIV?

A. I'd have to count. I just don't know. If you want to give me the CV, I can count them for you.

Q. Well, how about this. How about we just agree that a significant number of them deal with HIV and AIDS?

A. That's probably true.

Q. Okay. And you were first retained by Monsanto when, ma'am?

A. It was around November -- October or November of 2018.

Q. You wrote your first report for Monsanto on November 26th. Does that ring a bell?

A. I would have to look at the date, but I will accept that for the moment.

Q. It was not this case, another case --

A. Yes.

Q. So you must have been working for them for a few months before then.

A. I'm not sure. I need the dates, and then I can tell you exactly when I started. And I can go into my book and my calendar as well.

Q. And you wrote a report in that case, and we'll talk about that more later. And then you've done this case.

Do you have a third case coming up soon?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Sustained.

MR. MILLER: We'll talk about it later.

THE COURT: Qualifications.

MR. MILLER: Well, we talked about money on the direct. I was just going to --

BY MR. MILLER:

Q. So you have 500 an hour on this case and 500 an hour on the other case as well?

A. That's correct.

Q. Do you want to tell us how much you've earned so far for the two cases?

A. I don't know about this case. The other case, it was probably about $80,000.

Q. Okay. And that money goes to you; it doesn't go to City of Hope, right?

A. That is correct.

Q. Okay. I think you told us you semi-retired at the end of 2016?

A. I retired from my administrative jobs but continued my clinical work.

Q. Now, when you were first hired and went to work for Monsanto, as late as March, when you testified in that first proceeding, you didn't know Roundup was a pesticide.

Do you remember that?

A. Do you want to show me what you were referring to?

Q. Sure. 1654 from Hardeman. And we have copies if you want.

THE REPORTER: Counsel, did you say Hardeman?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Counsel, approach.

(Sidebar discussion not reported.)

BY MR. MILLER:

Q. Doctor, this is from a proceeding on March 11th, 2019. And we're at page 1654. And I'm starting at line 7.

THE COURT: No. Allow her to refresh her recollection --

MR. MILLER: Absolutely.

THE COURT: -- and ask her the question. Do not read from the transcript.

MR. MILLER: I understand, Your Honor. Thank you.

THE COURT: Okay.

BY MR. MILLER:

Q. If you'd read, please, from line 7 through line 11, the questions you were asked and the answers you gave.

Does that refresh your recollection that you did not know Roundup was a pesticide on March 11th?

A. Yes. It clarifies my language.

Q. Okay.

A. Do you want me to read it?

THE COURT: No.

BY MR. MILLER:

Q. I don't think the Court does. Thank you.

But you do know now that Roundup is a pesticide?

A. Roundup is an herbicide. Herbicides are under the classification of pesticides, so it's really both.

MR. MILLER: You can keep that up there. I might be using that later.

Let me just look and see if I'm done here.

BY MR. MILLER:

Q. So that was March 11th. You wrote your report in this case in January, the case we're on here today, right?

A. I'll look at the date. If you can give me a copy of my report, I can tell you what the date was.

Here it is. The date is not listed.

Do you have a copy that says the date on it?

Q. I do. I'm just going to approach with the Court's permission. I'm going to have a copy for you later.

A. I'm so sorry. It's on the last page. It was written January 24th -- it was signed off, final, January 24th of 2019.

MR. MILLER: Subject to the points raised, I don't object to the doctor as an expert in non-Hodgkin's lymphoma; but, as to its relationship to pesticides, I do object.

THE COURT: Okay. Overruled.

You may resume.

MR. ISMAIL: Thank you, Your Honor.

DIRECT EXAMINATION (resumed)

BY MR. ISMAIL:

Q. So, Dr. Levine, I want to give an overview for the jury about the topics we're going to cover and just sort of give the headline of your opinions, and then we're going to go back and explain how you got there. Okay?

A. Sure.

Q. So are we showing here a summary of what we're going to talk about today?

A. Yes, we are.

Q. And did you look at Mr. Pilliod's medical records to identify whether he has any risk factors for the development of his diffuse large B-cell lymphoma?

A. Yes, I did.

Q. And did you identify whether he had it?

A. Yes, I did. He had a very prominent risk factor for his non-Hodgkin's lymphoma.

Q. And what was that?

A. That was a fully abnormal immune system, an immune system that was not normal.

Q. And was his abnormal immune system, as you identified it, an identifiable risk factor for his non-Hodgkin's lymphoma?

A. Perhaps the most important risk factor for non-Hodgkin's lymphoma is an abnormal immune system, the inability of the immune system to recognize that first cell as cancerous and foreign and get rid of it.

Q. Did that increase his risk for developing the condition he eventually received?

A. That would absolutely increase his risk of having developed diffuse large B-cell lymphoma.

Q. And, Doctor, based on your review of both Mr. Pilliod's records and the other information you described, do you have an opinion as to whether or not

Roundup played any role in Mr. Pilliod's development of cancer?

A. Yes. The bulk of the data from valid scientific studies indicate that the Roundup did not cause Mr. Pilliod's diffuse large B-cell lymphoma.

Q. Now, let's go back and talk about -- you described the immune system and its role in cancer surveillance and protection just generally. And we're going to go in and describe some of that more fully.

One second, Doctor.

So if we can advance the slide.

Doctor, let's start with a description of non-Hodgkin's lymphoma, and then we're going to get into how it relates to the immune system.

A. Okay.

Q. So if you would, please explain sort of generally non-Hodgkin's lymphoma, as you would to a patient or a family member.

A. Non-Hodgkin's lymphoma is actually a cancer of the immune system itself. And these can be cancers either of B lymphocytes or T lymphocytes. There are many different components of the immune system, but two of those components are B or T lymphocytes.

B-cells make a protein called an antibody against the germ, and that's what kills the germ or the

foreign cancer cell. T lymphocytes directly recognize that germ or cancer cell and directly kill it.

So this non-Hodgkin's lymphoma is actually a cancer of these very important components of the immune system itself. But it turns out that non-Hodgkin's lymphoma is not one disease at all. At this point, there are over 60 different types of non-Hodgkin's lymphoma.

And it's really important to understand that because these are different diseases. They have different presentation, the way the patient first comes to you. They have different causes. They have different treatments. They have different prognosis, how likely you are to do well or not. These are totally different diseases.

If you're talking about diffuse large B-cell lymphoma in Mr. Pilliod, you need to talk about diffuse large B-cell. It's not the same as non-Hodgkin's lymphoma overall.

Usually, these malignancies, the non-Hodgkin's lymphoma, begin in the lymph glands. But lymphocytes, B-cells or T-cells, have to be all over the body all the time because they're always looking for foreign germs or foreign cells.

So those cells travel very quickly into other

areas of the body. When they do, that's called metastatic, or spreading, of that cancer. And it's very common for lymphoma to spread in that way simply because normal B or T lymphocytes spread in that way and always are moving throughout the body.

Q. And if you look at all subtypes of non-Hodgkin's lymphoma, how prevalent or common of a cancer is that?

A. It's about 2 to 4 percent of all cancers. It's not the most common, but it's not rare either.

Q. And when we talk about how cancer develops, let's talk about it from the cellular level.

A. Okay.

Q. Can you help teach us how a cell progresses and -- or what the evolution or journey is to becoming a cancer?

A. Actually, it's a long journey. There are several steps that are required to actually get a cancer.

First of all, one needs something that's called a driver mutation in the DNA, an accident in the DNA of a given B or T lymphocyte in this example. The errors -- there are all kinds of errors that can occur in the DNA, accidents.

But your body has a way to deal with that. We

have these DNA repair mechanisms. So you're always making little errors in the DNA, and your repair mechanisms take care of that for you.

The mutation that occurs that causes a cancer eventually has to be what we call a driver mutation. That's very specific. It's not just an abnormality; it's a very specific abnormality, a mutation in the DNA, that is then taken forward into every single cell that divides from that cell.

So the one cell will divide. Both of them have the same driver mutation. Those two cells divide. Now all four of them have the same driver mutation. So it is a permanent mutation, a very specific mutation in a specific area of the DNA that controls how cells divide and how they die.

And that driver mutation is going to say to that cell, "Divide, divide, divide, and never stop." And it's also going to say to the cell, "And don't die either."

So first you need a driver mutation. Not any old error, not only any old abnormality, you need a driver mutation.

The second thing you need for a real cancer to develop is some problem with the repair mechanism. So the person's ability to repair damage to the DNA has

been impaired, faulty in some way.

And then the third thing that you need is an abnormal immune system. Because even if you have a driver mutation, even if you don't have an ability to repair it, you still have an immune system.

And the immune system should see that cell and get rid of it. So, really, you need a very specific driver mutation. You need the inability to correct that. And then you need an immune system that was also abnormal and unable to recognize that abnormal cell.

Q. So let's make sure that I understand this.

Is any DNA damage -- is that necessarily a driver mutation?

A. No. A driver mutation is very specific. It is a mutation that is hereditable. It passes on through all the generations of those cells. It doesn't die, doesn't cause death of the cell. And specifically says to the cell, "Divide, divide, divide, divide, and do not die."

Q. Are there mutations that can occur even if they're passed on to daughter cells that are not one of these driver mutations that is necessary for the development of cancer?

A. Would you repeat that? I'm not sure.

Q. Sure.

Are there other mutations that are not driver mutations?

A. Yes, there are.

Q. What do we call those in medicine?

A. They're called passenger mutations. It's kind of interesting, because 90 to 99 percent of all of the mutations are really passenger. They're irrelevant. They don't really matter to the person or to that cell.

So it's a very specific kind of mutation that will eventually lead to cancer if those other factors are present as well.

Q. Does glyphosate, or Roundup, cause mutations?

A. No study has shown that glyphosate causes mutations in those cells.

Q. And you said the second part -- and we're going to talk about that a bit this morning -- is, for a cancer to develop, the immune system failed in some way to detect or destroy that cancer; is that correct?

A. That is correct.

Q. Now, the jury has heard this term "genotoxicity." Obviously, you've heard that in your 40-plus years as an oncologist?

A. Yes, I have.

Q. Is genotoxicity the same thing as cancer?

A. No. Genotoxicity is a nonspecific word. It

just means some abnormality at that DNA, some problem. But it doesn't mean anything more than that.

Q. And did you help us put together this explanation, and can you tell us what you're communicating here with this slide?

A. Yes. I just wanted to explain what genotoxicity really meant.

And, again, it's a very nonspecific term. It just means any kind of DNA damage. It is not -- it doesn't mean driver mutation, by any means. So it's a nonspecific term. It is not the same as a mutation, and it's not the same as a driver mutation.

Q. So you have some examples that you put here, double-strand breaks or single-strand breaks.

What are those examples of?

A. Those are examples of nonspecific DNA damage. We get one bit of our DNA, half from your mother and half from your father. So one strand from your mother, one strand from the father. And both strands could break, a single strand could break. That's what it means. And those are nonspecific examples of DNA damage.

Q. And does double-strand break or single-strand break, does that mean the same thing as a mutation or a driver mutation?

A. No, it doesn't.

Q. Now, we have -- the next bullet you put here were tests. And these are some tests that the jury may have seen earlier in this trial.

Do those tests that are described here testing for mutations?

A. No. Those tests -- comet assays and binucleated micronuclei -- big words -- those are not tests of mutations at all; those are tests of nonspecific genotoxicity.

Q. Now, the jury has heard about a couple of studies that involve aerial spraying of formulated glyphosate in areas of Ecuador, the border of Ecuador, I believe?

A. Yes.

Q. And the study authors were Paz-y-Mino and Bolognesi.

Have you read those articles as well?

A. Yes, I have.

Q. And the jury has seen them several times. We're not going to walk through them yet again. But, in your review and opinion, Doctor, do they show that formulated glyphosate causes driver mutations, first of all?

A. Neither of those studies show that glyphosate

causes driver mutations. They do not show that.

Q. Can you even read those articles to conclude that glyphosate-based formulations cause specific genetic damage?

A. The papers were interesting to me. The Bolognesi article was careful to look at individuals who were exposed to glyphosate and those who were not exposed. And there was no difference at all in these nonspecific changes that they described between those who were exposed and those who were not exposed to glyphosate.; i.e., it did not show me that the patients -- the individuals in the city -- one was in Columbia -- the Bolognesi was in Columbia. The other, Paz-y-Mino, was in Ecuador, I believe -- so the populations there.

Q. And in the Paz-y-Mino study, after the initial tests, did those researchers go back to those communities and do additional genetic testing of the citizens there?

A. Their first testing was done somewhere between two weeks and two months from the time that the spraying was done. We have no idea what else those individuals might have been exposed to during those two weeks to two months. It did not show statistical evidence of any chromosomal mutations, no mutations, and no nonspecific

DNA damage. When they went back two years later, there was nothing there at all.

Q. So have you also heard the term "oxidative stress"?

A. Yes, I have.

Q. Is oxidative stress the same thing as cancer?

A. No, oxidative stress is not the same thing as cancer.

Q. Is oxidative stress even the same thing as mutation?

A. No, oxidative stress is not the same thing as a mutation.

Q. Is oxidative stress something that is happening in our bodies even without exposure to any chemicals?

A. Yes. We're always making these oxygen radicals, they're called, and we have mechanisms to suppress them, to get rid of them. And there's a balance of what we make and what we destroy.

It's happening all the time in our bodies. When you have a cold, the oxygen radicals will go up. When you run a marathon, the oxygen radicals will go up. So it happens quite commonly in all of us.

Q. Does that mean having a cold causes cancer?

A. No, having a cold does not cause cancer,

thankfully.

Q. So can oxidative stress lead to genotoxicity?

A. Yes, it can lead to nonspecific DNA damage.

Q. As you described for us, is that the same thing as having one of these important necessary mutations that is part of cancer development?

A. It is not at all the same thing.

Q. And now I just want to sort of summarize this part of your testimony here, Doctor.

So with respect to these concepts of genotoxicity and oxidative stress, is genotoxicity cancer?

A. No, it is not.

Q. Is genotoxicity the same thing as a driver mutation?

A. No, it is not.

Q. Is oxidative stress cancer?

A. No, it is not.

Q. And is oxidative stress the same thing as a driver mutation?

A. No, it is not.

Q. You described that the necessary step in cancer development is this important mutation.

Do you need to have exposure to something like a chemical for one of these driver mutations to occur?

A. No, not at all.

Q. And can you explain why that is.

A. Maybe I'll use an example with Mr. Pilliod. Mr. Pilliod had something called ulcerative colitis, and that means that his immune system is abnormal and sees his own colon cells, his own gut cells, as foreign to him. And because of that, his own cells are trying to knock off his own internal colon cells all of the time. That's all internal. He doesn't need any other external force to allow this to occur. His immune system is seeing something foreign all the time and reacting against it.

Q. And as those lymphocytes react to that inflammation or, in his case, inability to recognize his own tissue as cell for foreign, what are the lymphocytes doing as they're stimulating?

A. So as those lymphocytes are stimulated all the time, they're dividing over and over and over. And as they divide over and over, every time they have the opportunity for an error to occur, in that, before the cell can divide, the DNA, the whole DNA has to divide.

And if those cells are dividing over and over and over again, there is an increased chance that there will be an error at that DNA as it's tying to divide itself, and one of those errors could certainly be a

driver mutation.

Q. This process of stimulation of the lymphocytes in response to either an autoimmune disease or a virus, does that have a name?

A. It's called chronic antigenic stimulation. So something foreign to you would be an antigen, a foreign protein, something foreign.

So chronic stimulation by something -- an abnormal protein, something foreign to you -- allows those lymphocytes, forces the normal ones to divide over and over to try to knock off that foreign thing, be it your own cell or be it another.

Q. This concept of product antigen stimulation that you've told us about, is this something that you teach medical students and something that you've looked at yourself outside this courtroom?

A. Yes, I have. It is well known, for example, that inflammation is one of the risk factors related to non-Hodgkin's lymphoma. Chronic antigenic stimulation is anything internal or external that will cause those immune cells, B- and C-cells, to divide over and over, makes them divide over and over and increases the risk that a mutation, a true driver mutation, could occur.

Q. So thus far, Doctor, you've talked about the first side of the equation, the necessary step of a

mutation.

You also mentioned that the immune system is also part of this development of cancer. I want to turn to that discussion now.

A. Okay.

Q. Okay. So when you -- before I get to the picture, let's just talk about the immune system generally.

I think everyone has a general understanding of what the immune system is supposed to do, but can you talk to us about what the cells are in the body that go about identifying foreign cells and their response to it?

THE COURT: Excuse me, Mr. Ismail. We have to take a morning break. So maybe before you launch into your next topic, we'll take a ten-minute break.

All right, ladies and gentlemen. We're going to take a break for ten minutes. We'll come back at 25 of the hour and resume. Thank you.

(Recess taken from 10:25 a.m. to 10:40 a.m.)

(The following proceedings were heard out of the presence of the jury:)

MR. EVANS: Your Honor, I believe what was raised at sidebar, Mr. Miller, both with Dr. Mucci when examining her, referenced the, quote, Johnson case and

her prior testimony in the Johnson case. And we objected and said you should not use the name of prior testimony in prior trials. And then this morning he did the same thing with this witness with the Hardeman case.

And they obviously are trying to tie both witnesses to prior trials that the jury has heard about. We know that from voir dire. And we think it's completely improper, prejudicial, violates motions in limine exactly on this that Your Honor has ordered, and we believe it is subject to and should result in a mistrial.

MR. WISNER: Your Honor --

MR. MILLER: Let me respond.

MR. WISNER: Get the witness.

THE COURT: You may step down, Dr. Levine. Thank you, Dr. Levine.

MR. MILLER: Your Honor, it was very clear, and we agreed the last with Dr. Mucci, don't say the word "trial" anymore.

THE COURT: No, don't say the word "trial" or Hardeman or whatever.

MR. MILLER: I haven't since, Your Honor, sidebar. I have not said the word Hardeman.

THE COURT: I know. But you weren't supposed to say it in the first place.

MR. MILLER: Well, I apologize, but I thought the admonition was to not say "trial."

THE COURT: No. But you know the admonition was to not reference those particular trials. I mean, I was very clear about not only trials, but you were only supposed to obliquely reference to prior proceedings.

MR. MILLER: I have to be very -- I can't --

THE COURT: You can't mention a prior trial that has taken place. Come on. You know I've said that more than once.

MR. MILLER: I understand, Your Honor. And I want to follow the Court's instructions. That's why I've been able to say -- have the privilege of practicing law for 40 years. I truly want to follow the Court's instructions.

I thought we agreed don't mention the word "trial." Now, at sidebar, Your Honor said -- and I've been following it since sidebar -- don't mention Mr. Hardeman's name. But I have to use the testimony from -- I'll call it a proceeding; I'll call it a sworn -- whatever the Court wants me to call it is what I'm going to call it.

THE COURT: I was fine with "proceeding." I said that last Thursday or Wednesday or Tuesday. I just have to remind you because, really, the objection was do

not specifically reference the trials by name. So we came up with "proceeding" so it doesn't actually reference trial.

MR. EVANS: And the date.

THE COURT: But the issue we talked about was you can't say Johnson, you can't say Hardeman because it does bring up trials that they are aware of. So "proceeding" was secondary to not mentioning specifically the Johnson trial, the Hardeman trial, which all of the jurors are aware of.

MR. MILLER: I understand. And that's the way we're going to do it.

MR. WISNER: Your Honor, just to clarify for the record, though, Mr. Miller specifically said the proceeding in March. She said, "What are you talking about?" He gave her the date. She still was confused.

THE COURT: Well, let her be confused. But don't mention --

MR. WISNER: No, I understand. I'm just saying. And he then said it was a proceeding in San Francisco. She again didn't understand, and he went to the Hardeman case, hoping that would refresh her recollection. It won't happen again, but the witness really drew that out. It wasn't Mr. Miller trying --

THE COURT: You're all skilled, very skilled.

You know how to figure that out, including having the jurors leave if you need to have them leave so that they're unaware of what's going on. That's fine too.

But no more Johnson, no more Hardeman. Absolutely. Mistrial denied, but -- we're at the end. Let's get there.

MR. MILLER: I understand.

MR. WISNER: Yes, Your Honor.

THE COURT: Let's just get there.

All right. We've going to have the jurors come back in.

(The following proceedings were heard in the presence of the jury:)

MR. ISMAIL: May I proceed, Your Honor?

THE COURT: You may.

MR. ISMAIL: Thank you.

BY MR. ISMAIL:

Q. Dr. Levine, I had just two quick follow-up questions for you before we turn to the subject of the immune system.

You mentioned that you were -- your clinical and research interest was piqued back in the early '80s when you saw this sort of unusual presentation of lymphomas in your clinical practice, and you described then your work to understand and help with that AIDS

epidemic crisis.

What was it about the presentation of those early patients before you even had a name for the condition that prompted your interest?

A. There were several things. First of all, lymphoma usually occurs in people who are older, in their 60s and so forth, and these people coming in were young men. And right away, that got my attention.

The second was that the lymphomas were widely disseminated, involving many, many, many organs in the body, widely disseminated.

I guess the next thing was it wasn't just that they had the lymphomas, these unusual, widely disseminated, fast-growing lymphomas; but they also had all kinds of interesting or difficult infections at the same time.

So it was all of those things that said to me what is this? Something is different here.

Q. Did the presentation of these patients with these infections on top of the lymphomas or concurrent with the lymphomas connect with your training in oncology that there might be an immune-related connection between what you were seeing?

A. Yes.

MR. MILLER: Your Honor, I would object.

Leading.

THE COURT: Overruled.

Go ahead. Finish your answer.

THE WITNESS: Yes. The infections and no real control over those lymphomas spreading so quickly and rapidly said to me is there something wrong with the immune system of these people? And I started to look at it.

BY MR. ISMAIL:

Q. Now, the second question I wanted to ask you from a prior discussion relates to this concept of driver mutations that you told us about.

And you told us -- well, let me ask it this way: Does showing genotoxicity, can that -- does that genotoxicity in a cell become a driver mutation?

A. No. The abnormality in the cell is a driver mutation or something else. One error doesn't lead to the other. One accident or aberration doesn't lead to a driver mutation. The mutation occurred or it didn't.

Q. So if you can't -- if all you're showing is genotoxicity generally but not mutations specifically, are you demonstrating the ability of some chemical to cause cancer?

A. Not really, no.

Q. Now, continuing to where I was right before

the break, we were just getting started about the immune system.

Can you just generally describe how the immune system works with respect to detecting and protecting us from foreign cells in our body.

A. So, first of all, the immune system is comprised of many, many different cells: The lymphocytes that we talked about, T lymphocytes, B lymphocytes. There are cells called macrophages, monocytes, dendritic cells, neutrophils, eosinophils, basophils. There are many, many different components of the immune system. And they work in concert, each doing the job in another different kind of way, a little different way, to try to keep us safe from these foreign invaders.

Q. And, Dr. Levine, have you used pictures of these immune system cells and their role and function when you are teaching medical students or other audiences?

A. Yes, I frequently use them.

Q. And have you brought some of those with you today to help teach us about the immune system?

A. I did.

Q. So the first picture we have here, can you describe what's going on? Well, first of all, how is

this picture even taken?

A. It's called an electron microscope. So it's a way to go way down on the cell level and actually see deeply into the cell when a regular microscope could not see that. And then stains are applied to show the different colors.

Q. This isn't a drawing or a cartoon; this is actually a photo?

A. This is a photograph taken from an electron micrograph.

Q. Can you tell us what you're showing here?

A. First of all, the green cell is something called a macrophage, and that's part of the immune system, one of those cells that's important. And what you see in orange are literally TB germs.

Q. Are those tuberculosis germs?

A. I'm sorry. Tuberculosis. Correct.

So what you're seeing is the macrophage has recognized these cells as being foreign. It's now literally eating them; it's engulfing them. And once it does, it will, quote, digest them. It will use all kinds of chemicals to kill that TB.

Right now we have drugs for TB. We use them. But prior to the time that we had medicines to treat TB, lots of people had it, but not everyone dies of TB. And

the reason is we have an immune system, and the immune system is supposed to do that for us, and very often it does. So this is just an example of one cell in the immune system recognizing a germ, a foreign germ as foreign, and killing it.

Q. Is a cancer cell a foreign cell that the body should recognize and protect us from?

A. Yes. That is exactly the point. A cancer cell is foreign to you and should be seen as foreign to you by that immune system. Should be able to see it, recognize it, attach to it, and kill it.

Q. Now, are these also photographs of the immune system working properly to identify a foreign cell?

A. Yes. And this is another example, something called a natural killer cell, another part of that immune system.

And the cancer cell is the one in yellow. The natural killer cell is in that grayish-blue color. And you see, first of all, it's attaching, parts of it are actually attaching to that cancer cell. They recognize it. They see that it's foreign, and they're attaching to it. And as soon as they attach to it, they literally are poking a hole in that cancer cell, and it just explodes. It dies.

So that's an example of a cancer cell being

seen as foreign and being destroyed by the immune system.

Q. Is this another photograph to help explain the concept of the role of the immune system in detecting foreign cells?

A. Yes. So what we're seeing here in the middle -- what you see in the middle is a dendritic cell, something called an activated dendritic cell, activated because it's seeing something as foreign to it.

And what it's doing here is relaying its message. This is what foreign looks like to a T-cell. So they're working in concert. The two kinds of cells are working together, one to recognize and say this is the foreign one; this is the one you better get rid of.

Now the T-cell has that message, and now the T-cell is going to go forward to do something to that foreign cell.

Q. And so if a patient has an abnormality in any one of these processes, might that affect the patient's ability to identify and protect that patient from cancer cells?

A. Yes. So you don't need all of your immune system to be gone to get in trouble here. You just need one or another of these elements of the immune system to

be abnormal.

Q. So is it simply a question of measuring T-cell count in a patient to determine whether their immune system is functioning properly or not?

A. Well, first of all, T-cells are one part of the immune system, and an important part; I don't deny. On the other hand, there are other parts of the immune system.

And, number one, you want each of these parts to be the right number. You know, if you didn't have any of these cells, you'd be in trouble. But even if you have the right number, they have to be functioning normally. They have to be working normally.

So the number is nice. But once you have a number, now you have to know do those cells actually work on not?

Q. So earlier in the trial, Dr. Nabhan was here talking with the jury, and he referenced that Mr. Pilliod had his T-cell count assessed at some point in time.

Is that T-cell count -- is that the only thing you have to consider when assessing whether Mr. Pilliod, in particular, immune system was operating normally?

A. No. First of all, the T-cells -- I did see that in his records, and the number of his T-cells were

normal, but there was no test done to look at the function. I believe that his immune system is clearly abnormal, and there are many, many different cells that compromise that immune system.

Q. And we'll show the reasons why you've arrived at that conclusion with respect to the immune system.

What are you depicting here in this next photograph?

A. I'm showing the T-cell now had that message, what is foreign? And in this example, the foreign was the cancer cell, and the big brown thing there is a cancer cell. And what you see is the T-cells are now -- they are armed with a message. They know what foreign is. Foreign has told them that is the cancer cell. They are attaching to the cancer cell, and they will kill that cancer cell.

Q. And if there's some malfunction in this process, either messaging or functioning of these cells, will that put that patient at increased risk of developing non-Hodgkin's lymphoma?

A. Sure. Those T-cells could be normal in number but not functioning.

Q. Now, has this concept of the immune system playing an important role in cancer risk resulted in new therapies that researchers are coming up with to treat

these cancers?

A. Yes. This is one of the most exciting times in all of oncology. We're using -- the immune system is so powerful in preventing cancer in us and recognizing this and preventing it, the next question was can we use the immune system to treat cancer? Can we develop -- we call them immunotherapies. And they're in the newspapers. I don't know if you've read.

But, in any event, one of those first immunotherapies, arming the immune system to fight the person's cancer, is something called CAR T-cells, chimeric antigen receptor. It's a big word; doesn't matter. But anyway, they are T-cells that we genetically modulate, engineer, to recognize diffuse large B-cell lymphoma.

And, as it turns out, there are two products that are licensed in the United States right now to treat diffuse large B-cell lymphoma, and these are CAR T-cells. They work. The T-cells, if they're working properly, or if we can give the patient T-cells that work properly, are remarkably effective. I can't tell you how exciting it is. This is remarkably effective.

And what you see here is the genetically engineered cell, the CAR T-cell directly attaching. Again, it points an attachment. It sees. And once it

sees, again, it will destroy that cell and has been able to provide survival in about 50 to 60 percent of patients who had no treatment more available to them at all with diffuse large B-cell lymphoma.

Q. Does the success of these immune-based therapies in cancer treatment speak to the importance of the immune system in protecting us all from cancer?

A. That is exactly the fact. And the reason that abnormal immune system is so prominent as a risk factor for non-Hodgkin's lymphoma is simply that fact.

Q. Now, there's a therapy called rituximab --

A. Correct.

Q. -- that has actually been referenced earlier in this trial.

Did Mr. Pilliod receive rituximab as part of his cancer treatment?

A. Yes, he did. It was part of his -- what's called the R-CHOP regimen, and the R was rituximab.

Q. Can you tell us what you're showing here with this drawing?

A. What I'm showing is the cancer cell looks different from other kinds of cells. In this example, the lymphoma cell has a protein on its surface. It's called CD20.

And what we have is an antibody. Here's

what -- a B-cell would make an antibody to kill that foreign germ. So here we have an antibody to CD20. Biology works by a lock and a key. One chemical fits into the other. And as soon as that happens, something happens. But it starts with recognizing a fit, a lock and a key.

So if the lock is the rituximab antibody, it was designed, it was developed, engineered so that it's a perfect fit to the CD20 on the non-Hodgkin's lymphoma cell.

Mr. Pilliod's lymphoma was CD20 positive. He was given the antibody. And what you can see is the attachment. Right away, it's going to attach into that lock and a key. And once it does that, by different mechanisms that I don't have to go into unless you want me to, the cell will die. By ADCC, by -- there are different mechanisms. The cell will die.

Q. Is this a form of immunotherapy?

A. It is a form of immunotherapy and has changed the prognosis in diffuse large B-cell lymphoma appreciably since that antibody was developed and used.

Q. Is this rituximab therapy that was used in Mr. Pilliod's case, was it effective as part of his other cancer treatments in allowing him to get into remission a few months after he was diagnosed?

A. Yes. This was a major component of the success of the therapy that was used. And it was successful therapy with Mr. Pilliod, which is really good.

Q. Now, I want to talk further about Mr. Pilliod and his medical history.

But, just generally, why do oncologists go about getting a patient's past medical history when they are newly diagnosed with cancer?

A. There are multiple different risk factors for lymphoma. There are multiple points in a history that might help me, guide me a little bit to know how I need to treat that patient or what special concerns I must be attentive to.

So I need to know the entirety of what is that medical history. It tells me who the patient is in a certain sense biologically, medically, gives me hints as to how I need to treat, gives me hints perhaps as to diagnosis or what I have to do.

Q. So let's just do an overview of Mr. Pilliod's medical history. And then we'll talk about how these factors led you to identify which risk factors he had for non-Hodgkin's lymphoma.

So if you can just give us an overview, a general picture of Mr. Pilliod's medical history as you

saw it from his medical records?

A. First of all, he has had a total of 22 different skin cancers. Those began in 1970 when he was in his late 20s. That would be quite unusual to be diagnosed with that many and starting so early in life.

Then I saw, in 1978, when he was 36, he had his first episode of infection in the brain. And we call that meningoencephalitis.

Encephalitis means infection and inflammation in the brain. The meninges are the tissues that cover the brain. So he had an infection of the brain, encephalitis, and of the tissues surrounding the brain.

They did not know what caused it at that time. They were thinking that it could be autoimmune; i.e., his own immune system saw his brain cells as foreign to him and tried to destroy them, or they thought it might be related to a virus of some sort. Did not know what the virus could be, but those were the two things that they were thinking about, something called herpes simplex virus and autoimmune.

In time, he's had multiple -- he had four other reoccurrences. So he's had five episodes of brain infection. This is due to the herpes simplex virus, HSV.

HSV, that herpes simplex virus, it causes cold

sores and is very, very common. I will assume that almost everybody in this room has had a cold sore every once in a while. And that's due to HSV type 1. HSV type 2 can cause sores, the same kind of sores, but in the anogenital region. But most of us certainly have had HSV 1; we have not had infection of the brain on five different occasions caused by the cold sore virus. That said to me, whoa, what is this? What is this?

That was formally proven to be due to the herpes simplex virus at a time when the spinal fluid was examined and found to have HSV in the spinal fluid. He was treated for HSV. He continues to be treated for HSV.

He then had recurrent genital warts. Now, that's also caused by a virus. It's called human papilloma virus, HPV. Again, this is a relatively common virus in the community in general; but, very often, the most common thing is that your own immune system clears it.

And then to have that herpes simplex virus causing these genital warts at the age of 60 and so forth, that would be very unusual. It was allowed to persist. His immune system did not clear it, as usually occurs.

In 2006, he was diagnosed with ulcerative

colitis. That was proven by a biopsy. And that means it's an autoimmune disease. His immune system is seeing his own colon tissue as foreign to him and trying to kill it. So that was another example to me that, oh, my gosh, this immune system, this is not normal here.

He does have a family history of cancer. And that's associated with the patient himself developing cancer.

He does have a history of 20-year pack-a-day smoker. And then he has other medical conditions, multiple brain injuries over the years in addition to the infections in his brain. A history of stroke, sleep apnea, high blood pressure, something called hemochromatosis, which is congenital, you're born with that, and you don't deal with iron normally.

Q. So let's break down some of the things you just talked about with the jury. Let's start with skin cancer.

Are these the dates and diagnoses and the locations of the skin cancers that Mr. Pilliod has experienced in his adult life?

A. Yes. These are.

Q. And have you looked at the medical records to identify each one of these diagnoses and the dates and locations?

A. Yes. I didn't have a location. I didn't have records from 1970 to tell me the location. But other than that, yes, we have the pathology reports, the dates where these biopsies were taken, and so forth.

Q. And is this an accurate summary, to the best of your ability, of those diagnoses?

A. Yes. And what you see on the left is all of them that occurred before he ever had lymphoma. And, on the right side, you see all of those that were diagnosed after he had lymphoma.

Q. So rather than go through 22 different sets of medical records, I want to work from this summary here.

So you said this first skin cancer diagnosis was back in 1970. And how old was he at that time?

A. He was 28.

Q. Is that an unusually early time to present with skin cancer?

A. Yes, it is. Skin cancer in general is not uncommon; but, at age 28, or many, many, those facts are uncommon.

Q. And that was a basal cell carcinoma?

A. Correct.

Q. Did he have repeated instances of basal cell carcinoma?

A. Yes, he did. He had a total of 16 basal cell

carcinomas.

Q. Did he have other forms of skin cancer besides basal cell carcinoma?

A. Yes. He also had something called squamous cell cancer of the skin.

Q. Is that a different type of skin cancer?

A. It's a different type of skin cancer, yes.

Q. Did he also develop melanoma of the skin?

A. Yes. He developed melanoma, which is a very aggressive kind of skin cancer, far more aggressive than these other two. He developed that about a year prior to the diagnosis of lymphoma.

Q. And you focused on this period the year prior to his diagnosis. If we look here, the jury has heard that Mr. Pilliod was diagnosed in June of 2011.

Did Mr. Pilliod develop basal cell carcinoma, squamous cell carcinoma, and melanoma within the year before he developed non-Hodgkin's lymphoma?

A. Yes. He developed three different kinds of skin cancer before he ever had lymphoma, and about a year before.

Q. In total, how many different skin cancers has Mr. Pilliod had in his adult life?

A. So, in total, he's had 22 skin cancers.

Q. Now, as someone who's been practicing medicine

for nearly 50 years, have you ever seen a patient with 22 skin cancers?

A. Actually, this would be extremely unusual. I really have not seen this before. I haven't really even seen this in my AIDS patients.

Q. Now, it has been described to the jury previously that skin cancer is associated with UV light exposure?

A. Yes, it is.

Q. Is that a risk factor for the development of skin cancer?

A. Yes. An important risk factor for skin cancer is exposure to UV sunlight.

Q. And if patients have lighter skin tone, has that also been reported as a risk factor for skin cancer?

A. Yes. That is an increased risk factor for skin cancer.

Q. Even given those risk factors, do you find it unusual that a patient would develop skin cancer on 22 separate occasions in their adult life?

A. Yes, I do. That is unusual.

Q. And what is this -- what is the relationship, as you see it, between the skin cancers and understanding Mr. Pilliod's risk for developing

non-Hodgkin's lymphoma?

A. Well, first of all, just -- let's just talk about the UV light for one moment.

UV light clearly is associated with an increased risk of all of these skin cancers. But the other interesting thing is that UV light is protective against lymphoma. So it isn't the UV light that's involved here. It's not that. That would have protected him from lymphoma.

What this means to me is something -- I need to look at his immune system. Something isn't right with the immune system. That would be one of my questions right away.

Q. Is the immune system part of the process of cancer detection and cancer protection even in the context of skin cancer?

A. Oh, yes. Absolutely. Immune system goes everywhere in the body. It has to. It has to find germs everywhere or foreign cells everywhere.

Q. Have researchers looked at the question of whether having recurrent skin cancer is a risk factor for developing non-Hodgkin's lymphoma?

A. Yes, they have.

Q. And have you reviewed that literature for your opinions in this case?

A. I did.

Q. And the jury has seen each one of those articles previously. And so we won't walk through them yet again.

But can you tell us, Dr. Levine, whether you've reviewed this literature and research yourself?

A. Yes, I have.

Q. Can you give us an overview of what these studies show with respect to the risk of developing non-Hodgkin's lymphoma in patients with recurrent skin cancer?

A. Yes. So the first two articles on your left talk about basal cell cancer. The next one talks about -- well, okay.

The first two talk about skin cancer. Both of them show statistically increased risk of developing lymphoma if you have had basal cell cancer prior.

The Cho article talks about the more basal cell cancers you have, the more increased risk you have, 0, 6, 12, and so forth.

The Nugent article talks about when you got those skin cancers. All of these are statistically significant, these relationships. They are scientifically valid.

So if the basal cell was diagnosed less than a

year from the lymphoma, or one to four years, again, significantly significant increase in lymphoma in those patients.

Nugent also looked at squamous cell cancer. And, again, squamous cell is associated with abnormal immune system in general. We can talk about that later if someone wishes.

In any event, the squamous cell is statistically more likely to be associated with an increased risk of lymphoma if diagnosed less than a year or one to four years before.

The Wheless article talks both about basal cell and squamous with the exact same data, statistically significant increase in lymphoma in patients who have had prior basal cell or prior squamous cell carcinoma. All of these are saying the exact same thing.

And then we have the melanoma work of Dr. Verner and then Dr. Lens. What we see there is kind of interesting. Because, if you have melanoma first, you are statistically more likely to develop lymphoma in time.

But the opposite is also true. If you develop lymphoma first, you are statistically more likely to develop melanoma over time.

In other words, it's going in both directions. And when it goes in both directions like that, I, as a clinician or a physician, will think of common cause. It goes in either direction. What's the common cause?

Q. So in terms of these various relative risks that you have reported here, this one is for basal cell skin cancers of six or more. Does Mr. Pilliod fall in that group?

A. Yes, he does.

Q. With respect to basal cell skin cancer, did Mr. Pilliod have basal cell skin cancer both one year and one to four years prior to his NHL?

A. Yes, he did.

Q. Same with the squamous cell?

A. Correct.

Q. This is an overall look at nonmelanoma skin cancer. He would fall in these relative risks?

A. He falls within those risks, yes.

Q. And then does he fall in both of these with respect to melanoma?

A. Well, he falls -- in other words, first he had melanoma, and then he developed lymphoma. So he falls in the first article there.

Q. Now, does this research mean that, if you have skin cancer, it causes the patient to develop

non-Hodgkin's lymphoma? Does the skin cancer turn into lymphoma?

A. No. It doesn't cause it at all; it's a risk factor. So it says to the physician be careful here, look for this, be careful for this. But it doesn't cause it at all.

Q. Now, have some of these researchers looked to see what the link is in terms of the risk of developing skin cancer and the risk of developing non-Hodgkin's lymphoma?

A. Yes, they have.

Q. Now, if you turn to Exhibit 6502 in your binder.

Is that the Wheless article that we're showing here?

A. Yes.

Q. Okay.

So this has been published previously.

And this is a look at the -- why don't you tell us in a few sentences what this paper is doing.

A. It basically -- it's looking at a big review of multiple articles, multiple studies that have been published in the literature. And they're looking in large numbers of patients at the risk of second primary cancers in people who have had either basal cell or

squamous cell carcinoma in the past.

And what they're showing is a very significant increased risk of a secondary primary -- another primary cancer, just a completely independent, additional cancer in these patients who have had nonmelanomatous skin cancer.

Q. In this review, did these authors look at 21 individual studies?

A. Yes, they did.

Q. Now, if you turn to page 6, Table 3.

Were these authors -- is this where you got the data that you showed on the prior slide with respect to nonmelanoma skin cancers?

A. Yes, it is.

Q. If we go across here, does it show non-Hodgkin's lymphoma and increased relative risks for basal cell carcinoma and squamous cell carcinoma?

A. Yes, it does, statistically significant, scientifically valid increases in non-Hodgkin's lymphoma among patients with squamous cell or basal cell cancer of the skin.

Q. Now, you told us that researchers have looked into what might be increasing the risk in both types of cancers.

I'll ask you to turn to page 8, and we'll see

if this is what you're referring to.

A. Yes.

Q. Are you there, Doctor?

A. I am.

Q. So you see the sentence that says "There are several plausible biological mechanisms --"

A. Yes, I do.

Q. -- "that could explain the association between nonmelanoma skin cancer and risk of other cancers, including immunosuppression."

Does that relate to the immune system?

A. Yes, it does, weakening of the immune system.

Q. "Chronic inflammation." Do you see that as a risk in both skin cancers and non-Hodgkin's lymphoma?

A. Yes, cells that are seen -- yes, turning over of these lymphocytes and the possibility of an error.

Q. Is ulcerative colitis an autoimmune condition that might relate in chronic inflammation?

A. Yes, it does.

Q. You see "variation in DNA repair efficiency."

Is that related to that process you were telling us about, how we can have genetic damage but our bodies repair it naturally?

A. Yes, that is what I meant. And in the Cho article that we looked at a moment ago, he looked at DNA

repair mechanisms in these patients with basal cell carcinomas and was able to show that 20 percent of them did have errors in DNA repair. And that could be consistent with other cancers as well.

Q. I want to look above.

Did they comment on the impact of controlling for potential confounders in this study?

A. Yes.

Q. And do they write "The association between nonmelanoma skin cancer and other cancers not only persisted but actually increased in strength among studies adjusting for potential confounders such as smoking status"?

A. Yes. That's what they say.

Q. If the association increases when you control for confounders, what does that mean to you as a researcher?

A. It makes it more possible that this is for real, that this is scientifically valid. So you get rid of the confounders, and it's even stronger that there's a relationship, an increased risk between these basal cell and squamous cell cancers and development of lymphoma.

Q. Now, we've talked about the 22 skin cancers and what that tells you about Mr. Pilliod's immune

system. Are there other indications in his medical history that lead you to conclude that his immune system may not be functioning properly?

A. Yes.

Q. Now, you talked about meningoencephalitis.

A. Correct.

Q. The infection of the lining and of the tissue of the brain.

A. Correct.

Q. You indicated that Mr. Pilliod first had meningoencephalitis back in 1978, is it?

A. Correct.

Q. And then did that brain infection come back, in Mr. Pilliod's case, more than once?

A. Yes, it did. It came back four different times.

Q. Is this a serious infection?

A. This is an infection that could certainly lead to death, could lead to serious long-term adverse effects. This is -- any infection of the brain is a big deal.

Q. Was it serious for Mr. Pilliod?

A. It was very serious. He was hospitalized, I believe, for about six weeks. He was comatose, as I understand, for about a month. He was exceedingly ill

with this.

Q. 1978, he was about 36 years old at that time?

A. Correct.

Q. Is developing meningitis, encephalitis at age 36 or 38 unusual?

A. HSV meningitis, the risk of that is basically 2 to 4 per million. It's very unusual. This germ is common. We all get cold sores. But our immune system takes care of it. We don't get brain infections, and we certainly don't get brain infections five different times.

Q. Now, you indicated that, at first, they didn't know what was -- "they" being Mr. Pilliod's doctors -- didn't know what was causing his brain infections.

Was that later identified as the herpes simplex virus?

A. Yes, it was.

Q. Will you turn to Exhibit 6417.

A. Yes.

Q. And specifically page 612.

A. 612.

Q. 612.

A. I see -- here it is, yes.

MR. ISMAIL: Permission to publish?

MR. MILLER: No objection, Your Honor.

BY MR. ISMAIL:

Q. So if we look up here at the top in this medical record, this relates to Mr. Pilliod. This is dated September of 2007, correct?

A. Correct.

Q. And discharge diagnoses, this was following a hospitalization?

A. Correct.

Q. So what's the first discharge diagnosis?

A. HSV meningitis.

Q. What's HSV meningitis?

A. Herpes simplex virus.

Q. And, here, it notes he had two episodes prior to this one in 2007.

Were there others documented in the medical records beyond the three that are shown here?

A. Yes. He had an additional episode, I believe, in 2001. I need to look it up to be sure.

Q. Does this discharge record show his diagnosis for ulcerative colitis?

A. Yes, it does.

Q. Now, how did they go about identifying that Mr. Pilliod's brain infections were coming from this herpes virus?

A. They did a spinal tap. And they removed

spinal fluid, and that feeds the brain. You can tell -- you can get a lot of information about what's going on in the brain by looking at the fluid that surrounds it and feeds it.

And that fluid was tested in a very, very sensitive and specific way for HSV, something called PCR, polymerase chain reaction. But in a very, very sensitive and specific assay, this spinal fluid was positive for HSV. And that proved what the doctors had believed all along: this was HSV.

Q. After this hospitalization, Mr. Pilliod received antiviral treatment?

A. Yes, he did.

Q. What did he receive?

A. He received something called valacyclovir. And that will treat HSV. So he's been on that valacyclovir since 2007 and has not had another event. He's been treated for HCV.

While he was actively infected with the meningoencephalitis, on three occasions, he was treated for HCV using specific drugs. One called acyclovir was not available when the first diagnosis was made, the first episode was made. But he's been treated for HCV on these later episodes and continues to be treated for HCV -- HSV. Sorry.

Q. You talked about the rarity of having one episode of HSV -- herpes simplex virus -- induced brain infection.

If you turn to Exhibit 6589, I'll ask you to identify that article.

A. 6589. 6809, I have.

Q. I'm sorry. 6569. I'm sorry.

MR. MILLER: Your Honor, we need to approach on that one.

THE COURT: Okay.

(Sidebar discussion not reported.)

BY MR. ISMAIL:

Q. Dr. Levine, you have that article in front of you?

A. Yes, I do.

Q. Is that the Bradshaw publication?

A. Yes.

Q. And in terms of the -- does this discuss the herpes simplex virus encephalitis in adults?

A. Yes, it does.

Q. And I would like you to turn to page 3 of this publication.

A. Yes.

Q. And in that carryover in the top left paragraph, do these authors talk about the prevalence of

having this brain infection due to the simple herpes virus?

A. Yes, it does.

Q. What do they say?

A. They say that the incidence, the development over time, of HSV encephalitis is estimated to be between two and four cases per million, both globally and in the United States.

Q. And so when you consider how rare of a condition a brain infection is from the herpes virus, is it significant to you that Mr. Pilliod had that on five separate occasions?

A. It would be significant to me if he had it on one occasion. If he had it on five occasions, it means to me something is wrong with his immune system.

Q. Now, has Mr. Pilliod experienced lasting consequences from the episodes of these brain infections that he has in his medical history?

A. Yes, he has.

Q. And what has he developed as a result?

A. He has developed, according to the records, seizure disorders, complicated seizure disorders. Recently some information related to his higher cerebral function -- his memory, his ability to use words properly.

Q. If you turn to Exhibit 6396 in your binder.

A. Yes.

Q. I'm going to ask if you can identify that medical record for us.

A. Yes. It is a record from Dr. Stan Lin, Mr. Pilliod's neurologist, one of them, and it's discussing a neuropsychiatric -- neuropsychologic evaluation, the function of Mr. Pilliod's brain function. And this is dated February 15 of 2011.

MR. ISMAIL: Permission to publish.

MR. MILLER: No objection, Your Honor.

THE COURT: Granted.

BY MR. ISMAIL:

Q. So we're looking up here at the top part of this. The date of this report is in February of 2011?

A. Correct.

Q. Is that before Mr. Pilliod developed non-Hodgkin's lymphoma?

A. Yes. He was diagnosed in June of 2012.

Q. 2011.

A. I'm sorry. 2011. I'm so sorry. Yes.

Q. So is this before he had chemotherapy?

A. Yes, it was.

Q. And do they note the reason for this referral?

A. It says "To evaluate the cognitive emotional

status due to changes in cognition" -- that's higher brain function -- "with history of grand mal seizure, status post viral meningitis."

Q. And was this all before the cancer diagnosis?

A. Yes, it was.

Q. And down below, I want to show the jury whether this is the incident that you were talking about with the jury earlier where it says "Briefly, Mr. Pilliod stated that he suffered his first grand mal seizure in 1978 and was apparently in a coma for four weeks, some of which was at Stanford and others at Washington Hospital."

A. Correct.

Q. Was that that first episode of meningoencephalitis that you talked to the jury about?

A. Yes, it was.

Q. Does this report describe that -- whether the seizure episodes and these brain infections has resulted in any complications for Mr. Pilliod?

A. It indicates he did have complications -- seizure disorder, number one, and abnormalities in his cognition, number two.

Q. And if you turn to -- all right.

If you turn to the third page of this exhibit, page 241 --

A. Yes.

Q. -- down at the bottom.

In January of 2011, does it say "The client reported that he was having sleep problem, concentration difficulties, word-finding difficulties with regards to nouns and numbers, and difficulty logging into an account as he was unable to spell his name"?

A. Yes. It states all of that.

Q. Is that before or after his cancer diagnosis?

A. That is before the cancer diagnosis in June of '11.

Q. Did you see other medical records that we don't have to go through here that are consistent with what is reported here by Dr. Lin with respect to how significant these brain infections were for Mr. Pilliod?

A. Yes, I did go through other records in addition.

Q. Now, we've talked about the 22 skin cancers. We've talked about the five brain infections from the herpesvirus.

Are there other indications from Mr. Pilliod's medical records that you found significant with respect to the immune system?

A. Yes.

THE COURT: Counsel, can we keep it to a

minimum.

MR. WISNER: I'm sorry.

MR. ISMAIL: Thank you, Your Honor.

BY MR. ISMAIL:

Q. What else did you find significant on this question of how well functioning is Mr. Pilliod's immune system?

A. The other finding was the diagnosis biopsy proven of ulcerative colitis, which is an autoimmune disease, which says to me that his immune system is abnormal. There may be a question of deficiency or abnormality, and this is an abnormality in the sense of his immune system seeing his own body cells as foreign to him and trying to kill them.

Q. If you turn to Exhibit 6376.

A. Yes.

Q. Can you tell us what that is?

A. This is a biopsy of Mr. Pilliod's colon. The date, I believe, is 9/22/2006.

Q. And what was the diagnosis given by the pathologist, if you turn to page 13, on this biopsy done on Mr. Pilliod?

A. Number 4, that fourth biopsy, says "Inflammatory bowel disease." That's what this whole topic -- kind of illness is. "IBD, inflammatory bowel

disease, consistent with ulcerative colitis."

Q. And if you look just above that, when they talk about the findings here, do they talk about whether there's any inflammation found in the left colon?

A. Yes. It says "There was marked chronic inflammation composed predominantly of plasma cells" and so forth.

Q. And does having inflammation, does that relate in any way to that chronic antigen stimulation you were talking about with the jury earlier in your testimony?

A. Yes. Those were all the immune cells going into that area trying to do their thing, dividing, dividing, trying to get there, and the opportunity for an accident as those cells and their DNA divide, an error that could be a very specific driver mutation.

Q. Now, did Mr. -- has Mr. Pilliod been dealing with other viruses other than the herpesvirus in his adult life?

A. Yes.

Q. What is that?

A. He also had human papilloma virus, causing genital warts.

Q. And has he had that on more than one occasion?

A. Yes, he has.

Q. And the jury -- we've talked about some of

these articles with other witnesses, so in the interest of time, are these the dates upon which Mr. Pilliod was diagnosed with genital warts as a result of the HPV virus?

A. Correct, beginning in 2003 when he would have been in his early 60s.

Q. Is it unusual of a gentleman of his age to present with this virus when he did?

A. Yes, it is. It should have been cleared by his immune system many, many decades before. That would be the usual outcome.

Q. Has there been research published that looks to see whether patients who have these genital warts from HPV are at an increased risk of non-Hodgkin's lymphoma?

A. Yes, there are studies that show a statistical increase in lymphoma.

Q. Are these two of the studies that you identified as part of your review of this case?

A. Yes. And both of them show that presence of HPV infection statistically increases the risk of subsequent development of non-Hodgkin's lymphoma.

Q. Do these authors comment about what might be the shared increased risk between non-Hodgkin's lymphoma and the outbreak of the genital warts?

A. Yes, they do.

Q. Are you showing that here on the next slide?

A. Yes. Both of them basically talk about the commonality as an indicator of immune impairment. It says Dr. Nordenvall and Dr. Blomberg is saying the author suggests underlying immunodeficiency as an explanation.

Q. Dr. Levine, have you helped us put together a timeline to put in context all the different things you've talked about thus far with Mr. Pilliod?

A. Yes.

Q. Is that what we're showing here?

A. That's what we're showing. It's impressive.

Q. So let's unpack this. What are you showing in blue?

A. In blue are all of the times that he developed either basal cell or squamous cell skin cancer.

Q. And you have this lighter blue color here. Why is that a lighter color?

A. I made that different simply because it's so -- it goes in either direction. It's so heavily an immune responsive tumor, and it's a far more aggressive tumor than the other skin cancers that he had. So I don't think of it exactly in the same way.

Q. And we know Mr. Pilliod's diagnosis was

somewhere in here.

A. Correct.

Q. Did many of these skin cancers occur before he was ever diagnosed with non-Hodgkin's lymphoma?

A. Yes. Most of them did occur prior to the time that he was diagnosed with lymphoma.

Q. So what do you have in the green?

A. The green are the examples of brain infection due to the herpes simplex cold sore virus.

Q. Was 2007 when he got put on that antiviral treatment to try to prevent further brain infections?

A. Yes. He was treated with the antiretroviral while he had the active infections prior to that, but the first time that he was ever treated continuously with antivirals to prevent recurrent meningoencephalitis was on that occasion in 2007.

Q. And then in the gray, what do you have depicted here in the gray?

A. The gray are his outbreaks of human papilloma virus, genital warts.

Q. And then in the yellow?

A. The yellow is the autoimmune disease ulcerative colitis.

Q. How was he treated for his ulcerative colitis?

A. He was treated with hydrocortisone enemas and

later received something called asacol.

Q. What is hydrocortisone?

A. Hydrocortisone is a type of -- I'll use the same word; forgive me -- corticosteroid. It's a steroid. And it has tremendously powerful effects to weaken the immune system. They were trying to weaken his immune system because his immune system was trying to destroy his colon, and that needed to be stopped. So they used hydrocortisone enemas to try to weaken the immune system and the inflammation in his colon.

It turns out that cortisone kills lymphocytes. Period. It kills them. It kills B-cells. It kills T-cells. He got hydrocortisone enemas.

Q. Were you able to determine from the records that were available for how long he was treated with hydrocortisone and at what doses?

A. No, I can't say. I never got those records, although I sure would have liked to have seen them.

Q. Is ulcerative colitis curable?

A. No. It waxes and wanes over time, gets worse, gets better, gets worse, gets better on its own.

Q. Doctor, when you look at Mr. Pilliod's history in total -- the skin cancers, the infections of different types, the autoimmune condition -- what is it telling you, as a cancer researcher and specialist?

A. What it tells me is that his immune system is absolutely not normal. There are components, I assume, that are normal. I don't know which specific component. And I don't even have a name for this. But it's all right. I've been in that situation before related to HIV. Didn't have a name, but that was not a normal immune system. I've seen that kind of thing before.

I don't know what his abnormality is. Don't think that it's everything. I know it isn't every component of his immune system. But to see this kind of a history over and over -- repeated serious infections; organisms that he should have been able to clear; repeated skin cancers, 22 of them; ulcerative colitis, an autoimmune disease -- this is not a normal immune system. And I say that based on 50 years of being a doctor who deals with this stuff. This is not a normal immune system.

Q. Did Mr. Pilliod's abnormal immune system, as you've described it, in your view, increase his risk of developing non-Hodgkin's lymphoma?

A. I think that his abnormal immune system substantially increased his risk of lymphoma. That is the most prominent risk factored for non-Hodgkin's lymphoma, abnormal immune system.

Q. At what age was Mr. Pilliod diagnosed with

non-Hodgkin's lymphoma?

A. It was in 6/20/11. He was 68, I believe.

Q. And are patients of that age at increased risk of developing non-Hodgkin's lymphoma?

A. Yes. Increasing age is definitely a risk factor for all cancer, actually, most cancer.

Q. And was he of that age that put him at an increased risk?

A. Yes.

Q. Does that mean that getting older, having another birthday, causes a patient to develop NHL?

A. No, not in any sense. There's an association of risk, but it has nothing to do with cause.

Q. Now, Doctor, prior witnesses here have talked with the jury about something they called a differential for non-Hodgkin's lymphoma.

And these -- I don't remember who was who, but one of these is Dr. Nabhan, and one of these is Dr. Weisenburger. Using the same board.

First of all, differential for non-Hodgkin's lymphoma. As someone who's been a practicing doctor in this area for 50 years, does that make sense to you?

A. I normally will undergo a differential diagnosis. A patient comes in, and I need to look at all the things that could cause those symptoms and come

up with what the real diagnosis might be.

But a differential cause, a differential ideology? No, this is not at all something that would -- that I've really seen before.

Q. So when you're taking care of your patients, do you go through an exercise of listing risk factors and crossing them out and circling others?

A. No. That doesn't help me. There's no point to that, really. That doesn't tell me anything.

Q. When you're teaching medical students, do you go through an exercise like Dr. Nabhan and Dr. Weisenburger do?

A. Well, if this is supposed to be a list of the possible etiologies, the causes, it isn't, because age, sex, race -- I'll go through all of them, but those are risk factors, but they aren't causes.

So if you're looking for causes, you really have to have causes. And I guess I'm impressed by the fact that many of these are not causes. Some are; some are not.

The other difficulty is that, in a general sense, about 90 percent of diffuse large B-cell lymphoma is idiopathic. We don't know what the cause is. Eventually, hopefully, we'll know, but we don't know right now.

And so, if you're dealing with a disease where 90 percent, we honestly don't know, it's very difficult to put a list that has a couple of things on it when 90 percent we don't know.

So, no, this is not valid methodology to me for many different reasons.

Q. What makes up that 10 percent of conditions of NHLs for which we do know the cause of?

A. The most prominent of all would basically be the infections. So there are a whole series of viruses that can cause a non-Hodgkin's lymphoma: HIV, Hepatitis C, Hepatitis B, adult T lymphoma leukemia, Epstein-Barr virus. There are a whole series of viruses that can cause lymphoma, that are known to be causes of lymphoma, that cause these very specific driver mutations.

There are other bacterial infections, believe it or not, that cause lymphoma. One of them is a germ called H. pylori. H. pylori causes stomach ulcers. No big deal. It also causes non-Hodgkin's lymphoma.

There are many parasites that cause lymphoma. And so if one looks at poorly resourced areas of the world, poor areas of the world, about 20 percent of all these lymphomas are due to these germs. In resource-rich areas of the world, about 10 percent of

all lymphomas are due to those germs.

Q. If you don't have one of those known causes, these viruses you talked about or bacteria, how do you properly characterize that patient's non-Hodgkin's lymphoma?

A. It's difficult to do so, especially when 90 percent we really don't know.

Q. Is that what the term you used earlier, idiopathic?

A. Yes. Idiopathic is there but we don't know why.

Q. Does the term "idiopathic" meaning nothing is causing that patient to develop non-Hodgkin's lymphoma?

A. Oh, no. They had to have all the requisites, the driver mutation, so forth. We just don't know what did it in that given person.

Q. Now, if you're going to go through this exercise of listing risk factors in Mr. Pilliod's case, is there any way to cross out age or sex or race as factors that he had?

A. No. They're well, well, well described. Increasing age is a risk factor for lymphoma. Male gender is a risk factor for lymphoma. Caucasian race is a risk factor for lymphoma. You can't cross them off as factors; they are real.

Q. Based on what you described, did Mr. Pilliod have any of the known causes of NHL in terms of the infectious processes or bacterias that you talked about with the jury?

A. No.

Q. So how would you describe or characterize his non-Hodgkin's lymphoma from a cause perspective?

A. From a cause perspective, it's really idiopathic. I can't say what caused his driver mutation. I don't know what caused his driver mutation.

Q. Did he, nevertheless, have risk factors for developing that condition?

A. He has a massive risk factor for developing lymphoma, and that was his immunodeficiency and abnormal immune system.

Q. Drs. Weisenburger and Nabhan had pesticide use on here, and I want to talk to you about that.

A. Okay.

Q. Are there certain pesticides that have been associated with non-Hodgkin's lymphoma?

A. Yes.

Q. In fact, does your hospital's website describe a link between pesticides, as a generic term, and NHL?

A. Yes, it does.

Q. Now, are there multiple types of pesticide?

A. Many, many. Hundreds. Maybe even more than that.

Q. Does City of Hope list Roundup or glyphosate as a cause or risk factor for NHL?

A. No, it does not.

Q. Do you believe, based on your review, that Roundup is a risk factor for non-Hodgkin's lymphoma?

A. I think the summary, the basis of all of the data together says to me that, no, Roundup is not associated with development of diffuse large B-cell lymphoma or non-Hodgkin's lymphoma itself.

Q. Now, after Mr. Pilliod was diagnosed, did you review those records to determine how his care management proceeded?

A. Yes, I did.

Q. And the jury has heard this previously. So if you can just give us a quick summary reminder of how his care progressed after his diagnosis in June of 2011.

A. He was treated with a regimen called R-CHOP. R was the rituximab we talked about, and then the CHOP chemotherapy. He had six cycles of R-CHOP chemotherapy. After about four cycles, he had a repeat scan, which showed the lymphoma was clearly going away, this PET scan. And after six cycles, he was said to be in complete remission. In other words, they repeated the

tests that were positive before for the lymphoma, and now all of those tests are negative.

He's had other scans since that time. I believe the last one that I saw was August of 2018. And he remains in complete remission, which is really good. So it's been over seven years in complete remission, highly unlikely that he would ever relapse.

Q. Now, was there anything about the presentation of Mr. Pilliod's DLBCL that you thought was unusual, just in terms of his symptoms, his scans, his imaging that looked unusual to you?

A. No. I've seen unusual cases, as I've discussed, in the past. This was kind of a regular diffuse large B-cell lymphoma, if you will.

Q. Is there anything in his medical records or test results that suggested to you that there must be some chemical exposure that caused his DLBCL?

A. No.

Q. Did you see anything in his workup, his history, his presentation that would lead you to rule in Roundup as a reason why he developed this cancer?

A. No.

Q. Now, did you consider the fact that Mr. Pilliod was exposed to Roundup?

A. Of course.

Q. That was part of the materials that were provided to you?

A. Absolutely.

Q. And based on everything that you've described, do you believe Roundup played any role in the development of Mr. Pilliod's NHL?

A. Based upon the entirety of the data, I feel that Roundup did not have anything to do with his development of non-Hodgkin's lymphoma.

Q. Are you aware that Mr. -- that Mrs. Pilliod developed central nervous system lymphoma in 2013?

A. I am.

Q. And you reviewed portions of her deposition testimony?

A. I did.

Q. Did you consider that fact when assessing Mr. Pilliod's case? Were you aware of that fact as you were assessing Mr. Pilliod's case?

A. Yes, I was aware of that fact.

Q. Now, in terms of -- first of all, is central nervous system lymphoma the same thing as systemic DLBCL?

A. No. They both are, quote, diffuse large B-cell lymphoma, but primary central nervous system diffuse large B-cell lymphoma is an entirely different

entity. It has a different classification system. It is a separate entity. It's not the same as diffuse large B-cell. It is -- the cause is different. The treatment is different. The prognosis is different.

So there's no relationship between primary central nervous system lymphoma and diffuse large B-cell lymphoma. They are completely different diseases.

Q. Now, in terms of the question of whether two people living in the same house or a husband and wife can develop the same cancer, based on the prevalence of non-Hodgkin's lymphoma, would you expect to see that occur from time to time?

A. Sure. You'll see it by chance.

Q. And has that been described in the medical literature as well?

A. Yes, it has.

Q. Have researchers looked to see whether that development of non-Hodgkin's lymphoma in a household occurs any more frequently than you would expect just based on the background rate of NHL?

A. Yes. There are several studies which have looked at that issue --

Q. And have you looked at that yourself?

A. Yes, I have.

Q. The jury has seen these papers before, so

we're not going to go through them in any great detail.

But, in terms of the research, there are couples who are reported here, both of whom have -- both husband and wife have non-Hodgkin's lymphoma.

A. Correct.

Q. I think, in one of these papers, it was nearly 100 couples, both husband and wife had the disease; and another, it was 50 or 60, to that effect?

A. Yes.

Q. And so when these researchers looked to see is that happening more common in couples than you would expect to see in the background rate, what did they find?

A. They found that it was the same as the background rate. There were other cancers, for example, tobacco-related that were related, both couples -- both people smoking, but not lymphoma. There was no increase in lymphoma beyond what would be expected in the normal population.

Q. So, based on this research, does this -- how do you assess this case here? Is it -- because Mr. Pilliod had one form of NHL and Mrs. Pilliod had a different form of NHL, does that mean it has to be some environmental cause that led them to develop their individual cancers?

A. No. Just as in these large studies, there's no evidence that there is any kind of commonality, if one member of a couple gets lymphoma, that the other will. The data doesn't show that.

Q. Now, one of the prior witnesses for the plaintiffs who testified here went through this exercise on a flip chart. And I'll just describe it to you and ask for your opinion.

It was described that the risk of getting DLBCL is 1 in 120 on a population basis. And then they multiplied 1 over 120 times 1 over 120, I guess to say two people getting the condition.

As I'm describing that opinion -- methodology to you, does that make sense to you as a cancer researcher?

A. No, it really -- I don't understand it, but it doesn't make sense to me. You have to look at these cases no matter what. Mr. Pilliod had a real increased risk for developing lymphoma due to his immune system.

And to take two numbers and multiply them together to give you a risk factor for a given couple, that doesn't make sense to me.

Q. So let's talk about that further.

So Mr. Pilliod has ulcerative colitis?

A. Correct.

Q. And what's the prevalence of that?

A. Oh, I don't know. I'd have to look it up.

Q. The prior witnesses said it was about 1 in 400.

A. Okay.

MR. MILLER: Object to leading.

THE COURT: Sustained.

MR. ISMAIL: Fine.

BY MR. ISMAIL:

Q. Mr. Pilliod's encephalitis, you said was --

A. 2 to 4 per million.

Q. And so you talked about the fact that he had multiple skin cancers, correct?

A. Correct.

Q. Approximately what's the -- let's pick one of them. Melanoma, for example -- the risk of getting a melanoma?

A. I'd have to look that one up too.

Q. Would it make any sense, Doctor, to say, well, Mr. Pilliod's risk of getting a brain infection from herpes is 2 in a million, and I'm going to multiple it by his risk of getting ulcerative colitis to see what are the odds that the same person would have both conditions?

A. No. That's just not scientifically valid in

any sense. It doesn't mean anything.

Q. What drives his risk of getting skin cancer or encephalitis?

A. What's driving his risk is his abnormal immune system.

Q. Doctor, I will not --

MR. ISMAIL: Your Honor, I was going to switch subjects. Did you have a 12:00 stop today or --

THE COURT: Okay. Since it's noon, and you're going to switch, let's do our lunch break now.

Ladies and gentlemen, we're going to have a 45-minute lunch today and resume at a quarter of the hour. Don't talk about anything you heard in the courtroom this morning. Go enjoy your lunch, and we'll see you at 12:45. Thank you.

(Recess taken from 11:58 a.m. to 12:55 p.m.)

THE COURT: You may proceed, Mr. Ismail.

MR. ISMAIL: Thank you, Your Honor.

BY MR. ISMAIL:

Q. Dr. Levine, let's finish up direct examination here this afternoon. I want to switch topics. We were talking about the effect of Mr. Pilliod's medical history.

And I want to briefly touch on some of the other things you've done as part of your review in this

case. Okay?

A. Sure.

Q. Now, did you, as part of your review, also look at the published literature regarding whether or not there's a relationship between glyphosate-based products and non-Hodgkin's lymphoma?

A. Yes, I did.

Q. And did you also review, as part of that, the regulatory review that summarized and assessed that same data?

A. Yes, I did.

Q. Now, the jury has heard a lot about that information. So one of the disadvantages of going last is we're not going to repeat everything the jury has heard. But I do want to get your opinion based on your review. Okay?

A. Sure.

Q. Now, when you looked at the various summaries of the scientific data, did you also review the summaries of the animal carcinogenicity studies and the mechanism studies?

A. Yes, I did.

Q. And did you factor those reviews into your opinions that you arrived at?

A. I certainly did.

Q. And did you read the epidemiology studies, the likes of which the jury is well familiar?

A. Yes, I did.

Q. Did you undertake that epidemiological review with the same process that you would as a peer reviewer or as a researcher looking at medical literature?

A. Yes. I looked at it very carefully.

Q. And when you looked at the totality of that information, what opinions did you arrive at with respect to whether or not there's a relationship between products like Roundup and non-Hodgkin's lymphoma?

A. When I looked at all of the data, reviewed it really carefully, the bulk of the data, to me, indicates that glyphosate is not a cause for non-Hodgkin's lymphoma, nor is it a risk factor.

Q. One of the things you talked about this morning is something called the SEER database.

A. Yes.

Q. Can you remind us what the SEER database is?

A. The SEER database is the United States' way of looking at cancer across the entire population to figure out if there are problems that we need to address, certain things going up that we did not expect or going down that we did not expect.

So I used that. And I've used it a great deal

in my entire career as I look at lymphoma and try to figure out new things that we didn't really understand in the past.

Q. So well before we came to you and asked for your opinions in this case, did you have experience looking at the SEER data in terms of these -- the rate of non-Hodgkin's lymphoma?

A. Yes, I had a lot of experience. I've worked with it a great deal, and I've used it many times in the research that I've done.

Q. Now, have you looked to see the trends -- the national trends in NHL in the United States over the last couple of decades?

A. Yes, I have.

Q. And did you look at that same time period to see what the usage of glyphosate-based products were over that same period of time?

A. I did.

Q. And have you helped us put together a graph to show that relationship based on your review?

A. Yes, I did.

Q. So let's just orient everyone here to what we're looking at.

What is the orange line here?

A. The orange line is the incidence, the

development over time, of new diagnoses of non-Hodgkin's lymphoma. It's given cases per 100,000 people. That's how the US, that's how the NIH, that's how SEER, that's how EPA, that's how others look at data of that sort, by -- per 100,000 people. That's what that shows.

Q. Okay. So I think you told us in that last answer, but what is the source of the data for the rate of NHL?

A. The source of the data are these mandated SEER registries, tumor registries, population-based, as well as other agencies. The National Cancer Institute, for example, will use those kinds of figures.

Q. Now, if we look back to 1974 and look at this 40-year trend line, I want to focus first on this initial sort of kink here in the data.

A. Yes.

Q. Were you actually a practicing oncologist in cancer research during this time period?

A. Yes, I was.

Q. And did you look at this data back then for any particular purpose?

A. Yes. I looked at it very carefully because I was seeing these unusual lymphomas in these young men. And my question was whether that was just me at the LA County Hospital or was that really a trend in the United

States.

So I went to this registry. And you can see there's an uptick there. And it went up between -- actually, it started early 1980s, up until 1990, significant increase year over year of these lymphomas.

From 1990 until about 2000, when it started to be flat. Little bit of an increase, but not like it was before.

And that, in my own view, was related -- and then, after that, 2000 and beyond, it's pretty flat. If anything, there's a little suggestion it might be going down a little bit, as you can see. But it certainly is not going up the way it did in the early 1980s and '90s.

Q. And what accounted for this increase in NHL that you observed in the late '80s into the early '90s?

A. What accounted for the increase turned out to be the AIDS-related lymphomas.

Q. And is it generally accepted, Doctor, that, over the last couple of decades, the national incidence of NHL has plateaued?

A. Yes. And what's really happened is, around 1996, we developed multiagent therapy, different drugs to treat HIV. They're very, very successful. And we cannot cure HIV, but we can control it so that people live normal life spans, almost normal life spans. And

the risk of these lymphomas substantially went down. And so that's the flat part.

Q. And what is the blue line?

A. The blue line is the use of glyphosate in the United States in pounds over years, over time.

Q. And where did you get that data?

A. I got it from the Environmental Protection Agency. The EPA had very specific data about glyphosate usage in the U.S.

Q. And so I think the jury has heard from other witnesses that, beginning in the mid 1990s, the usage of glyphosate went up across the United States.

Is that consistent with the graph that you're showing here?

A. Yes. Definite increase, significant increase in the use of glyphosate over time.

Q. And what opinions, if any, Doctor, do you draw from the juxtaposition of these two rates as you've shown them here in this graph?

A. My understanding of this is that, as glyphosate use increased substantially in the United States -- and, as I understand it, one of, if not the most commonly used, substance of its kind in the United States -- if this really were a major cause or a cause of non-Hodgkin's lymphoma, I should have seen the rate

of new non-Hodgkin's lymphomas going up, just as I saw the rate go up when something new called HIV came along. But I don't see that.

Q. And is this one piece of the puzzle, as it were, that informs your opinion in this case?

A. Yes. It's population-based data, and it's helped me many times in my career to understand what might be going on with different potential causes of lymphoma.

Q. Now, with respect to the epidemiology that you looked at in this case, have you looked at studies that -- some of which control for other pesticide exposures and some of which that do not?

A. I have.

Q. Doctor, which do you believe to be the more reliable data to look at if a study reports adjusted and unadjusted data?

A. That's a really important issue. If you don't -- if there are many pesticides, herbicides, other kinds of factors that a person has been exposed to, and you're looking at the results for one specific pesticide or herbicide, you have to factor in everything else that the patient has been exposed to.

And if you don't factor it in, it's not a valid assessment of what's really happening. You have

to look at each independently. You can't look at a whole glop and say that this one thing caused what was -- actually, the exposure was much, much bigger than that one thing.

Q. And so would you look at adjusted or unadjusted data?

A. I would always look at adjusted data, adjusted for those other factors that may be involved.

Q. Now, are these all studies that you reviewed as part of your work in this case?

A. Yes.

Q. And the jury is familiar with all of them, so we won't go through again and show the data.

But if you look through here, it says "relative risk adjusted for other pesticides, if available." Is that correct?

A. Correct.

Q. Is it the case that not every study reports adjusted data?

A. That's true.

Q. But where there was adjusted data, which -- did you look to that for the relative risks?

A. That would be most important to me. That would be most reliable and valid scientifically, certainly.

Q. And when you looked at -- across this collection of studies, looking at the adjusted data, did any of them report a statistically significant increased risk?

A. No. Every one of these studies, the -- none of these are statistically significant, none of them.

Q. And the McDuffie and De Roos 2003 studies that the jury has heard a great deal about, where do you report that data on this graph?

A. Well, that was reported -- they are included within the North American Pooled Project. So that was an ability to take McDuffie and De Roos, add some other cases, and make it a larger study. So that's where they are.

Q. Do some of these studies report on diffuse large B-cell lymphoma in particular, the likes of which Mr. Pilliod had?

A. Some do, yes.

Q. And have you helped put together a chart to look at that data as well?

A. Yes, I did, because that's what Mr. Pilliod had.

Q. Now, again, some of these are not adjusted; for example, Orsi, I believe, Eriksson, and Chang.

But even when you look at adjusted and some

unadjusted data, did any of them show a statistically significant increased risk of DLBCL?

A. No, none of them, even at the higher dose level or exposure levels on Andreotti's paper.

Q. And how do you assess the Leon paper down here at the bottom?

A. Leon includes some of Andreotti, not all of it. So I think it went through 2011, and Andreotti several years beyond that. So it includes some of it but not the full data set.

Q. Is that properly characterized as a borderline finding?

A. Yeah. The confidence interval is 1.00 to 1.85. So it's very gray; it's borderline.

Q. But when you look at the totality, what does it tell you as a cancer researcher?

A. The totality tells me that there is no relationship here as far as use of glyphosate and the subsequent development of diffuse large B-cell lymphoma. It's not there.

Q. Did you also consider the regulatory reviews as part of your work in this case?

A. I did.

Q. And did you find them significant in arriving at your own opinions?

A. I certainly did.

Q. How so?

A. Well, the regulatory agencies, their job is to assure the public health, to do whatever they can to make sure that the public is not exposed to things that are toxic or difficult to the health of the people who live here.

That's a big responsibility. They're going to look at these data very, very carefully. They are responsible, ultimately. And I was impressed with the findings around the world.

Q. Did you look at the findings of the various agencies in the European Union?

A. Yes, I did.

Q. Did you look at the findings of the health agencies in Canada?

A. Yes, I did.

Q. And in the United States?

A. Yes, I did, the EPA.

Q. In New Zealand?

A. Yes, I did.

Q. And what did you find when you looked at those reviews?

A. I looked at the reviews, which were very, very carefully done. They analyzed all of the data. They

analyzed more data than other studies had.

And, basically, what they conclude, there is no -- glyphosate does not cause cancer in humans. And that is the United States EPA. That is Japan. That is New Zealand. That is Australia. That is Germany. That is France. That is the entire European Union, all of those countries.

The regulatory agencies in all of those countries come to the conclusion that glyphosate is not a hazard in terms of cancer to humans. And that, to me, was very weighty data.

Q. Now, let's wrap up what we've talked about today, and then we'll have Mr. Miller have an opportunity to ask questions.

So in summary, Doctor, when you look at Mr. Pilliod's case and his presentation and the testing that he had done for his DLBCL, did you see anything there to suggest any unusual course of his disease or presentation of his disease?

A. No. It was very typical. I've seen new things when they arise. This wasn't new; it was usual.

Q. And when you look at his medical records, did you determine whether or not his immune system, as evidenced by his various diseases and conditions he's developed over the years, was functioning normally?

A. I felt very strongly that his immune system could not be functioning normally to have had all of those illnesses that he's had over all of those years. And that would dramatically increase his risk of non-Hodgkin's lymphoma.

Q. Do you believe that the evidence supports that Roundup at relevant human doses causes non-Hodgkin's lymphoma?

A. No. I believe that the totality of all the evidence together says that there is no relationship between Roundup and the increased risk of lymphoma or causing lymphoma.

Q. Did Roundup contribute to Mr. Pilliod's DLBCL?

A. No, Roundup did not contribute to his lymphoma.

MR. ISMAIL: Thank you, Doctor.

No further questions, Your Honor.

THE COURT: Cross-examination.

MR. MILLER: Thank you, Your Honor.

CROSS-EXAMINATION

BY MR. MILLER:

Q. Hi, Doctor. Did you have a good lunch?

A. Yes, I did.

Q. Great. Great. Give me a second, if you would.

We've got a lot to cover. We'll try to do it as efficiently as we can. Okay?

A. Sure.

Q. I'm going to grab a piece of paper and put it up here and sort of write some stuff down.

What we're really going to be talking about is what caused or contributed as a cause to Mr. Pilliod's diffuse large B-cell lymphoma, right?

A. Right.

MR. ISMAIL: May I relocate, Your Honor, so I can see?

THE COURT: Sure.

BY MR. MILLER:

Q. All right. Getting set up is work.

You and I agree that age didn't cause Mr. Pilliod's non-Hodgkin's lymphoma, right?

A. I agree.

Q. All right. "Did not cause non-Hodgkin's lymphoma."

All right. Age did not cause it, right?

All right. We agree his race did not cause it, right?

A. Correct.

Q. Okay.

All right. "Did not cause." All right.

All right. His gender didn't cause it? Being a male didn't cause him to get non-Hodgkin's lymphoma?

A. No, it didn't. You're right.

Q. All right. "Did not cause." All right.

You and I agree obesity didn't cause his non-Hodgkin's lymphoma?

A. That's true.

Q. Right.

Okay. So, as I understand it, you went through Mr. Pilliod's extensive medical history, right?

A. Correct.

Q. And you went through it with the idea that -- not that it caused his non-Hodgkin's lymphoma, but what it does, it shows you he had a weakened immune system.

Is that fair?

A. It shows he had a weakened immune system and an abnormal immune system.

Q. Right. So what you told us was you didn't know what caused his non-Hodgkin's lymphoma, right?

A. That's correct.

Q. Okay.

L-E-V-I-N-E?

A. That's right.

Q. And I'm pronouncing it right, Levine?

A. Yes, you are.

Q. All right. "Does not know what caused Al Pilliod's non-Hodgkin's lymphoma," right?

A. Correct.

Q. Where you and I will, with respect, disagree with each other is you did not consider Roundup as a cause, right?

A. No, that's not true. I definitely considered Roundup. I have to keep an open mind.

And because I do keep an open mind, I'm able to see things over the years. For example, HIV; for example, Hepatitis C and others. No, I look at everything. You can't close your mind as a doctor. You have to look at everything, and I did.

Q. That's absolutely well said.

You considered it, looked at the evidence, and then completely rejected that Roundup could cause it. Is that a --

A. I didn't reject it. The data -- my interpretation of the data allowed me to reject it as the cause of his lymphoma.

Q. And of anyone's lymphoma. You think there's anybody in the world that has non-Hodgkin's lymphoma as a result of Roundup?

A. The totality of the data, as I have gone over it extremely closely and carefully, does not suggest

that glyphosate is associated with an increased risk of diffuse large B-cell lymphoma or of non-Hodgkin's lymphoma.

Q. All right. Okay. That's your opinion. I know that's where we're going to have respectful disagreement.

But just to sum up, though, age didn't cause it, race didn't cause it, gender didn't cause it, obesity didn't cause it; and, as far as medical history, you're not suggesting it caused it, but you're suggesting it showed a weakened immune system, right?

A. Which allowed the cancer, whatever caused it, to occur at an increased risk.

Q. And you and I agree that everyone who gets non-Hodgkin's lymphoma, by definition, has a weakened immune system?

A. Yes. That's part of any cancer, an abnormality of mild or more. That's what age is all about, for example.

Q. All right. So Al Pilliod fits squarely in -- 75,000 people a year in America get non-Hodgkin's lymphoma, right?

A. Did you say "dead of"? I don't know what you're saying.

Q. I'm sorry. 75,000 people, approximately, a

year in America get told they have non-Hodgkin's lymphoma; is that fair?

A. Yes, it is.

Q. And every one of them, by definition, has a weakened immune system because they couldn't fight it off?

A. That is true.

Q. Okay. And you and I agree that a chemical can be the damage that starts the DNA problem that leads to non-Hodgkin's lymphoma, right?

A. Sure. It depends on the chemical and so forth, but certainly.

Q. And we agree a chemical can cause DNA damage resulting in non-Hodgkin's lymphoma, right?

A. Yes.

Q. Okay. Can we agree that Roundup is a chemical?

A. Roundup is a chemical.

Q. Okay. And although you didn't know it was a pesticide on March 11th, you and I agree now Roundup is a pesticide?

A. What I believe I said is that Roundup is an herbicide, and herbicides are considered under the larger umbrella of pesticides, so one could say it is both.

Q. But you learned that on March 11th?

A. No, not on March 11th. I said it on March 11th. I made an error when I initially said it was an herbicide and not a pesticide. In fact, it's both.

Q. So we do now agree that it is a pesticide?

A. It is a pesticide.

Q. And we agree that the website at the City of Hope lists pesticides as a cause of non-Hodgkin's lymphoma?

A. Yes, in a general statement.

Q. Okay. And you agree with that?

A. Yes, I do.

Q. I bet you and I can agree, but let's find out. There's a scientist at City of Hope that knows more about the relationship between pesticides and non-Hodgkin's lymphoma than perhaps 99.99 percent of the world?

A. I don't know, but I think it's probably true.

Q. Who would that be?

A. That's Dr. Weisenburger, someone I respect highly.

Q. Sure. I hope I don't misspell his name.

All right. Now, you put in your report, and I think you'll agree right here, that ulcerative colitis in and of itself is not a cause of diffuse large B-cell

lymphoma?

A. That's true.

Q. And although everyone that gets non-Hodgkin's lymphoma has a problem in the immune system, there's got to be a defect or a hit at the cellular level in order to cause cancer, right?

A. There are many steps along the pathway to get cancer. Certainly one of them involves specific types of driver mutations at the DNA, if that's what you meant. I wasn't sure.

Q. Sure. Driving mutation. You've also called it a hit-and-run mutation, right?

A. I have in other circumstances, yeah.

Q. Sure. And for that first hit to DNA, before the cancer appeared, it can often takes years and years, right?

A. The first hit can happen anytime, theoretically.

Q. Sure. If someone came into your office, and you found out -- I know lung cancer isn't your specialty, but somebody -- and you had to diagnose that person with lung cancer, and they said, "But, Doc, I haven't smoked in 20 years," you'd probably tell them, "Well, look, the damage is done, and it led 20 years later to the cancer." That's what scientists

understand, right?

A. Well, yes. Actually, it's 15 years, but yes.

Q. Okay. Now, although you don't agree with the conclusion, you do know that there was this International Agency for the Research of Cancer, right?

A. Forgive me. Which one?

Q. International Agency for the Research of Cancer.

A. IARC. Yes, I do.

Q. In fact, in the other report you did for Monsanto, you cited them about nine times as authority for about nine different issues.

Do you remember that in that report?

A. I don't remember nine, but I certainly did speak to it.

Q. And you know that that agency, also called IARC, invited 17 experts from around the world to look at this issue of Roundup and non-Hodgkin's lymphoma, right?

A. Yes.

Q. Let's just look for a minute. It's been a long trial, so some of us -- I have -- have forgotten who the folks are.

MR. MILLER: May I approach, Your Honor?

THE COURT: Yes.

BY MR. MILLER:

Q. Here, Doc.

A. Thank you.

MR. MILLER: Can we put that up on the screen? It's been up on the screen before, I believe. Permission to publish, your Honor.

THE COURT: I believe it has.

MR. MILLER: 3029.

BY MR. MILLER:

Q. This is the members of the working group that looked at the issue of whether Roundup was a probable human carcinogen, right?

A. They were looking at the issue of glyphosate in relationship to the development of lymphoma, yes.

Q. Yes, ma'am. And not just glyphosate, but glyphosate formulations, right?

A. Correct.

Q. And this is not -- you have a very distinguished career, and we've talked about what you've done. But you were not invited to this meeting, right?

A. No, I was not.

Q. And you wouldn't expect to be because you don't really research pesticides and non-Hodgkin's lymphoma?

A. That's true.

Q. Some of the people that were invited and voted to conclude that, in fact, Roundup did cause non-Hodgkin's lymphoma was a gentleman from the Environmental Protection Agency. You see that? Peter Egeghy?

A. Yes.

Q. And from --

A. Although it does say he was unable to attend.

Q. Oh, you're right. There were two people from the EPA. One attended; one did not. I'll rephrase.

Aaron Blair, you understand he was on that committee, right?

A. Yes.

Q. Gloria Jahnke from the National Institute of Environmental Health Sciences?

A. Yes, she's listed.

Q. And Dr. Jameson. He got the chance to visit with us here in the courtroom.

Have you read his expert report or deposition?

A. I don't remember whether I have, actually. I think I did.

Q. Matthew Martin from the Environmental Protection Agency did, in fact, attend, right?

A. Yes.

Q. But you're also right. The other gentleman, I

believe, did not attend.

Matthew Ross from Mississippi State. We have someone from Texas A&M. And you mentioned New Zealand. We have someone from New Zealand, don't we?

A. Yes.

Q. And we have someone from Canada too, don't we?

A. Yes.

Q. And we have Lauren Zeise from the California Environmental Protection Agency, right?

A. Yes, she's listed.

Q. All right. And you understand that they voted unanimously to conclude that Roundup was a probable human carcinogen, right?

A. A probable human carcinogen, yes.

Q. And that was not an unscientific conclusion, was it?

A. No, it wasn't at that time.

Q. I understand. And here's what they decided.

MR. MILLER: If I could approach, Your Honor.

BY MR. MILLER:

Q. Here is Exhibit 2048. I'm going to hand this to you, Doctor.

MR. MILLER: Your Honor, did I hand you two copies or one?

THE COURT: You did.

MR. MILLER: Sorry.

BY MR. MILLER:

Q. Okay. And have you reviewed this publication in the Lancet where they reported the news of their finding about Roundup and non-Hodgkin's lymphoma?

A. Yes. Well, glyphosate.

Q. Right. Well, it's also about glyphosate and its formulations. Are you aware of that ma'am?

A. Yes.

Q. All right. Let's go, if we can, to the back page. The jury has seen this before. Indulge me, but it's been a while. Let's look at the bottom right side. Let's go to the glyphosate and glyphosate-based formulations. I'm going to highlight that for you so we can all look at it together there.

Glyphosate and glyphosate formulations induced what kind of damage?

A. It says DNA and chromosomal damage in -- that's what it says.

Q. In mammals and in human and in animal cells, right?

A. Yes.

Q. And DNA damage is what can ultimately lead to cancer, right, if the body can't repair and fight it off?

A. It's not DNA damage in general that can lead to cancer. It is a very specific kind of mutation called a driver mutation that can lead to cancer. Nonspecific abnormalities, nonspecific chromosomal or DNA damage, that does not necessarily lead to cancer at all.

Q. Sure, Doctor. And you figure these 17 experts in cancer from around the world know what DNA damage can and cannot cause cancer, right?

A. I would believe so.

MR. ISMAIL: Speculation, Your Honor.

THE COURT: Overruled. She can answer.

THE WITNESS: I believe so.

BY MR. MILLER:

Q. Sure. And what they said was this kind of DNA damage made Roundup a probable human carcinogen, right?

A. I disagree with their conclusions. Even on the bottom of what you're showing, bacterial mutagenesis tests were negative. Glyphosate has not been shown to be a mutagenesis. It does not cause mutations. They're saying that right there.

Q. One study reported increases in blood markers of chromosomal damage, right?

A. Yes.

Q. In residents of several communities after

spraying of glyphosate formulations, right?

A. Right.

Q. And the working group classified glyphosate as a probable carcinogenic to humans.

A. Yes, they did. I just don't agree with their conclusions.

Q. I understand. Now, let's continue. Okay.

The DNA defect that can cause cancer can be a double-strand break, right?

A. No. A double-strand break in itself is not a driver mutation, no. It doesn't equal the option for cancer, given the other factors.

Q. Let me show you --

MR. MILLER: Your Honor, may I approach. I'm going to hand the witness her deposition.

BY MR. MILLER:

Q. This is from March 15th when a lawyer from my office came, I believe, down to Los Angeles and took your deposition.

Do you remember that?

A. Yes, I do.

Q. Okay. Let's look at page 143, line 4. Were you asked this question, and did you give this answer?

"Q. So that defect in the DNA could be a hit-and-run kind of mechanism,

which means the agent, some agent, is causing mutations in someone's DNA."

What was your answer?

A. I said, "That's correct."

Q. And the next question was "That defect could be a double-strand break, correct?"

A. I said, "It could be."

Q. "Could be a single-strand break?"

A. I said, "It could be any number of defects, many different defects."

Q. That's the truth, isn't it, Doctor?

A. If the defect is a specific kind of driver mutation, is it causing mutation? That was what I said. Which means the agent, some agent, is causing mutations in someone's DNA.

Q. Okay. You know, while everybody else was having lunch, I read your reports in both cases and reread your deposition.

Do you agree with me, or do you want to look? You don't use the phrase "driving mutation" in either report or in this deposition.

Are you aware of that?

A. I am aware.

Q. Okay.

A. Reasons --

Q. I didn't mean to interrupt you.

And one of the reasons you never used that phrase was, as of March 11th, you hadn't even looked at the genotox material in this case.

Do you remember saying that in a sworn proceeding?

A. I had looked at some of that data.

Q. Do you remember saying in a sworn proceeding on March 11th you had not reviewed it?

A. On March 11th I had reviewed some of the data, not all of the data. I had reviewed the epidemiologic data carefully.

Q. You know, there's a difference between epidemiologic data which you -- I'll take you at your word, you reviewed carefully. But you did not review, as of March 11, the genotox data, right?

A. I was not asked to do so.

Q. That's right. You gave your opinion without being asked or looking at that material. That's what the Monsanto lawyers wanted you to do, right?

A. No.

Q. I'm sorry. I'm confused.

You gave your report in this case in January, right?

A. Yes.

Q. And as of March 11th, in a sworn proceeding, you advised you hadn't reviewed the genotox data yet because you had not been asked to review it, right?

A. I had not been asked to comment upon it.

Q. And you hadn't done a Bradford Hill causality review of this case, right?

A. That is true.

Q. You hadn't been asked to do that either, had you?

As of March 11th, you had not looked at the animal studies on Roundup. That's true, isn't it?

A. That is true.

Q. And as of March 11th, you had no opinion on the mechanistic data, or the genotox data. That's true, isn't it, Doctor?

A. At that time.

Q. So was it this weekend that you came up with the genotox opinion?

MR. ISMAIL: Objection. Argumentative.

THE COURT: Sustained.

BY MR. MILLER:

Q. When did you come up with an opinion on the genotox?

A. When I got into the data and read it carefully and saw what it really said.

Q. But you wrote a report telling the world that my client's cancer wasn't caused by Roundup, and you hadn't read that stuff?

A. There is no study that shows that Roundup causes mutagen. It is nonmutagenic. IARC says it is nonmutagenic. EPA says it is nonmutagenic. Every one of the regulatory agencies says it does not cause mutations. You need a driver mutation. Another patient -- never mind.

Q. Do you know who Chris Portier is?

A. I don't know him.

Q. Have you read his deposition?

A. Yes, I have.

Q. Do you know who Dr. Jameson is?

A. Yes.

Q. Did you read his deposition?

A. Yes, I did.

Q. When did Roundup come on the market?

A. I believe it came on the market around 1974.

Q. What else is in Roundup besides glyphosate?

A. My understanding is that there are solvents, there are other substances in the formulation. They are not exactly the same in many different varieties of this substance.

Q. Do you know what people that know pesticides

call the other ingredients in Roundup?

A. I'm not sure what word they use.

Q. Have you ever heard the word "surfactant" before?

A. I most certainly have.

Q. What's your understanding of a surfactant?

A. Surfactant allows a given substance to get into the body more carefully and the skin more carefully and so forth, more completely.

Q. Substance to get into the body more completely.

Is there a surfactant in Roundup?

A. I believe there is.

Q. Can you tell us what it is?

A. No, I can't.

Q. Can you -- have you ever heard this acronym before, POEA?

A. Yes, I have.

Q. What is it?

A. I can't tell you what it stands for. I believe it is one of the surfactants that is included in Roundup.

Q. Is it included in Roundup in Europe or just America?

A. I don't know.

MR. ISMAIL: Objection, Your Honor. Lack of foundation.

THE COURT: Sustained.

BY MR. MILLER:

Q. Did you give us an opinion about regulatory issues in Europe?

A. Yes, I did.

Q. Before you would give us regulatory opinions about what Roundup is used in Europe and whether it's safe, would you want to know what surfactant they used in Europe?

A. I might.

Q. Do you know?

A. I don't.

Q. Would it surprise you to learn they don't use POEA in Europe?

A. No, it wouldn't surprise me.

MR. ISMAIL: Objection, Your Honor.

THE COURT: Sustained. Stricken.

BY MR. MILLER:

Q. You and I agree -- and I respect and thank you -- you said you took Al Pilliod at his word, right?

A. Yes, I did.

Q. You and I agree that Al Pilliod started spraying Roundup in 1982?

A. That's what was said.

Q. Do you believe him?

A. I have no reason to disbelieve him.

Q. Okay. And you and I agree that Al sprayed Roundup nine months a year for 28 years?

MR. ISMAIL: Objection, Your Honor. Lack of foundation. There's no way for the witness to know.

THE COURT: Okay. If she knows or how she knows.

BY MR. MILLER:

Q. Did you read Al Pilliod's deposition?

A. I did.

Q. Then you would know that Al sprayed Roundup for nine months a year for 28 years before he got non-Hodgkin's lymphoma.

A. I thought it was 36 years, but I could have been mistaken. It could have been 28.

Q. He sprayed it for 36 years, but I was trying to be fair. It was 28 years of spraying before he got --

A. I see.

Q. And in what year did he get non-Hodgkin's lymphoma?

A. In 2011 he was diagnosed.

Q. And while we're there, he was Stage 4, right?

A. Yes.

Q. Which is -- Stage 4 is the worst it can be, right?

A. Luckily, he's in complete remission and has been cured of the disease.

Q. And we're all thrilled. And I'm sure you are as well. But my question was it was the worst stage it could be at that time?

A. Yes. It was in extranodal sites. It wasn't just in lymph nodes. It was in the organ, and the organ was the bone and the bone marrow.

Q. And you agree that, not only was he diagnosed with diffuse large B-cell lymphoma, but his wife four years later was diagnosed with a form of diffuse large B-cell lymphoma.

A. Well, she was diagnosed with primary CNS lymphoma, which is a different disease, but it's a kind of lymphoma.

Q. Isn't it true that PCN, the kind of lymphoma that Alberta Pilliod had, is a form of diffuse large B-cell?

A. No. It's not a subtype. It is a different distinct entity and is considered a distinct entity in all of the classification systems.

Q. So your testimony is that Alberta Pilliod did

not have diffuse large B-cell lymphoma?

A. She had primary central nervous system lymphoma, DLBCL.

Q. DLBCL stands for what?

A. Diffuse large B-cell lymphoma --

Q. Okay.

A. -- of her primary CNS, which is a different disease.

Q. Do you know if -- how many properties Al Pilliod sprayed?

A. I think they had three properties and one house.

Q. So that's four properties total?

A. I believe that was my recollection.

Q. Do you know if he sprayed concentrate?

A. I believe he -- he may have -- no, I don't think he really sprayed the concentrate, although I don't remember.

Q. Do you know if he ever spilled it on him?

A. Yes, I do recall I believe he spilled a bit on his hands two times, I think it was.

Q. Do you know if he ever wore protective gear?

A. I don't believe he did, but I'm not sure.

Q. Now, non-Hodgkin's lymphoma can start in the bone marrow, right?

A. It can.

Q. Sure. And do you know if Roundup gets into the bone marrow?

A. I assume it does, yes.

Q. Okay. Do you know how long it stays in the bone marrow?

A. No, I don't.

MR. ISMAIL: Objection, Your Honor. Beyond the scope.

THE COURT: Overruled. If she knows.

THE WITNESS: I don't know.

BY MR. MILLER:

Q. Okay. Wouldn't that be important?

A. Not necessarily.

Q. Okay. Now, you talked about regulators around the world. You talked about New Zealand. You talked about Australia and Japan. Right here in the state of California, they have a mechanism by which they turn to IARC, right?

A. They do.

Q. Okay. And that's because, at least here in this -- not in New Zealand, but in California, they believe IARC and their scientists set a pattern that they follow here.

Is that your understanding?

A. My understanding is that any statement from IARC that something is a probable carcinogen or carcinogen would automatically go onto Proposition 65 of California.

Q. Okay. And you know that, as we sit here, in California, Roundup is a known cause of cancer for non-Hodgkin's lymphoma?

A. No. I know that the State of California puts anything that IARC calls a probable or carcinogen onto their Proposition 65 list.

Q. Let me show you what's been marked as Exhibit 1093.

A. Thank you.

Q. Now, this is from the California Environmental Protection Agency?

A. Yes, it is.

Q. It says "Chemicals listed as known to the State of California to cause cancer."

And what is listed there?

A. Glyphosate.

Q. So you and I can agree that glyphosate is known to the State of California to cause cancer?

MR. ISMAIL: Objection. Asked and answered.

THE COURT: Sustained.

///

BY MR. MILLER:

Q. Do you disagree with the State of California on this?

A. I agree that the State of California states that glyphosate is a potential carcinogen. I disagree with that opinion.

Q. Now, you know that Al had treating physicians, right?

A. Yes.

Q. And Dr. Raj, did you read her deposition?

A. Yes, I did.

Q. She's an oncologist. Are you aware of that?

A. Yes, I am.

Q. And a hematologist?

A. Yes, she is.

Q. The same formal training that you have, right?

A. I assume so. I'm not sure exactly what her training was.

Q. Sure. And she was able to meet the patient and talk to the patient and examine the patient, right?

A. Yes.

Q. Okay. You never talked to Al Pilliod?

A. No, I didn't.

Q. Never examined him?

A. No, I didn't.

Q. And never talked to Dr. Raj?

A. No, I did not.

Q. Okay. And you know, though, that Monsanto's lawyers asked Dr. Raj if Al was immunocompromised.

Do you remember reading that?

A. No, I don't.

Q. You're not aware that she said he was not immunocompromised?

A. I would be happy to look at it. I don't recall that off the top of my head.

Q. Now, you told us there's no genetic signature from the pathology that tells us Roundup caused it, right?

A. That's true.

Q. It's also true that there's no signature under the microscope to say AIDS caused a particular non-Hodgkin's lymphoma, right?

A. I would disagree there, because, under the microscope, you could see HIV virus.

Q. But without seeing the HIV virus, there's no way to tell? Non-Hodgkin's lymphoma looks like non-Hodgkin's lymphoma?

A. That's true.

Q. And Hepatitis C as a cause of non-Hodgkin's lymphoma, there is no way to look under the microscope

and say Hepatitis C caused that non-Hodgkin's lymphoma?

A. No. You can look in the lymphoma tissue and see the Hepatitis C in the lymphoma tissue, but that's done by other means other than microscope.

Q. Right. And the last matter that you wrote a report on, gave testimony on, you said Hepatitis C caused his lymphoma, and there was nothing under the microscope to prove that Hepatitis C caused it, right?

A. If I'm allowed to opine, the individual had been infected with Hepatitis C for decades.

Q. And he hadn't had Hepatitis C for ten years --

MR. ISMAIL: Objection.

THE COURT: Approach.

MR. MILLER: I'll withdraw it, Your Honor. I'll withdraw it.

MR. ISMAIL: Well, perhaps...

THE COURT: Yes. Come up.

(Sidebar discussion not reported.)

BY MR. MILLER:

Q. So, going back to what we were talking about, with AIDS or Hepatitis C or Hepatitis B, you can be negative -- well, let's just narrow it down to hepatitis. You can be negative for ten years and still have it be a cause; is that right?

A. Are you talking about Hepatitis B causing a

lymphoma, or explain.

Q. Yes. Or C.

A. So if -- so the question is you can have Hepatitis B or C for many years and get lymphoma? Is that the question?

Q. It is.

A. Yes.

Q. Okay. Now, we're going to go through some of these studies about the weakened immune system.

But there are many people that get encephalitis who are immunocompetent. That's true, isn't it?

A. Yes. Well, I don't know about many. It's 2 to 4 per million.

Q. Well, 2 to 4 per million get encephalitis. But out of the people that get encephalitis, in fact, many of them are immunocompetent?

A. That's true.

Q. Okay. Right. So just because one has encephalitis does not mean someone is immunosuppressed, right?

A. One episode? No. It doesn't mean somebody is immunosuppressed.

Q. Okay.

Do you want some water?

A. No. I have some right here.

Q. All right. I want to be a gentleman.

All right. Hang on here.

Now, tell the folks what a CD4 count is.

A. A CD4 count is -- it's a count of the number of T-cells that are called helper T-cells, T4 or CD4. Lymphocytes are helper lymphocytes and an important part of the immune system.

Q. And as an important part of the immune system, it can be measured -- right? -- in patients?

A. The number can be measured, and the function can be measured also.

Q. And you agree that a low CD4 count would show someone had an immunocompromise situation?

A. Yeah. A low CD4 count would show someone was deficient in CD4 cells and therefore would be compromised.

Q. And when we say low CD4 count, we mean anything lower than 440?

A. It depends on the lab that you're dealing with. Every lab has different cutoffs. But, in general, that would be the right range.

Q. Okay. Around that range, 400, 420, 440, something like that?

A. Yes.

Q. Now, how many pages of medical records do you think you reviewed for Al Pilliod?

A. I was asked that before, and I couldn't answer. There were thousands of pages. Some of them came on CD. Some of them were on pages. I've never counted them. It's thousands of pages.

Q. Okay. And you didn't see one page where Al Pilliod had a low CD4 count?

A. I saw one page where the doctor was concerned enough to get a CD4 count, and the count was normal.

Q. And when we took your deposition, you said you never considered Al's CD4 records when forming your opinions in this case, true?

A. I was being informed by the entire totality of his medical history. And the CD4 count, the number per se, did not inform my opinion.

Q. You didn't know there had been a CD4 lab test when we took your deposition, right?

A. The records kept coming in as the months went on. I'm not exactly sure when I got that one, but it was more recently.

Q. And we can both agree now that there was a CD4 count done on Al Pilliod and it was above normal?

A. Yes. It was good and normal.

Q. And that was done in the year 2000, right?

A. I would have to check that, but I'll take your word for it.

Q. Let's all take a look at it.

A. Okay.

Q. It's Exhibit 3112.

A. Thank you.

MR. MILLER: Permission to publish?

MR. ISMAIL: No objection, Your Honor.

BY MR. MILLER:

Q. So this was in the hospital with one of those encephalitis problems he had, right?

A. Yes.

Q. Okay. And his CD count was normal, right?

A. His CD count was 1,814. Normal is up to 1,440.

Q. Answer, yes, it was normal?

A. It was high; it wasn't normal.

Q. Okay. It was high, but it sure wasn't low?

A. It was not low. The number was not low.

Q. And, just to be clear, you would expect a low CD4 count for someone who had an immune problem, right?

A. No. I disagree with that totally.

Q. Are you aware that -- well, you do a lot of HIV work, right?

A. I do.

Q. And they have immunocompromise problems, right? When you have HIV, that's part of the problem, right?

A. The specific problem in that entity is that the HIV virus infects the CD4 cells and kills them. So in that particular issue, the specific kind of immune deficiency is CD4, but that doesn't mean that that's the only kind of immune deficiency that could be possible.

Q. AIDS, acquired immune deficiency syndrome, right?

A. Correct.

Q. And with those immune deficiency syndromes, you're looking at CD4 counts of 100, 150, or even lower?

A. I'm not sure what your question is, but there are many different types of abnormalities of the immune system. I showed you dendritic cells. I showed you macrophages. I showed you T-cells, T8 cells.

I showed other things -- other things that I didn't show but I talked about were neutrophils, eosinophils, monocytes, and so forth. There are many components of the immune system and many ways for the immune system to be abnormal.

One of those ways that is classic of HIV is a deficiency of CD4 cells. Mr. Pilliod did not have a deficiency of CD4 cells. And, as we have discussed, he

did not have AIDS.

Q. And no doctor ever diagnosed him with having a T-cell abnormality?

A. No, no doctor did.

Q. Okay. And you're aware that Dr. Raj testified that Al had never been on immunosuppressive therapy?

A. I will disagree with that. He was on hydrocortisone for several different reasons. That is immunosuppressive therapy.

Q. But Dr. Raj didn't think so?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Sustained.

BY MR. MILLER:

Q. You agree that none of -- well, I'll agree with you. None of the treaters said, hey, skin cancer caused his non-Hodgkin's lymphoma?

A. I think one of the interesting things about this, as I read the record, is that he's had so many doctors. And they're all specialists. And everybody is looking at their little section.

I had the opportunity to look at the whole thing. And when I put the whole thing together, he is immunosuppressed.

Q. Immunosuppressed is a diagnosis made by Dr. Levine?

A. I am saying he was immunosuppressed. I have not, in 50 years, seen this kind of a history in someone whose immune system is fully normal.

Q. Okay. But everyone that gets non-Hodgkin's lymphoma has a problem with their immune system?

A. The interesting thing is that the immune system weakens in all of us as we age. It's not enough to cause, you know, all these opportunistic infections and so forth like AIDS patients do. But it is enough to give a little bit of an advantage to that cancer cell. It's weak enough. That's why age is associated with increasing cancer of almost all types.

Q. But you and I agree age didn't cause Mr. Pilliod's non-Hodgkin's lymphoma?

A. No, it did not.

Q. All right. And how many people get skin cancer a year?

A. I don't know, but I know it's a lot.

Q. About 3 million, right?

A. It's a common occurrence.

Q. The number one cause of skin cancer is being out in the sun?

A. That's true.

Q. And, in fact, being out in the sun reduces your risk of non-Hodgkin's lymphoma?

A. It does.

Q. Okay. All right.

And fair-skinned people have a higher risk of skin cancer, right?

A. That's true.

Q. And you didn't look at a picture of Mr. Pilliod or look at him before you made your conclusions about immunosuppression, right?

A. I knew he was Caucasian, and I knew that Caucasian was an increased risk as opposed to other races.

Q. You didn't know he was a really fair-skinned person?

A. No, I didn't.

Q. Did you know how much time he spent in the sun?

A. I knew he had a boat. I knew he said he did spend significant time in the sun. I don't know how much time per se that was.

Q. Do you agree that people with blue eyes are at increased risk of skin cancer, right?

A. That's true.

Q. What color are Al's eyes?

A. I don't know. I subsequently learned that they are probably blue, but I don't know that.

Q. All right. All right.

You did not know what color Al's hair was, that he had red hair, before you reached your opinions, right?

A. I didn't know. You're right.

Q. And you had never seen a color picture of Al, a photograph of him before?

A. You're right. I had not.

Q. Okay. All right.

And you and I can agree, though, that, if somebody has a family history of skin cancer, they're more likely to get skin cancer as well?

A. Yes.

Q. Did Al have a family history of skin cancer?

A. Yes, he did.

Q. But he did not have a family history of non-Hodgkin's lymphoma? That's true as well?

A. That is true.

Q. Okay. Now, you know that the melanoma he had was superficial?

A. Yes.

Q. Okay. And you know what I mean when I say clean margin, right?

A. Yes, I do.

Q. And when his superficial skin melanoma was

removed, he had a clean margin?

A. Yes, he did.

Q. Okay. Let's look at Exhibit 3118.

Let me show you an article I would like to look at with you real quick on melanoma and skin cancer and subsequent cancer risk, Exhibit 3118.

MR. MILLER: Permission to publish, Your Honor?

THE COURT: Any objection?

MR. ISMAIL: No, Your Honor.

THE COURT: Granted.

BY MR. MILLER:

Q. Now, this is a study -- when you looked at skin cancer studies, you were looking at population-based studies, weren't you?

A. Yes. You need large studies because this is so common.

Q. Right. And the reason they can't do a relative risk or an odds ratio out of a population-based study, they do an SIR, right?

A. I can't agree to -- that's a big blanket statement. A standardized incidence ratio is one of the tests used to look at significance of data.

Q. Right. It's not relative risk and it's not odds ratio; it's a standardized incident ratio, right?

A. That's correct.

Q. All right.

Now, this study looked at this issue of skin cancer and subsequent risk of cancer.

And if we could please turn to Table 3, this is a 2014 study. And it shows risk of non-Hodgkin's lymphoma -- way at the bottom there. There you go, non-Hodgkin's lymphoma.

And it shows, for basal cell carcinoma, 1.08. Not statistically significant, right?

A. Non-Hodgkin's lymphoma controls in BCC. It was -- you are correct, 1.08, and not significant clinically -- or statistically. Forgive me.

THE COURT: Doctor, if you could raise your voice just a bit and speak into the mic.

THE WITNESS: Sorry.

BY MR. MILLER:

Q. And for squamous cell, it's not statistically significant either, is it?

A. That's true.

Q. It's not even close, is it?

A. It's not statistically significant.

Q. You wouldn't want to draw any conclusions from that kind of data, would you?

A. No. I wouldn't draw conclusions from any one

piece of data anywhere. I'd like the entirety of the data to review and look at carefully.

Q. Let's look at the Herr study, 3156.

You know who Dr. Lindsay Morton is, right?

A. Yes, I do.

Q. You've published with Dr. Morton, haven't you?

A. Yes.

Q. Fine doctor?

A. Yes.

Q. Let's take a look at Exhibit 3156.

I have a copy for you, Doctor.

A. Thank you.

Q. Yes, ma'am.

MR. MILLER: Permission to publish?

MR. ISMAIL: No objection.

THE COURT: Granted.

BY MR. MILLER:

Q. Now, in this article published in 2018 -- that's pretty recent, right?

A. Yes.

Q. And by one of your coauthors, Dr. Lindsay Morton, right?

A. Well, not on this particular paper.

Q. Right. But you've published papers with her?

A. Yes, in the past.

Q. All right. So let's go to the risks for CM. It's in the abstract section there. What Dr. Morton tells us is "the risk for cutaneous melanoma was significantly elevated after diffuse large B-cell lymphoma," right? Which means to say, if you get non-Hodgkin's lymphoma, she did find an increased risk of subsequent melanomas, right?

A. I'll look at this and confirm it.

Do you want to show me where the table is?

Q. Well, let me finish that sentence, and we'll look anywhere you want.

"But the reciprocal relationship was not observed," meaning she did not find an increased risk of non-Hodgkin's lymphoma after cutaneus melanoma.

That's what she reports, right?

A. I have to look at this carefully before I can answer.

Q. Well, go right ahead.

A. So tell me your question again.

Q. Sure. Did I read this correctly? Dr. Morton says, "Risk for cutaneus melanoma was statistically elevated after a diffuse large B-cell lymphoma."

A. Where are you reading so I can confirm to you that that's what she said?

Q. It's on the screen, Doc. I'm in the abstract.

"Risk for cutaneus melanoma was statistically elevated after diffuse large B-cell and Hodgkin's lymphoma, but the reciprocal relationship was not observed," right?

A. Yes, that's what she said.

Q. And what that means is she did not see a relationship where you would be at increased risk for non-Hodgkin's lymphoma after a cutaneus melanoma?

A. That's what she says.

Q. And let's look at her tables to be precise about it. In fact, what she finds in her tables is that, if you've had cutaneus melanoma, you're at less risk for diffuse large B-cell lymphoma.

Let's look at Table 3. Let's look at what Table 3 is about first.

It says "Standardized incidence ratios for a second primary lymphoid neoplasm incidence adjusted by age, sex, latency, and stage among greater-than-one-year Caucasian adult survivors for first primary cutaneus melanoma," right?

A. Yes.

Q. And they're talking about what is your risk for a second primary lymphoid neoplasm.

Do you see that?

A. I do.

Q. So what Dr. Morton is telling us, if you're 60 to 69, let's look at that category.

That would apply to Mr. Pilliod, right?

A. Yes, it would. Uh-huh.

Q. Okay. And you have a diffuse large B-cell.

Do you see that?

A. I do.

Q. So for people of his age who have a diffuse large B-cell, they're at decreased risk of getting...

A. That's what that shows.

Q. Okay. And that was just 2018, right?

A. Yes.

Q. From a very well-respected person that you coauthor articles with?

A. Yes.

Q. Okay. Let's look at the Song study.

Here we go. I'm going to hand you what we've marked as Exhibit 3157.

A. Thank you.

Q. Are you familiar with this study --

MR. MILLER: First ask for permission to publish.

MR. ISMAIL: No objection.

THE COURT: Granted.

///

BY MR. MILLER:

Q. What these scientists do in this journal, they write an article about the "Risk of a Second Primary Cancer After a Nonmelanoma Skin Cancer in White Men and Women: A Prospective Cohort," right?

A. Yes.

Q. So unlike the population-based studies, these folks are now looking at a cohort study, right?

A. That's true.

Q. Yes, ma'am.

And to answer a question we had earlier, look at the introduction section real quick, and then we'll go to the heart of the matter. Down here at the bottom. Just highlight that.

Nonmelanoma skin cancer is the most common cancer in the United States, right?

A. Yes.

Q. It consists mainly of basal cell and squamous cell. Its incidence has been rapidly increasing over the past several decades with about 6,000 in 100,000 in the year 2006, right?

A. That's what it says, yes.

Q. That sounds about right in your estimation; is that fair?

A. I have no reason to disagree.

Q. Okay. If we can go to page 2, top left corner, these scientists actually talk about those population studies. We'll start with "Most previous reports."

It says, "Most previous reports, however, were based on cancer registry data without adjustment for potential confounding lifestyle factors."

That's what population studies do, right?

A. Yes.

Q. Okay. "So the only cohort study was limited by its sample size and lacked adequate power to assess individual cancer sites. So we carried out a cohort analysis to evaluate the association between personal history of nonmelanoma skin cancer and subsequent malignancy in the Nurses' Health Study and the Professionals' Follow-up Study," right?

A. That's what it says.

Q. So they took that cohort database. And what they did -- please turn to Table 3.

Risk of -- let's look at what this table is about. "Risk of subsequent primary cancers at different sites according to personal history of nonmelanoma skin cancer in men and women," right?

A. That's what it says, yes.

Q. "Non-Hodgkin's lymphoma, our cancer of

interest, not statistically significant, 17 percent," right?

A. It is not statistically significant. That's what it shows.

Q. You know, I'm going to show you in a bit studies on the association between Roundup and non-Hodgkin's lymphoma that show a doubling of the risk for diffuse large B-cell.

You're aware of that, right?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Sustained. Is that a question?

MR. MILLER: I'll rephrase.

BY MR. MILLER:

Q. Have you looked at the Chang and Delzell study?

A. Yes, I have.

Q. And you understand that was funded by Monsanto?

A. I'd have to look at it to be sure. Don't know that off the top.

Q. Do you remember -- we'll look at it in a minute -- that it has a statistically significant doubling of the risk for people exposed to Roundup who get diffuse large B-cell lymphoma?

A. I would need to look at that data to be able

to answer the question properly.

Q. All right. And just last point on this before we move on.

If we can please turn to page 7. Page 7, "the strengths of our study."

"The strengths of our study included a prospective cohort design," right?

A. That's what it says.

Q. Okay. And they did a detailed analysis, right?

A. That's what it says, yes.

MR. MILLER: If Your Honor wants to take an afternoon break now, we can take it. I've got another 45 minutes or so.

THE COURT: It's a little too early for the break. I think we probably need to go to 20 of the hour or so. Another 30 minutes.

MR. MILLER: Good.

Anybody want to stretch or anything? Okay.

All right. Okay. Let's move on.

BY MR. MILLER:

Q. Just to be clear, before I leave the -- none of the doctors say genital warts caused this non-Hodgkin's lymphoma, right?

A. No. It's a risk factor like many others.

Q. And none of the doctors say that cold sores cause non-Hodgkin's lymphoma, right?

A. I'm not sure what you mean by "none of the doctors," but I don't think that cold sores cause lymphoma, no.

Q. And you agree with the treaters that the cold sores didn't cause the NHL. Maybe that's a better way to say it. All right.

And I think we've been over this. I apologize. I'm trying to finish up my notes. But we'd agree that also colitis did not cause the development of the diffuse large B-cell, right?

A. Right. Did not cause it but caused an increased risk.

Q. Caused an increased risk as part of this immune problem?

A. Yes.

Q. Okay. And just to be fair, though, there are certain drugs, like TNF inhibitors, that sometimes ulcerative colitis patients take, and they do cause non-Hodgkin's lymphoma, right?

A. TNF inhibitors are drugs that are specific suppressors of the immune system. They don't cause lymphoma, but they are clearly associated with an increased risk of lymphoma.

Q. And Al Pilliod did not ever take a TNF inhibitor?

A. No, he didn't. You're right.

Q. Let's look at Exhibit 6002. Okay.

Are you a member of the InterLymph Consortium?

A. No, I'm not an official member at this point.

Q. Do you know what the InterLymph Consortium is?

A. Yes. It's a group of investigators around the globe interested in lymphoma and doing studies on lymphoma.

Q. You know Dr. Weisenburger is a member?

A. Yes, I do.

Q. He was one of the founders, I believe?

A. I don't really know that, but I think it may be true.

Q. Well, let's look at this study from the InterLymph Consortium, Exhibit 6002.

MR. MILLER: Permission to publish.

MR. ISMAIL: No objection.

THE COURT: Granted.

BY MR. MILLER:

Q. Have you reviewed this before, Doctor?

A. Yes, I have.

Q. And it's "Autoimmune Disorders and Risk of non-Hodgkin's Lymphoma Subtypes," right?

A. Right.

Q. And this was published in 2008?

A. Correct.

Q. And let's go to Table 3. And what they look at is personal history of selected autoimmune disorders and pooled relative risk of non-Hodgkin's lymphoma, right?

A. Yes, sir.

Q. And they look at ulcerative colitis, right?

A. They do.

Q. And for ulcerative colitis, they show a 2 percent increased risk, not statistically significant.

A. That is true.

Q. All right. Exhibit 6193. One more ulcerative colitis study, and I promise we'll move on. Okay. I apologize.

Thanks, Doctor. We'll keep this moving. Exhibit 6493.

Do you know who Dr. Siegel is?

A. I don't know him.

MR. MILLER: Permission to publish.

MR. ISMAIL: No objection, Your Honor.

THE COURT: Granted.

BY MR. MILLER:

Q. What this doctor is looking at is a risk of

lymphoma in inflammatory bowel disease, right?

A. Yes.

Q. What he's published in 2009, a couple points I wanted to ask you about here.

In the abstract, "A general message to convey to patients is that there is likely an increased risk of lymphoma associated with the treatment for inflammatory bowel disease but that the substantial benefit of therapies outweighs the very small risk incurred," right?

A. Yes, that's what it says.

Q. And you agree with that?

A. I agree that there is an increased risk of lymphoma. And, depending on the patient, I can't really say. It depends on the given patient. There certainly will be patients in whom the risk of the lymphoma is lesser a risk than the disease being experienced by this patient with this inflammatory bowel disease.

In other words, these decisions are made on a case-by-case basis. If the inflammatory bowel disease is severe in causing real symptoms and problems to the patient, yes, it's worth it to get it treated.

Q. Right. Let's see what he says on page 785.

This scientist tells us "Standardized incidence ratios were calculated based upon the rates of

lymphoma in these populations compared to the expected rates in the general Swedish population. For patients with ulcerative colitis, there was no statistically significant increased risk of lymphoma," right?

A. I see where it says that.

Q. I'm sorry. This is on page 785. Excuse me.

This scientist tells us, "For patients with ulcerative colitis, there was no statistically significant increased risk of lymphoma," right?

A. That's what it says, yes.

Q. Whether breaking down the analysis by Hodgkin's disease or non-Hodgkin's lymphoma, right?

A. That's what it says.

Q. By inpatient or outpatient registries, right?

A. That's what it says, yes.

Q. And you yourself have not written on the relationship between ulcerative colitis and non-Hodgkin's lymphoma?

A. No, I haven't.

Q. Let's go to one more point in this paper, and we'll move on.

Page 796, right side. "In summary, although some studies examining the risk of lymphoma associated with IBD have revealed subgroups that may be at risk" -- IBD is inflammatory bowel disease?

A. Yes, it is.

Q. -- "the vast majority of studies, including those from large population-based cohorts, do not confirm these findings." That's what he reports, right?

A. That's what he says.

Q. "Based upon the available data, it is likely safe to assume that the baseline risk of lymphoma in irritable bowel disease patients mirrors that of the general population," right?

A. That's what it says, yes.

Q. And that's why, at the City of Hope website, you don't list ulcerative colitis as a cause of non-Hodgkin's lymphoma.

A. Well, I would never think that any of these autoimmune diseases would cause lymphoma; they would be risk factors.

Q. Okay. All right. Let's look at 3160.

This is on the issue of -- I think you said something about corticosteroids increase the risk of non-Hodgkin's lymphoma or something?

A. No. I said that corticosteroids suppress the immune system. What they do is kill lymphocytes.

Q. All right. I don't need to show you, then, if we can agree that corticosteroids do not cause non-Hodgkin's lymphoma?

A. No, they don't.

Q. Okay.

A. They cause immune deficiency.

Q. Okay. Now, since you've been retained as an expert by Monsanto, have you taken any steps to -- well, let me back up.

You're now aware that, A, Roundup is a pesticide, right?

A. Say that again.

Q. Roundup is a pesticide, right?

A. Yes.

Q. And I think you told us -- correct me if I'm wrong -- you're aware that it's the number one pesticide in America?

A. I know it's used very commonly. It may be the number one or right up there.

Q. Okay. So my question is have you -- you've told us you feel pretty strongly Roundup does not cause non-Hodgkin's lymphoma.

Have you taken any steps to see that that information is conveyed on the website at City of Hope, which now says pesticides can cause non-Hodgkin's lymphoma?

MR. ISMAIL: Objection, Your Honor.

THE COURT: I understood the question.

Overruled. She can answer if she knows.

THE WITNESS: I think the website is correct. Pesticides can cause. On the other hand -- repeat your question again. Now I'm forgetting what it was.

BY MR. MILLER:

Q. I'm sorry.

A. Now I'm forgetting what you were asking. Tell me what your question was. Would you repeat it.

Q. Have you done anything to say, "Hey, get that off the website"?

A. Oh, yeah, yeah. That's it.

No, I have not. And I could go on for a thousand pages on all of the things that don't cause lymphoma. That would not be an appropriate thing to put on a statement of that sort.

Q. Exhibit 3135, if I could.

Look at this, if you could, Exhibit 3135.

A. Thank you.

Q. All right.

MR. MILLER: Permission to publish.

MR. ISMAIL: Objection, Your Honor. We object.

THE COURT: You object?

MR. ISMAIL: Yes.

MR. MILLER: I'll pull it down.

THE COURT: Pull it down.

(Sidebar discussion not reported.)

BY MR. MILLER:

Q. We're not going to publish it; we're going to talk about it. Okay?

A. What are we -- I'm sorry.

Q. We're going to talk about the first invoice -- or is this the first invoice you sent to Monsanto for your work on Al Pilliod's case?

A. It's the only invoice I've sent to date.

Q. And the invoice covered from Christmas Day, right --

A. Yes.

Q. -- of 2018 through February 10th of 2019, right?

A. That's what it says, correct.

Q. And we know that you wrote your report in late January, right?

A. Yes.

Q. Okay. And what you do, you get paid by the hour. So you tell them how many hours you spent here and how many hours you spent there, right?

A. Yes.

Q. You told us there were thousands of pages of medical records, right?

A. Yes.

Q. Tell the ladies and gentlemen how many hours you spent reviewing the medical records before you wrote this report.

A. 17 1/2 hours.

Q. It says, "Review medical records, 7.5."

A. I'm sorry. I'm sorry. "Review medical records, 7.5 hours." Sorry.

Q. So you spent 7.5 hours reviewing the medical records --

A. Yes.

Q. -- thousands of pages?

A. Yes.

Q. And then wrote a report that Roundup was not implicated in any way, shape, or form, right?

A. The medical records have nothing to do with the -- I don't understand your question. Please repeat it.

Q. I just wanted to know the amount of time and energy you put into this before you wrote a report to say my client -- his problem is not related to Roundup. You spent seven and a half hours reviewing medical records, right?

A. I spent seven and a half hours reviewing those records. And many, many, many hundreds of pages were

related to nursing notes, vital signs, and so forth. It's easy to go through those.

Q. You spent 17 1/2 hours collecting and reviewing pertinent literature?

A. Yes.

Q. All right. You spent 13 1/2 hours reviewing expert reports, right?

A. Yes.

Q. You spent 16 1/2 hours preparing and editing your report?

A. That's what it says, yes.

Q. Six and a half hours in conference calls and meetings with lawyers?

A. Yes.

Q. 61 1/2 hours total by February 10?

A. Correct.

Q. $30,000 and 750, right?

A. That's what it says, right.

Q. How many more hours have you put in since then?

A. I really have not counted them. I've not done this kind of analysis yet.

Q. We asked you for a new invoice at your deposition?

A. You didn't.

Q. We did not?

A. Did you ask me?

Q. This young man sitting right here, Curtis Hoke, did he ask you for an updated invoice?

A. I don't recall that he did.

Q. Okay. Have you finished invoicing for the other matter that you're handling for Monsanto?

A. Yes.

Q. What's your total bill there?

A. It was about $80,000.

Q. So no reason to believe your total bill here is going to be any less than 80,000?

A. I don't know.

Q. Might be more?

A. I don't know.

Q. Okay. Now, you agree that hay fever and food allergies decrease the risk of non-Hodgkin's lymphoma, right?

A. The data show that.

Q. And you put that in your other report, right?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Approach did you say, Counsel?

MR. ISMAIL: I said objection to the question. I objected to the question.

THE COURT: I know. Basis?

MR. ISMAIL: Conversation we had at sidebar.

THE COURT: So I think Mr. Miller has his direction as to what he can do. Clarify all of that.

BY MR. MILLER:

Q. You put that in a report not involving Mr. Pilliod, right?

A. I can review it. I don't totally recall that, but I may have.

Q. At some point, the Court will take a break, and here's what I'm going to ask you. First, Al Pilliod had a food allergy. Are you aware of that?

A. I'm not aware of what it was.

Q. Okay. Would there be some reason why you put that in a report not involving Mr. Pilliod, that food allergies decrease the risk of non-Hodgkin's lymphoma and not put it in Mr. Pilliod's report?

A. I assume I forgot other things as well. There's no particular reason in any sense.

Q. You sort of used the same computer program for both reports?

A. I use the same computer. I don't have a program for these reports.

Q. I guess "program" is the wrong word. But, I mean, there's a lot of similarity between the two reports, that's --

A. There's some similarity.

Q. Okay. Well, now, have you heard of Aaron Blair?

A. I've heard of Aaron Blair.

Q. You understand he's an epidemiologist?

A. That's what I understand.

Q. And you understand he was the chairman of the IARC? Are you aware of that?

A. I am.

Q. Was he also involved as an author in the Agricultural Health Study?

A. I believe he was, yes.

Q. Did you read Aaron Blair's sworn deposition in this case?

A. I believe I did, yeah.

Q. Okay. Then you know that he believes Roundup is a probable human carcinogen?

MR. ISMAIL: Objection, Your Honor.

THE COURT: If she knows. You can lay a foundation as to whether she does or does not know.

BY MR. MILLER:

Q. You've told us that you've read his deposition?

A. Yes.

Q. Okay. And you're aware, then, that he says

Roundup is a probable human carcinogen?

A. Yes. And, as chairman of IARC, I would expect that he would say that.

Q. Sure. You disagree?

A. I do disagree.

Q. I'm ready to look at Exhibit 2107. And I promise to make this quick because we've looked at it a few times here in this courtroom. But this is a meta-analysis performed by Drs. Chang and Delzell at the request of Monsanto.

A. Do not know.

MR. MILLER: Permission to publish.

MR. ISMAIL: No objection, Your Honor.

BY MR. MILLER:

Q. You reviewed this, right?

A. Yes.

Q. And you know that this was prepared at the request of Monsanto?

A. Actually, I did not. But I have no reason to doubt you.

Q. Right. Okay.

So this is a meta-analysis -- well, let's be precise.

It is a systematic review and meta-analysis of glyphosate exposure and the risk of lympho and

hemopoietic cancers, right?

A. Yes.

Q. And lympho and hemopoietic cancers include non-Hodgkin's lymphoma?

A. Yes.

Q. And they include diffuse large B-cell lymphoma?

A. Yes.

Q. Which, of course, we all know is what Al had, right?

A. Correct.

Q. So let's go then together to page 12, bottom right. The meta relative risk for the association between any use of glyphosate.

When we say "any use," that means just a little bit or a lot, right?

A. Yes.

Q. And the risk of B-cell lymphoma based on two studies was 2.0, according to random effects and fixed-model estimates. That's what they report here, right?

A. That's what they say.

Q. The results are the same as reported by Schinasi and Leon, right?

A. That's what they say.

Q. Did you ever speak to Dr. Chang or Dr. Delzell about this case?

A. No.

Q. All right.

One more study. Okay. Here we go. Exhibit 2006. Thanks for your patience, Doctor.

A. Thank you.

Q. Thanks very much.

MR. MILLER: Permission to publish?

MR. ISMAIL: No objection, Your Honor.

MR. MILLER: All right.

THE COURT: Granted.

MR. MILLER: Might be break time? All right.

BY MR. MILLER:

Q. We've seen it before. Let's look at it again, Doctor, with you, and then probably take a break.

What these folks are doing in this peer-reviewed journal, Dr. Schinasi and Dr. Leon, and they're looking at non-Hodgkin's lymphoma and occupational exposure to agricultural pesticide, a systematic review and a meta-analysis, right?

A. Yes.

Q. And this was published in 2014. To put it in context, the year before IARC, right?

A. Correct.

Q. Okay. And just to look real quick at the abstract, the first sentence, "This paper describes results from a systematic review and a series of meta-analyses of nearly three decades worth of epidemiologic research on the relationship between non-Hodgkin's lymphoma and occupational exposure to agricultural pesticide ingredients and chemical groups," right?

A. That's what it says, yes.

Q. That's what they're studying, right?

A. Yes.

Q. And they go on down a couple sentences. "In a handful of papers, associations between pesticides and NHL subtypes were reported, B-cell lymphoma -- in a handful of papers, associations between pesticides and non-Hodgkin's lymphoma subtypes were reported. B-cell lymphoma was positively associated with phenoxy herbicides and the organophosphorous herbicide glyphosate," right?

A. No, it doesn't really say that. It says that NHL subtypes were reported. "B-Cell lymphoma was positively associated with phenoxy herbicides and the organophosphorous herbicide glyphosate." It doesn't say that diffuse large B-cell.

Q. Okay. Take a look back here. Diffuse large

B-cell is a form of B-cell lymphoma, right?

A. Yes, it's within that category, but the category of B-cell lymphoma is very heterogeneous. It's not one entity; it's many.

Q. Let's go to page 64.

"The strongest meta RR," relative risk estimates.

Blow that up.

"The strongest meta relative risk estimates were associated with subtypes of non-Hodgkin's lymphoma. There was a positive association between exposure to organophosphorous herbicide glyphosate and B-cell lymphoma," right?

A. That's what it says there, yes.

Q. And then, to be precise, it goes down to diffuse large B-cell, right?

MR. ISMAIL: Objection, Your Honor.

THE COURT: Hold on one second. Don't answer the question once there's an objection.

The objection is?

MR. ISMAIL: So if the question --

MR. MILLER: I'll withdraw that question. Yeah. Fair enough.

BY MR. MILLER:

Q. So "the strongest relative risk estimates were

associated with subtypes of non-Hodgkin's lymphoma. There was a positive association between exposure to organophosphorous herbicide glyphosate and B-cell lymphoma." There was a doubling of risk. It was statistically significant, right?

A. That's what it says, yes.

Q. Thank you. All right.

MR. MILLER: Your Honor, you want to take that break now?

THE COURT: Yes, I do.

MR. MILLER: You look like you're ready for a break.

THE COURT: I think a 15-minute break. We'll start at five of the hour.

(Recess taken from 2:41 p.m. to 2:58 p.m.)

THE COURT: You may proceed.

MR. MILLER: Thank you, Your Honor.

BY MR. MILLER:

Q. Doctor, I want to go to the chart work that you did with counsel on population-based increases in non-Hodgkin's. You remember that general line of questioning?

A. Yes, I do.

Q. Okay. I'm going to show you that exhibit. I've attached that information to it.

I have one for counsel as well.

If we can put that up on the screen.

MR. MILLER: Permission to publish?

MR. ISMAIL: What's the number?

MR. MILLER: This was published by the defendants, Your Honor.

MR. ISMAIL: Well, no objection to the first page. I don't know what the rest of it is.

MR. MILLER: Well, I intend to publish the front page created by the defendant and the second page that we just created.

THE COURT: Do you have any objection? Clearly, the first page is -- I guess if he lays foundation.

Why don't we just wait and see if he lays a foundation. If you have an objection after that.

MR. ISMAIL: Thank you, Your Honor.

BY MR. MILLER:

Q. This first page is what you showed the jury, right?

A. Yes.

Q. And, as we look on the scale on the side, the right side, you list 0 to 100.

Now, what does that mean?

A. That was basically kilograms, amount of

glyphosate used, based upon data from the EPA.

Q. All right. And then, on the left side, what are those numbers?

A. Those are the new non-Hodgkin's lymphoma cases in the United States per 100,000. And, again, that's the way the NIH provides data and looks at data of that sort.

MR. ISMAIL: They've cut off the exhibit, Your Honor.

THE COURT: Why don't we just hold on one second.

BY MR. MILLER:

Q. I think this is the one that you showed through defense, right?

A. I can't tell.

Q. The question is, just wanted you to confirm that this is the one that Monsanto's lawyer and you talked about in the direct examination.

A. On the screen -- yes. It's not what I have printed here.

Q. Yes, ma'am.

And so, to be clear, the pounds per acres on the left side, right?

A. Okay.

Q. And the right side represents, per 100,000

persons, the incidence rate of non-Hodgkin's lymphoma, right?

A. Yes. Forgive me, because what was given to me did not have either of those edges of the slide.

Q. That's why I'm glad we switched to the other one. I wanted to make sure we had that. And I wasn't trying to trick you; I was just trying to get oriented.

So pounds is on the left side and incidence of non-Hodgkin's lymphoma is from 0 to 100 on the right side, right?

A. Right. 100,000 persons.

Q. So look at the one we created that is page 3. And you'll see, ma'am, all we did was change the per 100,000 persons, instead of going from 0 to 100, we have it go from 10 to 22.

Do you see that?

A. Yes, I do.

Q. Okay.

MR. MILLER: Permission to publish, Your Honor?

MR. ISMAIL: Well, Your Honor -- you know what? It's fine.

BY MR. MILLER:

Q. When you change --

THE COURT: Okay.

MR. ISMAIL: No objection --

THE COURT: Okay. No objection.

MR. ISMAIL: -- is the response I should have said.

THE COURT: That's fine. Okay.

Permission to publish.

BY MR. MILLER:

Q. So everybody knows, all we did here was change from 0 to 100 for the incidence rate for non-Hodgkin's lymphoma, and we changed it to 10 to 22.

Do you see that?

A. I do.

Q. So what it shows -- some colored pens here -- oh, it's in color.

New cases of non-Hodgkin's lymphoma go up -- when you do 10 to 22, go up dramatically until 1990, and they keep going up until about 2005, right?

A. Yes.

Q. And the use of glyphosate is in blue, isn't it?

A. On this slide, yes, it is.

Q. All right. And so, in 2005, what we have -- by then we have the new drugs for AIDS, right?

A. We do.

Q. And so that reduces the risk in non-Hodgkin's

lymphoma, right?

A. It does.

Q. Okay. So this would explain -- part of the dramatic increase could be explained by AIDS, but there's something else going on to keep the risk of non-Hodgkin's lymphoma going up and up until, I think it says, 2005, right?

A. Yes.

Q. And then it plateaus off effectively. It hovers around -- and it's at a rate of around 20, right?

A. That's true. The new drugs were licensed for the first time in 1996, and a few people were taking them at that point. More and more people were taking them over time, and the incidence of lymphoma going down over time in that group.

Q. Okay. We're happy to report there are less non-Hodgkin's lymphoma cases with the AIDS people because of these new drugs, right?

A. Right.

Q. All right. Let's move on to spousal concordance.

And you showed the jury a -- there's another blowup I want to talk to you about for a second.

Here it is. Okay.

All right. You said large studies, and they

show no spousal concordance. And I think we all know that spousal concordance is risk of one spouse getting it if the other spouse has it. Generally, that's the topic, right?

A. It means the risk of one spouse getting the same disease as the second spouse.

Q. All right. And you and I agree that Roundup came on the market in '75?

A. I thought it was '74. '75 is fine.

Q. Yeah, I think it was commercially available in '75. I'm not going to argue with you.

A. Okay.

Q. I want to let the jury know when they started looking at the data. Was it before Roundup was on the market, or was it after Roundup was on the market?

A. Some of these studies are clearly before Roundup was on the market.

Q. Let's be more precise, if we can. Wallach studied people at a Jewish hospital from 1960 to 1992.

Can we agree on that?

A. Could I see the paper before I could absolutely confirm that?

Q. That's the paper you were referring to in your PowerPoint, right?

A. Yes.

Q. Okay. So this paper, we started looking in 1960, right?

A. That is correct.

Q. Okay. And you and I agree that's 15 years before Roundup was on the market, right?

A. Yes.

Q. Okay. The Hemminki paper, do you remember what year they started looking at it?

A. No, I don't remember offhand.

Q. Will you accept my representation '58, or do you want to see the paper?

A. I will agree.

Q. Okay. 1958.

I'm terrible with math, but that's 17 years before Roundup got on the market?

A. Well before Roundup was on the market, you're right.

Q. And the Weires paper -- all right. I want to make sure I get this right.

The Weires paper, they started looking at people -- hang on. Oh, yeah -- 1958 as well, right? Or do you want to look at the paper?

A. 1958 through what?

Q. Point well taken.

From '58 to 2006.

A. Right. So some were prior to and some were after the availability of Roundup.

Q. Start date, we agree, 1958, right?

A. Fine. Uh-huh.

Q. But even the Weires paper said, even starting in '58, they're starting to see a novel cluster of non-Hodgkin's lymphoma.

Do you remember them saying that?

A. They may have been. There was no increased risk statistically among those spouses.

Q. Right. The good news for us is there was a study done right here in Berkeley, California, of concordance among spouses that started after Roundup was on the market. You're aware of that, aren't you?

A. Which study did you mean?

MR. ISMAIL: Objection, Your Honor. Lacks foundation.

THE COURT: Well, if you can lay the foundation, that's fine.

MR. MILLER: All right, Your Honor.

BY MR. MILLER:

Q. Have you heard of the Freeman study?

A. You'll have to show it to me. I don't remember off the top.

Q. Okay.

Here's a copy of the Friedman study. And it's Exhibit 2335.

A. Thank you.

Q. Yes, ma'am.

Did you review this?

A. Yes.

Q. In fact, we showed it to you at your deposition, right?

A. I believe so, although I'm not sure.

Q. And, now agree with me, it was done here in Berkeley, California?

A. Yes, it is. Oakland, California, actually.

Q. Oakland. Excuse me. That's right.

Published to the American Cancer Society?

A. Published in a journal called "Cancer," which is published by the American Cancer Society. Yes, you're right.

Q. And that's a peer-reviewed journal?

A. Yes, it is.

Q. By Dr. Friedman?

A. Yes.

Q. And a Dr. Queensberry, right?

A. Right.

Q. At the Division of Research, Kaiser Permanente Medical Care, right?

A. Yes.

Q. Okay. And let's look at it for a second.

First let's go to the heart of the matter and come back and look.

What they did, they followed -- I'm on page 2414.

They followed 25,000 married couples who were in the membership database as of June each year starting in 1976, right?

A. Correct.

Q. Right after Roundup came on the market, right?

A. Correct.

Q. Okay.

MR. ISMAIL: Your Honor, for optional completeness, counsel is skipping the first part of the method section, "subjects were selected."

MR. MILLER: Speaking objection, and we'll be happy to get to it.

THE COURT: So just state the objection. If you need a sidebar, we can do that.

MR. ISMAIL: Under optional completeness, reading the first part of the method section, which references the subjects.

THE COURT: Okay. That's not an objection.

But if you want to read it, go ahead;

otherwise, we'll do a sidebar.

MR. MILLER: I'll be more than happy to.

MR. ISMAIL: Thank you.

MR. MILLER: Sure.

BY MR. MILLER:

Q. Let's put it up on the ELMO. And we'll make sure to explain it all.

If we could put up 2535. Let's go back to the front page.

And then, like most papers, it has a method section, right?

A. Yes.

Q. And it says "The authors identified 25,670 cancer-free married couples in Northern California," right?

A. Yes.

Q. And what they did, they followed them from 1976. But what counsel wants me to point out to you, and I will, "subjects were selected from among persons who had received multiphasic health checkups between '64 and '72."

Do you see that on top?

A. Yes, I do.

Q. "Because the extensive data collected during the checkup would permit investigation" --

Do we have that? It's on page 2. Let's go to page 2. All right. There we are.

-- "would permit investigation of the reasons for spousal aggregation of cancer if they were present. Of the 175,000 persons who received at least one checkup during an eight-year period, 175,000 were identified as being free of cancer" -- right? -- "except for nonmelanoma skin cancer" -- right?

A. Yes.

Q. -- "at the time of the first checkup using cancer occurrence data described below. Follow-up was considered ended if a person either left the program or died of another cancer," right?

A. Yes.

Q. "The study was narrowed down to 25,600 married couples, who were identified as follows: For persons who were in the membership database, which consists of a list of subscribers as of each June for a year of" -- starting when? When did they start?

A. 1976, it says.

Q. Right. And they followed them for 31 years after that, right?

I'll show you.

Let's go down to about -- four sentences down, "Follow-up ended when a subject developed cancer or when

a subject was no longer in our membership database or on November 30th, 1995, whichever first occurred."

A. Okay. It says that.

Q. So they started following them in '76, and they followed them right through 1995, right?

A. That's what it says.

Q. Let's continue. Let's go back to the first page.

Let's go to the first sentence. Keep going.

"Cancer is known to have many environmental causes."

That's true, isn't it?

A. Yes.

Q. Okay. "Because married couples share at least their home environment, usually for many years, the study of spousal aggregation of cancer might provide clues to unsuspected etiologic factors."

That's true, isn't it?

A. It says that, yes.

Q. Sure. And how many years did Al and Alberta Pilliod share a home?

A. Many years. I don't recall when they were actually married.

Q. A long time?

A. A long time.

Q. Okay. Let's look at what they find.

Table 1, if we could, please.

In this only marital concordance study done after Roundup was on the market, they show, quote, an association of cancer occurrence within married couples, right?

A. Yes.

Q. And let's go down and look at what they find for non-Hodgkin's lymphoma.

Non-Hodgkin's lymphoma. All right? That's what we're dealing with here, isn't it, non-Hodgkin's lymphoma?

A. Well, we're dealing with a very specific entity within that. We're dealing with diffuse large B-cell lymphoma. There are at least 60 different kinds of lymphoma.

Q. This will be the easiest question I ask all day. Did Al Pilliod have non-Hodgkin's lymphoma?

A. Yes, he did.

Q. Did Alberta Pilliod get non-Hodgkin's lymphoma four years later?

A. Yes, she did.

Q. Okay. Non-Hodgkin's lymphoma, they find a relative risk of 2.78, right, Doctor?

A. That's what it says, yes.

Q. Statistically significant, right, Doctor?

A. Yes.

Q. It's almost tripling of the risk, isn't it?

A. That's what they show.

Q. All right. And, in fact, they point out that "one of the couples with non-Hodgkin's lymphoma had lived in Mexico for many years" --

If we could pull that out. Thank you.

-- "for many years, and both husband and wife were said to have been exposed there to pesticides, a suspected cause of lymphoma."

Do you see that?

A. I do.

Q. Okay. So of all the cancers they looked at, they only found concordance in four types, right?

A. Well, they found concordance in follicular lymphoma in the couple that you just described.

Is that what you mean?

Q. Well, I'll go to -- if we could, to page 2416.

In the discussion section, second paragraph, "For all cancers combined, there was no evidence of spousal concordance. The only cancer sites which statistically significant concordance was noted were the tongue" -- right?

A. Yes.

Q. -- "stomach and non-Hodgkin's lymphoma," right?

A. That's what they say, right.

Q. And that makes sense because, if they both smoke, they could both get tongue cancer, right?

A. It's conceivable. Sure.

Q. Sure. All right. Okay.

Well, how come the Friedman study didn't get on your blowup -- we could go back to the ELMO -- of marital concordance studies?

A. Simply because these people did not have the same disease that Mr. Pilliod did. Lymphoma is not one disease; it's at least 60 different diseases.

Q. Gotcha. Two more.

I'm going to do this real quick because the jury will start throwing stuff at me if I don't. But the Zhang study. All right? Just last topic.

Exhibit 2333, you've read it, right?

A. I did.

Q. It came out in February, right?

A. Yes.

Q. And you told us that what the EPA did was important to you. Remember that general conversation?

A. Yes.

Q. You know that -- I'll hand you a copy of it,

2333. Here you go.

A. Thank you.

Q. Yes, ma'am.

The jury could probably tell us all three authors right now, but I need to go over it anyway.

These three authors were on the scientific advisory panel for the issue of Roundup and non-Hodgkin's lymphoma, weren't they?

A. Yes.

Q. Okay. And after they were on that panel, they wrote a really long report. It looks like it's over 50-some, 60-some pages, right?

A. Yes.

Q. Peer-reviewed?

A. Yes.

Q. Okay. And it was published in February 2019.

Again, one of the scientists -- that's where I got Oakland and Berkeley mixed up. Right here in Berkeley, right?

A. Yes.

Q. Right down the road.

And what they say -- just to cut to the chase, if we could go to page 3. All right. "We documented." If we could start there.

"We documented further support from the

studies of malignant lymphoma incidence in mice treated with pure glyphosate as well as potential links between glyphosate-based exposure and immunosuppression," right?

A. That's what it says. I disagree.

Q. They saw a potential link between glyphosate-based formulas and the cause of immunosuppression in some.

That's what they report.

A. They did. I disagree.

Q. I know you do.

And they report genetic alterations that are commonly associated with non-Hodgkin's lymphoma, right?

A. That's what they say, but I disagree.

Q. I know you do. Okay. All right.

"Overall, in accordance with evidence from experimental animal and mechanistic studies" -- and again, just to remind ourselves, you didn't look at the animal or mechanistic studies before you wrote your report in this case, right?

A. I have reviewed them carefully at this point.

Q. At this point, right.

A. Yes.

Q. Okay.

A. And to some extent when I wrote the paper. I had more months. I read it more, read it over. But I

read them before.

Q. What these scientists who were on the SAP and attacked by the EPA for that and gone on to do their own study say, "Overall, in accordance with the evidence from experimental animal and mechanistic studies, our current meta-analysis of human epidemiologic studies suggest" what?

A. Do you want me to read it?

Q. Please.

A. "Suggest a compelling link between exposure to GBHs and increased risk for NHL." That's what it says.

Q. And you and I can agree that they spent a lot more time looking at this issue than you have.

MR. ISMAIL: Objection, Your Honor.

THE COURT: Argumentative, Counsel.

BY MR. MILLER:

Q. Let's look at a couple more points here, and we'll move on. All right.

Let's go to page 32 on the bottom right.

On that page, these scientists, who studied this issue, say -- regarding immunosuppression, right? They talk about immunosuppression. "Strongest factors known to increase non-Hodgkin's lymphoma are congenital and acquired states of immunosuppression. Several studies suggest that glyphosate alters the gut

microbiome, cytokine IFN-y and IL-2 production."

These changes could impact the what?

A. It says, "These changes could impact the immune system, promote chronic inflammation, and contribute to susceptibility of invading pathogens, such as H. pylori."

Q. So page 34. Almost done. The last sentence these authors say, quote, the overall evidence.

Do you see that?

A. I do.

Q. "The overall evidence from human, animal, and mechanistic studies presented here supports a compelling link between exposures to glyphosate-based herbicides and increased risk for non-Hodgkin's lymphoma."

You disagree with them?

A. Yes, I do. I certainly know that they said that, but I do disagree.

Q. Okay. Okay. All right.

Well, so I don't know if you looked at the Leon study that came out since we started this trial.

A. Yes.

Q. Can we agree, without pulling the study out, that, in fact, it shows a statistical increased risk of non-Hodgkin's lymphoma for diffuse large B-cell lymphoma?

A. No, I think we should look at it.

Q. All right. Let's look at it. Here we go. All right. All right. Here we go. Exhibit 294.

A. Thank you.

Q. Yes, ma'am.

You reviewed it, right?

A. Yes, I did.

Q. Let's take a look at it real quick, then. We don't need -- we know what it's about. Let's go to Table 2.

This is a table where they talk about ever use. Just ever use, not 1400 times, but ever using any one of these 14 pesticides. And they tell you what they found. If you go down to glyphosate, right?

A. Yes.

Q. For glyphosate, ever used it, 36 percent increased risk, right?

A. That's what it says, but I don't agree with the methodology that led to that statement.

Q. All right. You can agree that this is a peer-reviewed paper, right?

A. Yes, it is.

Q. By one, two, three, four -- 17 scientists?

A. Yes.

Q. Who are all published in the area of pesticide

and its causes on non-Hodgkin's lymphoma?

A. I disagree that we actually know who was exposed to glyphosate and who was not exposed.

Q. I'm sorry. I didn't hear you.

A. I disagree based on the fact that we don't -- you said we agree that we know the people exposed to glyphosate and this is what the outcome was, and I don't agree that we know who was actually exposed to glyphosate.

Q. All right. Have you looked at the Lamure paper that came out while Dr. Nabhan was here visiting us?

A. Which one? I'm sorry.

Q. Lamure study. It's Exhibit 3104.

Just real quick. While Dr. Nabhan -- the same scientists, some of them who were on this last paper -- at least Dr. Baldi, published in JAMA.

That's a good journal, right?

A. Yes, it is.

Q. It's is Journal of the American Medical Association?

A. Yes.

Q. All right. And let me get you a copy, and just a few questions, and we'll wrap up. Exhibit 3104.

A. Thank you.

Q. All right.

MR. MILLER: Permission to publish. It's been previously published.

THE COURT: This has been previously published?

MR. MILLER: Yes, it has.

THE COURT: What hasn't?

MR. MILLER: Right?

THE COURT: I'm sorry.

BY MR. MILLER:

Q. All right. This paper, published April of '19, Journal of American Medicine. Have you reviewed it before?

A. Yes, I have.

Q. What it tells us is -- this is kind of a segue into damages, but I want to ask you about this first.

It says, "Professional use of pesticides is a risk factor for non-Hodgkin's lymphoma."

A. That's what it says.

Q. It's true, isn't it?

A. Some of them, yes.

Q. Yeah. And one of the professions it talks about is gardening.

Are you aware of that?

A. Yes.

Q. The heart of what they're really studying here in this paper that came out two weeks ago is whether people who get non-Hodgkin's lymphoma from exposure to Roundup have as good a prognosis as people who get their non-Hodgkin's lymphoma from some other source.

That's generally what they're inquiring about, right?

A. Yes.

Q. And, unfortunately, they find that it's more refractory; that is to say, it simply doesn't do as well as people who get their non-Hodgkin's lymphoma from some other source.

A. Well, that's what it says. But, luckily, Mr. Pilliod didn't read this. He's done really well.

Q. He has. And you and I -- we'll all keep our fingers crossed. His odds of getting non-Hodgkin's lymphoma again are small. We both agree with that, right?

A. Yes.

Q. Great. We hope that happens.

But, I mean, let's not kid ourselves. His whole body was riddled with non-Hodgkin's lymphoma, right?

A. He had Stage 4 in his bone, bone marrow, and lymph nodes.

Q. Right. It was in his bones. It was in his bone marrow. It was in his lymph nodes. Right?

A. Yes.

Q. And he had to have what we call R-CHOP, right?

A. He had R-CHOP, chemotherapy and immunotherapy.

Q. Yes, ma'am. This is not something you would give somebody unless they were -- they needed it, right? You don't like to give R-CHOP to people?

A. I like to give it because it is capable of curing malignant diseases that are very difficult. So I love R-CHOP, to be very honest with you.

Q. I think you misunderstood my question. I love it too, and I think Mr. Pilliod loves it more than any one of us.

I didn't say I didn't like it. I said there are risks to drugs, and there's big risks to these drugs, but you have to do it, right?

A. Absolutely. Sure. You're right.

Q. What are some of the risks of R-CHOP?

A. Well, one could have lowering of the red blood cell count. You could be anemic, and you might need a transfusion.

The concept is with the chemotherapy, it's very nonspecific. It kills any cell that divides quickly. So the bone marrow cells, red cells, platelets

that make your blood clot, white cells that prevent infection, they will all go down because they divide very, very fast. Hair cells divide fast enough that they're going to be affected by the chemo, and that could be a side effect. A drug called Oncovin can cause peripheral neuropathy, numbness and tingling in the fingers and toes.

There are many side effects of each of these drugs.

Q. Sure. But if he didn't take R-CHOP, he would have died?

A. I expect that he would, statistically.

Q. Sure.

And I know Mr. Pilliod's neurologic problems, they're not at all related to the chemotherapy. Nobody is trying to say they are, right?

We've looked at some records that show he had some problems beforehand, right?

A. Yes.

Q. Sure. But there is this thing called chemo brain, isn't there?

A. Yes, there is.

Q. Sure. If I were to go to your website, that is City of Hope, I would be able to learn about chemo brain, right? It's on your website.

A. Yes.

Q. Explain to us what chemo brain is.

A. It's kind of interesting because there's no real definition of what chemo brain is. During chemotherapy, the patients can -- certainly some drugs directly affect neurologic function in a certain sense. Prednisone. Some people go kind of goofy. They don't think necessarily properly on prednisone.

So during chemotherapy or immediately after, there might be a sense that you're not just quite the same. Not like a stroke or a seizure; you're just not quite the same.

And in time, that basically goes away. There was one story published, actually, with R-CHOP looking carefully at neurologic function and this chemo brain. And they found in that particular study no evidence of change before or after.

I think the most important is that Mr. Pilliod had significant brain dysfunction that was proven way before he ever got the chemotherapy.

Q. You say "significant." Was Mr. Pilliod able to sale to Maui and back by himself?

A. I believe he was.

MR. MILLER: Listen, I thank you for your time, and I thank you for your patience.

THE WITNESS: Thank you very much.

THE COURT: Redirect?

MR. ISMAIL: Yes, Your Honor.

REDIRECT EXAMINATION

BY MR. ISMAIL:

Q. Okay, Dr. Levine. We'll get you back home treating your patients here momentarily.

A. I'm getting nervous.

Q. Okay. I'm going to go essentially in reverse order from what Mr. Miller covered.

Any indication you saw in the medical records that suggest this concept of chemo brain was operating with respect to Mr. Pilliod?

A. Absolutely not. He had several episodes of brain trauma starting when he was young. He had concussions. He lost consciousness. We know from the football players and so forth that that in itself is an issue. He had much more than that. No, this is not chemo brain.

Q. Did you look at medical records from as recently as last year where his physicians are working him up for his complex epilepsy and what that is doing to the structure of the brain?

A. Yes, I've seen that.

Q. What was the results of that workup?

A. The result is they believe that his seizure disorder, his abnormalities in the temporal lobes in his brain are a result of the herpes simplex infection repeated over time; that the major cause of the neurologic dysfunction related to five different episodes of very severe brain infection, one of which put him comatose for an entire month, apparently.

Q. Were those before he ever had non-Hodgkin's lymphoma?

A. Those were way before he ever had non-Hodgkin's lymphoma.

Q. So Mr. Miller asked you about the Leon paper that came out recently.

A. Yes.

Q. And he directed your attention to Table 2.

A. Yes.

Q. I want to go over some of the information he didn't show you. I believe this is the data from that table. Okay?

A. Yes.

Q. So they have a column here for non-Hodgkin's lymphoma malignancies overall.

A. Correct.

Q. Do you see that?

And with respect to glyphosate -- and this was

data published last month -- what was the hazard ratio for glyphosate?

A. The hazard ratio was less than 1. It was 0.95.

Q. And what is the significance, if any, to you that this large recent study showed a hazard ratio of .95?

A. It means to me that this large study has not been able to show a statistically significant relationship between glyphosate use and subsequent development of non-Hodgkin's lymphoma.

Q. And with respect to the diffuse large B-cell number that he directed your attention to, is that even statistically significant by traditional statistical measures?

A. No, it isn't. The confidence interval starts at 1.00. So the most you can say is it's gray or equivocal. It's a soft finding.

Q. Have you looked at other data on DLBCL and, in fact, showed the jury this morning, in addition to the Leon study, that looked at this question of DLBCL?

A. Yes, I have.

Q. Is there any indication of an increased risk with that subtype?

A. No, not at all. Eriksson was mentioned as a

reference in one of the papers that I was asked, and that paper was very clear. There was no statistical incidence -- increase of diffuse large B-cell lymphoma.

Q. With respect to the methodology of looking at epidemiology, would you just look at one number to the exclusion of all the other studies that you reviewed?

A. No. These are really complicated areas. I feel badly for anyone who has to look at this. It's hard. And, really, you have to look at the entirety of everything. Any one study doesn't mean a lot. You have to look at all of it together, decide which was best done, which was most valid, which wasn't. And it's the entirety that gives you the idea of what the truth might be.

Q. Was that your approach here?

A. Yes, it was.

Q. Mr. Miller asked you about the Zhang paper?

A. Yes.

Q. Now, the Zhang paper, is -- is that new data or is that looking back at older data?

A. It's looking back at older data. It's not new data.

Q. You talked about this morning the importance of looking at adjusted data when adjusting for other pesticide use?

A. Yes.

Q. With respect to some of the conclusions that Mr. Miller spoke about, do the Zhang authors limit themselves to just adjusted data in their review?

A. No. It's mixed up. Some of this is adjusted, some isn't, and it makes the whole thing less than I would want for real validity.

Q. Do the Zhang authors include the entirety of the Agricultural Health Study?

A. No. It says it does, but, really, the Zhang went up to -- I believe it was 2010, 2011. So it's a portion. It wasn't the updated Agricultural Health Study database set.

Q. Do the Zhang authors include the Leon finding of .95 that is reflected here?

A. Yes.

Q. I'm sorry. Does the Zhang study include the updated Leon paper that just came out last month?

A. Oh, I see. No. I'm sorry.

Q. So continuing forward, counsel asked you some questions about the spousal concordance paper, Friedman?

A. Yes.

Q. Do you recall that?

A. I do.

Q. There was what? Four couples in that paper?

A. Yes, there was four couples.

Q. And the studies that you talked about with the jury showing no spousal concordance, were those larger analyses?

A. Yes. Those were large population-based studies.

Q. With more couples that they were analyzing?

A. Yes, with many more couples.

Q. Is there increased reliability when you're looking at larger and larger pools of data than, say, a four-person or four-couple study like Mr. Miller was talking about?

MR. MILLER: Your Honor, I object. Leading.

MR. ISMAIL: I'll rephrase, Your Honor.

THE COURT: Rephrase, please.

MR. ISMAIL: Sure.

BY MR. ISMAIL:

Q. Doctor, based on your experience reviewing epidemiology studies, can you tell us about the importance of study size when interpreting the results?

A. If you're looking for a rather unusual outcome, you really need large numbers to be able to see it and to find it.

Q. And with respect to the papers that he showed you, I think he only wrote the start years on the slide.

Do those papers continue to follow those patients over time?

A. Yes. They continued to follow the patients over time after the time that glyphosate would have been available.

Q. Thank you.

Now, he asked you about the SEER data, and he gave you -- if I can find my copy. Uh-huh.

So first of all, you -- this was the graph that you talked about with the jury?

A. Yes.

Q. Where did the line that you reported for non-Hodgkin's lymphoma come from?

A. That comes from the SEER database and the National Cancer Institute as well.

Q. And is this how the SEER database reports their data?

A. Yes. They report it per 100,000 persons.

Q. Is there any dispute in the lymphoma scientific community that the rate of non-Hodgkin's lymphoma has plateaued in this country?

A. There's no question at all. It was a very interesting finding and exciting one. It has clearly plateaued.

Q. And with respect to the usage of glyphosate,

where was that source from?

A. I got that from the data from the EPA. They gave year by year the amount of glyphosate that was used.

Q. And is this exactly how they report that data?

A. Yes. That's why I made it that way.

Q. What do you think about what Mr. Miller did, changing the scale to change the slope of the curves?

A. I don't think that's valid. You can't do that. That's just not valid. You're changing what the data showed and trying to make it look a different way.

This is how it was reported. And so that's how we -- I wanted that to be shown in that way.

Q. And so when -- is this a fair look at the incidence rate of non-Hodgkin's lymphoma, the one that Mr. Miller showed you?

A. I need to see it again. I'm sorry.

Q. Sure. I can put it on the ELMO.

So when Mr. Miller changes the scale here, is this a -- did it come up?

THE COURT: It did. It doesn't want to stay up.

BY MR. ISMAIL:

Q. By changing the scale, Doctor, is this a fair look at the data?

A. Well, I really don't think so. Could you pull it up a little bit on the -- so I can see the bottom of the slide? I just want to see the years.

Yeah, I don't think it's a fair representation. One small point is that it looks to me as if the incidence of lymphoma is slightly going up again. I don't believe that's true. That's one thing.

But, again, if you're looking on a scale, it should be from 0 to 100. That's what it's supposed to be. You can change the way it looks, but the data are continuous, whether you graph it or not. What's happened in lymphoma is a tremendous increase early in the '80s, leveled off a bit in the '90s. By the 2000s, it's flat; it's not increasing.

But at the same time -- just forget the curve. At the same time, the use of glyphosate in the United States is growing. It's more and more and more.

So however you want to put that on a graph, lymphoma is not increasing and glyphosate use is.

Q. Thank you, Doctor.

Counsel showed you this article by Song on the risk of developing other cancers following a nonmelanoma skin cancer.

A. Yes.

Q. It's Exhibit 3157. And I just want to direct

your attention to what these authors say in their conclusion here.

Trying to do this upside down is never a good idea.

MR. EVANS: Your other left.

BY MR. ISMAIL:

Q. Let me back up here. We've --

Actually, can you pull up Exhibit 3157. Turn to page 7 of the article and call up the last paragraph above "References."

Does it say "We cannot estimate the recurrence rate of nonmelanoma skin cancer or subsequent cancer risk among people with multiple nonmelanoma skin cancers because we only recorded the first report of each type of skin cancer in both cohorts"?

A. Yes, that's what it says.

Q. Does that describe Mr. Pilliod?

A. No. He had 22 -- well, he had 21 nonmelanoma skin cancers; he didn't have one.

Q. And when you were looking at the data, did you report on studies that showed what happens if you have multiple skin cancers and the risk of non-Hodgkin's lymphoma?

A. Yes. As the numbers of basal cell cancers, as an example, goes up, the lymphoma risk goes up. And

it's not shown here. But, after 12 basal cells, it was even a higher risk in that particular paper.

Q. And, Doctor, counsel asked you about red hair and blue eyes.

By any measure of those additional demographic factors, is 22 skin cancers an unusual finding?

A. Yes, it is an unusual finding.

Q. Now, he asked you about CD4 count.

A. Yes.

Q. Is that the only measure that you would look to to understand whether a patient's immune system is functioning?

A. No. So the CD4 count would tell me the number of CD4 cells. It doesn't tell me the function of those cells, just the number. And his number was normal.

But the more important issue is that there are many, many, many components of the immune system. And the CD4 cell, the T4 cell, is only one example.

So I will say that the number of his T4 cells is normal. I don't know about their function. And I don't know that he's ever been studied for any of the other immunologic abnormalities that might be really underlying this.

Q. Okay. So counsel -- almost done here.

Counsel asked you about this Chang paper.

Do you recall that?

A. Yes.

Q. So, first of all, he directed you to a particular sentence here about B-cell lymphomas.

Do you recall that?

A. Yes, I do.

Q. What do these authors say immediately under that?

A. They say "Bias and confounding may account for the observed associations." Right in the abstract, they're saying that this could be due to chance or bias alone.

Q. And what do these authors say on their final conclusion based on their review of this meta-analysis data?

A. Their conclusion is "Thus a causal relationship has not been established between glyphosate exposure and risk of any type of lymphohematopoietic cancer"; i.e., lymphoma would be in that category.

Q. And he asked you about B-cell lymphoma in this study. But did they also report on diffuse large B-cell lymphoma in this study?

A. Yes, they did.

Q. And if you turn to page 15, what was the data they reported here for diffuse large B-cell lymphoma?

A. They reported that, for diffuse large B-cell lymphoma, the meta RR was 1.1, but the 95 percent confidence interval showed that this was not significant.

Q. And so when we're talking about Mr. Pilliod's risk, Doctor, is it more important to look at the type of cancer he had, diffuse large B-cell, or this heterogeneous cancer called B-cell lymphoma?

A. I said it at the beginning. And I can't even emphasize it enough. Lymphoma is not one disease. And to say lymphoma is associated with X, Y, Z, that's one piece of information.

But he has a very specific disease: diffuse large B-cell lymphoma. And it's important to know that these are different diseases. And we need to concentrate on what Mr. Pilliod had.

Q. Thank you, Doctor. A couple last topics real quick.

Mr. Miller asked you some questions about surfactants and their role in formulating a product. You told us that Roundup is an herbicide; is that correct?

A. Yes.

Q. So, with respect to the role of surfactants and what they do, would that relate to the efficacy or

the effectiveness of it as a herbicide?

A. Yes, because it can get into the leaves of the plants, for example.

Q. Is that what you were referring to earlier about getting into the body of the leaf?

A. Yes. That's what I meant, the leaf.

MR. MILLER: Objection. Leading.

BY MR. ISMAIL:

Q. Can you talk to us about that?

A. It's used in agricultural use. And it stops the plants from making certain proteins, amino acids. And the ability to get this into the plant leaf would be -- or into the plant itself would be important for that product.

Q. Does it have anything to do with getting it into a person?

A. No. It's hard to get through the skin of people, actually, glyphosate.

Q. Okay.

So, with respect to what Mr. Miller wrote up here -- I think he wrote it up here.

I have no idea whether he wrote it up here, but thank you for that clarification.

Last topic, Doctor. With respect to the things that Mr. Miller wrote up here on this chart -- I

don't know if you can still see it -- you wrote down a couple of the factors he talked about on direct examination.

Did he leave off a very important risk factor that you talked with the jury about?

A. Immunity, I guess. But it's hard for me to read. I can't read his handwriting.

Q. I can't either. I think that says "obesity," actually.

A. Okay.

Q. But let me ask it this way: What was Mr. Pilliod's most significant risk factor for developing NHL?

A. There's just no question in my mind. Taking the whole totality of this, his immune system was clearly abnormal. He had an autoimmune disease where his own immune system thinks that his colon is foreign to him. He had severe infections of the brain, five of them, due to the virus that causes cold sores in most of us. He had 22 skin cancers. And multiple skin cancers is a risk factor for eventual development for lymphoma.

All of these working through the immune system in a sense.

Q. And Mr. Miller asked you whether it's true that, anytime people develop the cancer, there's some

failure of the immune system to protect that person.

A. Yes.

Q. Is it something different when we're talking about Mr. Pilliod with respect to his immune system?

A. Yes. Mr. Pilliod's immune system has been abnormal at least since his 20s, developing diseases that just don't occur in -- over and over again, and so forth.

Age was mentioned. And age is a risk factor for cancer. Our immune systems, all of us, the immune system gets weaker as we age, not enough to cause opportunistic infections and so forth, but weak enough to get some of us in trouble with cancer and not recognizing the cancer.

Q. And you told the jury that, because Mr. Pilliod doesn't have one of the known causes of cancer, you'd characterize it as idiopathic.

A. That's correct.

Q. And what percentage of DLBCL patients have idiopathic cancers?

A. 90 percent. Some people say as high as 95 percent.

Q. So, with respect to what he wrote here, "Dr. Levine did not know what caused Al Pilliod's NHL," where would that put Mr. Pilliod in the population of

patients who develop his type of cancer?

A. It would put him within 90 percent of all patients with lymphoma. None of us know. We don't know what that cause is in the vast majority, in 90 percent of people.

And that's true with him. His abnormal immune system -- whatever caused this, his abnormal immune system would have truly allowed this to occur, without question.

Q. Would that increase his risk?

A. It would increase his risk substantially.

MR. ISMAIL: Thank you.

Your Honor, no further questions.

MR. MILLER: Real quick.

RECROSS-EXAMINATION

BY MR. MILLER:

Q. It sounds like you think Mr. Pilliod is more susceptible to getting non-Hodgkin's lymphoma because of his immune system.

MR. ISMAIL: Objection.

THE COURT: I'll allow it.

BY MR. MILLER:

Q. So are you saying that his immune system problem was a substantial contributing factor to causing non-Hodgkin's lymphoma or not? Or are you saying you

don't know the cause?

Which is it?

A. I'm saying we don't know the cause in Mr. Pilliod or in the other 90 percent of people who have diffuse large B-cell lymphoma. We don't know.

I don't know what his specific mutation -- his specific type of mutation was. I don't know what it was.

I do know that his very abnormal immune system would not have allowed him to recognize the first malignant cell as foreign, and it would have been allowed to continue.

Q. Have a safe trip back to Los Angeles.

A. Thank you very much.

THE COURT: Thank you, Dr. Levine.

THE WITNESS: Thank you.

THE COURT: So, ladies and gentlemen, we are -- I'm sorry.

We're done for the day. Hold on just a second.

We're done for the day. We are not going to be in session tomorrow because I need to talk to the lawyers about a number of issues to get prepared for closing and submission of final evidence. So we will not be in session tomorrow. We will start at 9:00 on

Wednesday morning with jury instructions and closing arguments.

Keep in mind we will not be here on Friday, even though you will begin deliberating before Friday, because we have that day off. I think we discussed that. So we're not going to be in session this Friday. So we will be in session Wednesday and Thursday.

So, while we have completed the evidence, you have not heard any jury instructions. I don't want you to begin thinking about all of the evidence yet until you've heard the jury instructions and the closing arguments of both plaintiff and defense counsel. So hold on for one or two more days, and then you can talk away about the evidence.

I'll see you on Wednesday morning at 9:00. Thank you.

(The following proceedings were heard out of the presence of the jury:)

THE COURT: How about 10:00 tomorrow morning? Does that work for everybody to start working on finishing up the jury instructions? I assume you guys need to meet and confer about evidence or to make final decisions about what's going to be admitted?

MR. EVANS: Yeah, there are a few defense exhibits that we've been back and forth on we probably

will have to raise with Your Honor. There's not too many, I don't think.

MR. ISMAIL: So we didn't formally rest, Your Honor; but, if we did, it would be subject --

THE COURT: You didn't actually rest. I just was talking to the jury about kind of where we were in terms of --

MR. ISMAIL: Subject to the admission of exhibits.

THE COURT: Sure. No, no, I'm not resting for you.

All right. So I'll see you guys tomorrow morning at 10:00.

MR. WISNER: And I think, Your Honor, based on your ruling today, we don't anticipate calling any rebuttal testimony. We're ready to go for closings.

THE COURT: Why don't I get an estimate on the time -- are you closing?

MR. WISNER: Yes, your Honor.

THE COURT: I want to get an idea of how long your closing is going to be. If we're really going to finish closing on Wednesday -- and I don't think the jury instructions are going to take too, too long, because there aren't that many. We'll probably have to have some hard stops. So figure out exactly how much

time plaintiffs will get and defense will get.

MR. WISNER: And for budgeting time, should we assume an hour for housekeeping and jury instructions?

THE COURT: You mean tomorrow morning?

MR. WISNER: Wednesday morning.

THE COURT: I think housekeeping needs to happen tomorrow. Hopefully, we're going to start with jury instructions right away on Wednesday morning.

MR. WISNER: Okay.

THE COURT: So once we finish up, I'll have copies of the instructions. Once we get a final set, I guess what will have to happen is, once I make all my final rulings, somebody will have to clean them up and send them back. I will make copies for the jurors.

MR. MILLER: We were going to request -- there's been a large demand for public attendance at the close -- if it was possible to consider a larger courtroom for closing argument.

THE COURT: There's no larger courtroom available. I'll try to set up a few extra chairs like I did for opening. But that's the best I can do.

MR. MILLER: Understand.

THE COURT: Sorry.

MR. MILLER: Thank you, Your Honor.

MR. WISNER: Thank you.

THE COURT: I was going to say something else, but now I've forgotten. I'll remember tomorrow. We'll chat tomorrow morning.

(Proceedings adjourned at 3:58 p.m.)

State of California)
)
County of Alameda)

I, Lori Stokes, Court Reporter at the Superior Court of California, County of Alameda, do hereby certify:

That I was present at the time of the above proceedings;

That I took down in machine shorthand notes all proceedings had and testimony given;

That I thereafter transcribed said shorthand notes with the aid of a computer;

That the above and foregoing is a full, true, and correct transcription of said shorthand notes, and a full, true and correct transcript of all proceedings had and testimony taken;

That I am not a party to the action or related to a party or counsel;

That I have no financial or other interest in the outcome of the action.

Dated: May 6, 2019

Lori Stokes, CSR No. 12732