Volume 8

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UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

Before The Honorable Vince Chhabria, Judge

EDWARD HARDEMAN,	)	
	)	
Plaintiff,	)	
	)	
VS.	)	**NO. C 16-00525 VC**
	)	
MONSANTO COMPANY,	)	
	)	
Defendant.	)	
	)	

San Francisco, California
Tuesday, March 5, 2019

TRANSCRIPT OF PROCEEDINGS

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I N D E X

Tuesday, March 5, 2019 - Volume 8

E X H I B I T S

Tuesday - March 5, 2019 **8:28 a.m.**

P R O C E E D I N G S

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(Proceedings were heard out of presence of the jury:)

THE COURT: Good morning, everybody. We lost our third juror. She -- turned out that she had the flu. It was not food poisoning. So we are down to six. And we are ready to call in the jury.

Does anybody else -- does anybody have anything to discuss briefly?

MR. STEKLOFF: No, Your Honor.No Your Honor.

MS. WAGSTAFF: No, Your Honor.

THE COURT: Okay. Go ahead and bring them in. Mr. Hardeman is coming on first?

MS. WAGSTAFF: Actually, we are going to play the clip of Dr. Portier.

THE COURT: Oh, yeah, okay.

MS. WAGSTAFF: It is about four minutes.

Your Honor, we may move to have Exhibit 168 entered. Can we reserve to do that outside the presence of the jury because there may be some --

THE COURT: Which exhibit is that?

MS. WAGSTAFF: It's the Parry report. You said we can discuss it later. I just didn't want to have attorney argument in front of the jury.

THE COURT: I think we decided that wasn't going to be admitted, but we can certainly discuss it.

MS. WAGSTAFF: Okay.

(Proceedings were heard in the presence of the jury:)

THE COURT: Good morning, everyone. I see your group has gotten a little bit smaller. Thank you for being here, and we are ready to resume today.

The Plaintiffs are going to play an additional clip of the testimony from Dr. Portier's testimony in Australia. That is the first thing that is going to happen today.

MS. WAGSTAFF: The Plaintiff calls Dr. Portier, and it is only about four to five minutes.

THE COURT: Okay. Plaintiffs want to call their next witness.

MS. MOORE: Yes, Your Honor. The Plaintiffs call Edwin Hardeman.

EDWIN HARDEMAN,

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

THE CLERK: For the record, please state your full and last name and spell both of them.

THE WITNESS: My first name is Edwin. And my last name is Hardeman. That is E-D-W-I-N, and the last name is Hardeman, H-A-R-D-E-M-A-N.

THE CLERK: Thank you.

THE WITNESS: You are welcome.

DIRECT EXAMINATION

BY MS. MOORE

Q. Good morning, Mr. Hardeman.

A. Good morning.

Q. Have you ever testified in court before?

A. No, I haven't. I'm a little nervous, I must admit. This is my first experience so please bear with me.

Q. We will get through it. I'm going to ask you some questions this morning about your use of Roundup, and I want to start at the very beginning.

How did you learn about Roundup?

A. Well, we first became aware of Roundup when we moved to the Mendocino coast and purchased a property in the town called Gualala. We wanted to move closer to my brother who lives up on the coast. So after visiting the area for several months, we decided to move up there.

Q. Did you say Gualala?

A. Yeah, G-U-A-L-A-L-A. It is on the Mendocino coast. The house needed some work, and there was a lot of outside work to do also. We decided that we wanted to start on the outside so we were trying to figure out how to deal with the weeds and the other growth that was on the property. Looked in the local paper, the Coast Observer, to see if there was anybody in there to help us; and we found a worker in there and we called him up

and he came out. And he had this device with him, and he started spraying the weeds. So we asked him what he was using. He said it was Roundup.

So after that, we figured, Well, maybe we can do this ourself. We are kind of do-it-yourselfers. We found out where we can purchase it. And got the pump-up sprayer and what we would need to apply it, and we were on our way.

Q. And what year was that?

A. It had to be -- we moved up there in 1985. So I'm thinking it was 1986 when we really started getting into applying the Roundup.

Q. So 1986 is the first time -- well, first of all, did you use Roundup yourself?

A. Yes.

Q. So when was the very first time you used Roundup?

A. Well, I used it in -- I would say it was 19 -- either late 1985 or early 1986 to my recollection I started spraying it.

Q. And where would you buy the Roundup that you would use back -- let's stay in 1986 -- but where do you remember buying that?

A. Well, we looked around locally to see if we could purchase it, and there is a hardware store up there in town. It is possible we bought it there or one of our trips into Santa Rosa, which is a couple hours away, where we were doing our shopping up in -- you know, of food and supplies for the house,

one of the hardware stores in Santa Rosa. It would be Yard Birds, which was a home improvement store, or Friedman's is kind of a ranch supply hardware store in Santa Rosa.

Q. And so were you able to buy that right off the shelf at Yard Birds or Friedman's?

A. Yes.

Q. You keep saying "we." When you say "we," who are you talking about?

A. I'm sorry. I mean my lovely wife, Mary, who is sitting there. Together we would do this.

Q. Okay. Do you know what type of Roundup that you would buy back in 1986?

A. Well, we -- we bought this concentrate so we could dilute it with water and get more yield out of it. Roundup Concentrate.

Q. And so why did you -- why did you pick the Roundup Concentrate versus Roundup?

A. Well, because you could get more yield. You could mix it and, you know, get more volume or yield out of the product; and it was less expensive that way.

Q. And who would actually mix the Roundup?

A. I mixed it.

Q. And can you tell the ladies and gentlemen of the jury how you would mix the Roundup?

A. Yeah, so -- in a measuring -- I would get my pump-up

sprayer and remove the plunger off of the top of it. Pour the Roundup into a measuring cup. I mean, 4 to 6 ounces per gallon, and I would pour it into the opening. And then I would start to put water into it with a hose slowly until it got to the first gallon indicator on the spraying device. And then I would put the next 4 or 5 -- 4 or 6 ounces in there and apply the rest of the water slowly. Sometimes it foams out over the top and you can get it on you.

Q. And so when you say "4 to 6 ounces," that is 4 to 6 ounces of what, Mr. Hardeman?

A. Of Roundup, 4 to 6 ounces of Roundup.

Q. And so in the device you are using, that is two gallons?

A. Two-gallon sprayer is what I would use, yes, that is the capacity.

Q. So that would be, what, 8 to 12 ounces of Roundup in the sprayer itself?

A. Approximately, yes.

Q. And did you use Roundup at your home in Gualala?

A. Yes, I did.

Q. And can you tell the ladies and gentlemen of the jury where you would spray the Roundup at your home?

A. Yes. We had, you know, a driveway road access off of Iverson Road. It was approximately 400 feet. It is a gravel driveway. And also the -- when you got to the house area, we expanded the parking area. That was another 200 --

approximately 200 feet.

So after we -- we had that graded and it was looking nice, and the gravel was put down. The weeds started popping up after the winter so I would have to spray that whole -- that whole driveway to kind of keep it nice and often into the field areas around the house, around the detached garage, into -- they had a little -- small little kind of recreation pond, and it would spray around the pond and into the rest of the property where it needed.

Q. And how often would you spray Roundup at the Gualala property?

A. I would walk that probably, once a month, you know, I would do that.

Q. And how long did you live at this property up on the Mendocino coast?

A. We would live there approximately -- I think we moved from there in 1988. So about three, three and a half years we lived there.

Q. So from 1986 when you started using Roundup until you moved in -- what was it -- 1988?

A. Yes.

Q. So from 1986 to 1988 how often did you use Roundup?

A. Well, I would probably do -- use it once a month, you know, from May -- I would start in May after the winter is finished when the weather is nice and temperatures come up,

like it gets warmer. I would go through the summer and into the early fall, you know, once a month, or September or October.

Q. And then you moved in 1988. Tell the jury where you moved to.

A. Well, we -- after living on the coast for three years, we found that -- it was a -- the work was a little -- it wasn't consistent employment and work was a little hard to find, so -- and we felt it was a little bit too remote for us. So we decided to -- we liked the Santa Rosa and Sonoma County area. So we started looking there and found another country property that was -- needed some work and we could get a good deal on it. So we moved to Forestville, which is right on the West Side Road out by the Russian River in the West County. Moved there in -- I think it was October of 1988.

Q. How old would you have been then?

A. I'm sorry.

Q. How old would you have been then?

A. 39, going on 40.

Q. And when you moved to the West Side Road property, did you use Roundup there?

A. Yes. There was a large piece of property, 56-acre property. It -- the terrain wasn't mild. It was steep, and it had beautiful, beautiful views. Needed a lot of work. There was a lot of overgrowth, and it was run as an exotic animal

preserve when we found it; but it was in disrepair. And a lot of the improvements that were there were rundown, so we came on the scene. We bought it. We negotiated a good deal and moved in October of '88 and started initiating our plans to bring it back to a level that, you know, we wanted to see it at.

Q. Let me --

MS. MOORE: And, Your Honor, if I can grab the binders, I apologize. I didn't do that at the beginning. May I, Your Honor?

THE COURT: Sure.

BY MS. MOORE

Q. Mr. Hardeman, if you could, turn to -- you will see in the binder there are tabs on the side. If you can turn to tab 23, please.

A. 23, okay.

Q. And I'm going to ask you, do you recognize this document?

A. Yes.

Q. Okay. And what -- can you tell the jury -- and we will show the jury in a second -- but what is this document you are looking at?

A. This is a reduced version of an original plot plan for the West Side Road property, 11995 West Side Road. It has got some surveyor marks there, monuments along the side.

Q. Is this -- hold on one second. We will get to this. Is this plot map a fair and accurate description of your West Side

Road property?

A. Yes.

MS. MOORE: Your Honor, may I have permission to publish this to the jury?

THE COURT: Any objection?

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Go ahead.

MS. MOORE: Thank you.

BY MS. MOORE

Q. Mr. Hardeman, I want you to explain to the jury a little bit more about what we are looking at. Would it be helpful for you to come down and use a blow-up to do so?

A. Sure.

MS. MOORE: Okay. Your Honor, would that be okay?

THE COURT: Sure.

BY MS. MOORE

Q. Mr. Hardeman, I'm going to have you come on down, and we will put this right here. Have you stand right here.

MS. MOORE: Your Honor, I'm sorry. I know you can't see it. It is exactly what is on --

THE COURT: What exhibit is it?

MS. MOORE: It is Exhibit 23. It is the exact same thing. I'm sorry.

THE COURT: That's fine.

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BY MS. MOORE

Q. We are kind of in tight quarters. Is that all right?

Okay. If you could -- Mr. Hardeman, if you could explain to the jury what it is that we are looking at in Exhibit 23.

A. Yes. This is -- the yellow, the part that is outlined in yellow, is the property line. And this is the -- what is the West Side Road. This is the entrance to the property. There is an easement here across these two properties here.

Q. What is this Parcel 2 here?

A. Parcel 2 is between the two houses there. There was a house here that was built in 2000. So before that house was built I was -- I had to use the --

COURT REPORTER: I can't hear him. I'm sorry.

BY MS. MOORE

Q. She is having a hard time hearing you. We are going to move this, figure out the best way to put it. I'm going to move it back here, so that way she can -- the sound is not blocked.

Just try to speak up if you can, Mr. Hardeman, so the court reporter can hear you. Thank you.

A. All right.

Q. So what is Parcel 2? Is that part of your property?

A. No. That's not part of our property. This is a property that accommodates two other houses. Actually, it is split in two now, two -- I think it is two 5-acre pieces below us.

Q. So where is your house at the West Side property?

A. Now, our house is represented right here. So it is about -- from the time we entered this off of the West Side Road, you come up the driveway, it's about 5 -- 4 or 500 feet, and there is a large parking lot here. There is a detached garage about 80 feet from the house, and it's a water tank behind it.

Q. I'm going to have you -- because I think she is having a hard time hearing you. I'm going to have you stand here so you can project your voice towards her. I think it might be a little bit better. There we go.

A. Okay. So to recap, this is the West Side Road. And you come up the driveway to our house, there is a big parking lot there; a lower parking lot and an upper parking lot. This is the south side, the east side. You would continue up the road -- it continues on up to the back -- the road takes you up to the back of the property. And there are some off-shoots, a couple hiking trails and service -- there is 22,000 gallons of water storage. This is a concrete tank, which I would spray and take care of the various poison oak growth, Scotch broom and weeds and other things that were growing.

Q. Let me ask you this: What is Scotch broom?

A. It's a plant that -- it has a little yellow flower on it, and it grows up wild and up through the road in the back. And unfortunately, you know, it accommodates ticks which hang onto

it and so on and so forth. So you can get them on -- I wanted to keep that under control.

Q. You mentioned poison oak. Was there poison oak on the West Side property?

A. Yes, there was prolific poison oak. It was well known in the '80s and '90s. They even have a poison oak festival. It was kind of a novelty little thing, but yeah.

Q. A poison oak festival?

A. Yeah, a poison oak festival. We used to have that. They discontinued that.

But, yeah, anyway, there was a lot of poison oak throughout the property. And I first encountered it when I started the project to clean up on the property. There was a lot of debris there. It was thrown over the side of the downslope part of the property. And I went to retrieve it so I could recycle it and get it out of there and clean it up. But when I was down there cutting brush and whatever, I -- when I came back up, I had poison oak all over my body. I had to go to a doctor twice and get a shot of Benadryl to clear it all up.

Plus my dogs were bringing it back in their paws. And the oil, you know, you can't see it. You don't know you are in it until you get it on you. You can contaminate yourself. Your tools get contaminated. Anyway, I just started off on this mission to go after it.

Q. And what is the size of this property that you and your wife own?

A. This was 56 acres. And it's a little less than three quarters of a mile long. And it's got beautiful, beautiful views and hiking trails and things that we --

Q. I'm sorry, Mr. Hardeman.

A. Yes.

Q. Explain to the jury, when we are looking at this, it looks flat. What was the terrain like? If you could just walk them through the property briefly as to how it looked when you-all lived there.

A. Yeah. It's a mild to steep terrain. So it would start to -- you know, an incline upwards. And it had some nice flat spots that you could enjoy in various spots of the property. The -- all the water system and everything in the back here is all on a gentle slope.

Q. Did you have your own water system?

A. Yeah. It was on a spring fed, a natural mountain spring. It was all gravity fed down to the house. The water was -- we had -- it is all based upon water storage. There is, like, 22,000 gallons of water. The largest being a 15,000-gallon tank. In the spring is -- we have a spring box up here, which takes the water from a short little fall off of a rock and collects it, and then distributes it to these other tanks. And the spring itself, the source of the spring, is further up. So

I would -- I would hike up in here to make sure all this was running right because you had to constantly keep an eye on it. If there is an interruption, you could lose your water.

Q. And then what you have got here, it says elevation 1302. What does that mean?

A. That is the highest. That is Black Mountain. So it would rise up to 1302 feet elevation, and go down 900 feet down the other side of the property. But I don't -- I wouldn't go up there very often.

Q. Okay. And you mentioned hiking trails?

A. Uh-huh.

Q. Where is the hiking trails on the property?

A. Well, the hiking trails are a little closer in. So there is -- as you walk up to the back of the property here, past the house and you start going into this area up here by the tanks, there was a trail that went off down to the creek, which we opened up and -- so we could utilize that. And we wanted to keep that spring. There is also other areas where you could hike up into, more gentle sloped areas. And up -- what is called the roundabout where you could turn around. And then up into -- there is a little more steeper areas where you could hike up into the beautiful redwoods that had a combination of second growth redwood trees, several species of oaks and madrones, pepperwood trees, very beautiful. The view -- and of course, the views were pretty fantastic so --

Q. Now, were these hiking trails there when you and Mary bought the property?

A. They were -- they were in place, yes. They were -- they were just overgrown. And this is an original, you know, road there. And then there was some -- you know, some trails there. We opened up some ourselves, but --

Q. And how would you re-open the hiking trails? Tell us the process.

A. Well, this one I used -- someone had a loader. I had them originally take that and open it, and then I maintained it. The other one I had a chainsaw, a small chainsaw, and go up and start -- I had to go up and process wood. We had four fireplaces. That was our main heat.

Q. And so how would you maintain the hiking trails once you had got it cleared out?

A. Well, I would maintain it; go in there and I would look for poison oak, which would grow up in the foot -- the footpath there. So I would go up with my sprayer and spray that. Other things that -- excuse me -- weeds and whatever I felt needed to be sprayed. And the road -- this roadway coming up to the back, I needed to access that. You could drive up here with four wheels. I used to drive it with my four-wheel truck.

Q. When you say "spray," what are you spraying?

A. I'm spraying a lot of poison oak.

Q. What are you using -- what is in the sprayer?

A. Oh, Roundup. Roundup was in the sprayer, yes.

Q. Okay.

A. Sorry, I didn't know what your terms --

Q. That's okay.

And just before I have you sit down, Mr. Hardeman, can you show the jury on the plot map where -- first of all, did you use Roundup on this property?

A. Oh, yes.

Q. Okay. And when was the first time you used Roundup on this property, on the West Side property?

A. Well, probably I would think starting clearing that right away. So after I got that poison oak experience and needed a shot, you know, I just started -- it had to be, what, late 1989 to where I started setting aside some time for plans and maintenance to go after this stuff.

Q. And how long did you, Mr. Hardeman, own this property?

A. Probably 25 years.

Q. And during those 25 years that you-all owned this property -- would you like some water?

A. Thank you.

Q. Here.

During this 25 years that you and Mrs. Hardeman owned this property, how many of those years did you use Roundup on the property?

A. I used it every year, so 25 years. It was a regular part

of my maintenance to -- when I went to take care of other things and, you know, the water system, which was important to keep that running, or process firewood, I would always, you know, put the Roundup in the container and go up and climb up in that area and start applying it to where I thought it needed it.

Q. And now are you saying to the jury that you sprayed Roundup on all 56 of these acres?

A. No, not practical.

Q. So where -- that is not practical?

A. No.

Q. So where in particular did you spray Roundup during your maintenance each year of those 25 years?

A. Well, I would say that in relationship to the map, I would -- it would be all the way up in here -- in this area.

Q. And what is that area, Mr. Hardeman?

A. Well, what we have here is -- this is the concrete a ton GREE tank and then past that is the clearing 2,500-gallon plastic tank. I sprayed all around there. Plumbing, all around this tank here, coming up here, the trails coming up to the spring box. I went to the spring box and made sure that that was free of poison oak. And then maybe up to about -- it could have been right about up until the roundabout, and then up through the water course with the piping. I'm up to about past here.

So yeah, it is not quite half -- somewhere in here.

Q. Mr. Hardeman, I'm going to have you go ahead and take a seat back on the witness stand.

You testified that -- that you would spray every year as part of your annual maintenance program. How often during a year would you spray Roundup?

A. Well, I would start in May when the temperature was right and the winter was over with; and I would spray into the summer; spray into September, October. And then I would stop in November more than likely.

Q. And when you were spraying Roundup on any particular day, approximately how long would you be spraying it for?

A. I would say three to four hours, probably my spraying time.

Q. Is it fair to say because we are talking, you know, I guess between the Gualala property and the West Side property, how many years do you believe that you used Roundup?

A. Oh, I would have to say probably 28 -- between 28 and 30 years, I suppose.

Q. And during that time, is it fair to say sometimes you would spray some days longer, some days shorter?

A. Yes. I would definitely spray longer some days than others, I would think, yes.

Q. You testified that you used Roundup Concentrate at the Gualala property. What type of Roundup did you use at the West

Side property for those 25 years?

A. It was a concentrate Roundup product you could mix with water.

Q. And what type of device would you use to spray at the West Side property?

A. It's a pump-up sprayer.

Q. Would it -- would it be -- I'm going to show you -- if you could turn in your binder, Mr. Hardeman, to tab 25.

MS. MOORE: Ms. Melen reminded me, before we move onto that, Your Honor, we would move to admit into evidence Exhibit 23.

THE COURT: Any objection?

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Admitted.

(Trial Exhibit 23 received in evidence)

MS. MOORE: Thank you, Your Honor.

Q. And what is -- do you recognize Exhibit 25, Mr. Hardeman?

A. Yes.

Q. And what is that?

A. That is a typical pump-up sprayer that you would buy at one of the local hardware stores.

MS. MOORE: Your Honor, permission to publish to the jury.

THE COURT: Any objection?

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Go ahead.

BY MS. MOORE

Q. And is this a fair and accurate depiction, Mr. Hardeman, of the type of pump-up sprayer you used to spray Roundup during those 26, 28 years?

A. Yes, this is.

MS. MOORE: Your Honor, we would move to admit this photo as Exhibit 25.

THE COURT: Any objection?

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Admitted.

(Trial Exhibit 25 received in evidence)

BY MS. MOORE

Q. Would it be helpful, Mr. Hardeman, for you to demonstrate with a sprayer, a brand-new sprayer, how you would actually apply Roundup on your property?

A. Yes.

MS. MOORE: Your Honor, if he can come off the bench.

THE COURT: Sure.

MS. MOORE: Thank you.

Q. Mr. Hardeman, if you could, if you could explain to the ladies and gentlemen of the jury what this is.

A. This is a two-gallon pump-up sprayer, typical that you can buy at Home Depot or any hardware store, different versions of them. This happens to be a Flow Master.

Q. I'm going to ask you to speak up just a little bit so she can hear you.

A. Yeah. It has a plunger where you remove it, and you put in your Roundup and your water, and you bring it up to your two-gallon level mark. And then you put the plunger back in and put pressure -- build up pressure in it.

This is your wand that you use to -- that you have your pressure in it. You have interchangeable nozzles so you can put a fan spray nozzle on it to get a wider spray or you can use a direct shot.

Q. Which type of nozzle would you use on your pump?

A. I use a fan sprayer. That is my preference, to give me a wide swath. So if you are going and spraying on the ground, you can get -- you can cover more ground with it. I had a different kind of sprayer for a more direct targeting. Use my hand sprayer, typical one you use in the household.

Q. When you say "hand sprayer," do you mean like a bottle about this size?

A. Yes. Yes, a little hand sprayer that I would take the Roundup out of it and use a funnel and put it into the hand sprayer. And I could take the hand sprayer out, and I could target the poison oak if I was up on a platform where it was in close proximity to me. Then I could, you know, spray it, either mist it or spray it directly, just turning the nozzle and adjusting it.

Q. If you could demonstrate to the ladies and gentlemen of the jury, how would you actually -- how would you spray with using the pump?

A. Well, if I was walking down my road down from the house, you know, the poison oak was up on the embankments; and it would come up, grow and sort of lean up over the road. So I would spray it like this; go along -- or on the embankments, spray it on the embankments or down on the gravel part where the weeds and grass and other things are.

Q. Thank you, Mr. Hardeman. Take a seat back up on the witness stand.

MS. MOORE: Your Honor, if I can put this on the ELMO.

THE COURT: What is it?

MS. MOORE: It is the picture of what we just showed.

THE COURT: Okay.

MS. MOORE: Thank you.

Q. And do you recognize this document, Mr. Hardeman?

A. Yes.

Q. Okay. Is this the sample sprayer we just showed to the jury?

A. Yes.

MS. MOORE: Your Honor, we would move to enter this into evidence as Exhibit 946.

MS. MATTHEWS JOHNSON: The picture, no objection.

THE COURT: Okay. Admitted.

(Trial Exhibit 946 received in evidence)

BY MS. MOORE

Q. Mr. Hardeman, just so the jury has an understanding, I want to show you, if you could flip in your binder to Exhibit 936. And in the interest of time, there is a series of photos from 930 to 936. Do you see those in your binder?

A. Yes. Uh-huh.

Q. Do you recognize these photographs marked as Exhibits 930 to 936?

A. Yes, I do.

Q. And what are these photos depicting?

A. Oh, these are photographs -- let's see. This is page 930. So this is the driveway going up to the gravel road driveway. These are the drainage ditches on the side that I would maintain.

Q. So are these photographs from the West Side property?

A. Yes.

Q. Okay. And are these fair and accurate depictions of the areas of where you would spray on the West Side property?

A. Yes.

MS. MOORE: Your Honor, we would move to publish these to the jury.

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Go ahead.

MS. MOORE: Then we would also move to enter them into

evidence as well.

MS. MATTHEWS JOHNSON: No objection.

THE COURT: Admitted.

(Trial Exhibits 940 through 945 received in evidence)

BY MS. MOORE

Q. We will start, Mr. Hardeman, with 936. Mr. Hardeman, what are we looking here at Exhibit 936?

A. Go back to it here, that is the entrance -- as you turn off of the West Side Road -- as I showed on the map -- and you would make a left turn, this is the beginning of the driveway here to access the house and the areas I would spray. I would spray down in here. A lot of this is overgrown now.

Right on the other side of that address sign in front of the -- on the front West Side Road frontage is our mailboxes. So there is a lot of growth there and poison oak up in there. And also directly across the street from the mailboxes was a little turnout that we used to just kind of park our car off the road to walk across and get our mail.

Q. Why would you -- why would -- would you spray in that area?

A. I would spray that because that was our parking, little parking space. We all utilized that to, you know, walk up and get your mail. I would drive up to the house. When you get out on the passenger side, there is poison oak on that side, on the riverside. I would want to, you know, keep that, you know,

poison oak-free so if somebody got out, they weren't stepping in it and so on and so forth.

Q. And then let's turn to -- sorry -- let's turn to 930, if we can publish.

A. 930.

Q. And what is this area of the property? Sorry, you have to go backwards in the binder.

A. 930 is a little further up from the first picture on 936 going into the -- well, the gravel part of the road. And I would, you know, spray into these -- up on the embankments. If you look further up the road, you will see there is a lot of growth coming off that embankment. There is poison oak up on the hillside just past that on the tree.

Q. And then let's go to 931. Where is this on the property? What are we looking at here?

A. That is my detached garage. It is about 80 feet from the house. And what I did -- there was poison oak coming off of that embankment and going on top of the roof. So we built that garage, and I wanted to get up and clean those gutters -- those rain gutters clean. So I built a plank system. It is a very tight space there. I built this plank that I could attach to the fence, and stand up on it, and I would stand up on it. And when I got up on it, I could clean the gutters out. It would be about chest high. Then I would turn around with my hand sprayer and try to spray the poison -- the poison oak. And

that's when sometimes I would spray it with some close proximity to it. And, you know, you could see it in the sunlight. It would, like, atomize and I had a sense I breathed something in, like you may have a steamer or something, you know. Anyway, that's what that is. So that is an area I maintained.

Q. Now, can you actually see poison oak in this picture 931?

A. I'm sorry?

Q. Can you actually see poison oak in 931, the picture we are looking at?

A. I don't understand your question.

Q. Can you see any poison oak in the picture?

A. On the other side of that -- this is a fig tree here on the front. On the other side of this -- my plank would be on the other side of this. And, you know, you can see -- you can see, if you look through it -- I can see it there, you know. A lot of times you have -- you know, when it loses its leaves, you know, it goes into a -- like a stick form. Still the root system is still there.

Q. Let's pull up 932. Mr. Hardeman, this may be a different view of 931. What is this?

A. Yes, that's just another angle of the garage. You can see the roof -- yeah, the roof is on the east side, and there is the embankment. And that's where I had the plank. And you can see -- you can see, you know, on the other side of that. I can

see the growth that I would spray, poison oak. It is starting to grow on top of the roof.

Q. Did you and Mrs. Hardeman ever hire anyone to help you take care of this property?

A. No. I did it myself.

Q. Why did you do it yourself?

A. We were do-it-yourselfers. We -- it is something we could do. And I enjoyed doing it, and, you know, hiking and taking care of it. I wanted to make sure that I was going to get everything up to my own personal standard which, you know --

Q. Let's go to picture 933 -- Exhibit 933.

Mr. Hardeman, if you want, you can look on the screen there in front of you. What is this that we are looking at in 933?

A. That is between the garage up to the concrete tank. That is an area we could go off into. It is a gentle slope. I would go up in there to process firewood. You can see some pieces, remnants of someone cutting wood down there. Also when you walk up in there, there is also -- there is poison oak that pops up and grows in different spots. I would spray up in there.

Q. Mr. Hardeman, I was just told that you can actually put your finger on the screen if you want.

A. Yes.

Q. And it will -- is that right?

A. Okay. Yes. So I would walk up in there and, you know, get whatever firewood I could, and cut it up and bring it down. At the same time I would spray any -- you know, any poison oak or anything that may inhibit me from doing that.

Q. Let's go to 934.

A. Okay.

Q. What are we looking at in this picture, Exhibit 934?

A. This is the concrete water system that -- it is a 15,000-gallon tank. You can see that there is remnants of poison oak popping up in the driveway there. It is there, and along the -- I would -- as you look up that road up there, that goes up another -- you know, several hundred feet to that roundabout, yeah, up to the top there; and that's kind of where that road ends. But off to the left there is that other tank, the other blue dot on there, it is a 2,500-gallon tank. So I would go up there and there was poison oak growing up around the plumbing. It was important to keep the plumbing -- because it would go up there, and you had shut off valves and whatever, and I didn't want to get it on my hands. There was a lot of stuff growing up in there.

And then as you go up to the roundabout, that's when I would have to go up and service the spring box and go up -- I would hike up in there and spray where I felt it was necessary. This tank here, I would spray around this tank, the side, the back, you know, so that -- because it was cut against a

hillside on the other side and growth comes up on top.

So it is all part of the maintenance routine that I did on a monthly basis.

Q. And then Exhibit 935, can you tell the ladies and gentlemen of the jury what this is?

A. Yeah, this is the -- you know, the plumbing coming off of that tank, that's the shut-off valve. There were two shut-off valves. You can see the poison oak around the valve, and that's what I'm talking about there. It -- there is -- so I would come in there and try to, you know, maintain that as best to keep it free of this type of stuff. And, yeah, that comes out of the front of the tank going down the road, that particular valve.

Q. During these 26, 28 years that you used Roundup at your two properties, Mr. Hardeman, did you ever get Roundup on your skin?

A. Oh, yes, quite a few times. I described the sense that I breathe something in. Also a couple of times when it was foaming out the top when you are mixing it, it could get on your hands.

Q. When you say "foaming up at the top" --

A. You know, when you are filling it up with water, sometimes you put too much in. It foams up the top of the canister. You have to put the plunger in it, and it drives the foam out. When you handle it, you can get it on your fingers and hands

and whatever.

Q. What about when you use the hand sprayer?

A. On the hand sprayer, sometimes that would drip and leak; and, of course, when I was spraying the poison oak, I had a ladder on the east side of my house outside the kitchen against the cyclone fencing, and the poison oak was coming over the cyclone fence. I would try to get creative and get up there so I could spray it, you know, more the same height. I would use a hand sprayer. And that -- you know, sometimes you could see -- be spraying it and an afternoon wind would come up. You get winds there. And you get a little blow back, and it would blow back up on you is some of the experiences I had with it coming in contact with my skin or breathing it in.

Q. Do you still use Roundup?

A. No.

Q. When was the last time you used Roundup?

A. Had to be 2012, right before we put the house on the market. Summer I would think. Yeah, maybe somewhere around there.

Q. I want to switch gears and ask you a few questions before I sit down.

A. Uh-huh.

Q. Have you ever worked with any pesticides other than Roundup?

A. No.

Q. Did you ever work in chemicals or the chemical industry?

A. No.

Q. The jury, Mr. Hardeman, has heard that you had hepatitis C?

A. Yes.

Q. Can you tell them when you were diagnosed with hepatitis C?

A. 2005, to the best of my recollection.

Q. And the jury heard from your doctors, Dr. Turk and Dr. Ye, last week, and that you received treatment for hepatitis C.

A. Yes.

Q. Can you tell the jury when you received that treatment?

A. In 2005.

Q. Did that treatment go into 2006?

A. It finished in 2006.

Q. And what is your understanding of how that treatment went for the hepatitis?

A. I was cured.

Q. And has your -- to your understanding, has the hepatitis C virus ever come back since you were cured in 2006?

A. No, never.

Q. Now, how do you know that, Mr. Hardeman?

A. Because I get a blood test every year and they check that. I get a complete blood count, and they check for viral load; and it is all clear.

Q. The jury also heard that you had been diagnosed with cirrhosis of the liver; is that right?

A. Yes.

Q. And what is your understanding of the condition of your liver now?

A. It is in great condition. I mean, I was in my doctor's office looking at the CT scan from the lymphoma, and we went down and you can go down inside the body and look and see the liver. It looks great. It is really smooth.

And I said, Okay. Yeah, I -- pretty happy with that.

Q. Have you ever been diagnosed with active hepatitis B?

A. No.

Q. Now, the jury heard that you had a couple instances of some skin cancer, some sun spots. Can you tell the jury about that?

A. Well, sun damage years and years ago. I mean, the outside of my calf I had a thing called a basal cell. It is a little round mark, raspberry-colored thing. And then I --

Q. Where was that, Mr. Hardeman?

A. It was on my left calf. And it was a little -- like the size of a dime or nickel on the side of my calf, outside of my calf.

Q. When did you have that sun spot?

A. 2001 -- well, I think I have had that -- I don't know if it was a birthmark. May have been. It was around for many,

many, many years. Didn't pay much attention to it.

Q. And did you have treatment for that sun spot on your calf?

A. I went in and they removed it. It was outpatient. I went in and they took it out, and I went home and that was the end of it.

Q. And the last 18 years, since 2001, have you ever had any problems with that spot on your leg?

A. No, never had any problems with it.

Q. And then any other sun spots or sun damage?

A. Well, yes, last August I had -- when going in for routine checkups with Dr. Turk, I always have him check me out with my skin and everything. They always check the sun damage and look at freckles. So he found something up on my shoulder. We have been looking at for quite a while, and he took a picture of it. He sent it up to dermatology, and they did a biopsy and it came back as an in situ, which is zero stage kind of a skin cancer. And -- but went in for the surgery and took it out. And I was home that afternoon. There was follow-up. There was nothing --

Q. And that was in August of 2018?

A. Yes, last year.

Q. And that was after you had already been diagnosed with non-Hodgkin's lymphoma?

A. Yes.

Q. Okay. Were either of these sun spots malignant?

A. I'm sorry. Can you repeat --

Q. Were either of those malignant?

A. No, no, they were not malignant.

Q. Mr. Hardeman, the jury has heard that you have been diagnosed with non-Hodgkin's lymphoma. When did you get that diagnosis?

A. Well, official diagnosis was in, I think, in February of 2014 -- '15, '15, yes.

Q. When did you first notice that something was wrong?

A. Oh, well, it was on Christmas morning in 2014. I was getting ready to go to my sister's. My sister just passed away so I wanted to support my family down there and was looking in the mirror. We're getting ready to travel down to the South Bay.

I was looking in the mirror, and I said, Mary, look at this. I have this gigantic thing sticking out of my neck on this side. I said, What the heck is that?

And so what I did was I continued on. And then the next day I immediately called up Kaiser Permanente. And there was an on-call physician there.

He said, Well, why don't you just observe it for the next 30 days. It could be a swollen lymph node. Okay. My doctor was on vacation.

So as soon as he come back, I started emailing him and saying, It is not going away. There is something going on

here.

He said, Come on in. Let's look at it.

So I went in. And he made an appointment with the head and neck surgeon, Dr. Turley, to look at it. And he did a biopsy, two biopsies actually, and a needle biopsy, which is inconclusive. And then so he had to bring me back, and he did what -- I guess they take out a whole live sample. Waited. Waited and waited. And anxiety until it came back. And then it was -- he gave me the diagnosis.

Q. And so you noticed it -- was it that Christmas 2014?

A. The --

Q. When you noticed the swollen lymph node, that was Christmas 2014?

A. It was 2014, yes.

Q. And I don't want to get into the details of this right now.

A. Okay.

Q. But did you go through chemotherapy?

A. Yes, I did.

Q. And I'm sorry, Mr. Hardeman. So when you went through chemotherapy, did -- is it your understanding, do you know whether the hepatitis B came back?

A. No, none of that ever -- no, that didn't come back.

Q. What about the hepatitis C, did it come back?

A. No.

Q. And how do you know that?

A. Because my doctor, Dr. Ye, my oncologist, monitored it. You know, in between the six rounds of chemo, which were a thing called the nadir. You have 21 days between treatments. So you go for 10 days. You are going completely downhill, fighting all the side effects from the chemo. And during that period in order to -- I would say "qualify" for the next round, you have to go in and get a blood test because your white blood cells have to be up to a certain level so you can take the next round. And so along with the white blood cell test, he was testing for hepatitis C viral load.

Q. And since 2006 when you were cured of hep C, has the hep C -- hepatitis C ever come back to your knowledge?

A. No, never.

MS. MOORE: Those are all the questions I have, Mr. Hardeman. Thank you for your time this morning.

THE COURT: Any cross?

MS. MATTHEWS JOHNSON: Thank you, Your Honor. We have no questions, sir.

THE COURT: Okay. You are done. Go ahead and step down.

This is probably a good time to take a short morning break. So why don't we resume at about 35 after the hour. Thank you.

(Proceedings were heard out of presence of the jury:)

THE COURT: Is Dr. Weisenburger next?

MS. MOORE: Yes. Yes, Your Honor. Would you like him to come in so we can have that discussion with him?

THE COURT: Yeah, why don't we resume in two minutes? We will take a couple minutes. Then I will come back out and we will talk to Dr. Weisenburger before we --

MS. MOORE: Thank you. I would appreciate that.

(Recess taken at 9:28 a.m.)

(Proceedings resumed at 9:33 a.m.)

(Proceedings were heard out of the presence of the jury:)

THE COURT: Hello, Dr. Weisenburger.

THE WITNESS: Good morning.

THE COURT: So why don't -- I think what it would be worth doing, why don't we do it this way. Who's doing the direct examination of Dr. Weisenburger?

MS. MOORE: I am, Your Honor.

THE COURT: Do you want -- just to make sure we're on the same page, why don't you articulate to me your understanding of my ruling that limits Dr. Weisenburger's testimony about dose-response?

MS. MOORE: Okay. I will come to the microphone.

THE COURT: If you want me to do it, that's fine --

MS. MOORE: No.

THE COURT: -- but I thought it would be useful to make sure that you understand and that Dr. Weisenburger

understands.

MS. MOORE: My understanding, Dr. Weisenburger will testify that in his opinion there is a dose-response; that the more you use Roundup, the greater the risk of developing non-Hodgkin's lymphoma. And that, in a nutshell, is how he's going to testify about dose-response.

And with respect to probably where you're coming from on McDuffie and Eriksson, he is going to explain the Epi, not in any detail like Dr. Ritz, but the Epi studies do show that that dose-response, it increases and I think they used more than 2 days or more than 10 days. So he is going to reference that, but --

THE COURT: In connection with his general causation opinion?

MS. MOORE: That's correct, Your Honor. That's correct, Your Honor.

With respect to his case-specific opinion, it's that in his opinion, Mr. Hardeman falls within a high-risk category based on his significant exposure over those 26 or so years, and that was the reason why -- one of the reasons why he ruled in Roundup as a result of his general causation opinion as well. But he's not going to quantify the number. He's not going to quantify it.

THE COURT: In other words, he's not permitted to quantify the risk factor for Mr. Hardeman based on the McDuffie

and Eriksson studies.

MS. MOORE: Right. He's going to use the McDuffie and Eriksson studies to show there's a dose-response and to show how they base that dose-response on that; and then he's going to say, "In my opinion, based on the significant exposure of Mr. Hardeman over those years, that he would fall into a high-risk category so he would have an increased risk of developing non-Hodgkin's lymphoma."

THE COURT: Okay. But the McDuffie study and the numbers emanating from the McDuffie study and the Eriksson study come in during the general causation --

MS. MOORE: That's correct.

THE COURT: -- opinion, and they do not -- they're not used -- other than sort of the general comment that there's a dose-response, they're not used -- they're not linked specifically to Mr. Hardeman.

MS. MOORE: That's correct, Your Honor.

THE COURT: Okay. Dr. Weisenburger, do you understand those ground rules?

DR. WEISENBURGER: Yes, Your Honor.

THE COURT: Okay.

MS. MOORE: Okay.

MR. STEKLOFF: Can I just raise a separate issue, Your Honor, which is to clarify in part based on yesterday, the IARC question?

THE COURT: Thank you.

MR. STEKLOFF: Depending on how you rule on that, I think you've now heard from Dr. Ritz and Dr. Portier the IARC classification so I'm not sure we need to -- I understand that Dr. Weisenburger can say that Dr. Blair was part of the Working Group from earlier; but other than that, I don't think IARC needs to play a role in his testimony.

THE COURT: Other than the fact of the classification, he can repeat that. It's been repeated enough times sort of where EPA stands on this and where the European regulators stand on this. So he can reference the fact of the IARC classification and, you know, who Dr. Blair is, but that's it.

MR. STEKLOFF: Okay.

THE COURT: And then I did go back and think about your point about the IARC's conclusion regarding genotoxicity, and I agree with your point. I believe that the Portier testimony is sufficient to address the issue that we discussed yesterday, and so there will not be testimony permitted on the IARC's specific conclusions about genotoxicity.

MS. MOORE: And just to clarify, Your Honor, to make sure I understand, Dr. Weisenburger is prepared, as he did during *Daubert*, with his general causation opinion to say that he reviewed the genotox literature and that in his opinion, Roundup is genotoxic, which is separate and apart from --

THE COURT: Of course, that's fine.

MS. MOORE: Okay. Okay. Thank you, Your Honor.

THE COURT: It's just that Dr. Weisenburger -- just to make clear, just to make sure Dr. Weisenburger is clear, it's just that he cannot testify about -- he can testify about the fact of the IARC classification but can't go into any of IARC's specific conclusions regarding genotoxicity, epidemiology, toxicology.

That's -- we're not -- the point is we're not going down the road of having a fight about whose analysis is better between the IARC and the EPA. We're going to have a fight about whose analysis is better, Dr. Weisenburger's or Dr. Mucci's. That's what this trial is about. Okay?

MS. MOORE: All right. Thank you, Your Honor.

THE COURT: All right. So, Dr. Weisenburger, you can come on up.

And you can bring in the jury.

MS. MOORE: Your Honor, I'm going to distribute binders here.

THE COURT: Okay.

(Proceedings were heard in the presence of the jury:)

THE COURT: Okay. You can resume.

MS. MOORE: Thank you, Your Honor.

Plaintiffs call Dr. Dennis Weisenburger.

THE CLERK: Dr. Weisenburger, can you stand, please? I need to swear you in.

DENNIS WEISENBURGER,

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

THE WITNESS: I do.

THE CLERK: Thank you. Please be seated.

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

THE WITNESS: My first name is Dennis, D-E-N-N-I-S. My last name is Weisenburger, W-E-I-S-E-N-B-U-R-G-E-R.

THE CLERK: Thank you.

DIRECT EXAMINATION

BY MS. MOORE:

Q. Good morning, Dr. Weisenburger.

A. Good morning.

Q. Can you introduce yourself to the jury and tell them a little bit about what you do?

A. Yes. I'm a pathologist and I have special training in diseases of the blood and the bone marrow and the lymph nodes of which non-Hodgkin's lymphoma is one of the diseases that I deal with every day.

And over the last 40 or so years, that's been the main topic of my research at the University of Nebraska Medical Center where I worked for 28 years and now at the City of Hope Medical Center in the L.A. area.

Q. What is pathology?

A. Pathology is the study of disease. Pathologists, one of their roles is to try to understand what causes disease, what are the mechanisms that are used to cause disease by organisms or chemicals; and then a big role that we have in the hospital is we are the ones who run the clinical laboratories, that do all the testing on specimens, blood, urine, fluids and tissue biopsies. And so we're in the background helping the doctors make the proper diagnosis.

Q. You mentioned biopsy, and the jury has heard that Mr. Hardeman had a couple of different biopsies. Can you explain to the jury what a biopsy is?

A. Yes. So a biopsy is just taking a piece of tissue from the patient. Mr. Hardeman had two biopsies. One was a needle aspiration where they just sucked some of the tissue out of the tumor, and it was all dead tissue so they couldn't really make a diagnosis.

And so then they went and did what's called a needle biopsy where they took a needle and put it into the tumor and pulled out a needle core of tissue. And then they process the tissue, make slides from the tissue so they can actually look at what the tumor looks like, and that's what pathologists do.

Q. When you say "needle core," what does that mean?

A. Well, it's -- you know, like if you stuck a needle in an apple and you pulled the needle back out, you would have a very thin core of the apple. It's the same principle.

Q. And you said you're a pathologist. Are you a hematopathologist? I never can get that right. I know I'm not saying it right.

A. Yeah. So I have special -- I spent two additional years of training learning about hematopathology, which, as I said, was diseases of the blood and the bone marrow and the immune system. So that's an area that I'm specialized in in pathology in terms of my research and my everyday work.

Q. And what is hematopathology?

A. Well, as I said, it's really the study and diagnosis of diseases that arise in the blood or the bone marrow or the immune system; like the lymph nodes, for example.

Q. You mentioned that your focus is non-Hodgkin's lymphoma. How did you end up focusing on non-Hodgkin's lymphoma, Dr. Weisenburger?

A. Well, when I finished my training in hematopathology, I went looking for a job and I eventually ended up at the University of Nebraska where I ended up working for 28 years. And I went there to interview. People told me that there was a high rate of lymphoma in Nebraska. So that really peaked my curiosity.

And so in the end, I decided to go to Nebraska and work as a hematopathologist, but I was also interested in trying to understand why would there be a high rate of lymphoma in Nebraska. And so that's how I started my research, my

epidemiology research, into lymphoma.

Q. So let's back up, then, a little bit before we get into your actual research and explain to the ladies and gentlemen of the jury what your educational background is and your medical training.

A. Yes. So I got my medical degree from the University of Minnesota in Minneapolis, and then I did a one-year medical internship in internal medicine at the Ohio State University in Columbus.

And then I decided to shift gears and do a year of pathology just to take a break from the tough internship, and I went to the University of Iowa in Iowa City. And I just fell in love with pathology, and so I just stayed at Iowa and finished my training in pathology. I didn't go back to internal medicine like I'd originally planned.

And then there I got a real interest, because of some of my teachers and professors, an interest in hematopathology. And so after I finished my residency, I took a fellowship at the City of Hope actually where there was a famous hematopathologist at that time and spent two and a half years there learning hematopathology.

Q. What is the City of Hope?

A. The City of Hope is a freestanding comprehensive cancer center in Duarte, California, which is actually a suburb of Los Angeles. So it's one of the 50 or so comprehensive cancer

centers that are recognized by the National Cancer Institute for doing important work in research and treatment of cancer.

Q. What is the National Cancer Institute?

A. Well, that's the -- that's the federal government agency that oversees cancer research in the United States. They do their own research, but they also fund research all across the United States, mostly at universities and academic medical centers like City of Hope.

Q. So would you consider the City of Hope to be a national major research center for the study and treatment of cancers, including non-Hodgkin's lymphoma?

A. Yes.

Q. And are you Board certified?

A. Yes. I'm Board certified in both anatomic and clinical pathology.

Q. If you could, based on your 40 years of studying the causes of non-Hodgkin's lymphoma, explain to the jury what is non-Hodgkin's lymphoma?

A. So non-Hodgkin's lymphoma is a kind of cancer that develops from cells of the immune system -- okay? -- the system that protects us from infections and protects us from cancer. There are these cells in the immune system that protect us. Okay? And they're cells called B cells -- just the letter B, B cells -- and those cells produce what we call antibodies or proteins that circulate in the blood and in the tissues and

protect us from infections and other things.

And then there's another type of cell called the T cell, which can react to a foreign material by mobilizing other cells to kill that material or actually killing that material itself. So it could kill bacteria or viruses or cancer cells.

And then there are a variety of other cells of the immune system, but those are the two main types.

Q. Is non-Hodgkin's lymphoma common among the general population of people in the United States?

A. Well, it's relatively common. It's I think the sixth or seventh most common cause of cancer in adults. In the men it's number six, and I think in women it's number seven. And there are about a little over 70,000 cases a year in the United States. So it's relatively -- it's a relatively common cancer.

Q. But is cancer common among the general population?

A. Yes, cancer is common among the general population.

Q. And then are there -- the jury has heard there's different subtypes of non-Hodgkin's lymphoma.

A. Yes. So there are non-Hodgkin's lymphomas that arise from the B cells that we talked about, as well as the T cells, and there are actually 60 or more different specific types of non-Hodgkin's lymphoma. So it's a complicated classification but we often talk about what type of lymphoma is it, what type of non-Hodgkin's lymphoma; and today we're going to talk about

one of the common types called diffuse large B-cell lymphoma.

Q. And where are you working now?

A. Well, I was the chairman of the department at City of Hope for six years, and then last fall after I had my 70th birthday, I decided I didn't want to work so hard and do all that administration so I went back to being just a diagnostic pathologist continuing my teaching of fellows and doing my research, as well as doing diagnostic work.

Q. So you gave up the administrative part?

A. Yeah.

Q. Do you miss that?

A. No.

Q. All right. And prior to coming to the City of Hope, is that when you were at the University of Nebraska Medical Center?

A. Yes.

Q. And did you also teach when you were at the University of Nebraska Medical Center?

A. Yes. I actually taught more because we had medical students, we had residents, and we had fellows, and so there was a lot more teaching and lecturing there at the University of Nebraska.

Q. And I saw on your curriculum vitae, Dr. Weisenburger, that you also had listed the Beckman Research Institute at the City of Hope. Can you tell the ladies and gentlemen of the jury

what that is?

A. Well, the Beckman Research Institute is more of a basic science institute. That's where most of the researchers are that are looking into what causes cancer and how cancer develops. So it's sort of the more basic research part of the City of Hope, and I'm a member of that group because of the research that I do.

Q. And are you still doing research into the causes of non-Hodgkin's lymphoma?

A. Yes.

Q. When you were in Nebraska, I saw on your curriculum vitae that you also were listed as the chief pathologist for the Nebraska Lymphoma Study Group; is that right?

A. Yes.

Q. Tell the ladies and gentlemen of the jury what that was.

A. Well, there's a group of people at Nebraska who were very interested in lymphoma, particularly non-Hodgkin's lymphoma, and there are clinicians who take care of patients, pathologists like myself, as well as researchers; and so we formed a research group and we organized the eastern part of Nebraska, all the community hospitals, into a group that would provide us with material from all the patients in eastern Nebraska. So we had lots of biopsies and material to do the research on.

And so this is a group that's been ongoing for over 40

years, that's been very productive, and one of the things that Nebraska is famous for is its research into lymphoma.

Q. I saw on your curriculum vitae also reference to something called InterLymph. Can you tell the ladies and gentlemen of the jury what InterLymph is?

A. Yes. When I first came to University of Nebraska, as I told you, I was interested in trying to figure out why there's an increased -- increase in non-Hodgkin's lymphoma or lymphomas in general in Nebraska so I got interested in epidemiology. And as part of that, I actually organized a large epidemiologic case-control study of non-Hodgkin's lymphoma in Nebraska and learned epidemiology kind of by doing it. And so that was one of the important research projects that I carried out in my career.

The InterLymph is a group of people like me who are epidemiologists, pathologists, biologists, and clinicians who are working together as a team to do research into what causes non-Hodgkin's lymphoma and the other lymphomas.

Q. And are you a founding member of InterLymph?

A. Yes, I am.

Q. And why did InterLymph actually come into existence?

A. Well, back in the 1980s, epidemiologists noticed that there was a rapid increase in the incidence of non-Hodgkin's lymphoma which was unexplained, and so they called a meeting at the National Cancer Institute and they invited people from

around the country and around the world to come to that meeting, it was an all-day meeting, to sort of discuss what could the cause be for this rapid increase in non-Hodgkin's lymphoma. Because from about 1970 to 1990, there was a rapid increase and we didn't understand why.

And so out of that meeting that was held, the InterLymph group grew out of that meeting as an organization to do research and try to address that question among other questions.

Q. And is InterLymph still active today?

A. It is, yes. It still -- we meet once a year and sometimes we meet at other meetings to talk about our research and design new studies. So it's a large group of about 40 or 50 researchers who are together working to try to understand better what causes lymphoma.

Q. And are you still active with InterLymph?

A. I am, yes.

Q. What is the Eppley Institute for Research in Cancer and Allied Disease?

A. So that's the basic science institute at the University of Nebraska that does basic research. That's where most of the Ph.D. researchers are that are researching what causes cancer and how cancer develops. And so I was a member of that group also because of my research in lymphoma.

Q. And I also saw that you listed the Center for

Environmental Health and Toxicology. What is that?

A. So that was a center at the University of Nebraska that was mainly focused on diseases that were caused by the environment. So in Nebraska, of course, it's an agricultural state so they were looking at asthma in farmers and we were looking at cancer in farmers, including non-Hodgkin's lymphoma, but there were a variety of things we were looking at mainly regarding agriculture because that's, you know, the main -- that's the main occupation there in Nebraska.

Q. So you mentioned "environmental." The jury has heard the term "environmental health." What does that mean?

A. Well, it's just what is the effect of environment on the health of people. You know, in some places, big cities, they look at air pollution. In some places, like Nebraska, we were concerned about pesticide contamination and fertilizer contamination of water, groundwater, lakes and rivers, and underground water. We were also interested in the use of pesticides by farmers.

Q. And how long have you been studying whether pesticides cause cancer?

A. Well, since -- almost since I was -- I started at the University of Nebraska in the mid-1980s because when I got there, I started sort of asking questions what could be causing this increase in Nebraska.

Q. And, Dr. Weisenburger, is Roundup a pesticide?

A. Yes, Roundup is a pesticide. It's specifically a herbicide, which is a chemical that kills weeds. It kills plants actually, and we want to put it on weeds because we don't like weeds, but if you put it on other plants, it will kill other plants too.

Q. I also saw that you listed that you had the National Cancer Institute Peer Review Group. What was that?

A. So I was invited to be on a panel of researchers and clinicians who -- to look at the research program and the future plans of the National Cancer Institute with regard to these hematologic cancers, and so I was an invited guest. We spent a day and a half together going over all the things the National Cancer Institute was doing in lymphoma and other diseases and trying to advise them about what we thought they should do in the future.

Q. And what is the Cancer and Leukemia Study Group B?

A. Yeah, so that's a large cooperative group of universities and hospitals that are doing clinical studies of patients. So patients with a certain disease, they may test a new drug or a new drug combination in those patients.

And I was involved in that group because someone has to review the biopsies and the pathology and make sure the diagnosis is correct, and so that was my role in that group.

Q. Have you published about non-Hodgkin's lymphoma?

A. Yes. That's been the major area that I've done most of my

publication in, in that disease.

Q. How many publications have you authored or co-authored over your career?

A. Well over 400 publications published in peer-reviewed journals, yes. So it's -- you know, as an academic person, that's what we're expected to do.

Q. How many of those 400 publications involved looking at the causes of non-Hodgkin's lymphoma?

A. Well, that's a tough question. A lot of them looked -- a lot of them were looking at the cause of non-Hodgkin's lymphoma; but I think the major ones, what you're asking me is, you know, with regard to epidemiology studies, we did over 50 -- we wrote over 50 papers on epidemiology and the research that we did at Nebraska and the research we've done in the InterLymph group to try to understand the causes of non-Hodgkin's lymphoma.

Q. Have you published on the causes of non-Hodgkin's lymphoma including studies of pesticides?

A. Yes, especially the studies that we did in Nebraska focused a lot on farming and farming practices and pesticide use.

Q. In addition to actually authoring -- writing publications, have you also served on editorial boards and participated in peer review?

A. Yes. So I've been on a number of editorial boards for

pathology journals and lymphoma journals, and I've been a peer reviewer for papers for many years.

Q. Well, Dr. Weisenburger, you've referenced a couple times your work in Nebraska, and if you could tell the ladies and gentlemen of the jury a little bit more about what that work entailed when you first got to Nebraska and noticed this increase in non-Hodgkin's lymphoma.

A. Yes. So, you know, how does one start? So I was -- you know, I was a new young hematopathologist trying to figure out how do I tackle this approach. So one of the things I did first, I realized there was some databases. So the Nebraska Department of Health had a database on a cancer registry, and I could look and see how many cancers occurred in each year in the different counties of Nebraska. So I made some maps in eastern Nebraska and I found out which were the counties that had high -- a high rate of non-Hodgkin lymphoma, Hodgkin lymphoma, leukemia, and other diseases like that.

And then I made some maps looking -- based on data published by the University of Nebraska on, you know, what were the counties where there was high pesticide use, herbicide use, insecticide use, fertilizer use, and corn production to try to see if I could correlate the counties with the high rates of lymphoma with counties that seemed to have very intense agriculture. And, in fact, there was a correlation, and that got me very interested in pursuing research.

And then about that time, a publication came out about an epidemiologic study from Kansas, and it showed that certain pesticides increase the risk for non-Hodgkin's lymphoma. So I got really excited about that, and I called up these researchers at the National Cancer Institute, who I didn't know, I called them up out of the cold and I said, "Look, you guys" -- it was Dr. Blair and his group -- I said, "Look, you guys, you need to come to Nebraska and do an epidemiology study."

And they said, "Well, we'd like to, but we don't have any money to come to Nebraska." They said, "If you raise the money, we'll come and help you do it."

So I did. I wrote grants and I raised the money to do the study; and then because we didn't have any epidemiologists at Nebraska at that time, they came to Nebraska, helped us organize the study, designed the questionnaire, trained our interviewers, did the quality control, and actually then analyzed the data for us when the study was over.

So I had a partnership with these people at the National Cancer Institute, and that's how we did the first study of lymphoma and other diseases like that at Nebraska, the so-called Nebraska study, which you'll hear about.

Q. So you said "they came." So tell the ladies and gentlemen of the jury who actually came from Kansas to Nebraska to help you.

A. Well, Aaron Blair came, one of his bright young students Sheila Zahm came, and a number of other researchers came over the three or so years that it took us to do the study. And I also went to the National Cancer Institute and met with them.

Q. And who is Dr. Aaron Blair?

A. So Dr. Aaron Blair is a very well-known epidemiologist who was the head of the occupational epidemiology branch at the National Cancer Institute. So his role there was to study what causes diseases by different occupations, and he was originally from Kansas so he was very interested in what causes cancer in farmers and had designed that Kansas study that I told you about.

So his team actually were an expert team with regard to trying to study cancer in different occupations, particularly farming.

Q. And, Dr. Weisenburger, I'm going to have you -- probably a blast from the past here, but look at 1569 in your binder.

A. (Witness examines document.)

Q. It's probably at the very back.

A. (Witness examines document.) Yes. This is a paper I wrote early on when I was at Nebraska just describing some of the findings and hypothesis that I had. So I don't know if you have it, but --

Q. Now, hold on. Do you -- so is this a publication that you authored, Dr. Weisenburger?

A. Yes.

Q. And what year was that?

A. Oh, 1985.

Q. Okay.

MS. MOORE: Your Honor, permission to publish to the jury.

MR. STEKLOFF: No objection.

THE COURT: Go ahead.

BY MS. MOORE:

Q. And is this your publication, Dr. Weisenburger?

A. Yes.

Q. Okay. And if you flip over to page 3, are these the maps you were referencing to the jury?

A. Yes.

Q. Okay.

A. This is my crude attempt to learn something about what was causing lymphoma in Nebraska.

Q. Okay. And this became the Nebraska story?

A. This was the start of the story, yeah.

Q. So what is the Nebraska story?

A. Well, the Nebraska story is that we did -- based on this research, I convinced the people at the National Cancer Institute to come and help me do a large epidemiologic case-control study of non-Hodgkin's lymphoma and Hodgkin lymphoma and other related diseases. And out of that study

came a lot of publications that I think were very important, some about pesticide use, others about a variety of other things that might cause non-Hodgkin's lymphoma.

Q. And what in particular were you looking at as to what was the cause of non-Hodgkin's lymphoma?

A. Well, we were, of course, mainly interested -- we were mainly interested in pesticides, but we also looked at other things that might cause non-Hodgkin's lymphoma. So we asked questions about family history. We asked questions about chemical use.

We asked questions about hair dye use because there was this idea that hair dyes could cause lymphoma. And, in fact, what we -- and one of the unique things about our study was that I insisted that we include women in our study. Okay? Because all the other studies, the Kansas study, were just men.

Q. Why is that?

A. Well, they thought that men would be the ones who would be the most exposed to pesticides, but I knew that women in rural communities work on the farm. They do -- sometimes they work just like a man. They drive the tractor, they do all those things. So I insisted that we include women in our study. So it was about half men and half women. And --

Q. Did you get some pushback about including women?

A. I did initially but since I was paying for the study, I got to say that. Okay?

And one of the things we found actually is that in women, the use of dark permanent hair dyes increased the risk of non-Hodgkin's lymphoma. So that was a really important finding. And as a result --

Q. Is that still the case today? Just asking.

A. No. Well, what happened after we published and other people published this, the hair dye industry decided to take a lot of the bad chemicals out of the hair dyes, and so later studies in the 1990s didn't find that finding anymore because people were using safe hair dyes. So it was one of the good things that happened as a result of the Nebraska study.

Q. On behalf of all women over the age of 40, we thank you for that.

Okay. And then is the Nebraska story -- I mean, we saw Exhibit 1569, one of your publications, Dr. Weisenburger; but this entire Nebraska story, has it been published?

A. Yes. I haven't counted how many papers, but there are at least probably a dozen papers just on the Nebraska study and what the findings were.

And then as you'll hear, some of the Nebraska data was combined with other studies as well to do more powerful kinds of research.

Q. Is that what was published by De Roos in 2003?

A. Yes. So -- yes, De Roos is one of the studies that was carried out by Aaron Blair and his team at the National Cancer

Institute. They did the Kansas study and then they did a study in Iowa and Minnesota, and then they came to Nebraska and did the Nebraska study. And so all three of those studies in those four states the data was combined together in that De Roos paper from 2003.

Q. Well, let's get into your opinions in this case, Dr. Weisenburger. And I want to show you Exhibit 880.

MS. MOORE: And permission to publish. It's the stool (indicating).

THE COURT: Go ahead.

BY MS. MOORE:

Q. And the jury has seen this when Dr. Portier testified last week, and if you could just kind of explain to the jury when you're studying the causes of non-Hodgkin's lymphoma, what do you look to to determine whether an agent like Roundup causes cancer?

A. Well, you want to look at all the literature on the chemical that you're interested in. So there are various different studies that tell you different things. So obviously I wanted to look at the epidemiology data because that's the data that would tell you does the chemical, in this case Roundup, cause cancer -- some kinds of cancer in people. Okay?

And so I looked at the epidemiologic data, and then there were also animal studies where they gave glyphosate or Roundup to animals in studies to see if those chemicals could cause the

cancer in animals. So I looked at the studies -- the animal studies to see what they said.

And then there were a lot of studies that looked at mechanisms of disease: Does Roundup cause DNA damage? Is it genotoxic? Does it cause other kinds of abnormalities in cells that might lead to cancer? And so I looked at all of the literature on the mechanisms of how Roundup and glyphosate could cause cancer.

So I looked at a wide body of data spanning animal studies to human studies and everything in between.

MS. MOORE: Ms. Melen, could I have the Elmo, please? Thank you.

Q. And, Dr. Weisenburger, explain to the jury why you looked at all three of these areas of science to form your opinion in this case.

A. Well, because I think you need to look at all the data. So, for example, if you just look at the epidemiology data, it might not be convincing. And if you look at the animal studies by themselves, they may or may not be convincing. And if you look at the mechanistic studies, again, depending on what you look at, you know, it may not be convincing.

So the way you do a general causation analysis is you want to look at all the information, analyze it, weigh it, and try to put -- put it together into a conclusion that's based on all the information rather than just pieces of the information.

Q. And so, Dr. Weisenburger, if someone came into the courtroom and told the ladies and gentlemen of the jury, "I only looked at epidemiology and I decided that Roundup causes or does not cause cancer," as someone who has been studying the causes of non-Hodgkin's lymphoma for over 40 years, what would you say to that?

MR. STEKLOFF: Objection, Your Honor.

THE COURT: Overruled.

THE WITNESS: Well, I would say that that was really inadequate in the sense that you should look at everything. You shouldn't just look at one piece of the puzzle because there are lots of important pieces of information, as you'll see today, besides the epidemiology studies.

BY MS. MOORE:

Q. So you want to look at all three?

A. Yes, and that's what I did.

Q. And, Dr. Weisenburger, after reviewing the literature for all three of these areas of science -- the epidemiology, the animal, and the mechanistic studies -- and weighing the evidence, based on your 40 years of studying the causes of non-Hodgkin's lymphoma, have you formed an opinion whether Roundup can cause cancer?

A. Yes, I have. And my opinion is that, you know, to the best of medical certainty, I believe that Roundup is a substantial cause of cancer in people who are exposed to it in

the workplace or in the environment.

Q. Well, what is Roundup itself? You mentioned earlier that it's a pesticide and an herbicide, but what is Roundup? The jury has heard about glyphosate, glyphosate-based formulations, and Roundup. Can you distinguish those for us?

A. Yeah. So Roundup is one of many glyphosate-based formulations. So it's thought that the active ingredient that actually kills the weeds is the glyphosate, but it's diluted in a liquid, probably water, and then there are other chemicals added to it to make it more potent. So there -- one of the chemicals that's added is a type of surfactant that allows --

Q. What's a surfactant?

A. Surfactant is just a chemical that allows a fluid to spread evenly over a surface. Okay? But the surfactants that they used not only did that, but they also helped bind the glyphosate to the leaves or to the plants and helped the glyphosate penetrate through the walls of the plants into the actual plant cells. Okay?

So glyphosate is the active chemical for these formulations, and different companies use different types of formulations but glyphosate is the basic chemical in all those formulations.

Q. And Roundup is a glyphosate-based formulation; is that right?

A. Yes.

Q. Well, how is one, then, exposed to Roundup?

A. Well, so the main way that I think in most cases the way people are exposed is when they use it to try to kill weeds or plants. Okay? So in farming, farmers would be exposed to it because they use it -- large amounts of it on corn and soybeans and other crops.

And people also use it for home use. So you can buy it in smaller bottles and use it to spray it on the weeds in your yard or to kill weeds in your garden. And, of course, you can be exposed to it by getting the chemical on your skin of your hands or your arms or, you know, you can even get it on other parts of your body, like your face if it's windy and it blows back on you.

You can expose -- get exposed if you get it on your clothes and you don't change your clothes that day or -- you know, farmers sometimes wear the same clothes more than one day because they wear it until it's dirty. Right? So they may be wearing clothes that already have pesticide in the fabric.

So those are the ways you get exposed to the pesticide. Mainly it's skin contact.

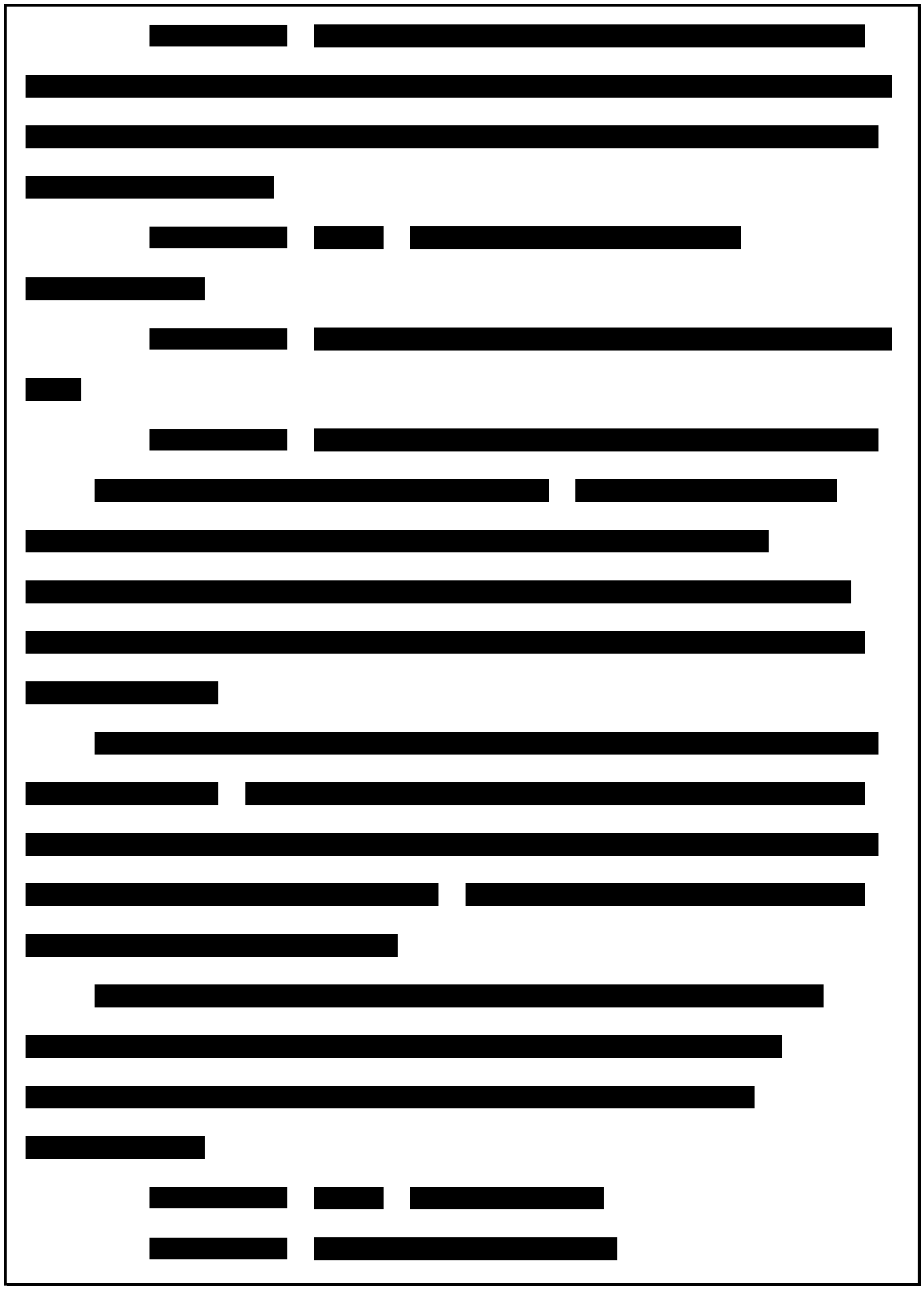
Q. And what happens when you're exposed to Roundup over and over again and it comes into contact with your skin?

MR. STEKLOFF: Objection, Your Honor.

THE COURT: Quick sidebar.

(The following proceedings were heard at the sidebar:)

SIDEBAR



(The following proceedings were heard in open court:)

BY MS. MOORE:

Q. So let me go back to my question then. What happens when you are -- when an individual is exposed over and over again to Roundup on their skin?

A. So when you get Roundup on your skin, just like the Roundup will penetrate the plant cells, it will penetrate the cells of the skin and it will get into the tissues and it will then get into the lymph system and into the blood, and eventually it goes through the kidneys and it gets excreted out in the urine. Okay?

But during that time, it's in the tissues, it's in the lymph, and it's in the blood, and so the tissues, all those tissues do get exposed to glyphosate as it's going through the body and out through the urine.

Q. When you say "in the lymph," what does that mean?

A. Well, we think about blood as being what circulates in our bodies, but in the tissues, the blood circulates but also other fluids without blood cells circulate and that's called the lymph. Okay?

So, you know, I don't know how it is for you, but if I sit a long time, my feet swell. Okay? And why are my feet swelling? Well, it's the fluid coming out of the blood and getting into the tissue and making my feet swell. And so if I get up and walk or run, that fluid will get mobilized and it

will get back into the circulatory system.

So the lymph is the circulatory system in the tissues that moves fluids around, but it's separate from the blood system although it's connected to the blood system. It empties into the blood system.

Q. So when Roundup penetrates the skin, it gets into the lymph system as well as the blood system?

A. Yes.

Q. Well, let's look at the first leg of the stool, the epidemiology. And the jury heard from Dr. Ritz last week, and I am not going to go through all those studies in detail, but I do want to focus on a couple of the studies that you're listed as the author, Dr. Weisenburger.

And the first one is -- it's Exhibit 451.

MS. MOORE: Permission to publish. De Roos 2003.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE:

Q. And, Dr. Weisenburger, can you tell the ladies and gentlemen of the jury what this publication is?

A. So this is the pooled study of De Roos 2003. This is the study where they pooled the data from the case-control studies done in Kansas, Iowa and Minnesota and Nebraska, and they put the data altogether into one study. They were able to do that because the studies had very similar designs, very similar

questionnaires because they were all designed by Aaron Blair and his team at the National Cancer Institute.

And so the purpose of this is to have a much larger study where you can have more statistical power to detect significant differences in people so, for example, who are exposed to one chemical or another chemical.

And the other thing about a bigger study like this is you can also do adjustments for confounding. And "confounding" means that if you're exposed to more than one chemical, how do you know which of those chemicals is actually causing the disease; right? So farmers use more than one chemical. They sometimes use -- they don't use a lot of chemicals but they usually use the same chemicals every year and they may use two, three, four different kinds of pesticides. Okay?

So a big study like this has the statistical power to actually adjust for the use of the other pesticides so you can focus on each of the pesticides individually and have a pretty good idea of whether it increases the risk or it doesn't, and that's what they were able to do in this De Roos study.

Q. And that was published back 16 years ago or so in 2003?

A. Yes.

Q. Okay. And the data that they were pooling, what period of time was that collected?

A. Well, the cancer study started in 1979, and they accrued cases till 1981.

Q. And, Dr. Weisenburger, I'm going to have Mr. Wolfe zoom out -- zoom back in -- sorry -- and then go down to the methods on page 1.

If you could bring that out. Thank you.

A. So there are three case-control studies. Here they're talking about Nebraska. So Nebraska was the last case-control study to be done in this group, and so we accrued cases from 1983 to 1986. Okay?

Q. That's the Nebraska story?

A. That's the Nebraska study, yep. Yes.

And then Iowa and Minnesota -- if we can go to the next page -- Iowa and Minnesota was done just before the Nebraska study. So here you can see for Iowa the cases were accrued from 1981 to 1983, and for Minnesota from 1980 to 1982, and then the last study was the Kansas study from 1979 to '81. So it was basically cases were accrued from 1979 through 1983 in those three different studies.

Q. I'm going to show you a slide, Dr. Weisenburger.

MS. MOORE: Ms. Melen, if I could have the Elmo, please.

Q. And this was shown to the -- I'm sorry.

This was shown to the jury during cross-examination of Dr. Ritz --

A. Okay.

Q. -- by Monsanto's attorney, and you see at the bottom it's

talking about Iowa, Minnesota, Kansas and Nebraska from De Roos 2003. Do you see that?

A. Yes.

Q. Okay. As someone who is a co-author on the De Roos 2003 study, what is your opinion about the information contained on this slide?

A. Well, there's a mistake. So in Kansas it says 1976 to 1982 and it was actually 1979 through 1981. So there's a mistake there.

Q. Are the dates of collection important, Dr. Weisenburger?

A. Well, the dates are -- the dates are important because glyphosate came on the market as a formulation in 1975, and so one of the questions I think that has been raised was: Was there enough time -- was there enough latency, was there enough time to develop lymphoma from the time glyphosate came on the market until the time these studies were started and stopped? Okay? Because it takes time sometimes to develop cancer. It doesn't happen -- it usually doesn't happen quickly.

Q. And I'm going to stop you right there because you mentioned the term "latency," and the jury has heard a little bit about that. But have you published a paper about latency?

A. Yeah. So when they had that meeting at the National Cancer Institute to try to understand what was causing the increase in non-Hodgkin's lymphoma, I was asked to talk about the pathology and I also was asked to talk about the latency.

And so I wrote this paper and drew some curves to sort of illustrate the principles of latency.

Q. And, Dr. Weisenburger, I'll ask you to turn to 1570 in your binder.

MS. MOORE: And permission to publish, Your Honor.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE:

Q. And is this, Dr. Weisenburger, a publication you authored back in 1992, "The Pathological Classification of Non-Hodgkin's Lymphoma for Epidemiological Studies"?

A. Yes.

Q. And would it be helpful for you to use your bell curve, and we will -- Mr. Wolfe, if you could flip over to page 6, please, of the publication.

Would it be helpful if you could use the bell curve to explain the concept of latency?

A. Yes. So --

Q. And I have a blowup.

A. Okay.

MS. MOORE: Your Honor, permission for him to come down.

THE COURT: Sure.

BY MS. MOORE:

Q. Okay. And, Dr. Weisenburger, I'm going to have you come

right over here so the court reporter can also hear you.

A. I'll speak up.

Q. And if you can explain to the ladies and gentlemen of the jury what we're looking at here.

A. So this is -- there's two latency curves here. Okay? And by latency we mean how long does it take -- from the first exposure to a chemical or an agent, how long does it take to actually get the disease. Okay? And for cancer, it's usually years -- okay? -- Because it requires a lot of exposure and genetic damage to develop into a cancer.

So I drew two different curves. One is kind of based on my knowledge of the literature on solvent exposure. So solvents like benzine and paint thinners and those kind of things can cause non-Hodgkin's lymphoma. There was a nice literature on that, and what it said is that on average it takes about 20 to 25 years to develop non-Hodgkin's lymphoma. Okay?

So what that means is about half of the cases of non-Hodgkin's lymphoma develop in the first 20 years or 25 years, and the other half more or less develop later. Okay? So the curve can go anywhere from two years for the first cases all the way out to 30 or maybe even 40 years.

So when we talk about latency, we usually talk about the median latency, what is the average time it takes to get a cancer. And so this curve is a curve for what I would consider

repeated low-dose chronic exposure over many years, like a mechanic would get or a machinist who is using a lot of solvents. Okay?

Q. And is that curve B? Is that what you're referring to?

A. The curve B, yeah, the lower curve that goes out a long time. Okay? Because with low-dose exposures to agents, usually it takes a longer period of time.

The other curve shows what the curve might look like if you had very high exposures to a very toxic or carcinogenic agent. Okay? And in that case you would expect -- because it's high dose and it's very toxic, you would expect to see the cancers come up much earlier, the peak or the median would be much earlier, and then they would trail off a little bit like this (indicating) with a bit of a longer tail.

So I think for glyphosate it's more likely that it has a curve like this B curve, like we saw for solvents, where it takes a fairly long time to develop the cancers and one has to wait a long time to see all the cancers, probably out to 30 or 40 years.

So in the De Roos study, we would be looking at cancer that developed in the first part of this curve. Okay? The latency is short, but we know that there are people who get cancer early and there are people who get cancer later; right?

And so --

Q. Meaning from their first exposure?

A. Meaning from their first exposure.

So, you know, there are people who get cancer early, like at two years, three years, four years, five, ten years after exposure. Okay? And that's this part of the latency curve (indicating).

But you have to wait a long time to see all the cases. So the cases in the De Roos study would have been on the early part of this curve, and I think that the De Roos study is a valid study because of that.

Q. And, Dr. Weisenburger, what's the conclusions that were drawn, then, from the De Roos study in 2003?

A. Well, the De Roos study looked at a lot of pesticides, and one of the conclusions or one of the findings was that glyphosate gave an increased risk for non-Hodgkin's lymphoma of about twofold increased risk.

And the nice thing about the De Roos study is they could do this adjustment for confounding by other pesticides. So they could really focus more on glyphosate, what is the real odds ratio for glyphosate; and it was statistically significant even after all the adjustments for the use of other pesticides.

Q. Thank you, Dr. Weisenburger. Why don't you take a seat back. I'll pull this down.

THE COURT: Is now a good time to take a break and make sure our feet don't start swelling?

MS. MOORE: Yes, Your Honor, it is.

THE COURT: Okay. Why don't we take a break. We'll resume at quarter to 11:00.

MS. MOORE: Thank you, Your Honor.

(Proceedings were heard out of the presence of the jury:)

THE COURT: Be back at quarter till.

MS. MOORE: Thank you, Your Honor.

(Recess taken at 10:34 a.m.)

(Proceedings resumed at 10:47 a.m.)

(Proceedings were heard out of presence of the jury:)

THE COURT: Just before I forget, on Friday, we will end the trial day at 1:00 o'clock and not take a lunch break. So we will maybe have one extra short break, and I think we will be able to get in almost the amount of time that we are anticipating; but that's how the schedule will go on Friday.

MS. MOORE: Okay. Thank you, Your Honor.

MR. STEKLOFF: We might raise -- Your Honor, we should not do it now, just where we are on all witnesses at the lunch break because of flights. And so we want to see how far we get and then we can discuss it, if that's okay.

THE COURT: Okay. Go ahead and bring the jury back in.

(Proceedings were heard in the presence of the jury:)

THE COURT: You can resume.

MS. MOORE: Thank you, Your Honor.

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BY MS. MOORE

Q. Dr. Weisenburger, I want to go back to the *De Roos* 2003 article. I just have a couple final questions on that before we move on. If you could -- if we could publish that, if we can turn over to page 7.

A. What number is that?

Q. It's 451. And it is up on the screen too, Dr. Weisenburger.

A. Okay.

Q. I want to draw your attention on page 7, that's the paragraph. It starts the very last sentence there. And, Dr. Weisenburger, this last sentence that we have highlighted, if you could, it says: These few suggestive findings provide some impetus for further investigation into the potential health effects of glyphosate, even though one review concluded that the active ingredient is noncarcinogenic and nongenotoxic.

First of all, what does it mean to say "noncarcinogenic"?

A. Well, that means a chemical does not cause cancer, either in people or in animals.

Q. And then it says nongenotoxic. What does that mean?

A. It means that the chemical doesn't damage the DNA or the chromosomes that govern the cell.

Q. And for glyphosate, based on your review of the literature and your study of the causes of NHL in the last 40 years, do you agree with where it says this one review concluded that the

active ingredient, meaning glyphosate, is noncarcinogenic and nongenotoxic?

A. That review was written some time ago, and it was a review that was written by -- it was sponsored by industry. You know, today we know a lot more about Roundup and glyphosate. My conclusion was that from my review of the literature, the old and the new literature, that glyphosate is both genotoxic and carcinogenic.

Q. So it has got a footnote there, footnote 50, and if we could flip over to page 9 of the *De Roos* study. And, Mr. --

MS. MOORE: Thank you, Mr. Wolfe, great.

BY MS. MOORE

Q. We will highlight footnote 50. It is citing to an article by a G.M. Williams from 2000. Are you familiar with this publication?

MR. STEKLOFF: Objection, Your Honor. May we approach?

THE COURT: Sure.

(The following proceedings were heard at the sidebar:)

[redacted]

[redacted]

(Sidebar ended.)

(The following proceedings were heard in open court:)

BY MS. MOORE

Q. And, Dr. Weisenburger, the *De Roos* 2003 study, what is the significance of it today, 16 years later?

A. Well, it is still a very important study because it was a large pooled study that looked at a large number of different pesticides, including glyphosate and Roundup. And the importance for today, here, is that even after adjusting for the use of the chemicals, there was an over twofold increase in risk associated with glyphosate that was statistically significant. And -- so that's an important finding.

Q. And for the epidemiological studies like *De Roos*, are we

looking at glyphosate itself or are we looking at the formulation like Roundup?

A. Formulations like Roundup, yes.

Q. And so when you say it is an over twofold risk for developing non-Hodgkin's lymphoma, what -- what is an over twofold risk?

A. Well, that means that people who were exposed to Roundup were twice as likely to develop non-Hodgkin's lymphoma as people who weren't exposed to Roundup.

MR. STEKLOFF: Objection. I move to strike.

THE COURT: Overruled.

BY MS. MOORE

Q. And, now, did you rely upon other case control studies in forming your opinion in this case?

A. Yes. There were six case control studies.

MS. MOORE: Your Honor, if I can have him come down. I have shown this to counsel for defense already.

THE COURT: Okay.

BY MS. MOORE

Q. Dr. Weisenburger, we blew up the six studies. And is this a chart that you created?

A. Right. This is the table from my report on general causation.

Q. And I will tell you, Dr. Weisenburger, that Dr. Ritz went through each of these studies, so I'm just going to ask you if

you can summarize for the ladies and gentlemen of the jury what about these six studies -- let me move it up a little bit. It is kind of small -- what about these six studies did you rely upon in forming your opinion as to whether Roundup causes cancer -- causes non-Hodgkin's lymphoma in humans?

A. So the important findings from the studies are that five of the six studies, with the exception of *Orsi*, showed an increased risk for non-Hodgkin's lymphoma, a two to threefold increased risk.

Q. Which ones are those?

A. It's one, two, three, four and six. So five out of the six studies showed an increased risk. Here you see threefold risk. Here you see a twofold risk. I bolded the statistically significant increased risk so that they stand out a little bit.

Q. And so why did you bold the statistically significant ones?

A. Well, because I think that one can have more reliance on the numbers if they are statistically significant, okay. There is less chance for random error, okay.

Q. If the numbers are not statistically significant, do you ignore those numbers?

A. No. You look at all the numbers because you can gain information from looking at the numbers and how the numbers trend and how the numbers change. So you also look at the numbers that are not statistically significant but still

perhaps increased.

So here is the *De Roos* study with the increased risk at 2.1. And it is statistically significant, okay. And it was adjusted for other pesticides. There are two other studies that were also adjusted for other pesticides to rule out this issue.

One was the *Hardell* study. It is a small study with only eight exposed people. The risk there was a threefold increased risk. And when they adjusted for the use of other pesticides, the risk went down to 1.85, almost a twofold increased risk. So the risk decreased, which is what you would suspect if there are other chemicals causing non-Hodgkin's lymphoma, right? The risk would decrease, but it didn't go down to 1. It is still almost a twofold risk, even though it is not statistically significant. So we would consider that in evaluation of that study.

And the same thing happened when they did adjustments in the *Eriksson* study where the risk went from about 2 down to 1.5. It was statistically significant, but then it became -- it was no longer significant. It was elevated, but it was no longer significant. So again, you see the risk going down, but not going down to 1. There is still a 50 percent increased risk there, okay.

The other really important thing about -- about these studies is that a couple of the studies were able to look at

dose response. In other words, by dose response what I mean is if people were exposed to the chemical for longer or more intense exposure, you would expect if there was a dose response, the people with the low dose would have a low risk and the people with the higher dose would have a higher risk, right. It makes sense. So there were two studies that did that -- they had the ability to do that. One was the *McDuffie* study, and what they found was that if people were exposed two days or less per year, they made it by definition 1, okay. It is 1.

Q. What does that mean?

A. It means that they didn't really have an increased risk, okay. But if they were exposed more than two days per year, the risk increased to over 2, and it was statistically significant.

So this is a dose response. You have people who -- they have low exposure. The risk is not increased. If they have a high exposure, the risk is increased. And this 1.2 is really an average of these two numbers, okay.

They did the same thing in the *Eriksson* study. They divided their cases and controls into those that had less than -- ten days or less cumulative exposure, and those that had more than ten days of exposure, and the same thing happened; that there was an increased risk here. It was not statistically significant for those who had less exposure, but

it was over twofold increased risk for those who had more than ten days of exposure to Roundup. And it was statistically significant. And dose response is really important because it is confirmatory evidence that that chemical is actually having an effect.

Let's see what else is important. The two studies that weren't statistically significant are really the small studies, and don't have much power to find statistically significant increases.

MS. MOORE: Mr. Wolfe, if you can go back to 451, *De Roos* 2003, and if you can turn to page 5, table 3.

BY MS. MOORE

Q. I'm going to ask you, Dr. Weisenburger, you have got *De Roos* 2003 on your chart here; and you testified a few minutes ago there was an over twofold risk, increased risk. Is that this 2.1 number?

A. Yes.

Q. Okay. And when you look at the table, it shows on *De Roos* the 2.1. It is under the column Logistics Regression. What does that mean?

A. So logistics regression is the statistical method that they used to adjust for the other pesticides and to deal with this issue of confounding, okay. So it is a statistical method. They also used another statistical method called hierarchical regression, and when they did that, the risks --

the risks were lower. So the risk went to -- if I can write this here -- 1.6, and the confidence intervals were like this 0.9 to 2.8.

So when they use this hierarchical regression method, which is a more conservative method, the risk decreased but it didn't go to 1. It went down to 1.6, so the risk was still increased. It just --

Q. So what is the significance that it didn't -- when you use a conservative methodology that it didn't go to 1?

A. Well, that -- it looks like there is still an increased risk of about 60 percent, even when you use a very -- a more conservative method to do your adjustment.

Q. Thank you, Dr. Weisenburger. I will have you take a seat on the stand.

In addition to the *De Roos* 2003 epidemiological study, did you -- have you also participated in another pooled project?

A. Yes. Another pooled project, a more recent project, was a project called the NAPP study, which stands for North American Pooled Project. And in the NAPP study, the cases -- we used the cases from *De Roos* from the three North American case control studies, and we used the cases from *McDuffie*, which was the Canadian -- across Canada case control study. So they pooled the data from *De Roos* and from *McDuffie* to get, again, more cases so we could have more power to detect significant differences. And it also allowed us to -- in that much larger

group -- do adjustments for confounding due to use of other pesticides.

Q. When you say it allowed you because you had a larger group, can you explain why that is?

A. It is hard to do adjustments when you have a small number of cases, because if you do the adjustments, everything goes away. So you have to have larger numbers to have the statistical power to detect differences. So if you have large numbers, you can detect small differences. If you have small numbers, at best you can detect large differences; but often you can't even detect differences. So the idea was to pool all the data together into one bigger study where you had more statistical power to -- to look at the data and also you could do the adjustment for confounding of use of other pesticides. So that's what we did in the NAPP study.

Q. And the NAPP study, is that currently published?

A. It is currently submitted to a journal; has been reviewed; been sent back for revisions. So we are hoping it will be published in the next month or two, but it has not actually been published yet in a journal. We are close.

Q. What is an abstract?

A. So an abstract is a summary of the research. Often one writes abstracts when you want to present your research at a meeting of scientists. And so the NAPP study has been -- the data from that study has been presented at three different

international meetings over the last few years. And for each of those meetings, in order to get it accepted for presentation, you have to write a summary of your research and your findings and your conclusions. And that's called an abstract.

Q. Are abstracts peer reviewed?

A. Yes, they are. For meetings like this, they are peer reviewed. And, you know, they select the ones they think are the most relevant or the most important or the best.

Q. And for the NAPP abstract, it's been reviewed by your peers and approved for the authors, then to present on the data from the NAPP; is that correct?

MR. STEKLOFF: Objection. Leading.

THE COURT: Sustained.

BY MS. MOORE

Q. Has the --

THE COURT: When I sustain an objection, that means you don't answer.

THE WITNESS: I see. Thank you.

MS. MOORE: I will rephrase it.

Q. Dr. Weisenburger, can you explain to the jury what it means that the abstract of the NAPP has been peer reviewed?

A. So before the meeting happens -- a few months before the meeting happens they put on an invitation to submit your research if you want to present it at the meeting. So what you

do is you write an abstract. You send it into the organization that is sponsoring the meeting, and then they find experts who will review a stack of abstracts and rank them -- score them. And then usually the abstracts with the best scores are the ones that get presented at the meeting. The results from the NAPP study were presented at three different international meetings over the last few years.

Q. And who attends these international meetings?

A. Some of the meetings are mainly epidemiologists. Other meetings are a mixture of epidemiologists and cancer researchers and sometimes clinicians. It depends on who sponsors the meeting.

Q. And, Dr. Weisenburger, I'm going to ask you to explain the results from the NAPP, and would it be helpful to you to use a blowup to do so?

A. Sure.

Q. And did you pull out one of the charts from the presentation to do so?

A. Yes.

Q. Okay.

MS. MOORE: Your Honor, Dr. Weisenburger, can you come on down. I'm not going to have you go through the entire presentation because I think the jury would not want to sit for that, but I do want to pull up this chart from the presentation and --

THE COURT: I take it there is no objection to this?

MS. MOORE: Your Honor, I should have said on a break we went through all the blowups, and my understanding is there is no objection to any of those.

THE COURT: Okay.

MR. STEKLOFF: That's correct, Your Honor.

MS. MOORE: I'm sorry, Your Honor. I should have told you that.

THE WITNESS: So as I said, in the NAPP study, we pooled the data from *De Roos*, the three North American studies and across Canada study, and then did similar analyses like in the other papers. And one of the important findings from the NAPP study was the analysis of dose response. And like in *McDuffie* -- and this is the data for overall -- all of non-Hodgkin's lymphoma. So if you look at the number of days per year that it was handled, these are the people who didn't use glyphosate. By definition their risk is 1.

Here you have people who used it two or less days per year, and here you have people who used it more than two days per year. And what you can see is that for non-Hodgkin's lymphoma as a whole, there wasn't an increased risk for use less than two days or less per year. It is about -- approximately 1. It is a little less than 1.

But if you look at those who used it more than two days per year, the risk was almost -- almost a twofold increase. It

was statistically significant. And you can see that this is a trend analysis, a value for trend analysis. And it shows that there is a dose response here that is statistically significant.

BY MS. MOORE:

Q. And the overall, who falls in the overall category?

A. That is all non-Hodgkin's lymphoma.

Q. Okay. And with all -- overall with non-Hodgkin's lymphoma, you still saw the dose response?

A. Yes.

Q. Okay. And what is a p-trend?

A. P-trend just -- it tells you that this number is significantly smaller than this number, and the risk is increasing with increasing dosage. So it is a way to do a statistical analysis of is this number really significantly different than this number.

Q. Okay. And then you also have a column here for DLBCL. What does that stand for?

A. So that is diffuse large B-cell lymphoma. So one of the other advantages of pooling all the cases together in the NAPP study is you can look at actual subtypes of non-Hodgkin's lymphoma. Remember, we said there were a lot of subtypes. Well, this is the standard. It stands for follicular lymphoma. This is diffuse large B-cell lymphoma. This is small lymphoblastic lymphoma. They are the three most common types

of non-Hodgkin's lymphoma. And then they group all the other ones together in sort of an enterogenous category.

If you focus on the diffuse large B-cell lymphoma, which is the disease that Mr. Hardeman has, again you see for low dose exposure the risk is not increased. But for higher dose exposure, you have almost a 2.5 times increase in risk for non-Hodgkin's lymphoma, using it greater than two days per year. And, again, the trend analysis is statistically significant with the p-value of .02.

So this study shows that there are significant increases in non-Hodgkin's lymphoma as a group as well as for diffuse large B-cell lymphoma as one of the -- really the most common subtype of non-Hodgkin's lymphoma in North America.

Q. So the results of the NAPP study, how does that factor into your opinion in this case as to whether Roundup -- whether Roundup increases one's risk of developing non-Hodgkin's lymphoma?

A. Well, because you see an increase that is statistically significant with increased dose. You can see a dose response. You see it for all the different subtypes. Although, for the other subtypes, the diffuse large B-cell lymphoma, it is not statistically significant, probably because of small numbers. But for diffuse large B-cell lymphoma, you see this dose response.

The other thing that is important about NAPP is in NAPP

they were able to adjust for a whole bunch of other things that could be confounders, okay. So they adjusted for age and sex and state or providence, whether there was a history of genetic cancer in first-degree relatives which increases risk, whether it was a proxy respondent rather than the individual case.

Q. A proxy respondent, meaning --

A. It would be a wife of a man who died of non-Hodgkin's lymphoma or the husband of a woman who died of non-Hodgkin's lymphoma.

Use of protective equipment because that decreases risk. And then most importantly they adjusted for three pesticides -- three herbicides -- 2, 4-D, dicamba, and malathion -- actually these are insecticides. So they adjusted for other pesticides that are known to cause non-Hodgkin's lymphoma, and the use was correlated with the use of glyphosates. So these are the important chemicals to adjust for so that we know we are looking mainly at the effect of glyphosate and not at the effect of 2,4-D or dicamba or malathion.

So these numbers are all adjusted to rule out confounders. And it is the most powerful study of the case control studies that does that, okay.

Q. All right. Thank you, Dr. Weisenburger. I will have you go ahead and take a seat.

The jury has heard testimony also about the Agricultural Health Study. Are you familiar with that?

A. Yes. I also reviewed both the early paper on the Agricultural Health Study as well as the recent one, which was published last year.

Q. And did you consider the publication -- the two publications from the AHS in forming your opinions in this case?

A. Yes, I did.

Q. And what is -- and how did these two publications from AHS factor into your opinion in this case?

A. Well, I considered them because I think the Agricultural Health Study is an important study of -- but it -- its results don't agree with the case control studies. It didn't really show an increased risk for non-Hodgkin's lymphoma.

Q. Is the AHS study a different type of study than the six case control studies that you have highlighted for the jury?

A. Yes. So the -- so the Agricultural Health Study is what we call a cohort study. So I don't know whether Dr. Ritz explained that.

Q. She did, but if you want -- if it is important for you just to briefly --

A. What they did in the Agricultural Health Study is they took licensed pesticide applicators -- so they were mainly farmers but also commercial applicators -- and they identified this group because they all had to take an exam to -- to have -- to use certain restricted pesticides. So they were

either farmers or pesticide applicators from either Iowa or North Carolina, I believe. And they were able to collect a large number, I think about 50,000 farmers and pesticide applicators.

And the idea was we are going to gather data on them at the time of the start of the study, and then we are going to follow them for 10, 20, 30 or more years and see which ones get non-Hodgkin's lymphoma; and they also looked at lots of other cancers. Non-Hodgkin's lymphoma is just one. And then see over time who gets the non-Hodgkin's lymphoma, and then see if you can relate back to their exposures to the different chemicals to try to figure out which of the chemicals were causing the non-Hodgkin's lymphoma.

So that was the design of the cohort study. It is a different design than the case control study.

Q. And in what way?

A. Well, in that -- in the cohort study you gather -- in the case control study you just gather your information at one point, and you are kind of looking backwards at what were the exposures before you got the non-Hodgkin's lymphoma, and you are comparing the exposures in the cases with lymphoma to those who didn't have lymphoma. So that's your cases in your controls.

In the cohort study you are starting with a whole group of people who don't have cancer, okay. So you kind of eliminate

all those people who have had cancer, and you just pick people who don't have cancer and haven't had cancer, okay. So it is kind of a -- it is a group that you can follow that doesn't have cancer, and then you see who is going to get the cancer over a long period of time.

So you gather data. At the initial registration you ask them -- you know, just like in the case control study, you ask them what did you use, how long did you use it, how many years did you use it, how many days per year did you use it, et cetera. And then once you have that baseline data, then the idea is that every few years you recontact them and you get new information. Now what are you using, how are you using it, did you stop using this, did you start using that, how much are you using. And you can calculate. You can see what happens over time, okay. So it's a prospective study.

So that's the design of the Agricultural Health Study.

Q. So when determining your opinion in this case, how much -- well, what weight did you give to the Agricultural Health Study?

A. Well, I didn't give it a lot of weight. I weighted it probably like I weighted each of the case control studies. And the reason I didn't give it a lot of weight, because there are some real significant issues and problems with the Agricultural Health Study, particularly with regard to Roundup, okay, because of how they did the study and how Roundup increased

dramatically during the middle of the first phase of the study.

Q. That was in and around 1996?

A. Yes.

Q. And what about the fact that Roundup sales increased in the '90s -- do you believe -- how did that impact the AHS?

A. Well, right at the end of the registration period when they were getting the initial information from the applicators, the genetically modified crops started being marketed by Monsanto. And so farmers liked using these new seeds because they could plant the crop and then use the pesticide to get rid of the weeds.

MR. STEKLOFF: Objection --

THE COURT: Hold on a second. There has been an objection, but it is overruled.

You can continue.

THE WITNESS: So anyway, it became very popular among farmers to use these genetically modified plants, corn and soybean and others, and then use glyphosate or Roundup to treat because they could -- they didn't have to worry about filling the corn or the soybeans because they were resistant. They just killed the weeds. So it wasn't -- I think it was a scientific breakthrough.

But this markedly increased use took off right at the latter part of the registration period, and then continued to go for years and years, okay, to go up. And I think you see --

you have seen the diagram of that earlier in the case.

So -- and then they did their first follow-up interview -- or follow-up questionnaire. So about five years later after the initial registration and gathering the first set of data, they contacted all of the people -- all the applicators, and they asked them, Well, what is -- what has happened in between?

But one of the problems is they didn't ask for each year in between. They just asked for the last year that they farmed. So they didn't get data on all of the -- all the information that they should have gotten.

And then the other really big problem with this study is that only 63 percent of the applicators responded to the questionnaire. So there was a large proportion of the applicators who didn't respond to the second questionnaire, okay. So they had no data on what happened to them in terms of their pesticide use and other things after that initial registration.

So to have a really good, successful cohort study, it is really dependent on getting good information at the different periods as you follow the people because of the changes -- their pesticide use changes. They use new pesticides. They stop using pesticides. They start using some pesticides more, like glyphosate. And so you have to gather all that data, okay, to really make sense of what happens in the end.

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BY MS. MOORE:

Q. And if you aren't able to gather all the data, then what happens?

A. Well, you misclassify people. So, for example, if somebody at the start of the study didn't use Roundup and then, say, in 1996 they say, Well, this is great stuff. I'm going to start using the Roundup, but they don't answer the follow-up questionnaire, you wouldn't know that they started using Roundup. And so when you use the data from the initial questionnaire, they would be one of your non-users but, in fact, they were using it, okay.

The same thing happens if they were using it -- say they were using it in small amounts, okay. And then suddenly decided, Well, I'm going to treat my corn with glyphosate and I'm going to use these new seeds, okay. So they go from a low user to a high user. But if you don't gather the information about what happened, you think they were a low user when they were really a high user, okay.

So there was a lot of this exposure misclassification that occurred in the people who didn't fill out the second questionnaire. And then they also had -- they had no real information on the people who did fill out the second questionnaire for all the years between the initial registration and the last year of farming, okay. So there was a lot of information that was missing, okay.

So this is a bad thing for a cohort study because you could say, Well, I'm just going to analyze the data for the people who filled out the first questionnaire and who filled out the follow-up questionnaire. I'm going to note all those people who didn't do the follow-up questionnaire, all right. But what happens then is you have a selection bias because the people who filled out the questionnaire the second time may be very different from the people who didn't fill it out, okay. And, in fact, this was true in this study.

So the alternative was to do this imputation, and probably Dr. Ritz told you about that. What they did is they -- based on the characteristics of the group that didn't fill out the questionnaire the second time, and the people who did fill out the questionnaire the second time, they tried to guesstimate -- basically guesstimate what the first group who didn't fill out the questionnaire would actually have done. So they attributed to them some use, okay, or no use. And they had to do that also for the people who filled out the second questionnaire because they didn't have use data for 6 to 12 years.

So there is a potential here for -- a significant potential for what we call exposure misclassification, okay. It was what we called nondifferential; that it could go either direction, okay. And when that happens, the power of the study is markedly decreased because you have got a lot of noise, and it becomes much more difficult to detect a true increased risk

because of all of the misclassification of the cases. And this is the major problem with the Agricultural Health Study.

So I didn't -- I evaluated it. I considered it. But I didn't give it any more weight than any one of the case control studies.

Q. And, Dr. Weisenburger, if someone only looked at the AHS study, the publication AHS study, and did not look at the case control studies, what would you say about that?

A. Well, it's not valid because you should look at all the epidemiologic data. And, I mean, if you take a superficial look at the Agricultural Health Study with regard to Roundup, you might think there is no increased risk. But if you really understand what happened in this study, you can say, Well, you know, maybe this is -- this study is a false-negative. Maybe there really was a risk there, but because of the fact that people didn't fill out the follow-up questionnaire and didn't gather all the data on the people who did, maybe this study isn't as informative as it could have been.

Q. And I want to switch gears now, and the jury has heard about something called meta-analysis. Did you review meta-analysis in forming your opinion in this case?

A. Yes. So there were a number of meta-analyses that were done, including the five case control studies that didn't include the Cocco study because it was a small study, so including the five case control studies that we talked about

already as well as the Agricultural Health Study.

Q. Let's turn to one of the first ones, and it's 1102.

MS. MOORE: Permission to publish.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE

Q. Are you familiar with the Chang publication from 2016?

A. Yes. So this is an industry-sponsored review of glyphosate exposure, and --

Q. Dr. Weisenburger, if I can stop you for a second. What do you mean when you say "industry sponsored"?

A. Well, these people were hired by industry to write this paper, okay.

Q. And I think if we turn to page 24, there is a disclosure as to the funding. And who funded the -- oh, I'm sorry. Who is listed under the funding section of the Chang meta-analysis?

A. Monsanto.

Q. And what is Chang -- what conclusions did you draw from Chang?

A. Well, Chang did a very detailed analysis that -- Chang and Delzell are both epidemiologists. They did a detailed analysis. They came to a different conclusion than I did, than the IARC did, with regard to the case control studies, and they --

MR. STEKLOFF: Objection, Your Honor.

THE WITNESS: -- put a lot of reliance --

THE COURT: Hold on. There is an objection.

Basis?

MR. STEKLOFF: Motion in limine Number 1 from the --

THE COURT: Overruled.

BY MS. MOORE

Q. You can go ahead.

A. So they did a meta-analysis including the -- and including the first AHS study, the one by *De Roos*, okay. And they found an increased odds ratio of 1.3 that was statistically significant. So taking all of the data from the case control studies and the AHS -- the AHS, the cohort study, they still found an increased risk of 30 percent that was statistically significant.

The IARC did the same thing and had the same finding, okay. And the first case -- and the first meta-analysis had a slightly higher finding, but they didn't do all the adjustments that IARC and Chang and Delzell did. So all of the --

Q. Which -- sorry, Dr. Weisenburger. Which is the first meta-analysis that you are referring to?

A. By Schinasi.

Q. Schinasi. Okay.

And you also mentioned that IARC did a meta-analysis, and what was the overall conclusion from IARC with respect to glyphosate?

A. Well, the overall conclusion was that it is a probable carcinogen. They gave it a rank of 2A. So we say it is probably carcinogenic in humans.

The IARC finding was the same as the Chang and Delzell finding because they did the analysis the same way.

Q. And then if we -- there was -- I think you mentioned there was a fourth meta-analysis. And which one is that?

A. So there was recently a meta-analysis that was done by some researchers from the University of Washington. Zhang is the first author.

MS. MOORE: If we could -- I would ask if we could publish 554.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

THE WITNESS: What is the number?

MS. MOORE: 554.

THE WITNESS: Okay.

BY MS. MOORE

Q. Is this the Zhang meta-analysis?

A. Yes, it is.

Q. And you said recent. When did the Zhang meta-analysis, when did it come out for publication?

A. Well, it -- it is -- it was just accepted for publication. So it hasn't actually been published, but they put the paper online so people could read it actually before it is published

in the journal. So this is the online version of the paper.

Q. And it looks like it is a month old today; right?

A. Yes.

Q. And in the -- and did you rely on the Zhang meta-analysis in forming your opinions in this case?

A. No, I didn't because it just came out so I only saw it a couple -- three weeks ag. But it --

Q. Have you reviewed it since then?

A. Yes, I reviewed it, and it supports all the other information that I reviewed that -- and we will look at the findings.

Q. Do you want to look at the findings in the tables, Dr. Weisenburger?

A. Yes. So I think we can go to table 5, it is --

MS. MOORE: Page 3, Mr. Wolfe.

THE WITNESS: -- gives you the -- sort of the meat of the paper. And so this is a new meta-analysis, but it is different than the other ones that were done in the sense that it includes the updated AHS, the 2015 AHS. It includes the updated AHS; although, they did look also at the 2005 AHS, like the other meta-analysis.

But what they did is they focused on the people who had high exposure, okay. The other meta-analysis just looked at ever-never. So they included everybody who was exposed, low exposed and high exposed. This study focused on the people who

seemed to have the most exposure. So if you look at the first column to the left, it says highest cumulative exposure.

BY MS. MOORE

Q. Then below that, Dr. Weisenburger, I think you said 2015 earlier. There's two publications out of the AHS, 2005 and 2018; is that right?

A. So they did the analysis for the data in both, but I think we should focus on the 2018 because that is the most recent data. So they tried to take the data on the higher exposed people in all of the studies, the case control studies, as well as the AHS 2018.

And if you look across you can see the odds ratio is 1.41, and it is statistically significant using a method called fixed effects. And they used another statistical method to also look at it called random effects, and again they saw an increased risk. It was a little bit higher, and it was statistically significant.

And the data was not too much different between the two AHS studies. You can see that the next line is the 2005 AHS, it is also pretty much the same. And that is for highest cumulative exposure. So these would be the people who had high intensity or frequent use, okay.

And then they did another analysis where they looked at the longest exposure, so people who had many days of exposure, looking at long exposure. And, again, they looked at the 2018

and the 2005 AHS. And, again, the numbers are very similar to what they saw above; that there was a 40 to 50, almost 60 percent increase in non-Hodgkin's lymphoma. And on -- all the numbers are statistically significant here, okay.

So I think that's all I want to talk about on this table. We should go to the next table because I think it is also informative, table 6. And let's focus on where it says other pesticides, adjusted, unadjusted, yes.

So what they did here is they -- where they could, they adjusted for the use of other pesticides to get around and to mitigate this issue of confounding the use of multiple pesticides. And if you look at the unadjusted odds ratio using 2005 AHS, there is about a 70 percent increase, okay. It is statistically significant. But when you adjust it, the odds ratio goes down. And that makes sense, because now they are taking away the effects of the other pesticides that could have caused non-Hodgkin's lymphoma, and they are focusing just on Roundup.

And, again, the numbers are about the same as what we saw before. It is 1.46, so a 46 percent increase risk of non-Hodgkin's lymphoma. Again, it is statistically significant.

So I think this is really important data because it looks at -- it is a meta-analysis. It looks at data a little bit differently. It focuses on the people who have high exposure.

And it uses the new data from the Agricultural Health Study. And it also attempts to do adjustment based on pesticide use. So I think it is an important, very informative study.

Q. And does the Zhang study, did it look at -- it is a meta-analysis of epidemiology. But did it look at the other legs of the stool?

A. So, yeah, they did an interesting thing in this paper. So they didn't just publish the meta-analysis. They also looked at the other two legs of the stool.

So they looked at the animal studies, just as I did. They looked at the mechanistic studies, just as I did. And they came to the same conclusion that I did, in that they felt that all of this data when put together makes a very compelling argument that glyphosate and Roundup cause non-Hodgkin's lymphoma in people. And so the study I think is a very informative, very important study.

Q. And let's look -- are you familiar with the conclusion from the Zhang meta-analysis?

Let's go to page 2. And at the end there, Dr. Weisenburger, do you agree with the conclusion from the Zhang meta-analysis that Overall in accordance with evidence from experimental animal and mechanistic studies, our current meta-analysis of human epidemiological studies suggests a compelling link between exposures to glyphosate-based herbicides, which is Roundup, right, and increased risk for

NHL?

A. I agree with it. I would even make a stronger statement and say that it is a compelling argument.

Q. Let's move to the second leg of the stool, and that's the animal studies. Did -- and they heard from Dr. Portier last week for a couple of days, and I want to just ask you: Did you consider the animal studies?

A. I did.

Q. Okay. And what about the animal studies -- we are not going to go through each one of them. What about the animal studies was important in reaching your opinion in this case?

A. Well, the animal studies were important because there were a number of studies that showed that feeding the -- either mice or rats glyphosate in their feed, or Roundup, increased the risk for tumors. I think I counted 13 studies that I wrote in my report. And in mice, for example, the chemical Roundup -- glyphosate actually caused the mice to get some rare tumors that they don't usually get, kidney tumors, okay, both benign and malignant tumors.

Q. When you say "benign and malignant," what is the difference there?

A. Well, there ware benign tumors that grow, that don't spread and kill the animal or the human; but they are often part of the stage of developing a malignant tumor. So you might develop first a benign tumor like a colon polyp, and then

you are at increased risk for developing colon carcinoma. That's why they take out the colon polyps.

So the animals would develop benign tumors and sometimes malignant tumors. Some of the animals got rare tumors that they usually don't get. Interestingly in a number of mice studies -- actually, four out of the six studies of mice -- glyphosate caused an increase in non-Hodgkin's lymphoma, the same cancer that we were seeing in the epidemiology studies.

Q. What is the significance of that?

A. Well, it is an interesting result because often you don't see that in animal studies; that the chemical causes the same tumors in the animal and in the human. But it is an interesting finding that I think gives some initial weight to my conclusion.

Q. So what conclusion then can you draw from the animal studies?

A. Well, I think the animal studies show that glyphosate -- glyphosate is carcinogenic in animals, in mice and in rats. It causes benign and malignant tumors at excess -- in excess numbers in these animals.

Q. You mentioned during the -- your testimony about the epidemiological studies, dose response.

A. Yes.

Q. And is that something that you can look for in animal studies?

A. Yes. So you have a group of animals that are your control animals that don't get the agent of interest. And then you have low dose, usually three or four doses: Low dose, intermediate dose, and high dose.

For high dose you try to give them a dose that they can tolerate that won't make them ill, okay. And usually what you see in the animal studies is a dose response. And in a number of the studies, they did see a dose response. In some of the studies they just saw the tumors in the animals that got the highest dose, okay, but that's what you would expect.

Q. Then the third leg of the stool -- is there anything else you want to say about animal studies, Dr. Weisenburger?

A. No.

Q. Okay. And then the third leg of the stool, the mechanistic data, did you also review the literature regarding mechanistic data?

A. I did. I did. So this is mainly information on the genotoxicity of glyphosate or Roundup; that is, you know, if you take cells or you treat animals with these chemicals, do they -- can you find evidence of DNA damage? And, of course, like all cancers, non-Hodgkin's lymphoma is a genetic disease. So genetic abnormalities occur in non-Hodgkin's lymphoma that actually are the -- are in the end what causes the disease.

So if you can show that the chemical is genotoxic in animals or in cultures of human cells, then this is just

another important piece of information. So I looked at the genotoxicity studies, and I also looked at the number of studies that looked at other effects of either Roundup or glyphosate in terms of does it affect other functions, like does it change how cells respond in culture and grow, or -- so I looked at a whole variety of things, including does the chemical cause oxidative stress because oxidative stress is another way that you can damage the DNA.

So by oxidative stress what I mean is that if you get a chemical, it causes a stress in the cells, okay. And one of the things that happens when the cells are stressed is they produce these things called oxygen radicals. And the oxygen radicals can damage the DNA. So it is sort of an indirect method for a chemical to damage the DNA. And glyphosate was found in many of these studies to cause oxidative stress and to produce these free radicals, and to -- it was also genotoxic.

Q. So let's look at a few of the studies that you relied upon, and I will have you turn to -- in your binder, it is 916.

MS. MOORE: Permission to publish?

THE COURT: Can I just ask, Ms. Moore, what are you -- about how much more time do you have? Should we break for lunch now or press ahead a few more minutes?

MS. MOORE: I will leave that up to the jury. But if I can get through the mechanistic, I can definitely do that in probably ten minutes.

THE COURT: Okay. Why don't we go ahead and do that and then we will take our lunch break.

MS. MOORE: Great.

Permission to publish.

MR. STEKLOFF: No objection, Your Honor.

MS. MOORE: 916.

Q. And, Dr. Weisenburger, tell the ladies and gentlemen of the jury what is this publication.

A. So this is a publication that looked at a variety of different pesticides and evaluated what was their potential to induce what we call double-strand breaks. These are breaks in chromosomes that cause the chromosomes to rearrange, okay. And that is a common finding in non-Hodgkin's lymphoma. So they looked at lymphocytes, human lymphocytes, the cells that are the precursor cells for non-Hodgkin's lymphoma. They put normal human lymphocytes from healthy donors in cultures and then they treated those cells with glyphosate.

Q. So you have got a petri dish in a lab, and they are putting the glyphosate in the petri dish? Is it glyphosate or Roundup in this instance?

A. I think this one is glyphosate.

Q. Okay. All right. And did you rely on the conclusions of this publication in forming your opinion in this case?

A. I did, because what they showed was an increase in double-strand breaks when the cells were treated with

glyphosate, and they showed a dose response that if they gave -- if they put more glyphosate into the cultures, there were more double-strand breaks, okay.

Q. And if we could, go to -- Dr. Weisenburger, in forming your opinions, did you then take that data and put it on a chart?

A. I did. It was part of my presentation to the judge at the Daubert hearing.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Okay.

THE WITNESS: So this just shows the data from this study, and what it shows is that glyphosate induces these double-strand breaks in the chromosomes of cultured human lymphocytes, even at low doses.

So what you see in this table is the data for glyphosate. And if you look across where it says zero under dose, those are cells that didn't -- they didn't put any glyphosate into the cultures. So you get a fairly low level of DNA damage.

At the bottom there is another compound called etoposide. Etoposide is the chemotherapy agent which they gave at a high dose just to show that they could cause damage in the cell. So that is what you call your positive control, and the negative control is no glyphosate in the cultures.

Then they increased the doses in micromolar doses, .4, 2.0, 10.0 and then high dose of 50. And what you see, if you

look at the next column under Mean percent cells, is you see the increase in DNA damage in double-strand breaks going from .33 with no exposure to a fivefold increase, 1.67. And then to a number -- another four or fivefold increase to 9.33 with a higher dose. So you see a dose response here.

As you give higher doses, it actually begins to go down, and that's because in this case the glyphosate was toxic to the cells and so the cells just die and you don't see as much. But if you look at the first three rows, you can see the dose response. And so -- and to the right you see the p-value, which is statistically significant.

So what the study shows is that glyphosate can cause DNA damage. It is genotoxic. It causes double-strand breaks. In human lymphocytes the same cells that develop -- the normal cells that become malignant in non-Hodgkin's lymphoma.

BY MS. MOORE:

Q. And, Dr. Weisenburger, what is the significance that glyphosate can cause DNA damage in the human lymphocyte cells in the petri dish?

A. Well, as I said, it shows that it is genotoxic. And it actually causes DNA in the normal cells that -- when they become malignant, they are called non-Hodgkin's lymphoma. So these are the same cells that we are talking about in the non-Hodgkin's lymphoma, same kinds of cells.

Q. All right. Let's go to 562.

MS. MOORE: Permission to publish?

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE

Q. And what publication is this, Dr. Weisenburger?

A. So this is another publication that did a number of different kinds of tests to determine whether glyphosate was genotoxic or not. And they also -- they looked at glyphosate and they also looked at Roundup. So they were evaluating both formulation and the -- the formulation and the active compound.

Q. And this is from 1997; is that right?

A. Yes, it is.

Q. If we could --

MS. MOORE: Mr. Wolfe, if you could go over to page 1960 and figure 3.

BY MS. MOORE

Q. And, Dr. Weisenburger, if you could explain to the jury what we are seeing in figure 3 -- and do you want to pull up one or two of the bar graphs?

A. Pull them both up. That would be all right.

So this is a test called sister chromatid exchange. They did a different kind of a test to look for DNA damage called sister chromatid exchange. Again, these are human lymphocytes from normal donors. And on the top scale they used glyphosate.

Q. I think you can touch the screen and show --

A. There it is.

So along the bottom you can see the dosages. The C is your control, no glyphosate in the cultures. And then they use low doses, .33 milligrams per mL; 1, 3, 4 and 6 milligrams per mL. So they used increasing doses. And what you see is a dose response. And the last three columns, because they have the little asterisks on top, are statistically significantly higher than the control.

So what you are seeing here is if you put just glyphosate into the cultures with the lymphocytes, you see increased sister chromatid exchanges, which is an indicator of DNA damage and genotoxicity.

And then the lower scale is Roundup. And here you see the same effect. You have the control and when you use very small amounts of Roundup, .01 milligrams per mL, and .33 grams per mL, you can see you get statistically significant increases in the sister chromatid exchange, and it is very similar to what you see for glyphosate. But the interesting thing is that you have got to use 3 milligrams per mL to get this effect where you see the fourth column with the asterisk.

See if I can show it here. Hang on. This is not working so well.

Q. Which one did you want highlighted? And we can have Mr. Wolfe do that for you.

A. The fourth one on the top.

Q. Okay.

A. That one, yeah. If you look down here, you see the same effect with Roundup; but you have to use ten times more glyphosate than Roundup to get the same effect. So what this shows you is that Roundup is much more genotoxic in human lymphocytes than glyphosate is.

Q. So the top -- the top graph is putting glyphosate in the petri dish?

A. Right. And if you just look at the 3 milligrams per mL, you can see that the sister chromatid exchange per cell is about 5. And then you go down to the lower one, they are using one-tenth of the dose of glyphosate in Roundup, and they get the same effect, telling you that Roundup in this study is ten times more genotoxic than glyphosate, okay. And that's been shown in many other kinds of studies; that Roundup seems to be more toxic than glyphosate. But here you see the genotoxicity.

Q. So the formulation is ten times more toxic?

A. Yes.

Q. All right. Then the last one, Dr. Weisenburger, 560 -- did you want to say anything else about this study, I'm sorry?

A. No.

Q. 563.

MS. MOORE: Permission to publish.

MR. STEKLOFF: No objection.

THE COURT: Go ahead.

BY MS. MOORE

Q. And what is this publication here?

A. So this is another study looking at peripheral blood mononuclear cells, which includes lymphocytes. And, again, they are looking at Roundup. And they are looking at glyphosate and they are looking at a different test for DNA damage called the common test. But it's another way to look at DNA damage in cells, including mononuclear cells in the blood and lymphocytes.

Q. And are there some graphs you wanted to point out in this publication?

A. Yes.

MS. MOORE: Can we turn to page 515, please. One more. Thank you.

THE WITNESS: This one. So let's just focus on the second graph first, if we could.

So this is a graph like I showed you before. On the lower axis you see concentrations of glyphosate in micromolar concentrations. And on the other scale you see DNA damage, okay. And so what you see is at low doses of glyphosate, you don't see much in the way of DNA damage. The dark bar consists of two types: Single-strand breaks, and the white bar is double-strand breaks.

But as you increase the dose there, you see -- if you look across at 250, you can see that the combination of

single-strand and double-strand breaks, which is the black bar, is statistically increased. If you can continue to increase the dosage of glyphosate to 1,000 micrograms per mole, you see a statistically significant increase in combination of single- and double-strand breaks, and the bar for double-strand breaks, which is the smaller white bar to the right is also statistically significant. So, again, it is another example of seeing a dose response and seeing genotoxicity in peripheral blood mononuclear cells that is significant.

BY MS. MOORE

Q. And then the glyphosate.

A. This is glyphosate. So let's look at Roundup --

MS. MOORE: So A, please.

THE WITNESS: -- which is the top one. And for Roundup, you see the same thing basically, except that, again, the doses of Roundup are much smaller than the doses for glyphosate. So if we just look at the last column, you see both the single- and double-strand breaks as well as the double-strand breaks are statistically significantly increased. There are only 10 micromolar concentration. And you remember the glyphosate one was 1,000 micromolar. So in this study the Roundup is 100 times more toxic than the glyphosate.

So when you do studies of just glyphosate, you might not find much in the way of effects. But if you do the studies on Roundup, you are much more likely to find genotoxic effects.

And that's what these recent studies did.

So those are just three examples of recent -- old and recent genotoxic studies that show that glyphosate, and particularly Roundup, are genotoxic in cells. I showed you data for lymphocytes because I think to me that is the most important data because those are the cells that eventually would become non-Hodgkin's lymphoma.

MS. MOORE: Your Honor, I'm going over my time, so I'm going to stop right there.

THE COURT: Okay. Very good. Why don't we break for lunch.

Remember all the admonitions about not talking about the case and not exposing yourself to other information about it. We will resume at 12:45. Thank you.

(Proceedings were heard out of presence of the jury:)

THE COURT: Remember, everybody has to stay in the room for five minutes so we can give the jury a chance to get out and use the elevators and stuff.

I had one thought that I was just pondering. We don't need to discuss it now, but I just wanted to plant the thought. You know, as I'm watching all this expert testimony and seeing all these excerpts from studies being pulled up, you know, I'm anticipating jurors wanting to bring the studies back with them in the jury room. I understand the problem with doing that.

But one thing I was curious about is would there -- would

there be any problem with sending back just the excerpts, just the portions that are published to the jury? That is something I would like you-all to ponder and get back to me on because I'm anticipating that that -- you know, that -- to me for the -- putting myself in the shoes of the jury, getting at least that, would be a lot more satisfying, potentially, it seems to me, than getting -- than being told you can't have anything at all. So think about that.

MS. MOORE: Thank you, Your Honor.

MR. STEKLOFF: Your Honor, can I just raise timing for our witnesses quickly?

THE COURT: Sure.

MR. STEKLOFF: So because of the delay yesterday, we told Plaintiff's counsel that we would be calling Dr. Mucci first tomorrow. So Dr. Mucci will be here tomorrow.

THE COURT: Okay.

MR. STEKLOFF: My sense of where we are right now -- and Dr. Weisenburger may not be happy to hear this -- and I will make every effort to be efficient -- that he might not finish today.

So if we go a little bit with Dr. Weisenburger tomorrow, plus I think there is an hour of deposition testimony between Dr. Reeves and Dr. Farmer -- I think it is almost exactly an hour -- 55 minutes and we have Dr. Mucci.

I'm sort of asking for permission that Dr. Arber does not

need to get on a plane tonight. He can get on a plane obviously to be here for Friday. Our plan would then be to fill the time with Dr. Mucci tomorrow; see if we even finish her, and then continue with either Dr. Mucci on Friday or Dr. Arber followed by Dr. Levine.

THE COURT: So you would have Arber and Levine ready on Friday?

MR. STEKLOFF: They are both ready -- but Dr. Arber just needs to know if he needs to get on a plane today under what I think is an unlikely possibility that he will get called tomorrow.

THE COURT: Somebody just walked out of the courtroom.

THE CLERK: Yeah.

THE COURT: So I don't know -- does anybody know who that was?

MS. MOORE: I didn't see, Your Honor.

THE COURT: All right. I remember who she is. So, Kristen, I'm going to ask you -- I will point her out to you, and I will ask you to speak with her if you see her again in the courtroom.

THE CLERK: Okay.

THE COURT: Disregarded my orders by leaving the courtroom.

Okay. So I mean, let's talk about it again at the end of the day. As I sit here right now, that sounds like it's okay;

but, you know, again, you are running a risk. I mean, if there is a significant amount of time -- if we end at 2:10 or something like that, fine, no big deal; but if you leave us with a significant amount of dead time, it's going to come out of your time.

MR. STEKLOFF: Well, we have a lot of time. I would rather be efficient and not waste the jury's time. I understand that risk. It is just the question of Dr. Arber's -- we are trying to be as flexible as we can, understanding that the jury takes priority for Dr. Arber as well.

THE COURT: Okay. Great, thank you. Sorry. I should have told you you could step down.

THE WITNESS: That's okay. I kind of like it up here.

THE CLERK: Court is in recess.

(Luncheon recess was taken at 12:04 p.m.)

AFTERNOON SESSION **12:45 p.m.**

(Proceedings were heard in the presence of the jury:)

THE COURT: All right. We are back. You can resume.

MS. MOORE: Thank you, Your Honor.

BY MS. MOORE

Q. Good afternoon, Dr. Weisenburger.

A. Good afternoon.

Q. When we broke for lunch, we were talking about genotoxicity; and my question for you is: You spent time

showing the jury these studies about what happens to the lymphocyte cells from humans in petri dishes, but what about the real world? How do we know Roundup is genotoxic out in the real world?

A. Yeah, so there are two studies done in South America that I think are informative to answer that question.

Q. If you want to turn to -- it looks like Exhibit 1438 in your binder.

MS. MOORE: And permission to publish.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Okay.

BY MS. MOORE

Q. Is this one of the studies that you are referencing, Dr. Weisenburger?

A. Yes, this is a --

Q. Can you --

A. -- a study of actual people in Ecuador who were exposed to large doses of Roundup herbicide sprayed from airplanes to eradicate cocaine plants.

Q. And what were the findings from this study?

A. Well, this was a relatively small study. These were rural -- rural inhabitants, farmers living right on the border of Ecuador and Colombia. And they didn't use pesticides in any of their farm work. And Colombia was spraying for these cocaine -- these cocaine plants trying to eradicate cocaine,

using Roundup -- a more -- a stronger formulation of Roundup. And they actually did three days of intensive exposure right on the border, so these people got exposed multiple times. The stuff drifted on over to their homes, and they were exposed. And then they -- after that three days of intense spraying, there were sporadic spraying for the next three weeks, so they were exposed over a period of months multiple times to high doses of Roundup that was sprayed from these airplanes.

MS. MOORE: And if we could, Mr. Wolfe, if we can go to the abstract on page 1.

BY MS. MOORE

Q. The last sentence in the abstract. Dr. Weisenburger, so what was the results -- what did the results show these authors in this particular study?

A. Well, what the results showed is that the formulation of Roundup that was used during aerial spraying had genotoxic effects on the peripheral blood cells from these -- of these people who lived in -- and were sprayed inadvertently.

Q. And I will show you -- I think that you extracted this data --

MS. MOORE: If we can go to your PowerPoint slide on that.

MR. STEKLOFF: No objection, Your Honor.

MS. MOORE: Thank you.

\\\

BY MS. MOORE

Q. What do we see here, Dr. Weisenburger?

A. So this is just the summary of the data. These individuals used the comet assay, one of the same assays that was used in the genotox studies I told you about before.

Q. You say "assay." What is that?

A. A test.

Q. A test?

A. Yeah, a test.

So here you can see there were two populations. There was an unexposed population of similar people who lived far away, 80 kilometers away from where the border between the two countries where the Roundup was sprayed. So you have an unexposed group. And then you have the exposed group who were exposed to this intensive spraying for three days followed by three weeks of intermittent spraying.

And what they did is they drew blood on these people who were exposed. It was 28 people over -- somewhere between two weeks and two months after they were exposed. And they did this comet assay to look for DNA damage, or in this case it's called DNA migration. But what it really means is it is a measurement of DNA damage. You can see that in the unexposed the number was 25.94.

Q. And what does that mean?

A. Well, that is just sort of the baseline abnormalities that

they saw in their so-called normal controls. And then the results increased to 35.5 in the people who were exposed. And this is, as you can see from the p-value, highly statistically significant.

So what this shows is that when innocent bystanders are sprayed with Roundup at high -- high concentrations, the -- some of these people actually got sick from the pesticide, okay. They were ill. And so they had very high doses that actually made some of them ill.

When you look at their lymphocytes, you can see evidence of genotoxic damage. So that -- this is sort of a real-world kind of animal study where you are giving the animals high doses.

Q. But in this case it is actually human beings?

A. Yes.

Q. Okay. And the next one, if you can turn to 1066.

MS. MOORE: Permission to publish.

MR. STEKLOFF: No objection.

THE COURT: Go ahead.

BY MS. MOORE

Q. And this is another study, Dr. Weisenburger. Did you review this and rely on it in forming your opinion in this case?

A. Yes, I did.

So this is the other study. This one was actually done in

Colombia. And, again, we are looking at basically agricultural workers. This study -- it was interesting -- they used couples. So they husband -- they had 30 couples in each group, and they looked at exposure then, in both men and women, some of which were agricultural workers, and they were actually working in the fields.

MS. MOORE: And if we could, Mr. Wolfe, if you would go over to 991 and pull up the graph at the bottom.

BY MS. MOORE

Q. What were the results of this, Dr. Weisenburger?

A. Yeah. So this is kind of a complicated slide. Let me walk you through it. So the first bar on the far left labeled underneath Santa Marta, this was a region in Colombia where they do organic farming for coffee. So there are no pesticides being used at all, okay. So this is your negative control, okay. This is results on people who were not exposed to pesticides at all.

The second group, Boyaca, is an area where they used pesticides but they sprayed them, like Mr. Hardeman would have, they sprayed them from tanks, manually walking along and spraying the pesticides. And they used a variety of pesticides. So this is sort of like your positive control, okay, in the sense that they did two measurements one month apart, and they found pretty much the same findings; that there was a significant increase in damage to cells.

In this assay they are looking at what is called binucleated micronuclei -- cells with binucleated micronuclei. And there was this significant increase compared to the control, but it was due just to the spraying of multiple pesticides manually, okay. They probably were exposed to glyphosate, but -- among many other pesticides that they used, okay.

And then there are the three other regions -- Putumayo, Narino and Valle -- and in these three areas they were spraying the glyphosate, the Roundup, aerially just like in the other studies. So they were trying to get rid of the cocaine plants in Colombia by spraying -- by spraying the Roundup from airplanes, okay. And they sprayed in these three regions.

And so what you see here is the first bar on each of these last three was the test drawn just before the spraying, okay.

Q. So this bar, the one that is not filled in, is that the one you are referring to?

A. The one right there, yeah. So that would be the first bar. So that is sort of the control for that person or that group of people because this is what the DNA damage was in these three groups before the aerial spraying of glyphosate. So you can see they all have increased DNA damage probably because they were using other pesticides, okay, in spraying other pesticides.

So then there was this intense spraying of glyphosate from

the air and --

Q. Is it glyphosate, Dr. Weisenburger?

A. It was Roundup, yeah. Roundup from the air.

And then they did the second measurement, which is the middle bar, within five days after the aerial spraying of Roundup, okay. And what you can see in each area is there is an increase in the DNA damage in each of the three groups, and it is statistically significant. So there was -- after -- shortly after the Roundup spraying, there was a statistically significant increase in all three of these groups after spraying, okay.

Q. What is the increase of?

A. It is an increase in binucleated micronuclei, which are -- it is a test to determine genotoxicity. Again, this was done in human lymphocytes, okay.

Q. What does this tell us if there was an increase five days after spraying Roundup?

A. It tells you there was an increase in genotoxic damage to the lymphocytes from the people in these three regions that was associated with the aerial spraying of Roundup.

And then the third bar, the dark bar, was drawn four months later. And what you can see is that in the first group it stays up. In the second and third groups, it goes back down a little bit. And so I don't know what to make of this. It seems like the genotoxic damage persisted in Putumayo, maybe

related to the use of other pesticides. But in the other two regions it went back down closer to what their baseline was.

So the meaning of this study is that it is a real-world scenario in which agricultural workers and their spouses were exposed to aerial spraying of Roundup, and you can see a correlation, an increase in genotoxic damage to the human lymphocytes related directly to that spraying of Roundup. So you can see genotoxic effects in experimental situations where you are looking at lymphocytes in a culture dish, and you can see the same effects in real human beings exposed to high amounts of Roundup.

Q. And have you formed an opinion based on your review of the animal studies, the human lymphocyte studies in the petri dish, and then the real-world exposures to Roundup as to whether Roundup is genotoxic?

A. Yeah. I think there is no question that Roundup is genotoxic because there are multiple positive studies now in different systems. I'm just showing you a few of the examples, but there are multiple studies. There were at least 11 studies in human lymphocytes that showed genotoxicity. There were studies in other human systems as well where they saw genotoxicity. So I don't think there is any question that Roundup is genotoxic.

Q. And, Dr. Weisenburger, after your review of the epidemiology -- back to our three legs -- the epidemiology, the

animal studies, and the mechanistic data, did you perform what the jury has heard of as a Bradford-Hill analysis?

A. Yes, I did. So I weighed all of that evidence and used the criteria that were proposed by Bradford-Hill to do a general causation analysis.

Q. And just briefly, remind the jury, who is Bradford Hill?

A. So Bradford Hill was an English epidemiologist who was a very influential -- quite a long time ago. He was involved a lot in analysis of smoking causing long cancer, okay. But he wrote these -- these eight criteria or guidelines to use in trying to determine whether a chemical or other agent can cause non-Hodgkin's lymphoma.

Q. So let's go through those criteria briefly. And what I want you to focus on, because the jury has heard what the criteria is, I want you to focus on what your analysis of each of the criteria is, what your conclusions were in forming your opinion?

MS. MOORE: And, Ms. Melen, can I have the ELMO, please. Thank you.

BY MS. MOORE

Q. Let's start with the first one, which is the temporal relationship.

A. All right. Temporal relationship, so what that means -- it is very straightforward. What that means is that you have to be exposed to the agent or chemical -- in this case,

Roundup -- before you get the disease to conclude that Roundup may have caused the disease. So you just have to be exposed to the agent before you get the disease which, of course, was the case in the case control studies, in the animal studies.

Q. So do we meet -- in your opinion is the temporal relationship criteria met in this case?

A. Yes, yes.

Q. Okay. The next one, strength of association.

A. So the strength of association means if you look at the odds ratios, do you see -- what is the strength of the association, what is the odds ratio? Is it high and is it statistically significant? And what we saw in the case control studies, in five of the six, the studies were positive with odds ratio greater than 2, and they were statistically significant. So I think the strength of the association is seen there as well as in the meta-analysis, where you also saw increased risks that were statistically significant.

Q. So should I write "yes" there?

A. Yes.

Q. And then the next one is dose response relationship.

A. So as we talked about, if something -- if there is an agent that causes cancer, usually the more agent you are exposed to, the higher your risk for the cancer. And so we saw in two of the case control studies that the risk of non-Hodgkin's lymphoma when -- was increased when there was

more days that the people were exposed, either per year or total days. So that shows a dose response relationship.

And we saw the same thing in the Zhang meta-analysis where, when they looked at the high dose, the people who were exposed to higher doses or more exposure, they had a -- they had a higher risk ratio than those that were just ever-never. Then you saw it also in the NAPP study where there was a very nice demonstration of dose response for non-Hodgkin's lymphoma overall and for diffuse large B-cell lymphoma.

So dose response is a really important criteria. If you see dose response, it gives you some assurance that that chemical is actually causing the disease.

Q. Should I write "yes" on that?

A. Yes.

Q. Then the next criteria is replication of results. Did you see replication?

A. Yeah. So what that means is you want to -- you don't want to just evaluate one study, okay. You want to see the same results or similar results in multiple studies. So with regard to the epidemiology studies, we saw it in five of the six studies, okay. In fact, the AHS study was an outlier compared to the other studies. And you want to see it -- the studies done in -- by different researchers in different countries and at different times.

So, you know, I think that there is a consistent

replication of results in the epidemiology studies and in the animal studies, too, because in many of the -- the tumors were replicated in other studies.

Q. And so do we have replication of results?

A. Yes.

Q. And then biological plausibility.

A. So what this means is, you know, based on what we know scientifically, does it all make sense? Is it plausible? Does it sound like it makes sense?

And I think what I have told you today is that there is definitely a biologic plausibility. We know that Roundup is genotoxic in lymphocyte cultures, in other tests. We know that it causes genotoxic damage in humans who are highly exposed like the studies in South America. We know that it causes tumors in animals, including non-Hodgkin's lymphoma. And finally, the epidemiology studies point towards non-Hodgkin's lymphoma as well.

So you have got a coherence of results that make it very plausible that -- that Roundup causes non-Hodgkin's lymphoma.

Q. So is biological plausibility met?

A. Yes.

Q. The next one is alternative explanations.

A. So when you look at epidemiology studies, you always want to think, Well, could there be another explanation for the results, okay. So some of the things that have been proposed

are things like selection bias or recall bias or confounding due to the use of other pesticides. And so, you know, we should talk a little bit about that.

You know, these studies were designed by very experienced epidemiologists who knew how to do case control studies. So the idea that there would be some selection bias in the cases -- the controls I don't think is plausible, okay?

The issue of recall bias I think is important -- do you know what recall bias is? That is when you have cases and controls -- the cases have the disease, and the idea is that because they have the disease, they are asking themselves, How could I have gotten this disease? What could have caused my disease? And so the idea is maybe they would remember the pesticides better than the people who didn't get the disease but who used the pesticides.

But, in fact, we looked at that in our study in Nebraska, and if you look at the frequency of the various pesticides that were recalled by the cases and by the controls, there was really no difference. The cases didn't recall more pesticide use or a higher number of pesticides than the controls did. So it is unlikely to be a recall bias. But even a more important argument is recall bias would be a systematic error, okay.

So you should see it in the case control studies of other cancers, and we have -- we have lots of case control studies of other cancers with Roundup, other hematological malignancies

like Hodgkin's lymphoma, myeloma, leukemia, and other solid tumors. And there was no evidence of increased risk in any of those other studies. The only studies that are positive are the studies of non-Hodgkin's lymphoma.

So recall bias couldn't possibly be the explanation for the increased risks in the non-Hodgkin's lymphoma studies because it should have caused the same kind of effect in the other studies and it didn't, okay.

Yeah, so I think that's all I want to say about that.

Q. So were you able to --

A. Oh, and then confounding. The other one was confounding, I forgot. So that is also an important one to consider, you know. Are we mistaking one pesticide, causing it and blaming the other pesticide?

So that's why we do the adjustments like we did in three of the case control studies we talked about, and -- in the NAPP study and in the Zhang meta-analysis. All of those showed that when you make adjustments for the use of other pesticides, the odds ratio sometimes go down, but they don't go to 1. In some of the studies they remain statistically significant like *De Roos* and Zhang and NAPP; whereas in other studies they go down and they become nonsignificant. But it is still important to look at all of that data?

So it can't be confounding. It can't be recall bias. It can't be selection bias. So we have no other explanation for

why the case control studies would be positive. And I have given some reasons why I think the AHS is probably a false-negative, okay, because there are lots of problems and issues with that study.

Q. So in your opinion is alternative explanations met?

A. There are no alternative explanations, so I would say yes.

Q. And then disease specificity.

A. So this is interesting. So as I mentioned to you, there have been lots of case control studies of other kinds of cancers, and they have all been negative. So the only studies that have been positive are the studies of non-Hodgkin's lymphoma. So I think that exposures to glyphosate and Roundup are very specific for this one cancer, non-Hodgkin's lymphoma. So there is disease specificity here.

Q. And then the last one is coherence. Have we met coherence?

A. Coherence just means does all the data fit together, and does it fit with what we know about the use of other pesticides. And, of course, there are other pesticides that cause non-Hodgkin's lymphoma in the same way that Roundup does. They are genotoxic and they cause oxidative stress and DNA damage. And so what we are learning about glyphosate fits with what we know about other pesticides and other chemicals that cause cancer.

Q. So that is met?

A. So that's met.

Q. And, Dr. Weisenburger, I'm just going to write on the top here your name so we will not forget that this was yours, and forgive my handwriting. I'm going to mark this for identification purposes only as Exhibit 948.

So taking into consideration all the criteria of Bradford-Hill and your review of the epidemiology, the animal studies, the mechanistic data, what then is your opinion within a reasonable degree of medical certainty now that we have gone through all the literature and your 40 years of experience in looking at the causes of NHL, what is your opinion regarding Roundup?

A. So my opinion is that I agree with -- that after looking at all this information, my conclusion is that based on all this data, that Roundup can cause non-Hodgkin's lymphoma in people who were exposed to it occupationally or exposed to high doses inadvertently.

Q. And you said "occupationally." And I want to make sure. So is it only in people who are using Roundup in the workplace?

A. No. Just like Mr. Hardeman. I mean, he used it frequently and had high exposures, and so I would include him in that group. It may not have been his occupation, but he spent a lot of time doing it.

Q. Is it fair to say that it's more about the amount of exposure than it is the occupation?

MR. STEKLOFF: Objection. Leading.

THE COURT: Sustained.

BY MS. MOORE

Q. What is a more important factor in determining whether -- what is a more important factor when you are looking at an exposure of an individual?

A. Well, I think when you look at the exposure of an individual, you want to really understand how much exposure he or she had. So you want to get an idea of what their dose was over time, were they exposed for many years, how often did they use it, did they wear protective clothing, et cetera, to get an idea of what their actual exposure was.

Q. Dr. Weisenburger, in your opinion does Roundup cause tumors in mammals?

A. Yes.

Q. In your opinion does Roundup cause malignant lymphomas in mice?

A. Yes.

Q. Does Roundup cause genetic damage in human lymphocytes?

A. Yes.

Q. Does Roundup cause oxidative stress in human cells?

A. Yes.

Q. Does Roundup cause DNA damage in people who are highly exposed to Roundup?

A. Yes.

Q. And in your opinion does Roundup cause non-Hodgkin's lymphoma in humans at real-world exposures?

A. Yes.

Q. Now, over your 40 years of studying non-Hodgkin's lymphoma, have you seen an increase or a decrease in the number of cases of non-Hodgkin's lymphoma?

A. Well, over the last 50 years, the incidence of non-Hodgkin's lymphoma has increased. In fact, there was a rather remarkable increase between 1970 and about 1990. And as I told you, that was -- that increase was what got people together to think about what could be causing this increase, okay.

Q. And in your opinion what was causing that market increase from 1970 to 1990?

A. Well, we don't have all the answers. We know that during that period of time is when the AIDS epidemic began, and people with HIV/AIDS have a more -- remarkably increased risk for non-Hodgkin's lymphoma. So we know some of that steep curve was probably due to AIDS.

Probably some of it was better diagnosis by pathologists, and probably some of it was due to other causes, which we don't really know what they are. It could be due to increased use of pesticides or solvents or petrochemicals, or it could have been an increase in infections, for example. But we don't really know, other than HIV, what caused that increase -- that

increase over time.

Q. And I'm going to show you --

MS. MOORE: If I could publish on the ELMO, what is called -- it is trends and incidence rates -- let me zoom in on this.

BY MS. MOORE

Q. Trends and incidence rates. It says 1975 to 2015. Can you explain to the ladies and gentlemen of the jury what this graph shows?

A. Well, first of all --

MS. MOORE: I'm sorry, hold on. May I have this published?

MR. STEKLOFF: No objection, Your Honor.

THE COURT: I saw the silent non-objection.

MS. MOORE: Sorry.

THE WITNESS: So the top curve shows the incidence rate in men, and the lower curve shows the incidence rate in women. And non-Hodgkin's lymphoma, just like many other cancers, is higher in men than women.

BY MS. MOORE

Q. So this yellow line here, that's --

A. That's men.

Q. -- men?

And this line here?

A. Is women, yes.

And you can see -- the far left is 1970, and you can see how --

Q. I think it -- hold on. Let me zoom in.

A. -- how it goes up.

Q. I think -- I don't mean to interrupt you, Dr. Weisenburger. I think it is 1975.

A. 1975, okay.

Q. Up to 2015.

A. Yes.

So you can see how it goes up in both men and women, a little more steeply in men than women. Between 19 -- it actually started in 1970, but -- so between 1970 and probably 1990 or 1995 you see a more steep curve. And then it begins to kind of level off after that, and it is pretty stable. For men it creeps up a little bit, and for women it is pretty stable.

So this early part of the curve where you see it increasing, at what would be kind of a worrisome rate, eventually leveled off and it is not increasing nearly as rapidly as it did during that early period.

Q. It looks like from 1975 the rate for men, it went from -- what is that -- 13 up to 1995 -- is it almost doubled then?

A. It is almost doubled, yes.

Q. Okay. And I want to show you a slide, Dr. Weisenburger, and this was shown to the jury during opening by Monsanto's attorney. Okay.

And from this slide -- well, first of all, what is this slide? What, if anything, does this slide tell you?

A. Well, it shows -- it is a drawing on a somewhat different scale, but it shows you in the brown -- brown line, it shows you that the number of cases of NHL, which is on the left side, and the number of cases per hundred thousand persons, which is the incidence, both of those are going up, again, between 1974 and about 1990, and then it begins to level off. So this is a combined curve for both men and women, okay.

And then what the blue curve shows is that glyphosate was introduced in 1974, and it was -- increasing -- was used increasingly up until about 1995 or 1994 when the use began to go up dramatically due to the use of the genetically modified crops, like corn and soybeans.

So what you can see is there has been a marked increase in the use of glyphosate over the 20 or 30 years or so there; whereas the rate of non-Hodgkin's lymphoma has gone up not very much, okay. So, you know, it is sort of misleading to say, Look how the glyphosate has gone up and yet the rate of non-Hodgkin's lymphoma hasn't gone up and that's because the rate of non-Hodgkin's lymphoma is due to a whole variety of things.

So think about it. If glyphosate caused an increased number of non-Hodgkin's lymphoma cases, but we learned how to treat the HIV infection, HIV/AIDS, and now we -- they don't get

nearly as many lymphomas as they did before, glyphosate could cause an increase in non-Hodgkin's lymphoma; treatment of HIV infection could cause a decrease in non-Hodgkin's lymphoma, and the curve wouldn't change very much at all, right, because you have got some things increasing risk and some things decreasing risk.

Q. Let me back up and ask you -- because you referenced HIV and AIDS as an example -- is HIV -- someone who is HIV, are they at an increased risk for developing non-Hodgkin's lymphoma?

A. Yes. Back in the '70s and '80s it was a markedly increased risk. And then when we found drugs to treat it, the incidence of non-Hodgkin's lymphoma went down dramatically in people with HIV/AIDS.

Q. So on this slide that was shown by Monsanto's attorney in opening, the title of it is "Glyphosate use increased, NHL did not," can we draw any kind of conclusions about Roundup and the correlation between Roundup causing an increase in non-Hodgkin's lymphoma from this graph?

A. I would say no. I think it is actually a rather misleading graph that can't let you conclude anything.

Q. It doesn't show a dose response, does it?

A. No.

MR. STEKLOFF: Objection. Leading.

THE COURT: Sustained. That answer will be stricken.

BY MS. MOORE

Q. Dr. Weisenburger, does it -- does this graph give you any information as far as the impact of Roundup on the journal of population?

A. It doesn't.

Q. Let's switch gears then, and I want to focus on Mr. Hardeman. Have you had an opportunity to review documents and form an opinion as to whether Roundup was a substantial factor in causing Mr. Hardeman's diagnosis of non-Hodgkin's lymphoma?

A. Yes. So I reviewed the medical records. I reviewed the doctors' depositions. I reviewed the deposition of Mr. Hardeman and his wife. I reviewed the pesticide fact sheet for Mr. Hardeman. And I actually talked to Mr. Hardeman on the phone for about an hour to get -- to answer my questions and to get a better idea of what happened to him and what kind of exposures he really had to Roundup.

Q. And based upon that review of depositions and the medical records of Mr. Hardeman, your interview with Mr. Hardeman, have you -- what is your opinion as to whether Roundup was a substantial factor in causing Mr. Hardeman's non-Hodgkin's lymphoma?

A. Well, I think after talking to Mr. Hardeman, he had really quite high exposures to Roundup over many years. You know, he used Roundup for 26 years before getting non-Hodgkin's

lymphoma, and 23 of those years he was using it for the -- for the six months of spring and summer, he was using it twice a month, for two to four hours every time, and spraying up to 20 gallons at one -- at one time in one day. And he did that -- and then -- then during the winter months, he would spray once a month using, you know, lesser amounts.

MR. STEKLOFF: Your Honor, I object to hearsay.

THE COURT: Overruled.

THE WITNESS: And so, you know, I did some crude calculations of how many days he was -- how many days during those 26 years he used Roundup based -- I used information from his deposition. So he was exposed to Roundup over 300 times during those 26 years. And he used -- my calculation -- he used around 5,900 gallons of Roundup in those 26 years.

So, you know, he had high exposure to Roundup. He didn't wear any protective equipment, didn't wear gloves, didn't wear a mask. He wore short-sleeved shirts. He didn't take any real precautions at all when he sprayed it, so he got it on his hands. He got it on his arms. He got it on his face. And when he was mixing it, he would sometimes even get it on his hands while he was mixing it.

So, you know, when you add it all together, he had -- he was -- he had a high exposure to Roundup over many years.

BY MS. MOORE

Q. And based upon that -- your determination that

Mr. Hardeman fit into the category of high exposure, what was your conclusion as to the cause of his non-Hodgkin's lymphoma?

A. Well, my conclusion after weighing all the evidence -- and we will talk about the differential diagnosis or differential etiology -- but after going through all of the -- this methodology, I came to the conclusion that Roundup was the substantial contributing cause for him with regard to his development of non-Hodgkin's lymphoma.

Q. But, Dr. Weisenburger, the jury heard testimony last week from Mr. Hardeman's treating physicians, Dr. Ye, his oncologist. And you have reviewed that deposition?

A. Yes.

Q. Okay. And the jury heard that Dr. Ye had -- did not form an opinion as to the cause of Mr. Hardeman's non-Hodgkin's lymphoma. Did that surprise you when you read that?

A. No, because once the patient --

MR. STEKLOFF: Your Honor.

THE COURT: Overruled.

THE WITNESS: So because once the patient has the disease, unless the cause is obvious, the oncologist is more concerned with treating the disease than he is trying to figure out what caused the disease. So it's not uncommon for physicians not to ask questions or to try to figure out what caused the disease unless it's obvious.

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BY MS. MOORE

Q. Now, you mentioned a differential. And so what is a differential?

A. So the methodology for doing this is the same methodology we use when we are diagnosing and treating patients in the hospital or the clinic. If the patient has a disease, you want to think about what are all of the potential causes and risk factors for that disease, and you want to go through the list and try to eliminate the things that are not applicable to the patient and to -- and then to just evaluate the things that may be causes and hopefully come to a conclusion that one or sometimes more than one thing are the cause.

So you want to go through the list of all the known causes and try to eliminate the ones that don't apply to the case, and then what you are left with are potential causes. Then you have to weigh them and see, well, which is the most important of these potential causes, or you can rule some of them out.

Q. Did you go through that process with respect to Mr. Hardeman?

A. Yes, I did.

MS. MOORE: And, Your Honor, with your permission if I can ask the witness to come off the stand and use the blowup.

THE COURT: Sure.

BY MS. MOORE

Q. Dr. Weisenburger, if you want to come this way.

Did you put together -- did you put together a list that you used in your differential?

A. Yes, I did.

Q. And is this your list?

A. Yes.

Q. And on the left-hand column there -- if you need a pen, I have one.

In the left-hand column, you have Known risk factors for NHL. Can you explain to the jury how you came up with what is in the left-hand column?

A. Well, these are all of the known and published risk factors for non-Hodgkin's lymphoma. So I made this list, and -- with Mr. Hardeman, and I went through them by reading his medical records and reading his doctors' depositions and talking to him. I went through this list and tried to rule in or rule out some of these causes, okay.

MS. MOORE: Before you do that, may I publish this to the jury, too, Your Honor, on the screen?

THE COURT: Any objection?

MR. STEKLOFF: No, Your Honor.

MS. MOORE: Thank you.

BY MS. MOORE

Q. Go ahead.

A. Okay. So age is a known risk factor for non-Hodgkin's lymphoma; that is, as you get older, your risk for

non-Hodgkin's lymphoma increases. It is true of many cancers, okay. There are some cancers that are more common in children, but for most cancers the risk increases as people get older.

And sex, I showed you that non-Hodgkin's lymphoma is more common in men than women; probably 20 to 30 percent more common. And we don't really understand why that is.

And then race also. Caucasians have a higher risk than blacks or Hispanics, and they have a higher risk than Asians. So, you know, Mr. Hardeman was 65, I think, when he got his non-Hodgkin's lymphoma. He was a male. And he was Caucasian. So he fit into this category, but I don't consider any of these causative risk factors. Did his age cause his non-Hodgkin's lymphoma? That doesn't really make sense.

Q. When you say "causative risk factor," what does that mean?

A. Well, it means that it is a factor that actually caused the lymphoma, okay. So I don't believe that age causes lymphoma or sex causes lymphoma or race causes lymphoma. Those are risk factors, but they are not causative risk factors.

Q. So in other words with respect to age, simply because you are at an older age, does that mean you are going to get -- does that mean age itself is going to cause you to get non-Hodgkin's lymphoma?

A. No.

Q. The same thing with sex and race, just because you are a man that doesn't mean you are going to -- cause you to get

non-Hodgkin's lymphoma?

A. No. We can't say Mr. Hardeman got non-Hodgkin's lymphoma because he is a man. That doesn't make sense, right. So they are risk factors. They tell you something about the disease, but they don't really tell you what caused the disease. So I eliminated these three as causative risk factors. And the other thing is you have no control over that, right. You have no control over that.

So the next thing I thought about was family history of malignancies because we do know if you have a family history of a hematologic cancer, like non-Hodgkin's lymphoma or Hodgkin's lymphoma or leukemia or myeloma, we know that those people have a two-time increased risk for non-Hodgkin's lymphoma, okay.

But in Mr. Hardeman's case he didn't have any family history of any of these cancers. So he probably didn't have any genetic predisposition to getting this.

Q. Let me -- before you do that, I want to ask you: You have "hematologic malignancy." What is that?

A. Well, those are the family of diseases that we are talking about: Non-Hodgkin's lymphoma, Hodgkin's lymphoma, myeloma, leukemia, things that hematologists are diagnosing. And it is a family history. It is usually first-degree relatives. So it is father, mother, brother, sister, and children. Those are the things that you look at to get this increased risk. So he didn't have any family history, okay.

Q. So you ruled that out?

A. So I ruled that out.

Q. Okay.

A. Pesticide use --

Q. Before you go there, I just want to ask you -- because the jury heard some testimony about Mr. Hardeman having a couple of sun spots, basal cell carcinoma on his leg, and melanoma last year on his shoulder -- I don't see that you have skin cancer up here. Can you explain to the jury why you didn't put that on here?

A. Well, because it is not really a causative risk factor. And Mr. Hardeman had one basal cell carcinoma. It is a very common tumor, okay. Melanomas are also fairly common.

So -- and neither basal cell carcinoma or melanomas are causes of non-Hodgkin's lymphoma. It is -- they are usually due to the sun damage, okay, ultraviolet light damage to the skin. And we know that people who get a lot of sun exposure have a lower risk of non-Hodgkin's lymphoma, not a higher risk. So I don't see that -- how that fits in at all. It is -- it doesn't -- I didn't -- I didn't add it into my differential, no.

Q. Go ahead with the next thing you put on your risk factor list.

A. So the next thing was pesticide use. And, of course, as we talked about, he had high exposures to Roundup for many

years. I asked about other pesticides because we know there are other pesticides that cause non-Hodgkin's lymphoma, and he didn't use any of those other pesticides. In fact, the only thing he ever used was he would occasionally, maybe once a year, spray for ants in the house, okay, buying something off the shelf.

So the only -- the only pesticide that he used in any amount was Roundup, okay. So I think we have to put Roundup over here -- excuse my writing -- over there --

Q. You are a doctor.

A. -- as a possible risk factor for his non-Hodgkin's lymphoma, okay.

Q. Let me stop you there because you moved Roundup over to this column right now, does that mean you have already determined that it is the cause?

A. No. We have to go through these other -- the rest of the list here to see if there were other important risk factors.

Q. Okay. All right. The next one?

A. So we know that obesity is sort of a weak risk factor for non-Hodgkin's lymphoma. Mr. Hardeman wasn't obese, but he is a bit overweight, okay. His average weight was about 190 pounds, and I think his height was only -- I can't even remember. But the calculation of his body mass index was in the overweight category, not in the obese category. But even overweight people can have an increased risk of non-Hodgkin's lymphoma.

But it is a small risk. It is not a big risk.

So, you know, we probably have to include obesity. His risk would have been maybe 1.3, maybe a 30 percent, increased risk. We will put obesity there, really meaning overweight.

Q. Okay. Did you want to write "overweight"?

A. No. It's okay.

Q. Then the next one?

A. The next one is viral infections. And there are a number of viral infections that actually cause non-Hodgkin's lymphoma. One of the best known ones is Epstein-Barr virus. It is the same one that causes infectious mononucleosis, but it also causes non-Hodgkin's lymphoma in some people, okay. But he didn't have any evidence of that in his biopsy or even in his blood testing.

And then there is some other less common viruses that also cause non-Hodgkin's lymphoma, but there are two viral infections that do cause non-Hodgkin's lymphoma. One is hepatitis C virus infection and one is hepatitis B virus infection; and he did have a history of both of those viral infections, okay. So we have to put both of those over here in the column and evaluate whether they could be the cause of his non-Hodgkin's lymphoma, okay. So we will say hepatitis C and hepatitis B.

Q. Okay. And then you want to finish off the list then?

A. And then bacterial infections. There are some bacterial

infections that can cause non-Hodgkin's lymphoma. The most common one is non-Hodgkin's lymphoma of the stomach related to a bacteria, but he didn't have any evidence of bacterial infections that would fit into that category. So I excluded that one.

People who have immune deficiency have an increased risk of non-Hodgkin's lymphoma. So we were talking about people with HIV/AIDS, for example. That is a good example. They have a very dramatic knockout of their immune system, and then they get infections and they can get lymphoma, okay. And people who have organ transplants, for example, they are put on drugs that could decrease their immunity and then they can get non-Hodgkin's lymphoma. But he didn't have any history of any of that kind of severe immunodeficiency. So I crossed that one out.

And then immunosuppression. So if you give certain drugs or chemotherapy, you can knock the immune system down and increase the risk for non-Hodgkin's lymphoma; but he really didn't have any history of immunosuppression by drugs or other things, so I crossed that one out.

Another thing that can cause non-Hodgkin's lymphoma is autoimmune diseases. So diseases like rheumatoid arthritis, for example, diseases like systemic Lupus or celiac disease, et cetera. There are a variety of autoimmune diseases where the immune system is altered and the body reacts against

itself, and people with those diseases can have an increased risk for non-Hodgkin's lymphoma.

Q. And based on your review of Mr. Hardeman's medical records, did he have any type of autoimmune disease?

A. He didn't so I crossed that one out.

And then chronic inflammation is a known cause. So people who have some kind -- the most recent one actually that I think is the best example is we now know that women who have breast implants have an increased risk for non-Hodgkin's lymphoma. And why is that? Well, they have chronic inflammation associated with that breast implant, and a small percentage of women actually get non-Hodgkin's lymphoma that comes out of that chronic inflammation.

And there's some other examples that are -- you know, that I could give you, but he didn't have any evidence that he had some kind of chronic inflammation going on in his body so I crossed that one out.

And then the last one is solvent use. We talked a little bit about that earlier. So people who use solvents, particularly people who use a lot of solvents in their work, like painters or machinists, people who are using solvents to clean their brushes or clean their equipment or their machines have an increased risk of non-Hodgkin's lymphoma; but he didn't have any history of solvent use, and so we crossed out that one as well.

So what we're left with is Roundup and obesity and hepatitis C and possibly hepatitis B as the possible causes for his non-Hodgkin's lymphoma.

Q. So once you've eliminated these other risk factors and now you've looked at, for Mr. Hardeman in particular, these four, what's the next step in your process?

A. So the next step is to take a hard look at each of these and decide whether they're strong risk factors or weak risk factors or maybe not even risk factors at all depending on the history of what happens with the viral infections.

Q. Okay. So which one do you want to start with, then, and explain to the jury what your process was next?

A. Well, I think we should start with the hepatitis B and hepatitis C and talk about those because those are -- those could also be substantial risk factors. Okay?

Q. And in Mr. Hardeman's case, let's start with hepatitis C, when you were looking to see whether it is a substantial factor, what did you look at?

A. So I had to go back and do a lot of research on hepatitis B and hepatitis C to really understand who's at risk and what is the risk -- okay? -- and what I found was that people who have chronic active hepatitis, they have the virus causing the disease in their liver, those are the people who are at risk for developing non-Hodgkin's lymphoma. So in some ways they have chronic inflammation that's causing liver damage

and eventually cirrhosis. Okay?

And when Mr. Hardeman was found to have hepatitis, first of all, he was found to have cirrhosis; and then they looked to see what could cause the cirrhosis, and they said, "Oh, my gosh. He's got hepatitis C -- hepatitis." So you have to look at the whole story here and try to figure it out.

But what the literature says is that you have to have chronic active hepatitis to be at risk for non-Hodgkin's lymphoma.

Q. So did Mr. Hardeman at one point in time have chronic active hepatitis C?

A. Yes, he must have because he had cirrhosis in 2005 when the cirrhosis was discovered; and then they did the test for hepatitis B and hepatitis C, and they found that he had an active infection of hepatitis C. He had lots of viral particles in his blood, and they tested him also for hepatitis B, and he was found to be immune to hepatitis B.

So he didn't have an active infection of hepatitis B. Because of his prior infection, he became immune to hepatitis B, and so he was immune to that virus.

Q. Well, that's what I wanted to ask you. You say he was immune to hepatitis B and you've got it over in this column. What does immune to hepatitis B mean?

A. Well, it means that he developed immunity. So, for example, if you get a vaccine to -- you know, when you're

little, you got vaccinated; right? And after they vaccinated you with, let's say, the polio -- attenuated polio virus, you developed immune response to that polio virus and it protects you from getting polio; right?

Well, the same thing happens with hepatitis B. If you get hepatitis B, you might be sick for a while but then your body develops immunity, gets rid of the hepatitis B, and you have an immunity and you're protected from hepatitis B in the future. Okay?

Q. Is that what happened with Mr. Hardeman?

A. And that's what happened with Mr. Hardeman. He didn't have any evidence of hepatitis through the hepatitis B. He was immune to hepatitis B virus.

Q. And then -- and when do you know that he was -- at what point in time, based on your review of the records, do we know that he was immune to hepatitis B?

A. Well, they did the test in 2005. At the time they were working him up for his cirrhosis, they did tests for hepatitis B and hepatitis C; and they thought because he had active hepatitis through the hepatitis C, that that was the main cause of his cirrhosis.

Q. All right. So you keep mentioning "active." Can you explain to the jury a little bit more? What does it mean to have active hepatitis?

A. So active hepatitis means you have inflammation in the

liver. The virus is infecting the liver cells. It's destroying the liver cells and eventually the liver gets scarred from all the inflammation and death of liver cells and you end up with cirrhosis, just scarring of the liver. Okay? And that's what he had in 2005, not a severe case but, you know, it definitely was there.

Q. Is liver damage an expected outcome with someone who has hepatitis C?

A. Yes.

Q. And based on your review of the literature, Dr. Weisenburger, people who have active hepatitis C, what percentage of those people go on to develop non-Hodgkin's lymphoma?

A. It's actually quite a low percentage. It's less than 1 percent. Probably less than a half a percent. The data is not very -- there's not very good data on exactly what percentage it is but it's low. It's much lower than liver cancer as a result of hepatitis and cirrhosis. It's probably 10 times less common than liver cancer.

Q. So what does that mean for Mr. Hardeman? What would you expect -- given that he had active hepatitis C, what would you expect that to develop in to --

A. Well, I think --

Q. -- based on those stats?

A. -- I think, you know, when we look back at his history, he

thought he probably contracted the hepatitis C and hepatitis B sometime in the 1960s. So he probably had the infection to hepatitis C since that time. So he had it probably for almost 40 years causing chronic hepatitis. Not making him very sick because he never really went to the doctor saying "I'm sick"; but by the time he did get to the doctor in 2005, he was found to have scarring of the liver and cirrhosis and some hepatitis as well. There was evidence of liver damage from blood tests.

So, you know, he probably had the hepatitis C for about 40 years -- 39, 40 years. He probably had the hepatitis B at sometime during that same time frame but became immune to it and, you know, kept that infection in check.

Q. So in the time period that you're talking about the active hepatitis C, what time period is that?

A. For Mr. Hardeman?

Q. For Mr. Hardeman.

A. It was about 39 years.

Q. So from what year to what year?

A. Probably 1996 to 2005, something like that. So 39, 40 years.

Q. Well, you said "1996," so --

A. Let's see...

Q. Now I've got you in a math problem here.

A. 1966. 1966 to 2005. I'm sorry.

Q. That's okay. That's okay.

And so during the time that Mr. Hardeman likely had active hepatitis C for those 39 years or so, did he get non-Hodgkin's lymphoma?

A. No. So that's when he would have been at risk for getting non-Hodgkin's lymphoma, and he didn't get non-Hodgkin's lymphoma and luckily he didn't get liver cancer either -- okay? -- which are the two cancers he would have been at high risk for.

Q. All right. So based on your review, then, what happened after he found out in 2005 that he had hepatitis C?

A. Well, by that time we have had fairly good treatments for hepatitis C so he was given a course of antiviral therapy, which included two drugs -- one called Interferon, another called Ribavirin -- and he was given a course of that treatment over a period of about 46 weeks. Okay?

And what happened to him was that within 12 weeks, his viral DNA in the blood, which is the way they made that, his viral DNA in the blood went away -- okay? -- and he no longer had evidence of the viral infection, but they continued to treat for up to 46 weeks because you know that you have to continue to treat for a long time afterwards to get rid of hepatitis C so that's what they did.

Q. Okay.

A. And during that period of treatment, once his viral testing in the blood became negative at 12 weeks, it stayed

negative through the whole treatment right out to the end of the treatment.

Q. And I think we have -- and, Dr. Weisenburger, is that based on your review of his test results?

A. Yes.

Q. Okay. And I think we have a summary of his test results, and I'm going to slide behind you and put that up on the board.

A. Take this off?

Q. It's fine. We're going to leave it there.

And this is a summary of the test results.

MS. MOORE: May we publish that to the jury, Your Honor, please?

THE COURT: Sure.

THE WITNESS: So this just shows you the chronology of his hepatitis C. So in 2005, January 2005, the test was found to be positive. Okay? And so at that point he was treated with this antiviral therapy to try to cure him of the hepatitis C. Okay? And, in fact, within just 12 weeks, his viral RNA in the blood became negative. Okay?

And then he was treated for, I think, 46 weeks and he stayed negative through that time. At the end of the treatment, he was still negative. And then they monitored him for over five years, and he stayed negative for the whole time. Okay?

\\\

BY MS. MOORE:

Q. What's the significance of once he started antiviral treatment for hepatitis C that he stayed negative during treatment and then after treatment?

A. Well, you would consider him to have a rapid response because he quickly became negative, and then you would consider him to have what we call the sustained virologic response because he stayed negative for six months and then for five or six years thereafter. Okay?

So most people who treat hepatitis C would say that he was cured of his hepatitis -- okay? -- in terms -- because his liver function tests became normal and the virus disappeared from the blood. Okay?

Q. And are these factors that were important for you in determining whether to rule in or rule out hepatitis C as a cause?

A. Well, they were because I realized that when he got his non-Hodgkin's lymphoma, he had already been free of the virus for nine years. Okay? He'd been cured of the virus for nine years.

And so part of the research I did was to say: Well, okay. We know that people who have active hepatitis C have an increased risk for non-Hodgkin's lymphoma, probably two- to threefold increased risk; but what about people who are treated with antivirals, have a sustained virologic response, and are

cured of the virus? Do those people -- are they still at increased risk for hepatitis C causing non-Hodgkin's lymphoma?

So that was the question I had to answer.

Q. Okay. And so I want to make sure. So the first factor you said was a rapid response; is that right?

A. Right. He had a rapid response in that he cleared the virus within the first 12 weeks.

Q. So I'm going to say within 12 weeks; is that right?

A. Yes.

Q. Okay. And then you said he was cured of hepatitis C?

A. Yes. So after you stay negative for six months during the -- six months after the therapy is completed, then you're considered cured, and he did meet that criteria. So he was considered by his doctors at Kaiser to be cured of the hepatitis C, and they stopped -- then they stopped the antiviral treatment after the first 46 weeks.

THE COURT: Can I interrupt for a minute? I think now would be a good time for a five-minute break.

MS. MOORE: Thank you, Your Honor.

THE COURT: Why don't we go ahead and take a break. Be back at 2:00 o'clock.

THE CLERK: All rise.

(Proceedings were heard out of the presence of the jury:)

THE CLERK: Please be seated.

THE COURT: All right. Back in a few minutes.

MS. MOORE: All right. Thank you, Your Honor.

(Recess taken at 1:56 p.m.)

(Proceedings resumed at 2:01 p.m.)

(Proceedings were heard out of the presence of the jury:)

THE COURT: Okay. You can bring the jury back in.

MS. MOORE: Thank you, Your Honor.

(Proceedings were heard in the presence of the jury:)

THE COURT: Okay. You can resume.

MS. MOORE: And, Your Honor, before I get right back into the questioning, a couple of housekeeping matters that Ms. Melen asked about.

And that is the summary of Mr. Hardeman's test results for hepatitis C viral load that we have up on the easel is Exhibit 940, and we would move to enter that into evidence as Exhibit 940.

THE COURT: Any objection?

MR. STEKLOFF: No objection, Your Honor.

THE COURT: It will be admitted.

(Trial Exhibit 940 received in evidence)

MS. MOORE: Thank you, Your Honor.

And then I also had shown this trend in incidence rates, and I did not mark it for identification, and we would mark it as identification 949, "Trends and Incidence Rates 1975 to 2015."

I apologize for that, Your Honor.

(Trial Exhibit 949 marked for identification)

BY MS. MOORE:

Q. Now, Dr. Weisenburger, let's go back to where we were, and you were on this summary that we marked as Exhibit 940, and we were getting back to the point where you said he had been considered cured of hep C. And then what was the next step in your process?

A. Right. So he was cured of hep C and he remained viral -- virus negative for nine years up until his diagnosis of non-Hodgkin's lymphoma in early 2015. Okay?

And --

Q. Well, hold on. You said "up until." Was he still considered cured when he was diagnosed with non-Hodgkin's lymphoma?

A. Yes. So he was tested at that time -- you can see the test right here -- and he was still negative. Okay? So at the time he was diagnosed with non-Hodgkin's lymphoma, his viral test in the blood was still negative so he was still considered to be cured. Okay?

And I don't know if I told you this, but he was found to be immune at the time of the diagnosis for hepatitis C. So he wasn't thought to have an active infection.

Q. Let me interrupt. You said hepatitis C.

A. Hepatitis B. Hepatitis B.

So they retested him for hepatitis B here. He continued

to be immune. Okay? There was no evidence of a viral infection due to hepatitis B or hepatitis C at the time of his non-Hodgkin's lymphoma.

Q. So can I say "immune hepatitis B" here?

A. Right.

Q. If I can spell it.

Okay. Is that right?

A. So one of the things that the clinicians worry about is when you treat people for non-Hodgkin's lymphoma and they have had a past infection for hepatitis C or hepatitis B, they sometimes have small amounts of that virus still in their body -- okay? -- but the immune system is holding it in check.

So he could have some virus in the liver. He could maybe even have some virus in B cells. Okay? But it's at a very low level, what we call latent -- present in the latent state, and it's held in check by the immune system.

But because they knew he had this history of hepatitis C and hepatitis B, they decided they should monitor him because when you give the chemotherapy, you cause immunosuppression and the viruses can reactivate and cause hepatitis. Okay? And they don't want that to happen when he's getting his chemotherapy. Okay?

Q. So let me stop you right there. What is immunosuppression?

A. So immunosuppression is what we talked about earlier where

we give drugs that knock the immune system out and increase the risk for infection. And because he had these two viral infections before and he might have some of that virus still in his body at a very low level, they decided, first of all, to give him antiviral drug for his hepatitis B -- okay? -- because there was a drug they could give him to make sure that if he had some hepatitis B still in his system, that it wouldn't reactivate during the chemotherapy.

And for hepatitis C there isn't a drug that you can give to prevent it so what they did is they just carefully monitored it every few weeks to make sure that he didn't reactivate his hepatitis C because if he did, they would have to give him the antiviral therapy like they did back in 2005.

Q. So right here where you have this shaded, is this the test they did to check him to see if the hepatitis C or B came back while undergoing chemo?

A. While undergoing chemo and -- yeah, while undergoing chemo. So he went under chemo for about six or eight -- about six weeks. So during that period of time, they -- six months. So during that period of time, they were checking him every few weeks for his hepatitis C infection. And they were also doing a test for his hepatitis B infection to make sure he didn't reactivate that; but because he was on antivirals for that, it was pretty unlikely that that would happen. But they monitored both of those infections, those possible infections.

Q. So with respect to the hepatitis C, there was no drug they could give him to make sure that didn't come back; right?

A. Right.

Q. Okay. So during the time he was undergoing chemotherapy when his system was weak, did the hepatitis C come back?

A. It didn't.

Q. And what is the significance of that?

A. Well, what that would tell you is that he didn't have it in his system at all or his immunity didn't get knocked down so low that the virus would come back. So we don't really know, but what we do know is that he didn't develop an active hepatitis infection even when he was getting the immunosuppressive drugs for the lymphoma. Okay?

Q. All right.

A. And the same is true for the hepatitis B infection as well.

Q. Let me take this down and let's go back.

So going back to your process to eliminate -- or, I'm sorry -- going back to your process to consider hepatitis B and C with respect to the non-Hodgkin's lymphoma, what did you do next?

A. Well, so I did some research and I read about hepatitis B and hepatitis C, and the main question was: If you -- if you have a cure -- if you're cured of your hepatitis C, do you still have an increased risk for non-Hodgkin's lymphoma?

And, in fact, the answer is no, which makes sense because the virus is what's causing the infection. And so if you get rid of the virus and you no longer have an active infection, then you wouldn't get the lymphoma from the virus because the virus is gone. Okay?

And the same is true -- and the same is true for hepatitis B. So I wanted to know: Well, are people who are immune to hepatitis B, are they still at increased risk for non-Hodgkin's lymphoma? And the answer for that was no too.

So if you're -- if you're cured of hepatitis C or if you're immune to hepatitis B, you no longer have an increased risk for non-Hodgkin's lymphoma -- okay? -- because you're cured of the virus. And even if there's small amounts of either B or C in your body, they're in a latent state held in check by the immune system; and the literature shows that when that occurs, there's no increased risk for non-Hodgkin's lymphoma with either hepatitis B or hepatitis C.

And I'm going to show you some -- some -- actually some --

Q. Do you want to do that now?

A. -- some data from the literature to convince you of that. Okay?

Q. Okay. All right. Go ahead. You can go back to the stand. Thank you.

Let's go to the Gianelli graph. It's actually -- don't kill me, Dr. Weisenburger, but I think you're going to have to

come off the stand because this is a blow-up. I'm sorry.

A. Oh.

Q. You're going to get your exercise.

Someone's got my stuff out of order. Oh, here we go.

MS. MOORE: Counsel, this is Gianelli.

MR. STEKLOFF: No objection.

MS. MOORE: And we'll mark this for identification purposes as 938.

(Trial Exhibit 938 marked for identification)

BY MS. MOORE:

Q. And, Dr. Weisenburger, can you explain to the jury what this chart is that we're looking at?

And if I could, Your Honor, may I publish this to the jury?

THE COURT: Sure.

THE WITNESS: So this is from one of the studies of hepatitis C and it shows what happens when patients have a complete virologic response like Mr. Hardeman had to the antiviral therapy for hepatitis C. Okay?

So here he has the infection, and -- I'm trying to see what -- the black box is one of the liver function tests called the ALC; and, of course, his liver function tests are remarkably abnormal and elevated at the time of the diagnosis of hepatitis. Now, Mr. Hardeman's weren't quite this high, but his tests were also elevated, his liver function tests.

And this shows the viral load. Okay? This diamond again is very high, about here 5 million copies of viral RNA in his -- per mil. Okay?

BY MS. MOORE:

Q. And what does viral load represent?

A. So viral load just tells you how much virus you have circulating in your blood. Okay? And Mr. Hardeman had about, I think, 2 million copies but still a significant number.

So --

Q. And this graph here, the Gianelli graph, this is from an actual patient?

A. This is an actual patient that was treated with antiviral therapy in that paper. Okay?

Q. Go ahead.

A. Yeah. So it shows you. So you can see when you give the antiviral therapy, what happens. You get a dramatic decrease in the liver enzymes and they come back to normal. Okay? And they stay normal over here (indicating) over a two-month -- over a two-year period.

And the same thing happens. There's a dramatic decrease in the viral RNA in the blood and it goes down to zero -- okay? -- which is exactly what happened to Mr. Hardeman, and then it stayed negative over the 24 months.

So Mr. Hardeman was very much like this patient who was treated in the paper. And here you see the viral RNA test.

It's initially positive and then it becomes negative over time. Okay?

So this is what --

Q. And is that --

A. -- this is what would have happened to Mr. Hardeman. Okay?

Q. And so if we go back to our Exhibit 940, Dr. Weisenburger, here --

A. Here's the positive (indicating) and then --

Q. So that's before the treatment?

A. Yep. And then here are all the negatives (indicating).

Q. And that's after the treatment?

A. After the treatment, yes.

And so this is the kind of response that Mr. Hardeman had to the treatment for his hepatitis C infection.

Q. So what does this tell us?

A. Well, what this tells us is that once you treat these patients with antiviral therapy and they have a sustained virologic response, their liver disease stabilizes and it doesn't get worse, and there's little or no continued damage to the liver and the virus is completely gone from the blood and is no longer causing disease. Okay?

And, in fact, what I'm going to show you is once you get to this state of sustained virologic response, you no longer have an increased risk for non-Hodgkin's lymphoma -- okay? --

which was what the state Mr. Hardeman was in for nine years before he got the non-Hodgkin's lymphoma.

So it's very unlikely, it's almost impossible that the hepatitis C could have caused his non-Hodgkin's lymphoma because he was cured of the hepatitis C nine years before he got the non-Hodgkin's lymphoma.

Q. Okay. If you want to go back to the stand, and I think there's some other literature.

A. Right.

Q. And I'll direct you to -- this is Tab 1531 in your binder.

MS. MOORE: And permission to publish, please.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

THE WITNESS: So this is some data on hepatitis B and hepatitis C viral infection and the risk of non-Hodgkin's lymphoma. It was a case-control study from Italy and, I mean, what the study showed is that if you have chronic active viral infection with either hepatitis B or hepatitis C, you are at about a two- to threefold increase risk of non-Hodgkin's lymphoma.

But as I'll show you, if you have a history of past infection and you no longer have the infection, then the risk goes away.

So let's -- there's a table in this paper that I want to show you. Can we hone in on table -- this table? Yeah.

So basically this -- the top half -- this doesn't really show very well. It shows hepatitis C. So she's got the bar where it says "HCV." That's hepatitis C. So we're going to talk about hepatitis C first. Okay?

So if you -- and this is -- it's done in a case-control format. So you have an odds ratio -- you have the cases, you have the controls, and then you have the odds ratio on the far right. Okay?

So the first category of people are the people who have immunity to hepatitis C virus. Okay? And they have the antibody but they don't have the viral infection. Okay?

No, these are the people who never -- I'm sorry. These are the people who don't have the antibody. So they have never been exposed to hepatitis C. So this is your control group. Okay? And they have a risk ratio of 1.

If you look at the ones that have the antibody to hepatitis C, then you see the risk goes up to over twofold like I told you. So if you have had an -- if you have a history of an infection with hepatitis C, you have an over twofold increased risk.

But then if we divide that into --

BY MS. MOORE:

Q. Dr. Weisenburger, can I stop you for a second?

So the first row where it says anti-HCV, anti-hepatitis C --

A. Then it says negative. There's a little negative there.

Q. Oh. There you go.

A. You can't see it.

Q. Off the V.

Okay. Sorry the copy is bad.

A. Yeah.

Q. So the anti-HCV negative there, those are people who do not have hepatitis C?

A. They never had hepatitis C --

Q. Okay.

A. -- right.

Q. So that's a control group?

A. That's sort of your --

Q. Like a control group?

A. -- control group, right.

Q. Okay. And then the next row are people who do have hepatitis C?

A. They have antibody to hepatitis C. So we know that they either have an active infection or they have had the infection in the past. We don't really know which of those is, and that's why you have to test for the hepatitis C RNA, which is the next line. Okay?

And so if you test -- so you take these people that have the antibody and you say, "Okay. Do they have active infection or not?"

So you measure the hepatitis C virus in the blood; and if it's negative, look across. There's no increased risk for non-Hodgkin's lymphoma. The number is essentially 1. Okay? Just like the people who never had it.

But if you look at the next line, people who have the virus in their blood, those are the ones who are at increased risk for non-Hodgkin's lymphoma.

So this makes the point that I told you that you have to have chronic active viral hepatitis at the time you're diagnosed with non-Hodgkin's lymphoma -- okay? -- to give you an increased risk for non-Hodgkin's lymphoma.

If you're immune to it, like the column above where they have the antibody but they don't have the virus, then you're immune to it. Okay?

Q. So in Mr. Hardeman's case, when he's cured of hepatitis C in 2006 and diagnosed with non-Hodgkin's lymphoma in 2015, what conclusions can you draw from this table here?

A. Well, from this table if you have -- if you don't -- if you have the antibody, which he did, and you don't have the virus, then you're actually on the line between the two yellow lines where you have an odds ratio of .98, which is essentially 1, which is the same for your people who never had a hepatitis C infection. Okay?

So just the fact that you had an infection in the past does not put you at any increased risk for non-Hodgkin's

lymphoma. It's only if you have an active infection that's ongoing right up until the time you get non-Hodgkin's lymphoma, which he didn't have.

Q. Okay.

A. So let's go to the bottom half of that table because it also then talks about hepatitis B, you know. So this is the same format, and there are three different tests here. Let's go across the first row. Okay?

So it's hepatitis surface antigen negative. Hepatitis -- so that's the antigen, that's the actual virus, the measure of the virus in the blood.

Q. What's an antigen?

A. It's the -- it's part of the coat -- the coat of the virus. It's a protein that's part of the coat of the virus, and you can measure it. And so it tells you the virus is there because you're finding the protein of the virus.

So in that first group, which is your control group, we have -- you can't really see it very well unfortunately, but it's hepatitis surface antigen negative and antibody surface negative and antibody core protein negative. So these are people who have never had hepatitis B. Okay? They have no evidence of infection. They have no evidence of immunity. Okay?

Q. Just like the line from the hepatitis C, the first line?

A. Right. So the odds ratio for them is 1. They're your

control group again. Okay?

Now, the next group is a group that has hepatitis surface antigen negative, does not have the surface antibody, but has the core antibody. Okay? And that's what Mr. Hardeman had. When they tested him, he was negative for the surface antigen, he was negative for the surface antibody, and he was positive for the core antibody showing that he had immunity.

And if you go across, you can see that the odds ratio is less than 1. So there's no increased risk for people who have immunity to hepatitis C virus, which is what he had at the time he was diagnosed with lymphoma.

If you go to the next one, again, it's hepatitis -- I mean, I can't read it because I have to pull up --

Q. Do you want the actual study?

A. I want the actual study because I can't read the --

Q. Sorry. If you turn in your binder to 1531 and it's page 4.

A. (Witness examines document.) All right. That's not much better.

(Witness examines document.) I don't have my copy.

(Witness examines document.) Yeah, so the second row, unfortunately, is -- the antigen is negative but both antibodies are positive. So he's immune. Okay?

So he was actually this -- the third one -- the third one is the antigen negative, the surface antibody positive, and the

core antibody negative. And, again, there's no increased risk. Okay? So he's immune.

The fourth one is again the antigen is negative, the surface antibody is negative, and the core antibody is positive. And that's what Mr. Hardeman had. I'm sorry. It was this fourth one.

So he didn't have any evidence of active viral infection, and he had at least the anti-core antibody saying that he was immune. And, again, you can see the risk ratio is 1.1. It's very close to 1 and it's not significant. So he would not have been at increased risk for non-Hodgkin's lymphoma based on this.

And then the last one is people who have -- are surface antigen positive. That indicates that they have active viral infection. And if you look across, those are the people -- that's the only group that has an increased risk of non-Hodgkin's lymphoma of about twofold increased risk. Okay?

Q. And does Mr. Hardeman fall in that last list?

A. No, he doesn't. He falls in the one above it.

Q. Okay.

A. Okay. So basically this is data that shows you if you're immune to hepatitis C and you're immune to hepatitis B, you're not at increased risk for non-Hodgkin's lymphoma. It's only when you have the active viral infection that you're at increased risk.

Q. Okay. All right. So then were there other studies that you relied on in forming your opinion regarding the hepatitis C?

A. So I just want to show you some examples of some of the studies that prove what I just told you.

Q. All right. Let's go to I think it's Tab 1291.

MS. MOORE: Permission to publish.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

THE WITNESS: So this is a study from Japan in which they had a large number of people with hepatitis C, many of which were untreated and some of which were treated. And of those that were treated, there was a group that had a sustained virologic response just like Mr. Hardeman, and there was a group that didn't have a sustained virologic response. They had a response but it wasn't a complete response. Okay?

And so let's look at the curve.

MS. MOORE: Page 1039, please.

THE WITNESS: And read the title too. It says "Viral" -- go back to the title. It's important to read the title actually because it tells you what the study shows. So "Viral elimination" -- they're talking about hepatitis C -- "reduces the incidence of malignant lymphoma in patients with hepatitis C."

Okay. Let's look at the curve.

MR. WOLFE: Sorry. Which table was it?

MS. MOORE: 1039, Figure 3. It's the top half. There you go.

THE WITNESS: So let's look at the figure. So what this shows you is the time, a period of 5, 10, and 15 years along the bottom of the curve; and on this upper side on the other axis you see the incidence rate of non-Hodgkin's lymphoma. Okay?

Yes. And what you can see is that the people with persistent infection over time had an increasing incidence of non-Hodgkin's lymphoma. So these are the people who were never treated and had chronic persistent infection, chronic active viral infection, and the people who were treated but who didn't have a sustained virologic response. Okay? So you can see that they over time are at increased risk for non-Hodgkin's lymphoma.

But if you look at the patients who had a sustained virologic response like Mr. Hardeman, it goes right along the base. There weren't any cases of non-Hodgkin's lymphoma, not one case in 15 years.

And actually if you go out to 20 years, there was only one case and it was a T cell lymphoma, which was probably unrelated to the virus at all.

So in this study, it's very dramatic, there's a statistic -- a marked statistically significant difference in

these curves showing that people who have a sustained virologic response don't have any increased risk for non-Hodgkin's lymphoma.

BY MS. MOORE:

Q. So let me ask you two things from that. When you keep saying SVR, the sustained virologic response, when we go back to Exhibit 940 and we have marked here from February 23rd, 2006, all the way down to June 7th, 2015, and it's written "Negative," what does that signify with respect to the sustained virologic response for Mr. Hardeman?

A. Well, what it tells you is that once he attained a sustained virologic response, he was no longer at increased risk for non-Hodgkin's lymphoma.

Q. So this negative is he no longer has a sustained -- he has a sustained virologic response; correct?

A. Yeah. So he's one year out there. So he -- you know, after six months of negatives, you consider it a sustained virologic response. And obviously he stayed in remission during that whole nine-year period.

Q. And then the second thing is you said that there was one person, I think about at the 20-year marker, that had the infection and it was the T cell.

A. Right. So it was probably a background case, not related at all to the non-Hodgkin's lymphoma because patients with hepatitis B and hepatitis C, they get B-cell lymphomas. They

don't get T-cell lymphomas.

Q. Okay. So that's not relevant then?

A. It's not relevant, no.

Q. Okay. All right.

Anything else from this graph that you relied on in forming your opinions in this case?

A. No.

Q. Okay. Let's go to 917.

MS. MOORE: And permission to publish, please.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

THE WITNESS: So this is another study that looked at the frequency of liver cancer and non-Hodgkin's lymphoma in patients with hepatitis C virus infection, and it's a cohort study from Denmark. Okay?

And I'm just going to show you the data for non-Hodgkin's lymphoma here.

MS. MOORE: So let's go -- Mr. Wolfe, if we could turn to page 2314 and the graph in the lower right corner for non-Hodgkin's lymphoma, please.

THE WITNESS: So, again, this is a similar graph as I showed you before. Time is on the bottom part of the curve, it goes out 10 years, and the cumulative incidence is on the other part of the curve.

And what you see here, if you look at the dashed line,

which is the second line from the top -- can you show that? The small dashes?

Right there. Yeah.

That's the line for people who have a history of hepatitis C infection. They have a positive antibody. Okay? So they either have active infection or they have a history of past infection. Okay?

But like we did on the table, we can split that into two groups -- right? -- the ones that have the active viral infection, that have the RNA in the blood, and that's actually the dark curve above, the solid curve. Okay? So they're the ones who have a high infection.

And, in fact, the people who have immunity to hepatitis C are the very low curve made up of the dots. Okay? So -- and then the large dashes are the expected rate of non-Hodgkin's lymphoma in the population.

So you can see that there's no difference in the curve -- the incidence curve for people who never had hepatitis C compared to the lower dotted curve of people who are immune to hepatitis C.

BY MS. MOORE:

Q. So what does --

A. It's only the people who have active infection who have an increased risk.

Q. So what does that mean with respect to Mr. Hardeman?

A. Well, it means that his risk for non-Hodgkin's lymphoma was no different than anybody else's who never had hepatitis C.

Q. Okay. And then those long dashes on this graph, that's the incident rate of people who have hepatitis?

A. Who never had hepatitis.

Q. Who never had hepatitis.

A. So that's sort of your background rate in the general population. Okay?

Q. So once you're treated for hepatitis and you're cured, then you go back to where you were as if anyone in the general population?

A. Yeah. So it's the line of dots, which is pretty much similar to the large dashes. Those two are identical curves. They look a little different, but statistically they're the same.

Q. All right. And so what percentage, then, would you put on someone who at one point in time had active hepatitis C, then became cured of hepatitis C, stayed in the sustained virologic response? What is the percentage risk that they might have to develop non-Hodgkin's lymphoma?

A. There wouldn't be any increased risk.

Q. Okay. All right. Let's turn to 918.

MS. MOORE: And permission to publish.

THE COURT: Before we go there, let me ask you. How much longer do you think you have with your direct of

Dr. Weisenburger?

MS. MOORE: I would say probably 15, 20 minutes.

THE COURT: Okay. In light of that, I think we should break -- we should end for the day before you finish with Dr. Weisenburger, but do you want to finish on the topic maybe of hep C and then we'll wrap up for the day?

MS. MOORE: I would leave that up to the jury, Your Honor. It doesn't matter.

THE COURT: Why don't you go ahead.

MS. MOORE: Okay.

THE COURT: It seems like you're almost done with hep C.

MS. MOORE: I am.

Okay. So let's go to 918. And permission to publish.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE:

Q. And what does this study tell us, Dr. Weisenburger?

A. So this is another study that looks at antiviral therapy and the risk of non-Hodgkin's lymphoma with hepatitis C infection. It's a recent study from China, and it also makes the point that I made before if we can look at the diagram.

Q. Well, let's look. The title of it is early antiviral therapy reduces risk of lymphoma in patients with chronic hep C; is that right?

A. Right.

Q. Okay. So let's go to page 336, and I think it's the diagram for non-Hodgkin's lymphoma, the B diagram, please.

A. So this is another similar curve like I've shown you with the years on the lower scale and the incidence of NHL on the other scale. And what you see for people who have untreated hepatitis C virus, they have the dotted line, which is the increasing incidence over time. Okay?

The small dotted line are people who never had hepatitis C viral infection. Okay?

And then the other -- the solid line just above that are the people who got a full course of Interferon and antiviral therapy, and again you can see that the curve pretty much matches the unexposed curve. Okay?

Probably it begins to stray a little bit because some of those just had a partial response; but, in fact, if you get treated, your risk of non-Hodgkin's lymphoma is the same as the general population. It's not increased.

So it shows you the same -- in three different studies I've sort of shown you the same thing, and there are actually another five studies out there that I'm not even going to show you, but there are eight studies that make this point very -- very well.

Q. And this -- Dr. Weisenburger, these studies and these graphs you've been showing to the jury, is this based on real

data of real people?

A. Yes. It's real data of real people who had hepatitis C infection and were treated with antivirals just like Mr. Hardeman.

Q. It's not hypothetical about what might happen if you have antiviral therapy following a diagnosis of hepatitis C?

A. No, this is real data.

MR. STEKLOFF: Objection.

THE COURT: Sustained.

MS. MOORE: Okay.

THE COURT: The answer will be stricken.

BY MS. MOORE:

Q. Dr. Weisenburger, is this information, the data that you've been showing in these last three studies -- Exhibits 1291, 917, and 918 -- is that based on real data or a hypothetical situation?

MR. STEKLOFF: Objection.

THE WITNESS: It's real data and real patients.

THE COURT: Sustained -- I mean, overruled. Sorry.

BY MS. MOORE:

Q. And, Dr. Weisenburger, if hepatitis C was really causing non-Hodgkin's lymphoma after someone is cured, would you see it in the literature in the data?

A. Well, you should see it because there's lots of data on large numbers of patients, and the data is very consistent

across all eight studies that there's no increased risk for non-Hodgkin's lymphoma for people who are cured or people who become immune spontaneously. If you don't have active infection, you're not at increased risk.

Q. Okay. And if you turn to 1302 in your binder.

MS. MOORE: Permission to publish.

MR. STEKLOFF: No objection, Your Honor.

THE COURT: Go ahead.

BY MS. MOORE:

Q. And what is this publication, Dr. Weisenburger?

A. Well, this is another study which shows similar findings, but this study shows them for hepatitis B. So the story is the same for hepatitis B, that if you don't have a chronic active viral infection at the time you get non-Hodgkin's lymphoma, then the hepatitis B is not the cause.

MS. MOORE: And let's go to the second page, please, Mr. Wolfe.

THE WITNESS: Now, let's hone in just on the top two diagrams.

Yeah. So this is now we're talking about hepatitis B, okay? So remember we said that if you have the hepatitis B surface antigen, which is the first one on the top diagram there, and you look across this bar graph, it's like the four spots that Dr. Ritz showed you, you can see there's an increased risk of 1.82 for non-Hodgkin's lymphoma with active

hepatitis B infection.

But then if you go down to the next one, it says anti-hepatitis C. So this person has the antibody, the hepatitis C, like Mr. Hardeman did, and you can see the risk is basically at 1. There's no increased risk. Okay?

And the same is true if you have the antibody to the surface antigen. The risk is close to 1. It's not elevated. They're immune -- these -- the lower two items are immune, and they have no increased risk of non-Hodgkin's lymphoma associated with hepatitis B infection.

And I won't belabor the point, but the curves -- the data is the same for diffuse large B-cell lymphoma here. You have an increased risk of over twofold with hepatitis -- with active hepatitis infection with hepatitis surface antigen; but if you have the antibodies, the risk is around 1 or less.

So both for hepatitis C and hepatitis B, if you're immune or you're cured, you don't have an increased risk of non-Hodgkin's lymphoma, and that's why I crossed out hepatitis C and hepatitis B as substantial risk factors for Mr. Hardeman -- okay? -- on my differential list.

BY MS. MOORE:

Q. Dr. Weisenburger, I'm going to have you, before we adjourn for the day, come down off the stand and we're going to put up the differential again.

THE COURT: Why don't we do that tomorrow morning?

MS. MOORE: Do it tomorrow morning? Okay. Great. Thank you.

THE COURT: Okay. So we're wrapping up for today. We'll resume again at 8:30 sharp tomorrow. And as I mentioned to you back there this morning, even though we lost yesterday, we're still a little bit ahead of schedule so that's the good news. We look forward to seeing all six of you at 8:30 sharp tomorrow.

Thank you.

(Proceedings were heard out of the presence of the jury:)

THE COURT: And, Dr. Weisenburger, you're free to step down.

THE WITNESS: Thank you.

THE COURT: Everybody, remember, nobody leaves the courtroom for five minutes to give the jurors a chance to use the elevators. So nobody is allowed to leave the courtroom until they hear from either me or Kristen.

Okay. Anything else to discuss?

MS. MOORE: I don't think so, Your Honor.

MR. STEKLOFF: No, Your Honor.

THE COURT: I mean, it seems pretty clear that you're not going to need to have Dr. Arber here tomorrow; right? I mean, we're going to have -- you're going -- how long do you anticipate your cross of Dr. Weisenburger to be?

MR. STEKLOFF: It might be a couple hours.

THE COURT: And then we have another hour of video testimony.

MS. MOORE: Yes, Your Honor.

THE COURT: And then you'll put on Dr. Mucci, and she likely will not finish tomorrow.

MR. STEKLOFF: I agree, Your Honor.

THE COURT: Okay. That's fine.

Okay. Anything else from anyone?

MS. MOORE: I don't think so, Your Honor.

THE COURT: Okay.

MS. MOORE: Thank you.

THE COURT: Sounds good. So everybody sit tight in the courtroom for a couple more minutes, and you'll hear from Kristen when you are permitted to leave.

(Proceedings adjourned at 2:42 p.m.)

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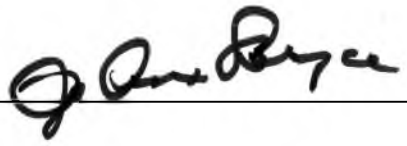
CERTIFICATE OF REPORTERS

I certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

DATE: Tuesday, March 5, 2019



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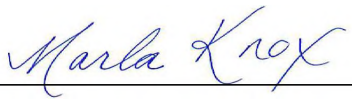

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