

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

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By Video Testimony (not reported)

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HARDEMAN, EDWIN

(SWORN)

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PROCEEDINGS

1 Tuesday - March 5, 2019

8:28 a.m.

2 P R O C E E D I N G S

3 ---000---

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

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

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

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

12 **MS. WAGSTAFF:** No, Your Honor.

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

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

17 **THE COURT:** Oh, yeah, okay.

18 **MS. WAGSTAFF:** It is about four minutes.

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

22 **THE COURT:** Which exhibit is that?

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

PROCEEDINGS

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

3 **MS. WAGSTAFF:** Okay.

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

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

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

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

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

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

17 EDWIN HARDEMAN,
18 called as a witness for the Plaintiff, having been duly sworn,
19 testified as follows:

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

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

25 **THE CLERK:** Thank you.

HARDEMAN - DIRECT / MOORE

1 **THE WITNESS:** You are welcome.

2 **DIRECT EXAMINATION**

3 **BY MS. MOORE**

4 **Q.** Good morning, Mr. Hardeman.

5 **A.** Good morning.

6 **Q.** Have you ever testified in court before?

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

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

12 How did you learn about Roundup?

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

18 **Q.** Did you say Gualala?

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

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

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

8 **Q.** And what year was that?

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

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

14 **A.** Yes.

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

16 **A.** Well, I used it in -- I would say it was 19 -- either late
17 1985 or early 1986 to my recollection I started spraying it.

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

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

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

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

6 A. Yes.

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

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

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

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

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

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

21 Q. And who would actually mix the Roundup?

22 A. I mixed it.

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

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

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

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

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

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

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

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

17 A. Approximately, yes.

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

19 A. Yes, I did.

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

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

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1 approximately 200 feet.

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

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

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

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

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

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

21 A. Yes.

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

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

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

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

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

16 **Q.** How old would you have been then?

17 **A.** I'm sorry.

18 **Q.** How old would you have been then?

19 **A.** 39, going on 40.

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

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

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

6 Q. Let me --

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

10 THE COURT: Sure.

11 BY MS. MOORE

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

15 A. 23, okay.

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

17 A. Yes.

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

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

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

1 Road property?

2 A. Yes.

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

5 THE COURT: Any objection?

6 MS. MATTHEWS JOHNSON: No objection.

7 THE COURT: Go ahead.

8 MS. MOORE: Thank you.

9 BY MS. MOORE

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

13 A. Sure.

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

15 THE COURT: Sure.

16 BY MS. MOORE

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

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

21 THE COURT: What exhibit is it?

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

24 THE COURT: That's fine.

25 \\\

HARDEMAN - DIRECT / MOORE

1 **BY MS. MOORE**

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

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

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

9 **Q.** What is this Parcel 2 here?

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

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

14 **BY MS. MOORE**

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

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

21 **A.** All right.

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

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

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

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

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

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

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

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

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

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

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

8 **Q.** A poison oak festival?

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

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

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

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

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

6 Q. I'm sorry, Mr. Hardeman.

7 A. Yes.

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

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

17 Q. Did you have your own water system?

18 A. Yeah. It was on a spring fed, a natural mountain spring.
19 It was all gravity fed down to the house. The water was -- we
20 had -- it is all based upon water storage. There is, like,
21 22,000 gallons of water. The largest being a 15,000-gallon
22 tank. In the spring is -- we have a spring box up here, which
23 takes the water from a short little fall off of a rock and
24 collects it, and then distributes it to these other tanks. And
25 the spring itself, the source of the spring, is further up. So

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

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

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

10 **Q.** Okay. And you mentioned hiking trails?

11 **A.** Uh-huh.

12 **Q.** Where is the hiking trails on the property?

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

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

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

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

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

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

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

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

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

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

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

2 Q. Okay.

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

4 Q. That's okay.

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

8 A. Oh, yes.

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

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

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

17 A. Probably 25 years.

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

20 A. Thank you.

21 Q. Here.

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

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

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

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

9 **A.** No, not practical.

10 **Q.** So where -- that is not practical?

11 **A.** No.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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1 Side property for those 25 years?

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

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

6 **A.** It's a pump-up sprayer.

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

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

12 **THE COURT:** Any objection?

13 **MS. MATTHEWS JOHNSON:** No objection.

14 **THE COURT:** Admitted.

15 (Trial Exhibit 23 received in evidence)

16 **MS. MOORE:** Thank you, Your Honor.

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

18 **A.** Yes.

19 **Q.** And what is that?

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

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

24 **THE COURT:** Any objection?

25 **MS. MATTHEWS JOHNSON:** No objection.

1 **THE COURT:** Go ahead.

2 **BY MS. MOORE**

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

6 **A.** Yes, this is.

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

9 **THE COURT:** Any objection?

10 **MS. MATTHEWS JOHNSON:** No objection.

11 **THE COURT:** Admitted.

12 (Trial Exhibit 25 received in evidence)

13 **BY MS. MOORE**

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

17 **A.** Yes.

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

19 **THE COURT:** Sure.

20 **MS. MOORE:** Thank you.

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

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

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

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

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

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

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

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

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

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

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

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

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

13 THE COURT: What is it?

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

15 THE COURT: Okay.

16 MS. MOORE: Thank you.

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

18 A. Yes.

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

21 A. Yes.

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

24 MS. MATTHEWS JOHNSON: The picture, no objection.

25 THE COURT: Okay. Admitted.

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1 (Trial Exhibit 946 received in evidence)

2 **BY MS. MOORE**

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

7 **A.** Yes. Uh-huh.

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

10 **A.** Yes, I do.

11 **Q.** And what are these photos depicting?

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

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

17 **A.** Yes.

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

20 **A.** Yes.

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

23 **MS. MATTHEWS JOHNSON:** No objection.

24 **THE COURT:** Go ahead.

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

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1 evidence as well.

2 **MS. MATTHEWS JOHNSON:** No objection.

3 **THE COURT:** Admitted.

4 (Trial Exhibits 940 through 945 received in evidence)

5 **BY MS. MOORE**

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

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

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

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

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

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

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

5 A. 930.

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

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

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

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

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

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

8 **A.** I'm sorry?

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

11 **A.** I don't understand your question.

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

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

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

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

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

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

5 A. No. I did it myself.

6 Q. Why did you do it yourself?

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

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

12 Mr. Hardeman, if you want, you can look on the screen
13 there in front of you. What is this that we are looking at in
14 933?

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

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

24 A. Yes.

25 Q. And it will -- is that right?

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

5 **Q.** Let's go to 934.

6 **A.** Okay.

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

8 **A.** This is the concrete water system that -- it is a
9 15,000-gallon tank. You can see that there is remnants of
10 poison oak popping up in the driveway there. It is there, and
11 along the -- I would -- as you look up that road up there, that
12 goes up another -- you know, several hundred feet to that
13 roundabout, yeah, up to the top there; and that's kind of where
14 that road ends. But off to the left there is that other tank,
15 the other blue dot on there, it is a 2,500-gallon tank. So I
16 would go up there and there was poison oak growing up around
17 the plumbing. It was important to keep the plumbing -- because
18 it would go up there, and you had shut off valves and whatever,
19 and I didn't want to get it on my hands. There was a lot of
20 stuff growing up in there.

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

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

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

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

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

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

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

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

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

1 and whatever.

2 Q. What about when you use the hand sprayer?

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

14 Q. Do you still use Roundup?

15 A. No.

16 Q. When was the last time you used Roundup?

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

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

22 A. Uh-huh.

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

25 A. No.

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

2 A. No.

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

5 A. Yes.

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

8 A. 2005, to the best of my recollection.

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

11 A. Yes.

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

13 A. In 2005.

14 Q. Did that treatment go into 2006?

15 A. It finished in 2006.

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

18 A. I was cured.

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

21 A. No, never.

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

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

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

3 A. Yes.

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

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

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

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

12 A. No.

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

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

19 Q. Where was that, Mr. Hardeman?

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

23 Q. When did you have that sun spot?

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

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

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

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

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

8 A. No, never had any problems with it.

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

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

20 Q. And that was in August of 2018?

21 A. Yes, last year.

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

24 A. Yes.

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

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

2 Q. Were either of those malignant?

3 A. No, no, they were not malignant.

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

7 A. Well, official diagnosis was in, I think, in February of
8 2014 -- '15, '15, yes.

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

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

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

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

21 He said, Well, why don't you just observe it for the next
22 30 days. It could be a swollen lymph node. Okay. My doctor
23 was on vacation.

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

1 here.

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

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

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

11 A. The --

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

14 A. It was 2014, yes.

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

17 A. Okay.

18 Q. But did you go through chemotherapy?

19 A. Yes, I did.

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

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

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

25 A. No.

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1 Q. And how do you know that?

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

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

14 A. No, never.

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

17 THE COURT: Any cross?

18 MS. MATTHEWS JOHNSON: Thank you, Your Honor.

19 We have no questions, sir.

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

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

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

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1 **THE COURT:** Is Dr. Weisenburger next?

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

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

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

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

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

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

11 **THE COURT:** Hello, Dr. Weisenburger.

12 **THE WITNESS:** Good morning.

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

16 **MS. MOORE:** I am, Your Honor.

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

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

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

23 **MS. MOORE:** No.

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

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1 understands.

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

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

13 **THE COURT:** In connection with his general causation
14 opinion?

15 **MS. MOORE:** That's correct, Your Honor. That's
16 correct, Your Honor.

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

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

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1 and Eriksson studies.

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

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

12 **MS. MOORE:** That's correct.

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

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

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

20 **DR. WEISENBURGER:** Yes, Your Honor.

21 **THE COURT:** Okay.

22 **MS. MOORE:** Okay.

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

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1 **THE COURT:** Thank you.

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

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

13 **MR. STEKLOFF:** Okay.

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

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

25 **THE COURT:** Of course, that's fine.

PROCEEDINGS

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

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

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

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

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

16 And you can bring in the jury.

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

19 **THE COURT:** Okay.

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

21 **THE COURT:** Okay. You can resume.

22 **MS. MOORE:** Thank you, Your Honor.

23 Plaintiffs call Dr. Dennis Weisenburger.

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

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1 DENNIS WEISENBURGER,

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

4 **THE WITNESS:** I do.

5 **THE CLERK:** Thank you. Please be seated.

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

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

10 **THE CLERK:** Thank you.

11 DIRECT EXAMINATION

12 **BY MS. MOORE:**

13 **Q.** Good morning, Dr. Weisenburger.

14 **A.** Good morning.

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

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

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

25 **Q.** What is pathology?

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

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

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

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

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

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

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

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

9 Q. And what is hematopathology?

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

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

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

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

1 epidemiology research, into lymphoma.

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

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

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

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

22 **Q.** What is the City of Hope?

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

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

3 **Q.** What is the National Cancer Institute?

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

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

12 **A.** Yes.

13 **Q.** And are you Board certified?

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

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

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

1 protect us from infections and other things.

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

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

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

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

16 **Q.** But is cancer common among the general population?

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

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

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

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

2 Q. And where are you working now?

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

9 Q. So you gave up the administrative part?

10 A. Yeah.

11 Q. Do you miss that?

12 A. No.

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

16 A. Yes.

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

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

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

1 what that is?

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

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

10 **A.** Yes.

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

14 **A.** Yes.

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

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

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

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

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

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

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

19 **Q.** And are you a founding member of InterLymph?

20 **A.** Yes, I am.

21 **Q.** And why did InterLymph actually come into existence?

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

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

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

10 Q. And is InterLymph still active today?

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

16 Q. And are you still active with InterLymph?

17 A. I am, yes.

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

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

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

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1 Environmental Health and Toxicology. What is that?

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

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

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

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

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

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

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

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

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

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

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

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

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

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

1 publication in, in that disease.

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

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

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

9 **A.** Well, that's a tough question. A lot of them looked -- a
10 lot of them were looking at the cause of non-Hodgkin's
11 lymphoma; but I think the major ones, what you're asking me is,
12 you know, with regard to epidemiology studies, we did over
13 50 -- we wrote over 50 papers on epidemiology and the research
14 that we did at Nebraska and the research we've done in the
15 InterLymph group to try to understand the causes of
16 non-Hodgkin's lymphoma.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

5 Q. And who is Dr. Aaron Blair?

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

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

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

18 A. (Witness examines document.)

19 Q. It's probably at the very back.

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

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

1 A. Yes.

2 Q. And what year was that?

3 A. Oh, 1985.

4 Q. Okay.

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

7 MR. STEKLOFF: No objection.

8 THE COURT: Go ahead.

9 BY MS. MOORE:

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

11 A. Yes.

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

14 A. Yes.

15 Q. Okay.

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

18 Q. Okay. And this became the Nebraska story?

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

20 Q. So what is the Nebraska story?

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

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

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

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

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

16 **Q.** Why is that?

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

23 **Q.** Did you get some pushback about including women?

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

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

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

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

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

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

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

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

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

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

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

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

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

10 **THE COURT:** Go ahead.

11 **BY MS. MOORE:**

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

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

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

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

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

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

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

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

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

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

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

7 **MR. STEKLOFF:** Objection, Your Honor.

8 **THE COURT:** Overruled.

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

14 **BY MS. MOORE:**

15 Q. So you want to look at all three?

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

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

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

1 the workplace or in the environment.

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

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

12 **Q.** What's a surfactant?

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

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

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

25 **A.** Yes.

SIDEBAR

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

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

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

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

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

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

23 **MR. STEKLOFF:** Objection, Your Honor.

24 **THE COURT:** Quick sidebar.

25 **(The following proceedings were heard at the sidebar:)**

SIDEBAR

1	[REDACTED]
2	[REDACTED]
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22	[REDACTED]
23	[REDACTED]
24	[REDACTED]
25	[REDACTED]

1 (The following proceedings were heard in open court:)

2 BY MS. MOORE:

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

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

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

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

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

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

1 will get back into the circulatory system.

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

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

8 **A.** Yes.

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

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

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

16 **MR. STEKLOFF:** No objection, Your Honor.

17 **THE COURT:** Go ahead.

18 **BY MS. MOORE:**

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

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

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

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

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

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

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

21 **A.** Yes.

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

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

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

4 If you could bring that out. Thank you.

5 A. So there are three case-control studies. Here they're
6 talking about Nebraska. So Nebraska was the last case-control
7 study to be done in this group, and so we accrued cases from
8 1983 to 1986. Okay?

9 Q. That's the Nebraska story?

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

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

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

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

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

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

24 A. Okay.

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

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1 talking about Iowa, Minnesota, Kansas and Nebraska from De Roos
2 2003. Do you see that?

3 **A.** Yes.

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

7 **A.** Well, there's a mistake. So in Kansas it says 1976 to
8 1982 and it was actually 1979 through 1981. So there's a
9 mistake there.

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

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

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

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

WEISENBURGER - DIRECT / MOORE

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

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

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

6 MR. STEKLOFF: No objection, Your Honor.

7 THE COURT: Go ahead.

8 BY MS. MOORE:

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

12 A. Yes.

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

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

18 A. Yes. So --

19 Q. And I have a blowup.

20 A. Okay.

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

23 THE COURT: Sure.

24 BY MS. MOORE:

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

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

2 **A.** I'll speak up.

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

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

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

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

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

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

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

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

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

15 So I think for glyphosate it's more likely that it has a
16 curve like this B curve, like we saw for solvents, where it
17 takes a fairly long time to develop the cancers and one has to
18 wait a long time to see all the cancers, probably out to 30 or
19 40 years.

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

24 And so --

25 **Q.** Meaning from their first exposure?

1 **A.** Meaning from their first exposure.

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

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

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

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

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

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

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

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

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

3 **MS. MOORE:** Thank you, Your Honor.

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

5 **THE COURT:** Be back at quarter till.

6 **MS. MOORE:** Thank you, Your Honor.

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

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

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

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

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

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

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

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

23 **THE COURT:** You can resume.

24 **MS. MOORE:** Thank you, Your Honor.

25 \\\

1 **BY MS. MOORE**

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

6 **A.** What number is that?

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

9 **A.** Okay.

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

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

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

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

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

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

SIDEBAR

1 active ingredient, meaning glyphosate, is noncarcinogenic and
2 nongenotoxic?

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

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

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

12 **BY MS. MOORE**

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

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

18 **THE COURT:** Sure.

19 (The following proceedings were heard at the sidebar:)

20 [REDACTED]

21 [REDACTED]

22 [REDACTED]

23 [REDACTED]

24 [REDACTED]

25 [REDACTED]

1 [REDACTED] [REDACTED] [REDACTED]
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10 [REDACTED] [REDACTED] [REDACTED]
11 [REDACTED]
12 [REDACTED] [REDACTED] [REDACTED]

13 (Sidebar ended.)

14 (The following proceedings were heard in open court:)

15 **BY MS. MOORE**

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

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

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

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

3 **A.** Formulations like Roundup, yes.

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

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

10 **MR. STEKLOFF:** Objection. I move to strike.

11 **THE COURT:** Overruled.

12 **BY MS. MOORE**

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

15 **A.** Yes. There were six case control studies.

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

18 **THE COURT:** Okay.

19 **BY MS. MOORE**

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

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

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

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

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

10 **Q.** Which ones are those?

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

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

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

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

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

1 perhaps increased.

2 So here is the *De Roos* study with the increased risk at
3 2.1. And it is statistically significant, okay. And it was
4 adjusted for other pesticides. There are two other studies
5 that were also adjusted for other pesticides to rule out this
6 issue.

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

17 And the same thing happened when they did adjustments in
18 the *Eriksson* study where the risk went from about 2 down to
19 1.5. It was statistically significant, but then it became --
20 it was no longer significant. It was elevated, but it was no
21 longer significant. So again, you see the risk going down, but
22 not going down to 1. There is still a 50 percent increased
23 risk there, okay.

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

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

11 **Q.** What does that mean?

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

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

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

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

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

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

12 **BY MS. MOORE**

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

17 **A.** Yes.

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

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

1 the risks were lower. So the risk went to -- if I can write
2 this here -- 1.6, and the confidence intervals were like this
3 0.9 to 2.8.

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

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

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

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

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

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

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

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

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

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

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

21 **Q.** What is an abstract?

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

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

6 **Q.** Are abstracts peer reviewed?

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

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

13 **MR. STEKLOFF:** Objection. Leading.

14 **THE COURT:** Sustained.

15 **BY MS. MOORE**

16 **Q.** Has the --

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

19 **THE WITNESS:** I see. Thank you.

20 **MS. MOORE:** I will rephrase it.

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

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

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

8 **Q.** And who attends these international meetings?

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

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

16 **A.** Sure.

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

19 **A.** Yes.

20 **Q.** Okay.

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

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

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

5 **THE COURT:** Okay.

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

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

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

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

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

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

5 **BY MS. MOORE:**

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

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

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

10 **A.** Yes.

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

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

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

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

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

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

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

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

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

25 The other thing that is important about NAPP is in NAPP

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

6 **Q.** A proxy respondent, meaning --

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

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

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

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

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

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

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

7 **A.** Yes, I did.

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

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

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

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

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

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

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

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

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

16 **Q.** And in what way?

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

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

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

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

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

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

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

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

2 Q. That was in and around 1996?

3 A. Yes.

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

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

12 MR. STEKLOFF: Objection --

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

15 You can continue.

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

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

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

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

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

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

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

25 \\ \\

1 **BY MS. MOORE:**

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

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

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

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

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

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

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

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

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

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

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

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

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

1 already as well as the Agricultural Health Study.

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

3 MS. MOORE: Permission to publish.

4 MR. STEKLOFF: No objection, Your Honor.

5 THE COURT: Go ahead.

6 BY MS. MOORE

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

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

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

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

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

17 A. Monsanto.

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

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

25 MR. STEKLOFF: Objection, Your Honor.

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

2 **THE COURT:** Hold on. There is an objection.

3 Basis?

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

5 **THE COURT:** Overruled.

6 **BY MS. MOORE**

7 **Q.** You can go ahead.

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

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

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

21 **A.** By Schinasi.

22 **Q.** Schinasi. Okay.

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

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

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

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

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

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

13 **MR. STEKLOFF:** No objection, Your Honor.

14 **THE COURT:** Go ahead.

15 **THE WITNESS:** What is the number?

16 **MS. MOORE:** 554.

17 **THE WITNESS:** Okay.

18 **BY MS. MOORE**

19 **Q.** Is this the Zhang meta-analysis?

20 **A.** Yes, it is.

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

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

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

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

3 A. Yes.

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

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

8 Q. Have you reviewed it since then?

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

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

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

15 **MS. MOORE:** Page 3, Mr. Wolfe.

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

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

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

3 **BY MS. MOORE**

4 **Q.** Then below that, Dr. Weisenburger, I think you said 2015
5 earlier. There's two publications out of the AHS, 2005 and
6 2018; is that right?

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

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

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

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

1 and the 2005 AHS. And, again, the numbers are very similar to
2 what they saw above; that there was a 40 to 50, almost
3 60 percent increase in non-Hodgkin's lymphoma. And on -- all
4 the numbers are statistically significant here, okay.

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

9 So what they did here is they -- where they could, they
10 adjusted for the use of other pesticides to get around and to
11 mitigate this issue of confounding the use of multiple
12 pesticides. And if you look at the unadjusted odds ratio using
13 2005 AHS, there is about a 70 percent increase, okay. It is
14 statistically significant. But when you adjust it, the odds
15 ratio goes down. And that makes sense, because now they are
16 taking away the effects of the other pesticides that could have
17 caused non-Hodgkin's lymphoma, and they are focusing just on
18 Roundup.

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

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

1 And it uses the new data from the Agricultural Health Study.

2 And it also attempts to do adjustment based on pesticide use.

3 So I think it is an important, very informative study.

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

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

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

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

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

1 NHL?

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

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

8 **A.** I did.

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

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

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

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

1 you are at increased risk for developing colon carcinoma.

2 That's why they take out the colon polyps.

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

9 **Q.** What is the significance of that?

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

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

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

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

23 **A.** Yes.

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

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

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

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

13 **A.** No.

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

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

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

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

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

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

19 **MS. MOORE:** Permission to publish?

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

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

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

3 **MS. MOORE:** Great.

4 Permission to publish.

5 **MR. STEKLOFF:** No objection, Your Honor.

6 **MS. MOORE:** 916.

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

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

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

21 **A.** I think this one is glyphosate.

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

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

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

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

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

9 **MR. STEKLOFF:** No objection, Your Honor.

10 **THE COURT:** Okay.

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

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

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

24 Then they increased the doses in micromolar doses, .4,
25 2.0, 10.0 and then high dose of 50. And what you see, if you

1 look at the next column under Mean percent cells, is you see
2 the increase in DNA damage in double-strand breaks going from
3 .33 with no exposure to a fivefold increase, 1.67. And then to
4 a number -- another four or fivefold increase to 9.33 with a
5 higher dose. So you see a dose response here.

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

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

16 **BY MS. MOORE:**

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

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

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

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1 **MS. MOORE:** Permission to publish?

2 **MR. STEKLOFF:** No objection, Your Honor.

3 **THE COURT:** Go ahead.

4 **BY MS. MOORE**

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

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

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

12 **A.** Yes, it is.

13 **Q.** If we could --

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

16 **BY MS. MOORE**

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

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

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

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

1 **A.** There it is.

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

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

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

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

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

25 **A.** The fourth one on the top.

1 Q. Okay.

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

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

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

17 Q. So the formulation is ten times more toxic?

18 A. Yes.

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

21 A. No.

22 Q. 563.

23 MS. MOORE: Permission to publish.

24 MR. STEKLOFF: No objection.

25 THE COURT: Go ahead.

1 **BY MS. MOORE**

2 **Q.** And what is this publication here?

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

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

12 **A.** Yes.

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

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

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

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

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

10 **BY MS. MOORE**

11 **Q.** And then the glyphosate.

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

13 **MS. MOORE:** So A, please.

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

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

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1 And that's what these recent studies did.

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

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

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

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

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

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

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

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

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

9 **MS. MOORE:** Thank you, Your Honor.

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

12 **THE COURT:** Sure.

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

16 **THE COURT:** Okay.

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

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

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

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

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

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

12 **THE COURT:** Somebody just walked out of the courtroom.

13 **THE CLERK:** Yeah.

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

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

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

21 **THE CLERK:** Okay.

22 **THE COURT:** Disregarded my orders by leaving the
23 courtroom.

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

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

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

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

14 **THE WITNESS:** That's okay. I kind of like it up here.

15 **THE CLERK:** Court is in recess.

16 (Luncheon recess was taken at 12:04 p.m.)

17 **AFTERNOON SESSION**

12:45 p.m.

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

19 **THE COURT:** All right. We are back. You can resume.

20 **MS. MOORE:** Thank you, Your Honor.

21 **BY MS. MOORE**

22 **Q.** Good afternoon, Dr. Weisenburger.

23 **A.** Good afternoon.

24 **Q.** When we broke for lunch, we were talking about
25 genotoxicity; and my question for you is: You spent time

1 showing the jury these studies about what happens to the
2 lymphocyte cells from humans in petri dishes, but what about
3 the real world? How do we know Roundup is genotoxic out in the
4 real world?

5 **A.** Yeah, so there are two studies done in South America that
6 I think are informative to answer that question.

7 **Q.** If you want to turn to -- it looks like Exhibit 1438 in
8 your binder.

9 **MS. MOORE:** And permission to publish.

10 **MR. STEKLOFF:** No objection, Your Honor.

11 **THE COURT:** Okay.

12 **BY MS. MOORE**

13 **Q.** Is this one of the studies that you are referencing,
14 Dr. Weisenburger?

15 **A.** Yes, this is a --

16 **Q.** Can you --

17 **A.** -- a study of actual people in Ecuador who were exposed to
18 large doses of Roundup herbicide sprayed from airplanes to
19 eradicate cocaine plants.

20 **Q.** And what were the findings from this study?

21 **A.** Well, this was a relatively small study. These were
22 rural -- rural inhabitants, farmers living right on the border
23 of Ecuador and Colombia. And they didn't use pesticides in any
24 of their farm work. And Colombia was spraying for these
25 cocaine -- these cocaine plants trying to eradicate cocaine,

1 using Roundup -- a more -- a stronger formulation of Roundup.
2 And they actually did three days of intensive exposure right on
3 the border, so these people got exposed multiple times. The
4 stuff drifted on over to their homes, and they were exposed.
5 And then they -- after that three days of intense spraying,
6 there were sporadic spraying for the next three weeks, so they
7 were exposed over a period of months multiple times to high
8 doses of Roundup that was sprayed from these airplanes.

9 **MS. MOORE:** And if we could, Mr. Wolfe, if we can go
10 to the abstract on page 1.

11 **BY MS. MOORE**

12 **Q.** The last sentence in the abstract. Dr. Weisenburger, so
13 what was the results -- what did the results show these authors
14 in this particular study?

15 **A.** Well, what the results showed is that the formulation of
16 Roundup that was used during aerial spraying had genotoxic
17 effects on the peripheral blood cells from these -- of these
18 people who lived in -- and were sprayed inadvertently.

19 **Q.** And I will show you -- I think that you extracted this
20 data --

21 **MS. MOORE:** If we can go to your PowerPoint slide on
22 that.

23 **MR. STEKLOFF:** No objection, Your Honor.

24 **MS. MOORE:** Thank you.

25 \\\

1 **BY MS. MOORE**

2 **Q.** What do we see here, Dr. Weisenburger?

3 **A.** So this is just the summary of the data. These
4 individuals used the comet assay, one of the same assays that
5 was used in the genotox studies I told you about before.

6 **Q.** You say "assay." What is that?

7 **A.** A test.

8 **Q.** A test?

9 **A.** Yeah, a test.

10 So here you can see there were two populations. There was
11 an unexposed population of similar people who lived far away,
12 80 kilometers away from where the border between the two
13 countries where the Roundup was sprayed. So you have an
14 unexposed group. And then you have the exposed group who were
15 exposed to this intensive spraying for three days followed by
16 three weeks of intermittent spraying.

17 And what they did is they drew blood on these people who
18 were exposed. It was 28 people over -- somewhere between two
19 weeks and two months after they were exposed. And they did
20 this comet assay to look for DNA damage, or in this case it's
21 called DNA migration. But what it really means is it is a
22 measurement of DNA damage. You can see that in the unexposed
23 the number was 25.94.

24 **Q.** And what does that mean?

25 **A.** Well, that is just sort of the baseline abnormalities that

1 they saw in their so-called normal controls. And then the
2 results increased to 35.5 in the people who were exposed. And
3 this is, as you can see from the p-value, highly statistically
4 significant.

5 So what this shows is that when innocent bystanders are
6 sprayed with Roundup at high -- high concentrations, the --
7 some of these people actually got sick from the pesticide,
8 okay. They were ill. And so they had very high doses that
9 actually made some of them ill.

10 When you look at their lymphocytes, you can see evidence
11 of genotoxic damage. So that -- this is sort of a real-world
12 kind of animal study where you are giving the animals high
13 doses.

14 Q. But in this case it is actually human beings?

15 A. Yes.

16 Q. Okay. And the next one, if you can turn to 1066.

17 MS. MOORE: Permission to publish.

18 MR. STEKLOFF: No objection.

19 THE COURT: Go ahead.

20 BY MS. MOORE

21 Q. And this is another study, Dr. Weisenburger. Did you
22 review this and rely on it in forming your opinion in this
23 case?

24 A. Yes, I did.

25 So this is the other study. This one was actually done in

1 Colombia. And, again, we are looking at basically agricultural
2 workers. This study -- it was interesting -- they used
3 couples. So they husband -- they had 30 couples in each group,
4 and they looked at exposure then, in both men and women, some
5 of which were agricultural workers, and they were actually
6 working in the fields.

7 **MS. MOORE:** And if we could, Mr. Wolfe, if you would
8 go over to 991 and pull up the graph at the bottom.

9 **BY MS. MOORE**

10 **Q.** What were the results of this, Dr. Weisenburger?

11 **A.** Yeah. So this is kind of a complicated slide. Let me
12 walk you through it. So the first bar on the far left labeled
13 underneath Santa Marta, this was a region in Colombia where
14 they do organic farming for coffee. So there are no pesticides
15 being used at all, okay. So this is your negative control,
16 okay. This is results on people who were not exposed to
17 pesticides at all.

18 The second group, Boyaca, is an area where they used
19 pesticides but they sprayed them, like Mr. Hardeman would have,
20 they sprayed them from tanks, manually walking along and
21 spraying the pesticides. And they used a variety of
22 pesticides. So this is sort of like your positive control,
23 okay, in the sense that they did two measurements one month
24 apart, and they found pretty much the same findings; that there
25 was a significant increase in damage to cells.

1 In this assay they are looking at what is called
2 binucleated micronuclei -- cells with binucleated micronuclei.
3 And there was this significant increase compared to the
4 control, but it was due just to the spraying of multiple
5 pesticides manually, okay. They probably were exposed to
6 glyphosate, but -- among many other pesticides that they used,
7 okay.

8 And then there are the three other regions -- Putumayo,
9 Narino and Valle -- and in these three areas they were spraying
10 the glyphosate, the Roundup, aerially just like in the other
11 studies. So they were trying to get rid of the cocaine plants
12 in Colombia by spraying -- by spraying the Roundup from
13 airplanes, okay. And they sprayed in these three regions.

14 And so what you see here is the first bar on each of these
15 last three was the test drawn just before the spraying, okay.

16 **Q.** So this bar, the one that is not filled in, is that the
17 one you are referring to?

18 **A.** The one right there, yeah. So that would be the first
19 bar. So that is sort of the control for that person or that
20 group of people because this is what the DNA damage was in
21 these three groups before the aerial spraying of glyphosate.
22 So you can see they all have increased DNA damage probably
23 because they were using other pesticides, okay, in spraying
24 other pesticides.

25 So then there was this intense spraying of glyphosate from

1 the air and --

2 **Q.** Is it glyphosate, Dr. Weisenburger?

3 **A.** It was Roundup, yeah. Roundup from the air.

4 And then they did the second measurement, which is the
5 middle bar, within five days after the aerial spraying of
6 Roundup, okay. And what you can see in each area is there is
7 an increase in the DNA damage in each of the three groups, and
8 it is statistically significant. So there was -- after --
9 shortly after the Roundup spraying, there was a statistically
10 significant increase in all three of these groups after
11 spraying, okay.

12 **Q.** What is the increase of?

13 **A.** It is an increase in binucleated micronuclei, which are --
14 it is a test to determine genotoxicity. Again, this was done
15 in human lymphocytes, okay.

16 **Q.** What does this tell us if there was an increase five days
17 after spraying Roundup?

18 **A.** It tells you there was an increase in genotoxic damage to
19 the lymphocytes from the people in these three regions that was
20 associated with the aerial spraying of Roundup.

21 And then the third bar, the dark bar, was drawn four
22 months later. And what you can see is that in the first group
23 it stays up. In the second and third groups, it goes back down
24 a little bit. And so I don't know what to make of this. It
25 seems like the genotoxic damage persisted in Putumayo, maybe

1 related to the use of other pesticides. But in the other two
2 regions it went back down closer to what their baseline was.

3 So the meaning of this study is that it is a real-world
4 scenario in which agricultural workers and their spouses were
5 exposed to aerial spraying of Roundup, and you can see a
6 correlation, an increase in genotoxic damage to the human
7 lymphocytes related directly to that spraying of Roundup. So
8 you can see genotoxic effects in experimental situations where
9 you are looking at lymphocytes in a culture dish, and you can
10 see the same effects in real human beings exposed to high
11 amounts of Roundup.

12 **Q.** And have you formed an opinion based on your review of the
13 animal studies, the human lymphocyte studies in the petri dish,
14 and then the real-world exposures to Roundup as to whether
15 Roundup is genotoxic?

16 **A.** Yeah. I think there is no question that Roundup is
17 genotoxic because there are multiple positive studies now in
18 different systems. I'm just showing you a few of the examples,
19 but there are multiple studies. There were at least 11 studies
20 in human lymphocytes that showed genotoxicity. There were
21 studies in other human systems as well where they saw
22 genotoxicity. So I don't think there is any question that
23 Roundup is genotoxic.

24 **Q.** And, Dr. Weisenburger, after your review of the
25 epidemiology -- back to our three legs -- the epidemiology, the

1 animal studies, and the mechanistic data, did you perform what
2 the jury has heard of as a Bradford-Hill analysis?

3 **A.** Yes, I did. So I weighed all of that evidence and used
4 the criteria that were proposed by Bradford-Hill to do a
5 general causation analysis.

6 **Q.** And just briefly, remind the jury, who is Bradford Hill?

7 **A.** So Bradford Hill was an English epidemiologist who was a
8 very influential -- quite a long time ago. He was involved a
9 lot in analysis of smoking causing lung cancer, okay. But he
10 wrote these -- these eight criteria or guidelines to use in
11 trying to determine whether a chemical or other agent can cause
12 non-Hodgkin's lymphoma.

13 **Q.** So let's go through those criteria briefly. And what I
14 want you to focus on, because the jury has heard what the
15 criteria is, I want you to focus on what your analysis of each
16 of the criteria is, what your conclusions were in forming your
17 opinion?

18 **MS. MOORE:** And, Ms. Melen, can I have the ELMO,
19 please. Thank you.

20 **BY MS. MOORE**

21 **Q.** Let's start with the first one, which is the temporal
22 relationship.

23 **A.** All right. Temporal relationship, so what that means --
24 it is very straightforward. What that means is that you have
25 to be exposed to the agent or chemical -- in this case,

1 Roundup -- before you get the disease to conclude that Roundup
2 may have caused the disease. So you just have to be exposed to
3 the agent before you get the disease which, of course, was the
4 case in the case control studies, in the animal studies.

5 **Q.** So do we meet -- in your opinion is the temporal
6 relationship criteria met in this case?

7 **A.** Yes, yes.

8 **Q.** Okay. The next one, strength of association.

9 **A.** So the strength of association means if you look at the
10 odds ratios, do you see -- what is the strength of the
11 association, what is the odds ratio? Is it high and is it
12 statistically significant? And what we saw in the case control
13 studies, in five of the six, the studies were positive with
14 odds ratio greater than 2, and they were statistically
15 significant. So I think the strength of the association is
16 seen there as well as in the meta-analysis, where you also saw
17 increased risks that were statistically significant.

18 **Q.** So should I write "yes" there?

19 **A.** Yes.

20 **Q.** And then the next one is dose response relationship.

21 **A.** So as we talked about, if something -- if there is an
22 agent that causes cancer, usually the more agent you are
23 exposed to, the higher your risk for the cancer. And so we saw
24 in two of the case control studies that the risk of
25 non-Hodgkin's lymphoma when -- was increased when there was

1 more days that the people were exposed, either per year or
2 total days. So that shows a dose response relationship.

3 And we saw the same thing in the Zhang meta-analysis
4 where, when they looked at the high dose, the people who were
5 exposed to higher doses or more exposure, they had a -- they
6 had a higher risk ratio than those that were just ever-never.
7 Then you saw it also in the NAPP study where there was a very
8 nice demonstration of dose response for non-Hodgkin's lymphoma
9 overall and for diffuse large B-cell lymphoma.

10 So dose response is a really important criteria. If you
11 see dose response, it gives you some assurance that that
12 chemical is actually causing the disease.

13 **Q.** Should I write "yes" on that?

14 **A.** Yes.

15 **Q.** Then the next criteria is replication of results. Did you
16 see replication?

17 **A.** Yeah. So what that means is you want to -- you don't want
18 to just evaluate one study, okay. You want to see the same
19 results or similar results in multiple studies. So with regard
20 to the epidemiology studies, we saw it in five of the six
21 studies, okay. In fact, the AHS study was an outlier compared
22 to the other studies. And you want to see it -- the studies
23 done in -- by different researchers in different countries and
24 at different times.

25 So, you know, I think that there is a consistent

1 replication of results in the epidemiology studies and in the
2 animal studies, too, because in many of the -- the tumors were
3 replicated in other studies.

4 Q. And so do we have replication of results?

5 A. Yes.

6 Q. And then biological plausibility.

7 A. So what this means is, you know, based on what we know
8 scientifically, does it all make sense? Is it plausible? Does
9 it sound like it makes sense?

10 And I think what I have told you today is that there is
11 definitely a biologic plausibility. We know that Roundup is
12 genotoxic in lymphocyte cultures, in other tests. We know that
13 it causes genotoxic damage in humans who are highly exposed
14 like the studies in South America. We know that it causes
15 tumors in animals, including non-Hodgkin's lymphoma. And
16 finally, the epidemiology studies point towards non-Hodgkin's
17 lymphoma as well.

18 So you have got a coherence of results that make it very
19 plausible that -- that Roundup causes non-Hodgkin's lymphoma.

20 Q. So is biological plausibility met?

21 A. Yes.

22 Q. The next one is alternative explanations.

23 A. So when you look at epidemiology studies, you always want
24 to think, Well, could there be another explanation for the
25 results, okay. So some of the things that have been proposed

1 are things like selection bias or recall bias or confounding
2 due to the use of other pesticides. And so, you know, we
3 should talk a little bit about that.

4 You know, these studies were designed by very experienced
5 epidemiologists who knew how to do case control studies. So
6 the idea that there would be some selection bias in the
7 cases -- the controls I don't think is plausible, okay?

8 The issue of recall bias I think is important -- do you
9 know what recall bias is? That is when you have cases and
10 controls -- the cases have the disease, and the idea is that
11 because they have the disease, they are asking themselves, How
12 could I have gotten this disease? What could have caused my
13 disease? And so the idea is maybe they would remember the
14 pesticides better than the people who didn't get the disease
15 but who used the pesticides.

16 But, in fact, we looked at that in our study in Nebraska,
17 and if you look at the frequency of the various pesticides that
18 were recalled by the cases and by the controls, there was
19 really no difference. The cases didn't recall more pesticide
20 use or a higher number of pesticides than the controls did. So
21 it is unlikely to be a recall bias. But even a more important
22 argument is recall bias would be a systematic error, okay.

23 So you should see it in the case control studies of other
24 cancers, and we have -- we have lots of case control studies of
25 other cancers with Roundup, other hematological malignancies

1 like Hodgkin's lymphoma, myeloma, leukemia, and other solid
2 tumors. And there was no evidence of increased risk in any of
3 those other studies. The only studies that are positive are
4 the studies of non-Hodgkin's lymphoma.

5 So recall bias couldn't possibly be the explanation for
6 the increased risks in the non-Hodgkin's lymphoma studies
7 because it should have caused the same kind of effect in the
8 other studies and it didn't, okay.

9 Yeah, so I think that's all I want to say about that.

10 **Q.** So were you able to --

11 **A.** Oh, and then confounding. The other one was confounding,
12 I forgot. So that is also an important one to consider, you
13 know. Are we mistaking one pesticide, causing it and blaming
14 the other pesticide?

15 So that's why we do the adjustments like we did in three
16 of the case control studies we talked about, and -- in the NAPP
17 study and in the Zhang meta-analysis. All of those showed that
18 when you make adjustments for the use of other pesticides, the
19 odds ratio sometimes go down, but they don't go to 1. In some
20 of the studies they remain statistically significant like *De*
21 *Roos* and Zhang and NAPP; whereas in other studies they go down
22 and they become nonsignificant. But it is still important to
23 look at all of that data?

24 So it can't be confounding. It can't be recall bias. It
25 can't be selection bias. So we have no other explanation for

1 why the case control studies would be positive. And I have
2 given some reasons why I think the AHS is probably a
3 false-negative, okay, because there are lots of problems and
4 issues with that study.

5 **Q.** So in your opinion is alternative explanations met?

6 **A.** There are no alternative explanations, so I would say yes.

7 **Q.** And then disease specificity.

8 **A.** So this is interesting. So as I mentioned to you, there
9 have been lots of case control studies of other kinds of
10 cancers, and they have all been negative. So the only studies
11 that have been positive are the studies of non-Hodgkin's
12 lymphoma. So I think that exposures to glyphosate and Roundup
13 are very specific for this one cancer, non-Hodgkin's lymphoma.
14 So there is disease specificity here.

15 **Q.** And then the last one is coherence. Have we met
16 coherence?

17 **A.** Coherence just means does all the data fit together, and
18 does it fit with what we know about the use of other
19 pesticides. And, of course, there are other pesticides that
20 cause non-Hodgkin's lymphoma in the same way that Roundup does.
21 They are genotoxic and they cause oxidative stress and DNA
22 damage. And so what we are learning about glyphosate fits with
23 what we know about other pesticides and other chemicals that
24 cause cancer.

25 **Q.** So that is met?

1 **A.** So that's met.

2 **Q.** And, Dr. Weisenburger, I'm just going to write on the top
3 here your name so we will not forget that this was yours, and
4 forgive my handwriting. I'm going to mark this for
5 identification purposes only as Exhibit 948.

6 So taking into consideration all the criteria of
7 Bradford-Hill and your review of the epidemiology, the animal
8 studies, the mechanistic data, what then is your opinion within
9 a reasonable degree of medical certainty now that we have gone
10 through all the literature and your 40 years of experience in
11 looking at the causes of NHL, what is your opinion regarding
12 Roundup?

13 **A.** So my opinion is that I agree with -- that after looking
14 at all this information, my conclusion is that based on all
15 this data, that Roundup can cause non-Hodgkin's lymphoma in
16 people who were exposed to it occupationally or exposed to high
17 doses inadvertently.

18 **Q.** And you said "occupationally." And I want to make sure.
19 So is it only in people who are using Roundup in the workplace?

20 **A.** No. Just like Mr. Hardeman. I mean, he used it
21 frequently and had high exposures, and so I would include him
22 in that group. It may not have been his occupation, but he
23 spent a lot of time doing it.

24 **Q.** Is it fair to say that it's more about the amount of
25 exposure than it is the occupation?

1 **MR. STEKLOFF:** Objection. Leading.

2 **THE COURT:** Sustained.

3 **BY MS. MOORE**

4 **Q.** What is a more important factor in determining whether --
5 what is a more important factor when you are looking at an
6 exposure of an individual?

7 **A.** Well, I think when you look at the exposure of an
8 individual, you want to really understand how much exposure he
9 or she had. So you want to get an idea of what their dose was
10 over time, were they exposed for many years, how often did they
11 use it, did they wear protective clothing, et cetera, to get an
12 idea of what their actual exposure was.

13 **Q.** Dr. Weisenburger, in your opinion does Roundup cause
14 tumors in mammals?

15 **A.** Yes.

16 **Q.** In your opinion does Roundup cause malignant lymphomas in
17 mice?

18 **A.** Yes.

19 **Q.** Does Roundup cause genetic damage in human lymphocytes?

20 **A.** Yes.

21 **Q.** Does Roundup cause oxidative stress in human cells?

22 **A.** Yes.

23 **Q.** Does Roundup cause DNA damage in people who are highly
24 exposed to Roundup?

25 **A.** Yes.

1 Q. And in your opinion does Roundup cause non-Hodgkin's
2 lymphoma in humans at real-world exposures?

3 A. Yes.

4 Q. Now, over your 40 years of studying non-Hodgkin's
5 lymphoma, have you seen an increase or a decrease in the number
6 of cases of non-Hodgkin's lymphoma?

7 A. Well, over the last 50 years, the incidence of
8 non-Hodgkin's lymphoma has increased. In fact, there was a
9 rather remarkable increase between 1970 and about 1990. And as
10 I told you, that was -- that increase was what got people
11 together to think about what could be causing this increase,
12 okay.

13 Q. And in your opinion what was causing that market increase
14 from 1970 to 1990?

15 A. Well, we don't have all the answers. We know that during
16 that period of time is when the AIDS epidemic began, and people
17 with HIV/AIDS have a more -- remarkably increased risk for
18 non-Hodgkin's lymphoma. So we know some of that steep curve
19 was probably due to AIDS.

20 Probably some of it was better diagnosis by pathologists,
21 and probably some of it was due to other causes, which we don't
22 really know what they are. It could be due to increased use of
23 pesticides or solvents or petrochemicals, or it could have been
24 an increase in infections, for example. But we don't really
25 know, other than HIV, what caused that increase -- that

1 increase over time.

2 Q. And I'm going to show you --

3 MS. MOORE: If I could publish on the ELMO, what is
4 called -- it is trends and incidence rates -- let me zoom in on
5 this.

6 BY MS. MOORE

7 Q. Trends and incidence rates. It says 1975 to 2015. Can
8 you explain to the ladies and gentlemen of the jury what this
9 graph shows?

10 A. Well, first of all --

11 MS. MOORE: I'm sorry, hold on. May I have this
12 published?

13 MR. STEKLOFF: No objection, Your Honor.

14 THE COURT: I saw the silent non-objection.

15 MS. MOORE: Sorry.

16 THE WITNESS: So the top curve shows the incidence
17 rate in men, and the lower curve shows the incidence rate in
18 women. And non-Hodgkin's lymphoma, just like many other
19 cancers, is higher in men than women.

20 BY MS. MOORE

21 Q. So this yellow line here, that's --

22 A. That's men.

23 Q. -- men?

24 And this line here?

25 A. Is women, yes.

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1 And you can see -- the far left is 1970, and you can see
2 how --

3 **Q.** I think it -- hold on. Let me zoom in.

4 **A.** -- how it goes up.

5 **Q.** I think -- I don't mean to interrupt you,
6 Dr. Weisenburger. I think it is 1975.

7 **A.** 1975, okay.

8 **Q.** Up to 2015.

9 **A.** Yes.

10 So you can see how it goes up in both men and women, a
11 little more steeply in men than women. Between 19 -- it
12 actually started in 1970, but -- so between 1970 and probably
13 1990 or 1995 you see a more steep curve. And then it begins to
14 kind of level off after that, and it is pretty stable. For men
15 it creeps up a little bit, and for women it is pretty stable.

16 So this early part of the curve where you see it
17 increasing, at what would be kind of a worrisome rate,
18 eventually leveled off and it is not increasing nearly as
19 rapidly as it did during that early period.

20 **Q.** It looks like from 1975 the rate for men, it went from --
21 what is that -- 13 up to 1995 -- is it almost doubled then?

22 **A.** It is almost doubled, yes.

23 **Q.** Okay. And I want to show you a slide, Dr. Weisenburger,
24 and this was shown to the jury during opening by Monsanto's
25 attorney. Okay.

1 And from this slide -- well, first of all, what is this
2 slide? What, if anything, does this slide tell you?

3 **A.** Well, it shows -- it is a drawing on a somewhat different
4 scale, but it shows you in the brown -- brown line, it shows
5 you that the number of cases of NHL, which is on the left side,
6 and the number of cases per hundred thousand persons, which is
7 the incidence, both of those are going up, again, between 1974
8 and about 1990, and then it begins to level off. So this is a
9 combined curve for both men and women, okay.

10 And then what the blue curve shows is that glyphosate was
11 introduced in 1974, and it was -- increasing -- was used
12 increasingly up until about 1995 or 1994 when the use began to
13 go up dramatically due to the use of the genetically modified
14 crops, like corn and soybeans.

15 So what you can see is there has been a marked increase in
16 the use of glyphosate over the 20 or 30 years or so there;
17 whereas the rate of non-Hodgkin's lymphoma has gone up not very
18 much, okay. So, you know, it is sort of misleading to say,
19 Look how the glyphosate has gone up and yet the rate of
20 non-Hodgkin's lymphoma hasn't gone up and that's because the
21 rate of non-Hodgkin's lymphoma is due to a whole variety of
22 things.

23 So think about it. If glyphosate caused an increased
24 number of non-Hodgkin's lymphoma cases, but we learned how to
25 treat the HIV infection, HIV/AIDS, and now we -- they don't get

1 nearly as many lymphomas as they did before, glyphosate could
2 cause an increase in non-Hodgkin's lymphoma; treatment of HIV
3 infection could cause a decrease in non-Hodgkin's lymphoma, and
4 the curve wouldn't change very much at all, right, because you
5 have got some things increasing risk and some things decreasing
6 risk.

7 **Q.** Let me back up and ask you -- because you referenced HIV
8 and AIDS as an example -- is HIV -- someone who is HIV, are
9 they at an increased risk for developing non-Hodgkin's
10 lymphoma?

11 **A.** Yes. Back in the '70s and '80s it was a markedly
12 increased risk. And then when we found drugs to treat it, the
13 incidence of non-Hodgkin's lymphoma went down dramatically in
14 people with HIV/AIDS.

15 **Q.** So on this slide that was shown by Monsanto's attorney in
16 opening, the title of it is "Glyphosate use increased, NHL did
17 not," can we draw any kind of conclusions about Roundup and the
18 correlation between Roundup causing an increase in
19 non-Hodgkin's lymphoma from this graph?

20 **A.** I would say no. I think it is actually a rather
21 misleading graph that can't let you conclude anything.

22 **Q.** It doesn't show a dose response, does it?

23 **A.** No.

24 **MR. STEKLOFF:** Objection. Leading.

25 **THE COURT:** Sustained. That answer will be stricken.

1 **BY MS. MOORE**

2 **Q.** Dr. Weisenburger, does it -- does this graph give you any
3 information as far as the impact of Roundup on the journal of
4 population?

5 **A.** It doesn't.

6 **Q.** Let's switch gears then, and I want to focus on
7 Mr. Hardeman. Have you had an opportunity to review documents
8 and form an opinion as to whether Roundup was a substantial
9 factor in causing Mr. Hardeman's diagnosis of non-Hodgkin's
10 lymphoma?

11 **A.** Yes. So I reviewed the medical records. I reviewed the
12 doctors' depositions. I reviewed the deposition of
13 Mr. Hardeman and his wife. I reviewed the pesticide fact sheet
14 for Mr. Hardeman. And I actually talked to Mr. Hardeman on the
15 phone for about an hour to get -- to answer my questions and to
16 get a better idea of what happened to him and what kind of
17 exposures he really had to Roundup.

18 **Q.** And based upon that review of depositions and the medical
19 records of Mr. Hardeman, your interview with Mr. Hardeman, have
20 you -- what is your opinion as to whether Roundup was a
21 substantial factor in causing Mr. Hardeman's non-Hodgkin's
22 lymphoma?

23 **A.** Well, I think after talking to Mr. Hardeman, he had really
24 quite high exposures to Roundup over many years. You know, he
25 used Roundup for 26 years before getting non-Hodgkin's

1 lymphoma, and 23 of those years he was using it for the -- for
2 the six months of spring and summer, he was using it twice a
3 month, for two to four hours every time, and spraying up to
4 20 gallons at one -- at one time in one day. And he did
5 that -- and then -- then during the winter months, he would
6 spray once a month using, you know, lesser amounts.

7 **MR. STEKLOFF:** Your Honor, I object to hearsay.

8 **THE COURT:** Overruled.

9 **THE WITNESS:** And so, you know, I did some crude
10 calculations of how many days he was -- how many days during
11 those 26 years he used Roundup based -- I used information from
12 his deposition. So he was exposed to Roundup over 300 times
13 during those 26 years. And he used -- my calculation -- he
14 used around 5,900 gallons of Roundup in those 26 years.

15 So, you know, he had high exposure to Roundup. He didn't
16 wear any protective equipment, didn't wear gloves, didn't wear
17 a mask. He wore short-sleeved shirts. He didn't take any real
18 precautions at all when he sprayed it, so he got it on his
19 hands. He got it on his arms. He got it on his face. And
20 when he was mixing it, he would sometimes even get it on his
21 hands while he was mixing it.

22 So, you know, when you add it all together, he had -- he
23 was -- he had a high exposure to Roundup over many years.

24 **BY MS. MOORE**

25 **Q.** And based upon that -- your determination that

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1 Mr. Hardeman fit into the category of high exposure, what was
2 your conclusion as to the cause of his non-Hodgkin's lymphoma?

3 **A.** Well, my conclusion after weighing all the evidence -- and
4 we will talk about the differential diagnosis or differential
5 etiology -- but after going through all of the -- this
6 methodology, I came to the conclusion that Roundup was the
7 substantial contributing cause for him with regard to his
8 development of non-Hodgkin's lymphoma.

9 **Q.** But, Dr. Weisenburger, the jury heard testimony last week
10 from Mr. Hardeman's treating physicians, Dr. Ye, his
11 oncologist. And you have reviewed that deposition?

12 **A.** Yes.

13 **Q.** Okay. And the jury heard that Dr. Ye had -- did not form
14 an opinion as to the cause of Mr. Hardeman's non-Hodgkin's
15 lymphoma. Did that surprise you when you read that?

16 **A.** No, because once the patient --

17 **MR. STEKLOFF:** Your Honor.

18 **THE COURT:** Overruled.

19 **THE WITNESS:** So because once the patient has the
20 disease, unless the cause is obvious, the oncologist is more
21 concerned with treating the disease than he is trying to figure
22 out what caused the disease. So it's not uncommon for
23 physicians not to ask questions or to try to figure out what
24 caused the disease unless it's obvious.

25 \\\

1 **BY MS. MOORE**

2 **Q.** Now, you mentioned a differential. And so what is a
3 differential?

4 **A.** So the methodology for doing this is the same methodology
5 we use when we are diagnosing and treating patients in the
6 hospital or the clinic. If the patient has a disease, you want
7 to think about what are all of the potential causes and risk
8 factors for that disease, and you want to go through the list
9 and try to eliminate the things that are not applicable to the
10 patient and to -- and then to just evaluate the things that may
11 be causes and hopefully come to a conclusion that one or
12 sometimes more than one thing are the cause.

13 So you want to go through the list of all the known causes
14 and try to eliminate the ones that don't apply to the case, and
15 then what you are left with are potential causes. Then you
16 have to weigh them and see, well, which is the most important
17 of these potential causes, or you can rule some of them out.

18 **Q.** Did you go through that process with respect to
19 Mr. Hardeman?

20 **A.** Yes, I did.

21 **MS. MOORE:** And, Your Honor, with your permission if I
22 can ask the witness to come off the stand and use the blowup.

23 **THE COURT:** Sure.

24 **BY MS. MOORE**

25 **Q.** Dr. Weisenburger, if you want to come this way.

1 Did you put together -- did you put together a list that
2 you used in your differential?

3 **A.** Yes, I did.

4 **Q.** And is this your list?

5 **A.** Yes.

6 **Q.** And on the left-hand column there -- if you need a pen, I
7 have one.

8 In the left-hand column, you have Known risk factors for
9 NHL. Can you explain to the jury how you came up with what is
10 in the left-hand column?

11 **A.** Well, these are all of the known and published risk
12 factors for non-Hodgkin's lymphoma. So I made this list,
13 and -- with Mr. Hardeman, and I went through them by reading
14 his medical records and reading his doctors' depositions and
15 talking to him. I went through this list and tried to rule in
16 or rule out some of these causes, okay.

17 **MS. MOORE:** Before you do that, may I publish this to
18 the jury, too, Your Honor, on the screen?

19 **THE COURT:** Any objection?

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

21 **MS. MOORE:** Thank you.

22 **BY MS. MOORE**

23 **Q.** Go ahead.

24 **A.** Okay. So age is a known risk factor for non-Hodgkin's
25 lymphoma; that is, as you get older, your risk for

1 non-Hodgkin's lymphoma increases. It is true of many cancers,
2 okay. There are some cancers that are more common in children,
3 but for most cancers the risk increases as people get older.

4 And sex, I showed you that non-Hodgkin's lymphoma is more
5 common in men than women; probably 20 to 30 percent more
6 common. And we don't really understand why that is.

7 And then race also. Caucasians have a higher risk than
8 blacks or Hispanics, and they have a higher risk than Asians.
9 So, you know, Mr. Hardeman was 65, I think, when he got his
10 non-Hodgkin's lymphoma. He was a male. And he was Caucasian.
11 So he fit into this category, but I don't consider any of these
12 causative risk factors. Did his age cause his non-Hodgkin's
13 lymphoma? That doesn't really make sense.

14 **Q.** When you say "causative risk factor," what does that mean?

15 **A.** Well, it means that it is a factor that actually caused
16 the lymphoma, okay. So I don't believe that age causes
17 lymphoma or sex causes lymphoma or race causes lymphoma. Those
18 are risk factors, but they are not causative risk factors.

19 **Q.** So in other words with respect to age, simply because you
20 are at an older age, does that mean you are going to get --
21 does that mean age itself is going to cause you to get
22 non-Hodgkin's lymphoma?

23 **A.** No.

24 **Q.** The same thing with sex and race, just because you are a
25 man that doesn't mean you are going to -- cause you to get

1 non-Hodgkin's lymphoma?

2 **A.** No. We can't say Mr. Hardeman got non-Hodgkin's lymphoma
3 because he is a man. That doesn't make sense, right. So they
4 are risk factors. They tell you something about the disease,
5 but they don't really tell you what caused the disease. So I
6 eliminated these three as causative risk factors. And the
7 other thing is you have no control over that, right. You have
8 no control over that.

9 So the next thing I thought about was family history of
10 malignancies because we do know if you have a family history of
11 a hematologic cancer, like non-Hodgkin's lymphoma or Hodgkin's
12 lymphoma or leukemia or myeloma, we know that those people have
13 a two-time increased risk for non-Hodgkin's lymphoma, okay.

14 But in Mr. Hardeman's case he didn't have any family
15 history of any of these cancers. So he probably didn't have
16 any genetic predisposition to getting this.

17 **Q.** Let me -- before you do that, I want to ask you: You have
18 "hematologic malignancy." What is that?

19 **A.** Well, those are the family of diseases that we are talking
20 about: Non-Hodgkin's lymphoma, Hodgkin's lymphoma, myeloma,
21 leukemia, things that hematologists are diagnosing. And it is
22 a family history. It is usually first-degree relatives. So it
23 is father, mother, brother, sister, and children. Those are
24 the things that you look at to get this increased risk. So he
25 didn't have any family history, okay.

1 Q. So you ruled that out?

2 A. So I ruled that out.

3 Q. Okay.

4 A. Pesticide use --

5 Q. Before you go there, I just want to ask you -- because the
6 jury heard some testimony about Mr. Hardeman having a couple of
7 sun spots, basal cell carcinoma on his leg, and melanoma last
8 year on his shoulder -- I don't see that you have skin cancer
9 up here. Can you explain to the jury why you didn't put that
10 on here?

11 A. Well, because it is not really a causative risk factor.
12 And Mr. Hardeman had one basal cell carcinoma. It is a very
13 common tumor, okay. Melanomas are also fairly common.

14 So -- and neither basal cell carcinoma or melanomas are
15 causes of non-Hodgkin's lymphoma. It is -- they are usually
16 due to the sun damage, okay, ultraviolet light damage to the
17 skin. And we know that people who get a lot of sun exposure
18 have a lower risk of non-Hodgkin's lymphoma, not a higher risk.
19 So I don't see that -- how that fits in at all. It is -- it
20 doesn't -- I didn't -- I didn't add it into my differential,
21 no.

22 Q. Go ahead with the next thing you put on your risk factor
23 list.

24 A. So the next thing was pesticide use. And, of course, as
25 we talked about, he had high exposures to Roundup for many

1 years. I asked about other pesticides because we know there
2 are other pesticides that cause non-Hodgkin's lymphoma, and he
3 didn't use any of those other pesticides. In fact, the only
4 thing he ever used was he would occasionally, maybe once a
5 year, spray for ants in the house, okay, buying something off
6 the shelf.

7 So the only -- the only pesticide that he used in any
8 amount was Roundup, okay. So I think we have to put Roundup
9 over here -- excuse my writing -- over there --

10 **Q.** You are a doctor.

11 **A.** -- as a possible risk factor for his non-Hodgkin's
12 lymphoma, okay.

13 **Q.** Let me stop you there because you moved Roundup over to
14 this column right now, does that mean you have already
15 determined that it is the cause?

16 **A.** No. We have to go through these other -- the rest of the
17 list here to see if there were other important risk factors.

18 **Q.** Okay. All right. The next one?

19 **A.** So we know that obesity is sort of a weak risk factor for
20 non-Hodgkin's lymphoma. Mr. Hardeman wasn't obese, but he is a
21 bit overweight, okay. His average weight was about 190 pounds,
22 and I think his height was only -- I can't even remember. But
23 the calculation of his body mass index was in the overweight
24 category, not in the obese category. But even overweight
25 people can have an increased risk of non-Hodgkin's lymphoma.

1 But it is a small risk. It is not a big risk.

2 So, you know, we probably have to include obesity. His
3 risk would have been maybe 1.3, maybe a 30 percent, increased
4 risk. We will put obesity there, really meaning overweight.

5 **Q.** Okay. Did you want to write "overweight"?

6 **A.** No. It's okay.

7 **Q.** Then the next one?

8 **A.** The next one is viral infections. And there are a number
9 of viral infections that actually cause non-Hodgkin's lymphoma.
10 One of the best known ones is Epstein-Barr virus. It is the
11 same one that causes infectious mononucleosis, but it also
12 causes non-Hodgkin's lymphoma in some people, okay. But he
13 didn't have any evidence of that in his biopsy or even in his
14 blood testing.

15 And then there is some other less common viruses that also
16 cause non-Hodgkin's lymphoma, but there are two viral
17 infections that do cause non-Hodgkin's lymphoma. One is
18 hepatitis C virus infection and one is hepatitis B virus
19 infection; and he did have a history of both of those viral
20 infections, okay. So we have to put both of those over here in
21 the column and evaluate whether they could be the cause of his
22 non-Hodgkin's lymphoma, okay. So we will say hepatitis C and
23 hepatitis B.

24 **Q.** Okay. And then you want to finish off the list then?

25 **A.** And then bacterial infections. There are some bacterial

1 infections that can cause non-Hodgkin's lymphoma. The most
2 common one is non-Hodgkin's lymphoma of the stomach related to
3 a bacteria, but he didn't have any evidence of bacterial
4 infections that would fit into that category. So I excluded
5 that one.

6 People who have immune deficiency have an increased risk
7 of non-Hodgkin's lymphoma. So we were talking about people
8 with HIV/AIDS, for example. That is a good example. They have
9 a very dramatic knockout of their immune system, and then they
10 get infections and they can get lymphoma, okay. And people who
11 have organ transplants, for example, they are put on drugs that
12 could decrease their immunity and then they can get
13 non-Hodgkin's lymphoma. But he didn't have any history of any
14 of that kind of severe immunodeficiency. So I crossed that one
15 out.

16 And then immunosuppression. So if you give certain drugs
17 or chemotherapy, you can knock the immune system down and
18 increase the risk for non-Hodgkin's lymphoma; but he really
19 didn't have any history of immunosuppression by drugs or other
20 things, so I crossed that one out.

21 Another thing that can cause non-Hodgkin's lymphoma is
22 autoimmune diseases. So diseases like rheumatoid arthritis,
23 for example, diseases like systemic Lupus or celiac disease,
24 et cetera. There are a variety of autoimmune diseases where
25 the immune system is altered and the body reacts against

1 itself, and people with those diseases can have an increased
2 risk for non-Hodgkin's lymphoma.

3 **Q.** And based on your review of Mr. Hardeman's medical
4 records, did he have any type of autoimmune disease?

5 **A.** He didn't so I crossed that one out.

6 And then chronic inflammation is a known cause. So people
7 who have some kind -- the most recent one actually that I think
8 is the best example is we now know that women who have breast
9 implants have an increased risk for non-Hodgkin's lymphoma.
10 And why is that? Well, they have chronic inflammation
11 associated with that breast implant, and a small percentage of
12 women actually get non-Hodgkin's lymphoma that comes out of
13 that chronic inflammation.

14 And there's some other examples that are -- you know, that
15 I could give you, but he didn't have any evidence that he had
16 some kind of chronic inflammation going on in his body so I
17 crossed that one out.

18 And then the last one is solvent use. We talked a little
19 bit about that earlier. So people who use solvents,
20 particularly people who use a lot of solvents in their work,
21 like painters or machinists, people who are using solvents to
22 clean their brushes or clean their equipment or their machines
23 have an increased risk of non-Hodgkin's lymphoma; but he didn't
24 have any history of solvent use, and so we crossed out that one
25 as well.

1 So what we're left with is Roundup and obesity and
2 hepatitis C and possibly hepatitis B as the possible causes for
3 his non-Hodgkin's lymphoma.

4 **Q.** So once you've eliminated these other risk factors and now
5 you've looked at, for Mr. Hardeman in particular, these four,
6 what's the next step in your process?

7 **A.** So the next step is to take a hard look at each of these
8 and decide whether they're strong risk factors or weak risk
9 factors or maybe not even risk factors at all depending on the
10 history of what happens with the viral infections.

11 **Q.** Okay. So which one do you want to start with, then, and
12 explain to the jury what your process was next?

13 **A.** Well, I think we should start with the hepatitis B and
14 hepatitis C and talk about those because those are -- those
15 could also be substantial risk factors. Okay?

16 **Q.** And in Mr. Hardeman's case, let's start with hepatitis C,
17 when you were looking to see whether it is a substantial
18 factor, what did you look at?

19 **A.** So I had to go back and do a lot of research on
20 hepatitis B and hepatitis C to really understand who's at risk
21 and what is the risk -- okay? -- and what I found was that
22 people who have chronic active hepatitis, they have the virus
23 causing the disease in their liver, those are the people who
24 are at risk for developing non-Hodgkin's lymphoma. So in some
25 ways they have chronic inflammation that's causing liver damage

1 and eventually cirrhosis. Okay?

2 And when Mr. Hardeman was found to have hepatitis, first
3 of all, he was found to have cirrhosis; and then they looked to
4 see what could cause the cirrhosis, and they said, "Oh, my
5 gosh. He's got hepatitis C -- hepatitis." So you have to look
6 at the whole story here and try to figure it out.

7 But what the literature says is that you have to have
8 chronic active hepatitis to be at risk for non-Hodgkin's
9 lymphoma.

10 **Q.** So did Mr. Hardeman at one point in time have chronic
11 active hepatitis C?

12 **A.** Yes, he must have because he had cirrhosis in 2005 when
13 the cirrhosis was discovered; and then they did the test for
14 hepatitis B and hepatitis C, and they found that he had an
15 active infection of hepatitis C. He had lots of viral
16 particles in his blood, and they tested him also for
17 hepatitis B, and he was found to be immune to hepatitis B.

18 So he didn't have an active infection of hepatitis B.
19 Because of his prior infection, he became immune to
20 hepatitis B, and so he was immune to that virus.

21 **Q.** Well, that's what I wanted to ask you. You say he was
22 immune to hepatitis B and you've got it over in this column.
23 What does immune to hepatitis B mean?

24 **A.** Well, it means that he developed immunity. So, for
25 example, if you get a vaccine to -- you know, when you're

1 little, you got vaccinated; right? And after they vaccinated
2 you with, let's say, the polio -- attenuated polio virus, you
3 developed immune response to that polio virus and it protects
4 you from getting polio; right?

5 Well, the same thing happens with hepatitis B. If you get
6 hepatitis B, you might be sick for a while but then your body
7 develops immunity, gets rid of the hepatitis B, and you have an
8 immunity and you're protected from hepatitis B in the future.
9 Okay?

10 **Q.** Is that what happened with Mr. Hardeman?

11 **A.** And that's what happened with Mr. Hardeman. He didn't
12 have any evidence of hepatitis through the hepatitis B. He was
13 immune to hepatitis B virus.

14 **Q.** And then -- and when do you know that he was -- at what
15 point in time, based on your review of the records, do we know
16 that he was immune to hepatitis B?

17 **A.** Well, they did the test in 2005. At the time they were
18 working him up for his cirrhosis, they did tests for
19 hepatitis B and hepatitis C; and they thought because he had
20 active hepatitis through the hepatitis C, that that was the
21 main cause of his cirrhosis.

22 **Q.** All right. So you keep mentioning "active." Can you
23 explain to the jury a little bit more? What does it mean to
24 have active hepatitis?

25 **A.** So active hepatitis means you have inflammation in the

1 liver. The virus is infecting the liver cells. It's
2 destroying the liver cells and eventually the liver gets
3 scarred from all the inflammation and death of liver cells and
4 you end up with cirrhosis, just scarring of the liver. Okay?
5 And that's what he had in 2005, not a severe case but, you
6 know, it definitely was there.

7 **Q.** Is liver damage an expected outcome with someone who has
8 hepatitis C?

9 **A.** Yes.

10 **Q.** And based on your review of the literature,
11 Dr. Weisenburger, people who have active hepatitis C, what
12 percentage of those people go on to develop non-Hodgkin's
13 lymphoma?

14 **A.** It's actually quite a low percentage. It's less than
15 1 percent. Probably less than a half a percent. The data is
16 not very -- there's not very good data on exactly what
17 percentage it is but it's low. It's much lower than liver
18 cancer as a result of hepatitis and cirrhosis. It's probably
19 10 times less common than liver cancer.

20 **Q.** So what does that mean for Mr. Hardeman? What would you
21 expect -- given that he had active hepatitis C, what would you
22 expect that to develop in to --

23 **A.** Well, I think --

24 **Q.** -- based on those stats?

25 **A.** -- I think, you know, when we look back at his history, he

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1 thought he probably contracted the hepatitis C and hepatitis B
2 sometime in the 1960s. So he probably had the infection to
3 hepatitis C since that time. So he had it probably for almost
4 40 years causing chronic hepatitis. Not making him very sick
5 because he never really went to the doctor saying "I'm sick";
6 but by the time he did get to the doctor in 2005, he was found
7 to have scarring of the liver and cirrhosis and some hepatitis
8 as well. There was evidence of liver damage from blood tests.

9 So, you know, he probably had the hepatitis C for about 40
10 years -- 39, 40 years. He probably had the hepatitis B at
11 sometime during that same time frame but became immune to it
12 and, you know, kept that infection in check.

13 Q. So in the time period that you're talking about the active
14 hepatitis C, what time period is that?

15 A. For Mr. Hardeman?

16 Q. For Mr. Hardeman.

17 A. It was about 39 years.

18 Q. So from what year to what year?

19 A. Probably 1996 to 2005, something like that. So 39, 40
20 years.

21 Q. Well, you said "1996," so --

22 A. Let's see...

23 Q. Now I've got you in a math problem here.

24 A. 1966. 1966 to 2005. I'm sorry.

25 Q. That's okay. That's okay.

1 And so during the time that Mr. Hardeman likely had active
2 hepatitis C for those 39 years or so, did he get non-Hodgkin's
3 lymphoma?

4 **A.** No. So that's when he would have been at risk for getting
5 non-Hodgkin's lymphoma, and he didn't get non-Hodgkin's
6 lymphoma and luckily he didn't get liver cancer either --
7 okay? -- which are the two cancers he would have been at high
8 risk for.

9 **Q.** All right. So based on your review, then, what happened
10 after he found out in 2005 that he had hepatitis C?

11 **A.** Well, by that time we have had fairly good treatments for
12 hepatitis C so he was given a course of antiviral therapy,
13 which included two drugs -- one called Interferon, another
14 called Ribavirin -- and he was given a course of that treatment
15 over a period of about 46 weeks. Okay?

16 And what happened to him was that within 12 weeks, his
17 viral DNA in the blood, which is the way they made that, his
18 viral DNA in the blood went away -- okay? -- and he no longer
19 had evidence of the viral infection, but they continued to
20 treat for up to 46 weeks because you know that you have to
21 continue to treat for a long time afterwards to get rid of
22 hepatitis C so that's what they did.

23 **Q.** Okay.

24 **A.** And during that period of treatment, once his viral
25 testing in the blood became negative at 12 weeks, it stayed

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1 negative through the whole treatment right out to the end of
2 the treatment.

3 Q. And I think we have -- and, Dr. Weisenburger, is that
4 based on your review of his test results?

5 A. Yes.

6 Q. Okay. And I think we have a summary of his test results,
7 and I'm going to slide behind you and put that up on the board.

8 A. Take this off?

9 Q. It's fine. We're going to leave it there.

10 And this is a summary of the test results.

11 MS. MOORE: May we publish that to the jury,
12 Your Honor, please?

13 THE COURT: Sure.

14 THE WITNESS: So this just shows you the chronology of
15 his hepatitis C. So in 2005, January 2005, the test was found
16 to be positive. Okay? And so at that point he was treated
17 with this antiviral therapy to try to cure him of the
18 hepatitis C. Okay? And, in fact, within just 12 weeks, his
19 viral RNA in the blood became negative. Okay?

20 And then he was treated for, I think, 46 weeks and he
21 stayed negative through that time. At the end of the
22 treatment, he was still negative. And then they monitored him
23 for over five years, and he stayed negative for the whole time.
24 Okay?

25 \\

1 **BY MS. MOORE:**

2 **Q.** What's the significance of once he started antiviral
3 treatment for hepatitis C that he stayed negative during
4 treatment and then after treatment?

5 **A.** Well, you would consider him to have a rapid response
6 because he quickly became negative, and then you would consider
7 him to have what we call the sustained virologic response
8 because he stayed negative for six months and then for five or
9 six years thereafter. Okay?

10 So most people who treat hepatitis C would say that he was
11 cured of his hepatitis -- okay? -- in terms -- because his
12 liver function tests became normal and the virus disappeared
13 from the blood. Okay?

14 **Q.** And are these factors that were important for you in
15 determining whether to rule in or rule out hepatitis C as a
16 cause?

17 **A.** Well, they were because I realized that when he got his
18 non-Hodgkin's lymphoma, he had already been free of the virus
19 for nine years. Okay? He'd been cured of the virus for nine
20 years.

21 And so part of the research I did was to say: Well, okay.
22 We know that people who have active hepatitis C have an
23 increased risk for non-Hodgkin's lymphoma, probably two- to
24 threefold increased risk; but what about people who are treated
25 with antivirals, have a sustained virologic response, and are

1 cured of the virus? Do those people -- are they still at
2 increased risk for hepatitis C causing non-Hodgkin's lymphoma?

3 So that was the question I had to answer.

4 **Q.** Okay. And so I want to make sure. So the first factor
5 you said was a rapid response; is that right?

6 **A.** Right. He had a rapid response in that he cleared the
7 virus within the first 12 weeks.

8 **Q.** So I'm going to say within 12 weeks; is that right?

9 **A.** Yes.

10 **Q.** Okay. And then you said he was cured of hepatitis C?

11 **A.** Yes. So after you stay negative for six months during
12 the -- six months after the therapy is completed, then you're
13 considered cured, and he did meet that criteria. So he was
14 considered by his doctors at Kaiser to be cured of the
15 hepatitis C, and they stopped -- then they stopped the
16 antiviral treatment after the first 46 weeks.

17 **THE COURT:** Can I interrupt for a minute? I think now
18 would be a good time for a five-minute break.

19 **MS. MOORE:** Thank you, Your Honor.

20 **THE COURT:** Why don't we go ahead and take a break.
21 Be back at 2:00 o'clock.

22 **THE CLERK:** All rise.

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

24 **THE CLERK:** Please be seated.

25 **THE COURT:** All right. Back in a few minutes.

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

2 (Recess taken at 1:56 p.m.)

3 (Proceedings resumed at 2:01 p.m.)

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

5 **THE COURT:** Okay. You can bring the jury back in.

6 **MS. MOORE:** Thank you, Your Honor.

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

8 **THE COURT:** Okay. You can resume.

9 **MS. MOORE:** And, Your Honor, before I get right back
10 into the questioning, a couple of housekeeping matters that
11 Ms. Melen asked about.

12 And that is the summary of Mr. Hardeman's test results for
13 hepatitis C viral load that we have up on the easel is
14 Exhibit 940, and we would move to enter that into evidence as
15 Exhibit 940.

16 **THE COURT:** Any objection?

17 **MR. STEKLOFF:** No objection, Your Honor.

18 **THE COURT:** It will be admitted.

19 (Trial Exhibit 940 received in evidence)

20 **MS. MOORE:** Thank you, Your Honor.

21 And then I also had shown this trend in incidence rates,
22 and I did not mark it for identification, and we would mark it
23 as identification 949, "Trends and Incidence Rates 1975 to
24 2015."

25 I apologize for that, Your Honor.

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1 (Trial Exhibit 949 marked for identification)

2 **BY MS. MOORE:**

3 **Q.** Now, Dr. Weisenburger, let's go back to where we were, and
4 you were on this summary that we marked as Exhibit 940, and we
5 were getting back to the point where you said he had been
6 considered cured of hep C. And then what was the next step in
7 your process?

8 **A.** Right. So he was cured of hep C and he remained viral --
9 virus negative for nine years up until his diagnosis of
10 non-Hodgkin's lymphoma in early 2015. Okay?

11 And --

12 **Q.** Well, hold on. You said "up until." Was he still
13 considered cured when he was diagnosed with non-Hodgkin's
14 lymphoma?

15 **A.** Yes. So he was tested at that time -- you can see the
16 test right here -- and he was still negative. Okay? So at the
17 time he was diagnosed with non-Hodgkin's lymphoma, his viral
18 test in the blood was still negative so he was still considered
19 to be cured. Okay?

20 And I don't know if I told you this, but he was found to
21 be immune at the time of the diagnosis for hepatitis C. So he
22 wasn't thought to have an active infection.

23 **Q.** Let me interrupt. You said hepatitis C.

24 **A.** Hepatitis B. Hepatitis B.

25 So they retested him for hepatitis B here. He continued

1 to be immune. Okay? There was no evidence of a viral
2 infection due to hepatitis B or hepatitis C at the time of his
3 non-Hodgkin's lymphoma.

4 **Q.** So can I say "immune hepatitis B" here?

5 **A.** Right.

6 **Q.** If I can spell it.

7 Okay. Is that right?

8 **A.** So one of the things that the clinicians worry about is
9 when you treat people for non-Hodgkin's lymphoma and they have
10 had a past infection for hepatitis C or hepatitis B, they
11 sometimes have small amounts of that virus still in their
12 body -- okay? -- but the immune system is holding it in check.

13 So he could have some virus in the liver. He could maybe
14 even have some virus in B cells. Okay? But it's at a very low
15 level, what we call latent -- present in the latent state, and
16 it's held in check by the immune system.

17 But because they knew he had this history of hepatitis C
18 and hepatitis B, they decided they should monitor him because
19 when you give the chemotherapy, you cause immunosuppression and
20 the viruses can reactivate and cause hepatitis. Okay? And
21 they don't want that to happen when he's getting his
22 chemotherapy. Okay?

23 **Q.** So let me stop you right there. What is
24 immunosuppression?

25 **A.** So immunosuppression is what we talked about earlier where

1 we give drugs that knock the immune system out and increase the
2 risk for infection. And because he had these two viral
3 infections before and he might have some of that virus still in
4 his body at a very low level, they decided, first of all, to
5 give him antiviral drug for his hepatitis B -- okay? -- because
6 there was a drug they could give him to make sure that if he
7 had some hepatitis B still in his system, that it wouldn't
8 reactivate during the chemotherapy.

9 And for hepatitis C there isn't a drug that you can give
10 to prevent it so what they did is they just carefully monitored
11 it every few weeks to make sure that he didn't reactivate his
12 hepatitis C because if he did, they would have to give him the
13 antiviral therapy like they did back in 2005.

14 **Q.** So right here where you have this shaded, is this the test
15 they did to check him to see if the hepatitis C or B came back
16 while undergoing chemo?

17 **A.** While undergoing chemo and -- yeah, while undergoing
18 chemo. So he went under chemo for about six or eight -- about
19 six weeks. So during that period of time, they -- six months.
20 So during that period of time, they were checking him every few
21 weeks for his hepatitis C infection. And they were also doing
22 a test for his hepatitis B infection to make sure he didn't
23 reactivate that; but because he was on antivirals for that, it
24 was pretty unlikely that that would happen. But they monitored
25 both of those infections, those possible infections.

1 Q. So with respect to the hepatitis C, there was no drug they
2 could give him to make sure that didn't come back; right?

3 A. Right.

4 Q. Okay. So during the time he was undergoing chemotherapy
5 when his system was weak, did the hepatitis C come back?

6 A. It didn't.

7 Q. And what is the significance of that?

8 A. Well, what that would tell you is that he didn't have it
9 in his system at all or his immunity didn't get knocked down so
10 low that the virus would come back. So we don't really know,
11 but what we do know is that he didn't develop an active
12 hepatitis infection even when he was getting the
13 immunosuppressive drugs for the lymphoma. Okay?

14 Q. All right.

15 A. And the same is true for the hepatitis B infection as
16 well.

17 Q. Let me take this down and let's go back.

18 So going back to your process to eliminate -- or, I'm
19 sorry -- going back to your process to consider hepatitis B
20 and C with respect to the non-Hodgkin's lymphoma, what did you
21 do next?

22 A. Well, so I did some research and I read about hepatitis B
23 and hepatitis C, and the main question was: If you -- if you
24 have a cure -- if you're cured of your hepatitis C, do you
25 still have an increased risk for non-Hodgkin's lymphoma?

1 And, in fact, the answer is no, which makes sense because
2 the virus is what's causing the infection. And so if you get
3 rid of the virus and you no longer have an active infection,
4 then you wouldn't get the lymphoma from the virus because the
5 virus is gone. Okay?

6 And the same is true -- and the same is true for
7 hepatitis B. So I wanted to know: Well, are people who are
8 immune to hepatitis B, are they still at increased risk for
9 non-Hodgkin's lymphoma? And the answer for that was no too.

10 So if you're -- if you're cured of hepatitis C or if
11 you're immune to hepatitis B, you no longer have an increased
12 risk for non-Hodgkin's lymphoma -- okay? -- because you're
13 cured of the virus. And even if there's small amounts of
14 either B or C in your body, they're in a latent state held in
15 check by the immune system; and the literature shows that when
16 that occurs, there's no increased risk for non-Hodgkin's
17 lymphoma with either hepatitis B or hepatitis C.

18 And I'm going to show you some -- some -- actually some --

19 **Q.** Do you want to do that now?

20 **A.** -- some data from the literature to convince you of that.
21 Okay?

22 **Q.** Okay. All right. Go ahead. You can go back to the
23 stand. Thank you.

24 Let's go to the Gianelli graph. It's actually -- don't
25 kill me, Dr. Weisenburger, but I think you're going to have to

1 come off the stand because this is a blow-up. I'm sorry.

2 **A.** Oh.

3 **Q.** You're going to get your exercise.

4 Someone's got my stuff out of order. Oh, here we go.

5 **MS. MOORE:** Counsel, this is Gianelli.

6 **MR. STEKLOFF:** No objection.

7 **MS. MOORE:** And we'll mark this for identification
8 purposes as 938.

9 (Trial Exhibit 938 marked for identification)

10 **BY MS. MOORE:**

11 **Q.** And, Dr. Weisenburger, can you explain to the jury what
12 this chart is that we're looking at?

13 And if I could, Your Honor, may I publish this to the
14 jury?

15 **THE COURT:** Sure.

16 **THE WITNESS:** So this is from one of the studies of
17 hepatitis C and it shows what happens when patients have a
18 complete virologic response like Mr. Hardeman had to the
19 antiviral therapy for hepatitis C. Okay?

20 So here he has the infection, and -- I'm trying to see
21 what -- the black box is one of the liver function tests called
22 the ALC; and, of course, his liver function tests are
23 remarkably abnormal and elevated at the time of the diagnosis
24 of hepatitis. Now, Mr. Hardeman's weren't quite this high, but
25 his tests were also elevated, his liver function tests.

1 And this shows the viral load. Okay? This diamond again
2 is very high, about here 5 million copies of viral RNA in
3 his -- per mil. Okay?

4 **BY MS. MOORE:**

5 **Q.** And what does viral load represent?

6 **A.** So viral load just tells you how much virus you have
7 circulating in your blood. Okay? And Mr. Hardeman had about,
8 I think, 2 million copies but still a significant number.

9 So --

10 **Q.** And this graph here, the Gianelli graph, this is from an
11 actual patient?

12 **A.** This is an actual patient that was treated with antiviral
13 therapy in that paper. Okay?

14 **Q.** Go ahead.

15 **A.** Yeah. So it shows you. So you can see when you give the
16 antiviral therapy, what happens. You get a dramatic decrease
17 in the liver enzymes and they come back to normal. Okay? And
18 they stay normal over here (indicating) over a two-month --
19 over a two-year period.

20 And the same thing happens. There's a dramatic decrease
21 in the viral RNA in the blood and it goes down to zero --
22 okay? -- which is exactly what happened to Mr. Hardeman, and
23 then it stayed negative over the 24 months.

24 So Mr. Hardeman was very much like this patient who was
25 treated in the paper. And here you see the viral RNA test.

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1 It's initially positive and then it becomes negative over time.

2 Okay?

3 So this is what --

4 Q. And is that --

5 A. -- this is what would have happened to Mr. Hardeman.

6 Okay?

7 Q. And so if we go back to our Exhibit 940, Dr. Weisenburger,
8 here --

9 A. Here's the positive (indicating) and then --

10 Q. So that's before the treatment?

11 A. Yep. And then here are all the negatives (indicating).

12 Q. And that's after the treatment?

13 A. After the treatment, yes.

14 And so this is the kind of response that Mr. Hardeman had
15 to the treatment for his hepatitis C infection.

16 Q. So what does this tell us?

17 A. Well, what this tells us is that once you treat these
18 patients with antiviral therapy and they have a sustained
19 virologic response, their liver disease stabilizes and it
20 doesn't get worse, and there's little or no continued damage to
21 the liver and the virus is completely gone from the blood and
22 is no longer causing disease. Okay?

23 And, in fact, what I'm going to show you is once you get
24 to this state of sustained virologic response, you no longer
25 have an increased risk for non-Hodgkin's lymphoma -- okay? --

1 which was what the state Mr. Hardeman was in for nine years
2 before he got the non-Hodgkin's lymphoma.

3 So it's very unlikely, it's almost impossible that the
4 hepatitis C could have caused his non-Hodgkin's lymphoma
5 because he was cured of the hepatitis C nine years before he
6 got the non-Hodgkin's lymphoma.

7 **Q.** Okay. If you want to go back to the stand, and I think
8 there's some other literature.

9 **A.** Right.

10 **Q.** And I'll direct you to -- this is Tab 1531 in your binder.

11 **MS. MOORE:** And permission to publish, please.

12 **MR. STEKLOFF:** No objection, Your Honor.

13 **THE COURT:** Go ahead.

14 **THE WITNESS:** So this is some data on hepatitis B and
15 hepatitis C viral infection and the risk of non-Hodgkin's
16 lymphoma. It was a case-control study from Italy and, I mean,
17 what the study showed is that if you have chronic active viral
18 infection with either hepatitis B or hepatitis C, you are at
19 about a two- to threefold increase risk of non-Hodgkin's
20 lymphoma.

21 But as I'll show you, if you have a history of past
22 infection and you no longer have the infection, then the risk
23 goes away.

24 So let's -- there's a table in this paper that I want to
25 show you. Can we hone in on table -- this table? Yeah.

1 So basically this -- the top half -- this doesn't really
2 show very well. It shows hepatitis C. So she's got the bar
3 where it says "HCV." That's hepatitis C. So we're going to
4 talk about hepatitis C first. Okay?

5 So if you -- and this is -- it's done in a case-control
6 format. So you have an odds ratio -- you have the cases, you
7 have the controls, and then you have the odds ratio on the far
8 right. Okay?

9 So the first category of people are the people who have
10 immunity to hepatitis C virus. Okay? And they have the
11 antibody but they don't have the viral infection. Okay?

12 No, these are the people who never -- I'm sorry. These
13 are the people who don't have the antibody. So they have never
14 been exposed to hepatitis C. So this is your control group.
15 Okay? And they have a risk ratio of 1.

16 If you look at the ones that have the antibody to
17 hepatitis C, then you see the risk goes up to over twofold like
18 I told you. So if you have had an -- if you have a history of
19 an infection with hepatitis C, you have an over twofold
20 increased risk.

21 But then if we divide that into --

22 **BY MS. MOORE:**

23 **Q.** Dr. Weisenburger, can I stop you for a second?

24 So the first row where it says anti-HCV, anti-
25 hepatitis C --

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1 A. Then it says negative. There's a little negative there.

2 Q. Oh. There you go.

3 A. You can't see it.

4 Q. Off the V.

5 Okay. Sorry the copy is bad.

6 A. Yeah.

7 Q. So the anti-HCV negative there, those are people who do
8 not have hepatitis C?

9 A. They never had hepatitis C --

10 Q. Okay.

11 A. -- right.

12 Q. So that's a control group?

13 A. That's sort of your --

14 Q. Like a control group?

15 A. -- control group, right.

16 Q. Okay. And then the next row are people who do have
17 hepatitis C?

18 A. They have antibody to hepatitis C. So we know that they
19 either have an active infection or they have had the infection
20 in the past. We don't really know which of those is, and
21 that's why you have to test for the hepatitis C RNA, which is
22 the next line. Okay?

23 And so if you test -- so you take these people that have
24 the antibody and you say, "Okay. Do they have active infection
25 or not?"

1 So you measure the hepatitis C virus in the blood; and if
2 it's negative, look across. There's no increased risk for
3 non-Hodgkin's lymphoma. The number is essentially 1. Okay?
4 Just like the people who never had it.

5 But if you look at the next line, people who have the
6 virus in their blood, those are the ones who are at increased
7 risk for non-Hodgkin's lymphoma.

8 So this makes the point that I told you that you have to
9 have chronic active viral hepatitis at the time you're
10 diagnosed with non-Hodgkin's lymphoma -- okay? -- to give you
11 an increased risk for non-Hodgkin's lymphoma.

12 If you're immune to it, like the column above where they
13 have the antibody but they don't have the virus, then you're
14 immune to it. Okay?

15 **Q.** So in Mr. Hardeman's case, when he's cured of hepatitis C
16 in 2006 and diagnosed with non-Hodgkin's lymphoma in 2015, what
17 conclusions can you draw from this table here?

18 **A.** Well, from this table if you have -- if you don't -- if
19 you have the antibody, which he did, and you don't have the
20 virus, then you're actually on the line between the two yellow
21 lines where you have an odds ratio of .98, which is
22 essentially 1, which is the same for your people who never had
23 a hepatitis C infection. Okay?

24 So just the fact that you had an infection in the past
25 does not put you at any increased risk for non-Hodgkin's

1 lymphoma. It's only if you have an active infection that's
2 ongoing right up until the time you get non-Hodgkin's lymphoma,
3 which he didn't have.

4 **Q.** Okay.

5 **A.** So let's go to the bottom half of that table because it
6 also then talks about hepatitis B, you know. So this is the
7 same format, and there are three different tests here. Let's
8 go across the first row. Okay?

9 So it's hepatitis surface antigen negative. Hepatitis --
10 so that's the antigen, that's the actual virus, the measure of
11 the virus in the blood.

12 **Q.** What's an antigen?

13 **A.** It's the -- it's part of the coat -- the coat of the
14 virus. It's a protein that's part of the coat of the virus,
15 and you can measure it. And so it tells you the virus is there
16 because you're finding the protein of the virus.

17 So in that first group, which is your control group, we
18 have -- you can't really see it very well unfortunately, but
19 it's hepatitis surface antigen negative and antibody surface
20 negative and antibody core protein negative. So these are
21 people who have never had hepatitis B. Okay? They have no
22 evidence of infection. They have no evidence of immunity.
23 Okay?

24 **Q.** Just like the line from the hepatitis C, the first line?

25 **A.** Right. So the odds ratio for them is 1. They're your

1 control group again. Okay?

2 Now, the next group is a group that has hepatitis surface
3 antigen negative, does not have the surface antibody, but has
4 the core antibody. Okay? And that's what Mr. Hardeman had.
5 When they tested him, he was negative for the surface antigen,
6 he was negative for the surface antibody, and he was positive
7 for the core antibody showing that he had immunity.

8 And if you go across, you can see that the odds ratio is
9 less than 1. So there's no increased risk for people who have
10 immunity to hepatitis C virus, which is what he had at the time
11 he was diagnosed with lymphoma.

12 If you go to the next one, again, it's hepatitis -- I
13 mean, I can't read it because I have to pull up --

14 **Q.** Do you want the actual study?

15 **A.** I want the actual study because I can't read the --

16 **Q.** Sorry. If you turn in your binder to 1531 and it's
17 page 4.

18 **A.** (Witness examines document.) All right. That's not much
19 better.

20 (Witness examines document.) I don't have my copy.

21 (Witness examines document.) Yeah, so the second row,
22 unfortunately, is -- the antigen is negative but both
23 antibodies are positive. So he's immune. Okay?

24 So he was actually this -- the third one -- the third one
25 is the antigen negative, the surface antibody positive, and the

1 core antibody negative. And, again, there's no increased risk.
2 Okay? So he's immune.

3 The fourth one is again the antigen is negative, the
4 surface antibody is negative, and the core antibody is
5 positive. And that's what Mr. Hardeman had. I'm sorry. It
6 was this fourth one.

7 So he didn't have any evidence of active viral infection,
8 and he had at least the anti-core antibody saying that he was
9 immune. And, again, you can see the risk ratio is 1.1. It's
10 very close to 1 and it's not significant. So he would not have
11 been at increased risk for non-Hodgkin's lymphoma based on
12 this.

13 And then the last one is people who have -- are surface
14 antigen positive. That indicates that they have active viral
15 infection. And if you look across, those are the people --
16 that's the only group that has an increased risk of
17 non-Hodgkin's lymphoma of about twofold increased risk. Okay?

18 **Q.** And does Mr. Hardeman fall in that last list?

19 **A.** No, he doesn't. He falls in the one above it.

20 **Q.** Okay.

21 **A.** Okay. So basically this is data that shows you if you're
22 immune to hepatitis C and you're immune to hepatitis B, you're
23 not at increased risk for non-Hodgkin's lymphoma. It's only
24 when you have the active viral infection that you're at
25 increased risk.

1 Q. Okay. All right. So then were there other studies that
2 you relied on in forming your opinion regarding the
3 hepatitis C?

4 A. So I just want to show you some examples of some of the
5 studies that prove what I just told you.

6 Q. All right. Let's go to I think it's Tab 1291.

7 MS. MOORE: Permission to publish.

8 MR. STEKLOFF: No objection, Your Honor.

9 THE COURT: Go ahead.

10 THE WITNESS: So this is a study from Japan in which
11 they had a large number of people with hepatitis C, many of
12 which were untreated and some of which were treated. And of
13 those that were treated, there was a group that had a sustained
14 virologic response just like Mr. Hardeman, and there was a
15 group that didn't have a sustained virologic response. They
16 had a response but it wasn't a complete response. Okay?

17 And so let's look at the curve.

18 MS. MOORE: Page 1039, please.

19 THE WITNESS: And read the title too. It says
20 "Viral" -- go back to the title. It's important to read the
21 title actually because it tells you what the study shows. So
22 "Viral elimination" -- they're talking about hepatitis C --
23 "reduces the incidence of malignant lymphoma in patients with
24 hepatitis C."

25 Okay. Let's look at the curve.

1 **MR. WOLFE:** Sorry. Which table was it?

2 **MS. MOORE:** 1039, Figure 3. It's the top half.

3 There you go.

4 **THE WITNESS:** So let's look at the figure. So what
5 this shows you is the time, a period of 5, 10, and 15 years
6 along the bottom of the curve; and on this upper side on the
7 other axis you see the incidence rate of non-Hodgkin's
8 lymphoma. Okay?

9 Yes. And what you can see is that the people with
10 persistent infection over time had an increasing incidence of
11 non-Hodgkin's lymphoma. So these are the people who were never
12 treated and had chronic persistent infection, chronic active
13 viral infection, and the people who were treated but who didn't
14 have a sustained virologic response. Okay? So you can see
15 that they over time are at increased risk for non-Hodgkin's
16 lymphoma.

17 But if you look at the patients who had a sustained
18 virologic response like Mr. Hardeman, it goes right along the
19 base. There weren't any cases of non-Hodgkin's lymphoma, not
20 one case in 15 years.

21 And actually if you go out to 20 years, there was only one
22 case and it was a T cell lymphoma, which was probably unrelated
23 to the virus at all.

24 So in this study, it's very dramatic, there's a
25 statistic -- a marked statistically significant difference in

1 these curves showing that people who have a sustained virologic
2 response don't have any increased risk for non-Hodgkin's
3 lymphoma.

4 **BY MS. MOORE:**

5 **Q.** So let me ask you two things from that. When you keep
6 saying SVR, the sustained virologic response, when we go back
7 to Exhibit 940 and we have marked here from February 23rd,
8 2006, all the way down to June 7th, 2015, and it's written
9 "Negative," what does that signify with respect to the
10 sustained virologic response for Mr. Hardeman?

11 **A.** Well, what it tells you is that once he attained a
12 sustained virologic response, he was no longer at increased
13 risk for non-Hodgkin's lymphoma.

14 **Q.** So this negative is he no longer has a sustained -- he has
15 a sustained virologic response; correct?

16 **A.** Yeah. So he's one year out there. So he -- you know,
17 after six months of negatives, you consider it a sustained
18 virologic response. And obviously he stayed in remission
19 during that whole nine-year period.

20 **Q.** And then the second thing is you said that there was one
21 person, I think about at the 20-year marker, that had the
22 infection and it was the T cell.

23 **A.** Right. So it was probably a background case, not related
24 at all to the non-Hodgkin's lymphoma because patients with
25 hepatitis B and hepatitis C, they get B-cell lymphomas. They

1 don't get T-cell lymphomas.

2 **Q.** Okay. So that's not relevant then?

3 **A.** It's not relevant, no.

4 **Q.** Okay. All right.

5 Anything else from this graph that you relied on in
6 forming your opinions in this case?

7 **A.** No.

8 **Q.** Okay. Let's go to 917.

9 **MS. MOORE:** And permission to publish, please.

10 **MR. STEKLOFF:** No objection, Your Honor.

11 **THE COURT:** Go ahead.

12 **THE WITNESS:** So this is another study that looked at
13 the frequency of liver cancer and non-Hodgkin's lymphoma in
14 patients with hepatitis C virus infection, and it's a cohort
15 study from Denmark. Okay?

16 And I'm just going to show you the data for non-Hodgkin's
17 lymphoma here.

18 **MS. MOORE:** So let's go -- Mr. Wolfe, if we could turn
19 to page 2314 and the graph in the lower right corner for
20 non-Hodgkin's lymphoma, please.

21 **THE WITNESS:** So, again, this is a similar graph as I
22 showed you before. Time is on the bottom part of the curve, it
23 goes out 10 years, and the cumulative incidence is on the other
24 part of the curve.

25 And what you see here, if you look at the dashed line,

1 which is the second line from the top -- can you show that?

2 The small dashes?

3 Right there. Yeah.

4 That's the line for people who have a history of
5 hepatitis C infection. They have a positive antibody. Okay?
6 So they either have active infection or they have a history of
7 past infection. Okay?

8 But like we did on the table, we can split that into two
9 groups -- right? -- the ones that have the active viral
10 infection, that have the RNA in the blood, and that's actually
11 the dark curve above, the solid curve. Okay? So they're the
12 ones who have a high infection.

13 And, in fact, the people who have immunity to hepatitis C
14 are the very low curve made up of the dots. Okay? So -- and
15 then the large dashes are the expected rate of non-Hodgkin's
16 lymphoma in the population.

17 So you can see that there's no difference in the curve --
18 the incidence curve for people who never had hepatitis C
19 compared to the lower dotted curve of people who are immune to
20 hepatitis C.

21 **BY MS. MOORE:**

22 **Q.** So what does --

23 **A.** It's only the people who have active infection who have an
24 increased risk.

25 **Q.** So what does that mean with respect to Mr. Hardeman?

1 **A.** Well, it means that his risk for non-Hodgkin's lymphoma
2 was no different than anybody else's who never had hepatitis C.

3 **Q.** Okay. And then those long dashes on this graph, that's
4 the incident rate of people who have hepatitis?

5 **A.** Who never had hepatitis.

6 **Q.** Who never had hepatitis.

7 **A.** So that's sort of your background rate in the general
8 population. Okay?

9 **Q.** So once you're treated for hepatitis and you're cured,
10 then you go back to where you were as if anyone in the general
11 population?

12 **A.** Yeah. So it's the line of dots, which is pretty much
13 similar to the large dashes. Those two are identical curves.
14 They look a little different, but statistically they're the
15 same.

16 **Q.** All right. And so what percentage, then, would you put on
17 someone who at one point in time had active hepatitis C, then
18 became cured of hepatitis C, stayed in the sustained virologic
19 response? What is the percentage risk that they might have to
20 develop non-Hodgkin's lymphoma?

21 **A.** There wouldn't be any increased risk.

22 **Q.** Okay. All right. Let's turn to 918.

23 **MS. MOORE:** And permission to publish.

24 **THE COURT:** Before we go there, let me ask you. How
25 much longer do you think you have with your direct of

1 Dr. Weisenburger?

2 **MS. MOORE:** I would say probably 15, 20 minutes.

3 **THE COURT:** Okay. In light of that, I think we should
4 break -- we should end for the day before you finish with
5 Dr. Weisenburger, but do you want to finish on the topic maybe
6 of hep C and then we'll wrap up for the day?

7 **MS. MOORE:** I would leave that up to the jury,
8 Your Honor. It doesn't matter.

9 **THE COURT:** Why don't you go ahead.

10 **MS. MOORE:** Okay.

11 **THE COURT:** It seems like you're almost done with
12 hep C.

13 **MS. MOORE:** I am.

14 Okay. So let's go to 918. And permission to publish.

15 **MR. STEKLOFF:** No objection, Your Honor.

16 **THE COURT:** Go ahead.

17 **BY MS. MOORE:**

18 **Q.** And what does this study tell us, Dr. Weisenburger?

19 **A.** So this is another study that looks at antiviral therapy
20 and the risk of non-Hodgkin's lymphoma with hepatitis C
21 infection. It's a recent study from China, and it also makes
22 the point that I made before if we can look at the diagram.

23 **Q.** Well, let's look. The title of it is early antiviral
24 therapy reduces risk of lymphoma in patients with chronic
25 hep C; is that right?

1 **A.** Right.

2 **Q.** Okay. So let's go to page 336, and I think it's the
3 diagram for non-Hodgkin's lymphoma, the B diagram, please.

4 **A.** So this is another similar curve like I've shown you with
5 the years on the lower scale and the incidence of NHL on the
6 other scale. And what you see for people who have untreated
7 hepatitis C virus, they have the dotted line, which is the
8 increasing incidence over time. Okay?

9 The small dotted line are people who never had hepatitis C
10 viral infection. Okay?

11 And then the other -- the solid line just above that are
12 the people who got a full course of Interferon and antiviral
13 therapy, and again you can see that the curve pretty much
14 matches the unexposed curve. Okay?

15 Probably it begins to stray a little bit because some of
16 those just had a partial response; but, in fact, if you get
17 treated, your risk of non-Hodgkin's lymphoma is the same as the
18 general population. It's not increased.

19 So it shows you the same -- in three different studies
20 I've sort of shown you the same thing, and there are actually
21 another five studies out there that I'm not even going to show
22 you, but there are eight studies that make this point very --
23 very well.

24 **Q.** And this -- Dr. Weisenburger, these studies and these
25 graphs you've been showing to the jury, is this based on real

1 data of real people?

2 **A.** Yes. It's real data of real people who had hepatitis C
3 infection and were treated with antivirals just like
4 Mr. Hardeman.

5 **Q.** It's not hypothetical about what might happen if you have
6 antiviral therapy following a diagnosis of hepatitis C?

7 **A.** No, this is real data.

8 **MR. STEKLOFF:** Objection.

9 **THE COURT:** Sustained.

10 **MS. MOORE:** Okay.

11 **THE COURT:** The answer will be stricken.

12 **BY MS. MOORE:**

13 **Q.** Dr. Weisenburger, is this information, the data that
14 you've been showing in these last three studies --
15 Exhibits 1291, 917, and 918 -- is that based on real data or a
16 hypothetical situation?

17 **MR. STEKLOFF:** Objection.

18 **THE WITNESS:** It's real data and real patients.

19 **THE COURT:** Sustained -- I mean, overruled. Sorry.

20 **BY MS. MOORE:**

21 **Q.** And, Dr. Weisenburger, if hepatitis C was really causing
22 non-Hodgkin's lymphoma after someone is cured, would you see it
23 in the literature in the data?

24 **A.** Well, you should see it because there's lots of data on
25 large numbers of patients, and the data is very consistent

1 across all eight studies that there's no increased risk for
2 non-Hodgkin's lymphoma for people who are cured or people who
3 become immune spontaneously. If you don't have active
4 infection, you're not at increased risk.

5 Q. Okay. And if you turn to 1302 in your binder.

6 MS. MOORE: Permission to publish.

7 MR. STEKLOFF: No objection, Your Honor.

8 THE COURT: Go ahead.

9 BY MS. MOORE:

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

11 A. Well, this is another study which shows similar findings,
12 but this study shows them for hepatitis B. So the story is the
13 same for hepatitis B, that if you don't have a chronic active
14 viral infection at the time you get non-Hodgkin's lymphoma,
15 then the hepatitis B is not the cause.

16 MS. MOORE: And let's go to the second page, please,
17 Mr. Wolfe.

18 THE WITNESS: Now, let's hone in just on the top two
19 diagrams.

20 Yeah. So this is now we're talking about hepatitis B,
21 okay? So remember we said that if you have the hepatitis B
22 surface antigen, which is the first one on the top diagram
23 there, and you look across this bar graph, it's like the four
24 spots that Dr. Ritz showed you, you can see there's an
25 increased risk of 1.82 for non-Hodgkin's lymphoma with active

1 hepatitis B infection.

2 But then if you go down to the next one, it says
3 anti-hepatitis C. So this person has the antibody, the
4 hepatitis C, like Mr. Hardeman did, and you can see the risk is
5 basically at 1. There's no increased risk. Okay?

6 And the same is true if you have the antibody to the
7 surface antigen. The risk is close to 1. It's not elevated.
8 They're immune -- these -- the lower two items are immune, and
9 they have no increased risk of non-Hodgkin's lymphoma
10 associated with hepatitis B infection.

11 And I won't belabor the point, but the curves -- the data
12 is the same for diffuse large B-cell lymphoma here. You have
13 an increased risk of over twofold with hepatitis -- with active
14 hepatitis infection with hepatitis surface antigen; but if you
15 have the antibodies, the risk is around 1 or less.

16 So both for hepatitis C and hepatitis B, if you're immune
17 or you're cured, you don't have an increased risk of
18 non-Hodgkin's lymphoma, and that's why I crossed out
19 hepatitis C and hepatitis B as substantial risk factors for
20 Mr. Hardeman -- okay? -- on my differential list.

21 **BY MS. MOORE:**

22 **Q.** Dr. Weisenburger, I'm going to have you, before we adjourn
23 for the day, come down off the stand and we're going to put up
24 the differential again.

25 **THE COURT:** Why don't we do that tomorrow morning?

PROCEEDINGS

1 **MS. MOORE:** Do it tomorrow morning? Okay. Great.
2 Thank you.

3 **THE COURT:** Okay. So we're wrapping up for today.
4 We'll resume again at 8:30 sharp tomorrow. And as I mentioned
5 to you back there this morning, even though we lost yesterday,
6 we're still a little bit ahead of schedule so that's the good
7 news. We look forward to seeing all six of you at 8:30 sharp
8 tomorrow.

9 Thank you.

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

11 **THE COURT:** And, Dr. Weisenburger, you're free to step
12 down.

13 **THE WITNESS:** Thank you.

14 **THE COURT:** Everybody, remember, nobody leaves the
15 courtroom for five minutes to give the jurors a chance to use
16 the elevators. So nobody is allowed to leave the courtroom
17 until they hear from either me or Kristen.

18 Okay. Anything else to discuss?

19 **MS. MOORE:** I don't think so, Your Honor.

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

21 **THE COURT:** I mean, it seems pretty clear that you're
22 not going to need to have Dr. Arber here tomorrow; right? I
23 mean, we're going to have -- you're going -- how long do you
24 anticipate your cross of Dr. Weisenburger to be?

25 **MR. STEKLOFF:** It might be a couple hours.

PROCEEDINGS

1 **THE COURT:** And then we have another hour of video
2 testimony.

3 **MS. MOORE:** Yes, Your Honor.

4 **THE COURT:** And then you'll put on Dr. Mucci, and she
5 likely will not finish tomorrow.

6 **MR. STEKLOFF:** I agree, Your Honor.

7 **THE COURT:** Okay. That's fine.

8 Okay. Anything else from anyone?

9 **MS. MOORE:** I don't think so, Your Honor.

10 **THE COURT:** Okay.

11 **MS. MOORE:** Thank you.

12 **THE COURT:** Sounds good. So everybody sit tight in
13 the courtroom for a couple more minutes, and you'll hear from
14 Kristen when you are permitted to leave.

15 (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



Jo Ann Bryce, CSR No. 3321, RMR, CRR, FCRR
U.S. Court Reporter



Marla F. Knox, RPR, CRR
U.S. Court Reporter