

# PORTIER\_DAY1\_SS\_PA\_01 FINAL PLAYED

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Portier, Christopher 02-21-2019

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6:3 - 6:15

**Portier, Christopher 02-21-2019 (00:00:26)**

CP1\_SS\_01.1

6:3 Q. Good morning.  
6:4 A. Good morning.  
6:5 Q. How are you doing?  
6:6 A. I'm doing fine today.  
6:7 Q. As you can see, we're doing a  
6:8 video testimony.  
6:9 Can you please tell the jury  
6:10 where we are right now?  
6:11 A. We're in Melbourne, Australia.  
6:12 This is a hotel. We're in a meeting room in  
6:13 the hotel, cameras, lawyers, staffers.  
6:14 Q. And, sir, why are we in  
6:15 Melbourne right now?

6:23 - 17:10

**Portier, Christopher 02-21-2019 (00:11:45)**

CP1\_SS\_01.2

6:23 THE WITNESS: I guess you're  
6:24 here because you want to hear my  
6:25 testimony in this case. I was  
7:1 supposed to be in San Francisco for  
7:2 the case. My wife and I came to  
7:3 Australia. She's on a sabbatical from  
7:4 the University of Bern for five  
7:5 months. And while we were here, I was  
7:6 in the gym, had a cardiac arrest,  
7:7 collapsed on the floor. I was very  
7:8 lucky, there were people there who  
7:9 knew what they were doing. Taken to  
7:10 the hospital. I spent a week in the  
7:11 hospital recovering. They put a  
7:12 pacemaker and an automatic  
7:13 defibrillator in my chest to  
7:14 kick-start my heart next time it  
7:15 stops.  
7:16 I'm really not in a position to  
7:17 travel all the way back to San  
7:18 Francisco at this time because of this  
7:19 health concern, and that's why you're  
7:20 here, I believe.  
7:21 QUESTIONS BY MR. WISNER:  
7:22 Q. Well, sir, thank you so much

7:23 for being here. I really appreciate it.  
7:24 A. Well, thank you for coming  
7:25 here. I do appreciate the defense's coming.  
8:1 Q. Could you please state your  
8:2 full name and introduce yourself to the jury?  
8:3 A. My name is Christopher Jude  
8:4 Portier. I currently live in Switzerland.  
8:5 I'm a citizen of the United States.  
8:6 What more do you want to know?  
8:7 Q. You know what, we'll get into  
8:8 it directly.  
8:9 Let's start off with your  
8:10 educational background.  
8:11 A. Okay.  
8:12 Q. Where did you go to college?  
8:13 A. I went to a little college in  
8:14 Louisiana called Nicholls State University.  
8:15 It was about 40 miles from my hometown. From  
8:16 there I went to graduate school at the  
8:17 University of North Carolina in Chapel Hill.  
8:18 My undergraduate degree was mathematics and  
8:19 my graduate degree was in biostatistics with  
8:20 a minor in epidemiology.  
8:21 Q. And following your Ph.D. --  
8:22 well, when you were at UNC, what did you  
8:23 focus on in your Ph.D.?  
8:24 A. My Ph.D. was on the optimal  
8:25 design and analysis for two-year animal  
9:1 cancer bioassays. These are studies done in  
9:2 animals to look at chemicals that might cause  
9:3 cancer in the animals. It was finding the  
9:4 design that worked best for evaluating the  
9:5 studies.  
9:6 Q. Was that what your dissertation  
9:7 was about?  
9:8 A. That's what my dissertation was  
9:9 about.  
9:10 Q. And in your work looking at the  
9:11 optimal design, how has that impacted the way  
9:12 we look at animal studies today?

9:13 A. Well, the National Toxicology  
9:14 Program still uses that particular design in  
9:15 all of their bioassays, and most people use  
9:16 variations on that particular design. It's a  
9:17 good practical guide.

9:18 Q. And, sir, just to give the jury  
9:19 a sense, what drew you to this area of  
9:20 science?

9:21 Why did you want to look at  
9:22 animal studies?

9:23 A. Well, to be honest, when I was  
9:24 in graduate school, I had a daughter and a  
9:25 wife that I had to support, and the National  
10:1 Institute of Environmental Health Sciences  
10:2 needed somebody to look at their cancer  
10:3 bioassay and find the way to create an  
10:4 optimal design for them so that they used --  
10:5 they were most efficient in the use of  
10:6 animals and at the same time got the most  
10:7 information out of it. They offered me  
10:8 part-time employment to work on it as my  
10:9 Ph.D. thesis. It was a great opportunity for  
10:10 me.

10:11 Q. Following your Ph.D., where did  
10:12 you begin working?

10:13 A. At the National Institute of  
10:14 Environmental Health Sciences, which I'll  
10:15 just call NIEHS now. NIEHS offered me a job  
10:16 to stay there after I got my Ph.D. to work  
10:17 with them and with the National Toxicology  
10:18 Program, which is physically in the same  
10:19 building and managed by the same  
10:20 organization, and so I took that position.

10:21 Q. Can you please explain to the  
10:22 jury what are these various institutions?  
10:23 How do they fit within our sort  
10:24 of scientific umbrella in the US?

10:25 A. So in environmental issues in  
11:1 the United States, you have -- let's just say  
11:2 there are four major players: The

11:3 Environmental Protection Agency, which is the  
11:4 regulatory authority, they interpret the laws  
11:5 and set standards and make sure that  
11:6 companies follow those standards that they  
11:7 set.

11:8 The Centers for Disease Control  
11:9 and Prevention does public health outlook.  
11:10 They try to find ways to prevent lead  
11:11 poisoning, prevent asthma attacks, so their  
11:12 job is to get out into the public and improve  
11:13 public health.

11:14 The FDA is in charge of food  
11:15 and the quality of food.  
11:16 And then the National Institute  
11:17 of Environmental Health Sciences is the  
11:18 research arm. They're part of the National  
11:19 Institutes of Health. They fund research in  
11:20 the NIEHS, about 10 percent of their budget,  
11:21 but then about 90 percent of their budget is  
11:22 sent out to researchers and universities  
11:23 around the country to -- competitive grants  
11:24 to look at environmental health hazards in  
11:25 the population.

12:1 They're also the home of the US  
12:2 National Toxicology Program. It's the  
12:3 world's largest toxicology program. Their  
12:4 job is on behalf of the federal agencies to  
12:5 do studies to look at the impact of  
12:6 chemicals, the potential impact of chemicals  
12:7 on people, and most of that work is done in  
12:8 laboratories either using human cells or  
12:9 animal cells or animals themselves.  
12:10 Q. Now, when you finished your  
12:11 Ph.D. and you started at the NIEHS and the  
12:12 NTP, National Toxicology Program, what did  
12:13 you do?

12:14 A. Well, when I first started out,  
12:15 I did the same thing I basically did as a  
12:16 graduate student: I did research into better  
12:17 ways to analyze and interpret laboratory

12:18 studies. So I continued to do a lot of work  
12:19 on cancer bioassays, came up with a method to  
12:20 analyze the data from a cancer bioassay that  
12:21 the National Toxicology Program is still  
12:22 using today as well as many other  
12:23 authorities.  
12:24 We did work on reproductive  
12:25 toxicology, developmental toxicology, so how  
13:1 infants develop through their life and how  
13:2 chemicals might affect that. Immunological  
13:3 changes that chemicals might cause. So I  
13:4 continue to do that type of work.  
13:5 Eventually I stepped away from  
13:6 that work and became much more interested in  
13:7 the laboratory work itself and how the  
13:8 mechanisms of carcinogenesis work, and I  
13:9 spent a lot of time working with laboratories  
13:10 on how we might interpret that, better ways  
13:11 to create things on the computer that can  
13:12 help us interpret it better.  
13:13 After a while, I started my own  
13:14 laboratory doing my own research, so I had  
13:15 actually scientists who were in the lab  
13:16 mixing chemicals and exposing cells and  
13:17 things like that for experiments that I  
13:18 wanted to do.  
13:19 And after that I went into much  
13:20 more administrative work. Still kept my lab  
13:21 through my entire time at NIH, but I also did  
13:22 a lot of other administrative work.  
13:23 Q. And while you were at the NIH,  
13:24 National Institute of Health, what -- did you  
13:25 elevate in position while you were there?  
14:1 A. Well, I was a principal  
14:2 investigator from the first day that I was at  
14:3 NIEHS, and that's an independent scientific  
14:4 researcher within the organization. You have  
14:5 your own resources. You can get graduate  
14:6 students and laboratory supplies and things  
14:7 like that. And that's the standard position

14:8 for anybody who is doing science within NIH.  
14:9 But as time went on, I also  
14:10 took on larger positions. I was in charge of  
14:11 an entire branch that did work on  
14:12 computational biology and risk assessment.  
14:13 Then I was in charge of an entire division.  
14:14 All of the toxicology research within the  
14:15 NIEHS was under my management and control and  
14:16 as well I took over management of the  
14:17 National Toxicology Program for six years.  
14:18 And then after that I became  
14:19 the senior scientific advisor to the director  
14:20 of NIEHS, and there I worked on issues such  
14:21 as starting a program for climate change and  
14:22 human health research at NIH, starting a  
14:23 series of centers on children's environmental  
14:24 health issues across the United States,  
14:25 things like that.

15:1 Q. Following your time at NIH, did  
15:2 you work at another agency?  
15:3 A. Yes. I then went on to the  
15:4 Centers for Disease Control and Prevention in  
15:5 Atlanta where I was director of their  
15:6 National Center for Environmental Health.  
15:7 That's the center that's concerned about  
15:8 environmental public health in the United  
15:9 States. So, like I said earlier, they do  
15:10 things like lead poisoning prevention, asthma  
15:11 prevention. They measure chemicals in  
15:12 people's blood in the United States on a  
15:13 routine basis to look and see trends in  
15:14 chemical exposures, so are they going down,  
15:15 are they going up, what should we be  
15:16 concerned about.  
15:17 They have climate change in the  
15:18 human health program. They have a number of  
15:19 different programs. They even inspect all  
15:20 the cruise lines that land in the United  
15:21 States. So if you ever fly -- go on a cruise  
15:22 ship, CDC's National Center for Environmental

15:23 Health has inspected that cruise ship for  
15:24 sanitary practices.  
15:25 I was also director of the  
16:1 Agency for Toxic Substances and Disease  
16:2 Registry, and that's also in Atlanta. It's  
16:3 also under the management of the CDC,  
16:4 although it's not part of the CDC. So it's  
16:5 sort of like the National Toxicology Program  
16:6 at NIEHS. So I had two jobs, running both  
16:7 organizations.  
16:8 ATSDR concerns itself with  
16:9 Superfund sites. So these are toxic dump  
16:10 sites in the United States, and their legal  
16:11 responsibility is to assess the potential for  
16:12 health impacts in a community from those dump  
16:13 sites and then advise the Environmental  
16:14 Protection Agency on whether these sites need  
16:15 to be cleaned up.  
16:16 And then it's EPA's  
16:17 responsibility to clean it, to sue and get  
16:18 money to -- for cleanup from anybody who  
16:19 actually caused the problem. And then at the  
16:20 end, it's our job to go back and certify that  
16:21 it is now safe for the community.  
16:22 Q. All toll, how long were you  
16:23 working in government service and public  
16:24 health issues?  
16:25 A. Let's see. 1978 to 2013.  
17:1 About 35, 36 years.  
17:2 Q. And during that time, what  
17:3 percentage of your work focused on the causes  
17:4 of cancer?  
17:5 A. Well, at NIH it was clearly 80,  
17:6 90 percent of my work dealt with cancer,  
17:7 causes of cancer and mechanisms of cancer.  
17:8 At CDC, it's a bigger public  
17:9 health problem, so bigger health issues, so I  
17:10 spent more time with a lot of other things.  
17:11 Q. And specifically when it comes

17:11 - 18:4

**Portier, Christopher 02-21-2019 (00:00:58)**

CP1\_SS\_01.3

17:12 to cancer or carcinogens, can you give the  
 17:13 jury some examples of some of the projects  
 17:14 you worked on when you worked at the National  
 17:15 Toxicology Program and NIH?  
 17:16 A. Sure. One thing I worked on  
 17:17 for a number of years was the carcinogenicity  
 17:18 of dioxin. It's a contaminant. It's not a  
 17:19 chemical that you really want to have around.  
 17:20 It gets created accidentally in the  
 17:21 production of certain things. I spent a lot  
 17:22 of time on trying to understand how dioxins  
 17:23 cause cancer. We did a number of studies on  
 17:24 various ways to see what's going on with the  
 17:25 cancer process from dioxins, and we also used  
 18:1 that as a stepping stone for understanding  
 18:2 how chemicals that interact with what are  
 18:3 called cellular receptors can cause cancer in  
 18:4 people.

18:7 - 19:5

**Portier, Christopher 02-21-2019 (00:01:18)**

CP1\_SS\_01.4

18:7 Let's see. What else did I do?  
 18:8 I spent time looking at the  
 18:9 potential of power lines and electric and  
 18:10 magnetic fields to cause cancer in children,  
 18:11 childhood leukemia. There was some  
 18:12 literature on that subject that had concerned  
 18:13 Congress and they tasked NIH with looking at  
 18:14 that, and NIH tasked me with leading that  
 18:15 effort.  
 18:16 I did some work on early cancer  
 18:17 development in the brains of rats from  
 18:18 exposure to a variety of different chemicals.  
 18:19 And then I did -- one of the final things I  
 18:20 looked at was not just cancer, but cancer was  
 18:21 a big part of it, but sort of all human  
 18:22 diseases, all chemicals, and the question was  
 18:23 whether we could use this whole area called  
 18:24 genomics and proteomics to go from  
 18:25 experiments in cells and animals and predict  
 19:1 on a huge basis all human disease that they  
 19:2 are associating with, and we created this

19:6 - 23:10

19:3 huge network linking about 4,000 chemicals to  
19:4 about 200 human diseases. That was a really  
19:5 nice project.

**Portier, Christopher 02-21-2019 (00:04:36)**

CP1\_SS\_01.5

19:6 Q. Did you ultimately retire, sir?  
19:7 A. Yes, in 2013 I retired from --  
19:8 Q. What did you do after that?  
19:9 A. I spent six months working at  
19:10 the International Agency for Research on  
19:11 Cancer in Lyon, France. I was there as a  
19:12 senior visiting scientist. I think that's  
19:13 the title they use for it. It's a grant  
19:14 position that they bring people in -- at six  
19:15 months at a time to work with them. I worked  
19:16 on ways to evaluate mechanistic studies in  
19:17 cancer evaluations.  
19:18 After that I was working for  
19:19 the Environmental Defense Fund in the United  
19:20 States. It's a nonprofit, nongovernment  
19:21 organization. Their goal is to encourage the  
19:22 better use of science in policy decisions.  
19:23 They fund a lot of scientific research, and  
19:24 they do a lot of policy arguments and pushing  
19:25 for policy goals.  
20:1 My job there was to help them  
20:2 design some of the studies they're doing,  
20:3 evaluate some of the science that they were  
20:4 funding, mostly in the area of climate change  
20:5 and air pollution, and a little bit in the  
20:6 area of fracking and a little bit in the area  
20:7 of looking at human exposures to chemicals.  
20:8 And then I've done some  
20:9 consulting work for federal, for governments  
20:10 around the world and some consulting with  
20:11 lawyers.  
20:12 Q. You mentioned you did some --  
20:13 you've been doing some work with the NRDC.  
20:14 Can you please -- has any of  
20:15 that work related to health issues in the Bay  
20:16 area?

20:17 A. So it's not NRDC.

20:18 Q. Oh, sorry.

20:19 A. NRDC is the National Resources

20:20 Defense Council, and I have worked with them.

20:21 But, no, this was with the Environmental

20:22 Defense Fund.

20:23 Q. Sorry.

20:24 A. EDF.

20:25 Q. EDF.

21:1 A. And, yes, they have -- we have

21:2 done work in the Bay area. We -- one of the

21:3 very first things I did at EDF was meet with

21:4 Google. Google has Street View cars. If any

21:5 of you ever go and look at Google's maps, you

21:6 can always go down to the level where all of

21:7 a sudden now you're standing on the street

21:8 looking around. Those are cars that drive

21:9 around with cameras at the top and take all

21:10 these pictures.

21:11 Well, we had the idea that we

21:12 could put air pollution monitors on those

21:13 same cars and while they are driving around

21:14 taking pictures, at the same time they would

21:15 be driving around and measuring air pollution

21:16 in local communities, and we could use that

21:17 to map out at the local level what air

21:18 pollution looks like.

21:19 They agreed to work with us on

21:20 that project, and we started in Oakland and

21:21 we did a lot of mapping and monitoring in

21:22 Oakland. We -- at the same time we brought

21:23 in a local insurance company for -- Kaiser

21:24 Permanente for northern California, and we

21:25 worked with them on health records of people

22:1 near where this air pollution was being

22:2 measured to see if we could see differences

22:3 in health impacts of the air pollution at the

22:4 local levels.

22:5 Now we're doing -- we've

22:6 expanded that study into the entire Bay area,

22:7 so I think we're doing 14 of the cities in  
 22:8 and around San Francisco Bay. We've expanded  
 22:9 it into Houston metropolitan area in Texas.  
 22:10 We've expanded it into London. We have a  
 22:11 large project in London right now, and we're  
 22:12 looking at expanding into two more cities in  
 22:13 the near future.

22:14 Q. Sir, I understand you're  
 22:15 retired. Why are you doing this work?  
 22:16 A. Well, you spend all your career  
 22:17 figuring out how to do something. You think  
 22:18 when you first get your Ph.D., you know  
 22:19 everything. By the time you are my age, you  
 22:20 realize that you don't know everything, and  
 22:21 you still continue to learn.

22:22 My passion for environmental  
 22:23 health has not waned simply because I  
 22:24 retired. So I still do it because it's  
 22:25 important. It's what I spent my entire life  
 23:1 training for. The American public paid for  
 23:2 me to learn all this stuff. I figured they  
 23:3 should get something back from it, so I  
 23:4 continue to work on these issues.

23:5 Q. Now, you mentioned that shortly  
 23:6 after your retirement you spent six months  
 23:7 with the International Agency for Research on  
 23:8 Cancer. Do you recall that?  
 23:9 Is that also known as IARC?

23:10 A. Yes.

23:11 - 24:12

**Portier, Christopher 02-21-2019 (00:01:16)**

CP1\_SS\_01.6

23:11 Q. And I don't want to spend too  
 23:12 much time talking about IARC, but just for  
 23:13 those of us who aren't familiar, what is  
 23:14 IARC?

23:15 A. So the United Nations is a big  
 23:16 organization that many, many nations belong  
 23:17 to, and the United Nations has several  
 23:18 underlying organizations, one of which is the  
 23:19 World Health Organization. The World Health  
 23:20 Organization's goal is to sort of improve the

23:21 health of everybody on the planet. And under  
23:22 the World Health Organization, there are  
23:23 other subgroups, there's divisions that worry  
23:24 about infectious diseases and AIDS and  
23:25 noncommunicable diseases.

24:1 A. semi-independent agency  
24:2 within WHO is the International Agency for  
24:3 Research on Cancer. They started out as an  
24:4 agency that was intended to help countries  
24:5 around the world develop cancer registries so  
24:6 they could figure out how much cancer risk  
24:7 there were in each of these countries. But  
24:8 it broadened into a research organization  
24:9 that does global research on cancer as well  
24:10 as an organization that evaluates causes of  
24:11 cancer and works in ways to prevent those  
24:12 cancers from occurring.

24:13 - 25:13

**Portier, Christopher 02-21-2019 (00:01:04)**

CP1\_SS\_01.7

24:13 Q. Have you personally  
24:14 participated in IARC programs to evaluate  
24:15 whether or not things cause cancer?

24:16 A. Oh, yes.

24:17 Q. How many times; do you recall?

24:18 A. Seven or eight times for  
24:19 different collections of things that might  
24:20 cause cancer.

24:21 Q. And are you paid when you  
24:22 participate in that?

24:23 A. No. No. It's nonpaid. They  
24:24 simply cover your expenses.

24:25 Q. Why did you do it?

25:1 A. Well, most of the time I was  
25:2 working for the US government, so it was, in  
25:3 essence, part of my job to participate in  
25:4 activities like that. Even though I'm not  
25:5 representing the US government when I do  
25:6 that, they encourage us -- the NIH encouraged  
25:7 us to be involved in issues that are  
25:8 important like the evaluation of agents that  
25:9 might cause cancer.

25:10 NIH also encouraged me to work  
25:11 on EPA science advisory board and EPA's  
25:12 science advisory panel, and I worked on an  
25:13 Australian science advisory board for years.

26:6 - 27:17 **Portier, Christopher 02-21-2019 (00:01:52)**

26:6 Q. All right, sir. Now we've kind  
26:7 of covered some of your background. I want  
26:8 to sort of get to why we're here today.  
26:9 How did you get involved with  
26:10 glyphosate?  
26:11 A. So IARC was -- IARC had decided  
26:12 to review several pesticides for their  
26:13 potential for causing cancer, one of which  
26:14 was glyphosate. And so they put together a  
26:15 panel of scientists who were going to review  
26:16 these chemicals and make some decisions about  
26:17 whether it would -- they cause cancer or not,  
26:18 and their basic approach to looking at that.  
26:19 They had asked me to join them  
26:20 specifically for -- for chemicals for which  
26:21 there was information coming out of a program  
26:22 I started when I was at the National  
26:23 Toxicology Program, running that program,  
26:24 that brought in a lot of mechanistic  
26:25 information in sort of a very large scale,  
27:1 and they weren't sure they knew how to  
27:2 approach that data and they wanted me there  
27:3 to help them sort of interpret it. This was  
27:4 the first time they were facing what is  
27:5 called this Tox21 dataset. And so they asked  
27:6 me to come and help them with that, and  
27:7 that's why I was involved.  
27:8 And after that evaluation, I  
27:9 was approached by a law firm I had already  
27:10 been providing free advice to, whether I  
27:11 would provide them with advice on the science  
27:12 underlying the glyphosate decision that was  
27:13 made by IARC.  
27:14 Q. Can you turn to Exhibit 230 in  
27:15 your binder? It should be numbered pretty

CP1\_SS\_01.8

27:16 easily.

27:17 A. Okay.

29:7 - 30:15

**Portier, Christopher 02-21-2019 (00:01:27)**

CP1\_SS\_01.9

29:7 Q. Okay. And if we go down here,

29:8 there's a bunch of different names. I want

29:9 to go down to where you're mentioned. It

29:10 says your name under Invited Specialists.

29:11 Do you see that?

29:12 A. Yes.

29:13 Q. What is an invited specialist?

29:14 A. So an invited specialist is, in

29:15 essence, a consultant to the working group.

29:16 So you have the core working group, which in

29:17 this case I think is 16 or 17 scientists,

29:18 they write the evaluation of the literature,

29:19 they come up with the opinion of what they

29:20 believe the potential for carcinogenicity is

29:21 for the chemicals they're looking at and

29:22 write their overall decisions. That's their

29:23 job.

29:24 Sometimes the IARC decides that

29:25 they need some extra expertise but sometimes

30:1 that expertise has potential conflicts of

30:2 interest, and so they bring that expertise as

30:3 invited specialists. They're not allowed to

30:4 write. They're not allowed to help with the

30:5 decision. They're there to provide expert

30:6 advice on individual studies and just general

30:7 science overall.

30:8 In my case because I was

30:9 working part time for the Environmental

30:10 Defense Fund, which is a nongovernment

30:11 organization that advocates for environmental

30:12 issues, they felt it was a potential conflict

30:13 of interest and so they didn't want me on the

30:14 working group; they wanted me there simply to

30:15 provide expertise to the committee.

34:20 - 34:25

**Portier, Christopher 02-21-2019 (00:00:13)**

CP1\_SS\_01.10

34:20 Q. So following the IARC monograph

34:21 on glyphosate and those other pesticides that

34:22 were reviewed, you stated that you were --  
34:23 you began working with a law firm; is that  
34:24 right?

34:25 A. That is correct.

35:1 - 35:18

**Portier, Christopher 02-21-2019 (00:00:43)**

CP1\_SS\_01.11

35:1 Q. Okay. Following the IARC --  
35:2 well, put simply, what was IARC's conclusion,  
35:3 sir?

35:4 A. IARC's conclusion was that --  
35:5 for glyphosate specifically. IARC's  
35:6 conclusion was for glyphosate was that it  
35:7 probably carcinogenic to human -- humans,  
35:8 which is a classification that has a full  
35:9 categorization to it and rules under which  
35:10 it's created.

35:11 Q. And just to give the jury some  
35:12 context, that classification as a probable  
35:13 human carcinogen, where does that fall?

35:14 Is it the highest? Second  
35:15 highest? Third highest?

35:16 A. IARC has five classification  
35:17 batches that they put things in. Probable is  
35:18 the second highest.

35:19 - 37:1

**Portier, Christopher 02-21-2019 (00:01:26)**

CP1\_SS\_01.12

35:19 Q. Okay. Now, following the IARC  
35:20 classification, do you know if there's been  
35:21 any scientific response by regulatory  
35:22 agencies to IARC?

35:23 A. There was a lot of response to  
35:24 the IARC monograph by regulatory agencies.

35:25 Q. And did you take any actions to  
36:1 defend the IARC decision?

36:2 A. I took actions to not so much  
36:3 defend the IARC decision as to highlight the  
36:4 differences in the scientific justification  
36:5 for the decisions that were made by IARC as  
36:6 compared to other groups.

36:7 Q. And is one of those groups the  
36:8 European Union's equivalent of EPA?

36:9 A. The European Food Safety

36:10 Authority -- Agency, yes. I had discussions  
36:11 with them and their management.

36:12 Q. And is that group called EFSA?

36:13 A. EFSA, yes.

36:14 Q. And I understand you actually

36:15 published an open letter to the scientific

36:16 community, along with some colleagues; is

36:17 that right?

36:18 A. That is correct.

36:19 Q. Okay. Please turn to

36:20 Exhibit 228.

36:21 A. Okay.

36:22 Q. Is that a fair and accurate

36:23 copy of the letter you published?

36:24 A. Yes, it is.

36:25 Q. Okay. I'll publish this

37:1 document.

37:18 - 39:13

**Portier, Christopher 02-21-2019 (00:02:04)**

CP1\_SS\_01.13

37:18 Q. Okay. So we have here this

37:19 document, it's titled "Differences in the

37:20 carcinogenic evaluation of glyphosate between

37:21 the International Agency for Research on

37:22 Cancer, IARC, and the European Food Safety

37:23 Authority, EFSA."

37:24 Do you see that?

37:25 A. Yes.

38:1 Q. All right. And I notice on

38:2 this signature line there are -- well, how

38:3 many -- how many people signed this letter

38:4 with you, sir?

38:5 A. There are 96 signatures, I

38:6 believe.

38:7 Q. Okay. And then if we just go

38:8 to the back of it -- well, what was the

38:9 ultimate conclusion from this article?

38:10 A. Well, we were -- in the article

38:11 we were challenging -- so when EFSA -- EFSA

38:12 was in the process of re-reviewing glyphosate

38:13 when IARC did their review. And the IARC

38:14 review -- EFSA had already said that they

38:15 didn't think there was a problem with  
 38:16 glyphosate, so when the IARC review came out,  
 38:17 it created a conflict with EFSA.  
 38:18 So EFSA's -- the way Europe  
 38:19 does these things is they get authorities in  
 38:20 each country in Europe -- one or two  
 38:21 countries in Europe to lead the effort. So  
 38:22 in this case, the German Federal Institute  
 38:23 for Risk Analysis was leading the effort.  
 38:24 I'll just refer to them as BfR. Stands for  
 38:25 Bundesinstitut f|r Risikobewertung.

39:1 Q. Okay.

39:2 A. So BfR then did an appendix  
 39:3 that walked through what they thought were  
 39:4 the differences between IARC and EFSA and  
 39:5 published that, that appendix.

39:6 We're responding to that  
 39:7 appendix more than anything else where we  
 39:8 point out some of the scientific flaws in  
 39:9 what they did.

39:10 Our final conclusion was that  
 39:11 EFSA's review was flawed scientifically,  
 39:12 IARC's was not, and that we believe the IARC  
 39:13 classification is the correct classification.

39:14 - 40:3

**Portier, Christopher 02-21-2019 (00:00:31)**

CP1\_SS\_01.14

39:14 Q. So if you look at the last page  
 39:15 here, I will call it out. Hopefully you can  
 39:16 read it on your screen. It reads, "The most  
 39:17 appropriate and scientifically based  
 39:18 evaluation of the cancers reported in humans  
 39:19 and laboratory animals as well as supportive  
 39:20 mechanistic data is that glyphosate is a  
 39:21 probable human carcinogen. On the basis of  
 39:22 this classification -- sorry. On the basis  
 39:23 of this conclusion and in the absence of  
 39:24 evidence to the contrary, it is reasonable to  
 39:25 conclude that glyphosate formulations should  
 40:1 also be considered likely human carcinogens."

40:2 Do you see that?

40:3 A. Yes, I --

Page/Line	Source	ID
40:8 - 40:21	<p><b>Portier, Christopher 02-21-2019 (00:00:32)</b></p> <p>40:8 Q. And I just want to draw your  40:9 attention, sir, to a couple of the authors  40:10 that joined you on this letter.  40:11 Specifically do you see here  40:12 Anneclaire De Roos?  40:13 A. Anneclaire De Roos, yes.  40:14 Q. Sorry, De Roos.  40:15 And Dr. De Roos, I understand,  40:16 she was an author on a recent AHS  40:17 publication?  40:18 A. At the time, yes, she was  40:19 author on several publications on glyphosate,  40:20 one of them the AHS publication specifically  40:21 on glyphosate.</p>	CP1_SS_01.15
41:7 - 41:14	<p><b>Portier, Christopher 02-21-2019 (00:00:18)</b></p> <p>41:7 Q. Okay. I also saw on here  41:8 there's another physician -- or another  41:9 researcher, Charles Lynch.  41:10 Do you see that?  41:11 A. Yes.  41:12 Q. Charles Lynch, he's also an  41:13 author on a recent AHS publication?  41:14 A. Well, that, I don't know.</p>	CP1_SS_01.16
41:18 - 42:5	<p><b>Portier, Christopher 02-21-2019 (00:01:03)</b></p> <p>41:18 Q. Well, let's just check.  41:19 I believe the AHS publication  41:20 should be in your binder. It is Exhibit 550.  41:21 Are you there?  41:22 A. Yes.  41:23 Q. And is Dr. Lynch an author on  41:24 the article?  41:25 A. Let me check real quick here.  42:1 University of Iowa, Department  42:2 of Epidemiology. It's the same name. Let me  42:3 see if it's the same affiliation.  42:4 Yeah, that would seem to be the  42:5 same person.</p>	CP1_SS_01.17
42:18 - 45:1	<p><b>Portier, Christopher 02-21-2019 (00:02:27)</b></p> <p>42:18 Q. Based on what I've shown you,</p>	CP1_SS_01.18

42:19 are there any authors that joined you in this  
42:20 letter who are also authors on the recent AHS  
42:21 publication?

42:22 A. Yes.

42:23 Q. Okay. Who are those?

42:24 A. Well, if you're talking about  
42:25 the Andreotti publication, I don't believe  
43:1 De Roos is on that publication.

43:2 Q. Well, let's take a look, sir.

43:3 It's 550.

43:4 A. Oh, yes, she is. You're right.

43:5 Absolutely. So two of them are on the most  
43:6 recent publication.

43:7 Q. Yeah. And so we're looking at  
43:8 Exhibit 550 on the screen, just so we can  
43:9 confirm this.

43:10 Do you see Dr. De Roos and  
43:11 Dr. Lynch?

43:12 A. Yes, I do.

43:13 Q. Okay. Great.

43:14 Okay. So after IARC, did you  
43:15 take a step further in looking at the science  
43:16 behind glyphosate?

43:17 A. Yes, I did.

43:18 Q. What did you do?

43:19 A. Well, in drafting this response  
43:20 to EFSA, of course I had to spend a lot of  
43:21 time reading through their evaluation, and  
43:22 they had evaluated studies that IARC did not  
43:23 evaluate. They were evaluating studies that  
43:24 were proprietary and not in the public  
43:25 domain, something IARC does not do. And so I  
44:1 had to spend a lot of time looking at those  
44:2 studies and other science. I spent just a  
44:3 lot more time looking at it.

44:4 I also responded to something  
44:5 done by the US EPA. That took a lot of time  
44:6 and effort for me to go through, not only  
44:7 looking at what EPA did but redoing the  
44:8 analyses and redoing some of the evaluations.

44:9 Q. And to be clear, sir, that work  
44:10 you did responding to the EPA, this open  
44:11 letter we just looked at responding to EFSA,  
44:12 were you being paid by attorneys to do that  
44:13 work?

44:14 A. No, I was not being paid by  
44:15 anyone to do that work.

44:16 Q. Why are you doing it then?

44:17 A. Again, I've spent 36 years of  
44:18 my life learning how to evaluate animal and  
44:19 human cancer data and make decisions about  
44:20 whether this is a carcinogen or not. That is  
44:21 sort of the primary thing my career has been  
44:22 aimed at, and I feel that having looked at  
44:23 the way these agencies looked at this  
44:24 particular pesticide, they've missed all the  
44:25 rules that are in place that they should have  
45:1 followed in doing the evaluation.

45:14 - 72:4

**Portier, Christopher 02-21-2019 (00:27:17)**

CP1\_SS\_01.19

45:14 Q. All right. Okay. So when it  
45:15 comes to looking at whether or not an agent  
45:16 causes cancer, what areas of science do you  
45:17 as a scientist look at?

45:18 A. I look at the human evidence,  
45:19 so studies that have looked at populations of  
45:20 humans exposed to the agent. That would be  
45:21 epidemiology.

45:22 I look at the animal -- the  
45:23 laboratory animal data, where we take whole  
45:24 animals and expose them to the agent and look  
45:25 to see if it causes cancer in them.

46:1 And then I look at shorter  
46:2 laboratory experiments aimed at looking at  
46:3 the mechanisms by which cancer may be arising  
46:4 in these studies in animals and humans.

46:5 Q. All right. So I've prepared a  
46:6 little picture that I want to use to sort of  
46:7 help -- get the document camera -- to sort of  
46:8 get a -- sort of get a view of the different  
46:9 things.

46:10 So at the top of this picture,  
46:11 on top of the stool, I'm going to write  
46:12 "causation."  
46:13 Okay?  
46:14 A. Okay.  
46:15 Q. And you mentioned there are  
46:16 these three areas of science that you look  
46:17 at. The first one you mentioned was  
46:18 epidemiology; is that right?  
46:19 A. That's correct, epidemiology.  
46:20 Q. Okay. So I'm going to write  
46:21 that here on one of the legs.  
46:22 All right. And then you said  
46:23 you looked at -- is that animal studies?  
46:24 A. Yes.  
46:25 Q. All right.  
47:1 A. Animal cancer studies.  
47:2 Q. Okay. So I'm going to write on  
47:3 this other leg "animal studies."  
47:4 And then the last one was what,  
47:5 sir?  
47:6 A. Mechanistic studies.  
47:7 Q. Okay.  
47:8 A. Mechanisms.  
47:9 Q. And what are you looking at in  
47:10 mechanistic studies?  
47:11 A. You're looking at -- as a  
47:12 general rule you're looking at things that  
47:13 happen at the level of the cell, inside the  
47:14 cell, that will start or enhance the  
47:15 carcinogenic process.  
47:16 Q. All right. So we're going to  
47:17 call those cell studies; is that okay?  
47:18 A. They're not always cell  
47:19 studies.  
47:20 Q. Okay.  
47:21 A. I'd call them mechanism  
47:22 studies.  
47:23 Q. All right. All right. So just  
47:24 generally speaking, sir, from a scientific

47:25 perspective what is the requirement of  
48:1 looking at all three of these legs?  
48:2 A. Well, they all contribute to a  
48:3 general decision about whether or not a  
48:4 chemical can cause cancer. Epidemiology is a  
48:5 very important part of this, but seldom by  
48:6 itself does epidemiology give you this is  
48:7 clearly a cause.  
48:8 Animal studies are an important  
48:9 part of this, but seldom by themselves do  
48:10 they give you a definitive answer that this  
48:11 can cause cancer in humans, and the same with  
48:12 mechanisms. Together they give you a better  
48:13 picture of the overall potential, and you can  
48:14 make a better overall decision.  
48:15 Q. Okay. So what I want to do  
48:16 today is really focus in on animal studies,  
48:17 mechanism studies and epidemiology.  
48:18 Okay?  
48:19 A. Okay.  
48:20 Q. And just for your benefit, the  
48:21 jury will have heard testimony from Dr. Beate  
48:22 Ritz.  
48:23 Do you know who she is?  
48:24 A. Yes.  
48:25 Q. And what is her specialty?  
49:1 A. Epidemiology.  
49:2 Q. Okay. So they're going to have  
49:3 heard a lot about epidemiology, so we're not  
49:4 going to spend much time on that. I don't  
49:5 want to, you know, repeat ourselves.  
49:6 But I want to focus primarily  
49:7 on these first two, the animal studies and  
49:8 the cell studies.  
49:9 Okay?  
49:10 A. Okay.  
49:11 Q. All right. Let's start off  
49:12 with these animal studies.  
49:13 What is an animal study?  
49:14 A. So an animal study is -- for

49:15 cancer, specifically for cancer, is you take  
49:16 an animal, you take a group of animals, a  
49:17 large number sometimes, and you expose them  
49:18 to the chemical that you're interested in for  
49:19 a good part of their lifetime, and you see if  
49:20 they have more cancer in them than a group of  
49:21 animals that are not exposed. So you can  
49:22 make a comparison and see if the chemical  
49:23 causes cancer in the animal.

49:24 Q. I understand actually in  
49:25 preparation for your testimony today, you  
50:1 helped put together a PowerPoint walking  
50:2 through this; is that right?

50:3 A. That's correct.

50:4 Q. Okay. So let's take a look at  
50:5 that PowerPoint. It's Exhibit 881. If you  
50:6 go to the computer.

50:7 So, sir, how are you physically  
50:8 doing? Is this a good time for a break or do  
50:9 you want to --

50:10 A. I'm fine.

50:11 Q. Okay. Great.

50:12 So let's start off at the top  
50:13 here. We have this first slide. It says  
50:14 "Rodent Studies."

50:15 Do you see that?

50:16 A. Yes, I see it.

50:17 Q. And the first bullet point  
50:18 reads, "Humans share 95 percent DNA with  
50:19 rodents."

50:20 What does that mean?

50:21 A. Well, it's just a reminder of  
50:22 the fact that humans and rodents have a lot  
50:23 of the similar biological pathways that make  
50:24 up our lives. We're both mammals, and so  
50:25 much of what goes on at the cellular level in  
51:1 rats and mice are very similar, if not almost  
51:2 identical in some cases, to what happens in  
51:3 humans.

51:4 All of that is controlled by

51:5 DNA and -- mitochondrial DNA and other  
51:6 things, but it's all controlled by our  
51:7 genetic heritage. And the genetic heritage  
51:8 of the mouse and the human, rodents and  
51:9 humans, is very close.

51:10 Q. "Since humans share similar  
51:11 pathways for toxin eradication," what is that  
51:12 referring to?

51:13 A. Well, when you -- when you  
51:14 ingest anything, be it a chemical or be it  
51:15 food or whatever it is, your body absorbs it,  
51:16 it distributes it throughout the body, it  
51:17 metabolizes it, meaning the molecular systems  
51:18 in the cells in the body break it down into  
51:19 things the cells can either use or get rid of  
51:20 because they don't want it around, and then  
51:21 the body eliminates it.

51:22 So this whole process of  
51:23 absorption, distribution, metabolism and  
51:24 elimination, there are great similarities  
51:25 between rodents and humans in those  
52:1 processes.

52:2 Q. And how is that relevant when  
52:3 you're looking at the issue of, for example,  
52:4 cancer?

52:5 A. Well, for a chemical to cause  
52:6 cancer, it has to be absorbed. It has to be  
52:7 distributed to the source of the cancer.  
52:8 Sometimes it needs to be changed into a new  
52:9 chemical that will cause the cancer, so  
52:10 that's metabolism. And to prevent the  
52:11 cancer, it has to be eliminated. It has to  
52:12 be gotten rid of somehow.

52:13 So it's very important to the  
52:14 idea that a chemical can cause cancer in  
52:15 humans. If it's not absorbed, it can't cause  
52:16 cancer in humans. If it's not distributed to  
52:17 the site where the cancer occurs, it's not  
52:18 causing that cancer.

52:19 If the cancer is caused by a

52:20 specific metabolite, and in humans that  
52:21 metabolite is not formed, it can't cause the  
52:22 cancer, et cetera.

52:23 Q. It says here, "a standard model  
52:24 for studying cancer." What does that refer  
52:25 to?

53:1 A. So typically, regulatory  
53:2 agencies will request corporations that want  
53:3 a chemical to go into the environment as a  
53:4 pesticide or even as pharmaceuticals, they'll  
53:5 request that they do a study for safety. And  
53:6 one of the safety studies they request is an  
53:7 animal cancer study. And these rodents are  
53:8 the typical way of doing it.

53:9 A. typical animal study includes  
53:10 rats and mice, males and females, in multiple  
53:11 groups for the life of the animals.

53:12 Q. It says, "Use specially bred  
53:13 mice and rats." And if you look to the right  
53:14 we have, it looks like, CD-1 mouse and Wistar  
53:15 rats.

53:16 What is that referring to?

53:17 A. So whenever you do science, you  
53:18 want to make sure you document exactly what  
53:19 you do. If I went outside and collected a  
53:20 bunch of mice from around the dumpster in the  
53:21 back of the hotel and did a study with them,  
53:22 it would be an interesting, valid study about  
53:23 how a chemical might affect mice in their  
53:24 normal environment, but nobody could repeat  
53:25 it unless they came and caught the same  
54:1 animals behind the same dumpster at the same  
54:2 hotel.

54:3 So what we try to do in science  
54:4 is we have these strains of rats and mice,  
54:5 even substrains. We label them. We breed  
54:6 them. We take care to try to keep them  
54:7 genetically the same over multiple years so  
54:8 that if I do a study with a CD-1 mouse and  
54:9 somebody else wants to repeat what I did,

54:10 they can get a CD-1 mouse, do the same study  
54:11 and hopefully get the same answer. That way  
54:12 we can see that the science is consistent,  
54:13 and it's stronger if you can repeat it.  
54:14 So we maintain these different  
54:15 strains of rats and mice to make sure it's  
54:16 repeatable.  
54:17 Q. All right. The next one says,  
54:18 "Mouse models are commonly used to develop  
54:19 drugs for lymphoma treatments."  
54:20 What is that referring to?  
54:21 A. So as I mentioned before, when  
54:22 you're developing a drug or something, you do  
54:23 safety assessments, and you want to make sure  
54:24 that drug is safe before you give it to  
54:25 people. But as another part, you want to  
55:1 make sure it's going to work. And you try to  
55:2 do that before you start giving it to people.  
55:3 There's a lot of work done with  
55:4 human cells, but typically they will also  
55:5 find a similar disease in a model, in this  
55:6 case for lymphoma. Malignant lymphoma seen  
55:7 in the mouse is a very similar disease to  
55:8 B-cell lymphomas which are a subset of  
55:9 non-Hodgkin's lymphomas seen in humans.  
55:10 And so if you have a mouse  
55:11 model that spontaneously, just because it  
55:12 lives, gets a lot of malignant lymphomas,  
55:13 then you can use that and start giving it  
55:14 your new treatment and see if you reduce the  
55:15 lymphomas arising in those animals or get rid  
55:16 of them after they've started. And if that  
55:17 works, then you've got a potential drug for  
55:18 using in humans.  
55:19 So you create a model of the  
55:20 drug -- of the disease that you can give the  
55:21 drug to to see if it's going to work. The  
55:22 mouse is a good model for lymphomas in  
55:23 humans.  
55:24 Q. All right. So I understand you

55:25 have developed a sort of walk-through of a  
56:1 typical rodent study, and we're going to  
56:2 focus on a mouse here.  
56:3 Okay?  
56:4 A. Okay.  
56:5 Q. Okay. So the first step, it  
56:6 says, "Mice are placed in groups where they  
56:7 are treated identically."  
56:8 What does that refer to?  
56:9 A. So when you're going to do one  
56:10 of these studies, you don't want to do it  
56:11 with one mouse, obviously, because it's not  
56:12 enough information that one mouse got cancer  
56:13 or didn't get cancer. So you have groups of  
56:14 mice that you work with.  
56:15 And you want to treat them  
56:16 identically because -- so I'm going to take  
56:17 the mice and I'm going to break them into  
56:18 groups. And some groups are going to get  
56:19 exposed to my chemical that I'm worried about  
56:20 and some are not going to be exposed.  
56:21 And what I want to be able to  
56:22 do is compare the exposed groups to the  
56:23 nonexposed group. But in order to do that  
56:24 clearly, without any problem, I have to make  
56:25 sure they're all treated exactly the same.  
57:1 Because if I give my unexposed group, say,  
57:2 bottled water and I give my exposed group --  
57:3 besides the chemical, I give them tap water  
57:4 straight out of the pipe, then I can't tell  
57:5 if the cancers are due to the chemical or the  
57:6 differences in the water.  
57:7 So I make sure that everything  
57:8 in these animals' lives are identical except  
57:9 for the exposure I'm interested in.  
57:10 Q. Okay. And it says each group  
57:11 typically contains 50 males and 50 females.  
57:12 What does that refer to, and  
57:13 what's the significance of 50?  
57:14 A. Well, 50 is a practical

57:15 limitation. These studies are fairly  
57:16 expensive to do. The more animals you have,  
57:17 the more expensive they get.  
57:18 Based on work I did in my  
57:19 thesis and other work and work by other  
57:20 people, 50 seems to be a good number for  
57:21 being sensitive enough to see things that  
57:22 might occur and not so small that you  
57:23 wouldn't see them if they're there.  
57:24 Q. Okay. And what's the  
57:25 significance of having males and females?  
58:1 A. Ah, yes. Well, males and  
58:2 females can respond differently to chemicals,  
58:3 if nothing else. The targets can be  
58:4 different. Males can have testicular cancer,  
58:5 females can't. Females can have uterine  
58:6 cancer; males can't. Females tend to get  
58:7 mammary tumors. Males tend to not get those  
58:8 breast cancers that women can get. In the  
58:9 animals it's mammary tumors, males or  
58:10 females, because of tissue size and different  
58:11 tissue functions.  
58:12 But even in typical organs like  
58:13 livers and lungs, males and females tend to  
58:14 get different sensitivities to different  
58:15 exposures. So you always break it down and  
58:16 look at both males and females so you can  
58:17 look at the entire human population, not just  
58:18 one gender.  
58:19 Q. Okay. So how many different  
58:20 treatment groups are there?  
58:21 It says here there are four  
58:22 treatment groups, typically 400 mice.  
58:23 What is that referring to?  
58:24 A. Well, typically you take 200  
58:25 males and 200 females, 50 per group. You  
59:1 break them into four separate groups. One of  
59:2 the group gets no chemical, and the other  
59:3 groups get the exposure to whatever chemical  
59:4 you're interested in.

59:5 And you have a group of females  
59:6 that get no chemical, a group of males that  
59:7 get no chemical. The same on the exposure  
59:8 groups.  
59:9 Q. And here -- well, let's use for  
59:10 this example glyphosate.  
59:11 Okay?  
59:12 A. Okay.  
59:13 Q. All right. So how then do we  
59:14 determine what dose we give -- so I  
59:15 understand the ones on the left don't get  
59:16 glyphosate.  
59:17 A. Right.  
59:18 Q. The three groups on the right,  
59:19 they do.  
59:20 How do you determine which dose  
59:21 they get?  
59:22 A. So it's not random. It's a  
59:23 very serious part of the design of a cancer  
59:24 bioassay. We're interested in protecting  
59:25 human health. That's the purpose of doing  
60:1 this. The purpose is not to protect the  
60:2 health of rats and mice from cancer. The  
60:3 goal is to protect human health.  
60:4 And you might allow a  
60:5 beneficial product onto the market if the  
60:6 cancer risk was low enough. So typically  
60:7 regulatory agencies will look for a risk  
60:8 that's below one in a hundred thousand or one  
60:9 in a million and say, "oh, that's a very  
60:10 small risk, and the benefit from this thing  
60:11 is bigger than the risk, so we're going to  
60:12 allow it in society."  
60:13 But you can't measure one in a  
60:14 hundred thousand. In order for me to be able  
60:15 to see that, I'd have to have 500,000 mice or  
60:16 rats.  
60:17 So instead, you -- you assume  
60:18 that as the exposure gets bigger, the  
60:19 probability of getting cancer gets bigger.

60:20 So there's going to be a dose that gives you  
60:21 1 in a hundred thousand in the mice, but  
60:22 maybe ten times that dose will give you 1 in  
60:23 10,000. And ten times that dose will give  
60:24 you 1 in a thousand. Ten times that, 1 in a  
60:25 hundred. Ten times that, 1 in 10.

61:1 And so what you try to do in an  
61:2 animal bioassay is you get the highest dose  
61:3 you possibly can in hopes that if this causes  
61:4 cancer, you'll be in this range of 1 in 20, 1  
61:5 in 30 probability of getting cancer so you  
61:6 can actually see it in your 50 animals.

61:7 So how do you find that dose?

61:8 Q. Let me ask you a question about  
61:9 that. So it says here the highest dose is  
61:10 usually the maximum tolerated dose.

61:11 What is that?

61:12 A. So that's the dose you try to  
61:13 find, but of course you can't be certain. So  
61:14 you have to get indications in advance of  
61:15 what that will be.

61:16 So what you typically do is a  
61:17 90-day study. That's the same basic outline,  
61:18 controls, multiple treated group, smaller  
61:19 numbers of animals and a lot more groups,  
61:20 usually six or so, maybe seven groups, and  
61:21 what you do is you expose them for 90 days.

61:22 And during that 90 days, you  
61:23 look to see if the exposure is harming them  
61:24 in any way, and I mean any way. You look for  
61:25 changes in body weight. You look for  
62:1 disorientation in the animals. You look for  
62:2 them eating less food or drinking less water.  
62:3 You look inside of them at the end and see if  
62:4 there's damage to tissues or organs.

62:5 What you're trying to find is  
62:6 the highest dose that in 90 days does not  
62:7 cause any harm at all to the animals that you  
62:8 can see, and that dose is the maximum  
62:9 tolerated dose. And then you use that dose

62:10 for the entire two years in the longer-term  
62:11 experiment.  
62:12 Q. But, I mean, Doctor, if you're  
62:13 using this maximum tolerated dose, I mean,  
62:14 doesn't that sort of make it no longer  
62:15 relevant to humans?  
62:16 A. No, of course not. In the long  
62:17 term, if the -- if -- if the mechanisms by  
62:18 which the cancer occurs at that high dose are  
62:19 the same mechanisms that work at low doses,  
62:20 then, in fact, it is relevant.  
62:21 And the whole purpose of doing  
62:22 the 90-day study is to try to avoid any other  
62:23 mechanisms that might not operate at the  
62:24 lower doses. So you're trying to avoid that  
62:25 by looking for toxicity in advance of doing  
63:1 the studies.  
63:2 But in most cases, it's  
63:3 relevant to the lower exposure that people  
63:4 would see.  
63:5 Q. So that gets us to the high  
63:6 dose.  
63:7 What about the rest of the  
63:8 doses, the low dose and mid dose?  
63:9 A. Well, there you're looking at  
63:10 fractions of the high dose, some percentage,  
63:11 because you want to see what happens at lower  
63:12 and lower doses. The idea would be that  
63:13 you're going to see some sort of pattern in  
63:14 those exposures, and that pattern also tells  
63:15 you something about further down that dose  
63:16 scale into the range where humans are  
63:17 exposed.  
63:18 The actual doses that are  
63:19 chosen are somewhat subjective, but most  
63:20 people work from the algorithm I did in my  
63:21 thesis, which would put you at about  
63:22 somewhere between one-tenth to one-third of  
63:23 the maximum dose for the lowest dose, and  
63:24 between one-third and one-half of the maximum

63:25 dose for the middle dose.  
64:1 Most of the studies we're  
64:2 looking at for glyphosate have one-tenth of  
64:3 the maximum tolerated dose at the lowest  
64:4 dose, one-third of the maximum tolerated dose  
64:5 at the mid dose.  
64:6 Q. Okay. So we've gone through  
64:7 how you set the doses for the groups, for the  
64:8 mice that are going to get glyphosate.  
64:9 Okay?  
64:10 How long does this sort of  
64:11 process run for?  
64:12 A. The whole bioassay and the  
64:13 start-up with the 90-day study and everything  
64:14 else?  
64:15 Q. Well, no, that's -- fair  
64:16 enough. That's probably too much to ask.  
64:17 How long does the study go for  
64:18 for the mice that you're studying?  
64:19 A. Once you start the study, it  
64:20 usually goes for two years, although some  
64:21 mice studies now are done for 18 months,  
64:22 depending on the strain of mouse and how long  
64:23 it naturally lives, but that's -- it's  
64:24 generally two years.  
64:25 Q. And how old are the mice at the  
65:1 beginning of the study?  
65:2 A. Typically the mice and the rats  
65:3 are six weeks old when they start the study  
65:4 because that's when they have just reached  
65:5 puberty. So you -- these studies were  
65:6 originally thought of as adult exposure  
65:7 studies, so you start when the animal reaches  
65:8 puberty, which is when people might start  
65:9 working in a job, and you take it for their  
65:10 whole lifespan.  
65:11 Q. Now, maybe -- I don't know if  
65:12 you know this, but if -- you have two years  
65:13 for a CD-1 mouse, right?  
65:14 How old would a 2-year-old

65:15 mouse be in equivalent human years?  
65:16 A. That varies by strain and  
65:17 species, but let's just say approximately 65  
65:18 to 70 years old.  
65:19 Q. Well, then, sir --  
65:20 A. In humans.  
65:21 Q. -- what if you have a cancer  
65:22 that, you know, comes out at later ages, like  
65:23 in the 70s or 80s? Would these mice studies  
65:24 capture those?  
65:25 A. If the -- if the thing you're  
66:1 looking at, the chemical agent you're looking  
66:2 at, shortens the time to cancer, yes, you  
66:3 would see it, because it would come before  
66:4 that 70 time point.  
66:5 If all the chemical does is  
66:6 increase the probability of getting that  
66:7 cancer in that time frame, then you wouldn't  
66:8 see it.  
66:9 Q. Okay. So we run the study for  
66:10 two years, and at the end of two years, what  
66:11 do we do? What do we look for in mice?  
66:12 A. So typically, in almost all the  
66:13 bioassays, at the end of -- at the end of the  
66:14 study, end of two years, they sacrifice all  
66:15 of the animals. They kill them humanely.  
66:16 And every animal, including the ones who have  
66:17 died earlier than the two years, just from  
66:18 natural causes during the course of the  
66:19 study, all of those animals are looked at  
66:20 very carefully. Every organ is examined by a  
66:21 pathologist who looks for tumors, little  
66:22 lumps and bumps in the organs.  
66:23 In addition, they take and --  
66:24 take slices of each tissue, very thin slices,  
66:25 put it on a microscope slide, and they look  
67:1 at them under the microscope to see if they  
67:2 can see cellular changes that look like  
67:3 cancer. So they examine very carefully all  
67:4 over the animals.

67:5 Q. And when they're taking these  
67:6 slices from the various animals, are they the  
67:7 same sort of portions of the organ for each  
67:8 animal, or does it change?  
67:9 A. Just like the feeding and just  
67:10 like everything else, you have protocols that  
67:11 specify exactly what slices you are to take  
67:12 in the animals, exactly what angle and across  
67:13 what part of the tissue and organ, yes.  
67:14 They're very much uniform.  
67:15 Q. What if there is a tumor in  
67:16 another part that wasn't part of the typical  
67:17 slicing?  
67:18 A. If the tumor is big enough that  
67:19 you can see it or feel it, there's a lump or  
67:20 a bump there, they will take a slice through  
67:21 that, and that's part of the protocol.  
67:22 But if it's smaller than that,  
67:23 what we would call microscopic, the only way  
67:24 you'd see it is under a microscope, then, no,  
67:25 there's no way you'd ever see it. Because  
68:1 you don't take a slice there, you just won't  
68:2 see it.  
68:3 Q. All right. So we have on the  
68:4 slides here, we have some red circles that  
68:5 have popped up.  
68:6 What are those supposed to  
68:7 reflect, sir?  
68:8 A. Well, that simply is intended  
68:9 to show you what you might see in a typical  
68:10 bioassay for a typical single cancer type.  
68:11 You would have an animal that has the cancer  
68:12 or doesn't have the cancer.  
68:13 Here, the little rats or  
68:14 mice -- these are mice -- that are circled  
68:15 with the red are mice that had a particular  
68:16 cancer. And what you're looking at here  
68:17 are -- for example, in the low dose group,  
68:18 these are 50 mice, and 2 of the 50 mice had  
68:19 tumors.

68:20 So that's sort of the basis for  
68:21 the analysis, 2 out of 50 animals with a  
68:22 specific tumor.

68:23 Q. Now, when you say two tumors,  
68:24 is that two tumors of a specific type or just  
68:25 two tumors generally?

69:1 A. Generally it's two tumors of a  
69:2 specific type. You analyze the data for each  
69:3 tumor type.

69:4 The argument is that the tumors  
69:5 are generally independent of each other, and  
69:6 you're interested in what this may mean to  
69:7 the human population. So you might have a  
69:8 chemical -- there are a number of chemicals  
69:9 out there that hit multiple organs and with  
69:10 multiple types of cancer. So I can think of  
69:11 one now that has five or six different cancer  
69:12 sites.

69:13 Each of those cancer sites are  
69:14 of concern to human populations, and so you  
69:15 treat them each separately rather than just  
69:16 did this animal get a cancer or not. No.  
69:17 This animal got a lung cancer, it got a liver  
69:18 cancer, it got an adrenal cancer, and so we'd  
69:19 be worried about all of those.

69:20 Q. And so when we look at all the  
69:21 various tumors that appear in the treatment  
69:22 groups, we have this slide here, and I  
69:23 actually think there's a typo. In the mid  
69:24 dose group it says 3 out of 50. It probably  
69:25 should say two. I only see two circles  
70:1 there.

70:2 Do you see that?

70:3 A. Yeah, that happens.

70:4 Q. Okay. In any event, what are  
70:5 you doing when you're looking at the various  
70:6 tumors in the group? What are you looking  
70:7 for?

70:8 A. Well, there are two ways to  
70:9 analyze this type of data. One way to

70:10 analyze the data is to compare the low dose  
70:11 group to the control, the mid dose group to  
70:12 the control, and the high dose group to the  
70:13 control.

70:14 So here you would compare, for  
70:15 the low dose, 2 out of 50 against 1 out of 50  
70:16 in the control and ask yourself, is this  
70:17 unusual, under the assumption that there  
70:18 actually is no carcinogenic risk to this --  
70:19 to this -- for this chemical. So if there's  
70:20 no risk for this chemical, would a difference  
70:21 between 1 out of 50 versus 2 out of 50 be  
70:22 important.

70:23 And the answer to that question  
70:24 would be no in this case.

70:25 But when you look at the high  
71:1 dose versus control, 5 out of 50 versus 1 out  
71:2 of 50, that 5 out of 50 may be very  
71:3 different. And so there's statistics that  
71:4 allows you to ask that question and calculate  
71:5 the probability that you would see 5 out of  
71:6 50 versus 1 out of 50, if truth was there's  
71:7 no effect going on in this population.

71:8 So that's one way.

71:9 The other way to analyze the  
71:10 data is if you look at this, you've got low  
71:11 dose, mid dose, high dose, and the question  
71:12 would be a slightly different question: As  
71:13 you increase the dose, is the risk of getting  
71:14 cancer increasing.

71:15 And so there you look to see  
71:16 if -- if I drew a line through all of these  
71:17 data, is that line going up as the dose goes  
71:18 up or is it, in fact, flat.

71:19 And here you do the same thing  
71:20 you did with the pairwise test. Here you  
71:21 do -- you ask yourself: If truth is there's  
71:22 nothing going on, truth is it's perfectly  
71:23 flat, what's the probability that I see this  
71:24 slope.

71:25 And if that probability is very  
72:1 small, then you reject the idea that it's  
72:2 flat in favor of the idea that there is  
72:3 indeed an increasing risk with increasing  
72:4 dose.

72:17 - 77:1

**Portier, Christopher 02-21-2019 (00:04:21)**

CP1\_SS\_01.20

72:17 Q. So, sir, you said 5, but I  
72:18 believe here in the high dose group there's  
72:19 4.

72:20 Do you see that?

72:21 A. That is correct, and thank you  
72:22 for correcting me on that. And I'm pretty  
72:23 sure 4 out of 50 versus 1 out of 50 is not  
72:24 going to be statistically significant in  
72:25 these data set.

73:1 Q. Okay. This whole process,  
73:2 though, where you have these 50 mice per  
73:3 group, where you're looking at the slope of  
73:4 the lines and comparing it statistically to  
73:5 the control, is that -- is that process  
73:6 something that you actually helped develop  
73:7 when you did your Ph.D.?

73:8 A. Some of it. Most of the simple  
73:9 pairwise comparisons of one group versus --  
73:10 that was known from the 1930s. Fisher's  
73:11 exact test has been around a very long time.  
73:12 Trend tests, which look at  
73:13 these slopes, that's something I worked on  
73:14 post-Ph.D. my first few years at NIH where I  
73:15 did a lot of work in that area.

73:16 Q. And this approach that you  
73:17 developed in your work, is it the approach  
73:18 that's still used today?

73:19 A. It is the standard way of  
73:20 analyzing these types of studies by the US  
73:21 National Toxicology Program and many  
73:22 toxicology programs around the world.

73:23 Q. All right. So I want to get  
73:24 real here. We've talked about a hypothetical  
73:25 experiment. Let's talk about an actual study

74:1 on glyphosate to give -- to explain to the  
74:2 jury how this actually works out.  
74:3 Okay?  
74:4 A. Okay.  
74:5 Q. I want to draw your attention  
74:6 to the Wood study from 2009.  
74:7 Okay?  
74:8 A. Okay.  
74:9 Q. Are you familiar with that  
74:10 study?  
74:11 A. Yes, I am.  
74:12 Q. All right. And what are we  
74:13 looking at here on the slide?  
74:14 A. So this is bigger mice, and  
74:15 you've only brought in the mice that actually  
74:16 have the tumor. So here you had three dose  
74:17 groups and one control group. The control  
74:18 saw no malignant lymphomas in 50 animals.  
74:19 Actually -- is it 50 or 51? I don't remember  
74:20 the study, but it's either 50 or 51. The low  
74:21 dose saw one animal with the tumor, the mid  
74:22 dose saw two animals with the malignant  
74:23 lymphoma, and the high dose saw five animals  
74:24 out of 50 with the malignant lymphoma.  
74:25 Q. So let's break what this is  
75:1 showing.  
75:2 So in this study on glyphosate,  
75:3 what, if any, is the significance of not  
75:4 having a single tumor or a single malignant  
75:5 lymphoma in the control group?  
75:6 A. It just means that in this  
75:7 particular case, which is an 18-month study,  
75:8 I believe, of -- in the mice, that as a  
75:9 matter of spontaneously appearing tumors,  
75:10 none have appeared of this type in these  
75:11 males in this study.  
75:12 Q. Okay. So then we have one in  
75:13 the low dose, two in the mid dose and five in  
75:14 the high dose. What -- what's the  
75:15 significance of that?

75:16 A. Well, the pattern's important  
75:17 here. You can see that as the exposure is  
75:18 increasing, the number of animals with the  
75:19 tumor is increasing out of a constant 50. So  
75:20 the proportion of animals with the tumor has  
75:21 increased, and that's very important to look  
75:22 at.

75:23 And at the highest dose, you  
75:24 have a fairly big number of animals with the  
75:25 tumor relative to the controls.

76:1 Q. And so you've plotted them out  
76:2 here, it looks like, in a bar graph.

76:3 Do you see that?

76:4 A. Yes.

76:5 Q. And if you go to the last  
76:6 slide, it reads, "Dose response or trend."  
76:7 What does that mean?

76:8 A. Well, again, that's -- now  
76:9 looking at the data and asking the question,  
76:10 do these data indicate a concern for  
76:11 malignant lymphomas, did this chemical cause  
76:12 malignant lymphomas in these mice in this  
76:13 study, that's the question you have to first  
76:14 ask yourself.

76:15 And there you do your  
76:16 statistical tests, the pairwise test, each  
76:17 group against control, and the trend test,  
76:18 like I said before. And here in the trend  
76:19 test, you're looking to see if that line that  
76:20 you're looking at has a slope. The slope of  
76:21 the line is the angle at which it climbs.  
76:22 You're asking is that slope greater than  
76:23 zero. A zero slope is a flat line. Any  
76:24 slope that's bigger than that is a positive  
76:25 line. You're testing whether it's not zero  
77:1 or not.

77:2 - 80:18

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CP1\_SS\_01.67

77:2 In this case, it is  
77:3 significantly different from zero. So this  
77:4 shows a significant increase in the

77:5 proportion of animals with tumor as the dose  
77:6 increases.  
77:7 Q. So what does this study show  
77:8 you when it comes to lymphoma?  
77:9 A. If this is the only study I  
77:10 have, it shows me that this study, for these  
77:11 animals, it's fairly clear that glyphosate is  
77:12 causing malignant lymphomas.  
77:13 Q. Well, hold on, Doctor. You say  
77:14 glyphosate's causing malignant lymphomas.  
77:15 How do you know these tumors  
77:16 wouldn't have just happened naturally, just  
77:17 because mice get tumors? How do you know  
77:18 it's not that?  
77:19 A. Well, that's the whole purpose  
77:20 of the study, isn't it? I've controlled  
77:21 everything else in the study. So all of  
77:22 these mice are being treated exactly the same  
77:23 way.  
77:24 So if it were spontaneous, if  
77:25 it were just random chance, it's unlikely  
78:1 they would line up like this, and that's what  
78:2 the statistics is telling you. That's why  
78:3 you do a statistical analysis. It's  
78:4 evaluating the probability that you see this  
78:5 sort of pattern by chance.  
78:6 Q. What is the -- what is the  
78:7 probability that you'd see something like  
78:8 this by chance?  
78:9 A. Well, if I remember the study  
78:10 correctly, I think this is .007 probability,  
78:11 which is about 7 in 1,000 chance that this  
78:12 arises by chance.  
78:13 You can also go look at --  
78:14 these are CD-1 mice, a certain substrain.  
78:15 You can look at other experiments that have  
78:16 been done in this same mouse strain, and  
78:17 every one of those other cancer experiments  
78:18 has a control group which gets no exposure.  
78:19 And so you can look at all those control

78:20 groups from the other studies and also see  
78:21 how much variation there is in the control  
78:22 response, and that can tell you also  
78:23 something about the probability of seeing  
78:24 this type of response.

78:25 Q. Well, you said this is an  
79:1 18-month study; is that right?

79:2 A. That's correct.

79:3 Q. So for an 18-month study for  
79:4 animals, these CD-1 mice that are not exposed  
79:5 to any chemicals, what is the rate that they  
79:6 spontaneously get lymphoma?

79:7 A. I do look that up, and it's  
79:8 probably about 1 in 50.

79:9 Q. Okay.

79:10 A. On average, 1 in 50.

79:11 Q. So you'd expect to see 1 in 50,  
79:12 and in this high dose you're seeing 5 of 50;  
79:13 is that right?

79:14 A. Correct.

79:15 Q. What's the significance of  
79:16 that?

79:17 A. Well, that's, again, what the  
79:18 statistics is telling you. The statistics is  
79:19 telling you the significance of it is you  
79:20 stand only a 7 in 1,000 part chance of ever  
79:21 seeing this type of pattern, given do you  
79:22 believe that there was nothing there.

79:23 Q. All right. We're going to take  
79:24 a break in a second. I really appreciate  
79:25 your endurance here.

80:1 I want to -- before we take a  
80:2 break, though, I want to just cover generally  
80:3 whether or not there are any guidelines that  
80:4 govern sort of how we look at animal studies.

80:5 A. There are many guidelines. The  
80:6 National Toxicology Program has guidelines.  
80:7 The EPA has guidelines. The European Food  
80:8 Safety Authority has guidelines. There's an  
80:9 international organization called the

Page/Line

Source

ID

80:10 Organization of Economic and Cooperative  
80:11 Development, OECD. OECD has guidelines.  
80:12 Most people follow all of these  
80:13 guidelines. And, yeah, they're there for not  
80:14 only how to design the study, how to run the  
80:15 study, how to do the pathology at the end of  
80:16 the study, but there's also rules on how to  
80:17 analyze the data from the study and how to  
80:18 interpret these studies.

80:19 - 80:21

**Portier, Christopher 02-21-2019 (00:00:05)**

CP1\_SS\_01.21

80:19 Q. All right. Look at Exhibit 388  
80:20 in your binder.

80:21 A. Okay.

81:19 - 82:2

**Portier, Christopher 02-21-2019 (00:00:18)**

CP1\_SS\_01.22

81:19 Q. And does this document go over  
81:20 some of the standard scientific approaches  
81:21 for looking at long-term animal  
81:22 carcinogenicity studies?

81:23 A. Yes, it does.

81:24 Q. All right. Let's take a look  
81:25 at those standards very quickly. It's a page  
82:1 ending in 2-21.

82:2 A. Okay.

82:7 - 82:15

**Portier, Christopher 02-21-2019 (00:00:22)**

CP1\_SS\_01.23

82:7 Q. All right. The very bottom of  
82:8 the page, Section 2.2.1.4, assessment of  
82:9 evidence of carcinogenicity from long-term  
82:10 animal studies. It reads, "In general,  
82:11 observation of tumors under different  
82:12 circumstances lends support to the  
82:13 significance of the findings for animal  
82:14 carcinogenicity."

82:15 Sir, do you agree with that?

82:17 - 82:21

**Portier, Christopher 02-21-2019 (00:00:04)**

CP1\_SS\_01.24

82:17 THE WITNESS: Yes.

82:18 QUESTIONS BY MR. WISNER:

82:19 Q. Can you explain what that  
82:20 means?

82:21 A. Well, it -- it --

82:24 - 83:18

**Portier, Christopher 02-21-2019 (00:00:38)**

CP1\_SS\_01.26

82:24 THE WITNESS: The -- it just  
82:25 says -- I mean, it's -- it's a  
83:1 statement that is so obvious, it's  
83:2 hard to even say what it means.  
83:3 I have to observe tumors in an  
83:4 animal study to be able to decide if  
83:5 tumors are caused in the animal study.  
83:6 So the observation of those tumors  
83:7 contributes to the decision about  
83:8 whether you have a significant finding  
83:9 of animal carcinogenicity in the  
83:10 animals.

83:11 QUESTIONS BY MR. WISNER:

83:12 Q. Okay. Great.

83:13 So the next sentence reads,

83:14 "Significance is generally increased by the  
83:15 observation of more of the factors listed  
83:16 below."

83:17 Do you see that?

83:18 A. Yes.

83:25 - 90:20

**Portier, Christopher 02-21-2019 (00:06:14)**

CP1\_SS\_01.26

83:25 Q. And if we turn to the next  
84:1 page, it has those factors listed.  
84:2 Do you see that?

84:3 A. Oh, sorry, they're at the top  
84:4 of this page.

84:5 Yes, I see that.

84:6 Q. Okay. Great.

84:7 I want to quickly run through  
84:8 these. First one, it says, "Uncommon tumor  
84:9 types."

84:10 What does that refer to?

84:11 A. So when you're doing an animal  
84:12 study, certain tumors almost never appear in  
84:13 animals. The classic example for me is  
84:14 fluoride. The National Toxicology Program  
84:15 did a study of fluoride to see if it caused  
84:16 cancer in the animals.

84:17 In two of the high exposure

84:18 rats in that study, we saw what's called an

84:19 osteosarcoma, which is a blood -- which is a  
84:20 bone tumor. But it didn't appear in bone; it  
84:21 appeared in the muscle of the rat. So you've  
84:22 got an odd tumor in the muscle of the rat.  
84:23 We'd never seen in 50 rat  
84:24 studies an osteosarcoma in any muscle tissue  
84:25 anywhere. So it's an extremely rare tumor.  
85:1 Almost certainly it arose because of the  
85:2 exposure to the fluoridation.  
85:3 Q. Great.  
85:4 It says, "Tumors at multiple  
85:5 sites."  
85:6 What does that refer to?  
85:7 A. So if I see a chemical that --  
85:8 in the rodents that only causes one tumor in  
85:9 liver, then the chances of this being a  
85:10 rodent carcinogen depends only on that one  
85:11 tumor. But if the chemical comes in and you  
85:12 see tumors in the liver, the lungs, the  
85:13 blood, the kidneys, the brain, then the  
85:14 chances of making a mistake and saying this  
85:15 chemical causes tumors in the animals and it  
85:16 really doesn't is lowered completely.  
85:17 Q. Okay. It says, "Tumors by more  
85:18 than one route of administration."  
85:19 What's that referring to?  
85:20 A. So you do a study and you give  
85:21 the chemical by feed to the animal. I do a  
85:22 study and I have the animal breathe the  
85:23 chemical in. In your study the animal gets  
85:24 liver tumors; in my study the animal gets  
85:25 lung tumors.  
86:1 Perfectly reasonable if it's a  
86:2 point-of-contact carcinogen. That  
86:3 strengthens the finding that this can cause  
86:4 cancer in rodents.  
86:5 Q. It says, "Tumors in multiple  
86:6 species, strains or both sexes."  
86:7 What's the significance of  
86:8 that?

86:9 A. So you do a study in rats; I do  
86:10 a study in mice. You see a cancer in the  
86:11 rat; I see a cancer in the mice. Chances are  
86:12 it's causing cancer in these animals. They  
86:13 may not be the same cancers, but it  
86:14 strengthens the overall call that this  
86:15 chemical can cause cancer in the rats and  
86:16 mice. Males and females, same thing.  
86:17 Q. It says, "Progression of  
86:18 lesions from preneoplastic to benign to  
86:19 malignant."  
86:20 What's that referring to?  
86:21 A. So very few cancers just, boom,  
86:22 pop up and you've got a cancer. They start  
86:23 as premalignant states. The classic example  
86:24 most people know about, skin tumors. Your  
86:25 skin tumor starts as a little bump on your  
87:1 skin. You might get a little worried about  
87:2 it, go to the doctor and they go, "oh, that's  
87:3 a nevi." That's a premalignant skin lesion.  
87:4 And if you don't do something about it, it  
87:5 gets worse and worse and turns into a real  
87:6 skin cancer that is very worrisome. So a lot  
87:7 of tumors arise that way.  
87:8 And when that's the case for  
87:9 those types of tumors, with the chemical you  
87:10 hope to see the progression in the animals.  
87:11 You'd like to see some animals with very  
87:12 early findings, some with beginning of a  
87:13 tumor and some with the real tumors there.  
87:14 Q. Okay. Great.  
87:15 The next one says, "Reduced  
87:16 latency of neoplastic lesions."  
87:17 Before we even get into that,  
87:18 is that really relevant to the glyphosate  
87:19 data?  
87:20 A. Yeah.  
87:21 Q. Okay. So what is it?  
87:22 A. I would have to argue that is  
87:23 relevant to the glyphosate data.

87:24 It's the thing you asked me  
87:25 about before. If it's only occurring after  
88:1 seven -- 70 years of life, will we actually  
88:2 see it.  
88:3 If you reduce the latency, if  
88:4 you reduce the time it takes to get the  
88:5 tumor, you'll see them earlier. And because  
88:6 you're looking at a fixed time, you might see  
88:7 an increase in risk if you look at the right  
88:8 time.  
88:9 Q. Okay. Metastasis, what is  
88:10 that?  
88:11 A. So when you get a real  
88:12 malignant tumor, what's called a malignant  
88:13 tumor, malignant tumors are known -- called  
88:14 that because they tend to invade the  
88:15 surrounding region. Malignant tumors also  
88:16 can metastasize. So pieces of the tumor, one  
88:17 cell, two, three cells, can break off and  
88:18 transport to other parts of the body and  
88:19 continue to become a tumor.  
88:20 So you can have a liver tumor  
88:21 that breaks off one liver cell and it gets  
88:22 caught in the lung, and you get a lung tumor.  
88:23 But the lung tumor is actually a metastasized  
88:24 liver tumor, and you can actually see that.  
88:25 Q. "Unusual magnitude of tumor  
89:1 response," what does that refer to?  
89:2 A. The controls have no tumors,  
89:3 the highest dose has 100 percent of the  
89:4 animal with tumor. That would be an unusual  
89:5 magnitude of response. You see such a  
89:6 massive response, it can't possibly be  
89:7 anything else but the chemical causing that  
89:8 massive response.  
89:9 Q. So a second ago we looked at  
89:10 the Wood study. There was nothing in the  
89:11 control and five in the high dose.  
89:12 Would that be an unusual  
89:13 response?

89:14 A. No.  
 89:15 Q. Okay.  
 89:16 A. That would be usual magnitude  
 89:17 of response.  
 89:18 Q. Gotcha.  
 89:19 "Proportion of malignant  
 89:20 tumors," what does that refer to?  
 89:21 A. There you're just looking at  
 89:22 the whole picture of the animals themselves,  
 89:23 what -- what proportion of the animals in the  
 89:24 whole study have malignant tumors of any  
 89:25 sort.  
 90:1 If that's increasing with  
 90:2 exposure, that's an indication of a concern.  
 90:3 Q. Okay. And the last one here is  
 90:4 "dose-related increases." I think you've  
 90:5 talked about this.  
 90:6 A. Correct.  
 90:7 Q. But can you -- is that what  
 90:8 we're talking about with the dose response?  
 90:9 A. Correct.  
 90:10 Q. Okay. Great.  
 90:11 In the last sentence here in  
 90:12 the first paragraph it says, "In these cancer  
 90:13 guidelines, tumors observed in animals are  
 90:14 generally assumed to indicate that an agent  
 90:15 may produce tumors in humans."  
 90:16 Is that your understanding of  
 90:17 the sort of science behind animal studies?  
 90:18 A. Correct. That's why they were  
 90:19 done in the first place, and I still hold  
 90:20 that's a reasonable assumption.

90:21 **Portier, Christopher 02-21-2019 (00:01:06)**  
 90:21 Q. Okay. And we're going to take  
 90:22 a break in a quick second, but before we do  
 90:23 that, I just want to show the jury these  
 90:24 charts that you've created.  
 90:25 All right, sir. So I want to  
 91:1 show you Exhibit 882. It's on the screen.  
 91:2 Do you see that, sir?

90:21 - 92:11

CP1\_SS\_01.27

91:3 A. Yes, I see it.  
91:4 Q. And just quickly, very quickly,  
91:5 what is this chart?  
91:6 A. These are five mouse studies,  
91:7 and these are the tumors that were  
91:8 significantly elevated in the five mouse  
91:9 studies.  
91:10 Q. Okay. And we also have a  
91:11 similar chart for the various rat studies; is  
91:12 that right?  
91:13 A. Yes. These are one, two,  
91:14 three, four, five, six -- yeah, the seven rat  
91:15 studies.  
91:16 Q. Okay. Great.  
91:17 And just after the break, I'm  
91:18 gonna go through what all these studies show  
91:19 and what this chart means.  
91:20 Does that sound good?  
91:21 A. Okay. Sure.  
91:22 Q. And what I'd like to do is  
91:23 during the break I'd like you to fill in  
91:24 these charts so we can save some time for the  
91:25 jury. All right?  
92:1 A. Okay.  
92:2 Q. All right. Great. Let's take  
92:3 a break.  
92:4 A. Fill it in with the --  
92:5 Q. The markers. I'll give you a  
92:6 marker.  
92:7 A. But significance of the  
92:8 findings --  
92:9 Q. Exactly, and then we'll walk  
92:10 through what your findings are.  
92:11 A. Okay. Good enough.  
93:7 Q. All right, Doctor. Thank you  
93:8 so much for coming back.  
93:9 You had a chance during the  
93:10 break to review those charts; is that right?  
93:11 A. That is correct.

93:7 - 94:21

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93:12 Q. Okay. We're looking at here --  
93:13 this is Exhibit 882, and it has all these  
93:14 black markings on it.  
93:15 Do you see that?  
93:16 A. Yes, I do.  
93:17 Q. Okay. And that black markings,  
93:18 are those -- were those done by you?  
93:19 A. Yes, they were.  
93:20 Q. Okay. And before we move on, I  
93:21 just want to clarify something.  
93:22 A. second ago when we were  
93:23 looking at those EPA guidelines and we were  
93:24 looking at those different factors, are those  
93:25 the same factors that you yourself consider?  
94:1 A. Yes.  
94:2 Q. Okay.  
94:3 A. Of course.  
94:4 Q. And it also occurred to me that  
94:5 you used a couple of words in the previous  
94:6 portion, and I want to make sure we don't  
94:7 have any misunderstandings.  
94:8 The first word is a pretty  
94:9 obvious one, but it's toxicology.  
94:10 What is toxicology?  
94:11 A. It's the branch of science that  
94:12 studies the toxic properties of chemicals in  
94:13 not just humans but anywhere, but generally  
94:14 my area, it's focused on humans.  
94:15 Q. And I'm not sure if the jury  
94:16 can hear, but there's a bit of noise going on  
94:17 in the background.  
94:18 Do you hear that, sir?  
94:19 A. Yes.  
94:20 Q. What is the meeting that's  
94:21 occurring over there?  
94:24 THE WITNESS: It's says  
94:25 "Australian pathologist" on the door.  
95:1 QUESTIONS BY MR. WISNER:  
95:2 Q. Okay. And that -- I asked you

94:24 - 104:4

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95:3 that because I want to ask you, what is  
95:4 pathology?  
95:5 A. Oh. A pathologist -- pathology  
95:6 is -- you might know it better by the word  
95:7 "anatomy." These are people who go into a  
95:8 body and look at it and discern what's going  
95:9 on in that body. They evaluate the pathology  
95:10 of the organs and tissues. Do they have  
95:11 normal -- do they look normal, do they appear  
95:12 to be functioning normal, or do they have  
95:13 manifestations that are different.  
95:14 It's a physical observational  
95:15 science as compared to something like  
95:16 molecular biology that's going in and looking  
95:17 at the chemical reactions within these cells.  
95:18 They're looking at the organization of the  
95:19 cells, the structure of the cells, how they  
95:20 relate to each other in terms of view.  
95:21 Q. And then the last word that was  
95:22 used earlier before the break was something  
95:23 called a bioassay.  
95:24 Well, what is that?  
95:25 A. Bioassay is just another word  
96:1 for an experimental study in toxicology.  
96:2 Basically a bioassay means I'm taking  
96:3 biological material and exposing it to  
96:4 something. So that's humans, animals, cells,  
96:5 and I'm doing an exposure study.  
96:6 Q. And so going back here to  
96:7 Exhibit 882, which is on the screen, all of  
96:8 these different columns, Knezevich and Hogan,  
96:9 Atkinson, Sugimoto, are those bioassays?  
96:10 A. Yes, each one of them is a  
96:11 bioassay.  
96:12 Q. Okay. And each one of these  
96:13 columns here listed, does that refer to what  
96:14 we went over earlier about what a rodent  
96:15 study looks like?  
96:16 A. Correct, each one of these is a  
96:17 rodent study.

96:18 Q. Okay. How many total rodent  
96:19 studies have been done on glyphosate?  
96:20 A. You know, I'm never certain  
96:21 I've got them all, but as of this point, I  
96:22 would count 24 rodent bioassays for cancer.  
96:23 Q. And my understanding is on  
96:24 these charts there's only 12 listed.  
96:25 Do you see that?  
97:1 A. That's correct.  
97:2 Q. Why is that?  
97:3 A. 12 of the studies are  
97:4 documented well enough, presented well  
97:5 enough, done in a way that is consistent with  
97:6 guidelines, well enough that I consider them  
97:7 worthy of part of an evaluation of this sort.  
97:8 The other 12, 10 of them are  
97:9 clearly limited in their interpretation,  
97:10 limited in the way that they presented the  
97:11 data, limited in such a way that I don't  
97:12 think they're adequate for an evaluation of  
97:13 this sort, so I have excluded them. All of  
97:14 those 10 have also been excluded by most of  
97:15 the regulatory authorities out there, so it's  
97:16 not unusual.  
97:17 The remaining two, one of them  
97:18 is a different type of study. It's what's  
97:19 called an initiation/promotion study, and if  
97:20 we want to talk about that, we can get there  
97:21 later.  
97:22 And the last one is an animal  
97:23 bioassay that I just found that looks like  
97:24 it's well conducted but it's really poorly  
97:25 documented, so I can't include it because I  
98:1 don't really know everything about it. So  
98:2 it's not included here.  
98:3 Q. Okay. So looking at these  
98:4 mouse studies, let's kind of walk through  
98:5 what -- what is being said on this chart just  
98:6 so the jury can sort of interpret it and  
98:7 understand it.

98:8 A. Okay.  
98:9 Q. So the first column, it says,  
98:10 Knezevich and Hogan, 1983.  
98:11 What does that refer to?  
98:12 A. So that's the two lead authors  
98:13 of the report from the animal cancer study.  
98:14 1983 is the year.  
98:15 And I've also written 24 in  
98:16 there because this particular study was a  
98:17 24-month study. The animals were exposed to  
98:18 glyphosate for two years.  
98:19 And this is in feed. All of  
98:20 these are feeding studies. The chemical is  
98:21 mixed in with the food, and the animals eat  
98:22 it.  
98:23 Q. Now, if we look at the top  
98:24 here, it says 1983. It says, Atkinson, 1993.  
98:25 Sugimoto, 1997.  
99:1 And what do those years refer  
99:2 to?  
99:3 A. The years in which the reports  
99:4 were completed or submitted to the regulatory  
99:5 agencies. I'm not absolutely certain. But  
99:6 it's the year associated with the information  
99:7 I have on that bioassay.  
99:8 The assays themselves were done  
99:9 before that date.  
99:10 Q. And of these five studies on  
99:11 this chart, which ones -- or which one was  
99:12 done by Monsanto?  
99:13 A. I think Knezevich and Hogan is  
99:14 a Monsanto study, but I'm really not certain  
99:15 because I -- it didn't matter to me as  
99:16 reviewing these who did the study. The  
99:17 question was, what's the quality of the  
99:18 study, what's it say, et cetera.  
99:19 Q. Okay. Great.  
99:20 So let's look at Knezevich and  
99:21 Hogan. So we have this 24-year -- you said  
99:22 that refers to the length of the study.

99:23 And then we have the blue box,  
99:24 and it says, "Kidney carcinomas and  
99:25 adenomas."  
100:1 Do you see that?  
100:2 A. Yes, I see it.  
100:3 Q. What is that referring to?  
100:4 A. So that's a finding from the  
100:5 study. This is one set of tumors, kidney  
100:6 tumors, and the tumors in the kidney come in  
100:7 two forms: carcinomas, which are the  
100:8 malignant tumors; and adenomas, which are the  
100:9 precursors to the carcinomas. So that's the  
100:10 premalignant tumors.  
100:11 And typically when you have  
100:12 them, you can analyze them separately and you  
100:13 can analyze them as combined. Here, I'm  
100:14 presenting the combined results.  
100:15 I've also got the individual  
100:16 results in a separate picture, but the  
100:17 combined results are good enough here.  
100:18 I've circled trend because they  
100:19 are statistically significant in their trend,  
100:20 which is that slope climb that we see before.  
100:21 There's a single plus there.  
100:22 If you slide down a little bit on the chart,  
100:23 you'll see I put a little legend down there.  
100:24 Q. Oh, down here.  
100:25 A. Yes.  
101:1 So the plus on the chart means  
101:2 that the statistical probability of seeing  
101:3 that trend is between .1 and .05. So I will  
101:4 refer to that as marginally significant.  
101:5 Typically in these studies,  
101:6 5 percent, .05, is what people refer to as  
101:7 statistically significant.  
101:8 Q. Is that referring to these two  
101:9 pluses right here?  
101:10 A. Correct.  
101:11 Q. Okay.  
101:12 A. So when it's two pluses, that

101:13 means it is below 5 percent but above  
101:14 1 percent.  
101:15 And people talk about highly  
101:16 significant as below 1 percent. So .01,  
101:17 that's the three pluses.  
101:18 Q. Okay. Great.  
101:19 A. So you're going from -- there's  
101:20 a trend, but it's not extremely strong.  
101:21 That's one plus. There's a trend, it's  
101:22 strong. And the bottom one, there's a trend  
101:23 and it's very strong. So that's what the  
101:24 three are broken down as.  
101:25 Q. You also have here HC,  
102:1 historical controls.  
102:2 What does that refer to?  
102:3 A. I'll explain that when we go  
102:4 back to kidney.  
102:5 Q. Okay. Let's go back to  
102:6 kidneys.  
102:7 So we're back to kidneys?  
102:8 A. Correct.  
102:9 Q. And so you've circled the trend  
102:10 and there's a plus?  
102:11 A. That's correct.  
102:12 Q. So that means it's a marginally  
102:13 significant trend?  
102:14 A. Correct.  
102:15 Q. Okay.  
102:16 A. In this case I think it was  
102:17 .062, 6.2 percent.  
102:18 Q. Okay.  
102:19 A. And it's in males, not in  
102:20 females.  
102:21 Q. And that's why you circled the  
102:22 M here?  
102:23 A. Right.  
102:24 And I did not circle dose. And  
102:25 that means that when you compare each dose  
103:1 group to the control group, there are none  
103:2 that were statistically significantly

103:3 different from control.  
103:4 If I circle dose, that means  
103:5 that at least one of those dose groups was  
103:6 different than the control all by itself.  
103:7 Q. Gotcha.  
103:8 A. Now, I put a little line to the  
103:9 side here and I've written "HC," and I put  
103:10 two pluses on top of that.  
103:11 So remember I told you, you can  
103:12 look back at other control groups from other  
103:13 studies in the same species, same strain,  
103:14 same sex, and look to see if this looks  
103:15 different than those control populations.  
103:16 Well, it turns out there are  
103:17 statistical ways of bringing in that  
103:18 historical evidence and evaluating the  
103:19 current study using that historical evidence  
103:20 from other control groups. And so I've done  
103:21 that here using what's known as the Tarone  
103:22 test for historical controls.  
103:23 In my expert report, I used a  
103:24 calculation that I had done on my own. It  
103:25 was criticized, so I went to one of the  
104:1 literature approaches and used one of the  
104:2 standard approaches, Tarone's test for  
104:3 historical controls. And I applied it here,  
104:4 and it shows a P value that's less than .05.

105:3 - 114:17

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CP1\_SS\_01.30

105:3 All right, sir. So we've  
105:4 looked at the kidney carcinomas and the  
105:5 Knezevich and Hogan tests from 1983. I want  
105:6 to jump forward to Sugimoto just to sort of  
105:7 keep it consistent.  
105:8 We again have kidney carcinomas  
105:9 and adenomas.  
105:10 Do you see that?  
105:11 A. Yes, I see that.  
105:12 Q. Okay. So let me see if I get  
105:13 my understanding of your symbols here.  
105:14 The circle with the plus, what

105:15 does that mean?  
105:16 A. Trend test was positive,  
105:17 marginally significant.  
105:18 Q. And then you again circled the  
105:19 M.  
105:20 Do you see that?  
105:21 A. For males, that's correct.  
105:22 Q. And so this is sort of the same  
105:23 sort of result. It's a trend, marginally  
105:24 significant in males?  
105:25 A. Correct.  
106:1 Q. Okay. And then you have the  
106:2 historical controls here?  
106:3 A. Correct.  
106:4 Q. And that one has three pluses?  
106:5 A. Correct.  
106:6 Q. So the difference between --  
106:7 A. Highly significant as compared  
106:8 to just significant.  
106:9 Q. Okay. So the difference  
106:10 between the Sugimoto and Knezevich and Hogan,  
106:11 when it comes to kidney carcinomas, is in  
106:12 Knezevich and Hogan it was just significant,  
106:13 the historical control result, but in  
106:14 Sugimoto it was highly significant?  
106:15 A. Correct.  
106:16 Q. Okay.  
106:17 A. And you only use historical  
106:18 controls in two situations. One situation --  
106:19 all the guidelines tell you that the best  
106:20 control group to use in evaluating cancer  
106:21 data is the concurrent control, the control  
106:22 that was used in the current experiment. And  
106:23 that's what you should use except in two  
106:24 situations, in my opinion.  
106:25 One situation is where you have  
107:1 a rare tumor. A rare tumor is defined in  
107:2 most toxicological literature as a tumor that  
107:3 occurs at less than 1 percent frequency in  
107:4 these animals.

107:5 The kidney tumors that we're  
107:6 looking at here are rare tumors by anyone's  
107:7 definition. They occur at about 1 per 400  
107:8 animals, roughly, about .25 percent of the  
107:9 time. And so it's appropriate here to look  
107:10 at the historical controls and compare them  
107:11 because it's a rare tumor.  
107:12 The other case of using  
107:13 historical controls is when you have an odd  
107:14 tumor response. And what you mean there is  
107:15 when you have a very low control response and  
107:16 then all of the treated groups have identical  
107:17 or close to identical response and it's much  
107:18 higher.  
107:19 And your question in that  
107:20 situation is should the historical  
107:21 controls -- should the controls have been up  
107:22 here, in which case it's perfectly flat, or  
107:23 is this reasonable, in which case you've got  
107:24 an increase but there's no trend. It's just  
107:25 increasing flat, which is an unusual  
108:1 response. And so those are the two cases  
108:2 you're looking at.  
108:3 But here we're looking at it  
108:4 because it's a rare tumor.  
108:5 Q. Okay. That's helpful.  
108:6 The other thing I want to  
108:7 clarify is in Knezevich and Hogan it was 24,  
108:8 and Sugimoto it was 18?  
108:9 A. That's correct. The 18 there  
108:10 refers to the number of months that these  
108:11 animals were exposed. So they were exposed  
108:12 for less time. When they finished the study,  
108:13 they were younger animals.  
108:14 The reason this historical  
108:15 control is now highly significant rather than  
108:16 just significant is because in 18 months you  
108:17 see even fewer kidney tumors in these  
108:18 animals. So their historical control rate is  
108:19 much lower, and you're still seeing a

108:20 positive response, and so it makes for a much

108:21 more significant finding.

108:22 Q. And if we -- I just want to

108:23 finish the loop here on the kidney tumors.

108:24 We have this last study here,

108:25 Kumar 2001.

109:1 Do you see that?

109:2 A. Correct.

109:3 Q. And you see it's shaded light

109:4 gray versus the white?

109:5 A. I can't really see the light

109:6 gray, but it should be shaded differently.

109:7 It's a different strain of mouse.

109:8 Q. And that's my question.

109:9 So why -- why is this study

109:10 slightly different than the others?

109:11 A. Yes, the others -- all four of

109:12 the others are CD-1 mice, one of the special

109:13 strains. This is a Swiss Webster mouse. It

109:14 is a different strain of mouse, and so you

109:15 would expect different historical responses,

109:16 different control responses, even different

109:17 responses to the chemical, potentially.

109:18 Q. So this is in a different

109:19 strain, and we see again a trend in males

109:20 that's positive; is that right?

109:21 A. Correct. It's marginally

109:22 significant.

109:23 Q. Just like the other two studies

109:24 were?

109:25 A. Correct.

110:1 Q. What, if any, significance is

110:2 the fact that you're seeing this same tumor

110:3 response across different strains of mice?

110:4 A. Oh, I will note I didn't do

110:5 historical controls in that one, not because

110:6 it's not rare, it's because I couldn't find a

110:7 historical control population --

110:8 Q. Oh.

110:9 A. -- for that particular type of

110:10 mouse. And I can't use the historical  
110:11 population from the CD-1 mice to do that  
110:12 calculation. So you have to find the  
110:13 appropriate group.  
110:14 The fact that you see the tumor  
110:15 in multiple studies from different  
110:16 laboratories strengthens -- it's one of the  
110:17 criteria we were looking at in EPA's cancer  
110:18 guidelines. It strengthens the belief that  
110:19 this is a positive finding.  
110:20 Q. And just to sort of tie the  
110:21 loop back, remember earlier we gave that  
110:22 example of the Wood study from 2009?  
110:23 A. Yes.  
110:24 Q. Is that it right there?  
110:25 A. That's it right there.  
111:1 Q. And we actually specifically  
111:2 discussed the malignant lymphoma finding,  
111:3 right?  
111:4 A. That's correct.  
111:5 Q. And what we have here is the  
111:6 trend, the dose and the M and three pluses.  
111:7 Can you explain to the jury  
111:8 what that means?  
111:9 A. In this case, you've seen the  
111:10 data. There was indeed a statistically  
111:11 significant trend in the data. In fact, it  
111:12 was less than .01, was the probability. I  
111:13 told you it was .007 out of -- 7 out a  
111:14 thousand, and that is in the highly  
111:15 significant group.  
111:16 The highest dose was, in fact,  
111:17 significantly different from the control  
111:18 group, and so I circled dose here. And it  
111:19 was only in males; it was not in females.  
111:20 Q. Okay. Great.  
111:21 So I don't want to spend all  
111:22 day going through all the different findings  
111:23 that you have here, but I do want to take a  
111:24 step -- well, I want to focus on a few more

111:25 just so we can understand what they're about.

112:1 I want to look at this yellow

112:2 box under Wood, multiple malignant tumors or

112:3 neoplasms.

112:4 Do you see that?

112:5 A. Yes.

112:6 Q. What's that refer to?

112:7 A. So that was an analysis they

112:8 did in the Wood study where they looked to

112:9 see how many malignancies there were per

112:10 animal in the study, and they looked to see

112:11 if that was increasing with exposure in the

112:12 study.

112:13 So they did a trend test

112:14 through that, and they found that to be a

112:15 statistically significant trend in the male.

112:16 So male animals, as you go up in exposure,

112:17 each animal is likely to have multiple

112:18 malignant tumors.

112:19 Q. And we have another multiple

112:20 malignant finding in the Sugimoto 1997 study.

112:21 Do you see that?

112:22 A. 1987, is that right?

112:23 Q. Sorry, it's 1997.

112:24 A. '97.

112:25 Q. Okay. And if you down here,

113:1 there's a lot of different tumors, but we get

113:2 down to the multiple malignant tumors.

113:3 Do you see that?

113:4 A. Yes.

113:5 Q. And this one -- the -- so you

113:6 have a significant -- a highly significant

113:7 trend, a highly significant dose and in

113:8 males?

113:9 A. This one has a highly

113:10 significant trend. I don't know about the

113:11 highly significant dose. I did not put the

113:12 pluses for the dose test, but it is in males.

113:13 Q. Fair enough.

113:14 A. The pluses on here are strictly

113:15 for the trend tests.  
 113:16 Q. Thank you. That's helpful.  
 113:17 All right. Well, taking a step  
 113:18 back and looking at all these studies, we  
 113:19 have all these tumors, and we've color-coded  
 113:20 the tumors to match up, right?  
 113:21 So we have the kidney ones in  
 113:22 light blue. Do you see that?  
 113:23 A. Yes.  
 113:24 Q. And we have this pink one that  
 113:25 appears in four of the five studies.  
 114:1 A. Correct.  
 114:2 Q. That's referring to malignant  
 114:3 lymphoma; is that right?  
 114:4 A. That's correct.  
 114:5 Q. What, if any, significance is  
 114:6 there that in four of the five mouse studies  
 114:7 you have a malignant lymphoma finding?  
 114:8 A. Again, it speaks to the  
 114:9 consistency of the finding across multiple  
 114:10 studies in multiple laboratories.  
 114:11 Two of those, both the 18-month  
 114:12 studies, both the most recent mouse studies,  
 114:13 are significant in and of themselves in each  
 114:14 of the two studies, and the other two are  
 114:15 marginally significant. It basically says  
 114:16 that this chemical is causing these tumors in  
 114:17 mice.

115:2 - 116:9

**Portier, Christopher 02-21-2019 (00:01:08)**

CP1\_SS\_01.31

115:2 Q. Where was the findings of these  
 115:3 tumors?  
 115:4 What type of mice were they  
 115:5 found in?  
 115:6 A. CD-1 mice for the three -- for  
 115:7 the Atkinson, Sugimoto and Wood, and in the  
 115:8 Swiss Webster mouse.  
 115:9 Q. And what were their genders?  
 115:10 A. All males.  
 115:11 Q. Does that have any significance  
 115:12 to you?

115:13 A. It's simply, again, repeating  
115:14 the finding from study to study. The fact  
115:15 that you don't see it in females, you do see  
115:16 it in males, speaks to a consistency of the  
115:17 actual finding itself.  
115:18 Q. Now, it's almost impossible to  
115:19 see, and I apologize because of the colors.  
115:20 We have this dark purple one here in  
115:21 Sugimoto. See if we can get in close enough  
115:22 to read it. It's hemangiomas.  
115:23 Do you see that?  
115:24 A. Yes.  
115:25 Q. Okay. And recognizing that it  
116:1 was hard to read, I see you wrote it to the  
116:2 side here; is that right?  
116:3 A. Correct.  
116:4 Q. And so what did you find for  
116:5 the hemangiomas in this study?  
116:6 A. Well, there was a highly  
116:7 significant trend in hemangiomas, it's in  
116:8 females, in females only, and there were no  
116:9 dose-related effects by themselves.

**Portier, Christopher 02-21-2019 (00:09:13)**

117:19 Q. All right. So we're looking at  
117:20 the Kumar study.  
117:21 A. Correct.  
117:22 Q. Is this the same strain of  
117:23 mice?  
117:24 A. No, it is not.  
117:25 Q. Okay. If we go down, we have  
118:1 the hemangioma finding. Is that what I'm  
118:2 seeing here?  
118:3 A. That is what you're seeing  
118:4 there.  
118:5 Q. And what did you find?  
118:6 A. Here we found a highly  
118:7 significant trend, increasing hemangiomas in  
118:8 females with an increasing exposure to  
118:9 glyphosate, and only in females, not in  
118:10 males.

117:19 - 127:16

CP1\_SS\_01.32

118:11 Q. And this finding between  
118:12 Sugimoto and Kumar, what significance is  
118:13 there to that?  
118:14 A. Oh, again, it's -- you're  
118:15 seeing the same tumor in multiple studies.  
118:16 In this case, two different laboratories. In  
118:17 this case, two different strains of mice.  
118:18 That adds to the overall finding that this is  
118:19 probably a positive finding.  
118:20 You don't see it in Wood, but  
118:21 these hemangiomas -- I'd have to go back and  
118:22 look at the Wood study to see why, but my  
118:23 recollection is that Wood saw none. This is  
118:24 a very rare tumor. And so that doesn't  
118:25 really subtract from the fact that she found  
119:1 it in the other study.  
119:2 Again, it's a highly  
119:3 significant finding.  
119:4 Q. Now, looking at all these  
119:5 tumors in these mice studies, which ones to  
119:6 you are the most compelling findings when  
119:7 you're assessing whether or not glyphosate  
119:8 can cause cancer?  
119:9 A. The kidney carcinomas and  
119:10 adenomas are important. They're repeated.  
119:11 Even though they're marginal, they're rare  
119:12 tumors. And as we saw with EPA's guidelines,  
119:13 when you see rare tumors occurring, you perk  
119:14 up and look at it very carefully. I think  
119:15 those are clearly caused by glyphosate here.  
119:16 The malignant lymphomas, I have  
119:17 no doubt in my mind that they are caused by  
119:18 glyphosate here. It's especially obvious in  
119:19 the 18-month studies.  
119:20 One you didn't mention were  
119:21 hemangiosarcomas. You saw it in one of the  
119:22 24-month studies in the Atkinson study. It's  
119:23 highly significant.  
119:24 When you look at the 18-month  
119:25 study, the hemangiosarcomas are significant.

120:1 But in 18 months, the historical controls, 26  
120:2 historical control groups, there were no  
120:3 hemangiosarcomas ever seen in 18 months, so  
120:4 that's a highly significant finding,  
120:5 biologically important, and that's quite  
120:6 obvious.  
120:7 So I think the hemangiosarcomas  
120:8 are important, and the hemangiomas that we  
120:9 just talked about in the females are  
120:10 important findings as well.  
120:11 Q. And just so we close the loop  
120:12 on this, this Atkinson study has the word  
120:13 "limited" in yellow.  
120:14 Do you see that?  
120:15 A. Oh, yes, I'm sorry, I didn't  
120:16 explain that.  
120:17 Q. Well --  
120:18 A. Would you like me to explain  
120:19 that?  
120:20 Q. Yeah.  
120:21 What does that mean?  
120:22 A. So the Atkinson study is  
120:23 different than the other studies because they  
120:24 didn't look at all of the animals by taking  
120:25 slices of the tissues. They -- they did  
121:1 something cheaper, less expensive, which was  
121:2 popular at the time. I don't want  
121:3 to think they were doing something very, very  
121:4 unusual.  
121:5 Several groups were exploring  
121:6 the possibility, including the National  
121:7 Toxicology Program, of reducing the amount of  
121:8 pathology you do. The idea would be that you  
121:9 do the control group and you do the high dose  
121:10 group, you do the entire evaluation, and then  
121:11 anything you see that's important in those  
121:12 two groups, you only look at those tissues in  
121:13 the interior groups.  
121:14 And so that's what Atkinson  
121:15 did. It turned out Atkinson didn't think any

121:16 of the tumors were important, so he didn't do  
121:17 any of the tissues in the intermediate groups  
121:18 except liver, lung and kidney, which they had  
121:19 decided to do in advance, that they would  
121:20 look at those tissues in advance, no matter  
121:21 what they saw.

121:22 So there were a bunch of  
121:23 animals in these studies that even though  
121:24 it -- the Atkinson study is multiple dose  
121:25 groups, it's really only a two-dose group  
122:1 study, high dose control.

122:2 Q. And even though in Atkinson  
122:3 they didn't look at lymphomas in the middle  
122:4 groupings, is there any significance to still  
122:5 having a lymphoma finding here?

122:6 A. Well, lymphomas are -- not  
122:7 really, okay? To be fair here, lymphomas are  
122:8 very aggressive tumors. You're going to find  
122:9 them. Even if you don't do pathology on  
122:10 every single tissue, you are going to find a  
122:11 malignant lymphoma if it's there. They're  
122:12 quite obvious from a pathological point of  
122:13 view.

122:14 So for malignant lymphomas, the  
122:15 proper denominator is probably all of the 50  
122:16 animals, 51 animals, that were in each of the  
122:17 dose groups from Atkinson because you would  
122:18 find them.

122:19 Q. Okay. And so it would be fair  
122:20 to say then that even though Atkinson was  
122:21 limited, it doesn't affect your opinion of  
122:22 the malignant lymphoma finding?

122:23 A. Correct.

122:24 Or the hemangiosarcomas,  
122:25 because it's the same thing. They are  
123:1 blood-based tumors, and you find them  
123:2 typically by seeing a tumor.

123:3 Q. Okay. Okay. Let's turn to  
123:4 Exhibit 883, which is the rat chart.

123:5 I don't know if you can see it

123:6 on the screen, sir, but as you -- maybe you  
123:7 can. But the last three studies are in a  
123:8 different shade than the first four studies.  
123:9 Do you see that?  
123:10 A. That's correct.  
123:11 Q. Okay. What --  
123:12 A. They should be.  
123:13 Q. -- does that signify?  
123:14 A. The first four studies were  
123:15 done in Sprague Dawley rats, one strain of  
123:16 rat. The second -- the last three studies  
123:17 were done in Wistar rats, a completely  
123:18 different strain of rat.  
123:19 Q. And I notice up here you have  
123:20 numbers written.  
123:21 What do those reflect?  
123:22 A. Number of months on study. So  
123:23 the Lankas study was 26 months' exposure in  
123:24 the rats, and all of the other studies are  
123:25 24 months of exposure.  
124:1 Q. Okay. And then you also have a  
124:2 little key down here.  
124:3 Is it the same plus chart we  
124:4 did from the previous one?  
124:5 A. That is correct.  
124:6 Q. Okay. And then we have -- two  
124:7 of these studies say "limited." It's  
124:8 Atkinson -- I mean, I'm confused. Atkinson  
124:9 was in the mouse study. Why is it on the rat  
124:10 chart?  
124:11 A. As I pointed out earlier,  
124:12 typically these studies are rats and mice,  
124:13 males and females. So Atkinson managed both  
124:14 sets of studies, rats and mice, males and  
124:15 females.  
124:16 You'll see also there's a Wood  
124:17 2009. There was a Wood 2009 in the mouse.  
124:18 Wood managed both of those studies. They  
124:19 were done in the same laboratory. So it's --  
124:20 that's not unusual to see.

124:21 And limited means exactly the  
124:22 same thing here. Atkinson, in their rat  
124:23 study, also did that same limited pathology.  
124:24 Suresh in 1996 also did the  
124:25 same basic limited pathology.  
125:1 Q. Okay. And Suresh, unlike all  
125:2 the other studies, you didn't find any  
125:3 significant tumor findings?  
125:4 A. That's correct. Suresh had  
125:5 absolutely nothing that appeared to be  
125:6 positive in the entire study.  
125:7 Q. Okay. So I want to go through  
125:8 a few of these, but let's just use the first  
125:9 one as just an example to sort of make sure  
125:10 we're reading it correctly.  
125:11 So this Lankas study is from  
125:12 1981; is that right?  
125:13 A. Correct.  
125:14 Q. And trend, dose, male, three  
125:15 pluses, what does that mean?  
125:16 A. So again, this is a highly  
125:17 statistically significant trend increase in  
125:18 these interstitial cell tumors in testicles  
125:19 in these Sprague Dawley rats after 26 months  
125:20 of exposure. The highest dose, or one of the  
125:21 dose groups, was statistically significant  
125:22 from the controls. And these are testicles,  
125:23 so it only occurred in the males.  
125:24 One thing about this study is  
125:25 that the doses in the study were  
126:1 significantly lower than all of the other  
126:2 studies here by a factor of at least 10 for  
126:3 even the lowest dose in the other studies,  
126:4 making this a very unusual study to have seen  
126:5 positive findings. But it is 26 months, so  
126:6 they went a little bit longer.  
126:7 And so your question earlier  
126:8 about 70-year-old people, this one's into  
126:9 that range. And so it's possible they're  
126:10 picking up things that other studies would

126:11 not pick up because they went a little  
 126:12 longer.  
 126:13 This testicular interstitial  
 126:14 cell tumor finding is in no other study.  
 126:15 It's a unique study by itself, but it's a  
 126:16 very strong finding.  
 126:17 Q. And if we look, just sticking  
 126:18 to Lankas, we have thyroid C-cell carcinomas  
 126:19 or adenomas and pancreatic islet cell tumors.  
 126:20 Do you see that?  
 126:21 A. Correct.  
 126:22 Q. And those are just, again,  
 126:23 types of tumors that are studied?  
 126:24 A. That's correct. The unique  
 126:25 thing here is the pancreatic islet cell  
 127:1 tumors, there is no dose-response trend  
 127:2 there. There's only a significant finding of  
 127:3 one of the groups to the control group.  
 127:4 The two pluses there refer to  
 127:5 that pairwise comparison, not the trend.  
 127:6 Q. Gotcha.  
 127:7 A. So there's no trend in that one  
 127:8 that is positive.  
 127:9 The thyroid C-cell carcinomas  
 127:10 were in females, and that was a marginally  
 127:11 significant finding.  
 127:12 Q. And if we look at the next  
 127:13 study, Stout and Ruecker, 1990, we again see  
 127:14 the thyroid one.  
 127:15 Do you see that?  
 127:16 A. Correct.

127:19 - 128:18

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CP1\_SS\_01.33

127:19 Q. And what is this reflection  
 127:20 that there's both M and F circled?  
 127:21 A. So this is, again, the same  
 127:22 tumors, thyroid C-cell carcinomas or adenomas  
 127:23 combined. When you look at thyroid C-cells  
 127:24 carcinomas here for the females, it's  
 127:25 significant all by itself, but I decided to  
 128:1 present the combined analysis here.

128:2 The trend test is marginally  
128:3 significant for both males and females, and  
128:4 for females, one of the dose groups is  
128:5 significantly different from the controls.  
128:6 Q. And then we see this pancreatic  
128:7 islet cell tumors.  
128:8 Do you see that?  
128:9 A. Correct. Again, the same  
128:10 tumors before, but -- and this time there's  
128:11 still no trend. You see a single dose group  
128:12 increased against the controls, and it's in  
128:13 males again.  
128:14 Q. And this is essentially the  
128:15 same finding?  
128:16 A. Exactly the same finding.  
128:17 Q. Okay.  
128:18 A. Or same kind of finding.

129:4 - 131:18

**Portier, Christopher 02-21-2019 (00:02:36)**

CP1\_SS\_01.34

129:4 Q. Okay. And they seem to be --  
129:5 well, how many times do they pop up in these  
129:6 studies?  
129:7 A. Three times in the Sprague  
129:8 Dawley rats and once in the Wistar rats.  
129:9 Q. What kind of tumor is that?  
129:10 A. A skin keratoacanthoma is a  
129:11 skin tumor. It's typically a benign skin  
129:12 tumor, although it can become malignant.  
129:13 It's not usually malignant, but it can become  
129:14 malignant. In some species it is highly  
129:15 malignant, depending upon the rat species,  
129:16 rat strain, you're looking at.  
129:17 But, yeah, it's a skin cancer.  
129:18 What else?  
129:19 Q. That answers my question.  
129:20 Are you familiar with the term  
129:21 "oncogenicity"?  
129:22 A. Yes, I am familiar with that  
129:23 term.  
129:24 Q. What does that mean?  
129:25 A. Oncogenicity means same as

130:1 carcinogenicity. It's the ability to cause  
130:2 cancer.  
130:3 Q. And specifically does it relate  
130:4 to tumor formation?  
130:5 A. Yes.  
130:6 Q. Okay. The fact that you're  
130:7 seeing these skin kera -- I can't say that  
130:8 phrase?  
130:9 A. Keratoacanthoma.  
130:10 Q. Okay. The fact that you're  
130:11 seeing so many of those in different studies,  
130:12 does that lend or not lend support to  
130:13 glyphosate being oncogenic?  
130:14 A. Oh, that lends support. Just  
130:15 because the tumor is benign doesn't mean it  
130:16 isn't an important oncogenic finding. So,  
130:17 yes, it does lend credence to that. It's  
130:18 quite clear that it's causing these skin  
130:19 keratoacanthomas in these rat studies.  
130:20 It's -- the fact that it's  
130:21 appearing in three of the four Sprague Dawley  
130:22 rat studies is an important finding.  
130:23 I don't remember what it was in  
130:24 Lankas. I did evaluate it. It's in my  
130:25 expert report. But I don't think the Lankas  
131:1 study made a big difference in what you were  
131:2 seeing here. I think this is quite clear.  
131:3 Q. Now, if we look at Endimoto,  
131:4 which is the middle study from 1997, we have  
131:5 a blue box.  
131:6 Do you see that?  
131:7 A. Yes.  
131:8 Q. What is this referring to?  
131:9 A. So again, we're looking at  
131:10 kidney carcinomas or adenomas, the same we  
131:11 saw as in the CD-1 mice. There's a  
131:12 significant trend only in males, and it's  
131:13 highly significant. It's P value is less  
131:14 than .01.  
131:15 Q. So if we just go back to the

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131:21 - 132:1	<p>131:16 mice chart briefly, we have kidney carcinomas  131:17 in Knezevich and Hogan and Sugimoto, and  131:18 that -- what kind of mice is that?</p> <p><b>Portier, Christopher 02-21-2019 (00:00:06)</b></p> <p>131:21 THE WITNESS: Sugimoto is the  131:22 CD-1 mouse.  131:23 QUESTIONS BY MR. WISNER:  131:24 Q. And then we have another  131:25 finding in Kumar.</p> <p>132:1 What kind of mouse was that?</p>	CP1_SS_01.36
132:4 - 132:11	<p><b>Portier, Christopher 02-21-2019 (00:00:10)</b></p> <p>132:4 THE WITNESS: The Kumar mouse  132:5 is a Swiss Webster mouse.  132:6 QUESTIONS BY MR. WISNER:  132:7 Q. And now we're into another  132:8 species altogether, and we have another  132:9 finding.  132:10 And what kind of mouse was --  132:11 what kind of rat was that?</p>	CP1_SS_01.36
132:14 - 134:3	<p><b>Portier, Christopher 02-21-2019 (00:01:42)</b></p> <p>132:14 THE WITNESS: That's a Sprague  132:15 Dawley rat.  132:16 QUESTIONS BY MR. WISNER:  132:17 Q. What is the significance of  132:18 seeing this popping up across species and  132:19 across strains?  132:20 A. Well, when you're -- when  132:21 you're looking at cancer bioassay data, one  132:22 thing that strengthens the belief that the  132:23 chemical can cause -- I'm using a very  132:24 general term. So I might say glyphosate  132:25 causes malignant lymphomas in CD-1 mice.  133:1 Okay? That's a very specific statement about  133:2 a specific tumor.  133:3 But you also have a general  133:4 statement about, you know, is it possible in  133:5 mammalian systems for glyphosate to cause  133:6 cancer. And since these are controlled  133:7 studies, we'd like to be able to say in  133:8 rodents, in rats and mice, does glyphosate</p>	CP1_SS_01.37

133:9 cause cancer.  
 133:10 So when you're trying to answer  
 133:11 that bigger question, there are things like  
 133:12 in the EPA evaluation you'd like to see.  
 133:13 Multiple studies with the same tumor,  
 133:14 multiple studies with the same tumor in  
 133:15 different species, that strengthens that  
 133:16 finding for that tumor, and it strengthens  
 133:17 that overall call that glyphosate can -- is  
 133:18 oncogenic, if you want to use that oncogenic  
 133:19 term. It can cause cancer of some sort in  
 133:20 mammalian systems.  
 133:21 And so on that big question,  
 133:22 when I see kidney tumors in Sprague Dawley  
 133:23 rats, CD-1 mice and Swiss Webster mice from  
 133:24 the same chemical, that strengthens the  
 133:25 finding that that chemical is oncogenic.  
 134:1 Q. How long have you been involved  
 134:2 in these exact type of rodent studies?  
 134:3 A. Oh, 40 years.

134:13 - 135:21

**Portier, Christopher 02-21-2019 (00:01:35)**

CP1\_SS\_01.38

134:13 Q. And when you look at all of  
 134:14 these tumor data in the rats and in the mice,  
 134:15 what is your conclusion about whether or not  
 134:16 glyphosate can cause cancer in animals?  
 134:17 A. There is no doubt in my mind  
 134:18 that glyphosate can cause tumors in  
 134:19 laboratory animals. There's just no doubt.  
 134:20 Q. Well, hold on a second. How  
 134:21 does that relate to humans then?  
 134:22 A. Well, most human -- in fact,  
 134:23 all human carcinogens that are chemical  
 134:24 carcinogens have been shown to be  
 134:25 carcinogenic in some sort of laboratory  
 135:1 animal. So you've got half of it. That's  
 135:2 the question of sensitivity.  
 135:3 Are animal models sensitive  
 135:4 enough to find human carcinogens? Yes.  
 135:5 Every human carcinogen has been seen in at  
 135:6 least one animal model. You don't have the

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	<p>135:7 specificity. Just because it's in the animal  135:8 model doesn't mean it's in humans.  135:9 So it tells you to be worried  135:10 about the human system. It's part of the  135:11 overall evaluation. It's not enough to be  135:12 absolutely certain this is going to cause  135:13 cancer in humans, but the fact that you can  135:14 see it causing cancer in mammals that are  135:15 95 percent genomically similar to humans  135:16 raises concerns and raises the bar to have  135:17 concern about the carcinogenicity,  135:18 oncogenicity of this particular product.  135:19 Q. And before a product is  135:20 approved, like glyphosate, are these types of  135:21 studies required?</p>	
135:24 - 136:16	<b>Portier, Christopher 02-21-2019 (00:00:39)</b>	CP1_SS_01.39
	<p>135:24 THE WITNESS: In the United  135:25 States they are definitely required.  136:1 QUESTIONS BY MR. WISNER:  136:2 Q. All right. So I want to go  136:3 back to Exhibit 880.  136:4 This is our cancer stool that  136:5 we've put together, our causation stool that  136:6 we've put together.  136:7 And we spent the morning so far  136:8 discussing animal studies; is that right?  136:9 A. That is correct.  136:10 Q. Okay. I want to move on to the  136:11 next topic, which is mechanism studies.  136:12 All right?  136:13 A. Okay.  136:14 Q. But you know what? Before we  136:15 do that, let's take a short break.  136:16 A. Okay.</p>	
137:5 - 137:8	<b>Portier, Christopher 02-21-2019 (00:00:09)</b>	CP1_SS_01.40
	<p>137:5 We were looking at this stool  137:6 here on animal studies, and so far the animal  137:7 studies we've looked at, were they looking at  137:8 glyphosate or glyphosate formulations?</p>	
137:11 - 137:25	<b>Portier, Christopher 02-21-2019 (00:00:28)</b>	CP1_SS_01.41

137:11 THE WITNESS: The studies that  
137:12 we've looked at were looking at  
137:13 glyphosate alone.  
137:14 QUESTIONS BY MR. WISNER:  
137:15 Q. What is --  
137:16 A. Pure glyphosate.  
137:17 Q. What is the difference between  
137:18 glyphosate and the glyphosate formulation?  
137:19 A. I am in no way, shape or form  
137:20 an expert on that, but roughly -- from my  
137:21 rough understanding, glyphosate formulations  
137:22 have other chemicals in them to help get the  
137:23 glyphosate into the plants and do other  
137:24 things that are necessary to make the  
137:25 glyphosate effective as a herbicide.

138:12 - 152:5

**Portier, Christopher 02-21-2019 (00:13:05)**

CP1\_SS\_01.42

138:12 Q. Okay. And to be clear, when we  
138:13 talk about the animal studies here, we've  
138:14 been talking so far about glyphosate; is that  
138:15 right?  
138:16 A. That is correct.  
138:17 Q. When we talk about mechanism  
138:18 studies, are we talking about just glyphosate  
138:19 or both?  
138:20 A. Both. There are mechanism  
138:21 studies which are pure glyphosate and  
138:22 mechanism studies which are glyphosate  
138:23 formulations.  
138:24 Q. And when we talk about  
138:25 epidemiology, are we talking about technical  
139:1 glyphosate or the formulation?  
139:2 A. Human studies are all technical  
139:3 glyphosate. The formulation -- sorry, the  
139:4 formulations. Yes, the humans are exposed to  
139:5 only the formulations.  
139:6 Q. And is that -- why is that?  
139:7 Why are humans exposed to the formulated  
139:8 product?  
139:9 A. Well, because these are not  
139:10 controlled studies, experimental studies in

139:11 humans. These are humans who are working or  
139:12 living near fields that are sprayed with  
139:13 glyphosate, who get ancillary exposure, and  
139:14 so they're being exposed to the commercial  
139:15 product, which is the formulation.

139:16 Q. Okay. Earlier in your  
139:17 testimony you talked about something called  
139:18 an initiation and promoter study.

139:19 Do you recall that?

139:20 A. Yes, I do.

139:21 Q. What is an initiator and  
139:22 promoter study?

139:23 A. So I do have a graphic on this.

139:24 Would you like to look at the  
139:25 graphic and I can walk through that?

140:1 Q. Sure.

140:2 Do you want to look at the  
140:3 carcinogenesis?

140:4 A. Yes.

140:5 Q. Okay. Great.

140:6 A. The mechanism graphic because  
140:7 that is -- pertains to the  
140:8 initiation/promotion study.

140:9 Q. Okay. This thing would be  
140:10 great, the trial pad.

140:11 In your binder is page 88 --  
140:12 well, I'll just put it up on the screen, and  
140:13 you tell me if this is what you're looking  
140:14 for.

140:15 Is this what you're looking  
140:16 for?

140:17 A. 885, it says.

140:18 Q. Okay. Great. This is  
140:19 Exhibit 885.

140:20 Using this diagram, explain to  
140:21 us what an initiation and promoter study is.

140:22 A. So this is a diagram, missing  
140:23 one line, of how cells go from being normal  
140:24 working cells to becoming cancerous cells.

140:25 It's a very simple picture of the overall

141:1 process.  
141:2 It's a multi-stage process, so  
141:3 cells don't go from being normal to cancer  
141:4 all in one shot. They go through a series of  
141:5 events that generally lead to a carcinogenic  
141:6 finding.  
141:7 The first part, you've got a  
141:8 whole bunch of normal cells. They're doing  
141:9 what they're supposed to do. They're happy.  
141:10 They're functioning. They're going along  
141:11 just fine.  
141:12 Something happens. Either  
141:13 something comes in or just normal to the  
141:14 cells, the DNA gets damaged. And there's  
141:15 supposed to be a line between normal cells to  
141:16 damaged cells, which somehow has disappeared.  
141:17 Q. I just drew a line.  
141:18 A. There you go.  
141:19 And all of a sudden now,  
141:20 instead of all of these normal cells --  
141:21 you've got a bunch of normal cells, and in  
141:22 the middle of them is one damaged cell. It's  
141:23 got a DNA that's different than the rest.  
141:24 Q. Is that this picture right here  
141:25 that you're referring to?  
142:1 A. Second picture.  
142:2 Q. Right here?  
142:3 A. Yes.  
142:4 Q. Okay.  
142:5 A. Now, the cell has a lot of  
142:6 machinery that can repair that DNA damage.  
142:7 And generally that happens when the cell  
142:8 replicates, but it can happen at any time.  
142:9 But it tries to repair that damage, and if it  
142:10 repairs it, fixes the DNA, then it's the same  
142:11 DNA as everybody else, and you go back to  
142:12 being a happy tissue with all the cells  
142:13 functioning in the right way.  
142:14 If, when the cell replicates,  
142:15 it doesn't fix that DNA repair, then -- if

142:16 you remember from high school biology, DNA is  
142:17 two strands. They wrap around each other  
142:18 like this, you know. When cells replicate,  
142:19 they break the strands, and then the  
142:20 individual strands replicate again so that  
142:21 you get two strands.  
142:22 Well, if this one's damaged,  
142:23 the sequence is different than that one.  
142:24 When it replicates, it replicates the damage.  
142:25 So now it's got a changed sequence over the  
143:1 other one. That's a mutation. So now that  
143:2 cell is a mutated cell.  
143:3 Q. So in this diagram, is that  
143:4 right here, the mutated cells?  
143:5 A. Correct.  
143:6 Q. Okay.  
143:7 A. That cell is very unlikely to  
143:8 be able to go back and become normal. It's  
143:9 going to remain being a mutated cell. And  
143:10 that process can repeat itself over and over  
143:11 again.  
143:12 Now, if we can go to the next  
143:13 slide...  
143:14 Q. Oh, the next slide.  
143:15 A. That one, correct. 885.  
143:16 Q. The next page?  
143:17 A. Oh, I'm sorry, the next page.  
143:18 Q. Okay.  
143:19 A. I think it's -- there should be  
143:20 another one.  
143:21 Q. I have it. It's 889. Or 890.  
143:22 Is that it?  
143:23 A. Correct.  
143:24 Now you're looking at how  
143:25 external things can affect this process. So  
144:1 a chemical, which is the thing at the  
144:2 bottom -- there you go. Chemicals can come  
144:3 in and change the rate at which cells get DNA  
144:4 damage. So the chemical itself can damage  
144:5 the cell or it can change the functioning of

144:6 the cell such that the damage is not repaired  
144:7 appropriately. But whatever the case, a  
144:8 chemical, by changing that rate, can increase  
144:9 the probability of a mutation.  
144:10 Q. So let me just slow you down  
144:11 there.  
144:12 So we have on this diagram here  
144:13 this chemical. Is that what you're referring  
144:14 to?  
144:15 A. Correct.  
144:16 Q. And then you're saying it can  
144:17 affect actual DNA damage?  
144:18 A. Correct.  
144:19 Q. It can affect replication?  
144:20 A. Correct.  
144:21 Q. And it can affect the  
144:22 uncontrolled growth?  
144:23 A. It can affect several things,  
144:24 but if it affects oxidative stress or DNA  
144:25 damage, genotoxicity, or it affects DNA  
145:1 repair down here, or it affects cellular  
145:2 replication without DNA repair, if it affects  
145:3 any of those three things adversely, then you  
145:4 can get an increased risk of a mutation.  
145:5 Q. Okay.  
145:6 A. Okay?  
145:7 Q. So hold on. You're using a lot  
145:8 of terms here. We have to define them all.  
145:9 Oxidative stress, what's that?  
145:10 A. So oxygen is common to cells.  
145:11 We breathe oxygen. There's a reason for it.  
145:12 We need it. It's the -- it's part of the  
145:13 energy that drives our bodies.  
145:14 Oxygen typically likes to bind  
145:15 to things, but when it's not bound, it's --  
145:16 it's wanting to bind to something. So think  
145:17 of it as a magnet next to metal. It wants to  
145:18 bind to the metal. That's an oxygen radical.  
145:19 It's not quite balanced because it isn't  
145:20 bound to anything.

145:21 Oxidative stress means that  
145:22 your cell has more oxygen radicals, unbound  
145:23 oxygen, than it normally should have. It's  
145:24 higher than it should be. And you can cause  
145:25 that in a number of ways, one of which is  
146:1 through chemical exposures.  
146:2 Q. Okay. So --  
146:3 A. And when that oxygen, that free  
146:4 oxygen, is running around and not bound to  
146:5 things it should bind to, it binds to things  
146:6 it shouldn't bind to, like DNA. And when it  
146:7 binds to DNA or parts of the -- to the  
146:8 machinery that works with DNA, it can affect  
146:9 the whole system and mess it up.  
146:10 Q. Okay. We're going to talk a  
146:11 lot more about oxidative stress and DNA  
146:12 damage later, but for now, how does this  
146:13 relate to that -- where we started,  
146:14 initiation and promotion studies?  
146:15 A. So that's what I wanted to get  
146:16 to. In toxicology chemical parlance, if a  
146:17 chemical causes an increase in mutations,  
146:18 it's called an initiator. So it is starting  
146:19 the chemical process. It's ini -- the cancer  
146:20 process. It is initiating the process.  
146:21 If the chemical comes in and  
146:22 enhances the process, so it takes something  
146:23 that's already started and makes it go  
146:24 faster, then it's called a promoter. It's  
146:25 promoting something that's already going on.  
147:1 So an initiator causes this  
147:2 mutation. A promoter enhances that mutation  
147:3 and makes it even come out more later to get  
147:4 more cancers.  
147:5 So an initiation/promotion  
147:6 study is one where you take a chemical that's  
147:7 an initiator, you give it to the animal for a  
147:8 short period of time, hopefully causing  
147:9 startup mutations in the animals, and then  
147:10 you come with another chemical, a promoter,

147:11 and you give it for a longer period of time,  
147:12 and that enhances that mutation and you begin  
147:13 to see the cancer.  
147:14 So a classical  
147:15 initiation/promoter study is used to try to  
147:16 understand some basic mechanisms of chemicals  
147:17 in causing cancer. If I have a chemical that  
147:18 I think might be an initiator, then I do a  
147:19 study where I give the animal that chemical  
147:20 for a short period of time, and then I --  
147:21 there are known promoters that we already  
147:22 know exist, and so then I give those same  
147:23 animals a promoter for a period of time and  
147:24 look to see if I see more cancers.  
147:25 If I do, then this was probably  
148:1 an initiator, the chemical I'm looking at.  
148:2 If I don't, then it's probably not an  
148:3 initiator. In this system at least.  
148:4 If I think the chemical is a  
148:5 promoter, then I give a classic initiator,  
148:6 something I already know will cause  
148:7 mutations, and then I follow it with this new  
148:8 chemical for a period of time and look to see  
148:9 if I see cancers.  
148:10 Okay. If you don't know  
148:11 anything about the chemical, you do both.  
148:12 You give it as an initiator with a classic  
148:13 promoter, you give it as a promoter with a  
148:14 classic initiator, and you see what happens.  
148:15 The George study, the one  
148:16 remaining study, is an initiation/promotion  
148:17 study with glyphosate.  
148:18 Q. Okay. Stop right there. Let  
148:19 me ask you some questions.  
148:20 A. Okay.  
148:21 Q. All right. Let's talk about  
148:22 the George study. If you turn to your binder  
148:23 to 559.  
148:24 A. Okay.  
148:25 Q. Is that a fair and accurate

149:1 copy of the George study?  
149:2 A. Yes, it is.  
149:3 Q. Okay. Great.  
149:4 So now it's up on the screen,  
149:5 and I just want to walk through a little bit  
149:6 what this says and ask you what it means.  
149:7 So the title of the document is  
149:8 "Studies on Glyphosate-Induced  
149:9 Carcinogenicity in Mouse Skin: A Proteomic  
149:10 Approach."  
149:11 What does that mean?  
149:12 A. It's proteomic.  
149:13 Q. Okay.  
149:14 A. So the key words here, it's  
149:15 glyphosate. They're looking for  
149:16 carcinogenicity. The study is not being done  
149:17 like the ones we looked at. This is done on  
149:18 mouse skin. So instead of the mouse eating  
149:19 the glyphosate, it's painted onto their skin.  
149:20 A. proteomic approach means that  
149:21 they're going to look at changes in proteins  
149:22 in the skin at the end of the study.  
149:23 Q. Okay. Great.  
149:24 And in this study it reads,  
149:25 "Glyphosate is a widely used, broad spectrum  
150:1 herbicide reported to induce various toxic  
150:2 effects in nontarget species, but its  
150:3 carcinogenic potential is still unknown.  
150:4 Here we showed the carcinogenic effects of  
150:5 glyphosate using two-stage mouse skin  
150:6 carcinogenesis model and proteomic analysis.  
150:7 Carcinogenicity study revealed that  
150:8 glyphosate has a tumor-promoting activity."  
150:9 Can you translate what I just  
150:10 read into English?  
150:11 A. The first sentence is obvious  
150:12 in their opinion.  
150:13 The second sentence deals with  
150:14 what they call a two-stage mouse skin  
150:15 carcinogenesis model. That is

150:16 initiation/promotion. First stage is  
150:17 initiation.  
150:18 Q. I see.  
150:19 A. Second stage is promotion.  
150:20 It's in the mouse skin, so they call that a  
150:21 two-stage mouse carcinogenicity study.  
150:22 Proteomic analysis is --  
150:23 Q. The protein?  
150:24 A. -- much more complicated.  
150:25 Q. Okay. And then it says,  
151:1 "Carcinogenicity study revealed that  
151:2 glyphosate has tumor-promoting activity."  
151:3 What does that mean?  
151:4 A. It means in this two-stage  
151:5 model where you give a known initiator and  
151:6 follow it with glyphosate for a fixed period  
151:7 of time, you see more skin tumors -- in this  
151:8 case they are skin papillomas -- than you  
151:9 would normally see, and so the glyphosate is  
151:10 promoting out the tumors that were started  
151:11 with the initiator.  
151:12 Q. All right. Now, I just want to  
151:13 turn to the second page here. This is -- it  
151:14 says, "Materials and Methods."  
151:15 Do you see that?  
151:16 A. Yes.  
151:17 Q. It says, "The commercial  
151:18 formulation of the herbicide glyphosate,  
151:19 Roundup original, copyright glyphosate  
151:20 41 percent, POEA, 15 percent, Monsanto  
151:21 Company, St. Louis, Missouri, was used."  
151:22 Is that your understanding in  
151:23 this study?  
151:24 A. Yes, that's -- that's the  
151:25 compound that was being painted on the  
152:1 animals.  
152:2 Q. So this -- is this different  
152:3 than pure technical glyphosate?  
152:4 A. Yes, this is different than  
152:5 pure technical glyphosate.

152:13 - 153:20

**Portier, Christopher 02-21-2019 (00:01:37)**

CP1\_SS\_01.43

152:13 Q. Okay. And then we have here  
152:14 all these different treatment groups. And I  
152:15 don't want to spend too much time on it, but  
152:16 you see Group 1, Group 2, Group 3.  
152:17 Do you see that?  
152:18 A. Yes.  
152:19 Q. And the one that I'm interested  
152:20 in is this Group 7 -- or Group 8, I'm sorry.  
152:21 It says, "DMBA plus glyphosate. Single  
152:22 topical application of DMBA followed one week  
152:23 later by topical treatment of glyphosate."  
152:24 Do you see that?  
152:25 A. Correct.  
153:1 Q. What is that referring to?  
153:2 A. DMBA is a chemical. It's a  
153:3 known initiator. So they're initiating the  
153:4 skin with DMBA and following it with  
153:5 glyphosate applications three times per week,  
153:6 25 milligrams per kilogram body weight on the  
153:7 backs of the mice.  
153:8 Q. And if we go to the results,  
153:9 it's on Table 1. And we see here that that  
153:10 group, Group 8, the DMBA plus glyphosate,  
153:11 what percentage of the animals had tumors on  
153:12 their skin?  
153:13 A. 8 out of 20 animals had  
153:14 papillomas on their backs.  
153:15 Q. And what percentage is that?  
153:16 A. Let's see. 40 percent.  
153:17 Q. Okay. And if you look at the  
153:18 rest of the results, the only other one that  
153:19 had tumors in the skin was Group 3.  
153:20 What does that reflect?

153:23 - 155:11

**Portier, Christopher 02-21-2019 (00:01:27)**

CP1\_SS\_01.44

153:23 THE WITNESS: Group 3 is the --  
153:24 what's called a positive control in  
153:25 this study. DMBA, the same initiator  
154:1 as they used with glyphosate, plus  
154:2 TPA. TPA is a known promoter, very

154:3 strong promoter, so that you would  
 154:4 expect to see lots of tumors. And  
 154:5 there they're seeing tumors in all the  
 154:6 animals.

154:7 QUESTIONS BY MR. WISNER:

154:8 Q. Okay. And if you look down  
 154:9 here, there's an asterisk on the Group 8, the  
 154:10 glyphosate group.

154:11 Do you see that?

154:12 A. Yes.

154:13 Q. And then it says, "P value less  
 154:14 than .5."

154:15 Do you see that?

154:16 A. Yes.

154:17 Q. "Versus untreated group"?

154:18 A. Yes.

154:19 Q. You mentioned P values earlier.

154:20 And in as simple terms as you can, what is a  
 154:21 P value?

154:22 A. It's the probability that the  
 154:23 observation you're seeing agrees with no  
 154:24 effect. So in this case it's the probability  
 154:25 that there's no increase in tumors from  
 155:1 glyphosate being used as a promoter in this  
 155:2 study.

155:3 If that probability is very  
 155:4 small, you reject the hypothesis that there's  
 155:5 no increase in favor of an alternative that  
 155:6 there in fact is an increase.

155:7 Q. So with this being a  
 155:8 statistically significant result, what does  
 155:9 that show you as a scientist?

155:10 A. That it's possible glyphosate  
 155:11 is a promoter of carcinogenesis.

155:12 - 155:14

**Portier, Christopher 02-21-2019 (00:00:03)**

CP1\_SS\_01.45

155:12 Q. And in this context we're  
 155:13 talking about commercial Roundup?

155:14 A. Correct.

155:18 - 155:24

**Portier, Christopher 02-21-2019 (00:00:16)**

CP1\_SS\_01.46

155:18 Q. All right. So let's -- let's

155:19 go back -- well, let's go back to this rat  
155:20 study, if you go back to the document camera.  
155:21 You know, in this rat study we  
155:22 have these repeated findings of skin tumors.  
155:23 Do you see that?  
155:24 A. Yes.

156:3 - 158:24

**Portier, Christopher 02-21-2019 (00:03:02)**

CP1\_SS\_01.47

156:3 Q. What, if anything, does this  
156:4 indicate to you as a scientist?  
156:5 A. In terms of the relationship to  
156:6 the skin painting study that was done, it  
156:7 would be far too speculative for me to go  
156:8 there.  
156:9 Q. Okay.  
156:10 A. In one case they're papillomas.  
156:11 These are skin keratoacanthomas. They're  
156:12 different mouse strains. The other study is  
156:13 very tailored for -- the initiation/promotion  
156:14 study is very tailored for a very fixed  
156:15 result.  
156:16 It would be too speculative for  
156:17 me to say they're related in any way.  
156:18 Q. Okay. Well, then let me ask  
156:19 you this question. The George study, this  
156:20 positive finding there, what -- what -- is  
156:21 that consistent with what you're seeing in  
156:22 the rodent data for glyphosate?  
156:23 A. Partially. Obviously it's --  
156:24 it's addressing the question of promotion,  
156:25 which means that you already have these  
157:1 initiated cells. Living can cause mutations  
157:2 to occur. And so it's conceivable that  
157:3 glyphosate, all of these tumor findings we  
157:4 are seeing here, are glyphosate promoting out  
157:5 already effects. I don't think it's likely,  
157:6 but it's conceivable that's the case.  
157:7 The initiation/promotion study  
157:8 is simply showing you that in one system, the  
157:9 skin, glyphosate has this ability to promote  
157:10 out cancer. That's all it really means.

157:11 Q. Well, let's -- hypothetically  
157:12 speaking, let's say an individual has a  
157:13 mutated cell caused by, like you said, life,  
157:14 or like a viral infection or something. Does  
157:15 the George study -- I don't know. You tell  
157:16 me. Does it have any influence on whether or  
157:17 not it could promote a mutation to lead to  
157:18 cancer?  
157:19 A. It certainly increases the  
157:20 chances that that might be the case because  
157:21 now you have evidence to suggest glyphosate  
157:22 can do that -- this. But I'd want to see a  
157:23 lot more evidence before I'd go there and  
157:24 start thinking about that.  
157:25 There are initiation/promotion  
158:1 studies you can do in the liver. There are  
158:2 initiation/promotion studies you can do in  
158:3 the brain. I'd like to see a little more  
158:4 work along those lines.  
158:5 And then looking at the other  
158:6 mechanistic evidence, I'd have to conclude  
158:7 that even though it wasn't an initiator in  
158:8 the skin, I'd want to look more closely at  
158:9 why it didn't come out as an initiator in the  
158:10 skin because theoretically it probably should  
158:11 have.  
158:12 Q. Okay. You mentioned that you'd  
158:13 like to see more initiation and promotion  
158:14 studies in other sort of organs.  
158:15 Have any of those been done?  
158:16 A. Not that I'm aware of. I would  
158:17 have hopefully picked them up in my search of  
158:18 the literature, and I haven't seen any.  
158:19 Q. Okay. All right. So going  
158:20 back to our causation stool here, we spent  
158:21 some time on animal studies. And we talked  
158:22 about the initiation and promotion study, and  
158:23 that kind of got us into this next section,  
158:24 which is the mechanism studies.

161:4 Q. We're talking about the  
161:5 mechanistic studies.  
161:6 How many known mechanisms are  
161:7 there between a known carcinogen and a cause  
161:8 in cancer?

161:9 A. It depends on how you want to  
161:10 break that down, but we recently wrote a  
161:11 paper that looks at ten different classes,  
161:12 let's call them classes, of mechanisms that  
161:13 we think relate to starting the  
161:14 carcinogenesis process or chemically  
161:15 modifying the carcinogenesis process.

161:16 Q. And for the purposes of  
161:17 glyphosate, how many have you looked at  
161:18 closely?

161:19 A. Two of those have sufficient  
161:20 data for us to really evaluate them for  
161:21 glyphosate.

161:22 Q. And what are those two?

161:23 A. One is DNA damage, causing DNA  
161:24 damage. The other is oxidative stress.

161:25 Q. And when you say "DNA damage,"  
162:1 is another term for that genotoxicity?

162:2 A. Yeah, that is another term for  
162:3 it, although genotoxicity can go beyond DNA  
162:4 damage. DNA damage is a subclass of the  
162:5 fuller class of genotoxicity.

162:6 Q. Okay. And I -- you know, I  
162:7 just want to make sure I understand. When  
162:8 you look at this cancer causation stool that  
162:9 we're talking about here, how important are  
162:10 the mechanistic studies, in your view?

162:11 A. Well, I was going to get back  
162:12 to your stool because the stool seems to  
162:13 imply that if you don't have one of these  
162:14 legs, the whole thing falls down.  
162:15 That's not true here. Having a  
162:16 mechanism strengthens the other data in terms  
162:17 of supporting a carcinogenic finding. Not  
162:18 knowing the mechanism doesn't subtract. It

162:19 simply leaves a question mark in your head  
162:20 about, well, how strong is this. So it  
162:21 may -- you won't have as strong of a finding,  
162:22 but you'll still have the finding there.  
162:23 There are a number of  
162:24 interesting carcinogens which the mechanism  
162:25 wasn't worked out until long after we were  
163:1 absolutely certain it was happening because  
163:2 we just couldn't find it out.  
163:3 Q. But here with glyphosate, have  
163:4 we figured out some mechanisms?  
163:5 A. We have indications of  
163:6 processes that support a mechanism that  
163:7 probably would work for glyphosate. I would  
163:8 not go so far as to say I'm absolutely  
163:9 certain this is exactly how the mechanism  
163:10 occurs.  
163:11 I'm absolutely certain it does  
163:12 certain things and that those things can lead  
163:13 to a carcinogenic finding, but I'm not  
163:14 absolutely certain that those mechanisms are  
163:15 the ones that are driving the carcinogenic  
163:16 finding for glyphosate.  
163:17 Q. Okay. Well, let's talk about  
163:18 the two that we've looked at. The first one  
163:19 was genotoxicity.  
163:20 I'd like to draw your attention  
163:21 to Exhibit 886 in your binder.  
163:22 And this is a picture that we  
163:23 put together to help explain genotoxicity; is  
163:24 that right?  
163:25 A. Yes. That's not what's on the  
164:1 screen, but...  
164:2 Q. I just wanted you to verify it,  
164:3 and then I'll put it on the screen.  
164:4 A. That's a specific type of  
164:5 genetic damage, DNA damage.  
164:6 Q. Perfect.  
164:7 So we have this picture up  
164:8 here, and I just kind of walk the jury

164:9 through what we're seeing here.  
164:10 So on the first thing we have a  
164:11 single-strand break. What's that referring  
164:12 to?  
164:13 A. Oh, you've got a -- yeah, I now  
164:14 see. You've got a whole bunch of different  
164:15 types of DNA damage here.  
164:16 Single-strand break means --  
164:17 like I said, DNA is double-twisted. It's a  
164:18 helix. So what you're looking at here with  
164:19 the bands of ribbon going around is a picture  
164:20 of what looks like DNA.  
164:21 A. single-strand break means you  
164:22 went in with something like a scissor and you  
164:23 cut one of the DNA strands.  
164:24 Q. Is that this area that I'm  
164:25 referring to?  
165:1 A. Yes.  
165:2 Q. Okay. And then we have  
165:3 mismatch.  
165:4 Do you see that?  
165:5 A. Correct.  
165:6 Q. What's that refer to?  
165:7 A. So DNA has these chemicals in  
165:8 it. There are four basic chemicals, and they  
165:9 tend to complement each other. On -- if one  
165:10 strand of DNA has -- let's give them letters.  
165:11 One is an A, one is a T, the two chemicals.  
165:12 If this strand of DNA has an A  
165:13 on it, the other strand of DNA will have a T  
165:14 on it. And they match together and they  
165:15 bind, and that's what makes this sort of  
165:16 ladder effect going up the DNA.  
165:17 But sometimes when the cell  
165:18 tries to repair itself, to repair the DNA, it  
165:19 mismatches. And so instead of putting an A  
165:20 across from a T, there may be another  
165:21 chemical, a molecule, in the cell called G --  
165:22 let's call it that -- and it's a G and a T,  
165:23 and they don't exactly fit together. So

165:24 that's a mismatch, and that happens with  
165:25 repair. That's a known DNA damage, mismatch  
166:1 repair.

166:2 Q. All right. And then we have  
166:3 all these other different mechanisms.

166:4 A. Correct.

166:5 Q. We have -- I want to talk about  
166:6 these cross-links.

166:7 What do these cross-links refer  
166:8 to?

166:9 A. So instead of the A and the T  
166:10 matching each other across the DNA, instead  
166:11 this T matches to that T and they -- they  
166:12 bind on the same DNA, and the two on the  
166:13 bottom might bind or not bind. So you're  
166:14 cross-linking within a single strand of DNA  
166:15 instead of across the DNA.

166:16 Q. Okay. And then down here we  
166:17 have a photograph or a picture of a  
166:18 micronucleus.

166:19 What is that?

166:20 A. So when you have some of these  
166:21 types of DNA damage, when the cell goes in to  
166:22 try to repair it, it ends up cutting off a  
166:23 piece of DNA, and it pulls it off to the side  
166:24 and you get these little micronuclei which  
166:25 indicate that DNA damage has been repaired.  
167:1 The more micronuclei you have,  
167:2 the more chances are that you have DNA damage  
167:3 that's unrepaired. So people measure  
167:4 micronuclear as a means of measuring  
167:5 potential DNA damage.

167:6 Q. All right. So when we look at  
167:7 these different types of genetic damage, are  
167:8 there different tests that measure different  
167:9 types of genetic damage?

167:10 A. Yes, there are. They can get  
167:11 very specific in terms of doing the types of  
167:12 damage you want to look at. Yeah, there are  
167:13 tests.

167:14 Q. Okay. All right. I want to --  
167:15 I prepared sort of a demonstrative to help us  
167:16 walk through -- sort of understanding  
167:17 genotoxicity data. This is Exhibit 887. And  
167:18 I want to sort of break things down for the  
167:19 jury. Okay?  
167:20 So are you familiar with the  
167:21 terms "in vivo" and "in vitro"?  
167:22 A. Yes, I am.  
167:23 Q. What do they refer to?  
167:24 A. In vivo refers to in the living  
167:25 organism, in viventem or whatever. It's a  
168:1 Latin term. Living organism.  
168:2 Q. All right. I wrote living  
168:3 there.  
168:4 And in vitro refers to what?  
168:5 A. In cells.  
168:6 Q. Okay. And is that often called  
168:7 a petri dish?  
168:8 A. Well, it's in cells,  
168:9 independent of the living organism.  
168:10 Q. So I'll put cells?  
168:11 A. Yeah.  
168:12 Q. Okay. Great.  
168:13 A. And that can be in a petri dish  
168:14 or in a flask or whatever.  
168:15 Q. A test tube or something?  
168:16 A. A test tube.  
168:17 Q. Okay. So we have in vivo and  
168:18 in vitro.  
168:19 Are there different types of  
168:20 tests that were done?  
168:21 A. Yes.  
168:22 Q. Okay.  
168:23 A. You wouldn't -- you wouldn't  
168:24 generally do the same test in living  
168:25 organisms that you do in cells in a petri  
169:1 dish.  
169:2 Q. All right. And then these  
169:3 different types of tests, are they done on

169:4 glyphosate in formulation?  
169:5 A. They can be.  
169:6 Q. Okay. And in the data that  
169:7 you've reviewed, have there been generally  
169:8 studies done on glyphosate and formulations?  
169:9 A. Correct. Both in vivo and in  
169:10 vitro.  
169:11 Q. All right. Okay. So then  
169:12 within the in vivo studies and the in vitro  
169:13 studies, are there studies done on different  
169:14 types of species?  
169:15 A. Yes, absolutely.  
169:16 Q. And how would you categorize  
169:17 those groups?  
169:18 A. Well, there are in vivo studies  
169:19 in humans.  
169:20 Q. Okay.  
169:21 A. There are in vivo studies in  
169:22 other mammals. And then there are in vivo  
169:23 studies in other animals and other things  
169:24 that are not mammals. So that can include  
169:25 bacteria and salmonella stuff, as well as  
170:1 fish and other things.  
170:2 Q. All right.  
170:3 A. Other animals/other stuff.  
170:4 Q. All right. I wrote "other  
170:5 non-mammals." Is that okay?  
170:6 A. That's fine.  
170:7 Q. Okay. Great.  
170:8 So it looks like then, when you  
170:9 look at the data here, there's in vivo, in  
170:10 vitro, glyphosate and formulations, and then  
170:11 the three categories of species in both --  
170:12 all four of those.  
170:13 A. Right, because you can derive  
170:14 cells from humans, you can derive cells from  
170:15 mammals that are not humans, and you can  
170:16 derive cells from other mammals.  
170:17 The main difference -- the only  
170:18 one is that in the in vitro side you can also

170:19 have single cellular organisms.  
170:20 Q. Oh, okay.  
170:21 A. Like bacteria.  
170:22 Q. Okay.  
170:23 A. Which you wouldn't put in the  
170:24 in vivo living side of it.  
170:25 Q. All right. So I put on  
171:1 bacteria as well. Okay. Great.  
171:2 For the purposes of sort of  
171:3 understanding the mechanism of carcinogenesis  
171:4 for glyphosate, what categories of species  
171:5 and formulation of glyphosate is the most  
171:6 helpful for understanding?  
171:7 A. Well, that's a tough question.  
171:8 If you're wanting to just look  
171:9 at glyphosate, if I wanted to address the  
171:10 question is glyphosate carcinogenic, then  
171:11 obviously I would look at the glyphosate  
171:12 studies.  
171:13 Irregardless, whether it's  
171:14 glyphosate or a formulation, I would rank  
171:15 human in vivo studies number one.  
171:16 Q. All right.  
171:17 A. That would clearly get my  
171:18 greatest attention because those studies are  
171:19 in the right organism, and they're in the  
171:20 living organism.  
171:21 Number two is a little tougher  
171:22 to call because in vitro studies in human  
171:23 cells are the right organism, but they're in  
171:24 cells in a petri dish so it's kind of removed  
171:25 from the human situation, the full working  
172:1 human situation, but still human cells in a  
172:2 petri dish.  
172:3 On the other hand, if I study  
172:4 mammals, it's in the living organism, and so  
172:5 that's closer to a living, breathing human  
172:6 being than cells in a petri dish.  
172:7 So it's hard for me to rank  
172:8 those two other than to say I'm going to

172:9 consider them both about the same importance.  
 172:10 So they would both get my number two ranking.  
 172:11 And then everything else is  
 172:12 falling down below that. Cellular studies in  
 172:13 mammals are interesting and important, but  
 172:14 they're not as interesting and important as  
 172:15 the human cellular studies.  
 172:16 Other mammals -- or other  
 172:17 non-mammal animals, studies in them are  
 172:18 important, but because they're so far removed  
 172:19 from the human experience, they're less  
 172:20 important than mammals that are closer to  
 172:21 humans.

172:22 - 174:20

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172:22 Q. Well, what about, for example,  
 172:23 the number one, in vivo human studies, so  
 172:24 living people studies. Are there different  
 172:25 levels of importance relative to what you're  
 173:1 studying in the human?  
 173:2 A. Yes. Yes.  
 173:3 Different studies carry  
 173:4 different quality of information. I'm going  
 173:5 to go to a slightly different subject for a  
 173:6 second to illustrate this. Tobacco's a good  
 173:7 example.  
 173:8 So there's all kinds of  
 173:9 different studies about smoking. One of the  
 173:10 most important smoking studies that was ever  
 173:11 done to really honestly prove beyond a shadow  
 173:12 of a doubt that smoking can cause lung cancer  
 173:13 was the study with doctors in the UK. And  
 173:14 what they did was they got the doctors to  
 173:15 quit smoking, some, and some didn't. And  
 173:16 what they were able to prove was that when  
 173:17 doctors quit smoking, their lung cancer rates  
 173:18 were lower than the doctors who continued to  
 173:19 smoke.  
 173:20 So you could show that doctors  
 173:21 who smoked got cancer at a certain rate. You  
 173:22 could show that doctors who never smoked got

Page/Line	Source	ID
	<p>173:23 cancer at another rate. And then you can  173:24 show that doctors who quit smoking, their  173:25 cancer risk went almost back down to the  174:1 nonsmokers if they quit early enough. And  174:2 that's a really strong study because you've  174:3 intervened in a human population and shown  174:4 that your intervention makes a big  174:5 difference.  174:6 Now, I can't do a study where I  174:7 force people to smoke and force some people  174:8 not to smoke and control everything else and  174:9 have them smoke, so I can't do that. But I  174:10 can do these intervention studies. We don't  174:11 have that here, but that's a strong study.  174:12 There are also weaker studies  174:13 than even the one where you look at smokers  174:14 versus nonsmokers. There are studies where  174:15 you look at Russians smoke more than  174:16 Americans. Let's look at Russian lung cancer  174:17 versus American lung cancer. That type of  174:18 study is a much more weaker study. So it  174:19 depends on the type of study you're looking  174:20 at.</p>	
175:19 - 175:20	<b>Portier, Christopher 02-21-2019 (00:00:01)</b>	CP1_SS_01.50
	<p>175:19 Did you want to say something  175:20 else, sir?</p>	
175:25 - 176:2	<b>Portier, Christopher 02-21-2019 (00:00:05)</b>	CP1_SS_01.51
	<p>175:25 THE WITNESS: The -- this is  176:1 discussed in my expert report with the  176:2 tobacco example and references.</p>	
176:6 - 176:9	<b>Portier, Christopher 02-21-2019 (00:00:07)</b>	CP1_SS_01.52
	<p>176:6 What about the actual organs  176:7 and cells that you're looking at, I mean,  176:8 does that influence your understanding of the  176:9 study?</p>	
176:12 - 182:13	<b>Portier, Christopher 02-21-2019 (00:05:49)</b>	CP1_SS_01.53
	<p>176:12 THE WITNESS: The -- the --  176:13 when you do these in vitro studies,  176:14 and even in the in vivo studies, yes,  176:15 it matters which target -- which</p>	

176:16 organs and cells you're looking at.  
176:17 QUESTIONS BY MR. WISNER:  
176:18 Q. So we're here to talk about  
176:19 glyphosate and non-Hodgkin's lymphoma.  
176:20 What would be the best thing to  
176:21 look at for whether or not mechanistically  
176:22 they're causing lymphoma?  
176:23 A. Well, you'd think you'd want to  
176:24 look at human systems and you'd want to look  
176:25 at hematopoietic cells, so cells that make up  
177:1 the blood, the lymphatic system. And there's  
177:2 a whole variety of cells that play a role in  
177:3 that system.  
177:4 Q. Okay. So turning to our sort  
177:5 of data over here on genotoxicity, are there  
177:6 any pure glyphosate in vivo human studies?  
177:7 A. No, there are not.  
177:8 Q. Are there any formulation in  
177:9 vivo human studies that look at genetic  
177:10 damage?  
177:11 A. Yes, there are.  
177:12 Q. Okay. And how many studies  
177:13 have looked at that?  
177:14 A. There are three studies that  
177:15 I'm aware of.  
177:16 Q. And one study was -- who were  
177:17 they done by?  
177:18 A. Two of them were done by a  
177:19 researcher whose last name is Paz-y-Miqo, and  
177:20 the third was done by a researcher called  
177:21 Bolognesi.  
177:22 Q. All right. Well, let's start  
177:23 up with Dr. Paz-y-Miqo.  
177:24 A. Okay.  
177:25 Q. What did that study show?  
178:1 A. The first study by Paz-y-Miqo  
178:2 was like my Russian versus US study. He  
178:3 looked at or she -- I actually don't know.  
178:4 Dr. Paz-y-Miqo looked at a group of people  
178:5 who lived near an area that was sprayed with

178:6 glypho -- with a glyphosate formulation and  
178:7 another group of people who lived  
178:8 80 kilometers away in an area that didn't  
178:9 experience any spraying.  
178:10 They asked questions to make  
178:11 sure there weren't other obvious things in  
178:12 the environment that might explain a  
178:13 difference.  
178:14 And then they went and took  
178:15 blood from those people who were in both  
178:16 locations and looked for DNA damage in the  
178:17 peripheral -- in that blood of those people.  
178:18 I think it was in lymphocytes.  
178:19 And they saw a significant  
178:20 difference with the people living near the  
178:21 sprayed area having more DNA damage than  
178:22 those living further away.  
178:23 Q. And non-Hodgkin's lymphoma, is  
178:24 that a blood cancer?  
178:25 A. It's a cancer of the  
179:1 hematopoietic system, yes. It's part of that  
179:2 whole system.  
179:3 Q. Did Dr. -- did Dr. Paz-y-Miqo  
179:4 do a follow-up study with these people?  
179:5 A. He did a follow-up study. I  
179:6 don't think it's the same exact people, but  
179:7 he did a follow-up study and looked later.  
179:8 Instead of soon after spraying, he looked at  
179:9 multiple times after spraying and didn't see  
179:10 the same effect. It disappeared.  
179:11 Q. How much later did he look at  
179:12 it?  
179:13 A. I think it was a year, a year  
179:14 or two.  
179:15 Q. Okay.  
179:16 A. I'd have to go back to the  
179:17 paper.  
179:18 Q. And so when you're looking at  
179:19 the mechanistic data and you have one study  
179:20 showing that immediately after exposure to

179:21 formulated Roundup or formulated glyphosate  
179:22 there's genetic damage, and then that genetic  
179:23 damage disappears after a few years, what  
179:24 does that indicate to you?

179:25 A. Well, in human blood it would  
180:1 be expected unless there were continued  
180:2 exposure.

180:3 If the exposure was periodic --  
180:4 human blood turns over fairly rapidly. Six  
180:5 months, give or take, most of the cells in  
180:6 your blood system have turned over and gone  
180:7 away. So they're -- they're differentiated.  
180:8 Unless you're looking down in  
180:9 the bone marrow where the cells begin, you  
180:10 wouldn't expect to see the DNA damage sitting  
180:11 around for a long period of time.

180:12 Q. And for people who are using or  
180:13 being exposed to a formulated glyphosate  
180:14 repeatedly, every couple of weeks, what does  
180:15 that indicate based on the Paz-y-Miqo study?

180:16 A. It would indicate that you'd  
180:17 probably see DNA damage consistently higher  
180:18 in those people as compared to others.

180:19 Q. And when you consistently have  
180:20 increased or elevated rates of genetic  
180:21 damage, does that increase the likelihood of  
180:22 developing lymphoma?

180:23 A. That is the theory, and that is  
180:24 usually what would occur, but there's  
180:25 absolutely no guarantee. It's part of the  
181:1 theoretical belief of how cancer arises.

181:2 Q. And you said there was another  
181:3 study that was done also in humans using  
181:4 formulations; is that right?

181:5 A. Correct.

181:6 Q. What was -- who did that study?

181:7 A. That study was done by  
181:8 Dr. Bolognesi, and that's a different study.

181:9 Q. What did that -- how was that  
181:10 study different?

181:11 A. Well, that study is, in my  
181:12 opinion, a stronger study. In this case, in  
181:13 the -- in the Paz-y-Miqo study, you're  
181:14 actually comparing communities. That's your  
181:15 sort of comparison group.  
181:16 Here, what Dr. Bolognesi did  
181:17 was they knew there was going to be spraying  
181:18 in the area, so they went and measured people  
181:19 for DNA damage before spraying and then after  
181:20 spraying. So they had five communities, four  
181:21 of them near areas that were going to be  
181:22 sprayed and one further away with no  
181:23 spraying, similar to Paz-y-Miqo, but they did  
181:24 before and after measurements.

181:25 And when you look at the  
182:1 analysis of the before and after, which is  
182:2 the strongest analysis, you see an increase  
182:3 of DNA damage after exposure -- after the  
182:4 spraying occurred, in the individual. You're  
182:5 comparing my now against my before. It's a  
182:6 much stronger comparison than my community  
182:7 against that community.

182:8 Q. Okay. And other than these  
182:9 three studies that look specifically at  
182:10 genetic damage in humans exposed to  
182:11 formulation products, has there been any  
182:12 other studies done?

182:13 A. Not that I'm aware of.

182:14 - 196:8

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CP1\_SS\_01.68

182:14 Q. Okay. And just looking at the  
182:15 in vivo human data, those three studies we  
182:16 just discussed, what does it tell you as a  
182:17 scientist?

182:18 A. It tells me that glyphosate  
182:19 formulations are -- can induce DNA damage.

182:20 Q. In human blood?

182:21 A. In human blood.

182:22 Q. Okay. Let's move on to the  
182:23 number 2 group. And I didn't prepare a chart  
182:24 for mammals, but I did look -- prepare a

182:25 chart for human in vitro studies.  
183:1 Okay?  
183:2 And have you looked at all the  
183:3 human in vitro studies that looked at  
183:4 glyphosate and formulations?  
183:5 A. Yes, I have.  
183:6 Q. And have you reviewed the  
183:7 peer-reviewed articles about that?  
183:8 A. Yes, I have.  
183:9 I also reviewed the -- any of  
183:10 the industry data that was available to me  
183:11 for review.  
183:12 Q. Okay. I want to take a look at  
183:13 our first chart here. This is Exhibit 874,  
183:14 sir. It's titled "Human in Vitro  
183:15 Genotoxicity Data."  
183:16 Do you see that?  
183:17 A. Yes, I see it.  
183:18 Q. And what does this chart  
183:19 reflect?  
183:20 A. So under the column "study" is  
183:21 the authors' name, or names, and the year in  
183:22 which the study occurred. All of these  
183:23 probably should have et als on them. There's  
183:24 more than one author.  
183:25 The second column reflects  
184:1 whether the study was done using glyphosate  
184:2 or, the third column, using a formulation.  
184:3 So the second column would be the findings  
184:4 for pure glyphosate, and the third column  
184:5 would be the findings for the formulation.  
184:6 Q. Okay. We have this key here on  
184:7 the right, a plus for positive.  
184:8 What does this key show?  
184:9 A. Well, if we're going to do what  
184:10 I think we're doing, we're going to sit down  
184:11 and put in positive, negatives. You see the  
184:12 NDs on there are already there. That means  
184:13 that in that particular study -- let's take  
184:14 the first one, Vigfusson and Vyse from 1980.

184:15 They studied only the formulation. They did  
184:16 not study the glyphosate pure forms. So  
184:17 there's no data on glyphosate pure in that  
184:18 study.  
184:19 Plus would mean it was a  
184:20 positive study in some way, shape or form,  
184:21 negative would mean it was a negative study  
184:22 completely, and ND means no data.  
184:23 Q. Okay. And then, for example,  
184:24 down here with Gasnier, Gasnier 2009, there's  
184:25 no ND.  
185:1 What does that mean?  
185:2 A. That means they studied both  
185:3 glyphosate and the glyphosate formulation. I  
185:4 will point out, however, that's wrong.  
185:5 In reviewing the way we did the  
185:6 chart, this chart, last night, Gasnier  
185:7 actually didn't do glyphosate. So there's no  
185:8 data on there for Gasnier. That's the  
185:9 only --  
185:10 Q. So I'll put an ND.  
185:11 A. -- one that's wrong.  
185:12 Q. Okay.  
185:13 A. It's an ND.  
185:14 Q. Okay. So I picked up on the  
185:15 one that was wrong. Okay.  
185:16 A. Bolognesi did both.  
185:17 Q. All right. What about Koller?  
185:18 A. Koller did both glyphosate and  
185:19 glyphosate formulations.  
185:20 Q. Okay. Great.  
185:21 Sir, how are you physically  
185:22 doing right now? Is this a good time for a  
185:23 break?  
185:24 A. 11:30. We can go to 12 --  
185:25 Q. Okay. Great.  
186:1 A. -- if you'd like.  
186:2 Q. Let's keep going.  
186:3 All right, sir. Well, let's go  
186:4 through these studies very quickly.

186:5 The first study. And I'll just  
186:6 call it the first study because I don't want  
186:7 to mispronounce these fine people's names.  
186:8 A. Okay.  
186:9 Q. The first study, was that  
186:10 positive or negative in the formulation?  
186:11 A. That was positive in the  
186:12 formulation.  
186:13 Q. Okay. Bolognesi 1997.  
186:14 A. Yes.  
186:15 Q. Was it positive in glyphosate?  
186:16 A. It was positive in glyphosate  
186:17 and positive in the formulation.  
186:18 Q. Lioi, 1998. In glyphosate,  
186:19 what was the results?  
186:20 A. Lioi, 1998, and it was  
186:21 positive.  
186:22 Q. Okay. Great.  
186:23 And the next one, 2004?  
186:24 A. Lueken did two different types  
186:25 of human cells. The previous ones did  
187:1 lymphocytes, but Lueken is looking at  
187:2 specifically cultured cells. He did two  
187:3 types of cultured cells.  
187:4 And it's a different study. I  
187:5 want to be fair here. They studied  
187:6 glyphosate with hydrogen peroxide. Now,  
187:7 hydrogen peroxide causes DNA damage. And  
187:8 what they were looking at was whether  
187:9 glyphosate, when you add it to hydrogen  
187:10 peroxide, makes it worse.  
187:11 Q. Gotcha.  
187:12 A. And it did.  
187:13 So when you say a positive  
187:14 here, it means that glyphosate, when added to  
187:15 hydrogen peroxide, made the DNA damage from  
187:16 hydrogen peroxide even worse.  
187:17 Q. Gotcha.  
187:18 A. Okay? So it was positive for  
187:19 both cell lines that they looked at.

187:20 Q. And there was two in there?  
187:21 A. There were two.  
187:22 Q. And you said these first three,  
187:23 they were all lymphocytes?  
187:24 A. There were all lymphocytes.  
187:25 Q. Human lymphocystic cells?  
188:1 A. Human lymphocytes from donors.  
188:2 Q. All right. I'm going to put an  
188:3 L next to those three.  
188:4 And if any of these other ones  
188:5 are lymphocytes, you let me know. Okay?  
188:6 A. Okay.  
188:7 Q. The next one, Munro 2005?  
188:8 A. Again, looking at two cell  
188:9 lines that are not lymphocytes, specific  
188:10 cultured cell lines, and both were positive.  
188:11 Q. Gasnier, there was no data for  
188:12 glyphosate, but for the formulation, what  
188:13 were the results?  
188:14 A. They claimed it was positive,  
188:15 but I have concerns about the study. I would  
188:16 call it inadequate.  
188:17 Q. So even though they said it was  
188:18 positive, you're saying you're not sure?  
188:19 A. I'm saying it's inadequate.  
188:20 I'm saying it's -- it's -- the way they did  
188:21 it, the limitations to the assay they used  
188:22 are such that -- and the way they presented  
188:23 the results are difficult to interpret  
188:24 appropriately. I think it's an inadequate  
188:25 study.  
189:1 Q. All right. So I'm going to put  
189:2 a question mark on it. Is that okay?  
189:3 A. That's perfect.  
189:4 Q. And then just because the  
189:5 authors, they concluded it was positive, I'll  
189:6 put that on there in parentheses.  
189:7 Okay?  
189:8 A. Okay.  
189:9 Q. And then Manas, 2009?

189:10 A. They did two different types of  
189:11 cells, one of which was lymphocytes --  
189:12 Q. Okay.  
189:13 A. -- and the other was a liver  
189:14 cancer cell line. The liver cancer cell line  
189:15 was positive; the lymphocytes were negative.  
189:16 Q. So we have a negative and a  
189:17 positive?  
189:18 A. Correct.  
189:19 Q. Okay. What about Mladinic? I  
189:20 said that wrong. Mladinic?  
189:21 A. I have no idea. Mladinic.  
189:22 That was lymphocytes. It was positive.  
189:23 Q. Okay. Now there's two here.  
189:24 Is this an error or --  
189:25 A. No, it's two separate  
190:1 publications, two separate sets of  
190:2 lymphocytes and two different ways of  
190:3 evaluating DNA damage.  
190:4 So the second publication was  
190:5 also lymphocytes, and it's also positive.  
190:6 Q. Koller 2012?  
190:7 A. That's a cell line, it's not  
190:8 lymphocytes. Both were positive, positive  
190:9 for glyphosate and positive for the  
190:10 formulation.  
190:11 Q. How about Alvarez-Moya, 2014?  
190:12 A. That was lymphocytes, and that  
190:13 was positive.  
190:14 Q. All right, sir. And I  
190:15 understand these were the studies that go  
190:16 through 2014; is that right?  
190:17 A. That is correct.  
190:18 Q. Have there been studies since  
190:19 then you've reviewed?  
190:20 A. Yes, there have been studies  
190:21 since then.  
190:22 Q. All right. Let's look at  
190:23 Exhibit 876. This is titled "Recent In Vitro  
190:24 Human Genotoxicity Data."

190:25 Do you see that, sir?  
191:1 A. Yes, I see that.  
191:2 Q. All right. We're going to do  
191:3 the same thing here. We're going to go  
191:4 through these studies, and we're going to see  
191:5 which ones were positive, negative, or I  
191:6 guess at least with one of these studies,  
191:7 uninterpretable.  
191:8 Okay?  
191:9 A. To be fair, these are 2017,  
191:10 2018 and 2019 is where I looked. I don't  
191:11 know if there are 2015, '16 and -- studies  
191:12 that I've missed. So to be fair, these are  
191:13 the most recent last two years.  
191:14 Q. Fair enough. So let's go  
191:15 through this.  
191:16 Townsend, 2017, this was on  
191:17 glyphosate. What was the results of that?  
191:18 A. That was positive.  
191:19 Q. And again, let me know if any  
191:20 of these are human lymphocytes.  
191:21 Okay?  
191:22 A. Okay.  
191:23 Q. Luo -- oh, by the way, just to  
191:24 go back, this Bolognesi study from 1997, is  
191:25 that a different study than the in vivo study  
192:1 we talked about earlier?  
192:2 A. I think they're connected.  
192:3 Q. Okay. So we have Luo 2017 in  
192:4 the formulated product.  
192:5 What were the results of that  
192:6 one?  
192:7 A. That was positive. But I will  
192:8 note in my opinion it's positive with a  
192:9 little bit of a question mark.  
192:10 Q. Okay. So I'm going to do a  
192:11 little question mark.  
192:12 A. Okay.  
192:13 Q. Okay.  
192:14 A. It's not as strong as some of

192:15 the others. I would -- if that was the only  
192:16 one I have, I would hesitate to use it.  
192:17 Q. Okay. The next one from 2017?  
192:18 A. This is leukocytes, not  
192:19 lymphocytes, so it's -- but it's drawn from  
192:20 human blood.  
192:21 Q. Okay. So I'll put "blood" on  
192:22 here.  
192:23 A. And that one was positive.  
192:24 Q. Okay. The next one from 2017,  
192:25 Kasuba?  
193:1 A. This one's positive. And the  
193:2 note -- the most notable thing about this one  
193:3 was it was positive at fairly low exposures.  
193:4 Q. Okay. Why is that important?  
193:5 A. They made -- they made a point  
193:6 of choosing exposures that they believed were  
193:7 at the levels that regulatory authorities  
193:8 were setting the exposures, setting the  
193:9 regulatory limits. And they made a big point  
193:10 of being very careful to match those  
193:11 exposures in doing their DNA damage studies.  
193:12 Q. And why is that relevant to  
193:13 your analysis?  
193:14 A. It's not really. It's relevant  
193:15 to the question of what happens at low --  
193:16 very low exposures, which is to some degree  
193:17 important in an evaluation of hazard.  
193:18 But in this case I'm being  
193:19 asked, is it possible that it can cause  
193:20 cancer, and the answer is yes. And I think  
193:21 the epidemiology studies speak very strongly  
193:22 to the question of can it occur in humans at  
193:23 the levels that we're currently exposed to.  
193:24 So I don't necessarily need  
193:25 this, but it is something to note from the  
194:1 study because it was important to them to  
194:2 note in doing their study.  
194:3 Q. Okay. This next one, Wozniak,  
194:4 2018?

194:5 A. That's, again, human  
194:6 leukocytes, so blood --  
194:7 Q. Okay.  
194:8 A. -- and it was positive for both  
194:9 the formulation and for glyphosate.  
194:10 Q. All right. The next one from  
194:11 2018?  
194:12 A. Santovito, that was  
194:13 lymphocytes. That one was positive as well.  
194:14 Q. Okay. 2018, the next one?  
194:15 A. De Almeida. They did three  
194:16 human cell lines.  
194:17 Q. Oh, wow.  
194:18 A. Two breast cancer cell lines  
194:19 and one endometrial cell line. That's the  
194:20 layer of cells that's sort of way below the  
194:21 basal part of the skin and other places in  
194:22 the body.  
194:23 It was negative for one of the  
194:24 breast cancer cell lines for glyphosate and  
194:25 positive for the other two, and it was  
195:1 negative for the same cell lines in the  
195:2 formulation and positive for the other two.  
195:3 So it's negative plus-plus in both cases.  
195:4 Q. Okay. Great.  
195:5 Then we have this next one from  
195:6 2018?  
195:7 A. This was human sperm, and it  
195:8 was negative.  
195:9 Q. Okay. All right, sir.  
195:10 So we're looking at these  
195:11 genotoxicity data that's in the peer-reviewed  
195:12 literature, and on the first chart here it's  
195:13 almost across the board positive. Again in  
195:14 the second chart, it's almost across the  
195:15 board positive.  
195:16 What significance does that  
195:17 have to you?  
195:18 A. Well, it's simply repeating the  
195:19 same thing over and over again, that

Page/Line	Source	ID
	195:20 glyphosate actually can cause DNA damage in 195:21 cells and so can the formulation. 195:22 Q. And I want to be very clear. 195:23 We've listed all these different studies 195:24 where there's lymphocytes involved. 195:25 Do you see that? 196:1 A. Yes. 196:2 Q. In your professional opinion 196:3 and expert opinion, do you believe that 196:4 glyphosate is genotoxic in human lymphocytes? 196:5 A. Yes. 196:6 Q. Do you believe the formulation 196:7 is genotoxic to human lymphocytes? 196:8 A. Yes.	
196:16 - 196:17	<b>Portier, Christopher 02-21-2019 (00:00:02)</b> 196:16 THE WITNESS: Santovito is 196:17 human lymphocytes.	CP1_SS_01.54
196:25 - 197:10	<b>Portier, Christopher 02-21-2019 (00:00:19)</b> 196:25 Q. Let's move on to the next 197:1 mechanism of carcinogenesis. 197:2 Well, actually, no, let's -- 197:3 let's actually stay with genotoxicity for a 197:4 second. I want to go back to that picture we 197:5 had up earlier. 197:6 And we were looking at these 197:7 different types of genetic damage, and we 197:8 spent some time talking about micronuclei. 197:9 Do you recall that? 197:10 A. Yes.	CP1_SS_01.56
198:10 - 198:20	<b>Portier, Christopher 02-21-2019 (00:00:19)</b> 198:10 Q. All right, sir. Just before 198:11 the break we were going back to this 198:12 genotoxicity diagram. This is Exhibit 886. 198:13 And I want to talk a little bit about the 198:14 micronucleus. 198:15 Okay? 198:16 A. Okay. 198:17 Q. Has there been -- and before we 198:18 get going, sir, how are you physically 198:19 feeling? I want to make sure we're not	CP1_SS_01.56

Page/Line	Source	ID
199:15 - 199:16	198:20 wearing you out. <b>Portier, Christopher 02-21-2019 (00:00:01)</b>	CP1_SS_01.57
199:23 - 212:1	199:15 A. All right. We're fine to 199:16 continue. <b>Portier, Christopher 02-21-2019 (00:12:51)</b> 199:23 Just before the break we were 199:24 talking about genotoxicity, and we were 199:25 looking at this Exhibit 886. I want to talk 200:1 specifically about micronuclei. 200:2 Okay? 200:3 A. Okay. 200:4 Q. Has there been a meta-analysis 200:5 specifically done on micronuclei studies with 200:6 glyphosate and formulated Roundup? 200:7 A. Yes, there has. 200:8 Q. Okay. And is that a study that 200:9 you reviewed in rendering your opinions in 200:10 this case? 200:11 A. Yes, it is. 200:12 Q. Okay. Why don't you turn to 200:13 Exhibit 560 in your binder. 200:14 A. Okay. 200:15 Q. Is this that meta-analysis that 200:16 you were referring to? 200:17 A. Yes, it is. 200:18 Q. Okay. Great. 200:19 So we have it up here on the 200:20 screen. 200:21 This document, it's titled 200:22 "Does exposure to glyphosate lead to an 200:23 increase in the micronuclei frequency? A 200:24 systematic and meta-analytic review." 200:25 What is this study about, sir? 201:1 A. This study takes all of the 201:2 peer-reviewed micronucleus assays and the 201:3 industry micronucleus assays that are 201:4 available and puts them into one global 201:5 analysis to see to what degree there is 201:6 positive findings for micronucleus. 201:7 Q. And the jury may have heard	CP1_SS_01.58

201:8 about this from Dr. Ritz, but what is your  
201:9 understanding of a meta-analysis?  
201:10 A. In a meta-analysis you're  
201:11 talking results from multiple studies using  
201:12 the -- the observed response and the noise  
201:13 around the observed response to bring them  
201:14 all together appropriately to look for a  
201:15 global observed response.  
201:16 Q. So if we dig into the study, if  
201:17 you go to the fifth page in the study,  
201:18 there's a chart. It's labeled "Table 1."  
201:19 It's also on the screen, so if  
201:20 you want to just follow along.  
201:21 A. Yes.  
201:22 Q. Okay. And this lists a bunch  
201:23 of different studies.  
201:24 Do you see that?  
201:25 A. Yes, I do see it.  
202:1 Q. What are these studies  
202:2 referring to?  
202:3 A. They are each individual dose  
202:4 groups in individual studies of micronucleus  
202:5 in exposure to -- after exposure to either  
202:6 glyphosate or glyphosate formulations.  
202:7 Q. And if we look on here, for  
202:8 example, here's a study that I think you  
202:9 might recognize, Bolognesi, 1997.  
202:10 Do you see that?  
202:11 A. Yes, I see it.  
202:12 Q. Okay. Great.  
202:13 And so if we go down here on  
202:14 the -- starting on the seventh page, there is  
202:15 this plot, and I've blown it up here for the  
202:16 jury.  
202:17 What kind of chart -- what  
202:18 would you call this chart?  
202:19 A. This would be in the parlance  
202:20 of statistics a forest plot.  
202:21 Q. And if you actually look at the  
202:22 bottom, is that what they call it?

202:23 A. Yes.  
202:24 Q. Okay. And walk the jury  
202:25 through how you read a chart like this. What  
203:1 are we seeing here?  
203:2 A. Okay. So let's look at the X  
203:3 axis first, which is the one across the  
203:4 bottom. That is the in log scale. Log is  
203:5 just a way of switching numbers around to  
203:6 sort of bring wide numbers into smaller  
203:7 numbers for the audience. It's a simple  
203:8 mathematical tool.  
203:9 The line that's going straight  
203:10 up in the middle of that is at zero. That is  
203:11 the point in this type of a plot where there  
203:12 is no effect. So any studies that lined up  
203:13 with that zero are showing no effect.  
203:14 Studies to the left of that  
203:15 zero are showing a reduction in micronucleus  
203:16 from exposure to either glyphosate or  
203:17 glyphosate formulations.  
203:18 Studies to the right, that have  
203:19 their -- that bulk to the right of zero in  
203:20 that plot are showing an increase in  
203:21 micronuclei from exposure to glyphosate or  
203:22 glyphosate formulation, depending on the  
203:23 study.  
203:24 Q. And the jury will have heard a  
203:25 little bit about epidemiology and maybe even  
204:1 seen some of these sorts of charts with  
204:2 epidemiology.  
204:3 In an epidemiology forest plot,  
204:4 is the no effect at zero or 1?  
204:5 A. It's always at 1. But when you  
204:6 take the log of 1, the log of 1 is zero,  
204:7 which is why this one's at zero, because  
204:8 they've got log on the horizontal axis.  
204:9 Q. Okay. And so if we look in  
204:10 here, it actually has these numbers next to  
204:11 each line.  
204:12 Do you see that?

204:13 A. Yes, I do see that.  
204:14 Q. What does that number refer to,  
204:15 for example, 93?  
204:16 A. That number corresponds to  
204:17 Table 1, where we just looked, and it  
204:18 corresponds to the 93rd study listed in  
204:19 Table 1.  
204:20 Q. Okay. And then if you see  
204:21 buried in here, it's kind of hard to see,  
204:22 there's something called the grand mean.  
204:23 Do you see that?  
204:24 A. Yes.  
204:25 Q. What is that?  
205:1 A. So this -- forest plots are  
205:2 used to do meta-analysis, and when you do a  
205:3 meta-analysis, as I mentioned earlier, you're  
205:4 bringing all that information to get one  
205:5 answer.  
205:6 This is the overall  
205:7 meta-analysis for all of these studies. It  
205:8 is what do all of these data tell me,  
205:9 regardless of whether they're in fish or  
205:10 frogs or humans or dogs or cats or mice or  
205:11 rats. What does all of this tell us as one  
205:12 bulk of data. That's what the grand mean is.  
205:13 Q. And if we look here on the  
205:14 chart, the grand mean is right there; is that  
205:15 right?  
205:16 A. That's correct.  
205:17 Q. And what significance, if any,  
205:18 is there to the fact that the grand mean is  
205:19 that far to the right of the line?  
205:20 A. It means that it's -- it's  
205:21 in -- on average, the -- the risk posed by  
205:22 glyphosate or glyphosate formulations in this  
205:23 entire class of body of evidence is positive.  
205:24 And the fact that the little  
205:25 lines that are stemming from the side, it  
206:1 looks like just a little plus mark for the  
206:2 grand mean, but that's actually the

206:3 95 percent confidence around the point.  
206:4 The fact that the bottom of  
206:5 that line does not cross over zero means that  
206:6 it's statistically significantly different  
206:7 from no -- no effect.  
206:8 Q. And that's kind of what we were  
206:9 talking about earlier with P values; is that  
206:10 right?  
206:11 A. Correct.  
206:12 Q. Okay. And now if we turn to  
206:13 the next page, there's some other -- there's  
206:14 some additional charts here.  
206:15 I want to sort of raise -- kind  
206:16 of ask you to explain what they refer to.  
206:17 Let's look at chart A, right?  
206:18 So here we have chart A, and  
206:19 you can see the grand mean is on here.  
206:20 Do you see that?  
206:21 A. Yes, I do.  
206:22 Q. All right. And what do these  
206:23 other things refer to?  
206:24 A. So chart A is the same type of  
206:25 chart. So zero, which is all the way to the  
207:1 left, is the no effect level. And you're  
207:2 looking at different classes of animals. So  
207:3 you've got fish, you've got amphibians,  
207:4 you've got crocodiles, which are reptiles,  
207:5 and then you've got mice. And they're  
207:6 showing the meta-analysis results just for  
207:7 those subclasses, again, for glyphosate and  
207:8 glyphosate formulations.  
207:9 Most of the fish studies are  
207:10 glyphosate formulations, although there are  
207:11 some laboratory. The amphibians and the  
207:12 crocodiles, they're all glyphosate  
207:13 formulations. The mice are a mixture.  
207:14 Q. And we spent quite a bit of  
207:15 time earlier today talking about the  
207:16 importance of mice studies.  
207:17 Is that significant to you,

207:18 that the mice study is all the way to the  
207:19 right?  
207:20 A. Well, I mean, it's significant  
207:21 that they're mammals and they are mice. Some  
207:22 of these studies, not all of them but some of  
207:23 them, are regulatory studies because the  
207:24 micronucleus assay in mice is a good general  
207:25 assay for DNA damage, regardless of the type  
208:1 of damage. So you're not looking for  
208:2 single-strand breaks or double-strand breaks;  
208:3 you're looking at general area of DNA damage.  
208:4 And so regulatory agencies  
208:5 require it, they ask people to do it. So  
208:6 there are a number of studies in here that  
208:7 were submitted by the regulators. So that's  
208:8 what makes it important, is that it's one of  
208:9 the key studies that regulatory agencies use  
208:10 to decide on the safety of a compound.  
208:11 Q. All right. And then, for  
208:12 example, on the next one, chart B, there is a  
208:13 distinction between -- what is the  
208:14 distinction between?  
208:15 A. Here, it's between mammals and  
208:16 non-mammals, so your fish and your crocodiles  
208:17 and your hairy armadillos are all to the left  
208:18 in the nonmammalian group. The mammalian  
208:19 group is up there.  
208:20 And what you're seeing again is  
208:21 zero, no effect, is way to the left, showing  
208:22 that these are all increased in their risk  
208:23 when you bring them together in the  
208:24 meta-analysis.  
208:25 Q. And the fact that we have here  
209:1 a much larger distance to the right from  
209:2 mammals than non-mammals, does that have any  
209:3 significance to you in assessing, you know,  
209:4 the genotoxicity of Roundup in humans?  
209:5 A. It just says the mammals are --  
209:6 the information is stronger that there's a  
209:7 DNA damage in the mammals.

209:8 Q. Okay. And then if we see down  
209:9 here -- and we don't have to spend too much  
209:10 time on this, but I do want to just show you  
209:11 we have, for example, another chart in here.  
209:12 They've broken -- how have they broken it  
209:13 down in this one?  
209:14 A. Okay. So these are different  
209:15 types of ways to expose -- to be exposed to  
209:16 glyphosate or glyphosate formulations.  
209:17 Oral is either by feed or --  
209:18 it's by feed. You eat it.  
209:19 Immersion is for fish; you're  
209:20 swimming in it.  
209:21 Spraying is for people and some  
209:22 of the ecological studies that were done in  
209:23 animals that are in the fields that are  
209:24 sprayed.  
209:25 Topical is on the skin.  
210:1 Intraperitoneal is injecting it  
210:2 into the peritoneum, which is the lower part  
210:3 of the cavity of these animals. The gut  
210:4 area, gut, stomach, liver.  
210:5 Q. And it looks like the chart B  
210:6 here is breaking it down by males and  
210:7 females.  
210:8 Do you see that?  
210:9 A. Correct.  
210:10 Q. And we have -- we have, for  
210:11 example, females that the line actually  
210:12 crosses the line.  
210:13 Do you see that?  
210:14 A. Correct. They have an  
210:15 increased risk in the meta-analysis, but it's  
210:16 not statistically significant, whereas the  
210:17 males are statistically significant.  
210:18 Q. Yeah. And if you look at the  
210:19 male one, it's way over here on the right.  
210:20 Do you see that?  
210:21 A. Yeah, that may reflect more the  
210:22 fact that there are a lot of male studies and

210:23 not a lot of female studies.  
 210:24 Q. And then what does this -- this  
 210:25 part in the middle, this both, what does that  
 211:1 refer to?  
 211:2 A. That's just the combination of  
 211:3 the male and female data at the same time.  
 211:4 Q. Okay. And was that  
 211:5 statistically significant?  
 211:6 A. Ignoring gender.  
 211:7 Q. Okay. Was that statistically  
 211:8 significant?  
 211:9 A. That one is statistically  
 211:10 significant.  
 211:11 Q. All right. And this process of  
 211:12 looking at all these studies in different  
 211:13 ways, is that commonly done in meta-analysis?  
 211:14 A. It should be done here.  
 211:15 There's definitely -- most meta-analyses are  
 211:16 done with epidemiology data, and they will  
 211:17 break it down into important characteristics.  
 211:18 You have different -- excuse  
 211:19 me, different types of studies or studies  
 211:20 from different continents or different  
 211:21 countries, and so you would break it down and  
 211:22 look at the individual continents or the  
 211:23 individual countries.  
 211:24 It's a sensitivity analysis.  
 211:25 You're looking at how sensitive the findings  
 212:1 are to subclassing the information.

212:4 - 212:5

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CP1\_SS\_01.69

212:4 Chart A, what does this  
 212:5 reflect?

212:8 - 215:18

**Portier, Christopher 02-21-2019 (00:03:12)**

CP1\_SS\_01.60

212:8 THE WITNESS: This is the  
 212:9 forest plot looking at glyphosate  
 212:10 technical versus -- they call it  
 212:11 Roundup, but it's actually glyphosate  
 212:12 formulations. It could be any  
 212:13 formulation. From my reading of this  
 212:14 document, it's not just Roundup -- and

212:15 comparing the grand means from the two  
212:16 subclasses.

212:17 QUESTIONS BY MR. WISNER:

212:18 Q. And what, if any, significance

212:19 is there to the fact that Roundup is

212:20 significantly farther to the right than just

212:21 glyphosate?

212:22 A. It would suggest that the

212:23 evidence for Roundup is stronger that there

212:24 is an increase in micronucleus in these data

212:25 for glyphosate formulations.

213:1 Q. Okay. Earlier you were talking

213:2 about regulatory studies and nonregulatory

213:3 studies.

213:4 Do you recall that?

213:5 A. Yes.

213:6 Q. What does this chart reflect?

213:7 A. For the most part it reflects

213:8 the regulatory studies versus the literature

213:9 studies. So peer-reviewed means those are

213:10 studies that have appeared in the

213:11 peer-reviewed literature. The

213:12 nonpeer-reviewed are those studies that they

213:13 were able to get that were regulatory

213:14 submission studies. And again, they're both

213:15 significantly different than no effect.

213:16 Q. And is there any significance

213:17 to the fact that the peer-reviewed data is

213:18 significantly farther to the right than the

213:19 nonpeer-reviewed data?

213:20 A. Again, it's the same thing.

213:21 The peer-reviewed data has stronger

213:22 indication that glyphosate can cause

213:23 micronucleus in these data.

213:24 Q. Let's take a quick step back,

213:25 sir.

214:1 I mean, have you ever been an

214:2 editor on a journal?

214:3 A. Yes, I have.

214:4 Q. Are you familiar with what peer

214:5 review is?

214:6 A. Yes, of course.

214:7 Q. What is peer review?

214:8 A. Peer review is when you -- when

214:9 you wish to have a paper put out in the

214:10 scientific literature for others to consider,

214:11 journals like to make sure that the paper

214:12 is -- appears to be scientifically sound and

214:13 based on sound strategies, sound arguments,

214:14 and it's complete. It's provided everything

214:15 you need to understand what's done.

214:16 So they will take that paper

214:17 and send it to several people who are

214:18 knowledgeable about that area of research,

214:19 who will read it and comment on the quality

214:20 and the -- the arguments used by the

214:21 scientists involved and whether they made

214:22 their case or didn't make their case, what

214:23 are the limitations.

214:24 Sometimes they will reject the

214:25 paper outright and say "this is just garbage,

215:1 you can't understand it, we don't know what

215:2 it means." Sometimes they love it and they

215:3 go, "we'll take it, it's perfect, you should

215:4 publish it like that."

215:5 Most times there's going to

215:6 be -- you -- we'd like to see this figure.

215:7 We don't think that one's very informative;

215:8 you should just remove it. Did you do this

215:9 analysis? If you did, could you show it,

215:10 because we'd like to see what the results of

215:11 that was. So there's some suggestions for

215:12 changes.

215:13 If the changes are made, then

215:14 it's usually published.

215:15 Q. And all things being equal,

215:16 sir, do you prefer -- all things being equal,

215:17 are peer-reviewed articles more reliable than

215:18 nonpeer-reviewed articles?

215:21 THE WITNESS: As a general  
215:22 statement, that would be correct.  
215:23 In the case of regulatory  
215:24 studies as compared to peer-reviewed  
215:25 studies, I would argue that they're  
216:1 probably of equal quality.  
216:2 There are requirements that go  
216:3 into developing things under peer  
216:4 review -- under regulatory guidelines  
216:5 that require astringency, that anybody  
216:6 peer reviewing it who read the notes  
216:7 that said "we did this under these  
216:8 guidelines" would probably accept it  
216:9 as a clean, reasonable study.  
216:10 They may not agree to the  
216:11 conclusions, they may not agree to the  
216:12 method of analysis or the analyses in  
216:13 a peer review, but at least they can  
216:14 agree to the quality of the study.  
216:15 So in a general rule, peer  
216:16 review is better than nonpeer review,  
216:17 but in a regulatory context, I would  
216:18 have to look carefully at the  
216:19 nonpeer-reviewed before I'd say, well,  
216:20 no, it's worse. I don't think as a  
216:21 general rule I would -- I would  
216:22 approach it as saying it's worse  
216:23 simply because it's not peer-reviewed.  
216:24 QUESTIONS BY MR. WISNER:  
216:25 Q. This meta-analysis by Dr. Ghisi  
217:1 and her colleagues, is this something that  
217:2 you relied on?  
217:3 A. I did. It's got its  
217:4 limitations, but certainly I -- it was part  
217:5 of the evidence I looked at in coming to my  
217:6 decision.  
217:7 Q. And what decision did you come  
217:8 to with respect to whether or not Roundup or  
217:9 glyphosate can cause micronuclei in cells?  
217:10 A. In mammalian systems, which is

217:11 the important one for me, I believe  
217:12 glyphosate can cause micronucleus in  
217:13 mammalian systems.  
217:14 Q. And the creation of  
217:15 micronuclei, is that a recognized mechanism  
217:16 through which something can cause cancer?  
217:17 A. Yes.  
217:18 Q. All right. So we've been  
217:19 talking about genotoxicity for a little bit  
217:20 now. I want to move on to the second one.  
217:21 What was the second one, sir?  
217:22 A. The second mechanism that was  
217:23 considered that -- where they had enough  
217:24 evidence was oxidative stress.  
217:25 Q. And you discussed what it was  
218:1 earlier, but let's just refresh everyone's  
218:2 recollection.  
218:3 What exactly is oxidative  
218:4 stress in a human cell?  
218:5 A. I'm going to try to make it as  
218:6 noncomplicated as I possibly can.  
218:7 Oxidative stress. So oxygen is  
218:8 the energy source of cells. I mean, it  
218:9 drives a lot of what we do in the cells to  
218:10 keep ourselves alive and moving and  
218:11 functioning and everything else. It's the  
218:12 energy source.  
218:13 Oxygen radicals are oxygen  
218:14 molecules that are not bound to anything.  
218:15 You know, water is H<sub>2</sub>O, so you've got two  
218:16 molecules of hydrogen bound to an oxygen, and  
218:17 that's a very stable chemical.  
218:18 But when you pull those  
218:19 hydrogens off, that oxygen becomes very  
218:20 reactive and it wants to bind to anything  
218:21 else. So if there's any oxygen around,  
218:22 hydrogen around, it's going to bind to the  
218:23 hydrogen, reform water.  
218:24 Okay. So in cells, that oxygen  
218:25 that's not bound to anything gets bound, then

219:1 it gets unbound, then it gets bound again,  
219:2 and that's doing the work of the cell. It's  
219:3 binding and unbinding energy sources. Oxygen  
219:4 is one of them.

219:5 There are things that receive  
219:6 that oxygen in the cell, and so you've got a  
219:7 balance. You don't want too many things  
219:8 there that are not bound to oxygen, because  
219:9 they can cause a problem, and you don't want  
219:10 too much oxygen that's not binding, because  
219:11 that can cause a problem. So you've got to  
219:12 balance.

219:13 Oxidative stress is when you go  
219:14 out of balance. Either you remove the things  
219:15 that the oxygen is binding to, reduce them,  
219:16 which causes more free oxygen around, or you  
219:17 make more free oxygen than can bind to what's  
219:18 there, and then more free oxygen is around.

219:19 That free oxygen can bind to  
219:20 micronuclei -- to mitochondria, it can bind  
219:21 to DNA, it can bind to other structures in  
219:22 the cell that can begin to damage the cell,  
219:23 and that damage to the cell can lead to  
219:24 mutations or other problems that can lead to  
219:25 cancer.

220:1 Q. But, sir, I mean, you're  
220:2 talking about oxygen in a cell.  
220:3 I mean, isn't there oxygen in  
220:4 our cells every day?

220:5 A. Absolutely.

220:6 Q. So why aren't I getting cancer?

220:7 A. Because too much of a good  
220:8 thing is too much of a good thing. You want  
220:9 to keep the balance. You want to make sure  
220:10 that you're not going overboard on the amount  
220:11 of free oxygen in the cell.

220:12 Q. So when we talk about oxidative  
220:13 stress in the context of glyphosate, are we  
220:14 talking about something that causes an  
220:15 imbalance?

220:16 A. That would be the root source  
220:17 of the oxidative stress, some sort of  
220:18 imbalance.  
220:19 Q. All right. So just like with  
220:20 genotoxicity, there's our in vivo studies and  
220:21 in vitro studies; is that right?  
220:22 A. Correct.  
220:23 Q. Have there been any in vivo  
220:24 human studies, like living people, that  
220:25 looked at oxidative stress with Roundup or  
221:1 glyphosate?  
221:2 A. No.  
221:3 Q. Okay. So that -- you know,  
221:4 that -- so we had that tier for genotoxicity.  
221:5 The number one, the humans in  
221:6 vivo, we don't have that for oxidative  
221:7 stress; is that right?  
221:8 A. That's correct.  
221:9 Q. Okay. What about number two,  
221:10 humans in -- human cells in vitro, do we have  
221:11 any data about that?  
221:12 A. Yes.  
221:13 Q. Did you actually help us  
221:14 prepare a chart similar to the genotoxicity  
221:15 for oxidative stress?  
221:16 A. Yes.  
221:17 Q. Okay. All right. So this is  
221:18 Exhibit 877, and it's titled "Human In Vitro  
221:19 Oxidative Stress."  
221:20 What does this chart reflect,  
221:21 sir?  
221:22 A. Similar to the previous chart,  
221:23 the first column gives studies. Each  
221:24 individual study is a peer-reviewed study of  
221:25 oxidative stress in cells, in human cells.  
222:1 The next column, labeled  
222:2 "glyphosate," is studies that is going to be  
222:3 a positive, negative or no data for technical  
222:4 glyphosate, pure glyphosate.  
222:5 And the last column,

222:6 "formulation," is for some glyphosate

222:7 formulation.

222:8 Q. Okay. And I noticed some of

222:9 these names are familiar from the previous

222:10 chart. So, for example, Wozniak.

222:11 Do you see that?

222:12 A. Yes.

222:13 Q. How are they on this chart and

222:14 on the previous chart?

222:15 A. It's the same study. Many

222:16 times when you do a study on oxidative

222:17 stress, you're also going to do a study on

222:18 DNA damage because the two are related.

222:19 Because the oxygen radicals can bind to DNA,

222:20 they can damage DNA, strand breaks that you

222:21 can then see.

222:22 And so the two are related to

222:23 each other, and it's not uncommon to see both

222:24 in the same paper.

222:25 Q. Now, I want to be clear. We're

223:1 here talking about human data, right?

223:2 A. Correct.

223:3 Q. Have there been studies done on

223:4 bacteria or mammals or reptiles?

223:5 A. Oh, yes. There's studies in

223:6 the animals. There's studies in crocodiles.

223:7 There's studies in all kinds of different

223:8 animals and then in various and sundry other

223:9 cell lines.

223:10 Q. So why then are we focusing on

223:11 human cell here?

223:12 A. Again, it's because -- well, if

223:13 we're setting my priorities, again, my

223:14 priorities are always -- for oxidative stress

223:15 it's -- this is real tough because the human

223:16 cells, again, those are cells from humans, so

223:17 they're close to the target I'm interested

223:18 in, but they're not in functioning organisms.

223:19 And the rodent models, the functioning

223:20 organisms, might be better here for oxidative

223:21 stress because they're in functioning  
223:22 organisms.  
223:23 And oxidative stress -- DNA  
223:24 damage is a single target. Oxidative stress  
223:25 is a target of an entire system. And so it  
224:1 might be that that's better, but they're,  
224:2 again, somewhat equal. So we're looking at  
224:3 human here because it's human cells.  
224:4 Q. All right. So let's go through  
224:5 this again. We have our positive and  
224:6 negatives in here.  
224:7 Before I get started, are any  
224:8 of these no datas incorrect?  
224:9 A. No.  
224:10 Q. Okay.  
224:11 A. This one is correct.  
224:12 Q. All right. So let's go for the  
224:13 first one starting in 2005.  
224:14 Did this look at both  
224:15 glyphosate and formulation?  
224:16 A. Yes, they did, and they were  
224:17 both positive in a very unique way -- unique  
224:18 type assays. But, yes, they were both  
224:19 positive.  
224:20 Q. Can you explain why it was  
224:21 unique?  
224:22 A. Yes.  
224:23 Instead of looking directly for  
224:24 oxidative stress, what they did was looked at  
224:25 reduction in cell death using antioxidants.  
225:1 And by showing that the antioxidants reduced  
225:2 toxicity in the cell, they're showing that  
225:3 there's too much free oxygen in the cell.  
225:4 And so their argument was that  
225:5 they're seeing oxidative stress because they  
225:6 can relieve it with the antioxidant.  
225:7 Q. Antioxidants, I mean, I hear  
225:8 about that all the time. What are those?  
225:9 A. They're --

225:12 THE WITNESS: They're chemicals  
225:13 or things that enter into the cell  
225:14 that bind out the free oxygen, let's  
225:15 put it that way, in a safe way.  
225:16 QUESTIONS BY MR. WISNER:  
225:17 Q. And so do they help reduce  
225:18 oxidative stress?  
225:19 A. Yes, they do.  
225:20 Q. Okay. All right. The next one  
225:21 from 2009, that was on glyphosate?  
225:22 A. Yes.  
225:23 Do you still want to know if  
225:24 it's in lymphocytes or not?  
225:25 Q. Oh, yes, please.  
226:1 A. So that one is in lymphocytes.  
226:2 Q. Okay.  
226:3 A. And that was positive. Not --  
226:4 no, not -- the Mladinic is in lymphocytes.  
226:5 The first one is not. And that one is  
226:6 positive.  
226:7 Q. Okay. Great.  
226:8 What about the 2010 one?  
226:9 A. Okay, they called it positive,  
226:10 but I don't like the assay they used. Plus  
226:11 their doses were extremely high, to the point  
226:12 of potentially suffocating the cells. I call  
226:13 this one inadequate.  
226:14 Q. Okay. So just like we did last  
226:15 time, I'll put a question mark.  
226:16 Does that work?  
226:17 A. That's fine.  
226:18 Q. And then I'll put --  
226:19 A. This one's clearly inadequate.  
226:20 I'm not even going to be wishy-washy on it.  
226:21 Q. All right.  
226:22 A. This one's clearly inadequate.  
226:23 I would never include this in my decisions.  
226:24 Q. Okay. So how do you want me to  
226:25 mark it so it's clear reflecting --  
227:1 A. Question mark is fine.

227:2 Q. Okay. I won't even put the  
227:3 plus, though.  
227:4 A. Yeah, I wouldn't put the plus.  
227:5 Q. Okay. Sounds good.  
227:6 All right. George and Shukla,  
227:7 2013?  
227:8 A. This one -- they were positive.  
227:9 They saw it as positive. I agree that --  
227:10 with what they did, they saw it as positive,  
227:11 but I'm a little iffy on this one, too.  
227:12 They used the same assay as the  
227:13 one by Elie-Caille. But what they -- they  
227:14 used much lower exposure, so the cytotoxicity  
227:15 is not such a big deal.  
227:16 So I'm in between this one  
227:17 saying, yeah, it's positive or it's  
227:18 inadequate. So I'd put a question mark next  
227:19 to that, too.  
227:20 Q. Does that work?  
227:21 A. Yep, that would work.  
227:22 Q. Okay. And before we move on,  
227:23 you said a word, cytotoxicity.  
227:24 What does that mean?  
227:25 A. Oh, the -- they were putting --  
228:1 in the Elie-Caille study, they were putting  
228:2 so much glyphosate into the petri dish with  
228:3 the cells that it was affecting the ability  
228:4 of the cells to survive.  
228:5 You know, cells need a  
228:6 nutritious buffer in which to live. They  
228:7 don't live in water. You've got to put in  
228:8 nutrients and all kinds of stuff. And when  
228:9 you add a chemical to it, it can block the  
228:10 access to those nutrients and cells start to  
228:11 die.  
228:12 They had so much chemical in  
228:13 there, I just can't imagine that the effects  
228:14 we're looking at are due to glyphosate.  
228:15 They're due to the fact that you've got a  
228:16 huge amount of chemical in there.

228:17 Q. Okay. And so what you mean by

228:18 cytotoxicity --

228:19 A. Is cell death.

228:20 Q. -- if you put in any chemical,

228:21 you'd have the same problem?

228:24 - 239:18

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228:24 THE WITNESS: Correct, but

228:25 it's -- in -- cytotoxicity technically

229:1 means cell death. And so when you see

229:2 increased cytotoxicity, that's okay

229:3 with an oxidative stress study because

229:4 oxidative stress can result from

229:5 cytotoxicity, and that's important.

229:6 And cytotoxicity can result from

229:7 oxidative stress. That's important.

229:8 But when you put in so much

229:9 chemical that you're killing it by

229:10 something other than slight changes in

229:11 oxidative stress, the cytotoxicity is

229:12 too high.

229:13 QUESTIONS BY MR. WISNER:

229:14 Q. Gotcha.

229:15 All right. This one from 2014?

229:16 A. It's negative for glyphosate

229:17 and positive for the formulation.

229:18 Q. What significance, if any, do

229:19 you see from that?

229:20 A. This is an interesting study

229:21 for that question, because the negative for

229:22 the glyphosate itself was at a fairly high

229:23 dose, whereas the positive for the

229:24 formulation was at a much lower equivalent

229:25 exposure. So this particular study would

230:1 suggest that the formulation in these cells

230:2 in this case is much more effective at

230:3 causing DNA damage than is the glyphosate

230:4 pure itself.

230:5 Q. All right. Let's go for the

230:6 next one. 2014?

230:7 A. Coalova. That one was

230:8 positive.

230:9 Q. All right. What about the next

230:10 one from 2014?

230:11 A. That was in red blood cells, in

230:12 humans.

230:13 Q. Okay.

230:14 A. And that one is positive.

230:15 Q. What about Luo from 2017?

230:16 A. Yeah, that one was positive.

230:17 That one was clearly positive.

230:18 Q. All right. Kasuba 2017?

230:19 A. That one was positive.

230:20 Q. And then the last one from

230:21 2018?

230:22 A. That's human leukocytes, and

230:23 both of those are positive.

230:24 Q. And by leukocytes, does that

230:25 mean blood?

231:1 A. A type of -- one of the blood

231:2 cells, yes.

231:3 Q. That's what we were doing

231:4 before. We called it blood, so I'll keep

231:5 doing that here.

231:6 They were both positive?

231:7 A. Yes.

231:8 Q. All right. Well, sir, I mean,

231:9 again, we're looking at this chart now for

231:10 oxidative stress in humans.

231:11 What does this data indicate to

231:12 you?

231:13 A. That both glyphosate and the

231:14 formulation can induce oxidative stress in

231:15 human cells.

231:16 Q. And we can't do a similar sort

231:17 of resolution for genotoxicity.

231:18 Is your opinion regarding

231:19 oxidative stress as strong?

231:20 A. Yes. When I look at not just

231:21 this but the in vivo data from animals and

231:22 other things, there's no doubt that the

231:23 oxidative stress data is strong and it's  
231:24 quite clear.

231:25 Q. All right. We'll go back to  
232:1 the stool that we were sort of using as a  
232:2 roadmap here.

232:3 And so far we've talked about  
232:4 animal studies and we've talked about  
232:5 mechanistic studies; is that right?

232:6 A. Correct.

232:7 Q. And, you know, I want to get a  
232:8 sense of your opinion about the strength of  
232:9 this evidence so far.

232:10 For the animals studies, do you  
232:11 think it's strong, or how would you  
232:12 characterize it?

232:13 A. I would characterize it as  
232:14 saying glyphosate can cause cancer in  
232:15 mammals.

232:16 Q. And then for the mechanism  
232:17 studies, what's the conclusion there?

232:18 A. Glyphosate can induce DNA  
232:19 damage in mammalian cells and in human cells,  
232:20 and it can induce oxidative stress in  
232:21 mammalian systems and in human cells.

232:22 Q. And when you reach that opinion  
232:23 about these two sort of groups of studies, is  
232:24 that opinion reached to a reasonable degree  
232:25 of scientific certainty?

233:1 A. Oh, yes. It's very little  
233:2 uncertainty.

233:3 Q. Okay. All right. I want to go  
233:4 to this last prong, epidemiology, and I'll  
233:5 let you know, Doctor, that Dr. Ritz has --  
233:6 will have already testified by the time the  
233:7 jury hears your testimony, so I don't want to  
233:8 spend too much time covering the basics.

233:9 Okay?

233:10 A. Okay.

233:11 Q. Have you reviewed the  
233:12 epidemiology in this case?

233:13 A. Oh, yes.

233:14 Q. Okay.

233:15 A. Oh, yeah.

233:16 Q. And what did that review

233:17 consist of?

233:18 A. Reading all the epidemiological

233:19 studies that relate to glyphosate in any

233:20 disease, but mostly focused on non-Hodgkin's

233:21 lymphoma. Reading the ancillary studies,

233:22 because when you do an epi study you don't

233:23 just publish one paper, you -- you publish

233:24 papers on how you measure dose and all kinds

233:25 of other things, and so you have to read

234:1 those as well. And so reading them as well.

234:2 Q. Okay. In the process through

234:3 which you reviewed the epidemiology, the

234:4 animal studies, the mechanism studies, is

234:5 that the process that you used when you

234:6 worked at the National Toxicology Program or

234:7 the National Institute of Health?

234:8 A. Yes, the National Toxicology

234:9 Program has the report on carcinogens, which

234:10 is the US government's official report on

234:11 what chemicals -- well, US Department of

234:12 Health and Human Services official list of

234:13 what chemicals cause cancer in humans, and we

234:14 used -- they used the same approach.

234:15 Q. And did you help, like, figure

234:16 out what substances should go on that list

234:17 when you worked there?

234:18 A. I was responsible for making

234:19 the final recommendation to the director, who

234:20 signed off on what should go on that list.

234:21 He usually just signed the list.

234:22 Q. So I don't want to spend too

234:23 much time going through the epidemiology, but

234:24 I want to talk about a few things.

234:25 I understand that you've placed

235:1 all of the studies onto a chart; is that

235:2 right?

235:3 A. That is correct.  
235:4 Q. Okay.  
235:5 A. All of the -- not -- well, it  
235:6 depends which chart you're going to bring up.  
235:7 There are several charts that I've made, some  
235:8 of which have all of the studies -- well, no  
235:9 one chart has all of the results from all of  
235:10 the studies because there are just too many  
235:11 results.  
235:12 So there are different charts.  
235:13 It depends which chart you want to bring up.  
235:14 Q. All right. Well, let's focus  
235:15 on meta-analysis.  
235:16 Okay?  
235:17 A. Okay.  
235:18 Q. Please turn to Exhibit 787 in  
235:19 your binder.  
235:20 A. 787?  
235:21 Q. That is incorrect. I'm sorry.  
235:22 It would be Exhibit 878.  
235:23 A. 878?  
235:24 Q. That's right.  
235:25 A. Okay.  
236:1 Q. Is that a copy of the chart you  
236:2 prepared with the meta-analysis?  
236:3 A. Oh, yes, it is.  
236:4 Q. Okay. So I'm going to put this  
236:5 up on the screen.  
236:6 Before we get started, where  
236:7 did you derive this chart from?  
236:8 A. Oh, a recent meta-analysis that  
236:9 was done on all of the epidemiology data by  
236:10 Zhang and coworkers published a couple of  
236:11 weeks ago. This is from -- directly from  
236:12 Table 7. This is a different way of looking  
236:13 at their Table 7.  
236:14 Q. Okay. Great. So let's break  
236:15 this down a little bit.  
236:16 So we have on the right here,  
236:17 we have this blue line.

236:18 Do you see that?  
236:19 A. Yes.  
236:20 Q. What does that blue line  
236:21 indicate?  
236:22 A. So like the forest plot we saw  
236:23 just a minute ago for the micronucleus  
236:24 assays, this is 1. This is where there's no  
236:25 effect in the data.  
237:1 Q. So if something is to the right  
237:2 of 1, what does that mean?  
237:3 A. If the -- so you have little  
237:4 black -- little squares and lines extending  
237:5 from the little squares.  
237:6 Q. I'll cull one out. Okay.  
237:7 A. Yeah, that's a good example  
237:8 right there, that black square in the middle,  
237:9 and then you've got lines extending from two  
237:10 sides.  
237:11 The black square is the mean of  
237:12 the relative risk, the risk ratio. So if  
237:13 that mean is directly on the blue line, then  
237:14 its value is 1, and that says there's no  
237:15 effect. If it's to the left, its value is  
237:16 below 1, that says there is an effect. It's  
237:17 a reduction of risk. If it's to the right,  
237:18 it says there is an effect, there's an  
237:19 increase in risk.  
237:20 The little spindly lines coming  
237:21 out of it are a 95 percent confidence bound.  
237:22 If the bottom end of that line touches the  
237:23 blue line, then it's not statistically  
237:24 significant but it's increased. If it  
237:25 doesn't touch it, it is statistically  
238:1 significant at the 5 percent level.  
238:2 Q. So looking at these two right  
238:3 here, the top one has a point to the right of  
238:4 the blue line but its whiskers don't touch  
238:5 the blue line.  
238:6 What does that mean?  
238:7 A. Are you talking about the black

238:8 square?

238:9 Q. Yeah, the one right up here on

238:10 the screen.

238:11 A. That top black square is

238:12 significantly increased risk from exposure to

238:13 glyphosate formulations in that study.

238:14 Q. Okay. Great.

238:15 What does the second black

238:16 square with these whiskers indicate?

238:17 A. It shows an increase in the

238:18 risk from exposure to glyphosate formulations

238:19 in that study, but it's not statistically

238:20 significant.

238:21 Q. Okay. And so when we look at

238:22 all these points and whiskers on this chart,

238:23 what do these all reflect?

238:24 A. Well, they reflect different

238:25 things because they're pulling from different

239:1 pieces of each of these epi studies.

239:2 I'm sure the jury has by now

239:3 seen Dr. Ritz talk about the fact that these

239:4 epi studies have different ways of looking at

239:5 exposure, so they might look at were they

239:6 exposed or not exposed. They might look at

239:7 were they exposed for ten years or not

239:8 exposed -- or exposed for less than ten

239:9 years. Were they exposed for two days or

239:10 less than two days. They're a way the -- the

239:11 epi studies will break it down.

239:12 And so one epi study might have

239:13 10 or 12 different evaluations in it. In

239:14 this table, Table 7, Zhang, et al., were

239:15 pulling out the pieces of these studies that

239:16 were used in various meta-analyses. So these

239:17 are parts of the individual epi studies that

239:18 are being displayed here.

240:19 - 245:11

**Portier, Christopher 02-21-2019 (00:04:25)**

CP1\_SS\_01.64

240:19 Q. Okay. So the top part, we have

240:20 different colors. So the first three lines

240:21 are red.

240:22 Do you see that, Doctor?  
240:23 A. Yes.  
240:24 Q. What do those refer to?  
240:25 A. Okay. The first three lines  
241:1 come from two different publications. Let me  
241:2 walk you through the columns real quick.  
241:3 Q. Okay.  
241:4 A. The column that says study is  
241:5 the name of the author and the year in which  
241:6 that particular epidemiology study was done.  
241:7 Q. Okay.  
241:8 A. The column that says RR, that  
241:9 is the relative risk. That's the mean value  
241:10 of the relative risk for that study.  
241:11 Q. I'll stop right there.  
241:12 And when we talk about relative  
241:13 risks or odds ratios, what does anything  
241:14 above 1 mean?  
241:15 A. Above 1 means there's a  
241:16 positive association between the exposure and  
241:17 the disease, in this case non-Hodgkin's  
241:18 lymphoma.  
241:19 Below 1 means there's a  
241:20 negative association, which means that the  
241:21 people who were exposed had less  
241:22 non-Hodgkin's lymphoma than the unexposed.  
241:23 And when it's exactly 1, it  
241:24 means there's no difference.  
241:25 Q. Okay. So then we have lower  
242:1 and upper.  
242:2 What do those refer to?  
242:3 A. So that's the 95 percent  
242:4 confidence bound. The lower is the lower  
242:5 part of that confidence bound. The upper is  
242:6 the upper part of the confidence bound.  
242:7 For simple purposes, the simple  
242:8 way to look at is if the lower bound is below  
242:9 1, that means it's not statistically  
242:10 significantly increased.  
242:11 If the upper bound is above 1,

242:12 that means it's not statistically  
242:13 significantly decreased.  
242:14 Q. Gotcha.  
242:15 A. And so you can draw those  
242:16 inferences from looking at the confidence  
242:17 bounds.  
242:18 Q. And would it be fair to say  
242:19 then that the lower and upper refer to the  
242:20 left and right side of the whiskers?  
242:21 A. Yes, that's exactly what  
242:22 they -- in fact, when you look at the plot,  
242:23 the -- you can see that with the first one,  
242:24 Andreotti, et al., 2018, the lower bound is  
242:25 .83, which is less than 1. And if you  
243:1 could -- if I had put .83 on the X axis, the  
243:2 bottom of it would match exactly with .83 at  
243:3 the bottom.  
243:4 Q. Okay. Great.  
243:5 And then -- so then for the  
243:6 first two colors you have the studies, the  
243:7 risk ratios, the lower and upper confidence  
243:8 bounds, and at the very bottom there's green  
243:9 ones.  
243:10 Do you see that?  
243:11 A. Correct.  
243:12 Q. And then it has letters to the  
243:13 right of it under included.  
243:14 A. So can I answer your other  
243:15 question first as to -- I didn't answer what  
243:16 would the red mean.  
243:17 Q. Okay. Fair enough. Let's take  
243:18 one step at a time.  
243:19 A. I told you what each column  
243:20 meant, but I didn't tell you what the red  
243:21 meant.  
243:22 Q. Okay. What does the red stuff  
243:23 refer to?  
243:24 A. So these are two separate  
243:25 publications in 2018 and 2015 from one study.  
244:1 It's called the Agricultural Health Study.

244:2 It's a cohort study. So they are following  
244:3 people over time who work in the agricultural  
244:4 industry, and every once in a while they look  
244:5 to see how many of them have a disease, in  
244:6 this case non-Hodgkin's lymphoma, but they  
244:7 look at all disease. But for NHL, they look  
244:8 to see how many people have it. And because  
244:9 they've asked these people questions about  
244:10 their exposure, they already know whether  
244:11 they've been exposed or not, and so they can  
244:12 relate the exposure to the study.  
244:13 So the first three lines, first  
244:14 three rows, are all from those cohort  
244:15 studies.  
244:16 The De Roos 2005 has two  
244:17 columns, B and C, the B and C columns. The  
244:18 first one relates to whether they were  
244:19 exposed or not exposed, which is used in some  
244:20 of the meta-analyses. The second relates to  
244:21 a grouping they did in the study of low,  
244:22 medium, high exposure, by grouping people  
244:23 into those exposures.  
244:24 And in one of the meta-analyses  
244:25 they only used the highest exposure group, so  
245:1 this is the result for that highest exposed  
245:2 group, which showed a relative risk below 1.  
245:3 Q. Okay. And then we have De Roos  
245:4 again underneath that.  
245:5 Do you see that?  
245:6 A. Correct.  
245:7 Q. And let's just clarify. This  
245:8 is the same De Roos that joined you in that  
245:9 letter we spoke about at the beginning of  
245:10 your testimony?  
245:11 A. That is correct.

245:15 **Portier, Christopher 02-21-2019 (00:02:13)**  
245:15 Q. And here we have De Roos 2003,  
245:16 and it's in a different color.  
245:17 Why is that?  
245:18 A. So from studies D through M,

245:15 - 247:19

CP1\_SS\_01.66

245:19 they're all in the same color. It's supposed  
245:20 to be dark blue, but it looks like black on  
245:21 my copy.  
245:22 But these are a different type  
245:23 of study. These are case-control studies.  
245:24 So in case-control studies what you've got is  
245:25 people with non-Hodgkin's lymphoma, those are  
246:1 your cases, and you have controls, which are  
246:2 people who don't have non-Hodgkin's lymphoma  
246:3 but they sort of match the cases with the  
246:4 controls.  
246:5 And then you ask them about  
246:6 their past exposures. And what you're really  
246:7 looking for is are the cases more likely to  
246:8 be exposed to glyphosate formulations than  
246:9 the controls.  
246:10 And so the relative risk you're  
246:11 looking at here is the risk of being exposed  
246:12 to glyphosate. And each of these, with a  
246:13 name and a number behind it, is a single  
246:14 finding from that study. And then if there  
246:15 are multiple findings like for Eriksson,  
246:16 which is F, G and H are two other findings  
246:17 that are different, that are used in  
246:18 different meta-analyses, so I extracted them  
246:19 from that paper as well.  
246:20 Q. And so just so we can  
246:21 understand this, if we look at line L, which  
246:22 is from the McDuffie study, do you see that?  
246:23 A. Yes, I see it.  
246:24 Q. And it has a risk ratio of  
246:25 2.12.  
247:1 Do you see that?  
247:2 A. Yes, I do.  
247:3 Q. And the lower bound is 1.2, and  
247:4 the higher bound is 3.37.  
247:5 Do you see that?  
247:6 A. 3.73, yes, I see that.  
247:7 Q. Sorry, I sometimes mix up  
247:8 numbers. I appreciate that.

247:9 What -- what does that  
247:10 indicate?  
247:11 A. Well, that indicates in this  
247:12 study that people in this study who had more  
247:13 than two days per year exposure, the cases  
247:14 were more likely to have that more than two  
247:15 days per year exposure than the controls,  
247:16 they were twice as likely as the controls to  
247:17 have that level of exposure.  
247:18 And it was statistically  
247:19 significantly different from 1.

248:3 - 250:5

**Portier, Christopher 02-21-2019 (00:02:16)**

CP1\_SS\_01.66

248:3 Q. Okay. So then at the very  
248:4 bottom we have the green.  
248:5 Do you see that?  
248:6 A. Yes, I see the greens.  
248:7 Q. All right. And what does the  
248:8 green refer to, and specifically what do  
248:9 these letters to the right of them refer to?  
248:10 A. So there are three published  
248:11 meta-analyses. Remember we just looked at a  
248:12 meta-analyses for micronuclei. This is the  
248:13 same thing, but now you're doing epidemiology  
248:14 studies and bringing them together.  
248:15 Q. I'm sorry, Doctor, you said  
248:16 there's three?  
248:17 A. Four.  
248:18 Q. Oh, okay.  
248:19 A. Sorry. Four published  
248:20 meta-analyses.  
248:21 These are the results from the  
248:22 four published meta-analyses that were  
248:23 mentioned in Table 7 by Zhang. The first  
248:24 three are for were you exposed ever or never.  
248:25 The Zhang paper looked at not  
249:1 ever, never, but they were interested in the  
249:2 highest exposed groups, so they're looking at  
249:3 a slightly different question. But that's  
249:4 what all of these are.  
249:5 The extra numbers -- the

249:6 letters, the B, D, F, I, K, N, for Schinasi  
 249:7 and Leon, that refers to which of the rows  
 249:8 from the studies went into that  
 249:9 meta-analysis. So I'm trying to give you a  
 249:10 feel for which studies went into which  
 249:11 numbers that you're looking at here.  
 249:12 Q. Okay. So if we actually look  
 249:13 at the data here on the points and the  
 249:14 whiskers, do you have an opinion about what  
 249:15 this data shows?  
 249:16 A. Well, as I pointed out in the  
 249:17 expert report, not for this graph but for the  
 249:18 graph that I had in there, which is similar  
 249:19 to this, most of the responses to the right  
 249:20 of the value of 1, that suggests that  
 249:21 generally the trend is toward an association  
 249:22 in these data.  
 249:23 Some of them are significantly  
 249:24 positive, some are not, but the general trend  
 249:25 is definitely toward a positive association.  
 250:1 If you look at ever, never,  
 250:2 which is some of the ones in this plot but  
 250:3 not all of these pictures, they're all either  
 250:4 1 or above, which is a very rare finding in  
 250:5 looking at these types of epi studies.

250:6 - 254:8

**Portier, Christopher 02-21-2019 (00:04:22)**

CP1\_SS\_01.69

250:6 Q. Okay. Why don't look at your  
 250:7 never, ever analysis. I believe it's  
 250:8 actually an exhibit here.  
 250:9 If you go into your -- in your  
 250:10 binder -- sorry, in your -- yeah, in your  
 250:11 binder, 893.  
 250:12 A. Oh, yes.  
 250:13 Q. Is that your never, ever  
 250:14 analysis?  
 250:15 A. That's the plot from the --  
 250:16 well, no, it's a modified version of the plot  
 250:17 from the expert report, but -- because it's  
 250:18 got Andreotti in it. But, yeah, that's  
 250:19 never, ever. That's the data.

250:20 Q. Okay. I'm going to push that  
250:21 up on the screen. So we're looking here at  
250:22 another plot summary.

250:23 This is just the never, ever  
250:24 data; is that right?

250:25 A. Correct. This is simply from  
251:1 the epi studies the comparisons of were you  
251:2 exposed or not exposed and looking at the  
251:3 relative risks.

251:4 Q. Now, Doctor, let's assume for a  
251:5 second that there actually is no relationship  
251:6 between Roundup exposure and non-Hodgkin's  
251:7 lymphoma.

251:8 Okay?

251:9 A. Okay.

251:10 Q. So let's assume that's the  
251:11 actual truth for a second.  
251:12 What is the likelihood that you  
251:13 would see data that looks like this?

251:14 A. So there's a way to address  
251:15 that question. It's one of the oldest  
251:16 statistical tests that exists.

251:17 So if truth is there's no  
251:18 effect whatsoever, then let's think of a  
251:19 coin. Coins, if it's fair, half the time  
251:20 it's heads, half the time it's tails.

251:21 If truth is there's no effect,  
251:22 then half the time you expect to see a little  
251:23 effect that's positive, and half the time you  
251:24 would expect to see a little effect that's  
251:25 negative.

252:1 And so if you turn this into is  
252:2 it positive, is it negative, simple question,  
252:3 then you'd expect to see about half and half.

252:4 Well, here what you see is  
252:5 everything's on the positive side except for  
252:6 Orsi, which is the -- down at the bottom,  
252:7 which is exactly on 1. And the probability  
252:8 of that happening can actually be calculated.  
252:9 It's one-half to the sixth power, because

252:10 there are six studies, and they're  
252:11 independent of each other.  
252:12 And that's a very small number,  
252:13 .03 or something along those lines. So it's  
252:14 a 3 percent chance that you'd see everything  
252:15 on the right-hand side. That's a very  
252:16 unusual finding.  
252:17 Q. What then is, in your opinion,  
252:18 the appropriate interpretation of this data?  
252:19 A. Well, I mean, you have to look  
252:20 at everything in interpreting all of this  
252:21 data. But when I look at everything I've  
252:22 seen in the epi data, including this, the  
252:23 meta-analyses, the understanding of how the  
252:24 studies were done, the strengths and the  
252:25 weaknesses of all of the studies, I see an  
253:1 association that's justified, there -- there  
253:2 is an association between NHL and glyphosate  
253:3 formulation exposure.  
253:4 I can't call it causal. And in  
253:5 my opinion, it's just not strong enough for  
253:6 me to bring me there all by itself.  
253:7 There's still potential for  
253:8 other things that could explain the results.  
253:9 I think the probability of those other things  
253:10 explaining the results is small, but I can't  
253:11 really rule it out.  
253:12 And so I'd say this is an  
253:13 association. It could be causal, but I can't  
253:14 absolutely say it's causal today with just  
253:15 this data.  
253:16 Q. So if we go back to this stool  
253:17 of causation, if I understand that correctly,  
253:18 if we got rid of the animal studies and got  
253:19 rid of the mechanism studies and you just  
253:20 look at the epi, it wouldn't be enough for  
253:21 you; is that right?  
253:22 A. To absolutely say this causes  
253:23 cancer in humans, it would not be enough.  
253:24 Q. That's not what we have here.

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253:25 A. That's correct.  
254:1 Q. We do have all this data.  
254:2 A. That's correct.  
254:3 Q. And when you look at all the  
254:4 data, sir, in your expert opinion, what does  
254:5 it show you?  
254:6 A. It shows me that glyphosate  
254:7 probably, with fairly high probability,  
254:8 causes non-Hodgkin's lymphoma in humans.

**Total Time = 03:41:41**

# PORTIER\_DAY2\_SS\_01 FINAL PLAYED

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Portier, Christopher 02-22-2019

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**Total Time 00:16:57**



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271:25 - 272:2

**Portier, Christopher 02-22-2019 (00:00:02)**

CP2\_SS\_01.2

271:25 Sir Bradford Hill; is that

272:1 right?

272:2 A. Correct.

272:6 - 273:15

**Portier, Christopher 02-22-2019 (00:00:50)**

CP2\_SS\_01.18

272:6 Q. Okay. And I don't want to

272:7 spend too much time talking about the history

272:8 of it, but I do want to talk about these

272:9 various points. It lists here consistency of

272:10 the observed association.

272:11 Do you see that?

272:12 A. Yes, I do.

272:13 Q. Strength of the observed

272:14 association?

272:15 A. Yes.

272:16 Q. Specificity of the observed

272:17 association.

272:18 Do you see that?

272:19 A. Yes.

272:20 Q. And then it has temporal

272:21 relationship of the observed association.

272:22 Do you see that?

272:23 A. Yep.

272:24 Q. Biological gradient.

272:25 Do you see that?

273:1 A. Yes.

273:2 Q. Biological plausibility.

273:3 Do you see that?

273:4 A. Yes.

273:5 Q. Coherence.

273:6 Do you see that?

273:7 A. Yes.

273:8 Q. And then experimental evidence.

273:9 But do we have experimental

273:10 evidence in this case?

273:11 A. Not from human populations, no.

273:12 Q. Why don't we? Why haven't

273:13 there -- why haven't there been a study, you

273:14 know, exposing people to glyphosate and other

273:15 people to placebo?

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274:11 - 274:23

**Portier, Christopher 02-22-2019 (00:00:30)**

CP2\_SS\_01.5

274:11 THE WITNESS: There are rules  
274:12 that govern how to treat human  
274:13 subjects in studies in the United  
274:14 States. Those rules are managed by  
274:15 the Department of Health and Human  
274:16 Services of the US government, which I  
274:17 was a member and senior manager for  
274:18 many years. And those rules would not  
274:19 allow you to administer a pesticide or  
274:20 something that has any indication of  
274:21 potential for human harm to humans in  
274:22 a controlled clinical trial over a  
274:23 long period of time.

275:14 - 278:25

**Portier, Christopher 02-22-2019 (00:03:19)**

CP2\_SS\_01.6

275:14 Q. All right. The last one here,  
275:15 sir, is analogy.  
275:16 Do you see that?  
275:17 A. Yes.  
275:18 Q. Okay. Great.  
275:19 So we've prepared a chart with  
275:20 these Bradford Hill factors to sort of go  
275:21 through them with you when it comes to  
275:22 glyphosate.  
275:23 Okay?  
275:24 A. Okay.  
275:25 Q. And as you can see here on the  
276:1 left, we have these considerations and then  
276:2 we have a blank area for strength.  
276:3 Do you see that?  
276:4 A. Yes, now I do.  
276:5 Q. Okay. Sorry, I guess it wasn't  
276:6 working yet. Great.  
276:7 So what I'd like to do is I'd  
276:8 like to go through these considerations one  
276:9 at a time, and as we go through them, kind of  
276:10 explain what they are to the jury so they  
276:11 understand what you're talking about.  
276:12 So let's start off with the  
276:13 first one, consistency of association.

276:14 What does that refer to?  
276:15 A. Consistency of association  
276:16 deals with the epidemiology data as a general  
276:17 rule.  
276:18 I should caveat this up front  
276:19 in saying that when Hill first proposed these  
276:20 criteria, which I now see was in 1965, he was  
276:21 interested in developing criteria for  
276:22 establishing causality of epidemiology data,  
276:23 in epidemiology data. Since then, most  
276:24 agencies have expanded this to establishing  
276:25 causality for a disease from the full set of  
277:1 data.  
277:2 So EPA's -- for example, EPA's  
277:3 criteria go beyond a bit what Bradford Hill  
277:4 had used, looking at a much more broad view  
277:5 of the animal data and the mechanistic data  
277:6 than Hill had in his presentation.  
277:7 That said, even in their  
277:8 evaluation, consistency deals with the  
277:9 epidemiology. The question is, do the  
277:10 studies show the same thing or approximately  
277:11 the same thing, one after the other. How  
277:12 consistent are they, both in magnitude and in  
277:13 direction.  
277:14 And in this case, the data is  
277:15 fairly strong on consistency. They all show  
277:16 the same general trend in a positive  
277:17 direction, with the exception of the  
277:18 Andreotti study, which has a number of  
277:19 failures that in my opinion would have put it  
277:20 into the potentially negative range to be  
277:21 expected. So in general, I think this is  
277:22 a -- there's strong consistency in these  
277:23 data.  
277:24 Q. Okay. So let's go to the  
277:25 screen. This was what we showed the jury  
278:1 yesterday, some of the epi studies that  
278:2 you're referring to?  
278:3 A. Yes.

278:4 Q. And we talk about the strength  
278:5 and -- strength of the consistency, is that  
278:6 reflected in nearly all of these points being  
278:7 to the right of the blue line?

278:8 A. That is correct.

278:9 Q. Okay. So let's go back to the  
278:10 chart here, and we have consistency of  
278:11 association. And I'll just put on here that  
278:12 we're talking about glyphosate.

278:13 The consistency of  
278:14 association -- sir, before I do that, is this  
278:15 the same thing for Roundup?

278:16 A. Yes.

278:17 Q. Okay.

278:18 A. My opinion on each of these is  
278:19 going to be the same for Roundup as it is for  
278:20 glyphosate.

278:21 Q. Okay. And for consistency of  
278:22 association, you said it was strong?

278:23 A. Yes.

278:24 And I should say, not Roundup,  
278:25 glyphosate-based formulations.

279:5 - 280:10

**Portier, Christopher 02-22-2019 (00:01:16)**

CP2\_SS\_01.7

279:5 Q. Okay. So we have this next one  
279:6 here, strength of association.  
279:7 What does that refer to in the  
279:8 Hill criteria?

279:9 A. It refers to the magnitude of  
279:10 the response originally when Hill was looking  
279:11 at it. Since then, because we've gotten  
279:12 better statistical methods and everything, it  
279:13 refers to the degree to which you have  
279:14 statistical significance in it as well as the  
279:15 magnitude of the actual observed effect.

279:16 In this case, because of the  
279:17 four meta-analyses, all of which are  
279:18 statistically significantly positive because  
279:19 of many of the studies being -- having some  
279:20 aspect of them that are statistically  
279:21 significant, I think, again, this is a strong

279:22 finding that there is -- a strength of  
 279:23 association in the epidemiology data is  
 279:24 strong enough to call it strong.

279:25 Q. If we can go back to the chart

280:1 here, this is the -- what you were talking

280:2 about, the bottom part here, these green

280:3 ones, that's the meta-analysis that you're

280:4 referring to?

280:5 A. Correct. All of them show

280:6 statistically significant findings above 1.

280:7 Q. Okay. Great.

280:8 So going back to the chart

280:9 here, this is strong; is that right?

280:10 A. Correct.

280:14 - 281:7

**Portier, Christopher 02-22-2019 (00:00:53)**

CP2\_SS\_01.8

280:14 Q. Okay. Biological plausibility,

280:15 what's that refer to?

280:16 A. Predominantly that refers to

280:17 the animal cancer data, the mechanism data.

280:18 Basically all of the laboratory data falls

280:19 into that category.

280:20 That data is extremely

280:21 convincing that glyphosate can cause tumors

280:22 in animal -- in mammalian systems, that there

280:23 are reasonable mechanisms by which that

280:24 occurs, and so I would label that very strong

280:25 in my opinion.

281:1 Q. And so I'm going to write that

281:2 in right now. "Very strong."

281:3 And just to, you know, go back

281:4 to what we've been doing in this examination,

281:5 you're talking about the mice studies; is

281:6 that right?

281:7 A. That is correct.

281:10 - 281:19

**Portier, Christopher 02-22-2019 (00:00:13)**

CP2\_SS\_01.9

281:10 THE WITNESS: And the rat

281:11 studies, that is correct.

281:12 QUESTIONS BY MR. WISNER:

281:13 Q. And then we have the -- what is

281:14 this referring to?

Page/Line	Source	ID
281:15 A. This is the genotoxicity data, 281:16 which is predominantly positive.		
281:17 Q. And again, this is in human 281:18 lymphocytes; is that right?		
281:19 A. That is correct.		
281:23 - 281:24	<b>Portier, Christopher 02-22-2019 (00:00:03)</b>	CP2_SS_01.10
281:23 Q. And then we have the recent 281:24 human genotox data?		
282:2 - 282:6	<b>Portier, Christopher 02-22-2019 (00:00:06)</b>	CP2_SS_01.11
282:2 THE WITNESS: And that is also 282:3 part of the opinion.		
282:4 QUESTIONS BY MR. WISNER:		
282:5 Q. And then we have the oxidative 282:6 stress data?		
282:9 - 284:2	<b>Portier, Christopher 02-22-2019 (00:01:36)</b>	CP2_SS_01.12
282:9 THE WITNESS: And that is also 282:10 part of the opinion.		
282:11 QUESTIONS BY MR. WISNER:		
282:12 Q. And so all this data, this data 282:13 we just went through really quickly, is that 282:14 what supports this idea of very strong?		
282:15 A. Yes, that is what supports the 282:16 very strong.		
282:17 Q. Okay. We have here gradient. 282:18 What does that refer to?		
282:19 A. Gradient refers to the concept 282:20 that as the exposure increases, the frequency 282:21 or the magnitude or the severity of the 282:22 cancer gets worse and worse.		
282:23 In this case, in the animal 282:24 evidence, it's quite clear that as you 282:25 increase the exposure, you're seeing		
283:1 increased cancer risk.		
283:2 In the human evidence, there's 283:3 some indication of that. Some of the studies 283:4 did not look at the issue; other studies did 283:5 look at the issue in some detail. Not all of 283:6 it was the same way every time or of the same 283:7 magnitude.		
283:8 I would argue that in this case		

283:9 that evidence is moderate.  
283:10 Q. Okay. So let's go to the --  
283:11 one of the exhibits we showed the jury  
283:12 yesterday. This was that never, ever  
283:13 analysis.  
283:14 Do you recall that?  
283:15 A. Yes.  
283:16 Q. But you also did the sort of  
283:17 time exposure response summary as well; is  
283:18 that right?  
283:19 A. Yes, this is a different --  
283:20 this is a different picture, yes.  
283:21 Q. Okay. And you refer to the  
283:22 gradient. So, for example, in McDuffie --  
283:23 well, I'll just cull it out.  
283:24 So in McDuffie, we have between  
283:25 zero and two days per year, and the risk  
284:1 ratio is 1.  
284:2 What does that mean?

284:5 - 286:21

**Portier, Christopher 02-22-2019 (00:02:46)**

CP2\_SS\_01.13

284:5 THE WITNESS: So in the  
284:6 McDuffie, et al., study, they tried to  
284:7 address the question of increasing  
284:8 exposure with increasing response. So  
284:9 they broke their exposed individuals  
284:10 into those receiving less than two  
284:11 days of exposure per year and those  
284:12 receiving greater than two days'  
284:13 exposure per year.  
284:14 The group getting less than two  
284:15 days' exposure per year had a relative  
284:16 risk of 1, which was clearly not  
284:17 significantly different from no  
284:18 effect, and the greater than two days  
284:19 per year had a relative risk of 2.12,  
284:20 which was statistically significant.  
284:21 So that does demonstrate an  
284:22 exposure response relationship.  
284:23 QUESTIONS BY MR. WISNER:  
284:24 Q. But sort of counteracting the

284:25 McDuffie one, let's look at De Roos.  
285:1 What does that show?  
285:2 A. The De Roos study, the 2005  
285:3 study from the Agricultural Health Study,  
285:4 showed, in fact, a -- they showed  
285:5 increasing -- well, the first was a drop. So  
285:6 basically they show nothing that's at all  
285:7 positive whatsoever. It's negative when they  
285:8 look at the concept of exposure response  
285:9 relationships. There's nothing there.  
285:10 Q. And when you said a second ago  
285:11 that this gradient is moderate, are you  
285:12 referring to these sort of conflicting  
285:13 results?  
285:14 A. That is correct.  
285:15 Q. Okay. Let's go back to the  
285:16 document camera. Put in moderate.  
285:17 All right. Temporality, what  
285:18 does that refer to?  
285:19 A. That refers to the concept that  
285:20 exposure must occur before the disease  
285:21 occurs. If that doesn't happen then, in  
285:22 fact, the disease can't be the cause --  
285:23 caused by the exposure.  
285:24 So it's a -- well, some -- many  
285:25 of these are not required to establish  
286:1 causality. This one is absolutely required  
286:2 to establish causality. I think it's  
286:3 satisfied in this case. Clearly people were  
286:4 exposed before the epidemiology studies were  
286:5 started, and in the animal studies that's  
286:6 quite obvious in the controlled situations.  
286:7 So this one is satisfied. I  
286:8 don't have to list it as strong or moderate.  
286:9 It's satisfied.  
286:10 Q. All right. What's specificity?  
286:11 A. Well, originally having read  
286:12 Bradford Hill's review, I thought specificity  
286:13 dealt with the fact that the disease that's  
286:14 being caused by the chemical agent has to be

286:15 unique, that the chemical agent is the only  
286:16 one that is known to cause it. That makes it  
286:17 very specific to that chemical, makes it very  
286:18 clear. And so in this case I would say that  
286:19 is not satisfied because NHL has a number of  
286:20 causes.

286:21 However,

286:23 - 288:4

**Portier, Christopher 02-22-2019 (00:01:09)**

CP2\_SS\_01.19

286:23 having heard some  
286:24 debate about this issue and going back and  
286:25 looking at several different articles on it,  
287:1 I have to concede the fact that there are two  
287:2 definitions for specificity.  
287:3 The second is that the chemical  
287:4 only has one disease which it appears to  
287:5 cause. That makes the epidemiology more  
287:6 specific.  
287:7 If the epidemiology were  
287:8 pointing to a bunch of different diseases,  
287:9 one would suspect, especially for  
287:10 case-control studies, one would suspect that  
287:11 maybe there's some recall bias going on, but  
287:12 that's not the case here. They're not  
287:13 pointing to all kinds of diseases; they're  
287:14 pointing at one disease.  
287:15 So here I would have to  
287:16 conclude that including that definition of  
287:17 specificity in here, I would say it's fairly  
287:18 strong.  
287:19 Q. Okay. So let's break that  
287:20 down.  
287:21 So the first one you're  
287:22 referring to whether or not NHL can only be  
287:23 caused by a chemical; is that right?  
287:24 A. By this chemical.  
287:25 Q. Okay. And then the second type  
288:1 of specificity is, of all the diseases that  
288:2 glyphosate could be causing, the data shows  
288:3 that it's causing just one specific one; is  
288:4 that right?

Page/Line	Source	ID
288:7 - 288:13	<p><b>Portier, Christopher 02-22-2019 (00:00:07)</b>            288:7 THE WITNESS: That is what I            288:8 was trying to portray, that is            288:9 correct.            288:10 QUESTIONS BY MR. WISNER:            288:11 Q. Okay. So for the first one,            288:12 it's not there, right?            288:13 A. It's not there.</p>	CP2_SS_01.14
288:16 - 289:3	<p><b>Portier, Christopher 02-22-2019 (00:00:30)</b>            288:16 QUESTIONS BY MR. WISNER:            288:17 Q. Okay. But for the second one,            288:18 and that is, what the glyphosate data is            288:19 showing in diseases, what is your            288:20 characterization of that?            288:21 A. It's strong.            288:22 Q. Okay. And I just want to            288:23 explore that issue on the epi a little bit            288:24 closer. I mean, Doctor, what is the            288:25 significance of the fact that in all these            289:1 different epidemiological studies, it's NHL            289:2 that keeps popping up, not some other type of            289:3 cancer?</p>	CP2_SS_01.15
289:6 - 290:21	<p><b>Portier, Christopher 02-22-2019 (00:01:48)</b>            289:6 THE WITNESS: Well, first you            289:7 have to remember that in case-control            289:8 studies, the cases are NHL. So in            289:9 those situations, you're not going to            289:10 be looking at any other disease.            289:11 But there are other            289:12 case-control studies here that looked            289:13 at the various other -- the end points            289:14 and other diseases for glyphosate and            289:15 really saw nothing. And it's those            289:16 studies that because there's nothing            289:17 going on there suggest that the NHL            289:18 findings are stronger than just random            289:19 chance.            289:20 QUESTIONS BY MR. WISNER:            289:21 Q. All right. Let's go to the            289:22 last one, coherence. What is that?</p>	CP2_SS_01.16

289:23 A. Coherence is a more complicated  
289:24 sort of thing. It's the catchall for  
289:25 everything else. Is the compound absorbed in  
290:1 humans. Is it metabolized to humans. Is it  
290:2 distributed to organs in humans. Are there  
290:3 similar pathologies in humans and animals.  
290:4 Does it make sense what you're seeing in the  
290:5 animal evidence to human evidence, the  
290:6 mechanistic evidence. Does all of it make  
290:7 sense. Does it stick together as one  
290:8 picture.  
290:9 And here I would have to say  
290:10 coherence is strong for two basic reasons.  
290:11 One is that the absorption, distribution,  
290:12 metabolism, the pharmacology of the compound  
290:13 as it enters human bodies is very similar to  
290:14 what happens with the other studies that  
290:15 we've looked at in the experimental evidence.  
290:16 And secondly, the malignant  
290:17 lymphomas in the mouse and the non-Hodgkin's  
290:18 lymphomas in the humans have commonalities  
290:19 that also add to the coherence argument.  
290:20 Q. So that's strong as well?  
290:21 A. That is strong as well.

290:25 - 292:16

**Portier, Christopher 02-22-2019 (00:01:43)**

CP2\_SS\_01.17

290:25 Q. Okay. So when you look at all  
291:1 these different Bradford Hill factors, right,  
291:2 you have strong, strong, very strong,  
291:3 moderate, satisfied, not there but strong,  
291:4 strong, what does that indicate to you as  
291:5 someone who has spent his career looking at  
291:6 whether or not stuff causes cancer?  
291:7 A. That the glyphosate and  
291:8 glyphosate-based formulations are probably  
291:9 causing non-Hodgkin's lymphoma in humans.  
291:10 Q. All right. I want to wrap up  
291:11 your testimony by sort of doing a summary. I  
291:12 wrote this up this morning, some questions.  
291:13 I just want to get a straight answer so we  
291:14 have a nice summary for the jury.

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291:15 So I have up here "does Roundup  
291:16 cause."  
291:17 Do you see that, sir?  
291:18 A. Yes.  
291:19 Q. All right. So the first  
291:20 question is, does Roundup cause tumors in  
291:21 mammals?  
291:22 A. Yes.  
291:23 Q. Does Roundup cause malignant  
291:24 lymphoma in mice?  
291:25 A. Yes.  
292:1 Q. Does Roundup cause genetic  
292:2 damage in human lymphocytes?  
292:3 A. Yes.  
292:4 Q. Does Roundup cause oxidative  
292:5 stress in human cells?  
292:6 A. Yes.  
292:7 Q. And finally, does Roundup cause  
292:8 non-Hodgkin's lymphoma in humans at real  
292:9 world exposures?  
292:10 A. Yes, with high probability.  
292:11 Q. And, sir, when you offer these  
292:12 opinions, do you offer them to a reasonable  
292:13 degree of scientific certainty?  
292:14 A. Yes.  
292:15 MR. WISNER: Thank you. I pass  
292:16 the witness.

**Total Time = 00:16:57**

# Portier Day 2 DC 0228-1400 FINAL PLAYED

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PORTIER, CHRISTOPHER 2019-02-22\_SS  
PORTIER, CHRISTOPHER 2019-02-22\_PIP

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[REDACTED]

Total Time 01:45:05



293:3 - 295:23

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:54)**

M20.1

293:3 Q. Doctor, good morning. My name  
293:4 is Paul Schmidt, and I represent Monsanto in  
293:5 this case.  
293:6 We met for the first time the  
293:7 other day, correct?  
293:8 A. Yes.  
293:9 Q. We have some time constraints  
293:10 today, so I'm going to do my best where I can  
293:11 to be simple and direct in my questions, and  
293:12 I'm going to ask you to help out by, where  
293:13 you can, being simple and direct in your  
293:14 answers.  
293:15 Is that fair?  
293:16 A. That's fair.  
293:17 Q. Thank you.  
293:18 Let me start off with just one  
293:19 of those -- what I hope is a simple question,  
293:20 simple yes/no question.  
293:21 Do you recognize that there are  
293:22 scientists who disagree with the views you've  
293:23 offered in this case on glyphosate?  
293:24 A. Yes.  
293:25 Q. There are independent  
294:1 scientists who disagree, correct?  
294:2 A. I'm sorry, what was the word in  
294:3 between?  
294:4 Q. Independent. There are  
294:5 independent scientists who disagree with the  
294:6 views you've offered in this case?  
294:7 A. I don't know what independent  
294:8 means.  
294:9 Q. Okay. But there are scientists  
294:10 out there in the published literature who  
294:11 have, correct?  
294:12 A. In the published literature?  
294:13 Q. And at regulatory agencies.  
294:14 A. I would say yes.  
294:15 Q. Okay. Let me just cover a few  
294:16 details about your background, and then I'll

294:17 go into some of your work on glyphosate and

294:18 your opinions on glyphosate, if that's okay.

294:19 I'd like to start with your

294:20 professional background.

294:21 As I understand it, you're a

294:22 biostatistician; is that correct?

294:23 A. My training and my Ph.D. is

294:24 biostatistics.

294:25 Q. You're not a medical doctor?

295:1 A. I am not a medical doctor.

295:2 Q. You've never diagnosed a

295:3 patient with NHL, for example?

295:4 A. No, I have not.

295:5 Q. You've never treated a patient

295:6 with NHL?

295:7 A. No, I have not.

295:8 Q. And you've never told a patient

295:9 the cause of their NHL?

295:10 A. No, I have not.

295:11 Q. And have you ever reviewed

295:12 individual patient's pathology slides to

295:13 determine whether they have NHL or something

295:14 else?

295:15 A. No.

295:16 Q. And last question in this area:

295:17 Because you're not a medical doctor, by

295:18 definition that means you're not an

295:19 oncologist?

295:20 A. Umm --

295:21 Q. Oncology being the field of

295:22 medicine that studies cancer.

295:23 A. Then by that definition, no.

296:15 - 296:24

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:17)**

M20.2

296:15 You're not here to talk about

296:16 whether Roundup or glyphosate actually caused

296:17 the cancer of the plaintiff in this case; is

296:18 that fair?

296:19 A. I think that's fair.

296:20 Q. And you haven't reviewed the

296:21 plaintiff's medical records or reviewed the

Page/Line	Source	ID
299:13 - 299:17	<p>296:22 medical testimony of doctors who have treated  296:23 the plaintiff in this case; is that correct?  296:24 A. That is correct.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</b></p>	M20.3
300:7 - 300:10	<p>299:13 Q. Okay. And you don't have any  299:14 knowledge on when the plaintiff did use  299:15 Roundup, how much they used at any one time?  299:16 A. I have no knowledge of the  299:17 plaintiff at all.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</b></p>	M20.4
302:24 - 303:5	<p>300:7 Are you aware that NHL is one  300:8 of the most common cancers in the United  300:9 States?  300:10 A. Yes.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</b></p> <p>302:24 Q. Okay. Other than NHL and  302:25 things that might be forms of NHL, you've not  303:1 given an opinion that glyphosate causes other  303:2 forms of cancer at this time?  303:3 A. In humans.  303:4 Q. In humans.  303:5 A. That is correct.</p>	M20.5
308:7 - 308:10	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</b></p> <p>308:7 So my question is simply, with  308:8 your understanding and your impression, do  308:9 you agree or disagree that the cause of most  308:10 lymphomas is not known?</p>	M20.6
308:13 - 308:19	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</b></p> <p>308:13 THE WITNESS: Again, I agree  308:14 with that statement --  308:15 QUESTIONS BY MR. SCHMIDT:  308:16 Q. Thank you, Doctor.  308:17 A. -- when the cause is genetic.  308:18 Q. Okay. Is it true that getting  308:19 older is a strong risk factor for lymphoma?</p>	M20.7
308:23 - 309:14	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26)</b></p> <p>308:23 THE WITNESS: Getting older, as  308:24 a general rule, is a risk factor for  308:25 most carcinomas, for most cancers.  309:1 QUESTIONS BY MR. SCHMIDT:</p>	M20.8

Page/Line	Source	ID
	309:2 Q. Is it for NHL?	
	309:3 A. I'm not certain.	
	309:4 Q. Okay. Do you know if most	
	309:5 cases of NHL occur with people in their 60s	
	309:6 or older?	
	309:7 A. I would not be surprised if	
	309:8 that were the case, but I have no direct	
	309:9 knowledge of it.	
	309:10 Q. Is gender a risk factor for	
	309:11 NHL?	
	309:12 A. I do understand that males have	
	309:13 a slightly higher incidence of NHL than	
	309:14 females.	
311:5 - 311:13	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</b>	M20.9
	311:5 Q. And so you would agree that	
	311:6 persistent immunosuppression presents a risk	
	311:7 of cancer, especially excess risk for	
	311:8 lymphoma?	
	311:9 A. I don't -- I don't -- I'm not	
	311:10 certain about the second half.	
	311:11 Q. Okay.	
	311:12 A. Immunosuppression is a known	
	311:13 risk factor for induction of cancers.	
311:14 - 311:15	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04)</b>	M20.10
	311:14 Q. I've put in front of you Trial	1501.1
	311:15 Exhibit 1501.	
311:21 - 312:14	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:43)</b>	M20.11
	311:21 Q. This is an article you wrote,	1501.1.1
	311:22 correct?	
	311:23 A. I am a coauthor on the article,	
	311:24 yes.	
	311:25 Q. We have it up on the screen	
	312:1 now. What I'd like to do is turn to page 716	1501.4
	312:2 of the document where you're listing some of	
	312:3 the characteristics you've spoken about with	1501.4.1
	312:4 us here today.	
	312:5 Do you see that?	
	312:6 A. Yes.	
	312:7 Q. And let me just cull out the	
	312:8 language I was reading to you.	

Page/Line	Source	ID
	312:9 Do you see where you write, 312:10 "Persistent immunosuppression presents a risk 312:11 of cancer, especially excess risk for 312:12 lymphoma"? 312:13 Did I read that correctly? 312:14 A. Yes, you did.	1501.4.2
313:16 - 314:16	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:47)</b> 313:16 Q. Okay. I'd like to ask you 313:17 about some of your work that you talked about 313:18 earlier on glyphosate. 313:19 I think when you were speaking 313:20 yesterday with the plaintiff lawyer, you 313:21 talked about your years of experience at 313:22 groups like NTP and NIEHS, correct? 313:23 A. Correct. 313:24 Q. As I understand what you were 313:25 saying, you have about 35 years of experience 314:1 there before you retired? 314:2 A. And CDC, yes. 314:3 Q. Yes. 314:4 And I might have missed the 314:5 exact percentage, but I think you said 314:6 somewhere in the neighborhood of 80 to 314:7 90 percent of your work was on carcinogens; 314:8 is that correct? 314:9 A. Especially when I was at NIH 314:10 and NTP. 314:11 Q. During that time, that 35 years 314:12 of work and that 80 to 90 percent of the time 314:13 on carcinogens, you never came to the opinion 314:14 that glyphosate was a carcinogen during that 314:15 time, true? 314:16 A. Not that I'm aware of.	M20.12 clear
315:9 - 316:3	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</b> 315:9 My question is simply, prior to 315:10 the IARC review, you never even thought about 315:11 glyphosate, correct? 315:12 A. That's correct. 315:13 Q. And just so the jury 315:14 understands, when you talk about that service	M20.13

Page/Line

Source

ID

315:15 that you have, NTP, NIH, NIEHS, CDC, you're

315:16 not here in court speaking for any of those

315:17 agencies, correct?

315:18 A. That is correct.

315:19 Q. You're offering your own

315:20 personal views?

315:21 A. That's correct.

315:22 Q. Now, when you were at NIEHS,

315:23 you had your own laboratory; is that true?

315:24 A. That is true.

315:25 Q. And you were able to do tests

316:1 on things of interest to you; is that

316:2 correct?

316:3 A. That is correct.

316:10 - 316:12

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:05)**

M20.14

316:10 You did not do any testing on

316:11 glyphosate at your laboratory at NIEHS?

316:12 A. No, I did not.

316:13 - 317:12

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:48)**

M20.15

316:13 Q. While you were at -- while you

316:14 were doing work with NTP, are you aware that

316:15 other scientists at NTP did do testing on

316:16 glyphosate?

316:17 A. No.

316:18 I am aware that NTP has a

316:19 document on glyphosate.

316:20 Q. And that dates from the time

316:21 when you were doing work with NTP, correct?

316:22 A. I don't recall.

316:23 Q. Were you doing work with NTP in

316:24 1992?

316:25 A. Yes.

317:1 Q. Okay. And to be fair, you

317:2 didn't do work on this document I'm about to

317:3 show you --

317:4 A. No.

317:5 Q. -- correct?

317:6 But you have seen it before?

317:7 A. I've seen it since I've been

317:8 working -- since the IARC review.

Page/Line	Source	ID
	317:9 Q. It's Trial Exhibit 1098.	1098.1
	317:10 And, sir, am I correct that you	
	317:11 recognize NTP as an authority?	
	317:12 A. Yes.	
317:21 - 318:1	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)</b>	<b>M20.16</b>
	317:21 Q. If we look in the upper corner,	1098.1.1
	317:22 you see this is a National Toxicology Program	
	317:23 document?	
	317:24 A. You're not showing it on there,	
	317:25 but -- there you go. Yes, I do see that it's	
	318:1 part of their toxicity report series.	
318:2 - 318:9	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)</b>	<b>M20.17</b>
	318:2 Q. Okay. And is that a regular	
	318:3 series that they would conduct, periodic	
	318:4 series?	
	318:5 A. Yes, it reports -- if you	
	318:6 remember yesterday I talked about 90-day	
	318:7 studies in order to set doses for -- this is	
	318:8 the reporting of findings from 90-day	
	318:9 studies.	
318:10 - 318:20	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:20)</b>	<b>M20.18</b>
	318:10 Q. Part of their -- part of NTP's	
	318:11 periodic work?	
	318:12 A. Correct.	
	318:13 Q. As a government agency?	
	318:14 A. Correct.	
	318:15 Q. And do you see that this is	
	318:16 dated July 1992, when you were doing work	1098.1.3
	318:17 with NTP?	
	318:18 A. Yes.	
	318:19 Q. I just want to show you a few	
	318:20 things from this document.	
319:19 - 319:23	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:16)</b>	<b>M20.19</b>
	319:19 Do you see on numbered page 12	
	319:20 where they talk about a study that they	1098.14.2
	319:21 conducted on rats and mice?	
	319:22 A. That is what it's talking	
	319:23 about, yes.	
320:7 - 320:23	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:53)</b>	<b>M20.20</b>
	320:7 Q. Do you see on page 16 they make	1098.18.3

Page/Line	Source	ID
	320:8 reference to mutagenicity studies they've 320:9 conducted?	
	320:10 A. Yes.	
	320:11 Q. And if we stay on the same 320:12 page, below that, do you see that they make 320:13 reference to a micronucleus test that they 320:14 conducted?	1098.18.4
	320:15 And I'll put it up on the 320:16 screen, if that helps as well.	
	320:17 A. Yes. No, that's a micronucleus 320:18 study, yes, correct.	
	320:19 Q. Specifically, they indicate 320:20 that 10,000 normochromatic erthrocytes from 320:21 each animal were scored for micronuclei. 320:22 Do you see that?	1098.18.5
	320:23 A. Correct.	
321:22 - 322:3	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</b>	M20.21
	321:22 Q. Do you mind looking at page 6 321:23 of the NTP study from 1992?	1098.8
	321:24 A. I'm looking at it.	
	321:25 Q. And do you see where it says, 322:1 "Glyphosate was not mutagenic in salmonella 322:2 and did not introduce micronuclei in mice"?	1098.8.2
	322:3 A. I see where it says that, yes.	
322:7 - 322:8	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08)</b>	M20.22
	322:7 Q. On page 36 of this study, down 322:8 near the bottom, do you see where they say,	1098.38 - 1098.38.2
322:9 - 323:21	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:02)</b>	M20.23
	322:9 "There was no evidence of genetic or 322:10 reproductive toxicity of glyphosate"?	
	322:11 Do you see that?	
	322:12 A. No.	
	322:13 Q. It's up --	
	322:14 A. Oh. I see.	
	322:15 Q. Do you see that?	
	322:16 Did I read that correctly?	
	322:17 A. Yes, you did read it correctly.	clear
	322:18 Q. Am I correct that you don't 322:19 disagree with the findings of this one study?	
	322:20 A. In Fischer rats and B63F1 mice,	

322:21 I do not disagree with the findings of this  
322:22 study.

322:23 Q. And you didn't recommend while  
322:24 you were working with NTP that they do any  
322:25 additional glyphosate testing, true?

323:1 A. I had nothing to do with this  
323:2 or with glyphosate.

323:3 Q. There were studies that existed  
323:4 on glyphosate when you were working at the  
323:5 government, correct?

323:6 A. Probably.

323:7 Q. In fact --

323:8 A. Most certainly, actually.

323:9 Knowing the literature now, of course they  
323:10 existed.

323:11 Q. Yeah. And in fact, you  
323:12 published talking about at least one of those  
323:13 studies while you were working with  
323:14 government, correct?

323:15 A. It's been pointed out to me  
323:16 before, but I don't recall it --

323:17 Q. Okay.

323:18 A. -- to be honest.

323:19 Q. If I may, let me point it out  
323:20 again.

323:21 A. Sure.

323:22 - 324:15

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:34)**

**M20.24**

323:22 Q. Doctor, I passed you an  
323:23 exhibit, 1657.

1657.1

323:24 Do you have that in front of  
323:25 you?

324:1 A. Yes, I do.

1657.1.4

324:2 Q. And do you recognize that this  
324:3 is an article that you published along with  
324:4 someone named David Resnik?

324:5 A. Yes, I do.

324:6 Q. And if you look at the  
324:7 disclosure after the document, you list  
324:8 yourself as being at the NTP at the time of  
324:9 this document.

1657.1.5

Page/Line	Source	ID
	324:10 Do you see that?	
	324:11 Just right up at the top after	
	324:12 your name, there's a 2, and then immediately	
	324:13 beneath it lists NTP.	
	324:14 Do you see that?	
	324:15 A. Yes.	
324:22 - 325:10	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</b>	M20.25
	324:22 And this is an article that you	
	324:23 wrote in 2005, correct?	
	324:24 A. Yes.	
	324:25 Q. If you look at page 3,	
	325:1 specifically in the bottom left-hand column,	1657.3.2
	325:2 do you see that there is reference to a study	
	325:3 by McDuffie from 2001? Do you see that?	
	325:4 It's also highlighted up on the	
	325:5 screen, if that helps.	
	325:6 A. Yes.	
	325:7 Q. And that's the study that	
	325:8 you've -- that's one of the studies that	
	325:9 you've discussed in this case, correct?	clear
	325:10 A. Correct.	
325:18 - 328:1	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:53)</b>	M20.26
	325:18 Q. Nowhere in this article citing	
	325:19 this study do you offer any conclusion about	
	325:20 glyphosate being carcinogenic, correct?	
	325:21 A. Correct.	
	325:22 Q. That's because you wrote this	
	325:23 article before you had come to any conclusion	
	325:24 about the carcinogenicity of glyphosate,	
	325:25 true?	
	326:1 A. Well, it wasn't the purpose of	
	326:2 this paper, to look at any specific	
	326:3 pesticide. It was just raising an issue	
	326:4 about pesticides in general.	
	326:5 Q. Is it true that you wrote this	
	326:6 article before you had reached a conclusion	
	326:7 regarding the carcinogenicity of glyphosate?	
	326:8 A. Oh, absolutely.	
	326:9 Q. Thank you.	
	326:10 You mentioned something	

326:11 yesterday called the Report on Carcinogens.  
326:12 Do you remember mentioning  
326:13 that?  
326:14 A. Yes.  
326:15 Q. And I think what you said is  
326:16 that you were responsible for making final  
326:17 recommendations about should -- what should  
326:18 go in the Report on Carcinogens while you  
326:19 were at NTP; is that right?  
326:20 A. For six of the years, yes.  
326:21 Q. And the Report on Carcinogens  
326:22 identifies -- I'm going to get the  
326:23 terminology wrong, but it identifies known or  
326:24 potential carcinogens, correct?  
326:25 A. Yeah, the terminology is "known  
327:1 or reasonably anticipated to be  
327:2 carcinogenic."  
327:3 Q. Okay. So let me see if I have  
327:4 that right.  
327:5 The purpose of the report on  
327:6 the carcinogens is for our National  
327:7 Toxicology Program to identify what is known  
327:8 or reasonably anticipated to be carcinogenic,  
327:9 correct?  
327:10 A. Not exactly. The purpose of  
327:11 the Report on Carcinogens, as established by  
327:12 law, is for the secretary of Health and Human  
327:13 Services to maintain a list of what is known  
327:14 or reasonably anticipated to be a human  
327:15 carcinogen. And she or he have designated  
327:16 the NTP to provide them with advice on what  
327:17 should be on that list, but they make the  
327:18 final decision.  
327:19 Q. Got it.  
327:20 Did you ever recommend  
327:21 glyphosate be on that list when you were at  
327:22 NTP?  
327:23 A. No.  
327:24 Q. When you had that  
327:25 responsibility you told us about yesterday?

328:22 - 330:13

328:1 A. No.

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:11)**

M20.27

328:22 You've offered an opinion today

328:23 that glyphosate can cause cancer; is that

328:24 right?

328:25 A. Yes.

329:1 Q. You've never carried out any

329:2 experiments on glyphosate, true?

329:3 A. True.

329:4 Q. You talked about the three legs

329:5 of the stool, Mr. Wisner's stool: human

329:6 epidemiology studies, animal studies and

329:7 mechanistic studies.

329:8 Do you recall that?

329:9 A. Yes, I do.

329:10 Q. To this date, you've never

329:11 conducted a human epidemiological study on

329:12 glyphosate, true?

329:13 A. On glyphosate, that is true.

329:14 Q. To this date, you've never

329:15 conducted an animal study on glyphosate; is

329:16 that true?

329:17 A. That is true.

329:18 Q. To this date, you've never

329:19 personally conducted an in vitro genotoxicity

329:20 assay on glyphosate; is that true?

329:21 A. That is true.

329:22 Q. I'd like to talk with you for a

329:23 moment about how you became involved in this

329:24 lawsuit.

329:25 You talked yesterday about

330:1 doing work with the working group of IARC.

330:2 Do you remember that?

330:3 A. Yes.

330:4 Q. That was in March of 2015 that

330:5 that culminated, correct?

330:6 A. I believe it is, yes.

330:7 Q. And shortly after that the

330:8 IARC -- a summary of the IARC view on

330:9 glyphosate was published in a journal called

Page/Line	Source	ID
330:22 - 330:25	<p>330:10 The Lancet.  330:11 Do you remember that?  330:12 A. Yes. It was about two or three  330:13 weeks after the working group meeting.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:05)</b></p>	M20.28
331:6 - 331:19	<p>330:22 Do you understand talking  330:23 yesterday about the exact rating that IARC  330:24 gave glyphosate?  330:25 A. Yes.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:29)</b></p>	M20.29
331:24 - 332:11	<p>331:6 Q. And the rating for the human  331:7 evidence was that there was limited evidence  331:8 in humans for the carcinogenicity of  331:9 glyphosate, correct?  331:10 A. Correct.  331:11 Q. And limited evidence means that  331:12 a positive association has been observed  331:13 between exposure to the agent and cancer for  331:14 which a causal interpretation is considered  331:15 by the working group to be credible, but  331:16 chance, bias or confounding could not be  331:17 ruled out with reasonable confidence,  331:18 correct?  331:19 A. That is the definition.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:28)</b></p>	M20.30
333:21 - 334:11	<p>331:24 Do you agree with that as a  331:25 correct description of the human data on  332:1 glyphosate?  332:2 A. That there is an association,  332:3 it is potentially causal, and that -- I'm not  332:4 so sure about bias, but confounding and  332:5 chance I can't really rule out, and so, yes,  332:6 I do disagree with the statement.  332:7 Q. And overall, you agree with the  332:8 overall designation that there's limited  332:9 evidence of human carcinogenicity, true?  332:10 A. If I applied that definition,  332:11 yes, it would be limited.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26)</b>  333:21 Q. We talked about that Lancet</p>	M20.31

333:22 publication.

333:23 Within a week or so of The

333:24 Lancet publication, you had had an agreement

333:25 with the plaintiff lawyers to consult with

334:1 them, correct?

334:2 A. It was a little longer than a

334:3 week after The Lancet publication, but, yes.

334:4 Q. I think it was about nine days,

334:5 right?

334:6 A. Yes.

334:7 Q. And those were lawyers you knew

334:8 from before, correct?

334:9 A. They were people who had called

334:10 me for my opinion, free of charge, on a

334:11 number of issues beforehand, yes.

335:7 - 336:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:00)**

M20.32

335:7 Q. Okay. And then after The

335:8 Lancet publication on glyphosate, they called

335:9 you back; is that right?

335:10 A. Correct.

335:11 Q. And you signed a contract with

335:12 them?

335:13 A. Well, they asked me to provide

335:14 them with advice again on this issue, and I

335:15 suggested that maybe it was becoming to take

335:16 up a lot more of my time than I had planned,

335:17 and so we wrote a contract on it, that is

335:18 correct.

335:19 Q. And from that time forward, you

335:20 got paid for your work for plaintiff lawyers

335:21 on glyphosate, true?

335:22 A. That would be true.

335:23 On the work I did for the

335:24 lawyers on glyphosate, yes.

335:25 Q. Yes, that's what I was asking.

336:1 Now, let me move forward a few

336:2 months after you signed that contract.

336:3 You talked yesterday about

336:4 something called EFSA.

336:5 Do you remember talking about

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336:6 EFSA yesterday?

336:7 A. Yes, I do.

336:8 Q. And EFSA stands for the

336:9 European Food Safety Agency, correct?

336:10 A. I think so. I get authority

336:11 and agency mixed up all the time, but --

336:12 Q. Fair enough.

336:13 A. It's one of the other.

337:3 - 338:9

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:01)**

M20.33

337:3 Q. You have presented your views

337:4 to the Europeans regarding what you think

337:5 EFSA is doing, correct?

337:6 A. I have presented my views in an

337:7 open letter that I'm absolutely certain EFSA

337:8 saw since they responded to it. I've

337:9 presented my views on some aspects of it to

337:10 the European parliament, but again, to EFSA

337:11 directly, no.

337:12 Q. Okay. Is this a copy of that

337:13 letter that you were just referencing, what

337:14 I've marked as Exhibit 1640 --

337:15 A. Yes.

337:16 Q. -- from November 27, 2015,

337:17 written by you to the Commissioner of Health

337:18 and Food Safety at European Commission?

337:19 A. It's written by me and my

337:20 colleagues to the Commissioner for Health and

337:21 Food Safety and the European Commission.

337:22 Q. Do you see that you've cc'd

337:23 various people?

337:24 A. Correct.

337:25 Q. And tell the jury who the third

338:1 cc is on this letter.

338:2 A. Dr. Bernhard Url, who is the

338:3 executive director of EFSA.

338:4 Q. Okay. So this did go to EFSA

338:5 by your direction?

338:6 A. Correct.

338:7 Q. Thank you.

338:8 A. It wasn't directed to them, but

1640.1

1640.1.4

1640.1.2

clear

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338:10 - 339:4	<p>338:9 you're correct. I stand corrected.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:37)</b></p>	M20.34
	<p>338:10 Q. Now, EFSA had come to the view,  338:11 or had expressed the view, that glyphosate is  338:12 unlikely to pose a carcinogenic hazard to  338:13 humans, correct?</p>	
	<p>338:14 A. Some wording along those lines,  338:15 that's correct.</p>	
	<p>338:16 Q. And in fact, you quote that in  338:17 the first paragraph of your letter, about  338:18 halfway or two-thirds of the way down.  338:19 Do you see that in your letter?</p>	1640.1.5
	<p>338:20 A. Yes, I do.</p>	
	<p>338:21 Q. EFSA's conclusion that  338:22 glyphosate is unlikely to pose a carcinogenic  338:23 hazard to humans?</p>	
	<p>338:24 A. That is correct.</p>	
	<p>338:25 Q. And you were obviously writing  339:1 because you disagreed with that, right?</p>	clear
	<p>339:2 A. We disagreed with -- we  339:3 disagreed with the scientific way in which  339:4 they arrived at that decision.</p>	
339:5 - 339:10	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</b></p>	M20.35
	<p>339:5 Q. You believed it should be  339:6 classified as a carcinogen, correct?  339:7 A. I believe they should have  339:8 followed their guidelines and done the  339:9 science the way they're supposed to have done  339:10 their job.</p>	
339:11 - 339:22	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:16)</b></p>	M20.36
	<p>339:11 Q. I'm going to ask you to focus  339:12 on my question.  339:13 Did you believe they should  339:14 have classified it as a carcinogen?</p>	
	<p>339:15 A. I believe they should have  339:16 classified it as 2B or 2A, absolutely, yes.</p>	
	<p>339:17 Q. Okay.</p>	
	<p>339:18 A. I don't know if we say that in  339:19 here.</p>	
	<p>339:20 Q. And they wrote back to you,</p>	

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	339:21 right?	
	339:22 A. They did write back to me.	
341:2 - 341:25	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:48)</b>	<b>M20.37</b>
	341:2 Q. Doctor, do you have in front of	1639.1
	341:3 you EFSA's letter to you dated January 13,	
	341:4 2016?	
	341:5 A. Yes, I do.	
	341:6 Q. And we've put it up on the	1639.1.4
	341:7 screen.	
	341:8 Do you see the EFSA logo in the	
	341:9 upper left corner?	
	341:10 A. Yes, I do.	
	341:11 Q. And if we look below that, you	
	341:12 can see the date, January 13, 2016.	
	341:13 Do you see that?	
	341:14 A. Yes, I do.	
	341:15 Q. And if you look below that,	
	341:16 they've written directly to you, "Dear	
	341:17 Professor Portier."	1639.1.5
	341:18 Do you see that?	
	341:19 A. Yes, I do.	
	341:20 Q. I want to just focus on a	
	341:21 couple things in this letter.	
	341:22 First of all, do you see that	
	341:23 they have a letter and then they have an	
	341:24 annex with specific responses?	1639.4.2
	341:25 A. Yes, I do.	
342:1 - 342:16	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)</b>	<b>M20.38</b>
	342:1 Q. And we see that up on the	
	342:2 screen, correct?	
	342:3 A. The annex, yes.	
	342:4 Q. Let's jump ahead to numbered	
	342:5 page 12 of the annex, which is up on the	1639.16
	342:6 screen, which says "Summary," and tell me	1639.16.5
	342:7 when you're there.	
	342:8 A. I'm there.	
	342:9 Q. Okay. I just want to call out	
	342:10 this first paragraph. Do you see where they	1639.16.6
	342:11 say, "EFSA considers that the arguments	
	342:12 brought forward in the open letter do not	

Page/Line	Source	ID
343:1 - 343:5	<p>342:13 have an impact on the EFSA conclusion on 342:14 glyphosate"?</p> <p>342:15 Did I read that correctly?</p> <p>342:16 A. You read it correctly.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</b></p> <p>343:1 QUESTIONS BY MR. SCHMIDT:</p> <p>343:2 Q. The open letter that they're 343:3 referencing, that is your letter, correct?</p> <p>343:4 A. That is the letter from me and 343:5 my colleagues.</p>	M20.39
343:12 - 343:13	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</b></p> <p>343:12 Q. They go on to say in the next 343:13 paragraph, "As reported in the EFSA</p>	M20.40 1639.16.7
343:14 - 343:20	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25)</b></p> <p>343:14 conclusion, there is very limited evidence 343:15 for an association between glyphosate-based 343:16 formulations and non-Hodgkin's lymphoma, and 343:17 overall evidence is inconclusive for a causal 343:18 or otherwise convincing associative 343:19 relationship between glyphosate and cancer in 343:20 human studies."</p>	M20.41
344:9 - 344:18	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)</b></p> <p>344:9 Did I read that statement 344:10 correctly?</p> <p>344:11 A. You read the statement 344:12 correctly.</p> <p>344:13 Q. Okay. Thank you, Doctor. 344:14 And that actually anticipated 344:15 my next question, which is, it's safe to say 344:16 you disagreed with EFSA and they disagreed 344:17 with you, correct? Is that true?</p> <p>344:18 A. That is true.</p>	M20.42
345:22 - 346:2	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</b></p> <p>345:22 Q. And I'm just going to pass you 345:23 a copy of this letter. I'm not going to put 345:24 it up on the screen, but just in fairness to 345:25 you so you have it handy.</p> <p>346:1 Do you recognize that as 346:2 Exhibit 1642?</p>	M20.43
346:3 - 346:3	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:01)</b></p>	M20.44

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346:4 - 346:9	<p>346:3 A. Yes, I do.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</b></p> <p>346:4 Q. And if you look at your  346:5 signature line this time -- you talked about  346:6 how some colleagues joined you in your  346:7 earlier letter. This time it's you alone,  346:8 correct? You're the only signatory?  346:9 A. That is correct.</p>	M20.45
346:25 - 347:9	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</b></p> <p>346:25 Q. My question, sir, simply: At  347:1 this point you were commenting on both EFSA  347:2 and on a group called ECHA, the European  347:3 Chemical Agency; is it true?  347:4 A. That is correct.  347:5 Q. Both of them had issued views  347:6 on glyphosate that you disagreed with,  347:7 correct?  347:8 A. I disagreed with the way they  347:9 interpreted the scientific evidence.</p>	M20.46
347:21 - 348:14	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:32)</b></p> <p>347:21 They did reach a conclusion,  347:22 correct?  347:23 A. They did reach a conclusion.  347:24 Q. Did you agree or disagree with  347:25 it?  348:1 A. I disagree with their  348:2 conclusion.  348:3 Q. Thank you.  348:4 And their conclusion was, and  348:5 you quote it in the executive summary for  348:6 your letter, was that the evidence does not  348:7 support a classification for glyphosate; is  348:8 that correct?  348:9 A. That was ECHA's conclusion;  348:10 that is correct.  348:11 Q. ECHA's also -- is it a public  348:12 health agency or scientific agency in Europe?  348:13 A. ECHA is -- I guess it's a  348:14 science agency in Europe.</p>	M20.47
356:19 - 357:18	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)</b></p>	M20.48

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356:19 Q. In addition to -- you spent  
356:20 some time talking about EPA.

356:21 Do you recall that?

356:22 A. Yes, I do.

356:23 Q. And in addition to EFSA, you  
356:24 also reached out to EPA, correct?

356:25 A. I sent public comments to an  
357:1 EPA document.

357:2 Q. For example, if you look back  
357:3 at that first letter you mentioned where you  
357:4 copied EFSA, you also copied EPA on that  
357:5 letter, correct?

357:6 A. That is correct.

357:7 Q. And then later you submitted  
357:8 public comments to them again, correct?

357:9 A. When the time was correct for  
357:10 its public comments, yes.

357:11 Q. And let's be precise. You  
357:12 understand that pesticides in the United  
357:13 States periodically go through a review  
357:14 process by EPA, correct?

357:15 A. That is correct.

357:16 Q. And that's happened for  
357:17 glyphosate as well?

357:18 A. That is correct.

358:4 - 358:4 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:02)**

M20.49

358:4 Do you understand that in 2016

358:5 - 358:15 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:16)**

M20.50

358:5 EPA conducted

358:6 another review of glyphosate?

358:7 A. The EPA conducted a review of  
358:8 glyphosate, that is correct.

358:9 Q. And the scientists at the EPA  
358:10 categorized glyphosate as not likely to be  
358:11 carcinogenic to humans, correct?

358:12 A. In their draft proposal.

358:13 Q. And it was that proposal that  
358:14 you made comments on, correct?

358:15 A. That is correct.

359:2 - 359:22 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:41)**

M20.51

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359:2 There were three sets of  
 359:3 comments you made that went to the EPA?  
 359:4 A. That went to the record, yes.  
 359:5 Q. Okay. That's what I was trying  
 359:6 to get at.  
 359:7 And among those comments was  
 359:8 your view that EPA should declare glyphosate  
 359:9 a probable human carcinogen, correct?  
 359:10 A. I don't remember saying that.  
 359:11 Q. You don't?  
 359:12 A. No, I don't.  
 359:13 My comments were towards the  
 359:14 science, again, the issues related to how  
 359:15 they evaluated the animal cancer data, how  
 359:16 they evaluated the epidemiology data, what  
 359:17 data was out there, et cetera.  
 359:18 Q. Sorry, I didn't mean to come  
 359:19 into your personal space.  
 359:20 Do you see Exhibit 1456 that I  
 359:21 put in front of you?

1456.1

359:22 A. Yes.

360:1 - 360:9

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:19)**

M20.52

360:1 Do you see that this is a  
 360:2 document titled "Comments of C. Portier on US  
 360:3 EPA"?

1456.1.2

360:4 A. Yes, I do see it.

360:5 Q. And this is one of those sets  
 360:6 of comments that we're talking about, this  
 360:7 one from October 4, 2016.

360:8 Do you see that?

360:9 A. That -- I do see it.

361:11 - 362:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:37)**

M20.53

361:11 Do you see on the bottom of  
 361:12 page 4 of your comments where it states in  
 361:13 bold language, "EPA should declare glyphosate  
 361:14 a probable human carcinogen"?

1456.4.2

361:15 Do you see that language in  
 361:16 bold there?

361:17 A. "And go on to do a risk  
 361:18 assessment to determine if human exposure is

Page/Line	Source	ID
	361:19 sufficient to warrant concern."	
	361:20 That was my statement. And	
	361:21 there are 32 justified scientific reasons why	clear
	361:22 I believe that to be the case.	
	361:23 Q. Okay. My question was simply:	
	361:24 Did I read that language correctly, in bold?	
	361:25 A. You did read it correctly.	
	362:1 Q. Thank you.	
	362:2 EPA subsequently issued a	
	362:3 subsequent report on glyphosate; is that	
	362:4 true?	
	362:5 A. That is correct.	
366:7 - 366:17	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)</b>	M20.54
	366:7 Q. Do you recall that after you	
	366:8 submitted those public comments, the EPA came	
	366:9 to the judgment that for cancer descriptors,	
	366:10 the available data and weight of evidence	
	366:11 clearly do not support the descriptors	
	366:12 "carcinogenic to humans," "likely to be	
	366:13 carcinogenic to humans" or "inadequate	
	366:14 information to assess carcinogenic	
	366:15 potential"?	
	366:16 Do you recall that?	
	366:17 A. No.	
367:7 - 367:14	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)</b>	M20.55
	367:7 Q. Okay. Did you read the EPA	
	367:8 report dated December 12, 2017?	
	367:9 A. Some of it.	
	367:10 Q. Okay. Let's look at that.	1184.2
	367:11 It's -- sorry, I just mangled your document.	
	367:12 It's Exhibit 1184.	
	367:13 Do you see that?	
	367:14 A. Yes.	
371:8 - 371:11	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</b>	M20.56
	371:8 Q. Does that refresh your	1184.2.1
	371:9 recollection that the EPA's ultimate	
	371:10 conclusion was the strongest support is for	
	371:11 not likely to be carcinogenic to humans?	
371:13 - 371:20	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:14)</b>	M20.57
	371:13 THE WITNESS: I do not	

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371:14 recollect the strongest support for  
371:15 it.

371:16 I do know -- I recollect that  
371:17 in this document their final statement  
371:18 was not likely to be carcinogenic to  
371:19 humans, which I still firmly disagree  
371:20 with.

clear

374:21 - 377:1

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:57)**

M20.58

374:21 Q. Okay. Let's talk about the  
374:22 first branch of evidence you discussed,  
374:23 animal studies. And I want to talk generally  
374:24 about some points and then go into some  
374:25 specific points, if that's okay.

375:1 A. Okay.

375:2 Q. Do you agree with me that  
375:3 animals models play an essential role in all  
375:4 toxicology testing?

375:5 A. All toxicology testing? I  
375:6 would disagree. It plays an essential role  
375:7 in toxicology testing.

375:8 Q. Do you agree with me that they  
375:9 do have some limitations due to differences  
375:10 in genetics, anatomy and physiology between  
375:11 humans and different animal species?

375:12 A. I would not agree with that  
375:13 general statement.

375:14 I would agree with the general  
375:15 statement that says for specific chemicals  
375:16 there would be differences in physiology that  
375:17 would make it -- that you would want to use  
375:18 cautiously in interpreting the animal versus  
375:19 the human: physiology, pharmacology,  
375:20 genetics, et cetera. It's going to be  
375:21 case-specific; it's not going to be a general  
375:22 statement.

375:23 Q. Could we put -- do you have in  
375:24 front of you Exhibit 1657? This is the  
375:25 article that you published with Dr. Resnik in  
376:1 2005.

1657.1

1657.1.6

376:2 Do you have that in front of

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	376:3 you still? And if you need help finding it, 376:4 I can help you find it.	
	376:5 A. Yep, I have it.	
	376:6 Q. If you go to the second page of 376:7 this document -- this is your publication, 376:8 correct?	1657.2
	376:9 A. Yes, it is.	
	376:10 Q. These are your words, correct?	
	376:11 A. Yes, they are.	
	376:12 Q. Let's look at your words in 376:13 this article. I'm in the third column, down 376:14 at the bottom, and I'm just going to read and 376:15 ask you if I've read this correctly. 376:16 "Although animal models play an 376:17 essential role in all toxicology testing" -- 376:18 Did I read that correctly, "all 376:19 toxicology testing"?	1657.2.1
	376:20 A. You did.	
	376:21 Q. -- "they do have some 376:22 limitations due to differences in genetics, 376:23 anatomy and physiology between humans and 376:24 different animal species." 376:25 Did I read that correctly?	
	377:1 A. You did.	clear
377:19 - 378:1	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:19)</b>	M20.59
	377:19 Q. In order to determine whether 377:20 or not glyphosate was causing NHL, we would 377:21 really need to look at the human 377:22 epidemiological evidence, right?	
	377:23 A. In my opinion, it would be 377:24 difficult to conclude that glyphosate is 377:25 causing NHL in humans using only animal 378:1 evidence.	
378:2 - 378:5	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08)</b>	M20.60
	378:2 Q. So that's a yes?	
	378:3 A. I'm not sure of the way you 378:4 stated the question. I'm trying to state an 378:5 answer that I'm comfortable with.	
378:6 - 378:12	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)</b>	M20.61
	378:6 Q. You would need to look at the	

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379:4 - 379:13	<p>378:7 human data, correct?  378:8 A. We would need human data in  378:9 order to make that leap from animals to  378:10 humans for a specific disease.  378:11 Q. Including glyphosate and NHL?  378:12 A. Including NHL and any agent.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:19)</b></p>	M20.62
383:12 - 383:18	<p>379:4 Q. When human data of high quality  379:5 and adequate statistical power are available,  379:6 they are generally preferable over animal  379:7 data and should be given greater weight and  379:8 hazard characterization and dose response  379:9 assessment, although both can be used.  379:10 Is that a correct statement in  379:11 your view?  379:12 A. Yeah, that would be a correct  379:13 statement in my view.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13)</b></p>	M20.63
384:25 - 385:10	<p>383:12 Q. You agree with the statement:  383:13 In the evaluation of human health risks,  383:14 sound human data, whenever available, are  383:15 preferred to animal data in the context of  383:16 risks?  383:17 A. When sound -- sound is the bold  383:18 word there. Yes, I agree.  <b>PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:20)</b></p>	M20.64
385:11 - 386:16	<p>384:25 Do you recall presenting  385:1 Exhibit 882 with five mouse studies?  385:2 A. Okay. So we're talking about  385:3 the cancer studies in mice. Yes, I remember  385:4 presenting that.  385:5 Q. And this is your handwriting on  385:6 it, correct?  385:7 A. Yes, it is.  385:8 Q. And then you also presented  385:9 seven rat studies, right?  385:10 A. That is correct.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:59)</b>  385:11 Q. Am I correct in understanding  385:12 from your testimony that it's not uncommon to</p>	M20.65

385:13 see tumors in rats and mice even when they're

385:14 not exposed to a potential carcinogen?

385:15 A. It depends on the tumor, but it

385:16 is --

385:17 Q. Okay.

385:18 A. There are some tumors which are

385:19 common and some are not. It varies by

385:20 species, by strain, yes.

385:21 Q. But the simple fact of seeing a

385:22 tumor doesn't answer the question for you,

385:23 correct?

385:24 A. That is correct.

385:25 Q. Because you can see tumors of

386:1 specific types without even being exposed to

386:2 a carcinogenic study -- substance in rats and

386:3 mice, correct?

386:4 A. Depends on the tumor, depends

386:5 on the species, depends on the strain. But

386:6 as a general rule, just seeing tumors is not

386:7 enough.

386:8 Q. Okay. Just seeing tumors is

386:9 not enough as a general rule?

386:10 A. As a general rule.

386:11 Q. And in fact, you saw tumors in

386:12 some of the rats and mice in the glyphosate

386:13 studies who were in the control groups that

386:14 were never exposed to glyphosate, correct?

386:15 A. There were tumors in unexposed

386:16 animals, certainly.

386:17 - 387:3

**PORTIER, CHRISTOPHER 2019-02-22\_PIP (00:00:33)**

M20.66

386:17 Q. All right. So let's talk for a

386:18 moment about the rat studies.

386:19 Do you remember preparing this

386:20 chart of the rat studies, Exhibit 883, where

386:21 you circled specific findings?

386:22 A. Yes.

386:23 Q. Am I correct that none of the

386:24 tumors identified here are -- in the rats are

386:25 lymphomas?

387:1 A. In this chart, that is correct.

Page/Line	Source	ID
387:4 - 387:17	<p>387:2 Q. As to the rats, correct?</p> <p>387:3 A. That is correct.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26)</b></p> <p>387:4 Q. And you understand that this is</p> <p>387:5 a case involving non-Hodgkin's lymphoma,</p> <p>387:6 correct?</p> <p>387:7 A. Correct, but there is no</p> <p>387:8 evidence in the literature to suggest that</p> <p>387:9 you must see the same results in laboratory</p> <p>387:10 animals that you see in humans for there to</p> <p>387:11 be a prediction --</p> <p>387:12 Q. My question --</p> <p>387:13 A. -- from the animal to human.</p> <p>387:14 I know what your question was.</p> <p>387:15 Q. My question was simply -- I'm</p> <p>387:16 not -- let me just ask it again to make sure</p> <p>387:17 I understand your answer.</p>	M20.67
387:18 - 387:20	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</b></p> <p>387:18 You understand that this case</p> <p>387:19 involves non-Hodgkin's lymphoma, right?</p> <p>387:20 A. Yes, I do.</p>	M20.68
388:7 - 388:20	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:33)</b></p> <p>388:7 Q. With the exception of growth</p> <p>388:8 and a few nonmalignant tumors, none of the</p> <p>388:9 rat studies showed any effect?</p> <p>388:10 A. No.</p> <p>388:11 Q. Okay.</p> <p>388:12 A. It's the nonmalignant tumors</p> <p>388:13 I'm disagreeing with.</p> <p>388:14 Q. Do you recall having a</p> <p>388:15 publication in a Swiss National Science</p> <p>388:16 Foundation called Horizons?</p> <p>388:17 A. Yes, I did. It's a National</p> <p>388:18 Science Foundation magazine, yes.</p> <p>388:19 Q. And that was in 2016?</p> <p>388:20 A. Yes, it was.</p>	M20.69
390:3 - 390:6	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</b></p> <p>390:3 Do you recognize this as that</p> <p>390:4 article we've been discussing, what I've</p> <p>390:5 marked as Exhibit 1667?</p>	M20.70
		1667.1

Page/Line	Source	ID
391:2 - 391:7	<p>390:6 A. Yes, I do.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:12)</b></p> <p>391:2 Q. Okay. I want to look at a  391:3 specific statement you make in this  391:4 publication. Look with me, if you would, at  391:5 the middle column.  391:6 Do you see that?  391:7 A. Yes, I do.</p>	M20.71
391:21 - 392:16	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</b></p> <p>391:21 Q. At the end of the paragraph --  391:22 I want to be complete in terms of the views  391:23 you express.  391:24 Do you see the end, the last  391:25 sentence of the paragraph?  392:1 A. I do see it, yes.  392:2 Q. You state, "The conclusion is  392:3 that glyphosate causes various tumors in  392:4 laboratory mice."  392:5 Do you see that?  392:6 A. I do see that.  392:7 Q. And that's the view you've  392:8 offered in this case, correct?  392:9 A. That is correct.  392:10 Q. Immediately above that you have  392:11 the sentence I read to you a few moments ago:  392:12 "With the exception of growth in a few  392:13 nonmalignant tumors, none of the rat studies  392:14 showed any effect."  392:15 Did I read that correctly?</p>	M20.72
392:17 - 392:20	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</b></p> <p>392:17 Q. Do you stand behind that  392:18 statement?  392:19 A. No, I do not.  392:20 Q. Okay.</p>	M20.73
393:2 - 393:11	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</b></p> <p>393:2 Six of  393:3 them are in rats, so there are more tumors in  393:4 rats than I knew in 2016. So that statement  393:5 in 2016 is no longer valid in 2019.</p>	M20.74

Page/Line	Source	ID
	393:6 Q. Okay. And that's my only 393:7 question, sir. 393:8 Do you stand behind this 393:9 statement that we've put up on the screen 393:10 from your 2016 publication?	clear
393:12 - 393:14	393:11 A. No. <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</b>	M20.75
	393:12 Q. Let's move on to the mouse 393:13 studies. And I want to ask you some 393:14 questions about mice, please.	
393:21 - 394:4	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</b>	M20.76
	393:21 Is it true that the genetic 393:22 alterations required for neoplastic 393:23 transformation sometimes differ for mice and 393:24 humans? 393:25 A. Yes.	
	394:1 Q. Is it true that there are 394:2 differences between the mouse and human 394:3 immune systems? 394:4 A. Yes.	
394:5 - 395:2	<b>PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:40)</b>	M20.77
	394:5 Q. I want to go to your mouse 394:6 chart and ask you a few questions about it. 394:7 It's Exhibit 882, and it's up on the screen. 394:8 Do you recognize that as the 394:9 chart you spent some time talking about in 394:10 your testimony with the plaintiff lawyer with 394:11 your handwriting on it? 394:12 A. Yes, I do. 394:13 Q. And I want to be clear I 394:14 understand it. One of the tumors that you 394:15 list here in three different places is kidney 394:16 carcinomas or adenomas. 394:17 Do you see those three 394:18 listings? 394:19 A. Yes, I do. 394:20 Q. The plaintiff in this case is 394:21 not claiming that Roundup caused kidney 394:22 cancer. 394:23 You understand that, right?	

394:24 A. I do understand that.

394:25 Q. And do you recognize the term

395:1 "renal" as a medical term for the kidneys?

395:2 A. Yes.

395:3 - 395:25

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:57)**

**M20.78**

395:3 Q. You were not aware of any

395:4 published article that conducts an analysis

395:5 to test whether the development of renal

395:6 tumors in mice is predictive of NHL in

395:7 humans, true?

395:8 A. Do I know of any article?

395:9 I only know of one article that

395:10 looks at prediction from mice to humans by

395:11 tumor site, and I just don't know if it

395:12 covers that or doesn't.

395:13 Q. There's no article you can

395:14 point me to that conducts an analysis to test

395:15 whether the development of renal tumors in

395:16 mice is actually predictive of NHL in humans;

395:17 is that true?

395:18 A. I don't know. I don't know of

395:19 any immediately.

395:20 Q. Let's focus on -- and there --

395:21 let's focus on lymphoma, please.

395:22 A. And to be fair, what I was

395:23 trying to say was I don't know of any article

395:24 for any tumor in mice, predictive of any

395:25 tumor in humans, except for one article.

396:1 - 397:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:56)**

**M20.79**

396:1 Q. Okay. You reported lymphoma in

396:2 five mouse studies, correct?

396:3 A. Four.

396:4 Q. Four. Okay.

396:5 And actually, that is

396:6 important --

396:7 A. I evaluated all five for

396:8 lymphoma, but four were reported as positive

396:9 of some weight, shape or form.

396:10 Q. And I'm glad for that

396:11 precision, and I appreciate that, because I

396:12 want to ask you about that, Doctor.  
 396:13 It's hard to read -- well, it's  
 396:14 not hard to read. These are the four  
 396:15 studies, Atkinson, Sugimoto, Wood and Kumar,  
 396:16 where you reported a difference in malignant  
 396:17 lymphomas.  
 396:18 Do you see that?  
 396:19 A. Yes.  
 396:20 Q. You did not report a difference  
 396:21 in malignant lymphomas for Knezevich,  
 396:22 correct?  
 396:23 A. That is correct. That is  
 396:24 correct.  
 396:25 Q. You did report something, and  
 397:1 this is where I have trouble reading it,  
 397:2 something called -- can you read the deep  
 397:3 purple box for me?  
 397:4 A. Spleen composite  
 397:5 lymphosarcomas.

397:6 - 397:16

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:31)**

M20.80

397:6 Q. Okay. Is that a type of  
 397:7 lymphoma?  
 397:8 A. That is a type of -- well, it's  
 397:9 a very old classification. I had to do a lot  
 397:10 of history lesson to try to understand what  
 397:11 it was.  
 397:12 The best I can find as an  
 397:13 explanation of that is it's an old  
 397:14 classification for some subpart of the  
 397:15 malignant lymphoma classification. But, yes,  
 397:16 it's some sort of lymphatic cancer.

397:17 - 398:1

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:17)**

M20.81

397:17 Q. This study did look at overall  
 397:18 lymphomas, correct?  
 397:19 A. Malignant lymphomas.  
 397:20 Q. Yes.  
 397:21 A. I think it did, yes.  
 397:22 Q. And it found no difference,  
 397:23 correct?  
 397:24 A. I'm not sure. I'd have to look

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397:25 at my documents on the individual study to be  
 398:1 able to answer that specifically.

399:16 - 400:1 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:29)** **M20.82**  
 399:16 Q. Okay. I want to come back to  
 399:17 something we were talking about a moment ago.  
 399:18 It is the case that you can see  
 399:19 lymphomas in mice that are not exposed to  
 399:20 Roundup, correct?  
 399:21 A. Depends on the mouse strain and  
 399:22 depends on the age of the mouse. They're  
 399:23 fairly rare when you get to the 18-month  
 399:24 study in CD-1 mice. It's about 2 percent or  
 399:25 something like that in controls. So you may  
 400:1 or may not see it, but you can see it.

400:2 - 401:3 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:56)** **M20.83**  
 400:2 Q. For example, if you look back  
 400:3 at your table, when it came to malignant  
 400:4 lymphomas in the Knezevich study, you saw as  
 400:5 many in the control group who had no Roundup  
 400:6 as you saw in the high dose group, correct?  
 400:7 A. I'm sorry, I put it away  
 400:8 already.  
 400:9 Q. Page 38, please, Doctor.  
 400:10 A. Yes.  
 400:11 Q. Okay. And that's not  
 400:12 remarkable, is it?  
 400:13 A. No, it's not remarkable.  
 400:14 Q. To see as many in the control  
 400:15 group as you see in the high dose group?  
 400:16 A. Well, if truth were there were  
 400:17 no effect, then, yes, it would not be  
 400:18 remarkable to see the same.  
 400:19 Now, the two mid dose groups  
 400:20 there had substantial different numbers.  
 400:21 Q. Okay. For that reason, some of  
 400:22 the tumors that you testified about were  
 400:23 probably false positives, correct?  
 400:24 A. You've introduced a new topic.  
 400:25 What do you mean by "false positives"?  
 401:1 Q. Is that a term you're familiar

407:2 - 408:14

401:2 with in your work?

401:3 A. Yes, I am.

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:40)**

M20.84

407:2 Q. Okay. Using that term, that

407:3 concept, as you define it, do you agree that

407:4 some of the findings that you discussed are

407:5 probably false positives, using that term as

407:6 you use it?

407:7 A. I'd still would like to define

407:8 the term.

407:9 Q. Why don't you define the term,

407:10 sir.

407:11 A. So false positive is a

407:12 situation where truth is there is no impact

407:13 of the chemical on the risk of getting

407:14 tumors, and you have decided, by whatever

407:15 means you've decided, that the -- there is

407:16 indeed a hazard. That would be a false

407:17 positive decision.

407:18 And with that definition, if

407:19 you were to draw a decision that every one of

407:20 the tumors I've cited here is, in fact, due

407:21 to glyphosate as a cause, then my statement

407:22 would be that some of them are probably false

407:23 positive findings, if you made that

407:24 statement.

407:25 Q. Okay. So you would agree with

408:1 me that some of the findings you talked about

408:2 with the jury, with the plaintiff lawyer, are

408:3 probably false positives, true?

408:4 A. Some of the findings on these

408:5 pages that outline statistical findings are

408:6 false positives. I would agree with that

408:7 statement.

408:8 Q. And to be fair to you, I think

408:9 you think it's a rare chance, but there could

408:10 be zero compound-related effects, true?

408:11 A. I really don't believe that's

408:12 the case. It would be so rare that I just

408:13 don't believe that's the case.

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408:15 - 408:18	<p>408:14 Q. Do you recall giving testimony  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08)</b>  408:15 in April of 2018?</p>	M20.85
409:10 - 409:12	<p>408:16 A. April?  408:17 Q. Of 2018.  408:18 A. A deposition of some sort?  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</b></p>	M20.86
411:2 - 411:15	<p>409:10 Q. One of my colleagues asked you  409:11 questions under oath, correct?  409:12 A. That is correct.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21)</b>  411:2 My question is, do you see on  411:3 page 404, line 6, you're asked the question:  411:4 "And there also could be zero  411:5 compound-related effects, right?"  411:6 Do you see that question?  411:7 A. Yes, I see that question.  411:8 Q. I'm going to read your answer.  411:9 "Answer: That is correct, both  411:10 there are rare chances, but, yes."  411:11 Did I read that correctly?  411:12 A. You read that correctly.  411:13 Q. And were you testifying  411:14 truthfully at the time?  411:15 A. Yes, I was.</p>	M20.87
411:21 - 411:24	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13)</b>  411:21 Q. Am I correct that many of the  411:22 tumors you talked about in the mouse studies  411:23 are seen at very high doses?</p>	M20.88
412:10 - 413:10	<p>411:24 A. No.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:44)</b>  412:10 Q. Do you see that the male mice  412:11 in the Knezevich study in the high dose group  412:12 were exposed to 4,841 milligrams per  412:13 kilograms per day?  412:14 A. Yes, I do see that.  412:15 Q. That's many, many fold higher  412:16 than humans are exposed to, correct?  412:17 A. Probably.  412:18 Q. Many hundreds or thousands of</p>	M20.89

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412:19 fold higher, correct?

412:20 A. I really don't know.

412:21 Q. You've not done that

412:22 calculation?

412:23 A. I've not done that calculation.

412:24 Q. Do you take issue with it being

412:25 hundreds or thousands of times higher than

413:1 what humans are exposed to?

413:2 A. It's much higher, I'll give you

413:3 that.

413:4 Q. Okay. Much higher.

413:5 The females were exposed to an

413:6 even higher level, correct, in the high dose

413:7 group, 5,874?

413:8 A. That is correct.

413:9 Q. If we look at Sugimoto, which

413:10 is in your report on page 42, Table 12?

413:11 - 413:25

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:25)**

M20.90

413:11 A. Yes.

413:12 Q. The males were exposed in the

413:13 high dose group to 4,348 milligrams per

413:14 kilogram per day.

413:15 Do you see that?

413:16 A. I do see that.

413:17 Q. Females, 4,116.

413:18 Do you see that?

413:19 A. I do see that.

413:20 Q. And some of the other ones, the

413:21 high dose groups in the studies were lower

413:22 than that, but they were all many times

413:23 higher than what humans are exposed to,

413:24 correct?

413:25 A. Yes, that is correct.

414:24 - 415:1

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:05)**

M20.91

414:24 Q. Okay. I want to ask you a

414:25 question. Let me just grab a pen and a piece

415:1 of paper.

415:2 - 415:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:07)**

M20.92

415:2 Doctor, do you have in front of

415:3 you -- you probably don't because I have it

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415:4 in my hands -- the edits that you've made to  
415:5 Exhibit 882?  
415:20 - 416:1 **PORTIER, CHRISTOPHER 2019-02-22\_PIP (00:00:14)** **M20.93**  
415:20 I'm going to ask you to work  
415:21 off your notes. I want to ask you some  
415:22 questions about the lymphomas that have been  
415:23 seen in these studies, if I could.  
415:24 A. Okay.  
415:25 Q. Fair? Is that fair, sir?  
416:1 A. Sure.  
416:2 - 419:6 **PORTIER, CHRISTOPHER 2019-02-22\_SS (00:02:38)** **M20.94**  
416:2 Q. As I understand these analyses,  
416:3 broadly speaking -- and here's where I'll ask  
416:4 you to bear with me -- there are two ways of  
416:5 analyzing the data. One way is something  
416:6 called a pairwise comparison; is that  
416:7 correct?  
416:8 A. That is correct.  
416:9 Q. And a pairwise comparison is  
416:10 where you compare two individual groups to  
416:11 see if one has a statistically higher rate;  
416:12 is that correct?  
416:13 A. Correct.  
416:14 Q. The other way is something you  
416:15 report called a trend analysis, correct?  
416:16 A. Correct.  
416:17 Q. And I'm probably going to  
416:18 butcher this horribly, but in lay terms,  
416:19 that's looking across the four groups to see  
416:20 if there's an increasing or other trend  
416:21 across the groups?  
416:22 A. Correct.  
416:23 Q. And you did those -- both of  
416:24 those analyses, correct?  
416:25 A. That is correct.  
417:1 Q. And you did them both in male  
417:2 mice and in female mice, correct, where the  
417:3 data was available?  
417:4 A. I have to be very specific, I'm  
417:5 sorry. I can't say correct to that.

417:6 For cases where I saw a  
417:7 positive tumor in any study on a specific end  
417:8 point, I made sure I looked at that same end  
417:9 point in other studies for the same sex,  
417:10 species group of the animal.  
417:11 Q. Okay.  
417:12 A. I also looked at all tumors  
417:13 greater than three in the total across all  
417:14 the dose groups in any of these studies.  
417:15 So there are some cases where  
417:16 I'm specifically looking at things that have  
417:17 nothing in them that are different than other  
417:18 cases.  
417:19 So I can't say I looked at  
417:20 everything and did that test on everything.  
417:21 It's a very specific rule that I used.  
417:22 Q. By and large, you looked at the  
417:23 male mice, right?  
417:24 A. Correct.  
417:25 Q. You did pairwise tests in the  
418:1 male mice?  
418:2 A. Sometimes.  
418:3 Q. You looked at trends in the  
418:4 male mice, right?  
418:5 A. I always did trends.  
418:6 Q. And one of the ways of looking  
418:7 at trends is something called the  
418:8 Cochran Armitage test, correct?  
418:9 A. That is correct.  
418:10 Q. And you looked at female mice?  
418:11 A. Correct.  
418:12 Q. And you did some trend analysis  
418:13 in female mice?  
418:14 A. That is correct.  
418:15 Q. And you did some pairwise  
418:16 analysis in female mice, correct?  
418:17 A. That is correct.  
418:18 Q. And you recognize when I'm  
418:19 talking about these two tests, pairwise and  
418:20 trend, that by convention for both tests --

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418:21 and I'm focusing on by convention for both  
 418:22 tests -- a statistically significant  
 418:23 comparison is one for which P is less than  
 418:24 .05 that the increased incidence is due to  
 418:25 chance.

419:1 Do you recognize that

419:2 convention I just quoted?

419:3 A. I'm not sure where you're

419:4 quoting it from, but modern use of statistics

419:5 doesn't just draw that, but that convention

419:6 stands.

419:7 - 419:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:12)**

M20.95

419:7 Q. Okay.

419:8 A. But most statisticians and

419:9 others now are starting to look at this in a

419:10 much more flexible fashion.

419:11 There was a nice article from

419:12 the American Statistical Association on this

419:13 issue.

419:16 - 419:20

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:08)**

M20.96

419:16 My question was simply, you

419:17 recognize that convention, right?

419:18 A. I recognize that some people

419:19 use that convention to a great degree, more

419:20 than they probably should.

423:11 - 425:12

**PORTIER, CHRISTOPHER 2019-02-22\_PIP (00:01:55)**

M20.97

423:11 Q. Okay. Using that .05 standard,

423:12 I just want to ask you about the findings of

423:13 those five mouse studies. I've put lymphoma

423:14 at the top because that's what I'm going to

423:15 focus on; .05, that standard that we just

423:16 read; and the two types tests for male and

423:17 for -- I'm sorry, male for pairwise and for

423:18 trend.

423:19 And I'm just going to ask you,

423:20 yes or no: Was there, under this standard, a

423:21 statistically significant finding at that

423:22 level for Knezevich, was there, at the .05

423:23 level?

423:24 A. No.

423:25 Q. What about for trend at the .05  
424:1 level?  
424:2 A. No.  
424:3 Q. Atkinson, the second study, was  
424:4 there statistical significance pairwise at  
424:5 the .05 level?  
424:6 A. No.  
424:7 Q. Trend?  
424:8 A. No.  
424:9 Q. Sugimoto, the third study you  
424:10 referenced, was there statistical  
424:11 significance pairwise at the .05 level?  
424:12 A. No.  
424:13 Q. I think we talked over each  
424:14 other. I didn't hear what you said, sir.  
424:15 A. There was no pairwise  
424:16 statistical significance.  
424:17 Q. And there was --  
424:18 A. Less than .05 P value for the  
424:19 pairwise comparisons in that study.  
424:20 Q. There was for trend, correct?  
424:21 A. There was for trend.  
424:22 Q. For Wood, the fourth study you  
424:23 talked about, there was on both tests,  
424:24 correct?  
424:25 A. That is correct.  
425:1 Q. And for the final study you  
425:2 talked about, Kumar --  
425:3 A. Yes.  
425:4 Q. -- was there statistical  
425:5 significance for pairwise?  
425:6 A. Yes.  
425:7 Q. Was there statistical  
425:8 significance for trend?  
425:9 A. No.  
425:10 It's yes for pairwise.  
425:11 Q. At the .05 level?  
425:12 A. Yes.

425:13 - 425:14

**PORTIER, CHRISTOPHER 2019-02-22\_PIP (00:00:10)**

M20.98

425:13 Q. Then why do you have 1 plus on

Page/Line	Source	ID
425:15 - 425:24	<p>425:14 your chart and no pairwise notation?  <b>PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:28)</b>  425:15 A. Kumar was .05. I'm sorry, yes,  425:16 it was statistically significant at .05.  425:17 The chart only shows the number  425:18 of pluses for the trend test. I made that  425:19 clear yesterday.  425:20 Q. Okay.  425:21 A. And I fully disagree with this  425:22 characterization of yes/no for these  425:23 findings, but you've created a table that is  425:24 indeed accurate.</p>	M20.99
426:15 - 427:23	<p><b>PORTIER, CHRISTOPHER 2019-02-22_PIP (00:01:11)</b>  426:15 Q. Did you do these same analyses  426:16 for female mice?  426:17 A. For malignant lymphomas?  426:18 Q. Yes.  426:19 A. No.  426:20 Q. Okay. You didn't look for  426:21 malignant lymphomas at whether there was  426:22 statistical significance in these studies?  426:23 A. Sometimes I didn't have the  426:24 data, and other times I -- I had a rule for  426:25 what I was looking at.  427:1 Q. Okay. So let me just ask you  427:2 the question.  427:3 When it comes to -- you do have  427:4 a notation on your chart for females; it's  427:5 just not circled, correct?  427:6 A. That's correct.  427:7 Q. Okay. When it comes to  427:8 females, can you point me to any findings as  427:9 to females in these studies that are  427:10 statistically significant on either the  427:11 pairwise or the trend?  427:12 A. In these studies?  427:13 Q. Yes.  427:14 A. No. If they were statistically  427:15 significant, they would be shown in the  427:16 table.</p>	M20.100

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427:17 Q. Okay. So there are no  
427:18 statistically significant findings for  
427:19 females in these studies?

427:20 A. In these studies for malignant  
427:21 lymphoma --

427:22 Q. Yes.

427:23 A. -- that is correct.

429:9 - 430:8

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:40)**

M20.101

429:9 Q. Do you recall talking yesterday  
429:10 about an author called De Roos? De Roos?

429:11 A. De Roos, yes.

429:12 Q. Yes. That's an author you said  
429:13 signed on to your letter.

429:14 Do you remember that?

429:15 A. That's correct.

429:16 Q. Do you recall Dr. De Roos  
429:17 actually publishing a study on epidemiology,  
429:18 human epidemiology?

429:19 A. Several, yes.

429:20 Q. And I'm going to focus on the  
429:21 2005 one.

429:22 You recall the 2005 study,  
429:23 correct?

429:24 A. Yeah, I do recall that she had  
429:25 a 2005 study.

430:1 Q. And that's a study that you  
430:2 have looked to. You've cited it in your  
430:3 report and you talked about it yesterday,  
430:4 correct?

430:5 A. That is correct.

430:6 Q. Let's take a look at that  
430:7 study, please. It's 528 in your binder, if  
430:8 you need to look at it.

528.1

430:9 - 430:11

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:06)**

M20.102

430:9 Do you recognize what I've put  
430:10 up on the screen as a copy of that study,  
430:11 Doctor?

528.1.1

430:12 - 430:18

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:13)**

M20.103

430:12 It's probably hard to read.  
430:13 It's the one that's in your binder as 528.

Page/Line	Source	ID
	430:14 A. Yes, I do recognize it.	
	430:15 Q. And we see the first author is	
	430:16 De Roos.	
	430:17 Do you see that?	
	430:18 A. Yes, I do.	
431:11 - 431:23	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)</b>	M20.104
	431:11 And if you look, do you see	
	431:12 that there's an abstract right at the top?	
	431:13 A. Yes, I do.	528.1.2
	431:14 Q. Do you see that they write in	
	431:15 their abstract, "Although there has been	
	431:16 little consistent evidence of genotoxicity or	
	431:17 carcinogenicity from in vitro and animal	
	431:18 studies"?	
	431:19 Do you see that?	
	431:20 A. I see that what's she writes.	
	431:21 Q. And I read that correctly,	clear
	431:22 right?	
	431:23 A. You read it correctly.	
445:9 - 445:18	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21)</b>	M20.105
	445:9 Are you familiar with the World	
	445:10 Health Organization Task Group on	
	445:11 Environmental Health Criteria on Principles	
	445:12 for Modeling Dose Response for the Risk	
	445:13 Assessment of Chemicals?	
	445:14 A. It's a very long name.	
	445:15 Q. Yeah, it is a very long name.	
	445:16 A. It sounds like something I	
	445:17 might have been involved in years ago. I	
	445:18 have no idea.	
446:2 - 446:15	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:43)</b>	M20.106
	446:2 Q. I'll pass you Trial	
	446:3 Exhibit 1278, please.	1278.1
	446:4 Do you see that this is a	1278.1.1
	446:5 document from the World Health Organization	
	446:6 International Programme on Chemical Safety?	
	446:7 A. Yes, this is an environmental	
	446:8 health criteria document.	
	446:9 Q. Yes.	
	446:10 And if you look at the inside	1278.2.2

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446:11 cover of that document, it states first draft  
446:12 prepared by the WHO task group that I  
446:13 mentioned.

446:14 Do you see that?

446:15 A. Yes.

447:21 - 448:6

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:16)**

M20.107

447:21 Do you see up on the screen

1278\_16.1

447:22 where it says "Task Group Members"?

447:23 A. Page 16, yes.

447:24 Q. Yes.

447:25 And if you look at the very

448:1 next page, under that listing do you see your  
448:2 name?

448:3 A. Yes, I do.

448:4 Q. Okay. And what I wanted to ask

448:5 you about this document and the quote I read

448:6 you earlier is on page 10 of this document.

448:9 - 448:17

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:13)**

M20.108

448:9 Q. And I've called out the bottom

1278.11.2

448:10 paragraph, and I just want to ask if I've

448:11 read this correctly from this working group

448:12 document.

448:13 "In the evaluation of human

448:14 health risks, sound human data, whenever

448:15 available, are preferred to animal data."

448:16 Did I read that correctly?

448:17 A. You read that correctly.

clear

455:16 - 456:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:24)**

M20.109

455:16 Q. I want to move on and talk to

455:17 you for a little -- talk with you for a

455:18 little bit about genotoxicity.

455:19 Do you recall testifying about

455:20 that? I think Mr. Wisner called it the

455:21 second leg of his stool.

455:22 Do you remember that?

455:23 A. I think I recall testifying

455:24 about that.

455:25 Q. And I think you mentioned two

456:1 potential mechanisms, if I understood you

456:2 correctly: One was genotoxicity; one was

Page/Line	Source	ID
457:20 - 458:6	<p>456:3 oxidative stress.  456:4 Is that accurate?  456:5 A. That is accurate.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</b></p>	M20.110
	<p>457:20 Do you see under their  457:21 conclusion EFSA writes to you, "Considering a  457:22 weight of evidence approach, taking into  457:23 account the quality and reliability of all  457:24 available data, it is concluded that  457:25 glyphosate is unlikely to be genotoxic in  458:1 vivo"?</p>	1639.15.3
458:7 - 459:8	<p>458:2 Did I read that correctly?  458:3 A. You read it correctly.  458:4 Q. And this is them writing back  458:5 to you; is that correct?  458:6 A. That is correct.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:53)</b></p>	clear M20.111
	<p>458:7 Q. You talked about a few studies  458:8 in this area, and I want to just look at a  458:9 couple of the studies, if we could.  458:10 One of the studies you  458:11 mentioned is by a lead author Bolognesi.  458:12 Do you remember that?  458:13 A. There were several, which -- it  458:14 depends which one.  458:15 Q. Okay. One of them was a study  458:16 that involved aerial spraying, correct?  458:17 A. I do remember that one.  458:18 Q. And if I recall your testimony  458:19 correctly, you compared that to two studies  458:20 by authors called Paz-y-Mino?  458:21 A. That's correct.  458:22 Q. And you said that the Bolognesi  458:23 study is the stronger study than either  458:24 Paz-y-Mino study, correct?  458:25 A. That's correct.  459:1 Q. The Bolognesi study showed that  459:2 genotoxic risk potentially associated with  459:3 glyphosate -- with exposure to glyphosate is  459:4 low, correct?</p>	

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459:5 A. I'd have to see the document,

459:6 but say it again so I can read it --

459:7 Q. Sure.

459:8 A. -- I can understand it.

459:9 - 459:20

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:26)**

M20.112

459:9 Q. "Genotoxic risk potentially

459:10 associated with glyphosate in the areas where

459:11 the herbicide is applied for coca and poppy

459:12 eradication is low."

459:13 A. I have to see it in the context of the statement

459:14 they're giving it in. I believe what they're

459:15 saying is that the magnitude of the effect

459:16 they saw was low --

459:17 Q. Okay. Let's take a look --

459:18 A. -- as compared to the -- the

459:19 strength of the evidence that there was an

459:20 effect.

459:21 - 460:7

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:26)**

M20.113

459:21 Q. Okay. Let's look at their

459:22 language and let their words speak for

459:23 themselves.

459:24 Do you mind if we go to

459:25 exhibit -- it's actually not in your binder.

460:1 I thought it was in your binder. I'll give

460:2 you a copy. It's 1066, please.

1066.1

460:3 Do you have that in front of

460:4 you, sir?

460:5 Do you recognize this as the

460:6 Bolognesi study we've been referring to?

460:7 A. Yes.

460:8 - 460:21

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:33)**

M20.114

460:8 Q. We've got it up on the screen.

460:9 Let's call out, just first, the authors.

1066.1.3

460:10 There we see the name of the Bolognesi

460:11 author, the leader author.

460:12 Do you see that?

460:13 A. Yes. That is the article.

460:14 Q. And if we look at the author

460:15 affiliations, their affiliations include the

460:16 National Cancer Research Institute in Genoa.

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460:17 Do you see that?

460:18 A. Yes.

460:19 Q. And various universities,

460:20 correct?

460:21 A. Correct.

461:3 - 462:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:11)**

M20.115

461:3 Q. And then at the end of their

461:4 abstract, do you see this language that I was

1066.1.4

461:5 just reading you?

461:6 "Evidence indicates that the

461:7 genotoxic risk potentially associated with

461:8 exposure to glyphosate in the areas where the

461:9 herbicide is applied for coca and poppy

461:10 eradication is low."

461:11 Did I read that correctly?

461:12 A. You read that correctly.

461:13 Q. And just so the jury

461:14 understands what we're talking about, this

clear

461:15 was a study that looked at aerial spraying

461:16 that was being done in South America to try

461:17 to eradicate crops relevant to the illegal

461:18 drug industry, correct?

461:19 A. Correct.

461:20 Q. And what they are saying is in

461:21 the context of their study, the genotoxic

461:22 risk potentially associated with that form of

461:23 exposure is low, correct?

461:24 A. That's what it says.

461:25 Interpretation that they put on

462:1 that is based upon the magnitude of the

462:2 effect, not the presence or absence of the

462:3 effect. So the low refers there to the

462:4 magnitude of the effect.

462:5 Q. Sir, have you ever talked with

462:6 the authors about this article?

462:7 A. It's in the article.

462:8 Q. Have you talked with the

462:9 authors about this article?

462:10 A. No, I have not.

462:11 Q. Okay. Let's look at what they

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	462:12 say later in the article.	1066.9
462:14 - 464:25	462:13 Could you flip to page 994 of <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:02:25)</b>	M20.116
	462:14 the document? And I think this might be the	
	462:15 point you were going to.	
	462:16 "Overall, these results suggest	1066.9.2
	462:17 that genotoxic damage associated with	
	462:18 glyphosate spraying, as evidenced by the MN	
	462:19 test, is small and appears to be transient."	
	462:20 Did I read that correctly.	
	462:21 A. You read that correctly.	
	462:22 Q. And the MN test, that's a test	
	462:23 of that metric you were talking about on	
	462:24 direct examination, micronuclei, correct?	
	462:25 A. Yes, the one they used here.	
	463:1 Q. Right.	
	463:2 And then do you recall that	
	463:3 this article, at least according to the terms	
	463:4 of these authors, purported to do a Bradford	
	463:5 Hill analysis of their data?	
	463:6 A. I don't recall that.	
	463:7 Q. Let's look at that. Could we	
	463:8 go to the next page, please, Doctor?	1066.10
	463:9 And I'll direct you, if I may,	
	463:10 to the right-hand column on page 995.	
	463:11 A. Okay.	
	463:12 Q. And if we look at the second	1066.10.3
	463:13 sentence it says, "Based on the	
	463:14 application" -- I'm sorry. It says, "Based	
	463:15 on the applicable Bradford Hill guidelines,	
	463:16 Hill 1965."	
	463:17 Do you see that?	
	463:18 A. Yes, I see it.	
	463:19 Q. And those are the same	
	463:20 guidelines you talked about on direct	
	463:21 examination, right down to the year, correct?	
	463:22 A. Yes, correct.	
	463:23 Q. And then they say, "Based on	1066.10.4
	463:24 the applicable Bradford Hill guidelines, it	
	463:25 is not possible to assign causality to the	

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464:1 increases in frequency of BNMN observed in  
464:2 our study."

464:3 Did I read that correctly?

464:4 A. You read that correctly.

464:5 Q. And BNMN is a measure of

464:6 micronuclei damage, correct?

464:7 A. It's a specific form of

464:8 micronuclei damage. Binucleated.

464:9 Q. Thank you, Doctor.

clear

464:10 I just referenced in our

464:11 discussion one of the Paz-y-Mino studies.

464:12 Do you recall that?

464:13 A. Yes.

464:14 Q. They did two studies, one back

464:15 in 2007 and then one -- a second one in 2011.

464:16 Do you remember that?

464:17 A. Yes, I do.

464:18 Q. And you reviewed and discussed

464:19 both of those on your direct; is that right?

464:20 A. They were certainly mentioned.

464:21 I discussed them a little bit, yes. I

464:22 remember that.

464:23 Q. Okay. Let me pass you the

464:24 second one, the one that was conducted in

464:25 2011, which is Exhibit 1437.

1437.1.3

465:1 - 465:2

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:07)**

M20.117

465:1 Do you recognize Exhibit 1437

465:2 as the second Paz-y-Mino study from 2011?

465:3 - 465:19

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:40)**

M20.118

465:3 A. Yes, sir.

465:4 Q. And if we look at the authors,

465:5 we see the first author is Paz-y-Mino,

465:6 correct?

465:7 A. That is correct.

465:8 Q. And this study is also looking

465:9 at aerial spraying, correct?

465:10 A. Yes.

465:11 Q. Let's look at their

465:12 conclusions. If we look at the right-hand

465:13 column -- the left-hand column, I apologize,

1437.1.2

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465:14 in the abstract, do you see where they state,  
 465:15 "In conclusion, the study population did not  
 465:16 present significant chromosomal and DNA  
 465:17 alterations"?

465:18 Did I read that correctly?

465:19 A. You read that correctly.

466:6 - 467:3

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:01)**

M20.119

466:6 Q. Page 50 is part of the

1437.6

466:7 discussion in the article, correct?

466:8 A. Yes.

466:9 Q. I want to show you two things.

466:10 First, they say at the bottom of the

1437.6.4

466:11 left-hand column, "Several research studies

466:12 related to glyphosate exposure have been

466:13 conducted in Colombia by Bolognesi, et al.,

466:14 Sanin and Solomon."

466:15 Do you see that?

466:16 A. Yes.

466:17 Q. And Bolognesi is what we were

466:18 just discussing, correct?

466:19 A. Yes, that's the same study.

466:20 Q. And have you read all three of

466:21 these studies that they reference?

466:22 A. I have not.

466:23 Q. Okay. They go on to say,

466:24 regarding these studies, "Which state that

1437.6.5

466:25 the studied populations have low genotoxic

467:1 risk associated with glyphosate."

467:2 Did I read that correctly?

467:3 A. Yes, you did.

467:8 - 467:17

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:19)**

M20.120

467:8 Do you see where they say,

1437.6.6

467:9 "Regarding our study, we obtained results

467:10 showing no chromosomal alteration in the

467:11 analyzed individuals"?

467:12 Did I read that correctly?

467:13 A. You read that correctly.

467:14 Q. And this is a study that you

467:15 relied on -- or that you discussed in your

467:16 report, correct?

468:2 - 469:22

467:17 A. Correct.

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:37)**

clear

M20.121

468:2 Q. Do you know if you've read,  
468:3 sir, all of the genotoxicity studies that

468:4 exist on glyphosate formulations?

468:5 A. I can't, of course, answer that

468:6 question. There's no way you can -- you

468:7 could answer a question that says "have you

468:8 read everything." I -- I've read everything

468:9 I've read --

468:10 Q. Okay. Fair enough.

468:11 A. -- and everything I've cited.

468:12 Q. Here's where I'm going with

468:13 that, sir.

468:14 If I go back to some of the

468:15 exhibits that you covered in your direct

468:16 examination with the plaintiff lawyer, for

468:17 example, Exhibit 876, do you see that?

468:18 A. Yes, I see it.

468:19 Q. Do you know if this represents

468:20 the full universe of in vitro human

468:21 genotoxicity data?

468:22 And actually, just in fairness,

468:23 I'm sorry, I don't want to -- there were two

468:24 of these that you did. The other one was

468:25 875.

469:1 Do you see that?

469:2 A. That's correct.

469:3 Q. Okay. And so let me ask the

469:4 question as to both of those.

469:5 Do you know between the two of

469:6 those whether those represent the full

469:7 universe of human in vitro genotoxicity data?

469:8 A. Those are the ones I was able

469:9 to find.

469:10 Q. Do you know if there are others

469:11 out there?

469:12 A. If I knew there were others out

469:13 there, they'd be in the list.

469:14 Q. Okay. You made reference, if I

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469:15 heard you right, and it's reflected on the  
469:16 charts, to -- you've got 1980 to 2014 on the  
469:17 first chart. You've got 2017 to 2018 on the  
469:18 second chart.

469:19 Did you look for things from  
469:20 2015 and 2016 and not find them, or do they  
469:21 not exist; do you know?

469:22 A. I don't -- I don't know.

469:23 - 470:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:30)**

M20.122

469:23 Q. Okay. If I were to ask you --  
469:24 let me try it this way.

469:25 There's a study by lead author  
470:1 Dutta, D-u-t-t-a, from 2017. I don't see it  
470:2 on your list.

470:3 Do you know one way or the  
470:4 other whether you've read it or not?

470:5 A. Was it in human cell lines?

470:6 Q. Do you know if you've read that  
470:7 study?

470:8 A. I'd have to look at my full  
470:9 list. This is the list of human cell lines.

470:10 Q. There was a study by --

470:11 A. I seem to recall a study by  
470:12 Dutta, but I don't think it was human cell  
470:13 lines.

471:2 - 471:23

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:42)**

M20.123

471:2 Q. Okay. In terms of this list,  
471:3 do you know if this list, or these two lists,  
471:4 the two that we've been looking at here, do  
471:5 you know if that represents 100 percent of  
471:6 the available human in vitro genotoxicity  
471:7 data? 50 percent? Some other number?

471:8 A. The only answer I can give you  
471:9 is that represents all of the human in vitro  
471:10 evidence that I was able to find.

471:11 Q. Okay. You were -- if I  
471:12 understand the documents you reviewed, you  
471:13 reviewed a deposition from a Monsanto  
471:14 scientist a couple years ago named Donna  
471:15 Farmer.

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471:16 Do you recall that?

471:17 A. That I reviewed a deposition by

471:18 her? I don't recall.

471:19 Q. Okay. You certainly haven't

471:20 reviewed a more recent deposition by her,

471:21 have you?

471:22 A. Again, I don't recall reviewing

471:23 any depositions by her.

472:7 - 472:11

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:10)**

M20.124

472:7 Q. Okay. You've not been shown a

472:8 list of documents that she prepared where she

472:9 listed the genotoxicity studies she's aware

472:10 of, have you?

472:11 A. I have not.

472:22 - 473:14

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:39)**

M20.125

472:22 It's not the purpose of

472:23 genotoxicity assays to establish that

472:24 glyphosate causes NHL?

472:25 A. Genotoxicity assays are not

473:1 used to establish that glyphosate causes NHL

473:2 in people.

473:3 Q. Thank you.

473:4 Just having a genotoxic

473:5 finding, in your view, does not lead to

473:6 cancer, correct?

473:7 A. Correct.

473:8 Q. And when we talk about

473:9 genotoxicity or damage to the DNA, it's fair

473:10 to say that you consistently have damage to

473:11 your DNA?

473:12 A. That is correct.

473:13 Q. A lot?

473:14 A. Quite a bit.

473:23 - 474:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:36)**

M20.126

473:23 Q. Okay. If I understand your

473:24 testimony, genotoxicity is what occurs when

473:25 there's damage to cells, correct?

474:1 A. And/or mutations.

474:2 Q. Okay.

474:3 A. It encompasses both.

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474:4 Q. Okay. Well, you do have -- in  
474:5 terms of this mechanism of causation, you  
474:6 have to have mutations to proceed to cancer,  
474:7 correct?

474:8 A. In this multistage model of  
474:9 carcinogenesis, that is correct.

474:10 Q. And just because a chemical can  
474:11 cause damage does not mean that it will cause  
474:12 mutations, correct?

474:13 A. That is correct.

474:25 - 475:8

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:34)**

M20.127

474:25 Q. So is it -- would you conclude  
475:1 that it's correct to say that the scientific  
475:2 evidence is insufficient to classify  
475:3 glyphosate as a mutagen or capable of causing  
475:4 mutations?

475:5 A. I would say that's incorrect.

475:6 Q. Do you recall giving testimony  
475:7 back in March 2018?

475:8 A. Yes.

475:9 - 476:11

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:07)**

M20.128

475:9 Q. Turn with me, if you would, to  
475:10 page 692. And I'm going to specifically  
475:11 direct your attention to line 16, and tell me  
475:12 when you're ready for me to read.

475:13 A. Okay. I'm ready.

475:14 Q. "Question: And you also agree  
475:15 that the scientific evidence is insufficient  
475:16 to classify glyphosate -- glyphosate as a  
475:17 mutagen or capable of causing mutations,  
475:18 correct?"

475:19 Did I read that correctly?

475:20 A. Correct.

475:21 Q. And then your answer: "Let me  
475:22 think about that one for a minute. I have to  
475:23 run through all of the assays that I looked  
475:24 at in my head.

475:25 "I would have to conclude that  
476:1 that is correct. It's genotoxicity; it's not  
476:2 mutations. I will point out that for most

476:3 evaluations of the genetic toxicity of  
476:4 chemicals, they don't sequence DNA and look  
476:5 for mutations."

476:6 Did I read that correctly, sir?

476:7 A. You did read it correctly.

476:8 Q. And were you being truthful in  
476:9 those answers?

476:10 A. The answer is incorrect as the  
476:11 question is stated.

477:21 - 480:16

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:02:16)**

M20.129

477:21 Q. Do you see where it says,

477:22 "okay," question on the next page?

477:23 A. Yes.

477:24 Q. And then do you see, "Answer:

477:25 So it would be rather unusual to have data

478:1 that would allow me to say, yep, it's a

478:2 mutation"?

478:3 Do you see that?

478:4 A. Correct.

478:5 Q. And then the testimony

478:6 continues, correct?

478:7 A. Correct.

478:8 Q. So is the testimony that I

478:9 read, including your statement: "I would

478:10 have to conclude that that is correct, it's

478:11 genotoxicity and not mutations," were you

478:12 being truthful when you gave that testimony;

478:13 yes or no?

478:14 A. It's truthful up to the point

478:15 where the question ends with the word

478:16 "mutagen." It is not truthful for the

478:17 "capable of causing mutations." Then that

478:18 statement would not be correct.

478:19 Q. Okay.

478:20 A. So I -- I misanswered because I

478:21 didn't take the "are" into account.

478:22 Q. The rest of the answer is

478:23 correct as to mutagen?

478:24 A. As to mutagen, per se. But as

478:25 to capable of causing mutations, that

479:1 answer's not correct.  
479:2 Q. What's a mutagen?  
479:3 A. What's a mutagen?  
479:4 Q. Uh-huh.  
479:5 A. That is something that is known  
479:6 to cause mutations.  
479:7 Q. And that doesn't apply to  
479:8 glyphosate?  
479:9 A. I don't have enough evidence  
479:10 that I would stand up and say absolutely it  
479:11 causes mutations.  
479:12 Q. In fact, the mutagenicity tests  
479:13 that exist for glyphosate are overwhelmingly  
479:14 negative, right?  
479:15 A. There are only two mutagenicity  
479:16 tests I know of that were used for  
479:17 glyphosate. One was a reverse mutation in a  
479:18 very -- in several strains of salmonella, and  
479:19 the other is a -- I'd have to look at my  
479:20 records what the other one was.  
479:21 Q. Are they overwhelmingly  
479:22 negative?  
479:23 A. The salmonella tests and  
479:24 bacteria were overwhelmingly negative.  
479:25 Q. Thank you.  
480:1 Let's switch quickly to  
480:2 oxidative stress, the second mechanism that  
480:3 you discussed.  
480:4 Is it fair to say that the fact  
480:5 that a chemical causes oxidative stress does  
480:6 not mean that it causes cancer? Is that a  
480:7 correct statement?  
480:8 A. That is a correct statement.  
480:9 Q. Oxidative stress is happening  
480:10 all the time in our bodies, correct?  
480:11 A. That is a correct statement,  
480:12 yes.  
480:13 Q. Exercise causes oxidative  
480:14 stress?  
480:15 A. Yes, in certain parts of the

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480:23 - 480:25	<p>480:16 body.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04)</b></p> <p>480:23 Having a cold causes oxidative 480:24 stress?</p>	M20.130
481:1 - 481:6	<p>480:25 A. That, I don't know. Probably.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:12)</b></p> <p>481:1 Q. I've passed you deposition 481:2 testimony from September 2017. 481:3 Do you see that? 481:4 A. Yes.</p>	M20.131
481:7 - 481:21	<p>481:5 Q. And if you would, look with me 481:6 at page 353, please. And tell me when you're</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)</b></p> <p>481:7 ready. I'm going to page -- line 10, sir. 481:8 A. Okay. 481:9 Q. Do you see on line 10 you were 481:10 asked: "And having a cold would cause 481:11 oxidative stress, correct?" 481:12 And you answer: "That's 481:13 correct." 481:14 Do you see that? 481:15 A. Yes. 481:16 Q. Did I read that correctly? 481:17 A. You read it correctly. 481:18 Q. Were you being truthful in that 481:19 testimony? 481:20 A. To be honest, I don't actually 481:21 know.</p>	M20.132
482:9 - 483:16	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:09)</b></p> <p>482:9 So back to where I was. Do you 482:10 agree with me, Doctor, that no oxidative 482:11 stress study on glyphosate that you reviewed 482:12 can establish in and of itself that 482:13 glyphosate causes non-Hodgkin's lymphoma? 482:14 A. Yes. 482:15 Q. Do you recall reviewing a 2018 482:16 analysis by NTP, where you used to work, 482:17 regarding the oxidative stress of glyphosate? 482:18 A. I read the study. I do 482:19 remember reading the study.</p>	M20.133

482:20 Or was it an abstract? I don't  
482:21 think there's a published study from them. I  
482:22 think it's an abstract or something along  
482:23 those lines.

482:24 Q. Do you recall that the NTP  
482:25 scientists who did this study, what they  
483:1 concluded was that the data suggests that  
483:2 glyphosate does not induce oxidative stress  
483:3 on its own?

483:4 A. If I could see the paper, it  
483:5 would be useful.

483:6 Q. I actually have your testimony  
483:7 on it. If you like, I can show your  
483:8 testimony on it. I don't have --

483:9 A. You don't have the paper?

483:10 Q. I don't think I have the paper.  
483:11 Not handy.

483:12 A. Or the abstract or whatever it  
483:13 was.

483:14 In the species that they  
483:15 tested, under the conditions they tested, I  
483:16 think they found it to be negative.

484:11 - 485:14

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:59)**

M20.134

484:11 Q. Let's look at the third leg of  
484:12 Mr. Wisner's stool: epidemiology.  
484:13 You did look at the human  
484:14 epidemiology in this case, correct?

484:15 A. Yes, I did.

484:16 Q. And so the jury is clear, human  
484:17 epidemiology data involves studies of people  
484:18 in the real world and their exposure to, in  
484:19 this case, glyphosate?

484:20 A. And many other things, yes.

484:21 Q. And there's been some talk  
484:22 about the formulated product Roundup versus  
484:23 glyphosate.

484:24 The epidemiological studies  
484:25 involved the formulated product, true?

485:1 A. That is correct.

485:2 Q. So I just want to walk through

485:3 quickly, as quickly as possible, the studies  
485:4 that you put up on the screen, or the -- I  
485:5 think they're called forest plots that you  
485:6 put up on the screen.

485:7 Do you recall showing the jury  
485:8 the forest plots?

485:9 A. A couple of them, yes.

485:10 Q. Let's look at them. Let's

485:11 start with Exhibit 878, which is in your --

878.1

485:12 which is in your binder, if you want to look  
485:13 at it directly.

485:14 A. I can see it here.

486:5 - 490:25

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:03:28)**

M20.135

486:5 Q. Okay. So below the line that

486:6 we have here, those are the meta-analyses,  
486:7 correct?

486:8 A. Those are the main results from  
486:9 the meta-analyses that were done, that is  
486:10 correct.

486:11 Q. And they combine the data from  
486:12 the studies above the line, correct?

486:13 A. That's correct. Selectively.

486:14 Q. Right. They pick out one  
486:15 finding and plug it in with other findings  
486:16 from the other studies, correct?

486:17 A. That is correct.

486:18 Q. And the studies above the line  
486:19 are the individual studies that you have  
486:20 reviewed and analyzed, and in some cases  
486:21 different analyses conducted in those  
486:22 studies, correct?

486:23 A. That is correct.

486:24 Q. So let's just walk through  
486:25 those very, very quickly.

487:1 The first one is a study called  
487:2 Andreotti 2018.

878.1.9

487:3 Do you see that?

487:4 A. I see that.

487:5 Q. That was not statistically  
487:6 significant, correct?

487:7 A. That particular finding, that  
487:8 is correct.

487:9 Q. The finding you report on this  
487:10 chart?

487:11 A. At your 5 percent level where  
487:12 you want to define yes and no, it's not.

487:13 Q. Okay. The next study is the  
487:14 De Roos study.

878.1.10

487:15 Do you see that?

487:16 A. Yes, I do.

487:17 Q. Those are your -- De Roos is  
487:18 the one you said joined your letter, correct?

487:19 A. That is correct.

487:20 Q. And De Roos reports two  
487:21 findings.

487:22 Do you see that?

487:23 A. That is correct, yes.

487:24 Q. The first De Roos finding is  
487:25 not statistically significant, correct?

878.1.11

488:1 A. That is correct.

488:2 Q. And then the second finding  
488:3 that they have is their highest exposure  
488:4 group, correct?

488:5 A. That's correct.

488:6 Q. And highest exposure means just  
488:7 what it sounds like, exposed to the most  
488:8 glyphosate?

488:9 A. Well, I mean, it has a very  
488:10 specific meaning --

488:11 Q. Yes, sir.

488:12 A. -- that they put into the  
488:13 document of how they calculate it, for which  
488:14 I have some concerns. But, yes, it means by  
488:15 their definition of exposure the highest  
488:16 exposure.

488:17 Q. Correct. Okay.

488:18 And that is not statistically  
488:19 significant, correct?

488:20 A. That is correct.

488:21 Q. In fact, that is below 1,

488:22 correct?

488:23 A. That is correct.

488:24 Q. It's on the side of 1

488:25 indicating that there's a reduced risk with

489:1 highest exposure of glyphosate, although it's

489:2 not statistically significant, correct?

878.1.12

489:3 A. That is correct.

489:4 Q. The next study is the earlier

489:5 De Roos study from 2003.

489:6 Do you see that?

489:7 A. Yes, I do.

489:8 Q. And here, too, there are two

489:9 analyses reported.

489:10 Do you see that?

489:11 A. Yes, I do.

489:12 Q. One is statistically

489:13 significant; one is not, correct?

489:14 A. That's correct.

489:15 Q. We then go to the next study,

489:16 the Eriksson study. This has, as I read it,

489:17 three analyses reported, correct?

489:18 A. That is correct.

489:19 Q. There's a general analysis.

489:20 Do you see that?

489:21 A. The general meaning -- the

489:22 first analysis, which is their primary

489:23 analysis uncorrected for other pesticides.

489:24 Q. Right.

489:25 And that is statistically

490:1 significant, right?

490:2 A. That is correct.

490:3 Q. And then they have their most

490:4 adjusted analysis.

490:5 Do you see that?

490:6 A. Yes.

490:7 Q. And that is not statistically

490:8 significant, correct?

490:9 A. That is correct.

490:10 Q. And among other things, that is

490:11 adjusting for just what you said, things like

878.1.13

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490:12 pesticides, correct?

490:13 A. It's -- the only difference

490:14 between that and F is correcting for

490:15 pesticides.

490:16 Q. Would you agree with me that

490:17 when comparing studies, the most reasonable

490:18 comparable is to use the most fully adjusted

490:19 risk estimates?

490:20 A. I would not agree with that.

clear

490:21 Q. Do you still have in front of

490:22 you Exhibit 1604? I'll have to give you

490:23 another copy. It's this report.

490:24 And look with me, if you would,

490:25 at page 15 of your report, please. And tell

491:1 - 491:2

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:02)**

M20.136

491:1 me when you're there.

491:2 A. I'm there.

491:24 - 491:25

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:01)**

M20.137

491:24 Q. Okay. Let's read the whole

491:25 sentence.

492:1 - 492:7

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:14)**

M20.138

492:1 "As noted by both the IARC

492:2 monograph 1/12/2015 and by Chang and Delzell

492:3 2016, when comparing studies, the most

492:4 reasonable comparison is to use the most

492:5 fully adjusted risk estimates."

492:6 Did I read that correctly?

492:7 A. You did read it correctly.

492:19 - 494:22

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:44)**

M20.139

492:19 Let's continue moving on with

492:20 the data up here.

492:21 They were -- staying with

492:22 Eriksson, they have a third analysis, right,

878.1.13

492:23 greater than ten days?

492:24 Do you see that?

492:25 A. Yes, I do see that.

493:1 Q. And that is statistically

493:2 significant, correct?

493:3 A. That is correct.

493:4 Q. Is that adjusted or unadjusted?

493:5 A. I think it's unadjusted, but  
493:6 I'd have to look again.  
493:7 Q. Just so the jury understands,  
493:8 I'm going to try something very hard and ask  
493:9 you to bear with me, which will simplify the  
493:10 con -- the -- what adjustment means.  
493:11 You have talked about the risk  
493:12 of confounders in studies, correct?  
493:13 A. Correct.  
493:14 Q. And a confounder is something  
493:15 that if it's in balance between the two  
493:16 groups you're looking at and it potentially  
493:17 influences the data, it may skew your data;  
493:18 is that accurate?  
493:19 A. No.  
493:20 Q. Okay. Pesticides are a  
493:21 potential confounder in these studies,  
493:22 correct?  
493:23 A. Some pesticides would be  
493:24 considered potential confounders.  
493:25 Q. And what does it mean for a  
494:1 pesticide to be a potential confounder?  
494:2 A. That it is related to both NHL  
494:3 and it is related to exposure to glyphosate,  
494:4 that the two are -- it's correlated in both  
494:5 areas.  
494:6 Q. And is it accurate to say that  
494:7 a concern about confounders is if you don't  
494:8 take account of them, they may make it look  
494:9 like there's a relationship when, in fact,  
494:10 it's due to the confounding?  
494:11 A. That would be a concern for  
494:12 confounders, absolutely.  
494:13 Q. And so, for example, when  
494:14 Eriksson in analysis D uses most adjusted --  
494:15 Do you see that?  
494:16 A. Yes.  
494:17 Q. -- they are trying to --  
494:18 A. In analysis?  
494:19 Q. G, I'm sorry.

clear

878.1.13

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494:24 - 495:11	<p>494:20 A. G.</p> <p>494:21 Q. G, as in gopher.</p> <p>494:22 A. Yes.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:30)</b></p> <p>494:24 That would be trying to adjust</p> <p>494:25 for potential confounders, correct?</p> <p>495:1 A. Well, what they're doing there</p> <p>495:2 is comparing it to F, and so they're looking</p> <p>495:3 at the degree to which other pesticides</p> <p>495:4 reduce the relative risk that you see for</p> <p>495:5 glyphosate.</p> <p>495:6 The interpretation there is not</p> <p>495:7 that the glyphosate is no longer important.</p> <p>495:8 The interpretation there is that some of the</p> <p>495:9 relative risk you see for glyphosate is</p> <p>495:10 associated with these other pesticides, so</p> <p>495:11 they are confounded.</p>	<p>M20.140</p> <p>clear</p>
495:12 - 496:9	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:37)</b></p> <p>495:12 Q. Okay. And I think we're saying</p> <p>495:13 the same thing, but I want to make sure I</p> <p>495:14 understand it in lay terms.</p> <p>495:15 In analysis G, most adjusted,</p> <p>495:16 what they're trying to do is take out the</p> <p>495:17 effect of potential pesticide confounders,</p> <p>495:18 correct?</p> <p>495:19 A. Or measure the effect of</p> <p>495:20 pesticide confounders on the effect they saw</p> <p>495:21 for glyphosate, without the confounders in</p> <p>495:22 there.</p> <p>495:23 Q. Okay. Exactly.</p> <p>495:24 Let's go to the next one. The</p> <p>495:25 next one is Hardell and Eriksson.</p> <p>496:1 Do you see that?</p> <p>496:2 A. Yes, I do.</p> <p>496:3 Q. And they report two results,</p> <p>496:4 right?</p> <p>496:5 A. Correct.</p> <p>496:6 Q. A regular -- a first result and</p> <p>496:7 a most adjusted result.</p> <p>496:8 Do you see that?</p>	<p>M20.141</p> <p>878.1.14</p>

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496:17 - 497:8	<p>496:9 A. Yes, I do.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)</b></p> <p>496:17 The first result is</p> <p>496:18 statistically significant, correct?</p> <p>496:19 A. Hardell and Eriksson, the lower</p> <p>496:20 bound for the confidence bound is above 1.</p> <p>496:21 Q. Right.</p> <p>496:22 The most adjusted result is not</p> <p>496:23 statistically significant?</p> <p>496:24 A. The lower bound is not above 1,</p> <p>496:25 that is correct.</p> <p>497:1 Q. McDuffie reports two analyses,</p> <p>497:2 correct?</p> <p>497:3 A. Yes, they do.</p> <p>497:4 Q. One is statistically</p> <p>497:5 significant; one is not?</p> <p>497:6 A. Again, one has a confidence</p> <p>497:7 bound, lower confidence bound, above 1; one</p> <p>497:8 does not.</p>	<p>M20.142</p> <p>878.1.15</p> <p>clear</p>
497:9 - 497:23	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:30)</b></p> <p>497:9 Q. Does that mean it's not</p> <p>497:10 statistical significant using a .05 level?</p> <p>497:11 A. Again, in understanding</p> <p>497:12 epidemiology, the epidemiologists don't</p> <p>497:13 always go to this yes/no statistically</p> <p>497:14 significant. There's quite a debate in the</p> <p>497:15 literature about that. You can -- you can</p> <p>497:16 set that bound, as you want to set it.</p> <p>497:17 Epidemiologists in the general rule would not</p> <p>497:18 do that these days.</p> <p>497:19 But if you're going to set that</p> <p>497:20 bound, then I will say, yes, one is</p> <p>497:21 statistically significant and one is not.</p> <p>497:22 Q. Thank you, Doctor.</p> <p>497:23 A. Okay.</p>	<p>M20.143</p>
498:23 - 499:12	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:40)</b></p> <p>498:23 Let's look at 893, which was</p> <p>498:24 another of the images you showed the jury</p> <p>498:25 reporting data from these six studies.</p> <p>499:1 Do you see that?</p>	<p>M20.144</p> <p>893.1</p>

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499:2 A. Yes.

499:3 Q. And to be fair, it's page 1 of  
499:4 893.

499:5 In the interest of time, let me  
499:6 see if I can short-circuit it.

499:7 Am I correct that according to  
499:8 this data, at least based on the data  
499:9 presented on this slide, at least one of the  
499:10 findings from every study is not  
499:11 statistically significant?

499:12 A. Correct.

499:13 - 500:4

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:38)**

M20.145

499:13 Q. And then I think you showed the  
499:14 jury page 2 of this document, and I'll ask  
499:15 you the same question for page 2.

893.2

499:16 Is it true that for every one  
499:17 of the studies shown on page 2, at least one  
499:18 of the results shown is not statistically  
499:19 significant?

499:20 A. That's correct.

499:21 Q. In fact, just looking  
499:22 numerically, most of the results shown here  
499:23 are not statistically significant, correct?

893.2.2

499:24 A. That would be correct.

499:25 Q. And a lot of them are actually  
500:1 on the protective side of the equation,  
500:2 correct?

500:3 A. Because there are a lot more  
500:4 done in those studies. But, yes, correct.

500:12 - 501:3

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:28)**

M20.146

500:12 Q. This chart, the one that we're  
500:13 looking at now, page 2 of Exhibit 893, it  
500:14 breaks the data out by different metrics.  
500:15 Do you see that?

500:16 A. Yes.

500:17 Q. So one of the metrics is how  
500:18 many days.

893.2.1

500:19 Do you see that?

500:20 A. Correct.

500:21 Q. One is cumulative exposure,

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500:22 intensity of exposure, latency, et cetera.

500:23 Do you see that?

500:24 A. Yes.

500:25 Q. Do you know which, if any, of

501:1 those buckets that the plaintiff in this case

501:2 fits into?

501:3 A. No.

501:4 - 502:7

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:58)**

M20.147

501:4 Q. I think you said this

clear

501:5 yesterday, but I want to make sure I

501:6 understand it.

501:7 Am I correct that when you look

501:8 at this data we've been looking at, the human

501:9 epidemiological data, you would say it could

501:10 be causal, but I can't absolutely say it's

501:11 causal today with just this data?

501:12 Is that accurate? Did I hear

501:13 that right yesterday?

501:14 A. Something like that. I guess I

501:15 would say it's reasonable to believe that the

501:16 association we see is causal, but there's not

501:17 enough -- there's questions that I have that

501:18 would not put me over that line right now.

501:19 Q. You can't make a firm statement

501:20 about glyphosate from the epidemiology data

501:21 alone?

501:22 A. That is correct. Other than

501:23 that there's an association, it's potentially

501:24 causal. That's a firm statement. It's not

501:25 the firm statement that glyphosate causes NHL

502:1 based solely on the animal -- human

502:2 epidemiology data.

502:3 Q. You can't rule out bias?

502:4 A. I come close to ruling out

502:5 bias, but I can't completely rule it out.

502:6 Q. You can't rule out confounders?

502:7 A. Not from all the studies.

510:6 - 511:9

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:04)**

M20.148

510:6 Do you recall talking earlier

510:7 about these comments that you submitted to

1456.1

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510:8 the US EPA in October 2016?

510:9 A. Yes, I do.

510:10 Q. And this is a document that you  
510:11 wrote?

1456.1.3

510:12 A. Yes, it is.

510:13 Q. In the document, you give your  
510:14 specific views on glyphosate data, correct?

510:15 A. I -- I give my comments to how  
510:16 EPA viewed the glyphosate data and my  
510:17 concerns about some of the things they did.

510:18 Q. Okay. If we flip ahead to  
510:19 page 5 of your comments. And you've put line  
510:20 numbers down the left-hand side.

510:21 Do you see that?

510:22 A. Yes, I do.

510:23 Q. Makes it quite helpful for our  
510:24 purposes. It's line 3. It says "human  
510:25 evidence."

1456.5.1

511:1 Do you see that?

511:2 A. Yes.

511:3 Q. If we go to the next page under  
511:4 human evidence -- human evidence is the  
511:5 epidemiological studies we've been  
511:6 discussing, right?

1456.6.1

511:7 A. That is correct.

511:8 Q. Let's go to the next page,  
511:9 talking about the human evidence.

511:15 - 513:25

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:01:43)**

M20.149

511:15 Q. You write, "However, it is fair  
511:16 to say that confounding could not be ruled  
511:17 out in these studies."

1456.6.2

511:18 Did I read that correctly?

511:19 A. You did. It's part of a  
511:20 broader comment, but, yes.

511:21 Q. And that's still your view  
511:22 today, correct?

511:23 A. When we're talking about these  
511:24 studies, we're talking about all of the  
511:25 studies, not just case-control, yes.

512:1 Q. Right.

512:2 And then it says, "4, recall  
512:3 bias is a concern, especially in the  
512:4 case-control studies." And it says,  
512:5 "Comment: I agree."  
512:6 Do you see that?  
512:7 A. So, yes, to put this in a  
512:8 little context, the 4, recall bias is a  
512:9 concern, is what EPA said.  
512:10 Q. Yes.  
512:11 A. And the comment is I'm agreeing  
512:12 with what their statement is.  
512:13 Q. Thank you. That was exactly  
512:14 what I wanted to elicit, Doctor.  
512:15 EPA is saying that recall bias  
512:16 is a concern, especially in the case-control  
512:17 studies, and you were saying, I agree with  
512:18 that?  
512:19 A. That's correct.  
512:20 Q. Let's go to the next page,  
512:21 please.  
512:22 And you've got a paragraph here  
512:23 that says "summary," starting at page 116.  
512:24 Do you see that?  
512:25 A. Yes, I do.  
513:1 Q. And I just want to read the end  
513:2 of this paragraph. It states, "So, is  
513:3 causality plausible here? Yes, absolutely."  
513:4 Did I read that correctly?  
513:5 A. Yes, you did.  
513:6 Q. And that's consistent with your  
513:7 views today, correct?  
513:8 A. Yes.  
513:9 Q. Next you say, "Is it  
513:10 demonstrated? No, clearly not."  
513:11 Did I read that correctly?  
513:12 A. You did read that correctly.  
513:13 Q. Do you stand behind that part  
513:14 of your statement to EPA?  
513:15 A. Yes.  
513:16 Q. It then says: "Are the

1456.6.3

1456.7.2

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513:17 findings possibly the result of chance, bias,  
513:18 and/or confounding?"

513:19 And your answer is: "Yes, but  
513:20 more unlikely than likely."

513:21 Did I read that correctly?

clear

513:22 A. That is correct.

513:23 Q. And do you stand behind that  
513:24 statement as well?

513:25 A. Yes.

514:4 - 514:14

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:21)**

M20.150

514:4 Earlier in the day, do you

514:5 remember me showing you the De Roos study?

514:6 A. Which one?

514:7 Q. Good question.

514:8 The 2005 study.

514:9 A. Okay.

514:10 Q. It's Exhibit 528. It's in your  
514:11 binder.

528.1

514:12 A. Yes, I do remember. I think we

514:13 looked at it, but certainly I remember the

514:14 study.

514:23 - 517:9

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:02:32)**

M20.151

514:23 I showed you language that

514:24 says, "Although there has been little

514:25 consistent evidence of genotoxicity from in  
515:1 vitro and animal studies."

528.1.3

515:2 Do you remember that?

515:3 A. Now I remember it.

515:4 Q. And they continue by saying, "A

515:5 few epidemiologic reports indicated potential

515:6 health effects of glyphosate."

515:7 Do you see that?

515:8 A. Potential health effects of

515:9 glyphosate, yes.

515:10 Q. Potential health effects of

515:11 glyphosate, yes.

515:12 And that's referring to some of

515:13 the same studies we've been looking at on

515:14 your forest plots, right?

515:15 A. I assume so. It's the

Page/Line	Source	ID
515:16	abstract, so there's no references, but I	
515:17	assume that's what they're talking about.	
515:18	Q. Let's look a little further	528.1.6
515:19	down the page. At the bottom of the first	
515:20	column, do you see where they say, "Results	
515:21	from genotoxicity studies of glyphosate have	
515:22	been conflicting"?	
515:23	Do you see that?	
515:24	A. Yes, I do.	
515:25	Q. Let's go ahead to their	
516:1	discussion of their data. It's on page 52 of	528.4
516:2	the study, please, Doctor.	528.4.3
516:3	In the middle paragraph under	
516:4	discussion, these authors state as to their	
516:5	results, "There was no association between	528.4.1
516:6	glyphosate exposure and all cancer incidence,	
516:7	or most of the specific cancer subtypes we	
516:8	evaluated, including NHL."	
516:9	Did I read that correctly?	
516:10	A. You read that correctly.	
516:11	Q. They go on to say that that	
516:12	statement is true, "Whether the exposure	528.4.2
516:13	metric was ever used, cumulative exposure	
516:14	days or intensity-weighted cumulative	
516:15	exposure days."	
516:16	Did I read that correctly?	
516:17	A. Yes, you did.	
516:18	Q. You talked -- I think you had	
516:19	on your forest plot some published	
516:20	meta-analyses.	
516:21	Do you remember that?	
516:22	A. Yes.	
516:23	Q. One of them was by some	
516:24	authors, Chang and Delzell.	
516:25	Do you remember that?	
517:1	A. Yes, I do.	
517:2	Q. I'd like to show you that	
517:3	published meta-analysis, Exhibit 1102.	1102.1
517:4	And do you recognize that as	
517:5	the Chang and Delzell study that you cite in	1102.1.2

Page/Line	Source	ID
517:10 - 518:18	<p>517:6 your report and that was on some of your  517:7 meta -- some of your human epidemiology  517:8 slides?  517:9 A. Yes, I do recognize it.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:32)</b></p>	M20.152
	<p>517:10 Q. Okay. I'd like to ask you a  517:11 few questions about this article. Look with  517:12 me, if you would, at page 422 of the article.</p>	1102.21
	<p>517:13 A. 422.  517:14 Q. And I'd like to direct your  517:15 attention to the upper right-hand corner.  517:16 Do you see where in the second  517:17 to last sentence of that carryover paragraph  517:18 they report their calculation of the relative  517:19 risk?</p>	1102.21.11
	<p>517:20 A. Yes.  517:21 Q. And they say specifically, "The  517:22 meta-RRs" -- that's relative risk from the  517:23 meta-analysis, correct?</p>	
	<p>517:24 A. Correct.  517:25 Q. "The meta-RRs calculated based  518:1 on at least four studies ranged from between  518:2 1.3 and 1.4."</p>	
	<p>518:3 Did I read that correctly?  518:4 A. You did.</p>	
	<p>518:5 Q. They go on to say, "These  518:6 associations are not of sufficient magnitude  518:7 to exclude modest bias or confounding as  518:8 reasonable explanations for the observed  518:9 results."</p>	
	<p>518:10 Did I read that correctly?  518:11 A. You did read it correctly.</p>	
	<p>518:12 Q. Just yes or no, is that a fair  518:13 statement in your view?</p>	
	<p>518:14 A. Assuming the meta-RRs they're  518:15 talking about are their models 1 through 4,  518:16 then, yes, that's true, but I can't be  518:17 certain that's the meta-RRs they're talking  518:18 about.</p>	
518:19 - 519:5	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</b></p>	M20.153

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	518:19 Q. Okay.	
	518:20 A. I might not agree to the word	clear
	518:21 "modest" bias, but...	
	518:22 Q. Okay. Other than that, would	
	518:23 it be a fair statement?	
	518:24 A. Okay. I would say -- yeah,	
	518:25 I -- I'm not sure reasonable explanations is	
	519:1 correct. Certainly they are potential	
	519:2 explanations.	
	519:3 Q. Okay.	
	519:4 A. Reasonable implies more	
	519:5 positive than I'm willing to accept.	
519:6 - 519:15	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</b>	M20.154
	519:6 Q. Okay. Are you aware that they	
	519:7 conducted a Bradford Hill analysis?	
	519:8 A. In this paper?	
	519:9 Q. Yes.	
	519:10 A. Vaguely recall something along	
	519:11 those lines.	
	519:12 Q. Okay. Let's take a look at it.	
	519:13 On the same page, in the bottom left-hand	1102.21.12
	519:14 corner, do you see that there's reference to	
	519:15 the Bradford Hill viewpoints?	
519:20 - 519:21	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:05)</b>	M20.155
	519:20 There is a Bradford Hill	
	519:21 viewpoints comment, yes.	
520:6 - 521:19	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:21)</b>	M20.156
	520:6 Q. One of the Bradford Hill	
	520:7 criteria that you talked about is	
	520:8 consistency, right?	
	520:9 A. Correct.	
	520:10 Q. And I believe you said on	
	520:11 your -- on your chart that there was	
	520:12 consistency.	
	520:13 Do you recall that?	
	520:14 A. Yes.	
	520:15 Q. Do you see what these authors	
	520:16 concluding -- concluded regarding	
	520:17 consistency? And let me just direct you to	
	520:18 it.	

Page/Line	Source	ID
520:19 Do you see on the second column 520:20 on 422, second paragraph?		1102.21.13
520:21 A. Yes, I do.		
520:22 Q. They write, "Results were not 520:23 consistent between case-control studies of 520:24 NHL and one prospective cohort study of NHL 520:25 which reported no association."		
521:1 Did I read that correctly?		
521:2 A. You did.		
521:3 Q. And having applied these 521:4 different Bradford Hill criteria, I'd like to 521:5 look at what the authors concluded.		
521:6 If you look at the bottom on 521:7 the left-hand side, still the same page, the 521:8 last paragraph, "overall evaluation."		1102.21.14
521:9 Do you see that?		
521:10 A. Yes.		
521:11 Q. And in the second sentence 521:12 under that they say, "In addition, an 521:13 evaluation of the association between 521:14 glyphosate exposure and risk of LHC based on 521:15 the Bradford Hill viewpoints does not favor a 521:16 causal relationship with NHL, any NHL 521:17 subtype, HL, MM or leukemia."		
521:18 Did I read that correctly?		
521:19 A. You read that correctly.		
521:20 - 521:21 <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</b>		M20.157
521:20 Q. Let's go to the next page,		1102.22
521:21 please, of this study.		
521:22 - 522:9 <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25)</b>		M20.158
521:22 Do you see where they talk 521:23 about the Bradford Hill criteria in the last 521:24 paragraph before the discussion?		1102.22.4
521:25 A. I see there's a discussion		
522:1 there, yes.		
522:2 Q. And they state, "In summary, 522:3 although none of the Bradford Hill viewpoints 522:4 can establish or disprove causality, we did 522:5 not find compelling evidence in support of 522:6 causality based on any of the nine		1102.22.5

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522:10 - 522:20	<p>522:7 viewpoints."</p> <p>522:8 Did I read that correctly?</p> <p>522:9 A. That is correct.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25)</b></p> <p>522:10 Q. And those are the same Bradford Hill viewpoints that you discussed with the plaintiff's attorney, correct?</p> <p>522:13 A. Not exactly. Again, I'm closer to the EPA interpretation of Bradford Hill and how they use it than what Bradford Hill himself wrote.</p> <p>522:17 I'm not sure how they were using it here in absolute certainty, so I can just simply say that's what they said.</p> <p>522:20 You're right, that's what they said.</p>	M20.159
523:8 - 523:19	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)</b></p> <p>523:8 Q. Okay. Let me show you one more thing in this article. I think I had stopped before we looked at the second sentence in this paragraph.</p> <p>523:12 Do you see their conclusion?</p> <p>523:13 "Thus, on balance, the existing epidemiological evidence does not favor a causal effect of glyphosate on NHL, HL, MM, leukemia, or any subtype of these malignancies."</p> <p>523:18 Did I read that correct?</p>	M20.160
523:20 - 525:1	<p>523:19 A. Let me look. That is what it says.</p> <p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:05)</b></p> <p>523:20 says.</p> <p>523:21 Q. Okay. And this is a study that you reference in your report and on some of your slides, correct?</p> <p>523:24 A. That is correct.</p> <p>523:25 Q. You also referenced a more recent meta-analysis by the lead author Zhang.</p> <p>524:3 Do you remember that?</p> <p>524:4 A. Yes.</p> <p>524:5 Q. And do you recall that in their</p>	M20.161
		clear

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524:6 primary meta-analysis, they included a 2018

524:7 study by the leader author Andreotti?

524:8 A. Yes.

524:9 Q. You would not put the Andreotti

524:10 study in a meta-analysis, true?

524:11 A. As a general rule, I probably

524:12 would not put it -- well, I certainly can't

524:13 put it in a yes/no meta-analysis.

524:14 In the meta-analysis they did,

524:15 it fits with their criteria for how they were

524:16 putting that meta-analysis together.

524:17 Q. I understand that. I'm talking

524:18 about your views.

524:19 In your views, you would not

524:20 put the Andreotti study in a meta-analysis,

524:21 partly because of what you view as failures

524:22 in the study, partly plus of an imputation

524:23 issue, correct?

524:24 A. The --

524:25 Q. Is what I said true?

525:1 A. Yeah, pretty much it's true.

527:10 - 527:24

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:27)**

M20.162

527:10 Q. Doctor, before we went off the

527:11 record, we touched briefly on a meta-analysis

527:12 by the lead author Zhang.

527:13 Do you recall that?

527:14 A. Yes.

527:15 Q. And if I recall correctly, that

527:16 was one -- you reported data from that on

527:17 some of your forest plots, correct?

527:18 A. At this deposition, yes.

527:19 Q. Yes.

527:20 During your testimony, I think,

527:21 yesterday, right?

527:22 A. Correct.

527:23 Q. I'd like to show you a copy of

527:24 that. It's marked as Exhibit 554, please.

554.1

527:25 - 528:13

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:31)**

M20.163

527:25 Do you see that this is a copy

528:1 of the Zhang publication?

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528:2 A. Yes.

528:3 Q. This is the one that in their

528:4 primary meta-analysis uses the Andreotti

528:5 study that we talked about briefly from 2018?

528:6 A. Amongst others, yes.

clear

528:7 Q. Yes, amongst others.

528:8 And I don't want to get into

528:9 details right now, but as I understand it,

528:10 you have critiques of the Andreotti in 2018,

528:11 correct?

528:12 A. I submitted a supplemental

528:13 report, yes.

528:16 - 529:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:45)**

M20.164

528:16 if we go to the Zhang meta-analysis.

528:17 First of all, this reports no

528:18 new original data, correct? It combines

528:19 previous existing data, correct?

528:20 A. That is correct.

528:21 Q. If we go to the tables in the

528:22 Zhang study, do you recall that they gave

528:23 quality scores to the different studies that

528:24 they evaluated?

528:25 A. Vaguely, yes.

529:1 Q. Let's look at that. I believe

529:2 it's numbered page 52 of the manuscript I've

529:3 given you.

529:4 Do you see that?

529:5 A. Yes.

530:7 - 532:12

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:02:32)**

M20.165

530:7 Q. And am I correct that their

530:8 highest overall quality score is for the

530:9 Andreotti 2018 study?

530:10 A. Yes.

530:11 Q. Let's look at that study,

530:12 please. It might be in your binder. It's

530:13 Exhibit 550.

530:14 Do you have that in your

530:15 binder?

530:16 A. Yes, I do.

530:17 Q. Let's take a look at that.

554.53

554.53.1

550.1

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530:18 Just a couple things regarding this study,  
530:19 just to orient us.  
530:20 First of all, this was 550.1.16  
530:21 available in 2017 online. It was published  
530:22 in 2018, correct?  
530:23 A. I believe that's correct.  
530:24 Q. And if we look at the authors 550.1.7  
530:25 of this study, do you see these authors?  
531:1 A. Yes, I do.  
531:2 Q. This includes two people,  
531:3 Dr. De Roos and I believe it's -- is it  
531:4 Dr. or Mr. Lynch? Doctor?  
531:5 A. I think it's Dr. Lynch.  
531:6 Q. Okay. Dr. Lynch, who you told  
531:7 us yesterday had signed on to your letter a  
531:8 couple years before this, correct?  
531:9 A. Correct.  
531:10 Q. And if we look at the 550.1.20  
531:11 affiliations of these authors, which is a  
531:12 little hard because the print is small, do  
531:13 you see that some of these authors have  
531:14 affiliations with the National Cancer  
531:15 Institute?  
531:16 A. Yes.  
531:17 Q. You see that some of them  
531:18 report affiliations with your former  
531:19 organization, NIEHS, the National Institute 550.1.21  
531:20 of Environmental Health Sciences?  
531:21 A. Yes.  
531:22 Q. And in fact, going back to that  
531:23 point about the National Cancer Institute, am  
531:24 I correct that this article was published in 550.1.22  
531:25 the Journal of the National Cancer Institute?  
532:1 A. The two are not related, but,  
532:2 yes, it's published in the Journal of  
532:3 National Cancer Institute, which is not the  
532:4 Journal of the National Cancer Institute.  
532:5 Q. The Journal of the National  
532:6 Cancer Institute is not the Journal of the  
532:7 National Cancer Institute?

Page/Line	Source	ID
533:15 - 533:21	<p>532:8 A. Correct. It's owned by Oxford  532:9 Press. It's a private journal.  532:10 Q. It's a peer-reviewed journal,  532:11 right?  532:12 A. It's a peer-reviewed journal.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21)</b></p>	M20.166
534:5 - 534:24	<p>533:15 Do you see on page 515 -- and  533:16 it carries over, which is going to be hard  533:17 for me with the screen. But do you see where  533:18 it identifies, starting at the bottom left of  533:19 page 515, do you see that it identifies who  533:20 funded it?  533:21 A. Yes.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:34)</b>  534:5 It's the Intramural Research  534:6 Program of the National Institutes of Health,  534:7 National -- and bear with me, I'm going to  534:8 turn the page so we can continue -- National  534:9 Cancer Institute, Division of Cancer  534:10 Epidemiology and Genetics.  534:11 Do you see that?  534:12 A. Yes, I do.  534:13 Q. It's also funded by the  534:14 National Institute of Environmental Health  534:15 Science, correct?  534:16 A. Correct.  534:17 Q. That's your former group,  534:18 NIEHS, right?  534:19 A. Correct.  534:20 Q. And then it gives some other  534:21 funding sources, including the University of  534:22 Iowa.  534:23 Do you see that?  534:24 A. Yes.</p>	557_1_558.1.1
535:12 - 535:15	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04)</b>  535:12 Q. It's not funded by Monsanto,  535:13 correct?  535:14 A. That is correct. As far as I  535:15 know.</p>	M20.168
535:18 - 536:14	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:13)</b></p>	M20.169

Page/Line	Source	ID
	535:18 Let's go back to page 1. And 535:19 if we look at the results in the abstract, 535:20 that's probably the easiest place to do it. 535:21 Do you see where it reports on 535:22 the number of individuals that looked at this 535:23 study. Among 54,000 applicators, 44,932 used 535:24 glyphosate. 535:25 Do you see that?	550.1.23
	536:1 A. I see that.	
	536:2 Q. Is it correct that this study 536:3 had more exposed NHL cases than in all the 536:4 published case-control studies combined?	
	536:5 A. If you're counting their 536:6 exposure, meaning also the people who are -- 536:7 have a statistically generated, imputed 536:8 exposure, then, yes.	
	536:9 Q. And these authors controlled 536:10 for specific pesticides, true?	
	536:11 A. They did.	
	536:12 Q. And just so that the jury knows 536:13 what we're talking about, if we go to 536:14 page 515 of the article, on the left-hand	550.7
536:15 - 536:21	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:27)</b> 536:15 side, do you see where these authors state, 536:16 "In this analysis, we controlled for the use 536:17 of correlated pesticides, which was not 536:18 possible in all previous studies"?	M20.170
	536:19 Did I read that correctly? 536:20 A. I have no idea what it means, 536:21 but, yes, you read it correctly.	550.7.6
539:20 - 539:21	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:01)</b> 539:20 Let's finish up with the 539:21 Andreotti study.	M20.171
542:14 - 542:25	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:42)</b> 542:14 Q. Under discussion it states, "In 542:15 this updated evaluation of glyphosate use and 542:16 cancer risk in a large prospective study of 542:17 pesticide applicators, we observed no 542:18 associations between glyphosate use and 542:19 overall cancer risk or with total	M20.172 550.5.2

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542:20 lymphohematopoietic cancers, including NHL

542:21 and multiple myeloma," correct?

542:22 A. Correct.

542:23 Q. That was their finding?

542:24 A. That's what it says.

542:25 Q. Let's go ahead to page 515.

550.7

543:1 - 543:21

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:48)**

M20.173

543:1 And I'd like to direct your attention on

543:2 page 515 to the left-hand side where these

543:3 authors state about, a couple lines down, the

543:4 lack of association.

550.7.7

543:5 Do you see where I'm reading?

543:6 A. Yes, I see where you're

543:7 reading.

543:8 Q. They state, "The lack of

543:9 association between glyphosate and NHL is

543:10 also consistent with the previous AHS

543:11 analysis."

543:12 Did I read that correctly?

543:13 A. That's what it says, that is

543:14 correct.

543:15 Q. And just so the jury knows what

543:16 we're talking about, the previous AHS

543:17 analysis they're referencing there is the

543:18 2005 De Roos study that you and I have talked

543:19 about, correct?

543:20 A. That is correct. By looking at

543:21 the references, that is correct.

544:5 - 545:3

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:56)**

M20.174

544:5 Q. And let's -- let's look at what

544:6 two of your coauthors on your 2016 paper said

544:7 in their 2018 publication.

544:8 Turn with me, if you would --

550.7

544:9 actually, stay with me, if you would, on this

544:10 page.

544:11 Do you see where they have a

544:12 concluding paragraph?

544:13 A. Page 515, the final paragraph

544:14 before funding?

544:15 Q. The final paragraph before

550.7.8

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544:16 funding, correct.

544:17 A. Okay.

544:18 Q. Do you see where they state,

544:19 "In conclusion, we found no evidence of an

544:20 association between glyphosate use and risk

544:21 of any solid tumors or lymphoid malignancies,

544:22 including NHL and its subtypes"?

clear

544:23 Did I read that correctly?

544:24 A. You did.

544:25 Q. Am I correct that this is the

545:1 most recent epidemiological study using

545:2 original data that exists?

545:3 A. Yes.

545:4 - 545:5

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:02)**

M20.175

545:4 MR. SCHMIDT: Thank you,

545:5 Doctor. That's all I have.

= 01:45:05

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# PORTIER\_REDIRECT\_01 FINAL PLAYED

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549:1 - 549:10	<b>Portier, Christopher 02-22-2019 (00:00:22)</b> 549:1 So Mr. Schmidt covered a lot of 549:2 different topics with you on 549:3 cross-examination, and I want to explore a 549:4 couple of them because we really didn't spend 549:5 too much time on it on your direct. 549:6 Let's start off exactly where 549:7 Mr. Schmidt left off, the Agricultural Health 549:8 Study. 549:9 Have you reviewed that study, 549:10 both from 2005 and 2018?	CP_SS_REDIRECT_01.1
549:13 - 549:13	<b>Portier, Christopher 02-22-2019 (00:00:00)</b> 549:13 THE WITNESS: Yes, I have.	CP_SS_REDIRECT_01.2
549:15 - 550:23	<b>Portier, Christopher 02-22-2019 (00:01:44)</b> 549:15 Q. And have you systematically 549:16 gone through and analyzed the strengths and 549:17 weaknesses? 549:18 A. Yes, I have. 549:19 Q. Okay. What is your opinion 549:20 about the reliability and value of the 549:21 glyphosate data for -- in the Agricultural 549:22 Health Study? 549:23 A. Well, the data from the 2005 549:24 study are fairly reliable. The entire cohort 549:25 responded. The analysis was done extremely 550:1 carefully. It's very well done. I think 550:2 it's a very reliable study. 550:3 The Andreotti study, the 2018 550:4 study, has some serious limitations in its 550:5 interpretation, partially due to the 550:6 nonresponse rate, which was 40 percent. 550:7 Their attempts to correct for 550:8 this nonresponse by using an imputation 550:9 algorithm failed to solve the problem because 550:10 their imputation algorithm introduced a bias 550:11 into the exposure classifications that could 550:12 have affected the overall response. 550:13 There are other issues with 550:14 that response which forces it towards the 550:15 null hypothesis based upon exposure --	CP_SS_REDIRECT_01.3

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551:24 - 552:10	<p>550:16 exposure misclassification, and that's very  550:17 well-addressed in several papers, the most  550:18 notable by Aaron Blair, one of the authors of  550:19 that as well.  550:20 I think it has serious  550:21 limitations. I think it's -- the result is  550:22 it's giving you exactly what you would expect  550:23 to see from it, that is, no effect.</p>	CP_SS_REDIRECT_01.4
	<b>Portier, Christopher 02-22-2019 (00:00:20)</b>	
	<p>551:24 Q. All right, sir. During  551:25 cross-examination, Mr. Schmidt, he showed  552:1 you, I believe, two meta-analyses; is that  552:2 correct?  552:3 A. Two papers with meta-analyses  552:4 in them, yes.  552:5 Q. One was by Chang and Delzell;  552:6 is that right?  552:7 A. That's correct.  552:8 Q. And the other one was by Zhang,  552:9 et al.?</p>	
557:24 - 558:2	<b>Portier, Christopher 02-22-2019 (00:00:03)</b>	CP_SS_REDIRECT_01.5
	<p>557:24 Q. You have  557:25 Dr. Chang and Dr. Delzell.  558:1 Do you see that?  558:2 A. Yes, I do.</p>	
558:21 - 559:11	<b>Portier, Christopher 02-22-2019 (00:00:36)</b>	CP_SS_REDIRECT_01.6
	<p>558:21 Q. And if we actually turn to the  558:22 disclosure page here, do you see this  558:23 statement here? It's on page 424 at the end.  558:24 A. 424, yes, I see 424.  558:25 Q. And there's a section that  559:1 says, "Funding."  559:2 Do you see that?  559:3 A. Correct.  559:4 Q. And it says, "This work was  559:5 supported by Monsanto Company, the original  559:6 producer and marketer of glyphosate  559:7 formulations."  559:8 Do you see that?</p>	

Page/Line	Source	ID
	559:9 A. I see that.	
	559:10 Q. Did Mr. Schmidt show the jury	
	559:11 this when he talked about this paper?	
559:14 - 559:14	<b>Portier, Christopher 02-22-2019 (00:00:01)</b>	CP_SS_REDIRECT_01.7
	559:14 THE WITNESS: No.	
559:16 - 559:21	<b>Portier, Christopher 02-22-2019 (00:00:09)</b>	CP_SS_REDIRECT_01.8
	559:16 Q. And it goes on to discuss, you	
	559:17 know, these -- these -- the disclosure	
	559:18 statement. It says, "The sponsors" -- stop	
	559:19 right there.	
	559:20 That's referring to Monsanto,	
	559:21 right?	
559:24 - 560:14	<b>Portier, Christopher 02-22-2019 (00:00:25)</b>	CP_SS_REDIRECT_01.9
	559:24 THE WITNESS: That would	
	559:25 generally be the interpretation of the	
	560:1 word "sponsors."	
	560:2 QUESTIONS BY MR. WISNER:	
	560:3 Q. Right.	
	560:4 So it says, "Monsanto was	
	560:5 provided the opportunity to review the	
	560:6 manuscript prior to journal submission, but	
	560:7 inclusion of their suggestions was left to	
	560:8 the discretion of the authors, who retained	
	560:9 sole control of the manuscript, content and	
	560:10 findings."	
	560:11 Do you see that?	
	560:12 A. I see that. You've inserted	
	560:13 Monsanto for the sponsors were provided, but,	
	560:14 yes, I see it.	
569:7 - 569:14	<b>Portier, Christopher 02-22-2019 (00:00:15)</b>	CP_SS_REDIRECT_01.10
	569:7 So we spent some time on this	
	569:8 overall evaluation section.	
	569:9 Do you recall that?	
	569:10 A. Yes.	
	569:11 Q. And they were -- there was some	
	569:12 discussions about the use of the Bradford	
	569:13 Hill criteria by Chang and Delzell, right?	
	569:14 A. Correct.	
569:15 - 569:23	<b>Portier, Christopher 02-22-2019 (00:00:25)</b>	CP_SS_REDIRECT_01.11
	569:15 Q. All right. First questions	

569:16 first. When they're looking at the Bradford  
569:17 Hill criteria in this context, are they just  
569:18 looking at epidemiology or are they looking  
569:19 at the full spectrum of science?

569:20 A. I would have to reread this  
569:21 whole section to see if they talk about the  
569:22 animal studies at all. So I can't answer the  
569:23 question without rereading everything.

569:24 - 573:6

**Portier, Christopher 02-22-2019 (00:03:19)**

CP\_SS\_REDIRECT\_01.12

569:24 Q. Okay. There was at one point  
569:25 here a discussion about consistency.

570:1 Do you recall that?

570:2 A. Yes, I do.

570:3 Q. And they -- and Mr. Schmidt

570:4 specifically asked you about what -- you  
570:5 know, they found that there was -- that the  
570:6 data was not consistent in the epidemiology.

570:7 Do you recall that?

570:8 A. Yes, that is the first

570:9 paragraph that starts with "results" right  
570:10 here.

570:11 Q. Okay. Sir, do you agree with  
570:12 what they're saying here about the  
570:13 consistency of the epidemiological data?

570:14 A. So it strikes me as  
570:15 interesting. They say the results were not  
570:16 consistent between case-control studies in  
570:17 NHL and the one prospective cohort study of  
570:18 NHL which reported no association.

570:19 I don't know what they mean  
570:20 there in terms of not consistent. The entire  
570:21 purpose of the meta-analysis is to look at  
570:22 the degree to which the studies are  
570:23 consistent with each other and give a  
570:24 consistent answer.

570:25 Now, in the analyses they did  
571:1 here, there was no heterogeneity. They  
571:2 tested for heterogeneity in response between  
571:3 the various studies. There was none  
571:4 whatsoever. So that would say the studies

571:5 were indeed consistent.  
571:6 I don't understand the  
571:7 statement they've made here in terms of their  
571:8 measure of consistency.  
571:9 Q. Now, if we can go to the -- one  
571:10 of the things that we discussed was this  
571:11 chart that was created that included the  
571:12 meta-analysis.  
571:13 Do you recall that? It's up on  
571:14 the screen here.  
571:15 A. This chart, yes, I still have  
571:16 it right here.  
571:17 Q. And this is page 878; is that  
571:18 right? Sorry, Exhibit 878?  
571:19 A. Yes.  
571:20 Q. And if we can go back to the  
571:21 document camera very quickly, it says here  
571:22 that it's from Table 7, so I just want to  
571:23 show the jury Table 7 from Zhang.  
571:24 Is this the table you're  
571:25 referring to?  
572:1 A. Yes, that is the table I'm  
572:2 referring to.  
572:3 Q. Okay. So let's go back to the  
572:4 iPad.  
572:5 So we're looking here at this  
572:6 analysis. And, you know, if we go down to  
572:7 the published meta-analysis, that's the green  
572:8 stuff; is that right?  
572:9 A. Correct.  
572:10 Q. Okay. What significance, if  
572:11 any, is there to the fact that every single  
572:12 one of them is to the right of the blue line  
572:13 and statistically significant?  
572:14 A. It basically tells you that all  
572:15 of these -- Mr. Schmidt talked about  
572:16 significant or nonsignificant.  
572:17 I look at these confidence  
572:18 bounds above the -- in the rest of that A  
572:19 through M analyses, and you see that the

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572:20 lower confidence bound is just barely  
 572:21 below 1. When you do a meta-analysis and  
 572:22 bring that all together, it tells you they're  
 572:23 all contributing to the positive finding.  
 572:24 And what we're seeing here with  
 572:25 these five findings down here is that the  
 573:1 data is consistent with each other, and  
 573:2 they're consistent with the finding that  
 573:3 there is indeed an association. And it is  
 573:4 statistically significant, above .05, because  
 573:5 the confidence bounds do not include 1 for  
 573:6 all of these meta-analyses.

573:15 - 573:19

**Portier, Christopher 02-22-2019 (00:00:09)**

CP\_SS\_REDIRECT\_01.13

573:15 When we talk about these  
 573:16 meta-analysis, sir, does that include the one  
 573:17 that was funded by Monsanto?  
 573:18 A. Yes, the Chang and Delzell  
 573:19 study, that is correct.

582:20 - 583:7

**Portier, Christopher 02-22-2019 (00:00:29)**

CP\_SS\_REDIRECT\_01.14

582:20 Q. All right, sir. So I want to  
 582:21 follow up on a few other things that were  
 582:22 discussed on cross-examination.  
 582:23 The first one was, there was  
 582:24 a -- a series of letters that were shown that  
 582:25 you had written to various regulatory  
 583:1 agencies.  
 583:2 Do you recall that?  
 583:3 A. Yes.  
 583:4 Q. Let me just ask you something.  
 583:5 Were you being paid by a law firm to submit  
 583:6 those letters?  
 583:7 A. No.

583:8 - 584:11

**Portier, Christopher 02-22-2019 (00:01:22)**

CP\_SS\_REDIRECT\_01.20

583:8 Q. Did those -- the preparation of  
 583:9 those letters and the statements you made,  
 583:10 did that take a lot of time?  
 583:11 A. Yes, it did.  
 583:12 Q. Why did you do it then?  
 583:13 A. Because I was to some degree  
 583:14 very surprised when I took time to look very

583:15 carefully at the regulatory reviews for  
583:16 glyphosate. I had spent my entire career  
583:17 working towards ways in which we evaluate and  
583:18 understand these types of data for making  
583:19 decisions, and many of the things that we had  
583:20 spent years working out that were part of the  
583:21 guidelines for both the agencies, EFSA and  
583:22 EPA, that they should have been following  
583:23 weren't being followed.

583:24 And, you know, when you spend  
583:25 your career trying to develop these things  
584:1 and all of a sudden you're finding out nobody  
584:2 is paying attention or using the things that  
584:3 are in their guidelines that make good solid,  
584:4 scientific sense, you're -- you want to fix  
584:5 it. You want to correct it.

584:6 And so that's why I took the  
584:7 time and effort to do it. I just could not  
584:8 believe that all of that effort that went  
584:9 into developing these guidelines and doing  
584:10 the science that led us to these excellent  
584:11 guidelines was being ignored.

586:18 - 587:17

**Portier, Christopher 02-22-2019 (00:00:47)**

CP\_SS\_REDIRECT\_01.18

586:18 Q. I want to go back to this  
586:19 letter that was brought in on -- on  
586:20 cross-examination. It was Exhibit 1456. And  
586:21 this is a letter that you wrote to the EPA.  
586:22 Do you recall talking about  
586:23 this?

586:24 A. Yes.

586:25 Q. And this is from 2016, right?

587:1 A. Correct.

587:2 Q. So over two years ago?

587:3 A. Yes.

587:4 Q. All right. And back here there  
587:5 was a series of lines that were read, and  
587:6 I -- he read them but didn't ask you any  
587:7 questions about them, so I want to now ask  
587:8 you those questions.

587:9 Okay?

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587:10 A. Okay.

587:11 Q. Specifically in the summary

587:12 section here, he read to you some lines, "So

587:13 is causality plausible here? Yes,

587:14 absolutely. Is it demonstrated? No, clearly

587:15 not."

587:16 Do you see that?

587:17 A. Yes.

587:21 - 588:16

**Portier, Christopher 02-22-2019 (00:00:50)**

CP\_SS\_REDIRECT\_01.17

587:21 Q. All right. So let's take a

587:22 quick step back here.

587:23 What are you saying here in

587:24 this summary statement when you look at the

587:25 whole paragraph?

588:1 And I can hand you a copy, if

588:2 you'd like, to look at it.

588:3 A. I'm sure I have a copy around

588:4 here.

588:5 Q. It's Exhibit 1456.

588:6 A. That's it.

588:7 Q. There it is.

588:8 We're on page 7 on the bottom.

588:9 Page 7.

588:10 A. Summary. Okay.

588:11 Q. Okay. So -- so they read

588:12 this -- this portion to you and it says, "Is

588:13 it demonstrated? No, clearly not."

588:14 Can you explain what you meant

588:15 when you wrote that, and what should we

588:16 understand from what you're saying here?

588:19 - 590:12

**Portier, Christopher 02-22-2019 (00:01:53)**

CP\_SS\_REDIRECT\_01.18

588:19 THE WITNESS: So I am

588:20 specifically responding to conclusions

588:21 that EPA made. One statement they

588:22 said was, "The association between

588:23 glyphosate exposure and risk of NHL

588:24 cannot be determined based on the

588:25 available data."

589:1 I was pointing out that this

589:2 is -- failed to use their 2005

589:3 guidelines. Their guidelines talk  
589:4 about consistency and significance and  
589:5 nonspecificity, temporality, et  
589:6 cetera. They never discussed any of  
589:7 that in what they had done.  
589:8 And so in answer to their  
589:9 statement about causality, I went on  
589:10 and answered, is it plausible, yes,  
589:11 absolutely.  
589:12 QUESTIONS BY MR. WISNER:  
589:13 Q. And what do you mean when you  
589:14 say it's plausible?  
589:15 A. So an example that's been given  
589:16 multiple times in looking at epidemiology  
589:17 data is the idea of reduction in birds in  
589:18 Europe during the 1950s to 2000 and linking  
589:19 it to the reduction in the number of storks.  
589:20 And there's the old, stoled wive's tales that  
589:21 babies come from storks being delivered them.  
589:22 So as the number of storks go down, the  
589:23 number of babies being delivered down -- goes  
589:24 down and the birth rate goes down. That is  
589:25 an association.  
590:1 But causality is not plausible  
590:2 in that situation because of the fact that  
590:3 children are not delivered by storks. So it  
590:4 makes no sense.  
590:5 Here, there is nothing that  
590:6 would inherently tell you this makes no  
590:7 sense. The human evidence is showing the  
590:8 association. The animal evidence, the  
590:9 mechanistic evidence, nothing in that says  
590:10 this makes no sense.  
590:11 And so causality is clearly  
590:12 plausible here. That's what it means.  
599:23 **Portier, Christopher 02-22-2019 (00:00:22)**  
599:23 And here is that -- one of  
599:24 those charts that we put together on direct.  
599:25 Do you recall that?  
600:1 A. Yes.

599:23 - 600:11

CP\_SS\_REDIRECT\_01.20

Page/Line	Source	ID
600:14 - 600:22	<p>600:2 Q. And this is reflecting data in 600:3 in vitro human cells? 600:4 A. Correct. 600:5 Q. And this is another part of 600:6 that data? 600:7 A. Correct. 600:8 Q. And Mr. Schmidt cross-examined 600:9 you for a couple of hours. 600:10 Did he ask you a single 600:11 question about any one of these studies? <b>Portier, Christopher 02-22-2019 (00:00:09)</b></p>	CP_SS_REDIRECT_01.22
600:25 - 601:5	<p>600:14 THE WITNESS: The specifics, 600:15 no. 600:16 QUESTIONS BY MR. WISNER: 600:17 Q. I mean, he showed you this 600:18 chart, right? 600:19 A. Correct. 600:20 Q. And when he showed you this 600:21 chart, did he show you anything that 600:22 challenged your assessment of these data? <b>Portier, Christopher 02-22-2019 (00:00:11)</b> 600:25 THE WITNESS: No. 601:1 QUESTIONS BY MR. WISNER: 601:2 Q. And if we look at the oxidative 601:3 stress data, again, did Mr. Schmidt ask you a 601:4 single question challenging all of these 601:5 positive findings?</p>	CP_SS_REDIRECT_01.23
601:8 - 601:8	<p><b>Portier, Christopher 02-22-2019 (00:00:00)</b> 601:8 THE WITNESS: No.</p>	CP_SS_REDIRECT_01.24
601:10 - 601:15	<p><b>Portier, Christopher 02-22-2019 (00:00:10)</b> 601:10 Q. All right. I want to go to the 601:11 lymphoma findings. And, you know, this is 601:12 the mouse chart that we talked about on 601:13 direct, and it was talked about on cross, 601:14 right? 601:15 A. Yes.</p>	CP_SS_REDIRECT_01.21
601:25 - 602:4	<p><b>Portier, Christopher 02-22-2019 (00:00:12)</b> 601:25 Q. And there was a question that 602:1 was asked you about whether or not kidney 602:2 tumors are predictive of human lymphoma.</p>	CP_SS_REDIRECT_01.22

602:8 - 603:6	<p>602:3 Is that an appropriate question  602:4 when you're looking at an animal study?  <b>Portier, Christopher 02-22-2019 (00:00:55)</b>  602:8 THE WITNESS: It -- it's --  602:9 it's a question you would  602:10 ask that's -- it's something you would  602:11 think about, but you wouldn't  602:12 necessarily require it. In fact, you  602:13 would not require that the tumors  602:14 you're looking at in the mouse matched  602:15 the tumors you were worried about in  602:16 humans. You would not require that  602:17 because the evidence is not there to  602:18 suggest that there is concordance.  602:19 Even when you look at mice  602:20 males to females, historically there's  602:21 not a great deal of concordance. Mice  602:22 to rats, historically, there's not a  602:23 great deal of concordance in the  602:24 sites. And mice and rats to humans,  602:25 there's not a great deal of  603:1 concordance in the sites.  603:2 The concordance is if you see  603:3 cancers in the rats and mice -- if you  603:4 see cancers in humans, you're almost  603:5 certain to see them in rats and mice.  603:6 In fact, you are certain.</p>	CP_SS_REDIRECT_01.33
603:10 - 606:3	<p><b>Portier, Christopher 02-22-2019 (00:02:19)</b>  603:10 Do we have concordance here  603:11 between lymphomas in mice and lymphomas in  603:12 humans?  603:13 A. In that regard, you do have  603:14 concordance.  603:15 Q. So let's talk about that  603:16 lymphoma data.  603:17 Do you recall there was a chart  603:18 that was put together with you and defense  603:19 counsel?  603:20 A. Yes.  603:21 Q. And I've made a photocopy of</p>	CP_SS_REDIRECT_01.33

603:22 it, so this is not the original. The  
603:23 original was 1675. And so we're going to  
603:24 call this 1675 B.  
603:25 Okay?  
604:1 A. Okay.  
604:2 Q. And as you can see, it's  
604:3 slightly cut off here because of the  
604:4 photocopying.  
604:5 Do you see that, sir?  
604:6 A. Yes.  
604:7 Q. All right. But can you still  
604:8 read what those are referring to?  
604:9 A. Yes.  
604:10 Q. Okay. So at the beginning of  
604:11 this chart, you started off with this premise  
604:12 of less than .05.  
604:13 Do you recall that?  
604:14 A. Yes.  
604:15 Q. Is that a valid thing to start  
604:16 off with?  
604:17 A. Not in my opinion.  
604:18 Q. Why is that?  
604:19 A. Because it's taking a very  
604:20 complicated picture and turning it from  
604:21 continuous evaluations of P values that give  
604:22 you some degree of information of the  
604:23 strength in each study to zero -- to yes or  
604:24 no. And so you've -- you've taken each study  
604:25 and thrown away all of the information you  
605:1 have for the study in favor of yes or no.  
605:2 Q. So here when it says .05,  
605:3 that's equivalent to a 95 percent confidence  
605:4 interval?  
605:5 A. Correct.  
605:6 Q. Okay. What if we -- we get a  
605:7 little more wild and go up to 90 percent,  
605:8 okay?  
605:9 Is that an analysis that you  
605:10 did?  
605:11 A. Yes.

605:12 Q. Okay. And what P value do you  
 605:13 get from that?  
 605:14 A. .1.  
 605:15 Q. Okay. So it would be less than  
 605:16 .1; is that right?  
 605:17 A. Correct.  
 605:18 Q. And that's 90 percent?  
 605:19 A. Correct.  
 605:20 Q. All right. And when you  
 605:21 characterize point -- something between .05  
 605:22 and .1, what do you call that?  
 605:23 A. I call it marginally  
 605:24 significant, and so does the literature.  
 605:25 Q. Okay. And so when we go to  
 606:1 your chart here, the marginal -- you specify  
 606:2 that exact point with your pluses.  
 606:3 A. Yes.

606:12 - 607:9

**Portier, Christopher 02-22-2019 (00:01:02)**

CP\_SS\_REDIRECT\_01.24

606:12 Q. -- two -- when you have two  
 606:13 pluses, what does that mean?  
 606:14 A. That means it falls inside the  
 606:15 95 percent confidence bound but not the most  
 606:16 extreme one, which would be 99 percent.  
 606:17 Q. And so like, for example, in  
 606:18 Wood, with lymphoma you have three pluses.  
 606:19 What does that mean?  
 606:20 A. The P value is less than .01.  
 606:21 Q. Okay. And so if we go back to  
 606:22 this chart, this modified version of  
 606:23 Exhibit 1675 B, first of all, did you do a  
 606:24 90 percent significance analysis for the  
 606:25 pairwise?  
 607:1 A. I did the pairwise evaluations.  
 607:2 I've only reported the 5 percent ones simply  
 607:3 as information for the reader.  
 607:4 Q. Okay. So I'm going to put not  
 607:5 reported, or NR, for those three. Okay?  
 607:6 And we're sticking to orange  
 607:7 here because it reflects the 90 percent, all  
 607:8 right?

Page/Line	Source	ID
607:10 - 607:14	607:9 A. Okay. <b>Portier, Christopher 02-22-2019 (00:00:09)</b>	CP_SS_REDIRECT_01.25
607:20 - 607:24	607:10 Q. So then if we go to the 607:11 90 percent instead of the 95 percent, 607:12 Knezevich and Hogan, does that change from no 607:13 to yes? 607:14 A. Correct. It changes to yes.	CP_SS_REDIRECT_01.26
607:25 - 608:6	607:20 Q. Yeah, we're talking about 607:21 lymphoma here. 607:22 A. Oh, lymphoma. I'm sorry. 607:23 Q. Does that change? 607:24 A. No. <b>Portier, Christopher 02-22-2019 (00:00:18)</b>	CP_SS_REDIRECT_01.27
609:5 - 609:13	607:25 Q. Okay. Does Atkinson change? 608:1 A. Yes, it does. 608:2 I should look at my chart. 608:3 Q. Well, Sugimoto is already yes. 608:4 What about Kumar? Does Kumar 608:5 change? 608:6 A. Yes. <b>Portier, Christopher 02-22-2019 (00:00:28)</b>	CP_SS_REDIRECT_01.28
609:19 - 609:21	609:5 Q. And so going back to the chart 609:6 that started this whole thing, do you specify 609:7 for each one of these tumors, those that are 609:8 99, 95 and 90 percent significant? 609:9 A. I specify for each of these 609:10 tumors the P value itself. And so you can 609:11 make the breakdown into each of these 609:12 categories if you'd like, but I specify the P 609:13 value in every single case.	CP_SS_REDIRECT_01.29
609:24 - 610:8	<b>Portier, Christopher 02-22-2019 (00:00:05)</b> 609:19 If you have a significance in 609:20 the pairwise or the trend, how does that work 609:21 when you analyze animal data? <b>Portier, Christopher 02-22-2019 (00:00:22)</b> 609:24 THE WITNESS: So by most of the 609:25 guidelines that are out there, if you 610:1 see either a trend or a pairwise 610:2 positive finding, you consider it as a	CP_SS_REDIRECT_01.30

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610:3 positive finding in the context of the  
610:4 study you're looking at.

610:5 In my evaluation, I relied on  
610:6 the trend test for my overall  
610:7 interpretation of the data, not on the  
610:8 pairwise comparisons.

613:2 - 613:7

**Portier, Christopher 02-22-2019 (00:00:16)**

CP\_SS\_REDIRECT\_01.31

613:2 Q. Standing here today, 2019, in  
613:3 your professional and expert opinion, do you  
613:4 believe that the use of glyphosate out in the  
613:5 real world can lead to people getting  
613:6 non-Hodgkin's lymphoma?  
613:7 A. Yes.

**Total Time = 00:20:24**

# Portier Day 2 DCC 0228 FINAL PLAYED

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PORTIER, CHRISTOPHER 2019-02-22\_SS

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Total Time 00:06:01



Page/Line	Source	ID
613:19 - 614:7	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:20)</b></p> <p>613:19 Q. Doctor, just a few concluding 613:20 questions. 613:21 Do you have in front of you 613:22 Exhibit 1456? 613:23 A. Yes, I do. 613:24 Q. These are your comments to the 613:25 EPA in 2016 that you just testified about on 614:1 redirect? 614:2 A. Yes, they are. 614:3 Q. Would you mind going with me to 614:4 page 7, which you testified about? 614:5 A. Okay.</p>	M22.2
614:8 - 614:12	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</b></p> <p>614:8 You talked with the plaintiff 614:9 attorney about your views on EPA and their 614:10 conclusion. 614:11 Do you remember that? 614:12 A. Yes.</p>	1456.1.2
614:13 - 615:10	<p><b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)</b></p> <p>614:13 Q. If we look at page 7 of the 614:14 document, you specifically focused on this 614:15 paragraph, this summary paragraph. 614:16 Do you remember talking about 614:17 that with the plaintiff attorney just now? 614:18 A. Yes. 614:19 Q. And you indicated that you were 614:20 responding to the conclusion by the EPA that 614:21 the association between glyphosate exposure 614:22 and risk of NHL cannot be determined based on 614:23 the available data. 614:24 Do you see that? 614:25 A. Correct. 615:1 Q. That's what you were objecting 615:2 to, correct? 615:3 A. It appears that's what I was 615:4 objecting to, yes. 615:5 Q. And they've not changed that</p>	clear M22.14

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615:20 - 616:5	<p>615:6 opinion to this date, correct?  615:7 A. Again, I don't know. I haven't  615:8 read the specifics on what their current  615:9 statement is with regard to the epidemiology  615:10 data.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</b></p>	M22.3
625:22 - 626:9	<p>615:20 Q. Okay. Now, when you were  615:21 making these comments to the EPA in  615:22 October 2014 -- '16, am I correct that you  615:23 had already agreed on that contract we talked  615:24 about with the plaintiff lawyers?  615:25 A. To provide them scientific  616:1 advice, yes.  616:2 Q. Yes.  616:3 And to be paid for that,  616:4 correct?  616:5 A. Correct.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</b></p>	M22.4
626:10 - 626:16	<p>625:22 well, you  625:23 have stated in your report in this case that  625:24 the meta-analysis done by Chang and Delzell  625:25 includes the same analysis as that done by  626:1 IARC and is an improvement over Schinasi and  626:2 Lyon, so I will focus my comments on using  626:3 the Chang and Delzell meta-analysis.  626:4 Do you recall saying that in  626:5 your report?  626:6 A. Yes, I do.  626:7 Q. And you stand behind that  626:8 statement?  626:9 A. Yes, I do.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13)</b></p>	M22.5
628:14 - 628:18	<p>626:10 Q. Last line of questions, sir.  626:11 Let's talk briefly about the most recent  626:12 epidemiological study, the Andreotti study.  626:13 Do you have that in front of  626:14 you? It's Exhibit 550.  626:15 A. I'm sure I have it somewhere  626:16 here.  <b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</b></p>	550.1 M22.6

Page/Line	Source	ID
628:14 Q. You made a point about 628:15 imputation of data in this study, correct?		
628:16 A. Correct.		
628:17 Q. Let's look at the fourth page		550.4
628:18 of the study, page 512.		
628:19 - 629:15	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:55)</b>	M22.7
628:19 Do you recall that they 628:20 actually conducted an analysis to see whether 628:21 imputation affected their results?		
628:22 A. They did some other analyses 628:23 that they argued told them whether imputation 628:24 affected their results.		
628:25 Q. Let's look at what we're 629:1 talking about.		
629:2 Do you see where it says in the 629:3 left-hand column, "To evaluate the impact of 629:4 using imputed exposure data for participants 629:5 who did not complete the follow-up 629:6 questionnaire, we limited the analysis to 629:7 34,698 participants who completed both 629:8 questionnaires, reducing the total number of 629:9 cases to 4,699"?		550.4.3
629:10 Did I read that correctly?		
629:11 A. You read that correctly.		
629:12 Q. Do they then report that when 629:13 they did that analysis, glyphosate use was 629:14 not associated with NHL?		
629:15 A. They didn't say that, yes.		clear
629:18 - 630:3	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:19)</b>	M22.8
629:18 Are you aware that just this 629:19 year they had a further publication 629:20 addressing this issue, just in the last month 629:21 or so?		
629:22 A. Are you talking about a 629:23 correspondence?		
629:24 Q. Yes.		
629:25 A. Yes.		
630:1 Q. And you've reviewed that?		
630:2 A. I have looked at it, yes, I 630:3 have.		

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632:11 - 633:5	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:48)</b>	M22.9 1031.1.1
	632:11 Q. It's Exhibit 1031. Let me give 632:12 you a copy, sir.	
	632:13 A. Thank you very much.	
	632:14 Q. And do you see that this paper 632:15 includes lead author Andreotti?	
	632:16 Do you see that?	
	632:17 A. Yes, I do.	
	632:18 Q. Do you see it's published in 632:19 the Journal of the National Cancer Institute, 632:20 2019?	
	632:21 A. I see that, yes.	
	632:22 Q. And do you understand that this 632:23 relates to this imputation question you 632:24 raised that we've been discussing?	
	632:25 A. It partially -- it relates to 633:1 other things, but it relates to the comments 633:2 sent by Dr. Shepherd and Dr. Shaffer.	
	633:3 Q. Which touched on imputation, 633:4 correct?	
	633:5 A. Correct.	
633:11 - 634:7	<b>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:47)</b>	M22.10 1031.1.2
	633:11 Do you see where they say, "The 633:12 patterns of risk are similar for those who 633:13 completed the follow-up questionnaire, i.e., 633:14 self-reported use, yes/no, and those who did 633:15 not, i.e., imputed use, yes/no."	
	633:16 Do you see that?	
	633:17 A. I see that, yes.	
	633:18 Q. And for that group they report 633:19 no statistically significant interaction 633:20 between glyphosate use and completion of the 633:21 follow-up questionnaire, correct?	1031.1.3
	633:22 A. I see that. That is correct.	
	633:23 Q. And above that they say -- they 633:24 talk about imputation.	1031.1.4
	633:25 Do you see that reference to 634:1 imputing exposure?	
	634:2 A. Yes.	
	634:3 Q. And then they say, "Although we	

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634:4 agree that this method could theoretically

634:5 bias risk estimates towards the null" --

634:6 Did I read that correctly?

634:7 A. You read that correctly.

634:8 - 634:18

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:15)**

M22.11

634:8 Q. And I understand that to be

634:9 similar to the point you were making, is that

634:10 correct, that it could bias results towards

634:11 the null?

634:12 A. No, the point that --

634:13 Q. Okay. Then I'll move on if

634:14 that's not the point you were making.

634:15 A. I'm sorry. The point that

634:16 Sheppard and Shaffer were making were a

634:17 different reason why this would go to the

634:18 null.

634:19 - 635:7

**PORTIER, CHRISTOPHER 2019-02-22\_SS (00:00:27)**

M22.12

634:19 Q. Got it. They then say -- and

634:20 this is the part I want to read to you.

634:21 "Based on sensitivity analyses" -- do you see

634:22 they're conducting additional analyzing?

634:23 A. Correct.

634:24 Q. -- "that we conducted and

634:25 reported in the manuscript and describe more

635:1 fully below, we demonstrate that our

635:2 imputation likely did not materially impact

635:3 risk estimates."

635:4 Did I read that correctly?

635:5 A. You read that correctly.

635:6 MR. SCHMIDT: Thank you,

635:7 Doctor. That's all I have.

1031.1.5

**Total Time = 00:06:01**

**Documents Shown**

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# REDIRECT\_01 - PORTIER\_RE- REDIRECT\_01 FINAL PLAYED

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Portier, Christopher 02-22-2019

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Total Time 00:01:51



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638:14 - 638:21	<p><b>Portier, Christopher 02-22-2019 (00:00:15)</b>  638:14 On his cross-examination, he  638:15 showed you the AHS study; is that right?  638:16 A. Yes.  638:17 Q. This is Exhibit 550.  638:18 And he asked you a question  638:19 about the credibility of the journal.  638:20 Do you recall that?  638:21 A. Yes, I do.</p>	CP_SS_RE.1
639:7 - 639:9	<p><b>Portier, Christopher 02-22-2019 (00:00:04)</b>  639:7 Q. Okay. He showed you this paper  639:8 on cross, right?  639:9 A. Yes, he did.</p>	CP_SS_RE.2
639:19 - 640:10	<p><b>Portier, Christopher 02-22-2019 (00:00:42)</b>  639:19 Q. Okay. So in this article, it  639:20 says right here, "Conclusion. In this large  639:21 prospective cohort study, no association was  639:22 apparent between glyphosate and any solid  639:23 tumors or lymphoid malignancies overall,  639:24 including NHL and its subtypes."  639:25 Do you see that?  640:1 A. Yes, I do.  640:2 Q. All right. Subtypes, what does  640:3 that refer to?  640:4 A. The various and different types  640:5 of lymphomas that make up the category of  640:6 non-Hodgkin's lymphoma.  640:7 Q. Okay. So if we go to Table 3  640:8 in the study, it lists out the various  640:9 results for these subtypes, is that right,  640:10 5-year and 20-year lag?</p>	CP_SS_RE.3
640:18 - 641:1	<p><b>Portier, Christopher 02-22-2019 (00:00:21)</b>  640:18 THE WITNESS: Yes, it does show  640:19 5-year and 20-year lags.  640:20 QUESTIONS BY MR. WISNER:  640:21 Q. All right. Looking at the  640:22 results here for non-Hodgkin's lymphoma  640:23 T-cell on the 20-year lag, and you see right  640:24 here, 2.97, 1.20 to 7.31.  640:25 Do you see that?</p>	CP_SS_RE.4

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641:5 - 641:7	641:1 A. Yes. <b>Portier, Christopher 02-22-2019 (00:00:05)</b> 641:5 QUESTIONS BY MR. WISNER:	CP_SS_RE.5
641:6 - 641:7	641:6 Q. That ratio of almost 3, is that 641:7 statistically significant?	
641:9 - 641:15	<b>Portier, Christopher 02-22-2019 (00:00:14)</b> 641:9 THE WITNESS: Yes, it is. 641:10 QUESTIONS BY MR. WISNER: 641:11 Q. For a subtype? 641:12 A. Yes, it is. 641:13 Q. So when it says right here that 641:14 there's no observed association with any 641:15 subtype, is that even factually true?	CP_SS_RE.6
641:18 - 641:20	<b>Portier, Christopher 02-22-2019 (00:00:04)</b> 641:18 THE WITNESS: No, it's not. 641:19 QUESTIONS BY MR. WISNER: 641:20 Q. Sir, is this a good study?	CP_SS_RE.7
641:23 - 641:23	<b>Portier, Christopher 02-22-2019 (00:00:00)</b> 641:23 THE WITNESS: No.	CP_SS_RE.8
641:25 - 641:25	<b>Portier, Christopher 02-22-2019 (00:00:01)</b> 641:25 THE WITNESS: It's not.	CP_SS_RE.9

**Total Time = 00:01:51**

# PORTIER\_RE-RECROSS FINAL PLAYED

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Portier, Christopher 02-22-2019

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**[REDACTED]**  
Total Time 00:00:22

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642:8 - 642:24

**Portier, Christopher 02-22-2019 (00:00:21)**

CP\_SS\_RE\_RECROSS.1

642:8 Q. Doctor, can I

642:9 ask a follow-up question on the Andreotti

642:10 study?

642:11 A. Yes, you may.

642:12 Q. You were asked a question about

642:13 non-Hodgkin's lymphoma T-cell.

642:14 Do you remember that?

642:15 A. Yes.

642:16 Q. Do you know if that has

642:17 anything to do with the facts in the

642:18 plaintiff's case -- of the plaintiff in this

642:19 case?

642:20 A. No, I do not.

642:21 MR. SCHMIDT: Thank you,

642:22 Doctor.

642:23 THE WITNESS: If you're talking

642:24 about the specific subtypes, yeah.

  
Total Time = 00:00:22