

PORTIER_DAY1_SS_PA_01 FINAL PLAYED

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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

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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

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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

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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.

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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

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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

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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.

26:6 - 27:17

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.

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CP1_SS_01.8

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

27:16 easily.

27:17 A. Okay.

29:7 - 30:15

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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

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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

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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

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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

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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

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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>Portier, Christopher 02-21-2019 (00:00:32)</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>Portier, Christopher 02-21-2019 (00:00:18)</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>Portier, Christopher 02-21-2019 (00:01:03)</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>Portier, Christopher 02-21-2019 (00:02:27)</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

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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

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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

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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

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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

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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

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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

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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

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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

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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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CP1_SS_01.28

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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CP1_SS_01.29

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

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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.

117:19 - 127:16 **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.

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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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

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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>Portier, Christopher 02-21-2019 (00:00:06)</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. 132:1 What kind of mouse was that?</p>	CP1_SS_01.36
132:4 - 132:11	<p>Portier, Christopher 02-21-2019 (00:00:10)</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>Portier, Christopher 02-21-2019 (00:01:42)</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

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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	Portier, Christopher 02-21-2019 (00:00:39)	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	Portier, Christopher 02-21-2019 (00:00:09)	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	Portier, Christopher 02-21-2019 (00:00:28)	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

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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

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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

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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

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

155:14 A. Correct.

155:18 - 155:24

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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

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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	Portier, Christopher 02-21-2019 (00:00:01)	CP1_SS_01.50
	<p>175:19 Did you want to say something 175:20 else, sir?</p>	
175:25 - 176:2	Portier, Christopher 02-21-2019 (00:00:05)	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	Portier, Christopher 02-21-2019 (00:00:07)	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	Portier, Christopher 02-21-2019 (00:05:49)	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

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	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	Portier, Christopher 02-21-2019 (00:00:02) 196:16 THE WITNESS: Santovito is 196:17 human lymphocytes.	CP1_SS_01.54
196:25 - 197:10	Portier, Christopher 02-21-2019 (00:00:19) 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	Portier, Christopher 02-21-2019 (00:00:19) 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

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

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

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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 H₂O, 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

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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 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

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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

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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

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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

Portier, Christopher 02-22-2019

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	Portier, Christopher 02-22-2019 (00:00:03)	CP2_SS_01.10
281:23 Q. And then we have the recent 281:24 human genotox data?		
282:2 - 282:6	Portier, Christopher 02-22-2019 (00:00:06)	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	Portier, Christopher 02-22-2019 (00:01:36)	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>Portier, Christopher 02-22-2019 (00:00:07) 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>Portier, Christopher 02-22-2019 (00:00:30) 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>Portier, Christopher 02-22-2019 (00:01:48) 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

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

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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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</p>	M20.5
308:7 - 308:10	<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> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</p>	M20.6
308:13 - 308:19	<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> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</p>	M20.7
308:23 - 309:14	<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> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26)</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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04)	M20.10
	311:14 Q. I've put in front of you Trial	1501.1
	311:15 Exhibit 1501.	
311:21 - 312:14	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:43)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:47) 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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35) 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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)	M20.16
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)	M20.17
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:20)	M20.18
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:16)	M20.19
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:53)	M20.20
	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
321:22 - 322:3	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23) 321:22 Q. Do you mind looking at page 6 321:23 of the NTP study from 1992?	M20.21
	321:24 A. I'm looking at it.	1098.8
	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
322:7 - 322:8	322:3 A. I see where it says that, yes. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08) 322:7 Q. On page 36 of this study, down 322:8 near the bottom, do you see where they say,	M20.22 1098.38 - 1098.38.2
322:9 - 323:21	PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:02) 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. 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,	M20.23
		clear

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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:53)	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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:05)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:29) 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.</p>	M20.29
331:24 - 332:11	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:28) 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.</p>	M20.30
333:21 - 334:11	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26) 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

Page/Line

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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

Page/Line	Source	ID
338:10 - 339:4	338:9 you're correct. I stand corrected. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:37)	M20.34
	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?	
	338:14 A. Some wording along those lines, 338:15 that's correct.	
	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?	1640.1.5
	338:20 A. Yes, I do.	
	338:21 Q. EFSA's conclusion that 338:22 glyphosate is unlikely to pose a carcinogenic 338:23 hazard to humans?	
	338:24 A. That is correct.	
	338:25 Q. And you were obviously writing 339:1 because you disagreed with that, right?	clear
	339:2 A. We disagreed with -- we 339:3 disagreed with the scientific way in which 339:4 they arrived at that decision.	
339:5 - 339:10	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)	M20.35
	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.	
339:11 - 339:22	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:16)	M20.36
	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?	
	339:15 A. I believe they should have 339:16 classified it as 2B or 2A, absolutely, yes.	
	339:17 Q. Okay.	
	339:18 A. I don't know if we say that in 339:19 here.	
	339:20 Q. And they wrote back to you,	

Page/Line	Source	ID
	339:21 right?	
	339:22 A. They did write back to me.	
341:2 - 341:25	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:48)	M20.37
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)	M20.38
	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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)</p>	M20.39
343:12 - 343:13	<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> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)</p>	M20.40 1639.16.7
343:14 - 343:20	<p>343:12 Q. They go on to say in the next 343:13 paragraph, "As reported in the EFSA PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25) 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15) 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10) 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. 346:1 Do you recognize that as 346:2 Exhibit 1642?</p>	M20.43
346:3 - 346:3	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:01)</p>	M20.44

Page/Line	Source	ID
346:4 - 346:9	<p>346:3 A. Yes, I do.</p> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:32)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)</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

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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:07)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:14)	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

clear

371:20 with.

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

1657.1

375:25 article that you published with Dr. Resnik in

1657.1.6

376:1 2005.

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

1657.2

376:7 this document -- this is your publication,

376:8 correct?

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

1657.2.1

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"?

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

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

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

PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08)

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

PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:15)

M20.61

378:6 Q. You would need to look at the

Page/Line	Source	ID
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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:19)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13)</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. PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:20)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:59)</p> <p>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
	387:2 Q. As to the rats, correct?	
	387:3 A. That is correct.	
387:4 - 387:17	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:26)	M20.67
	387:4 Q. And you understand that this is	
	387:5 a case involving non-Hodgkin's lymphoma,	
	387:6 correct?	
	387:7 A. Correct, but there is no	
	387:8 evidence in the literature to suggest that	
	387:9 you must see the same results in laboratory	
	387:10 animals that you see in humans for there to	
	387:11 be a prediction --	
	387:12 Q. My question --	
	387:13 A. -- from the animal to human.	
	387:14 I know what your question was.	
	387:15 Q. My question was simply -- I'm	
	387:16 not -- let me just ask it again to make sure	
	387:17 I understand your answer.	
387:18 - 387:20	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)	M20.68
	387:18 You understand that this case	
	387:19 involves non-Hodgkin's lymphoma, right?	
	387:20 A. Yes, I do.	
388:7 - 388:20	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:33)	M20.69
	388:7 Q. With the exception of growth	
	388:8 and a few nonmalignant tumors, none of the	
	388:9 rat studies showed any effect?	
	388:10 A. No.	
	388:11 Q. Okay.	
	388:12 A. It's the nonmalignant tumors	
	388:13 I'm disagreeing with.	
	388:14 Q. Do you recall having a	
	388:15 publication in a Swiss National Science	
	388:16 Foundation called Horizons?	
	388:17 A. Yes, I did. It's a National	
	388:18 Science Foundation magazine, yes.	
	388:19 Q. And that was in 2016?	
	388:20 A. Yes, it was.	
390:3 - 390:6	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)	M20.70
	390:3 Do you recognize this as that	
	390:4 article we've been discussing, what I've	
	390:5 marked as Exhibit 1667?	1667.1

Page/Line	Source	ID
391:2 - 391:7	<p>390:6 A. Yes, I do.</p> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:12)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</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? 392:16 A. You did read it correctly.</p>	M20.72
392:17 - 392:20	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:06)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)	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?	
394:5 - 395:2	394:4 A. Yes. PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:40)	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.

Page/Line	Source	ID
408:15 - 408:18	<p>408:14 Q. Do you recall giving testimony PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:08) 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? PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21) 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13) 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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:44) 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

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425:15 - 425:24	<p>425:14 your chart and no pairwise notation? PORTIER, CHRISTOPHER 2019-02-22_PIP (00:00:28) 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>PORTIER, CHRISTOPHER 2019-02-22_PIP (00:01:11) 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.

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	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:43)	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

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457:20 - 458:6	<p>456:3 oxidative stress. 456:4 Is that accurate? 456:5 A. That is accurate. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:17)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:53)</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

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

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

1066.1.4

clear

Page/Line	Source	ID
	462:12 say later in the article.	1066.9
462:14 - 464:25	462:13 Could you flip to page 994 of PORTIER, CHRISTOPHER 2019-02-22_SS (00:02:25)	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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:12)</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. 481:5 Q. And if you would, look with me 481:6 at page 353, please. And tell me when you're</p>	M20.131
481:7 - 481:21	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:09)</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 --
 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.

878.1

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.

487:3 Do you see that?

487:4 A. I see that.

487:5 Q. That was not statistically
 487:6 significant, correct?

878.1.9

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

Page/Line	Source	ID
494:20	A. G.	
494:21	Q. G, as in gopher.	
494:22	A. Yes.	
494:24 - 495:11	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:30)	M20.140
494:24	That would be trying to adjust	
494:25	for potential confounders, correct?	
495:1	A. Well, what they're doing there	clear
495:2	is comparing it to F, and so they're looking	
495:3	at the degree to which other pesticides	
495:4	reduce the relative risk that you see for	
495:5	glyphosate.	
495:6	The interpretation there is not	
495:7	that the glyphosate is no longer important.	
495:8	The interpretation there is that some of the	
495:9	relative risk you see for glyphosate is	
495:10	associated with these other pesticides, so	
495:11	they are confounded.	
495:12 - 496:9	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:37)	M20.141
495:12	Q. Okay. And I think we're saying	
495:13	the same thing, but I want to make sure I	
495:14	understand it in lay terms.	
495:15	In analysis G, most adjusted,	
495:16	what they're trying to do is take out the	
495:17	effect of potential pesticide confounders,	
495:18	correct?	
495:19	A. Or measure the effect of	
495:20	pesticide confounders on the effect they saw	
495:21	for glyphosate, without the confounders in	
495:22	there.	
495:23	Q. Okay. Exactly.	
495:24	Let's go to the next one. The	878.1.14
495:25	next one is Hardell and Eriksson.	
496:1	Do you see that?	
496:2	A. Yes, I do.	
496:3	Q. And they report two results,	
496:4	right?	
496:5	A. Correct.	
496:6	Q. A regular -- a first result and	
496:7	a most adjusted result.	
496:8	Do you see that?	

Page/Line	Source	ID
496:17 - 497:8	<p>496:9 A. Yes, I do.</p> <p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:24)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:30)</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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:40)</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."

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

528.1.3

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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:32)</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?</p>	
	<p>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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:35)</p>	M20.153

Page/Line	Source	ID
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:05)	M20.155
	519:20 There is a Bradford Hill	
	519:21 viewpoints comment, yes.	
520:6 - 521:19	PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:21)	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		1102.21.13
520:20 on 422, second paragraph?		
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		1102.21.14
521:7 the left-hand side, still the same page, the		
521:8 last paragraph, "overall evaluation."		
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 PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:03)		M20.157
521:20 Q. Let's go to the next page,		1102.22
521:21 please, of this study.		
521:22 - 522:9 PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25)		M20.158
521:22 Do you see where they talk		
521:23 about the Bradford Hill criteria in the last		1102.22.4
521:24 paragraph before the discussion?		
521:25 A. I see there's a discussion		
522:1 there, yes.		
522:2 Q. And they state, "In summary,		1102.22.5
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		

Page/Line	Source	ID
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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:25)</p> <p>522:10 Q. And those are the same Bradford</p> <p>522:11 Hill viewpoints that you discussed with the</p> <p>522:12 plaintiff's attorney, correct?</p> <p>522:13 A. Not exactly. Again, I'm closer</p> <p>522:14 to the EPA interpretation of Bradford Hill</p> <p>522:15 and how they use it than what Bradford Hill</p> <p>522:16 himself wrote.</p> <p>522:17 I'm not sure how they were</p> <p>522:18 using it here in absolute certainty, so I can</p> <p>522:19 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:22)</p> <p>523:8 Q. Okay. Let me show you one more</p> <p>523:9 thing in this article. I think I had stopped</p> <p>523:10 before we looked at the second sentence in</p> <p>523:11 this paragraph.</p> <p>523:12 Do you see their conclusion?</p> <p>523:13 "Thus, on balance, the existing</p> <p>523:14 epidemiological evidence does not favor a</p> <p>523:15 causal effect of glyphosate on NHL, HL, MM,</p> <p>523:16 leukemia, or any subtype of these</p> <p>523:17 malignancies."</p> <p>523:18 Did I read that correct?</p> <p>523:19 A. Let me look. That is what it</p>	M20.160
523:20 - 525:1	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:05)</p> <p>523:20 says.</p> <p>523:21 Q. Okay. And this is a study that</p> <p>523:22 you reference in your report and on some of</p> <p>523:23 your slides, correct?</p> <p>523:24 A. That is correct.</p> <p>523:25 Q. You also referenced a more</p> <p>524:1 recent meta-analysis by the lead author</p> <p>524:2 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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:21)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:34) 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:04) 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>PORTIER, CHRISTOPHER 2019-02-22_SS (00:01:13)</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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:27) 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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:01) 539:20 Let's finish up with the 539:21 Andreotti study.	M20.171
542:14 - 542:25	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:42) 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

Documents Shown

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PORTIER_REDIRECT_01 FINAL PLAYED

Portier, Christopher 02-22-2019

Total Time 00:20:24



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549:1 - 549:10	<p>Portier, Christopher 02-22-2019 (00:00:22) 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?</p>	CP_SS_REDIRECT_01.1
549:13 - 549:13	<p>Portier, Christopher 02-22-2019 (00:00:00) 549:13 THE WITNESS: Yes, I have.</p>	CP_SS_REDIRECT_01.2
549:15 - 550:23	<p>Portier, Christopher 02-22-2019 (00:01:44) 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 --</p>	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> <p>Portier, Christopher 02-22-2019 (00:00:20)</p> <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.? 552:10 A. Correct.</p>	CP_SS_REDIRECT_01.4
557:24 - 558:2	<p>Portier, Christopher 02-22-2019 (00:00:03)</p> <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>	CP_SS_REDIRECT_01.5
558:21 - 559:11	<p>Portier, Christopher 02-22-2019 (00:00:36)</p> <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>	CP_SS_REDIRECT_01.6

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	Portier, Christopher 02-22-2019 (00:00:01)	CP_SS_REDIRECT_01.7
	559:14 THE WITNESS: No.	
559:16 - 559:21	Portier, Christopher 02-22-2019 (00:00:09)	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	Portier, Christopher 02-22-2019 (00:00:25)	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	Portier, Christopher 02-22-2019 (00:00:15)	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	Portier, Christopher 02-22-2019 (00:00:25)	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

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? Portier, Christopher 02-22-2019 (00:00:09)</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? Portier, Christopher 02-22-2019 (00:00:11) 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>Portier, Christopher 02-22-2019 (00:00:00) 601:8 THE WITNESS: No.</p>	CP_SS_REDIRECT_01.24
601:10 - 601:15	<p>Portier, Christopher 02-22-2019 (00:00:10) 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?</p>	CP_SS_REDIRECT_01.21
601:25 - 602:4	<p>601:15 A. Yes. Portier, Christopher 02-22-2019 (00:00:12) 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

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602:8 - 603:6

602:3 Is that an appropriate question
 602:4 when you're looking at an animal study?
Portier, Christopher 02-22-2019 (00:00:55)

CP_SS_REDIRECT_01.33

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.

603:10 - 606:3

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

CP_SS_REDIRECT_01.33

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

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?

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607:10 - 607:14	607:9 A. Okay. Portier, Christopher 02-22-2019 (00:00:09)	CP_SS_REDIRECT_01.25
	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?	
607:20 - 607:24	607:14 A. Correct. It changes to yes. Portier, Christopher 02-22-2019 (00:00:04)	CP_SS_REDIRECT_01.26
	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?	
607:25 - 608:6	607:24 A. No. Portier, Christopher 02-22-2019 (00:00:18)	CP_SS_REDIRECT_01.27
	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?	
609:5 - 609:13	Portier, Christopher 02-22-2019 (00:00:28)	CP_SS_REDIRECT_01.28
	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.	
609:19 - 609:21	Portier, Christopher 02-22-2019 (00:00:05)	CP_SS_REDIRECT_01.28
	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?	
609:24 - 610:8	Portier, Christopher 02-22-2019 (00:00:22)	CP_SS_REDIRECT_01.30
	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	

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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

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PORTIER, CHRISTOPHER 2019-02-22_SS

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613:19 - 614:7	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:20)</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
	<p>614:6 Q. And let's go ahead and put 614:7 those up.</p>	1456.1.2
614:8 - 614:12	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:09)</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>	M22.13
614:13 - 615:10	<p>PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:45)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:23)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:13)</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. PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:10)</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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:55)	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	550.4.3
	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"?	
	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:19)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:48)	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	PORTIER, CHRISTOPHER 2019-02-22_SS (00:00:47)	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

Portier, Christopher 02-22-2019

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638:14 - 638:21	<p>Portier, Christopher 02-22-2019 (00:00:15) 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>Portier, Christopher 02-22-2019 (00:00:04) 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>Portier, Christopher 02-22-2019 (00:00:42) 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>Portier, Christopher 02-22-2019 (00:00:21) 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. Portier, Christopher 02-22-2019 (00:00:05) 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	Portier, Christopher 02-22-2019 (00:00:14) 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	Portier, Christopher 02-22-2019 (00:00:04) 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	Portier, Christopher 02-22-2019 (00:00:00) 641:23 THE WITNESS: No.	CP_SS_RE.8
641:25 - 641:25	Portier, Christopher 02-22-2019 (00:00:01) 641:25 THE WITNESS: It's not.	CP_SS_RE.9

Total Time = 00:01:51

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Portier, Christopher 02-22-2019

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642:8 - 642:24

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

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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