SUPERIOR COURT OF CALIFORNIA

COUNTY OF ALAMEDA

BEFORE THE HONORABLE WINIFRED Y. SMITH, JUDGE PRESIDING

DEPARTMENT NUMBER 21

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COORDINATION PROCEEDING SPECIAL TITLE (RULE 3.550) ROUNDUP PRODUCTS CASE	**JCCP No. 4953**
THIS TRANSCRIPT RELATES TO: Pilliod, et al. vs. Monsanto Company, et al.	**Case No. RG17862702** **Pages 1533 - 1833** **Volume 12**

Reporter's Transcript of Proceedings

Tuesday, April 2, 2019

Reported by: Kelly L. Shainline, CSR No. 13476, RPR, CRR
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I N D E X

Tuesday, April 2, 2019

Tuesday, April 2, 2019 8:45 a.m.

(Proceedings commenced in chambers out of the presence of the jury.)

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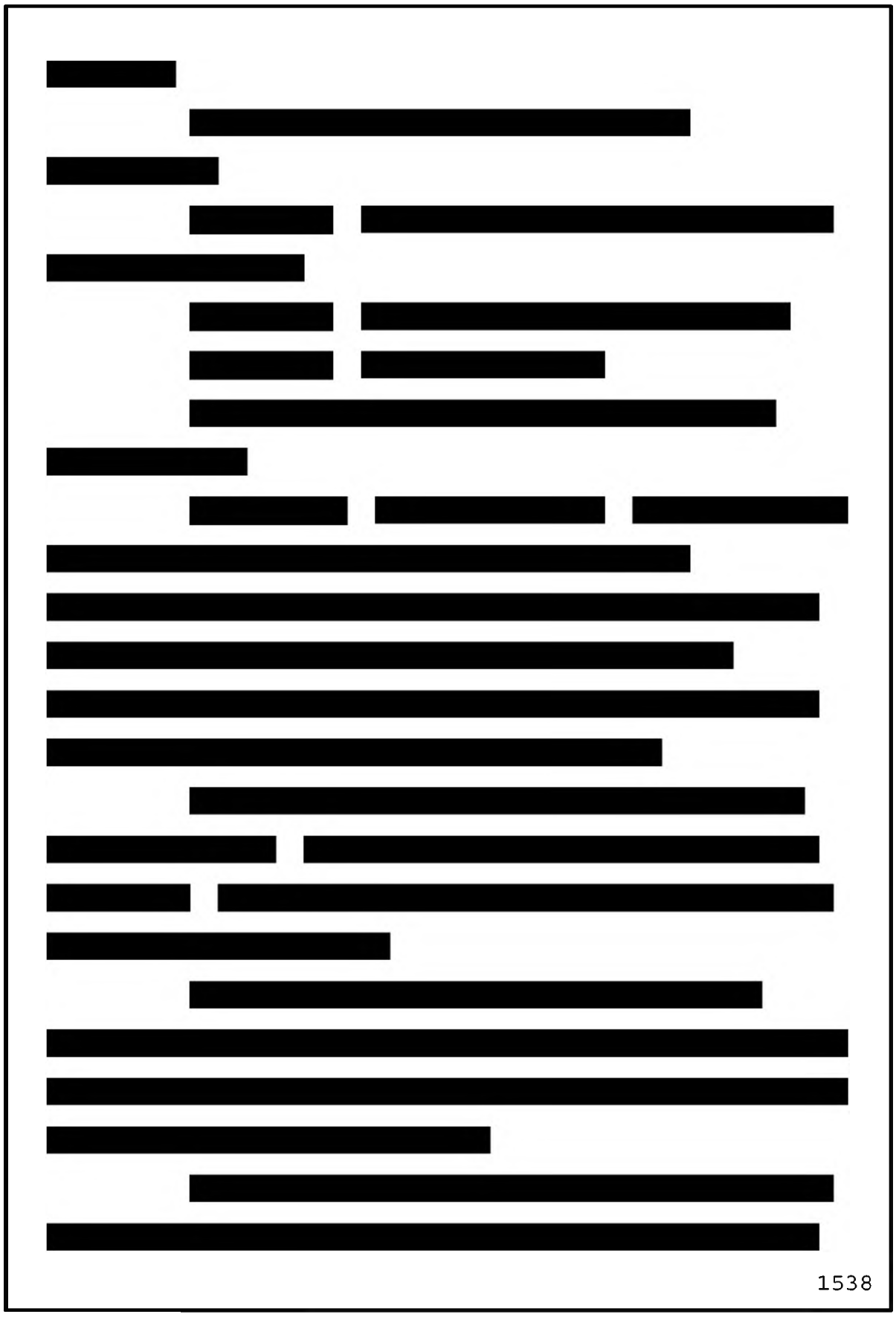
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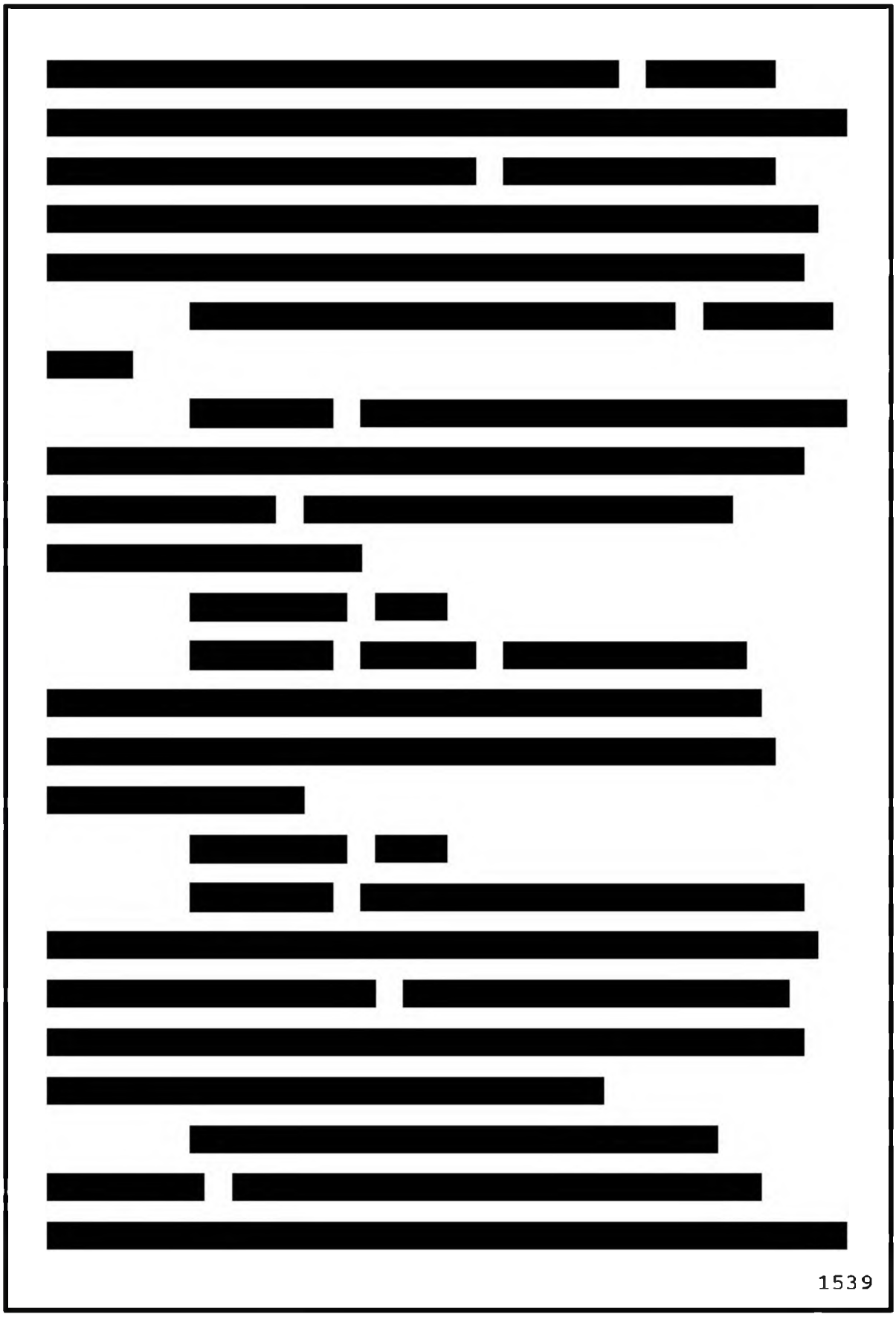
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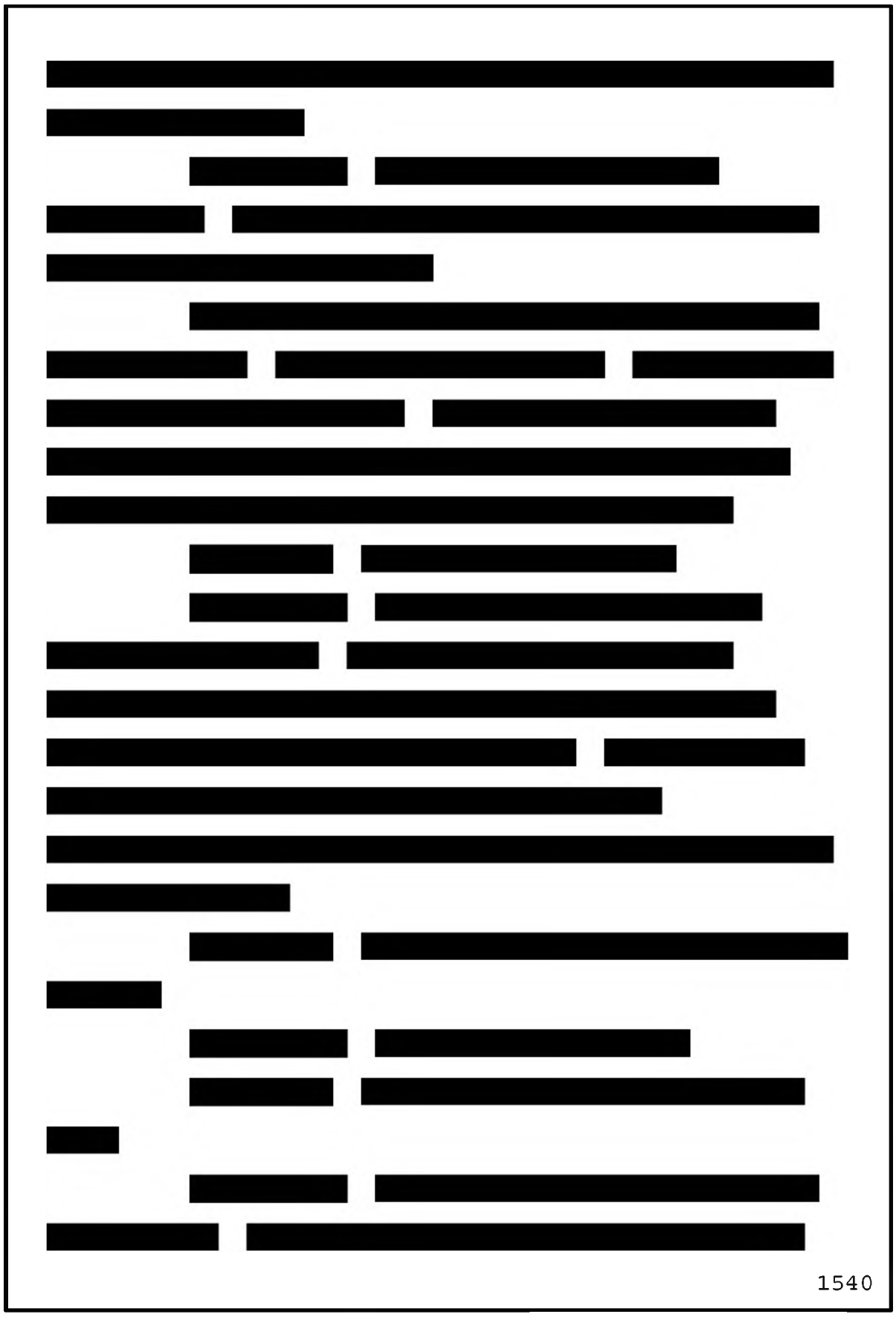
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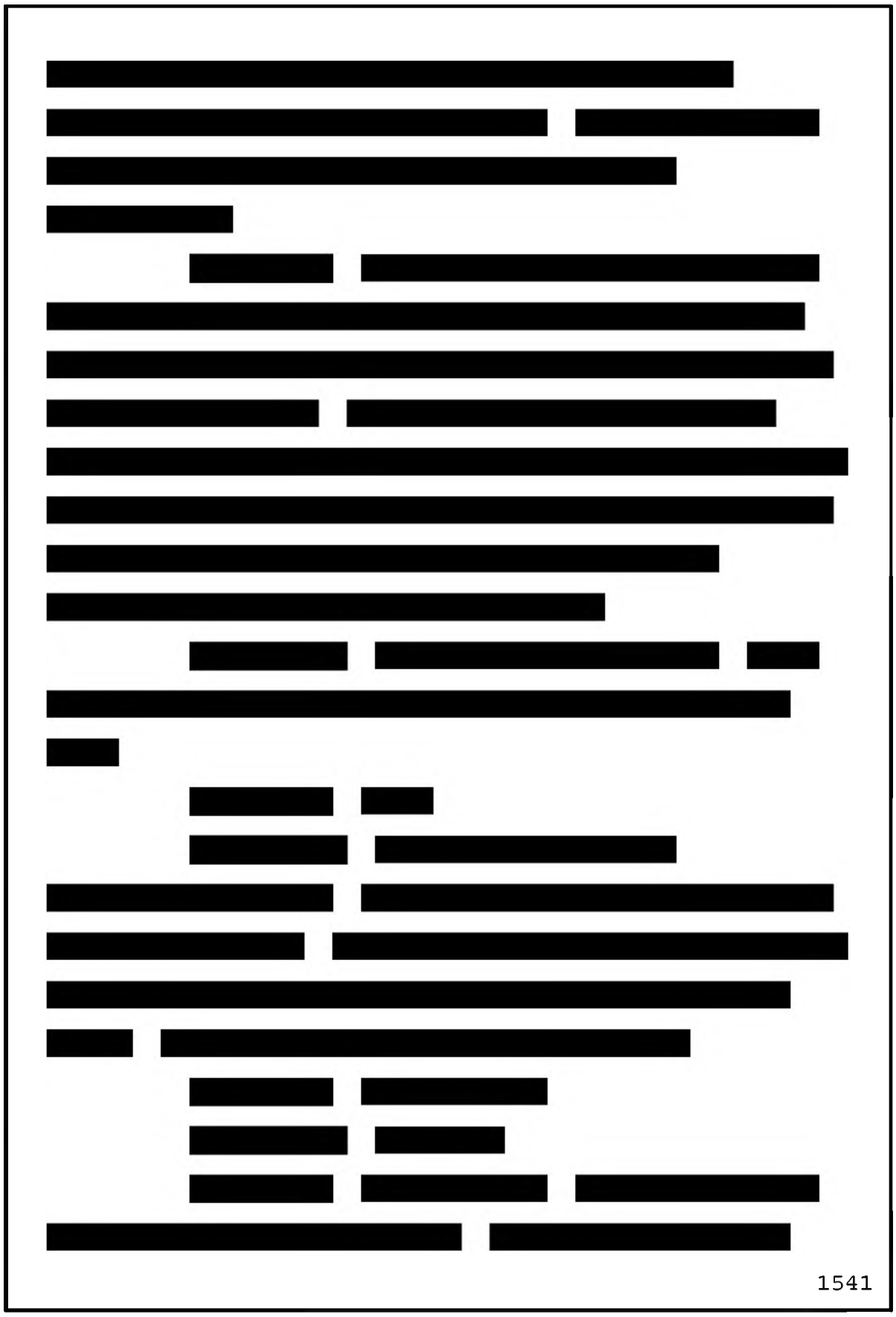
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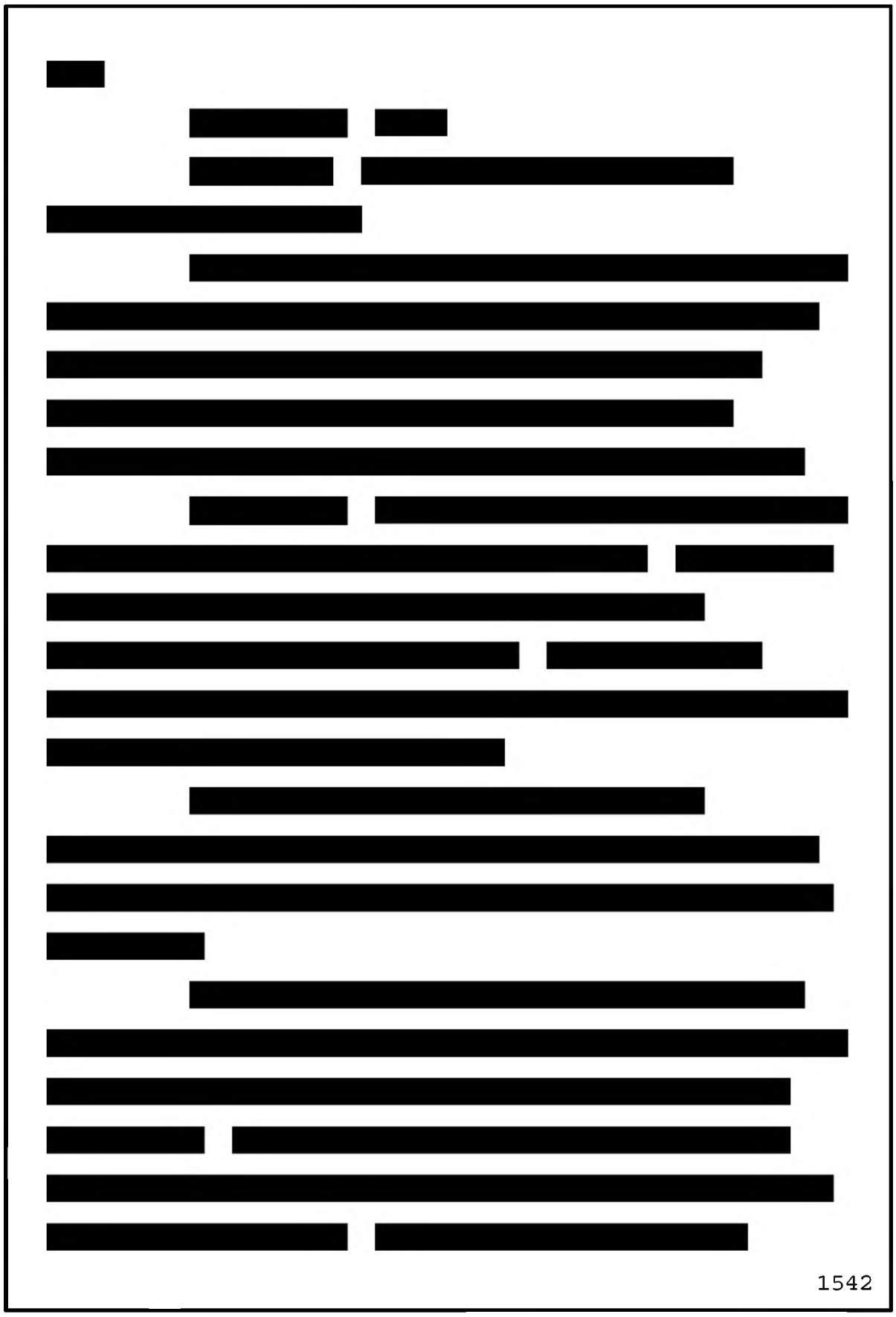
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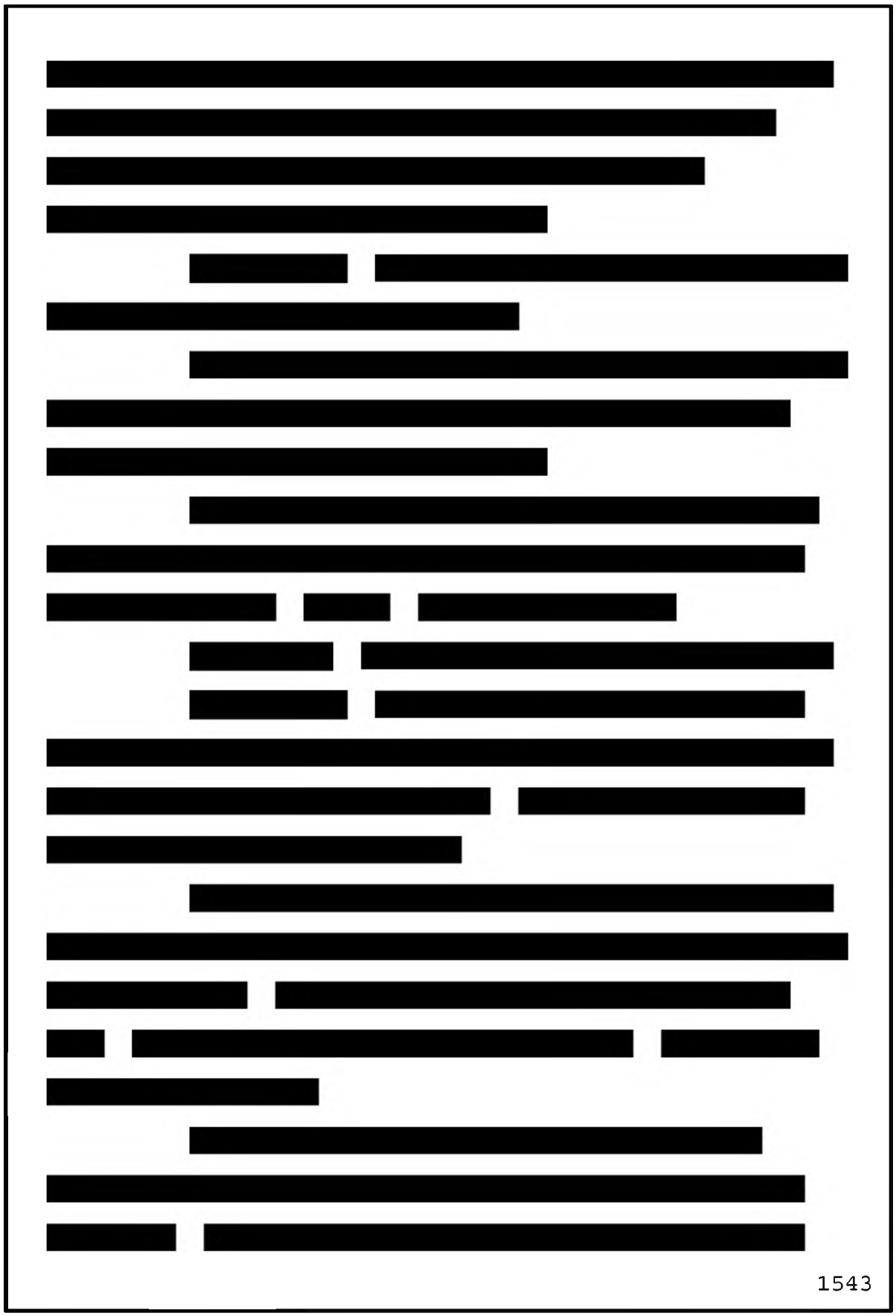
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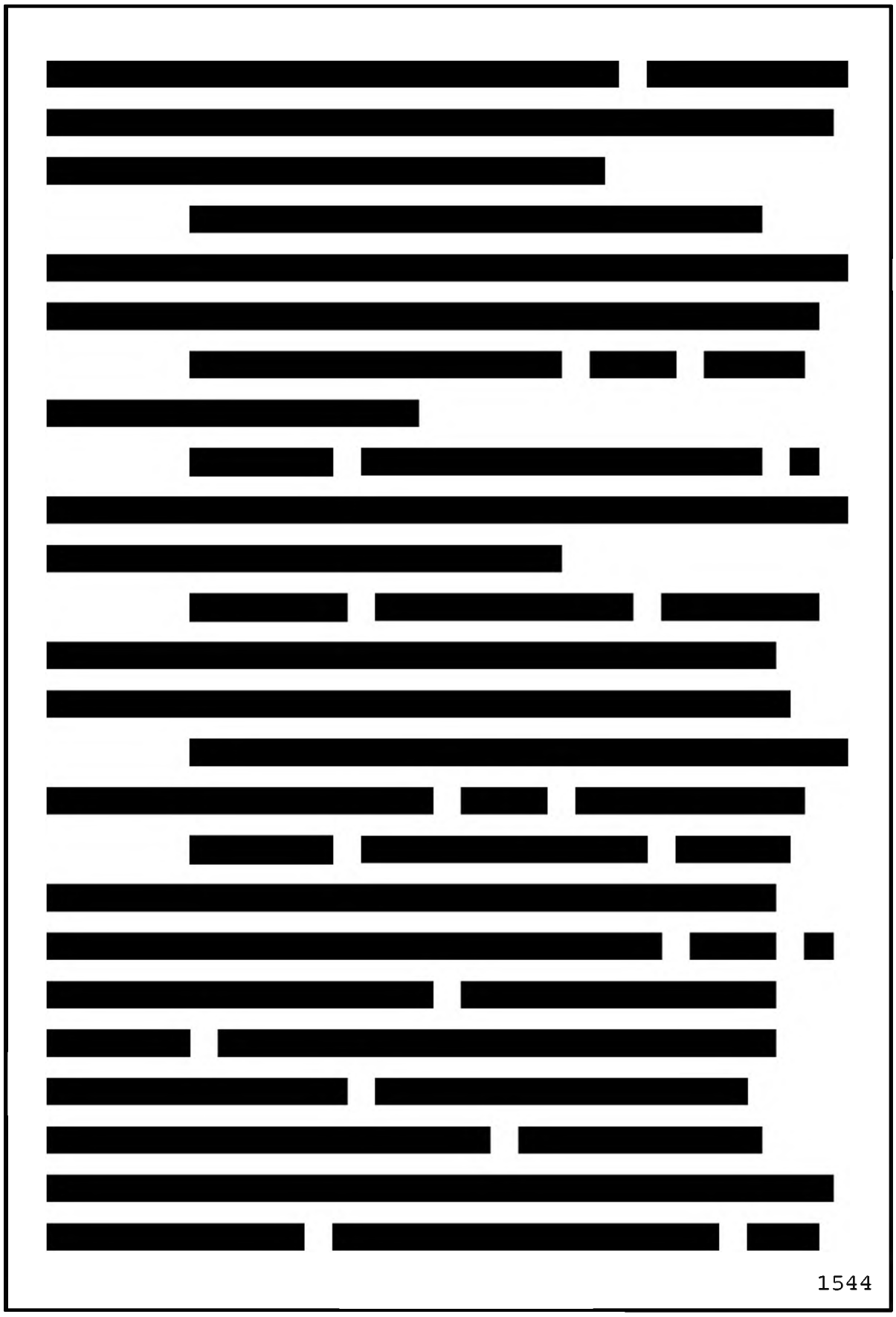
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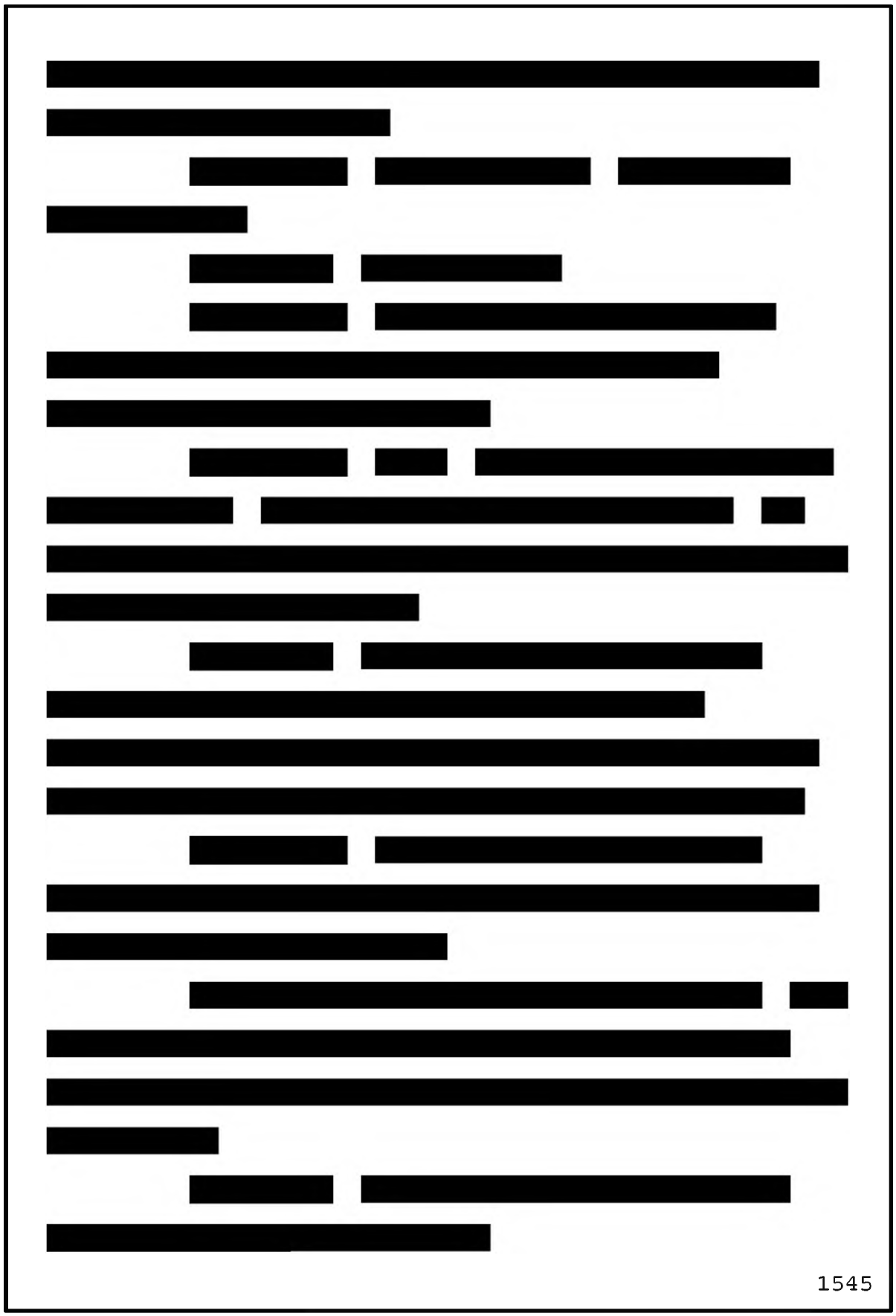
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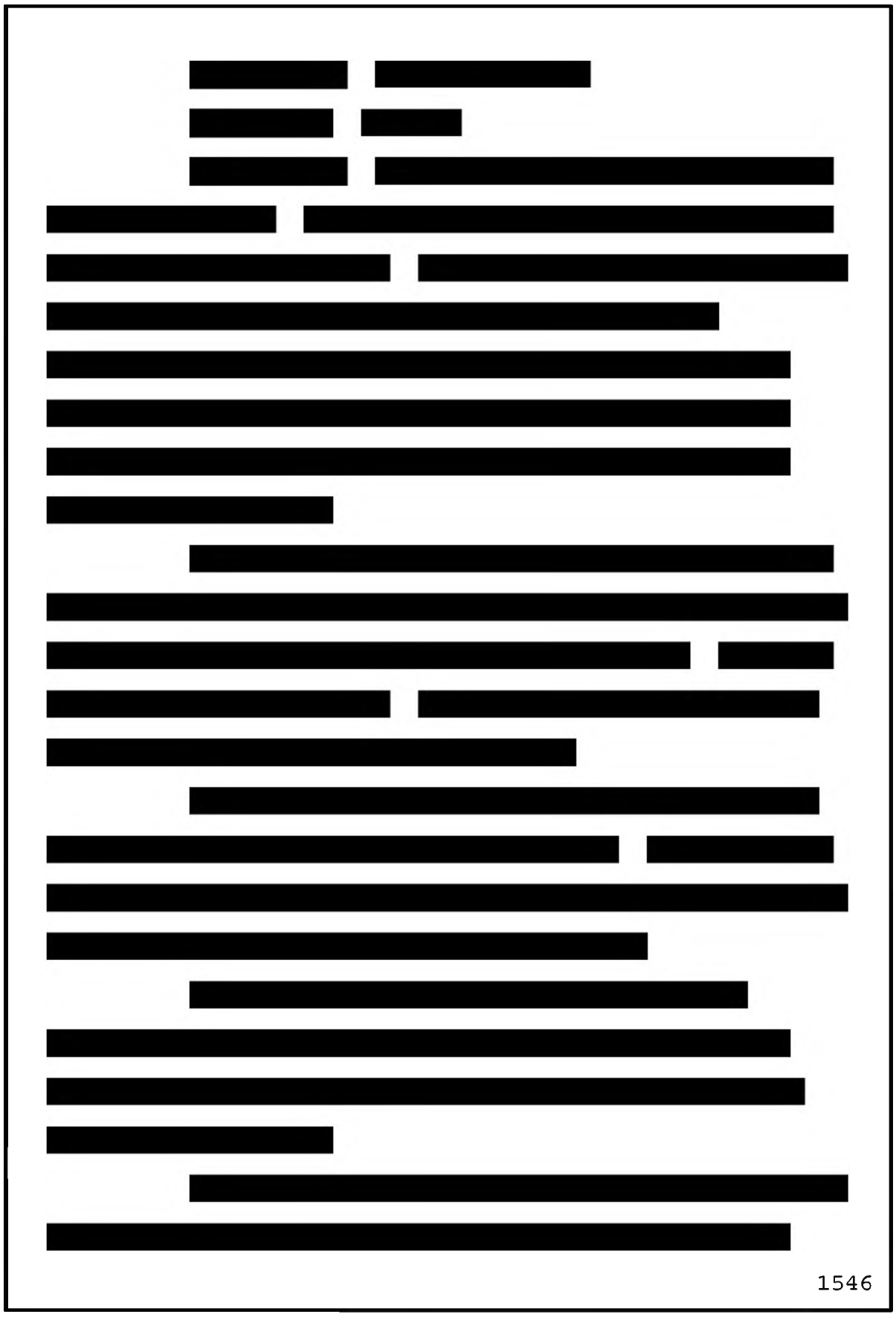
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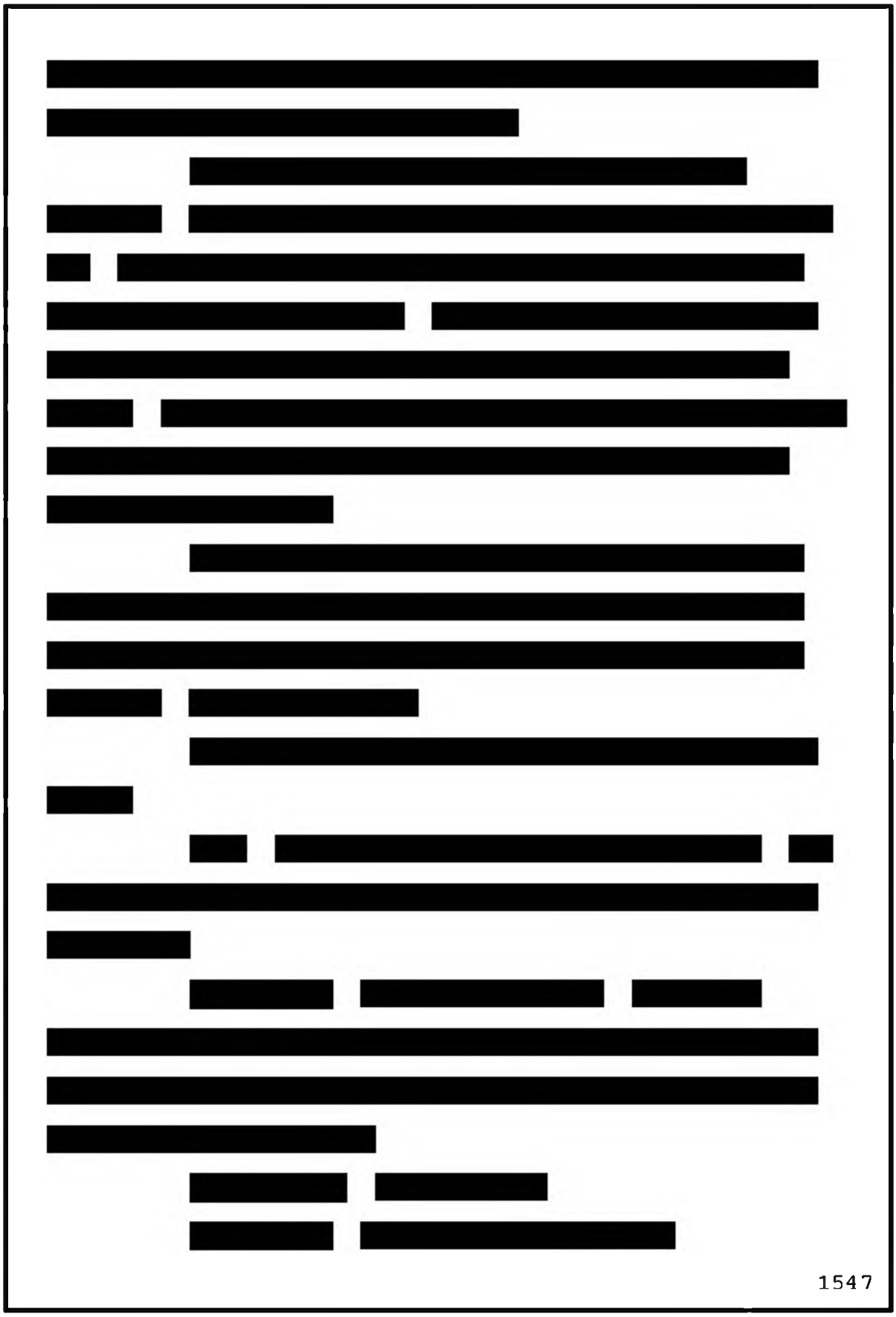
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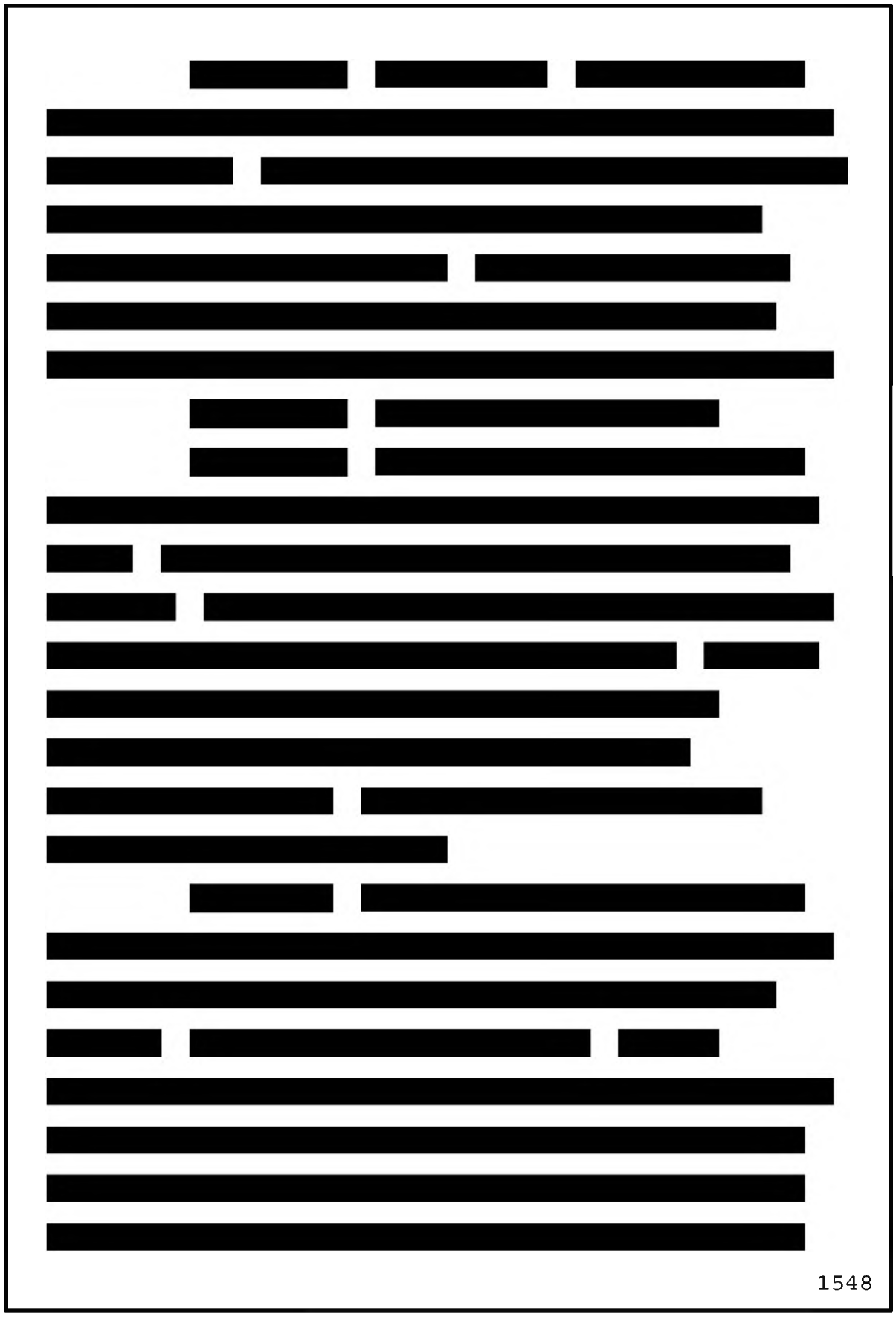
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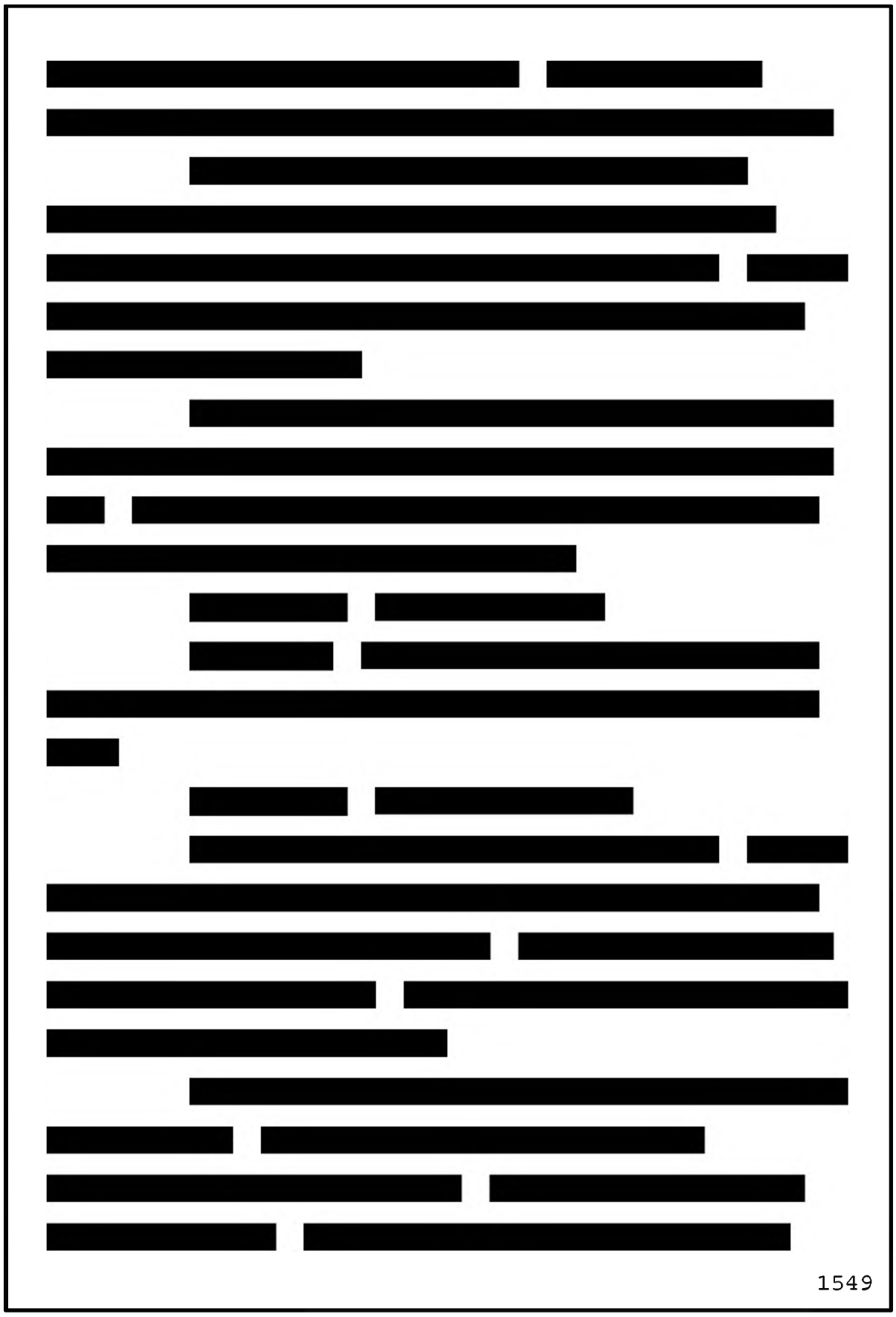
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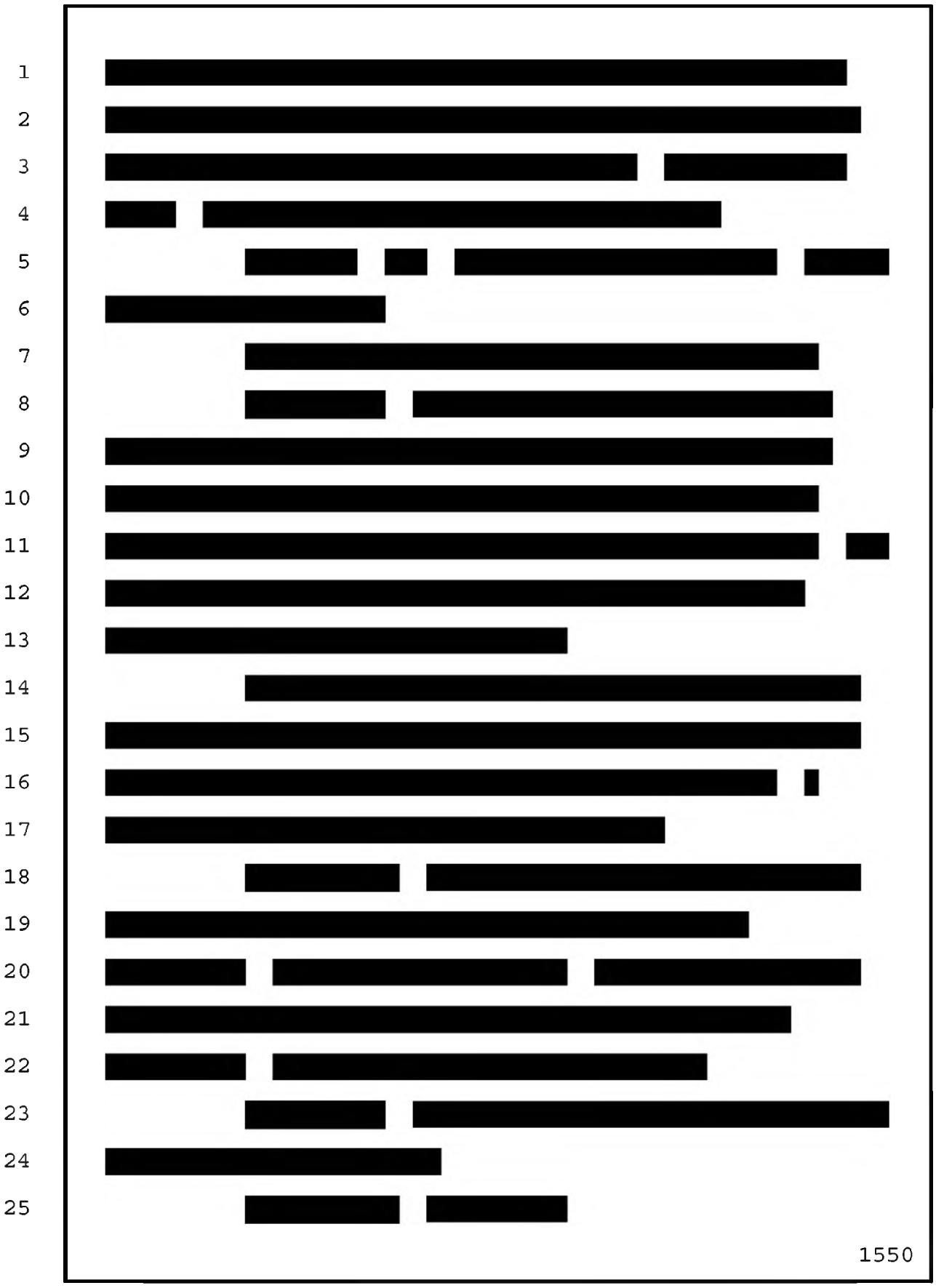
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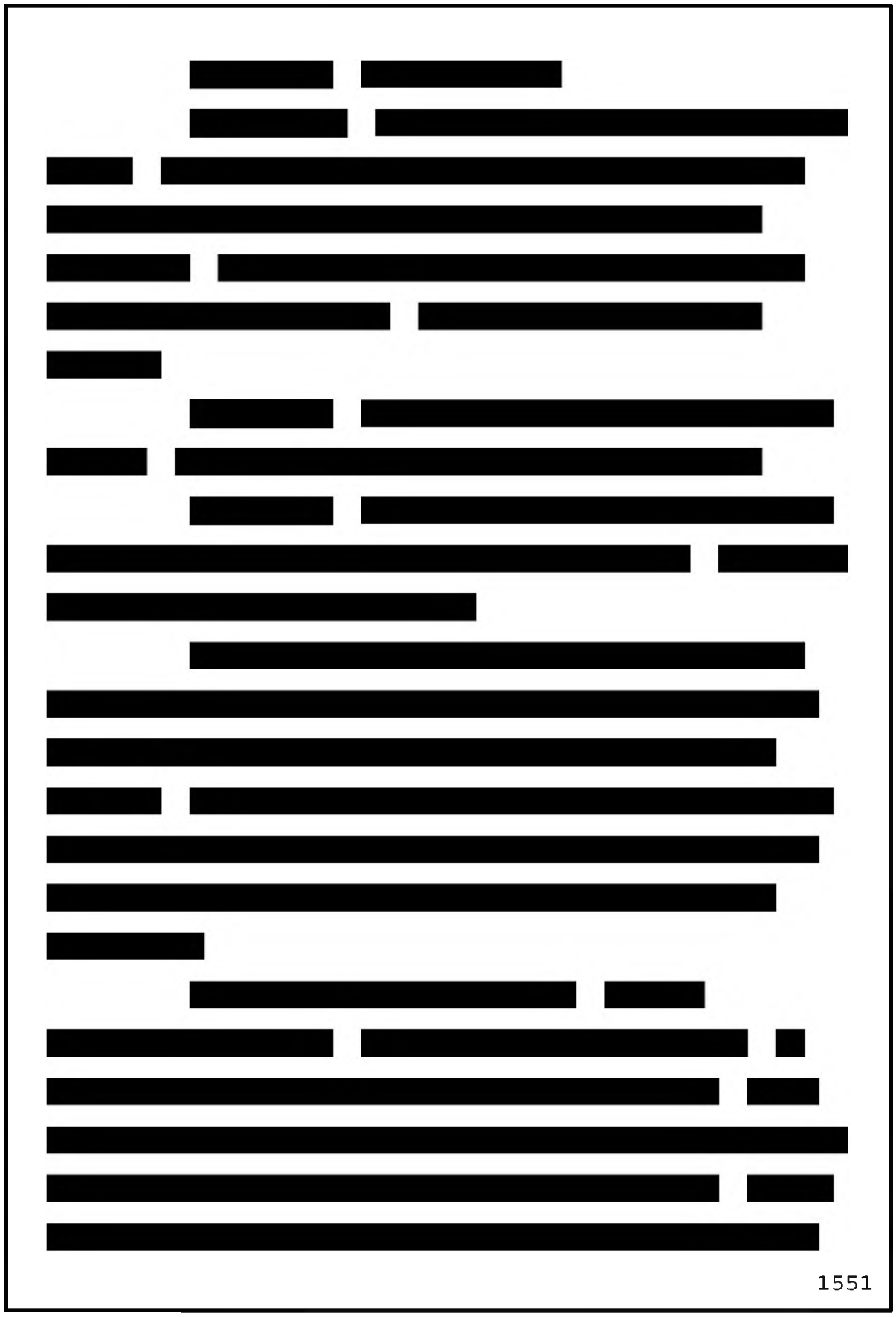
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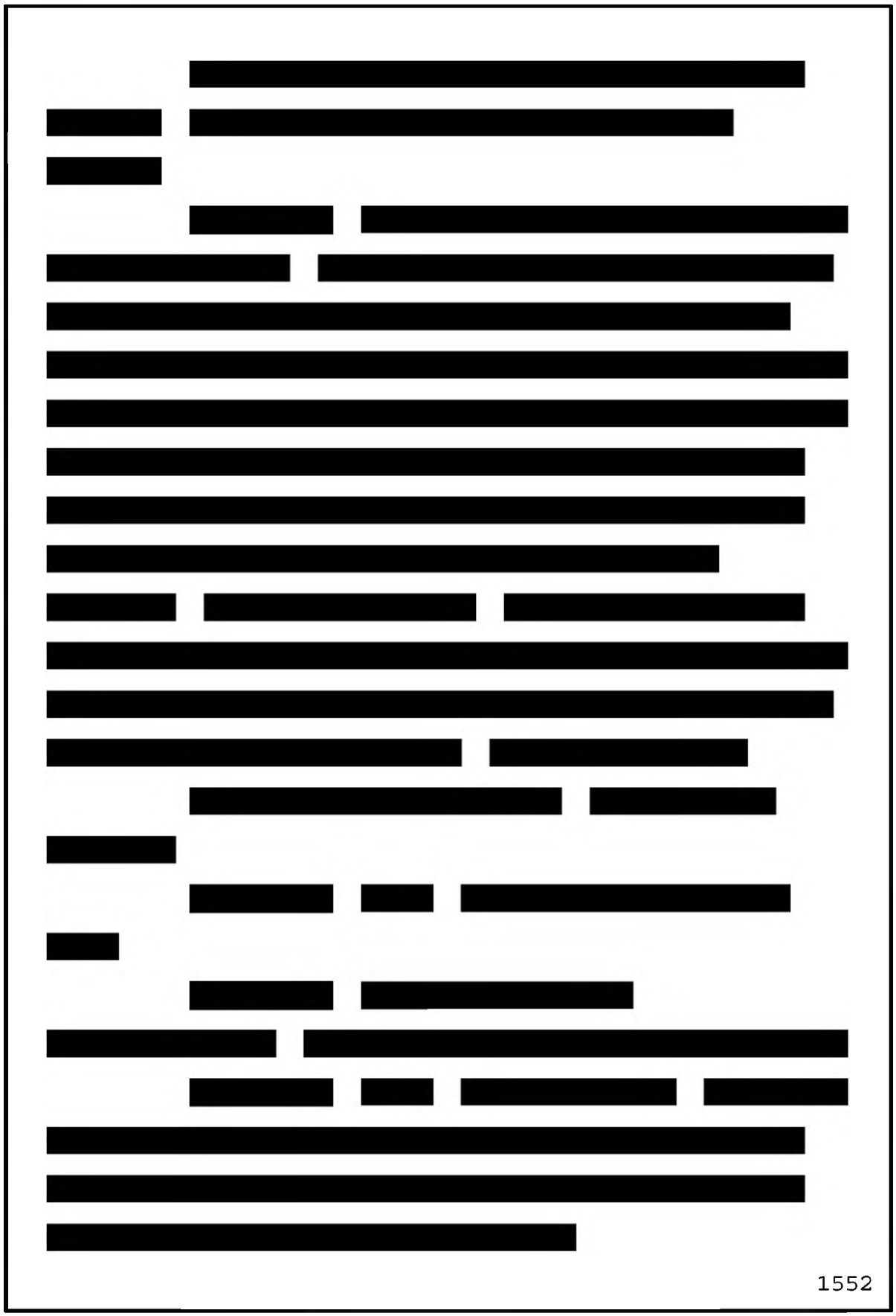
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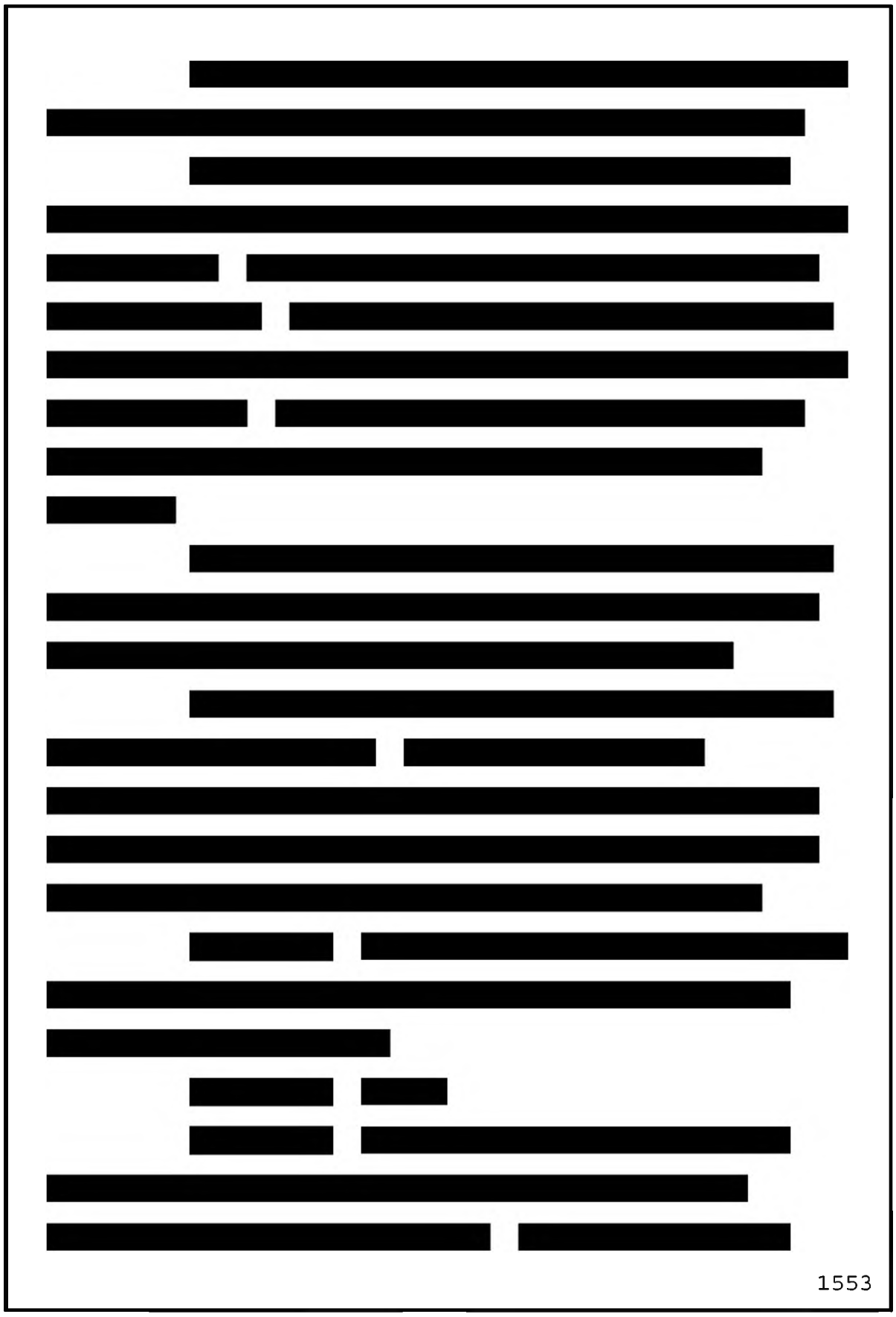
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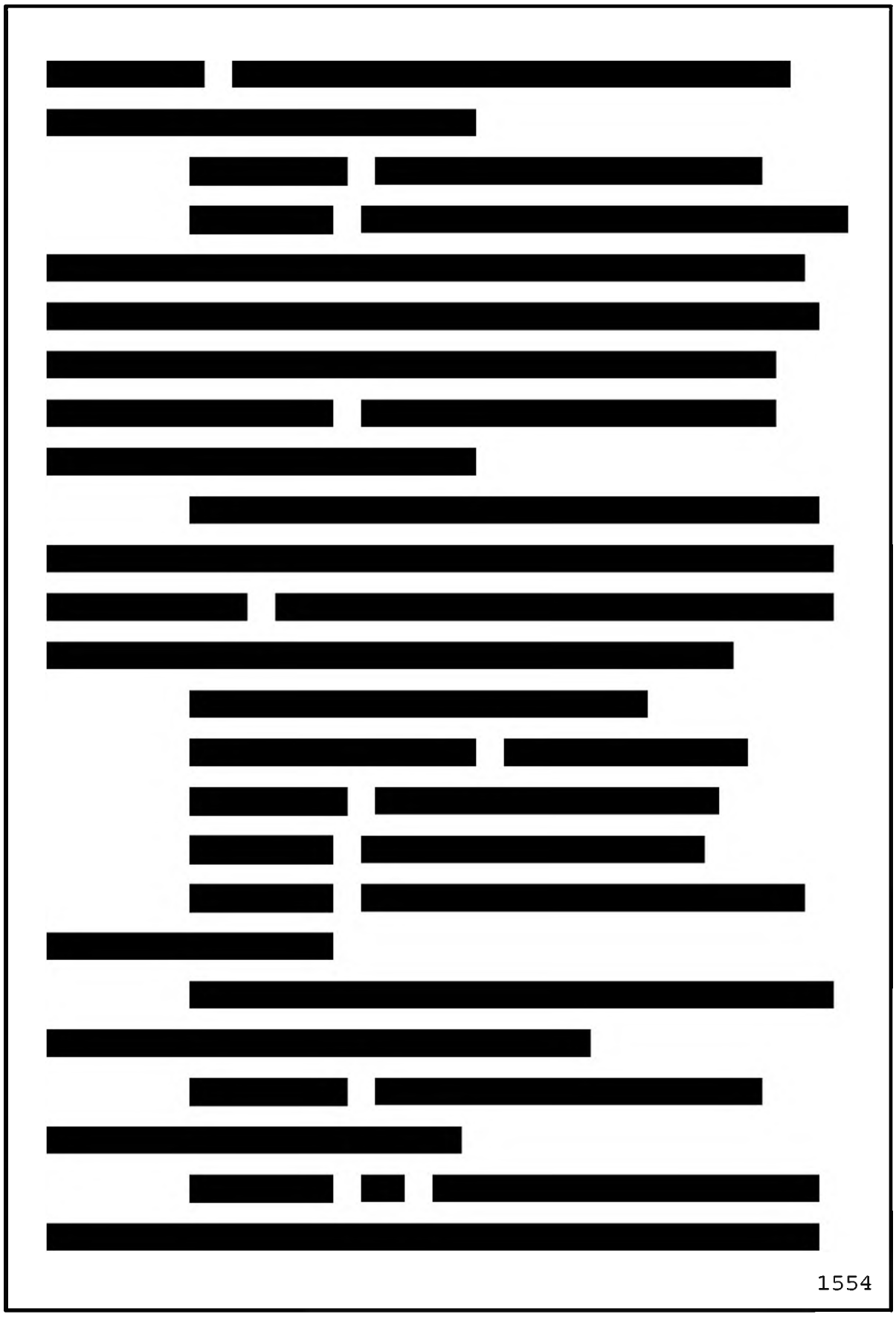
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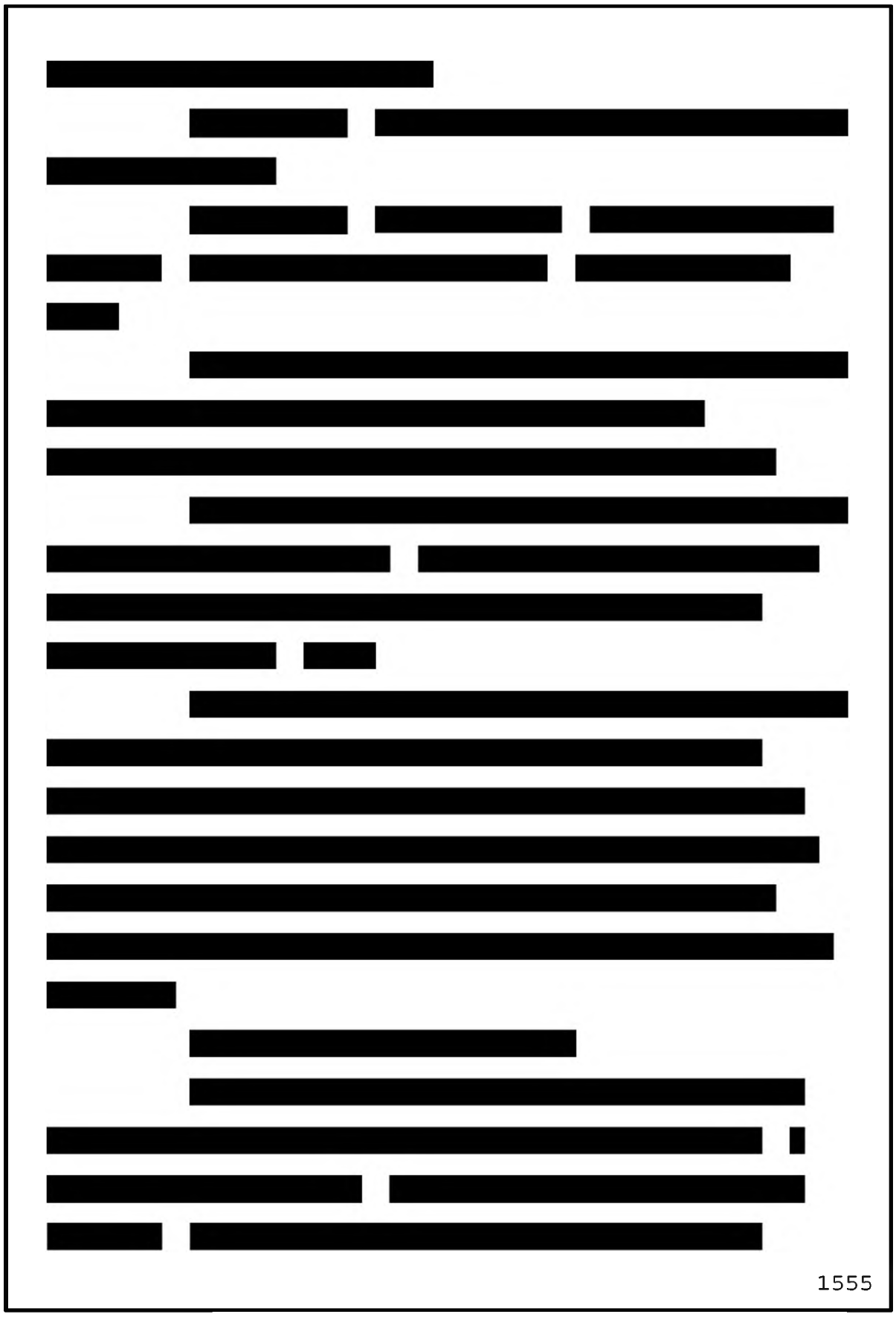
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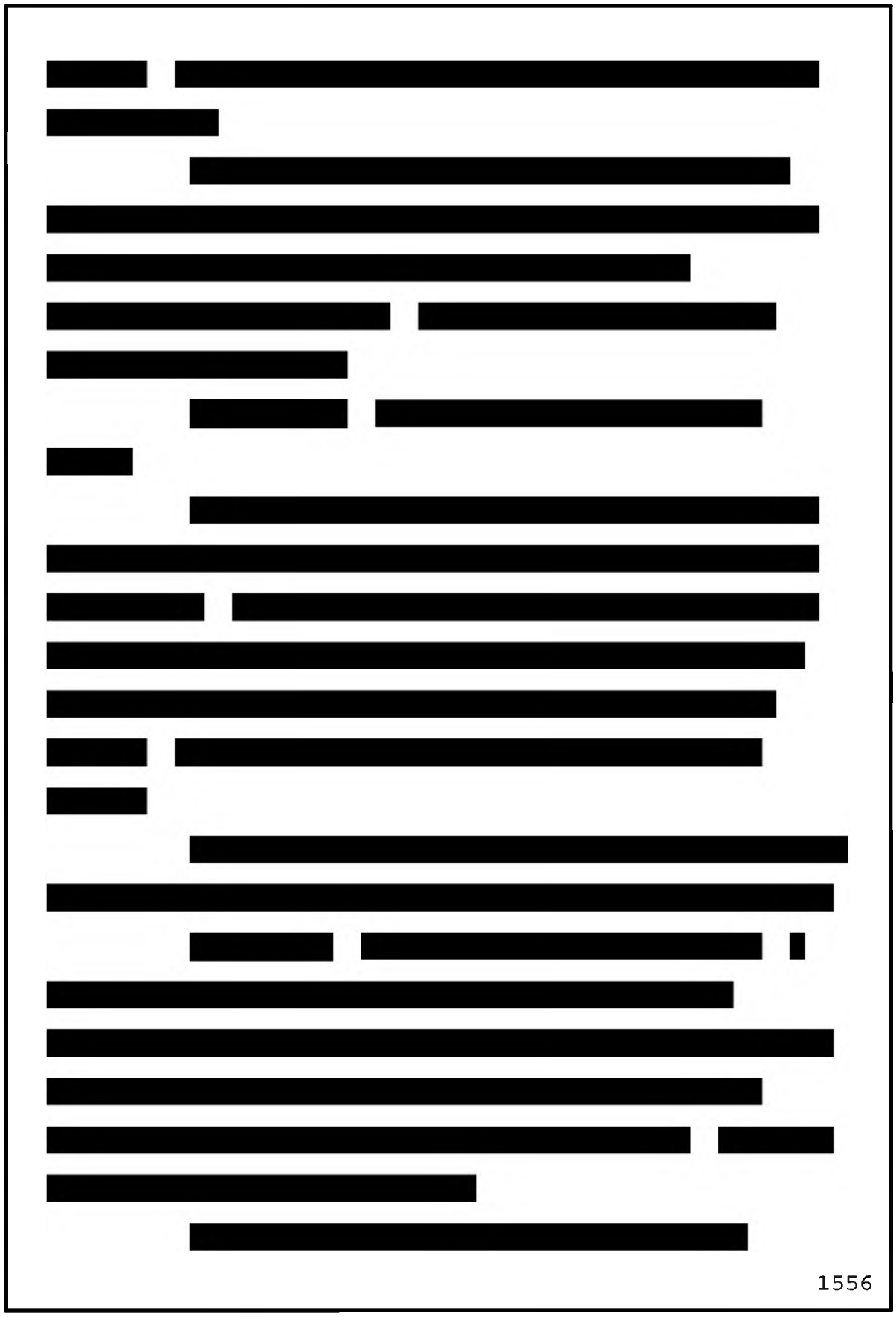
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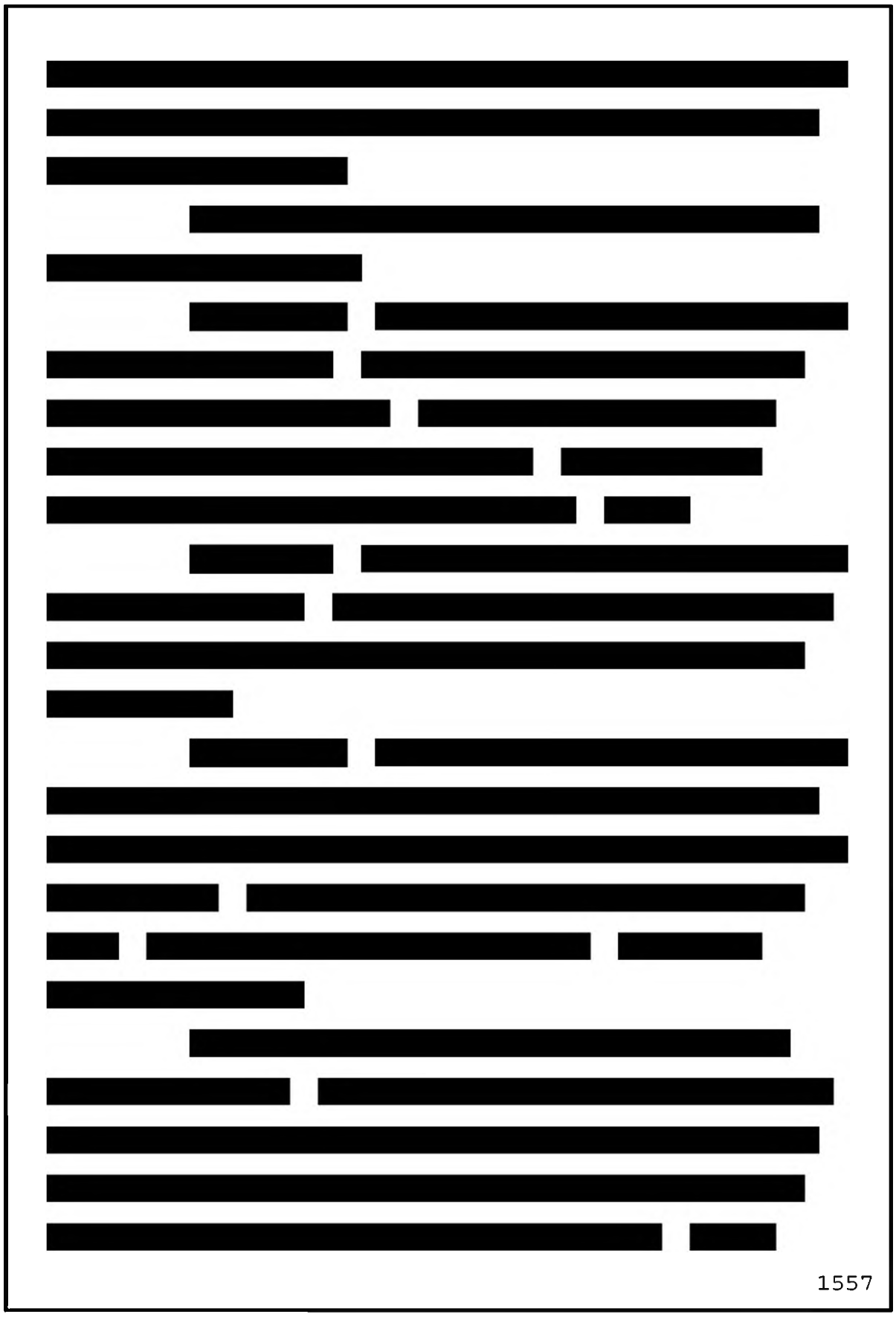
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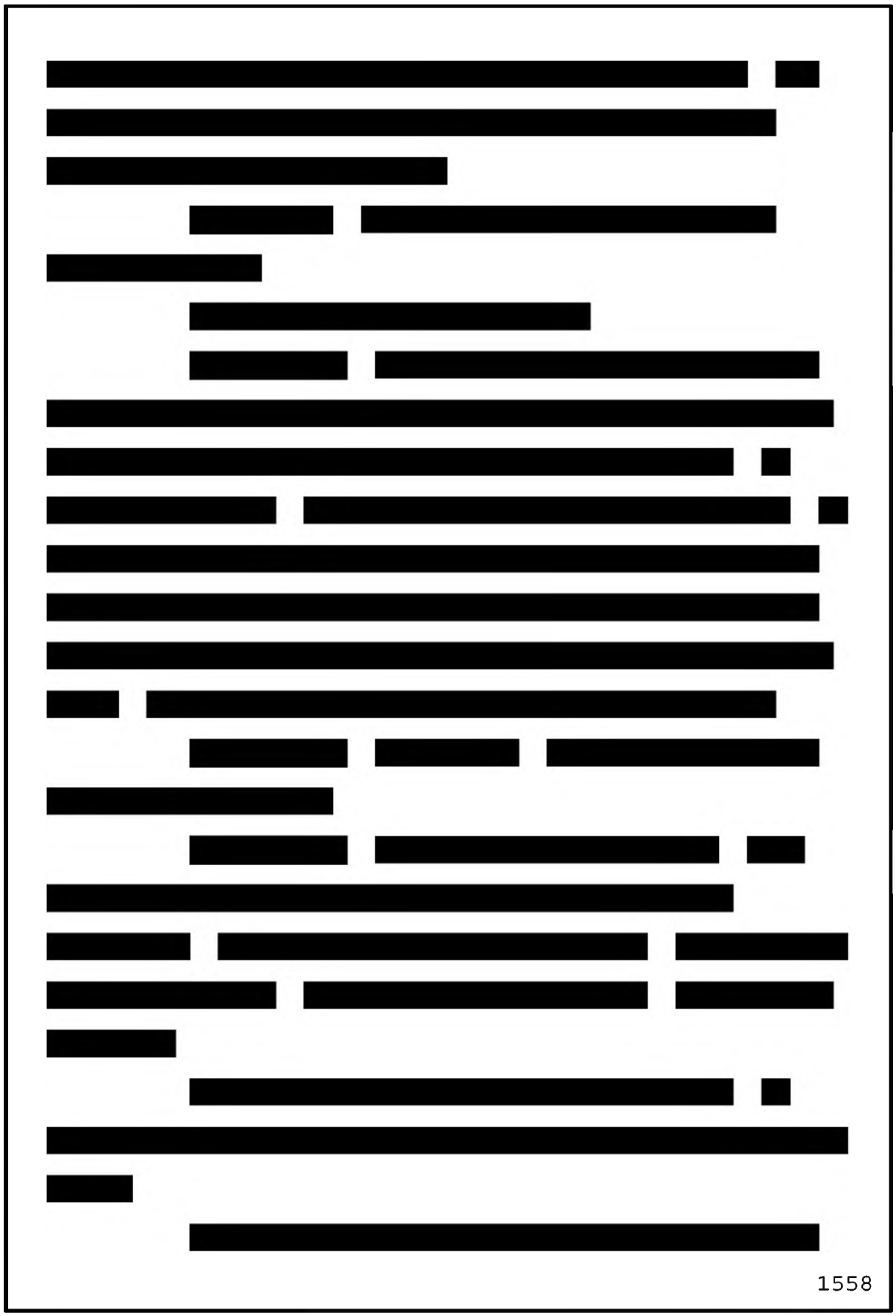
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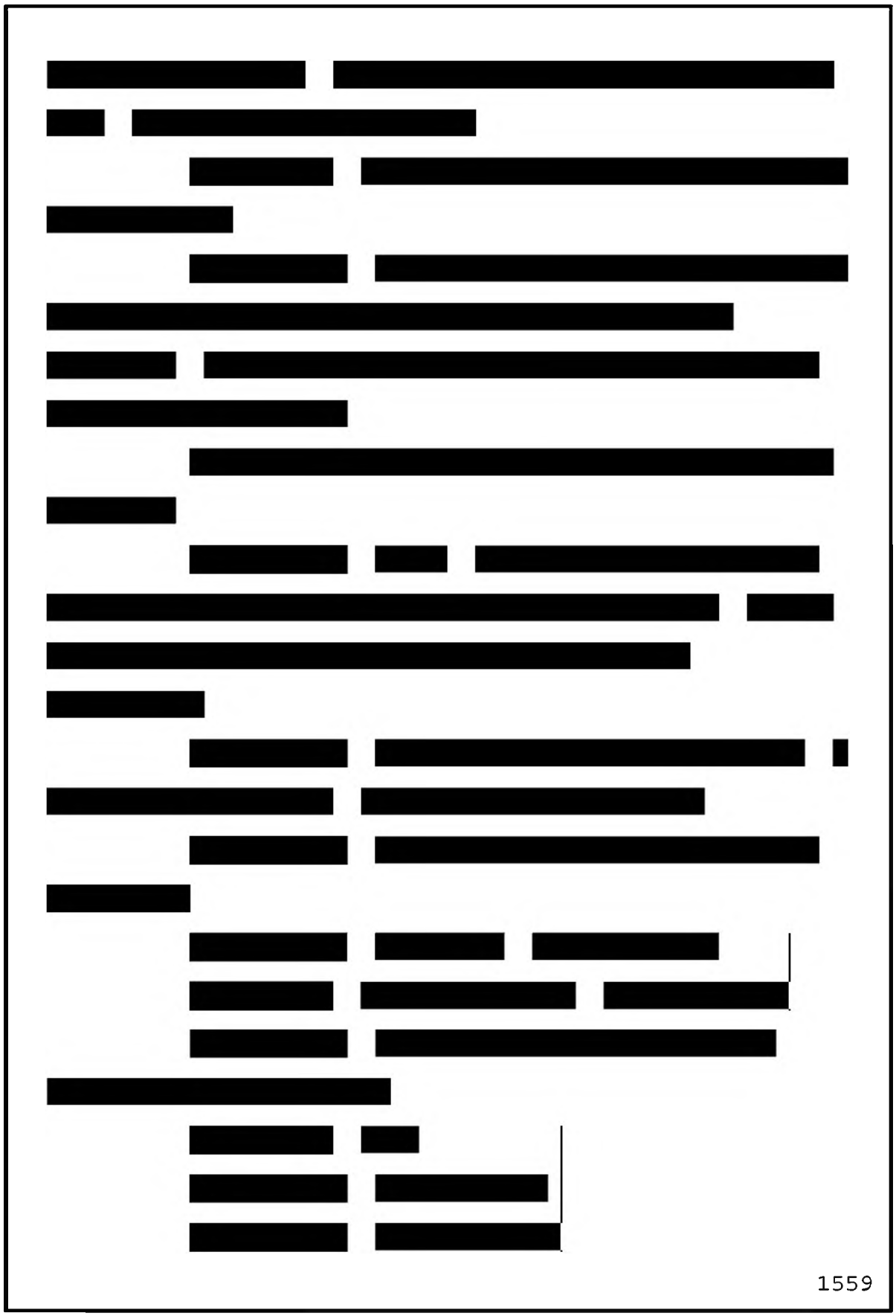
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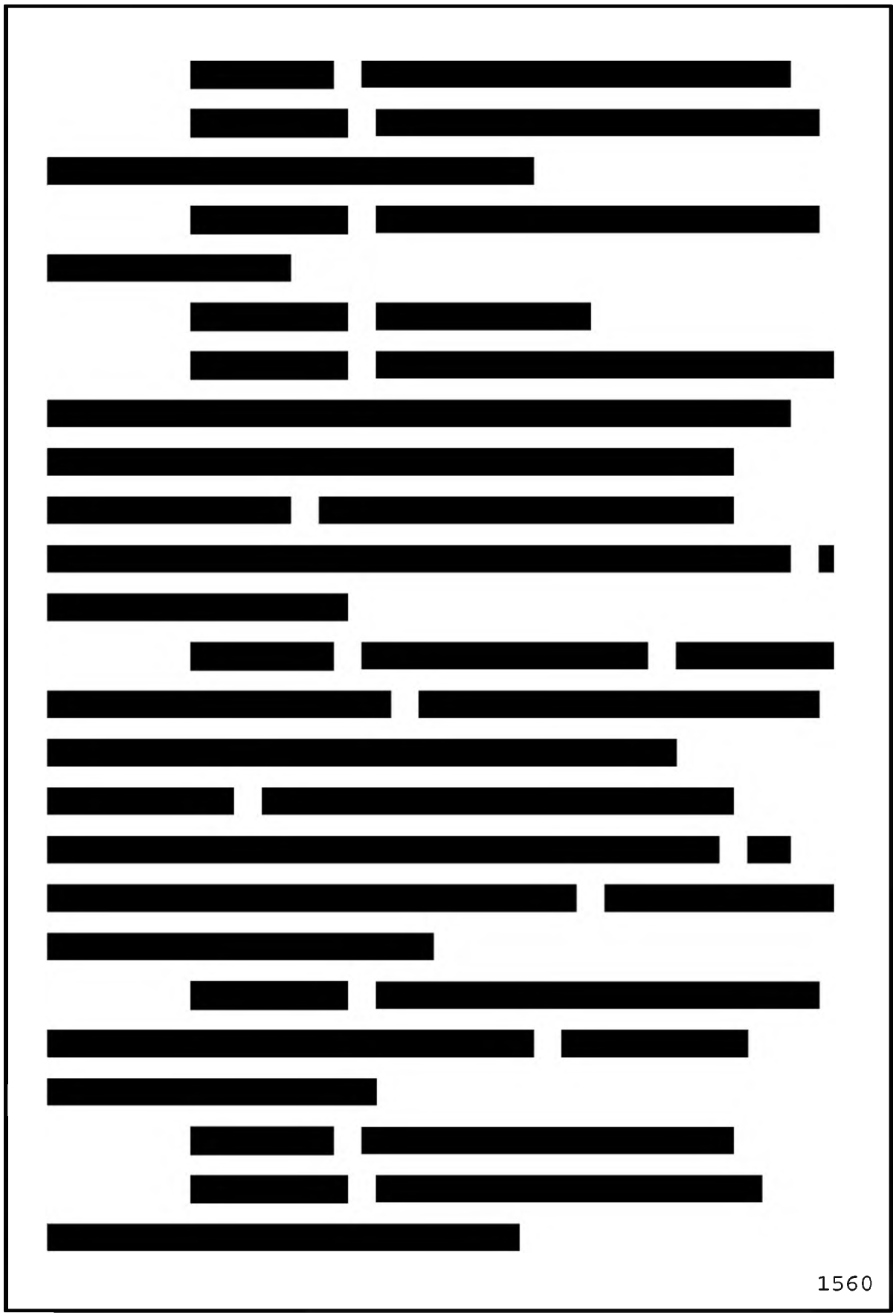
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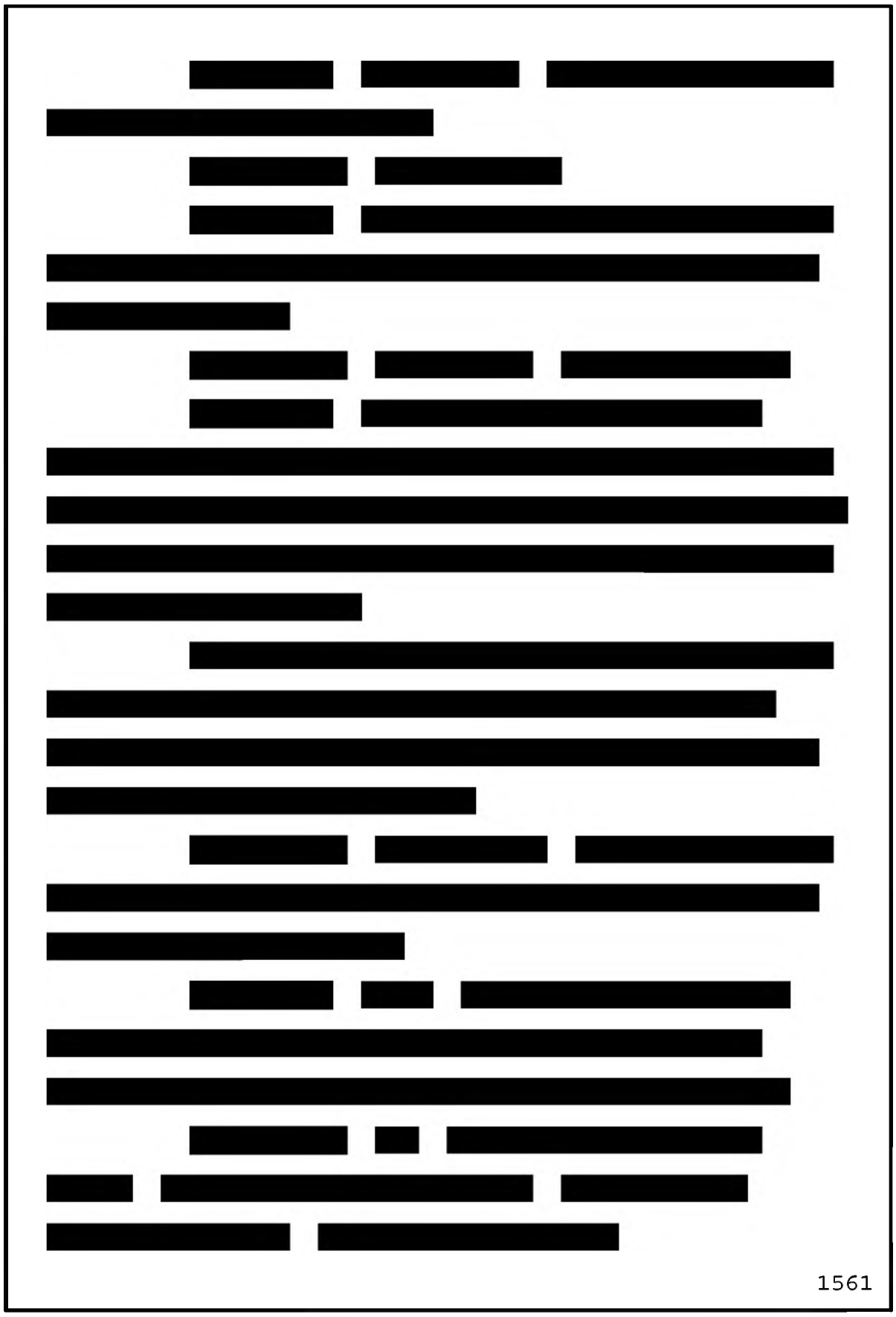
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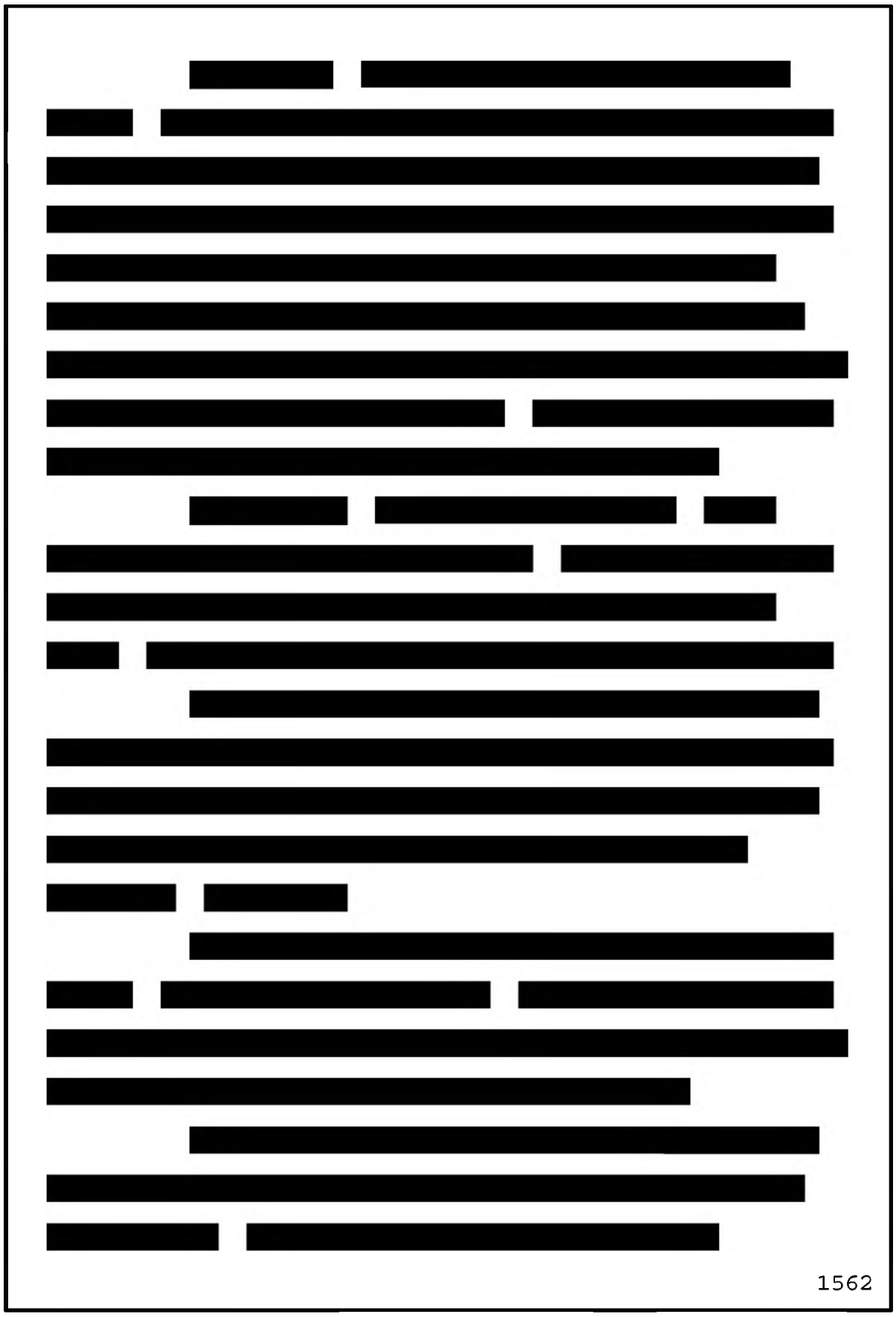
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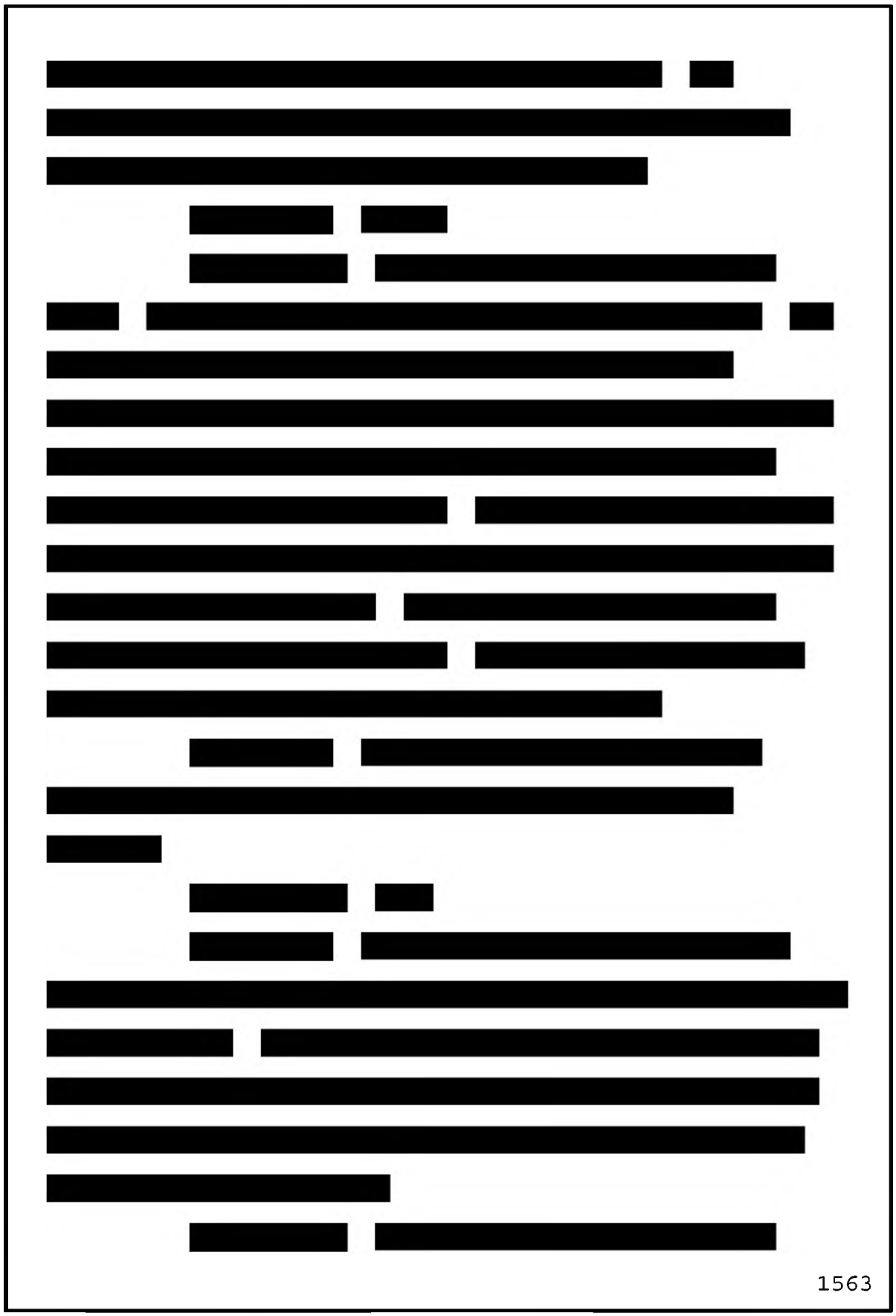
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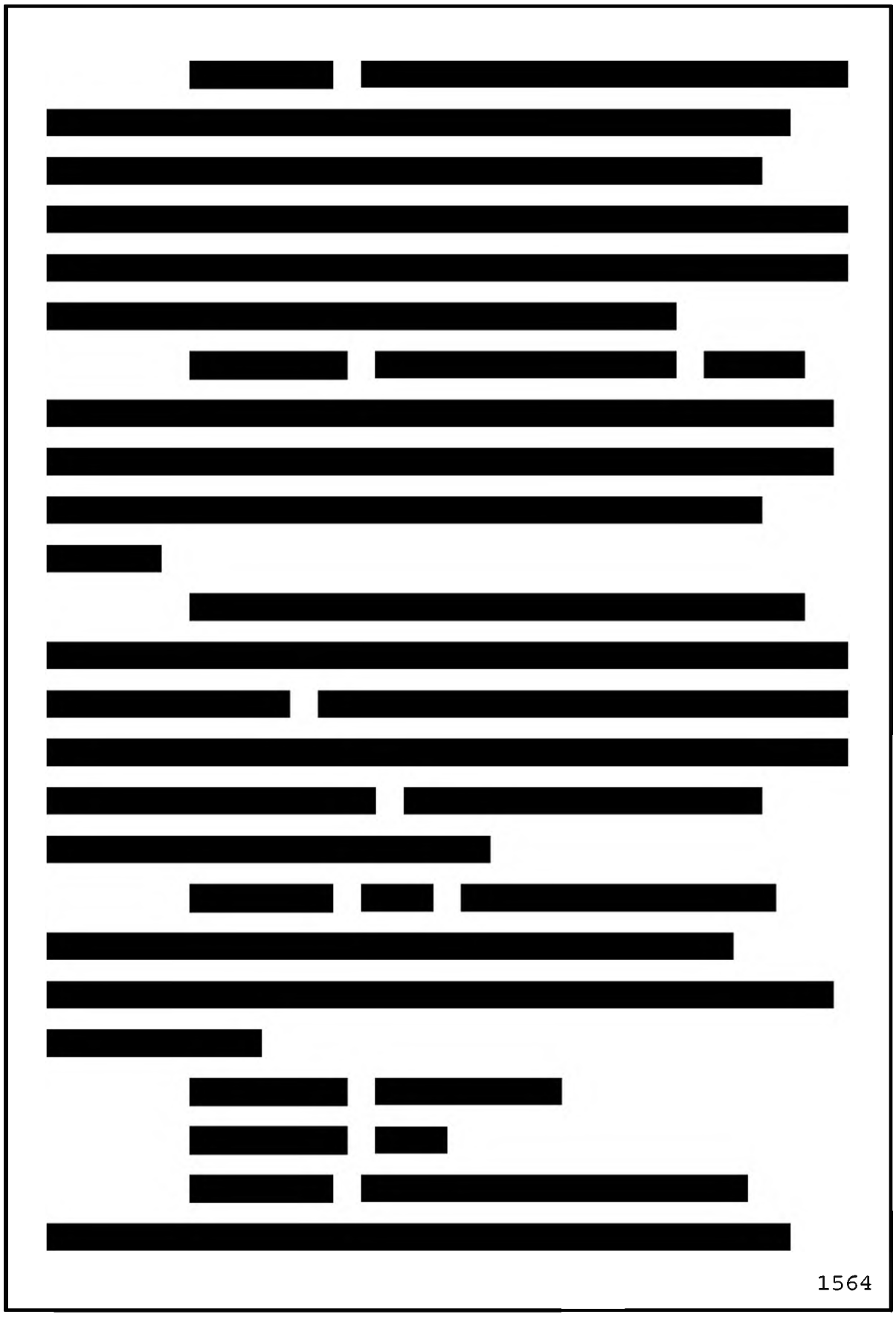
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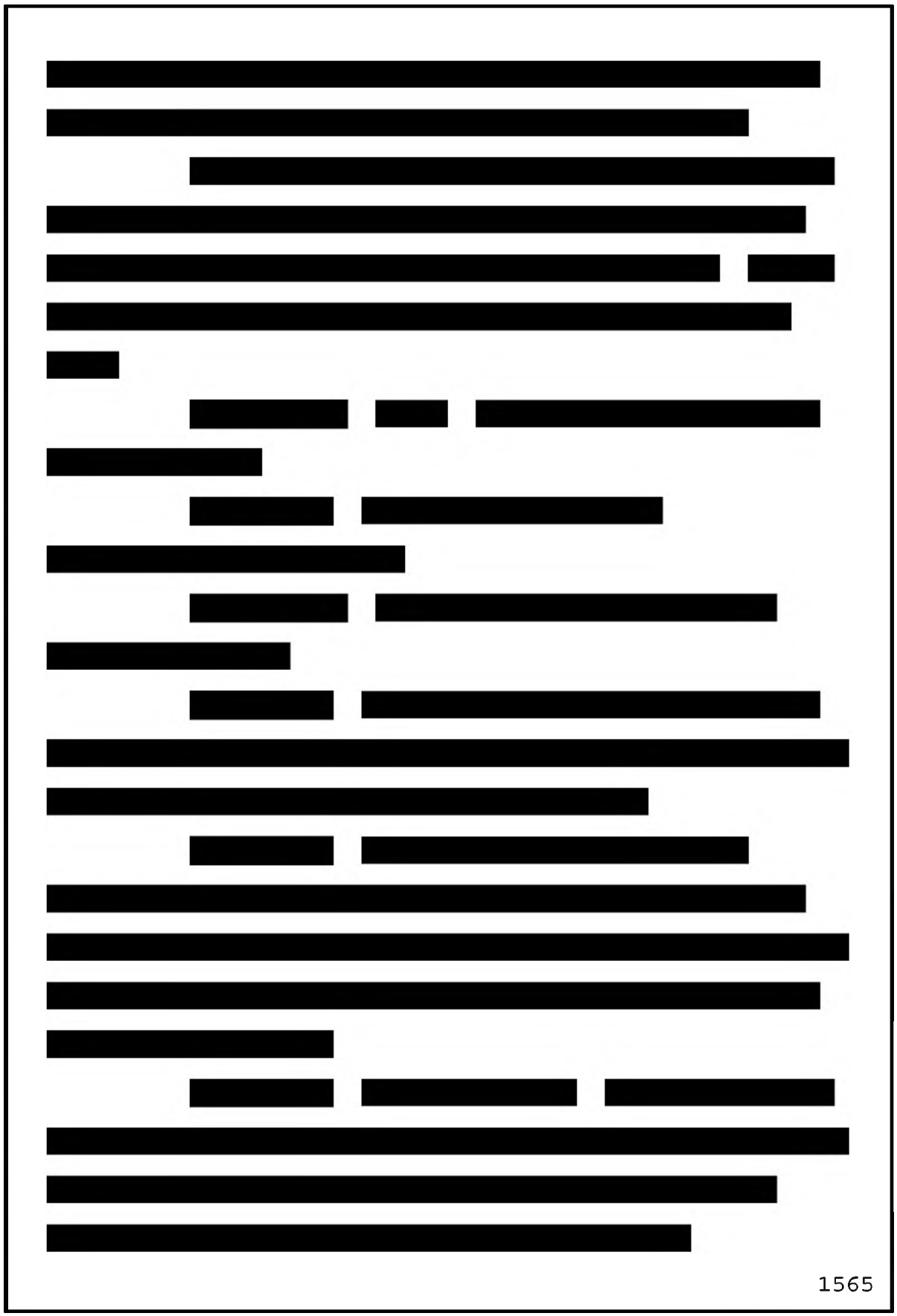
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[redacted]

(End of proceedings in chambers.)

(Recess taken at 9:19 a.m.)

(Proceedings resumed in open court out of the presence of the jury at 9:29 a.m.)

THE COURT: We're back on the record.

Pilliod versus Monsanto. And Mr. Wisner, we were going to conduct a short hearing this morning.

MR. WISNER: Yes, Your Honor. We'd like to conduct a 402 hearing. We'd like to have Christopher Portier take the stand and testify outside the presence

of the jury.

THE COURT: All right. Will you swear in Dr. Portier.

THE WITNESS: Good morning, Your Honor.

THE CLERK: Sir, if you would please raise your right hand.

CHRISTOPHER PORTIER,

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

THE WITNESS: I do.

THE CLERK: Thank you. Please be seated.

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

THE WITNESS: Christopher Portier.

MR. WISNER: Spell your last for the record.

THE WITNESS: Oh, sorry. I didn't hear that. P-O-R-T-I-E-R.

MR. WISNER: May I proceed, Your Honor?

THE COURT: Yes, you may.

DIRECT EXAMINATION

BY MR. WISNER:

Q. Good morning, Doctor.

A. Good morning.

Q. The purpose of this short hearing here is to discuss a specific issue we discussed yesterday, and I

want to give the Court a chance to hear what you have to say about it.

Specifically yesterday did you have a chance to review some of the testimony by Dr. Bill Reeves, Michael Koch, and Donna Farmer?

A. Yes, I did.

Q. And was that testimony specifically about whether it's possible to conduct a long-term rodent study on a formulated product like Roundup?

A. Yes, it was.

Q. Do you have an opinion about that?

A. In terms of being able to do that type of study, yes, of course, you can do that study.

Q. And why is that, sir?

A. Well, there are -- you can administer the material to the rats and mice. Even if it's somewhat nauseating, you can deal with that in the process of doing the material. But you've got a good example of just that type of study with the study that was done by Seralini.

Q. And what does that study show you about the feasibility of being able to conduct a long-term rodent study on formulated Roundup?

A. Well, the main concern by Dr. Farmer and her colleagues was mortality. They figured the animals

would die. But when you look at the Seralini study, there's no indication that he saw excess mortality in the exposed animals. So it clearly appears that that's just not a problem.

Q. And in your work history working at the National Toxicology Program and elsewhere, which we'll discuss in more detail later, have you ever had an occasion to test a substance in rodents in a long-term study where there was concern about its effects on the digestive tract?

A. Oh, yes, absolutely.

Q. How did you overcome those problems in those tests?

A. Well, usually you can overcome those problems by gavaging the animals. So you put the test substance into corn oil and you use a tube and you put the corn oil directly into the stomach of the animal.

And then when you do the pathology at the end, you put less weight on what you're going to see in the stomach because you know you will have some concerns about what's happening in the stomach, but you're looking for systemic effects into other parts of the body.

Q. In your opinion, sir, would a long-term rodent carcinogenicity study on formulated Roundup be helpful

in assessing whether or not Roundup causes cancer?

A. It would be unique in the literature so it would probably be very helpful.

MR. WISNER: Thank you, Your Honor. That's all we have.

MR. ISMAIL: Thank you, Your Honor.

CROSS-EXAMINATION

BY MR. ISMAIL:

Q. Good morning, Dr. Portier.

A. Good morning.

Q. The testimony you referred to in response to Mr. Wisner's questions was taken in January of this year; correct?

A. I can't be certain.

Q. Sure. And you know you testified in the *Johnson* trial; is that correct?

A. Yes, that is correct.

Q. And you recall from that trial that experts for Monsanto raised this very issue about the question of whether a long-term rodent study in a formulated product would be feasible?

A. I wouldn't know that.

Q. That occurred after you left the stand. But that trial was about a year ago; right?

A. The *Johnson* trial?

Q. Yes.

A. Nine months.

Q. Sure.

A. Eight months.

Q. And with respect to the substance of your testimony, sir, you said that a long-term carcinogenicity study on the formulated product would be unique; is that what you said?

A. Yes, in the literature.

Q. Because that's never been done in over 40 years that glyphosate formulations have been on the market; correct?

A. As far as I know.

Q. Are you aware of any regulatory agency in the world who's ever requested a long-term carcinogenicity study on formulated products?

A. I am not.

Q. When IARC did its review, did it note that it would like to see a long-term rodent study in the formulated product, to the best of your recollection?

A. They don't usually put that type of recommendation into their reports.

Q. So the answer to my question is what, sir?

A. It wouldn't be in there.

Q. And it isn't in there; correct?

A. Correct.

Q. Are you aware of any regulatory body in the world who has requested of any manufacturer of a glyphosate formulation that they do a formulated product long-term rodent study?

A. I'm unaware of any request of that type.

MR. ISMAIL: Thank you, Doctor.

MR. WISNER: Short redirect, Your Honor.

THE COURT: Yes.

REDIRECT EXAMINATION

BY MR. WISNER:

Q. Doctor, are you familiar with the recent EPA issue paper in 2017?

A. Yes, I am.

Q. All right.

MR. WISNER: This is Exhibit 3036, Your Honor. I'm going to quickly show it.

(Exhibit displayed.)

BY MR. WISNER:

Q. And Dr. Portier, is that the issue paper we discussed just a second ago?

A. Yes, it is.

Q. Okay. I'm going to turn to the conclusions of the paper.

Specifically I'm going to look at page 145.

And as you can see right here, there is a paragraph talking about collaborative research plan for glyphosate and glyphosate formulations. Do you see that?

A. Yes.

Q. And then the first paragraph reads:

> As previously mentioned, some have believed that glyphosate formulations may be more toxic than glyphosate alone. Glyphosate has been studied in a multitude of studies and there are studies that have been conducted on numerous formulations that contain glyphosate; however, there are relatively few research projects that have attempted to directly compare glyphosate and the formulations in some experimental design -- in the same experimental design. Furthermore, there are even less instances of studies comparing toxicity across formulations.

Have you read this before?

A. Yes, I have.

Q. And does the EPA go on to say that they're actually going to be working with the National Toxicology Program to study formulated Roundup?

A. Yes, it does.

MR. WISNER: Thank you. No further questions, Your Honor.

THE COURT: Did you --

MR. ISMAIL: No, Your Honor, just on the question of the adequacy of the disclosure, counsel referred to a document in 2017 which apparently goes to the substance of this newly disclosed opinion.

This was an issue in the *Johnson* trial. This was an issue specifically as referenced in the testimony from three months ago. So with all the reasons we've already previously -- this late disclosed opinion is not proper.

MR. WISNER: Your Honor, we stand on our argument. They're raising impossibility rebuttal to their failure to test. Dr. Portier is uniquely qualified to offer opinions about that. His opinion, I think, is well within the 10-yard lines of what he's allowed to testify about. And so I asked -- be asked to be allowed to offer this testimony in his direct examination in lieu of having to recall him for rebuttal.

And maybe we should have Dr. Portier excused.

THE COURT: Yes, I'm sorry. Dr. Portier, you're excused from the stand.

THE WITNESS: Thank you.

THE COURT: You can return to your seat.

All right. So we had a discussion in chambers and we've now had the 402 hearing regarding the issue of whether or not Dr. Portier should be permitted to offer a rebuttal opinion and whether or not it is true rebuttal opinion on the feasibility of a carcinogenicity study on Roundup, the formulated product.

I will allow limited testimony, and that is just the opinion that he offered regarding of the ability -- I don't want any references to Seralini at all. And I think it would be limited to really the last two or three paragraphs of his testimony, and that's it.

MR. WISNER: Yes, Your Honor.

THE COURT: Okay.

So I'm going to call the jury in and we'll get started.

(Proceedings continued in open court in the presence of the jury at 9:42 a.m.:)

THE COURT: We're back on the record.

Welcome back, ladies and gentlemen. I hope you all had a long restful weekend.

So we are going to proceed with testimony this morning. The plaintiffs will begin to present their case.

The alternate juror, [redacted], had a medical

emergency, and I have spoken with her doctor and verified the medical emergency. So she will not be able to participate. So we're going to proceed without her.

So, [redacted] if you would like to move your seat to see better, feel free to do that.

All right. Mr. Wisner, you may proceed.

MR. WISNER: Thank you, Your Honor.

At this time the plaintiffs call Dr. Christopher Portier to the stand.

CHRISTOPHER PORTIER,

called as a witness for the plaintiffs, having been previously duly sworn, testified further as follows:

THE COURT: He's already been sworn in. You may proceed.

MR. WISNER: Your Honor, I have a binder of the exhibits that will be used during direct. May I hand them up to the Court?

THE COURT: Yes.

MR. WISNER: Your Honor, also permission to approach the witness with a copy of the binder as well.

THE COURT: Yes.

MR. WISNER: May I proceed?

THE COURT: Yes, you may.

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DIRECT EXAMINATION

BY MR. WISNER:

Q. Good morning.

A. Good morning.

Q. Would you please introduce yourself to the jury.

A. Good morning. I'm Dr. Christopher Portier. What else would you like to know?

Q. We'll go through your background in a minute. Where did you come from? Where are you living right now?

A. Currently I'm temporarily living in Sydney, Australia.

Q. And what are you doing there?

A. My wife is on sabbatical from the University of Bern, which means she's taking a break from being a professor and doing some research and other things, and I went along with her.

Q. What's the time zone difference between here and Australia?

A. Six hours.

Q. Is it ahead or behind?

A. That's complicated. They're ahead of us by 18 hours. So it looks like they're behind us by six, but they're a day ahead.

Q. Okay. So it's really like 3:00 a.m. in the morning for you time-zone-wise?

A. Roughly.

Q. If you need any breaks, let me know as we go through this; all right?

A. Okay.

Q. Let's talk a little bit about your educational background. Are you a doctor?

A. Yes, I am.

Q. What sort of doctor are you?

A. I got my Ph.D. in biostatistics from the University of North Carolina in Chapel Hill.

Q. And what is biostatistics?

A. It is the use of statistical methods in the evaluation of biological experiments and biological observations. So it's a specialty in statistics.

Q. Okay. And when did you get your Ph.D.?

A. 1981.

Q. What did you do before that?

A. I went to school at Nicholls State University in Thibodaux, Louisiana where I got a bachelor's degree in mathematics with a minor in computer science.

Q. What drew you to biostatistics coming out of college?

A. My brother was at the University of

North Carolina in biostatistics, and he convinced me that's where I needed to go.

Q. And while you were at the University of North Carolina, what did your research focus on?

A. My Ph.D. thesis was on the optimal design of a two-year animal cancer study. What I was looking at was you have a limited amount of resources and you have two questions you really want to ask from that one study. One question is: Does it cause cancer in these animals? And the second is: Will it cause cancer at lower exposures? And they compete for the resources to answer the two questions so it's a question of balancing those resources to get the best possible design.

Q. And that dissertation you wrote on long-term rodent studies, has that been used in any way after your time at North Carolina?

A. Yes. It's still pretty much the standard design that's used for animal cancer bioassays globally.

Q. You said "cancer bioassay." What is that?

A. A bioassay -- an assay is a test. A bioassay is a biological test. A cancer bioassay is a two-year of chronic exposure, it's long-term exposure in rats and mice, study where the chemical is administered.

Q. Now, Doctor, what did you do after you finished your Ph.D.?

A. I went to work for the National Institute of Environmental Health Sciences in Research Triangle Park, North Carolina. I was a staff biostatistician, what they call a principal investigator.

So I was doing more work on how to analyze animal studies that pertain to environmental issues, how to design those studies to get the most information you can get out of it, things like that. That was the first few years.

Q. Just for the jury's education, what is the NIEH?

A. The National Institute of Environmental Health Sciences is one of the National Institutes of Health of the United States. NIH gives research grants to everybody in the United States doing work on medical issues, issues relating to human health. NIH's focus is on environmental issues related to human health. And so they have an in-house research unit, and then they give the grants to universities around the country. And so I was part of that in-house research unit.

Q. And so you said you were a principal investigator for the first few years. Did your role ever change?

A. My role at NIEHS changed over multiple years, but I was always a principal investigator during the

time I was there.

Q. What was your next position as well as principal investigator?

A. Next position was to be the branch chief of the laboratory of quantitative biology and risk analysis. I have to remember, that's a long time ago.

We basically had computational chemistry where you take chemical molecules and try to figure out why they're causing biological effects so you look to see how they bind to certain proteins or things within the body.

We had a molecular biology group in there. We had my unit which was risk assessment and analysis and figuring out how much the effect is on the human population.

Q. Let's cut to the chase. How long were you at the NIEH?

A. 34 or 33 years, something like that.

Q. And during -- and before you left NIEH, what was your role back then at the institution?

A. Well, I was at one point associate director of the National Toxicology Program. That's the largest toxicology program in the world. And the associate director is really the person who makes all the decisions.

The director of NIEHS is also the director of the National Toxicology Program, but they give all the responsibility to the associate director.

I was also the associate -- I was also the director of the Environmental Toxicology Program. So the National Toxicology Program is sort of outside of NIEHS, it has its own budget. But within NIEHS, they have research units that feed back and forth to the National Toxicology Program. That's the Environmental Toxicology Program. So in essence I was in charge of all toxicology for NIEHS.

Then after that, I was associate director of the NIEHS. I served as the senior scientific advisor to the director on what programs to do, how to move forward. We looked at things like children's health and set up a program for children's health, climate change in human health, we set up a program for that. Those types of issues.

Q. I want to talk to you about some of the products you worked on.

Before I do that, during your time in education, did you ever have any focus in any way on epidemiology?

A. Yes. My Ph.D. is in biostatistics but with a minor in epidemiology. My master's thesis was designing

an epidemiology study to look at the health impacts of power lines, for example.

Q. So I want to talk about some of the work that you did at the NTP and NIEHS. I said that wrong -- no, that's correct.

The first thing I want to talk about is did you, in your capacity as a researcher and associate director, look at the causes of human cancer?

A. Oh, yes. Most -- probably the majority of my published works deal with cancer-related issues.

Q. And during your time -- I'd like to talk about some of the products you've worked on. You mentioned power lines and childhood leukemia. What was that project about?

A. Well, that was funny because I did it in my master's thesis, and then 20 years later there were actually epidemiology studies showing a relationship between childhood leukemia and power lines. And NIEHS was given the task of researching this issue, and at the end of that research, presenting a report to Congress on what we found and what we believed to be the level of certainty around the safety -- the health safety of power lines.

My task was to write that report after all of the research was done. And I also was involved in what

research we would fund and things like that.

Q. Did you ever have occasion to work on an herbicide while you were at those programs?

A. We did do a lot of work on dioxin which is a contaminant of herbicides that were used back in the 1970s and earlier '80s. And so we did a lot of work on that. And I worked with the State Department on that on some issues of international agreements on persistent organic compounds which dioxin is one.

Q. And did you do any work related to the design of animal studies?

A. I continued to work on that issue, yes, looking at timing of exposure and we did some work on, as the science advances you get new tools to advance that science, and so one thing that came in in the late -- in the '90s is what's called microarrays. These are little slides that allow you to look at thousands and thousands and thousands of genes simultaneously and see how they change in a living organism and tissue.

And so we worked on the design of putting that into a bioassay so you could use that information as part of the interpretation of the data for health.

Q. What about animal mortality? Did you do any work about how our government actually researches on animals?

A. I don't know what you mean.

Q. Specifically related to rodent studies.

A. Again, I'm not sure. They do a lot of rodent studies.

Q. Sure. So I guess my question is did you implement any procedures or policies to help reduce the number of animals that have to be sacrificed in these studies?

A. Oh, yes. Yes. Back in 2003 when I was running the National Toxicology Program, I was actually sitting and having a beer and I asked myself a simple question. If I was given the money I have now to build a National Toxicology Program, would this be it? And I concluded no, it would not.

Science had changed. The direction of science had changed. We were doing technology that was 30 years old. So I implemented an entire program, a 10-year program to just completely redo the way we approached toxicology at the NTP.

And I guess 2014 we wrote a paper talking about what happened in the 10 years. And it was fairly successful. It did change the face of toxicology, and still changing.

And it uses a lot of in vitro work. So cells, doing studies in just cells to characterize toxicity

before you start thinking about doing any animal studies. And you use what we've already learned from animal studies and these in vitro studies to categorize things before you even start so that sometimes you just don't have to do a study.

Q. All right. I want to talk about a little bit after you finished at the NTP. Where did you go after that?

A. From NIEHS I went to the Centers for Disease Control and Prevention in Atlanta. There are centers there like NIH has institutes, CDC has centers. I was director of the National Center for Environmental Health, their environmental health center. And I was also the director of the Agency for Toxic Substances and Disease Registry, ATSDR.

And like the NTP is sort of a separate entity from NIEHS, ATSDR is sort of separate from CDC but it's also connected to CDC. So I ran both organizations.

Q. So double the work, same pay?

A. Exactly.

Q. Okay. Are you retired now?

A. From that, yes.

Q. And when you were at the CDC and the ATSDR overseeing the environmental health programs, what sort of projects did you focus on at that point?

A. Well, they do national biomonitoring study. So every two years they take sample of blood from people in the United States and they characterize what chemicals are in the blood and they provide that as information for everyone to look at.

We had lead poisoning prevention program. We had an asthma prevention program. We did Superfund sites around the country and evaluated the potential for human health impacts at Superfund sites and then advised EPA on whether to clean them up or not. There's a formal linkage there, legal linkage. Things like that.

And then we responded to a number of national emergencies. Fukushima, we were involved with dealing with the Fukushima problems. We were still dealing -- when I started, we were dealing with the Deepwater Horizon accident in the Gulf of Mexico. Other things like that. Public health work.

Q. What year did you leave -- what year did you retire?

A. 2013.

Q. And since 2013, have you continued to do research in the areas of human health?

A. Yes, I have.

Q. I'd like to talk to you about one of these projects that I was looking through your CV, it was

actually a project here in Oakland; is that right?

A. That's correct.

Q. Could you please describe to the jury what that project was.

A. I work part-time for the Environmental Defense Fund. It's a nonprofit, nongovernment organization that is basically dedicated to bringing greater science into policy decisions in the United States.

We -- when I started working for them, they were doing a project with Google looking at methane gas and measuring, using Google Street View cars to measure, methane gas in towns to try to find leaks and then to fix the leaks. It's a win-win for everybody. You fix the leaks from the methane gas. The methane is a greenhouse gas so it's bad for the environment. So you get it out of leaking into the environment. And Google had the cars. It was great. It was a great project.

And so I worked with their chief science officer and said, well, why don't we do the same for air pollution. So we went to Google and we got three Street View cars and we equipped them with air monitoring equipment, things like particulate matter and ozone and things like that we measured in the air. And we drove them around Oakland for two years.

We wanted to see how feasible that was.

There's some serious science involved in trying to make that actually work well. Because you have cars in front of you emitting pollutants, you've got trucks emitting pollutants, you've got the dock, the ports that emit pollutants, and you want to try to figure out all of this stuff. But after two years, we did a pretty good job of working here in Oakland and figuring that out.

And then we did a human health study. We worked with Kaiser Permanente and looked at all of their insurees in Oakland area and compared their exposure based on our Street View car runs against the diseases they've seen and showed that you could actually see differences by neighborhoods which had never been seen in the literature before because no one ever did this high quality of a map of the air pollution in the city ever before.

Q. So after you were able to map air pollution in Oakland to specific health outcomes in the town, did you expand that study in any way?

A. Yes. We're now doing the Bay Area. So we've got two cars driving up and down all over the place from Richmond to San Jose. We're doing Houston, Texas. And we've got a project going in London, England right now that's not with the Google Street View cars, it's a different partner, but nonetheless it's the same basic

idea. And we're now looking for a fourth city.

Q. Now just to be clear, using this data that you've collected, could I type in my address and see, oh, what's the pollution right there in that area? I mean, how detailed is this?

A. Oh, it's detailed. You can go on our maps and take a picture, take a look. You don't have to type it in. You can just look. And based upon the levels, you can figure out what you're exposed to.

Q. Doctor, you've been retired for almost six years. Why are you doing this work study?

A. It's fun. I like doing what I want to do. It's a challenge. It's a new field for me. I was not -- I did not do a lot of work in air pollution before then. So it was an interesting challenge.

EDF has some other interesting challenges for me that I wanted to work on. So I worked with them.

And then I do some other things here and there as well.

Q. Now, I was looking through your CV and I notice some awards. I just want to ask you about them. The Society of Risk Assessment, Risk Assessor of the Year. What was that?

A. I don't remember exactly why they gave it to me, whether it was for a paper or something like that.

But they give that award to one person every year for the person who's sort of contributing the most to the practice of evaluating risks.

Q. And what is cancer risk assessment, just as a field?

A. It's taking all of the scientific literature, looking through it and figuring out does this data tell you you have a chemical that can cause cancer in humans, and if yes, how much do you need to be exposed to that in order to -- what's the probability of getting cancer at certain exposure levels.

Q. In the last 40 years, how much of your career, whether it be the government or even in retirement, has focused on cancer risk assessment?

A. 90 percent, I'd say, something in that range.

MR. WISNER: At this time, Your Honor, I'd like to tender Dr. Portier as an expert in cancer risk assessment.

THE COURT: Voir dire, counsel?

MR. ISMAIL: Your Honor, subject to prior briefing and the Court's rulings, we'll reserve for cross-examination.

THE COURT: You may proceed.

MR. WISNER: Thank you, Your Honor.

Q. All right. So I want to talk to you about

obviously glyphosate and Roundup. But before I do, have you ever testified as an expert about whether or not something causes cancer, in litigation?

A. Yes.

Q. I mean, not related to Roundup, before Roundup?

A. Before Roundup?

Q. Yeah.

A. No, never.

Q. So how did you get involved in all this?

A. In the Roundup cases. There was a review of five chemicals, five pesticides by the International Agency for Research on Cancer.

Q. I'll stop right there. What is that?

A. IARC. I-A-R-C. It's part of the World Health Organization. It is a separate agency within WHO. And they do a lot of things internationally to look at cancer rates and risks.

But they have what's called the monograph program. And the monograph program produces these thick volumes where they review the cancer evidence for a hazard for chemicals and other things, power lines and whatever.

And they had a working group. So they bring in a bunch of independent scientists to look over the

literature and provide an opinion on the cancer hazard for the particular compounds.

So they had a working group for these five pesticides, one of which was glyphosate. And I will point out that in the international field, glyphosate is a pesticide. And then they break pesticides up into insecticides and herbicides. So if I say pesticide for glyphosate, it's because I'm using this umbrella of what it is.

But they asked me to join them. Because of my linkage to EDF, they brought me in as a special -- in a special position where I was just providing advice to the working group but not actually voting or writing on anything. But I became familiar with the data on glyphosate.

And after that IARC review and when IARC had made a conclusion that said it was a probable human carcinogen, I was approached by lawyers I'd already been talking to for free on other things to get involved with them on this issue.

Q. So to be clear, you got involved in this because IARC asked you to be a specialist for their assessment of glyphosate?

A. Eventually, yes.

Q. Okay. I want to talk briefly about IARC.

We're going to be calling another witness later this week to get into IARC in more detail so I don't want to spend too much time on that with you, Dr. Portier, but I do want to talk a little bit about what you did. Okay?

A. Okay.

Q. Turn in your binder to Exhibit 3029. It's at the very end. It's a big binder, sorry.

A. Okay.

Q. What does this document reflect?

A. This is the first few pages of the IARC monograph for the five chemicals I was talking about. This lists membership and who was at the meeting.

Q. Okay. So it's the members -- is it a fair and accurate copy of those people that participated in the IARC monograph related to glyphosate?

A. It appears to be, yes.

MR. WISNER: Your Honor, permission to publish.

MR. ISMAIL: No objection, Your Honor.

THE COURT: Granted.

(Exhibit published.)

BY MR. WISNER:

Q. All right. Doctor, so we're looking at this document here, and as we see up here at the top, get in closer, it has IARC monographs on the evaluation of

carcinogenic risk to humans. And it says Volume 112. And as we go through, you'll see that it does reference glyphosate. Do you see that?

A. Yes.

Q. Okay. And as we go through here, there's some of the people who participated in the meeting; is that right?

A. That is correct.

Q. Now there's people called members of the working group. What does that refer to?

A. So the opinion that is offered in this monograph is their opinion. It's their opinion of the science and their overall opinion of the carcinogenicity of these five compounds.

Q. And are they the ones who vote for the ultimate classification?

A. Yes.

Q. All right. And if we go through here, we see a couple of these people, Dr. Aaron Blair; do you see that?

A. Yes, I do.

Q. It says he's the overall chair. What does that mean?

A. He ran the meeting.

Q. Okay. Do you know Dr. Blair?

A. Yes, I do.

Q. How do you know him?

A. He was at the National Cancer Institute for many years. I was at the National Institute of Environmental Health. We interacted on cancer-related issues and research.

Q. And if we go down here, we have, you know, some interesting -- we have Frank Le Curieux. Do you see that?

A. Yes.

Q. European Chemicals Agency. Do you see that?

A. Yes.

Q. Is that ECHA?

A. That is ECHA.

Q. Okay. What is ECHA?

A. The European Chemicals Agency is -- it's not like the U.S. EPA. It's sort of slightly different. Let's say it's the repository for regulatory decisions within the EU. They don't necessarily make those decisions themselves, but they own the rules by which those decisions are made.

They also are in charge of the REACH program, which could take a long lecture and I'm not going to go there, in Europe which is looking at chemicals used in commerce and registering them. So that's basically what

they do.

Q. And I see here just below the ECHA person, we have someone from the U.S. Environmental Protection Agency. Do you see that?

A. Yes, I do.

Q. And are you familiar with the EPA?

A. Yes, I am.

Q. Did you interact with them as part of your work in government for 35 years?

A. Quite a bit.

Q. Down here we have Dr. -- or Lauren Zeise from the California EPA. Do you see that?

A. Yes, I do.

Q. And did you interact with Dr. Zeise when you were at the meeting?

A. I've known Dr. Zeise for 30-plus years, yes.

Q. Now I see that there's a lot of people listed on this member list who are from governmental organizations. Is that typical in an IARC monograph?

A. From where?

Q. From various government organizations. We have National Cancer Institute. We just kind of went through them all.

A. Yes.

Q. Is that typical?

A. Yes, that's typical.

Q. Why? Why are these scientists from these government agencies brought in for IARC?

A. Well, most of these are -- they're not regulators, they're researchers at those government organizations. And they've built connections with IARC over the years. I can't tell you anything more. They're excellent researchers.

Q. Sure. And we have your name here at the bottom. Do you see that?

A. Yes.

Q. And it says you're an invited specialist.

A. Correct.

Q. What does that mean?

A. It means that I served basically as a consultant to the working group. I can look at all the science that they look at. I can provide my opinion on that science. When they provide an opinion, if mine differs, I can explain it. But the final decision is theirs in terms of what they're going to put. I'm not allowed to vote, and I'm not allowed to write anything.

Q. Okay. I also noticed up here someone by the name of Charles Jameson. Do you see that?

A. Yes.

Q. Do you know Dr. Jameson?

A. Yes, I know him very well.

Q. How do you know Dr. Jameson?

A. He worked at the National Toxicology Program, and I knew him from the minute he was there. So we'd started about the same time at NIH.

Q. And do you see it says "Subgroup Chair." What does that refer to?

A. So there are four subgroups. They break the scientists up into subgroups for part of the meeting to get through all of the scientific literature. So there's one for exposure, one for cancer and experimental animals, one for cancer in humans, and one for mechanisms. And Bill was the chair of the subgroup on cancer in experimental animals.

Q. All right. So down here under with your name you have a footnote. Do you see that?

A. Yes.

Q. It says "Christopher Portier receives a part-time salary from the Environmental Defense Fund, a United States-based nonprofit environmental advocacy group. What is that referring to?

A. That is the conflict of interest they were concerned about by having me on the working group. So instead of putting me on the working group, they put me as a specialist because I have what they perceived as a

conflict of interest.

Q. You're talking about the work you were doing like measuring air pollution, that created a conflict of interest?

A. Yes. But they worry about the entire organization. And since it's the Environmental Defense Fund, they were worried they might be doing something related to regulation of any of these products, and it gives them concern.

Q. I guess that gets to my question, then, is: To participate in IARC, do you have to not have a conflict of interest?

A. That is correct.

Q. Okay. And then in the last page here we have -- on the second page here we have a group of people, we have representatives of national and international health agencies. Do you see that?

A. Yes, I do.

Q. And it looks like there was people from the EPA that had attended.

A. Yes.

Q. That's Jesudosh Rowland. Do you see that?

A. Yes.

Q. And then we had observers. Do you see that?

A. Yes.

Q. Who are the observers? What's their role in the program?

A. Observers are parties who are interested in the outcome of the evaluation. Usually they're representing -- if the agents being looked at are propriety, they're representing the owners of the patents on those agents. If we're looking at something like viruses, it might be a medical authority or something. But they're people who have interest.

They are allowed to sit in on every -- all the meetings of the subgroups and the big group and everybody else. They're allowed to comment at certain points. But, again, they can't write anything and they can't vote or anything like that.

Q. And I see here Thomas Sorohan from the Monsanto Company. Do you see that?

A. Yes, I do.

Q. Was Dr. Sorohan an observer for Monsanto to the best of your recollection?

A. I knew he was an observer there. I didn't know where he was from.

Q. And did he participate in -- or did he make comments as you're allowed to do as part of the process?

A. Yes.

Q. All right. Do you know ultimately what the

IARC group found with regards to glyphosate?

A. Yes. They found that glyphosate was what is known as a probable human carcinogen, and that has a very specific definition by IARC's review rules.

Q. And did they look at specific categories of science before arriving at that conclusion?

A. Yes. They -- as I said, they break into four specialty groups. Exposure is off by itself. It's not part of this decision. It's just to collect the information and make sure it's there for everybody to look at.

But the other three groups, all the literature is reviewed very systematically: Human cancer health risk, animal studies, and mechanism studies.

Q. And is that process of looking at those three pillars of science, is that how people typically look at cancer risks?

A. Yes, that's quite common worldwide.

Q. Okay, great. So I want to put up a document.

All right. So this is a demonstrative we've put together here. It's Exhibit 107.

MR. WISNER: I'm sorry, Your Honor. Do you want to see it before I showed it? I don't think they object.

MR. ISMAIL: We do not, Your Honor.

THE COURT: If you don't object, that's fine.

(Exhibit published.)

BY MR. WISNER:

Q. So we have here what we call the -- and I showed this to the jury in my opening statement -- the three pillars of causation. Okay.

And you said there's three of them. And the first one you discussed is -- we'll start off with animal studies. Is that a fair thing?

A. That's fine.

Q. Okay. What are animal studies?

A. Basically you are looking -- the concept is this. If you see that a compound can cause cancer in an animal, your concern about it causing concern in humans is much higher. If that animal is a mammal, it's much higher.

So what animal studies are, are typically they're rats and mice. They're exposed for a large portion of their lifespan. And at the end of that period, they're examined to see if they've gotten any cancers and they're compared against animals that are not exposed so that you can figure out whether the chemical is causing cancers in these animals.

Q. We're going to talk a lot about them in a minute.

A. Yes.

Q. But I just want to get an overview.

All right. And the next one, what would you put in the middle here, Doctor?

A. I would put epidemiology in the middle.

Q. Okay. And what is epidemiology, sir?

A. Typically -- it can go beyond this, but typically epidemiology is the study of human populations to understand what causes disease in those human populations.

Q. Okay. And then in the last one, what would you call that, sir?

A. Mechanism.

Q. Mechanism. Okay.

And can you please explain to the jury what mechanism studies are, data is?

A. Sure. When you suspect something causes cancer, you're trying to build a case scientifically to convince yourself and other scientists that indeed it really does cause cancer.

So you want to -- you want to try to figure out why it's causing cancer. And so you do a series of studies that tell you about what the chemical is doing in the cell at the molecular level, at the chemical level in the cell, and you use that to try to paint a

picture of how that cell is going from being normal to becoming a cancer cell.

And so that's what mechanism is looking at.

Q. Now, Doctor, did you look at all three of these pillars?

A. Yes, I did, for glyphosate.

Q. Fair enough. And Roundup as well?

A. Yes.

Q. And just from a scientific and methodological perspective, would it be appropriate to opine about whether or not glyphosate or Roundup causes cancer, ignoring any one of these three pillars?

A. No.

Q. Why?

A. Because all of the scientific evidence plays a part in building the picture of whether there's support for the concept of cancer from glyphosate or no support for cancer from glyphosate. You've got to look at all of that information simultaneously.

Now, that doesn't mean it all has to be there. If I have positive animal studies, some epidemiology, but no mechanism, I still may convince myself that this is really a cancer hazard.

So it's -- but you want to look at everything to get a real decent feel for what the literature is

telling you. And that's what all the regulatory agencies supposedly do.

Q. And have you looked at all these different pillars of science as it relates to Roundup and glyphosate?

A. Yes, I have.

Q. And we're going to go through everything you looked at in a second. But let's just be very clear. In your opinion, does Roundup cause non-Hodgkin's lymphoma in humans?

A. Probably, yes.

Q. All right. Let's go through each one of these one at a time. Let's talk about animal studies.

And I understand, sir, that you've actually helped put together a little short PowerPoint to walk us through what animal studies are; right?

A. Yes.

Q. Okay. And if you go to in your binder it's Exhibit 106. It's a printed out copy of that.

A. Okay.

Q. So is that a fair and accurate copy of this tutorial you put together?

A. Yes.

MR. WISNER: Your Honor, permission to publish?

THE COURT: Any objections?

MR. ISMAIL: As a demonstrative, no objection.

MR. WISNER: It works.

(Exhibit published.)

BY MR. WISNER:

Q. All right, Doctor. So we have this PowerPoint. It starts off with rodent studies, and it says humans share 95 percent DNA with rodents.

Why do we use mice and rats when we're looking at issues like cancer?

A. There's a lot of reasons. First of all, they are very similar to humans in the makeup of their DNA. They're very similar to humans in the biochemistry of what's happening in the body.

There are clearly differences. Rats and mice are not humans. But they're similar enough that they can be used as bellwether animals to test hypotheses about dangerous toxic chemicals.

They don't live as long as humans. So they're shorter lived, which makes them good for a laboratory experiment. If we used dogs or cats, it would be much longer experiments because they're very long-lived. These are very short-lived animals.

Q. Can I ask you a question about that?

A. Sure.

Q. You say they're short-lived, but do they live an entire lifetime?

A. Almost, in these studies. Most of them are in old age when the studies are ended. And when the studies are ended, any remaining animals are usually sacrificed.

Q. So, for example, humans live, you know, 80, 90, whatever years. What is the equivalent of that in mouse years, for example?

A. It depends on the strain of mouse or rat, but as a typical rule a mouse will live 26, 28 months at most. And a rat may live a little longer than that.

Q. Okay. So we're looking at two, two and a half years for the lifespan of these rodents?

A. Correct.

Q. All right. Sorry. You were explaining why we use them. You said the life span helps us actually study them. Why is that?

A. Oh, because you can do it in a reasonable period of time. If you have to do every single chemical you're going to study and have to do a study that's going to last eight years before you look at the results, that's a very long time. And if we're talking about a toxin, then it remains in the environment or people continue to get exposed while you're waiting for

this study. It's much better to do it faster.

These are standard models for studying cancer. They're used worldwide. They're widely accepted. You use not just the rat or mouse that lives on the corner under your house, these are specially bred animals. You know a lot about their genetics. They've been studied for years. There's somewhere around 200 strains that are commonly used in laboratories.

One of the things we did while I was at NTP was do a complete gene sequence on all 200 of those species of rats and mice, strains of rats and mice.

That's basically it.

Q. Now it says right here at the bottom, it says "Mouse models are commonly used to develop drugs for lymphoma treatments."

What does that refer to?

A. So unlike testing for toxicity like we're doing -- we do mostly in toxicology, in medicine you want to develop a new drug. And so you begin to develop that drug. Let's say you want to develop a drug for lymphoma. You want to treat people.

So then what you do is you develop the drug. You do some work with cells, make sure you think it's working right. And then you have to do some work in animals to show that it's going to be efficacious and

safe. So you have to have both of those. It's got to do what you say it does, and it's got to be safe.

And there are mouse strains that get lymphomas very readily. So 50, 60 percent of the animals will get lymphomas. So that's a useful strain for studying human treatments of lymphomas because, one, you've got a bunch of mice with the disease and they get it readily, and you can treat them and see if you can control it, stop it, make it go away, whatever. So that's what that type of model is used for.

Q. All right. And let's walk through a typical rodent study. Okay? We'll use a mouse, a CD-1 mouse as an example.

What is a CD-1 mouse?

A. Again, it's a substrain of mice. I guess I didn't look exactly where it derives from, but it's going to be derived from some other substrain and crossed, and then it's maintained like that for years so that if I do a study in my laboratory with a CD-1 mouse and you do a study in your laboratory with a CD-1 mouse, we hopefully will get the same results because it's the same mouse. They're genetically very close to each other.

Q. All right. So mice are placed in groups where they are treated identically. What does that refer to?

A. So when you do a chronic cancer study, a cancer bioassay, you're going to take a bunch of rats and mice. Well, in this case we'll take mice. And you want them to be somewhat identical so you're choosing CD-1 because it's an inbred strain, it's very identical animals.

You're going to have males and females. They don't get housed together. They get housed in separate rooms because it messes up the study if they're too close to each other. That's biology.

And you put about 50 of them in each group. They're not all put in one cage. The caging is very complicated in terms of how you cage these animals. But they're not all thrown in one cage.

And they're randomized to which group they're going to go to. So you get on the computer and you generate a random number, and it says 17, and you take animal number 17 and that's in the control group. And then generate another one, it's animal number 25. And that goes in the control group. And you do that until you have 15 in the control group. Then you generate more random numbers and you fill the other groups.

The idea would be that if there's any slight difference between the animals, you've gotten rid of it by just randomly putting them around.

Q. Now, they're treated identically. I mean, how carefully controlled is the living environment for these animals?

A. If you're following OECD guidelines or NTP guidelines, then it's extremely controlled. You control the air flow. You control the entrance and exit of the -- from the cage -- from the room where the animals are kept. You control their feed. You check their feed every day. You check their weight every day. You check to make sure they're not looking ill every day. It's just a completely controlled process.

So that the idea is the only thing that can affect cancer rates in these animals is the exposure itself and not something else that you're doing that you shouldn't be doing.

Q. All right. So these animals, you say -- you mentioned this now, that they're broken up into different groups. How many groups are they typically broken into?

A. The studies we're going to see, it's typically four groups. There is one study with five groups, but all the rest have four treatment groups. One is control, and those are animals that get exactly the same thing as the other animals but without the glyphosate.

And then they have low-, middle-, and

high-dose groups. And those get glyphosate.

Q. And why are they fed glyphosate? Why aren't they given some other form of exposure?

A. It's convenient. It's the route that humans will, to some degree, be exposed to glyphosate. Beyond that, I don't know what their reasoning for choosing feed was. I don't have access to the original reports on these. But I assume it's just convenient and it works well.

Q. Now, for the purposes of this experiment, these animal studies, are the animals in the control groups treated any differently than the animals in the other dose groups other than glyphosate?

A. No.

Q. Okay. So now we have low dose, mid dose, high dose. How are those doses established?

A. Usually in some sort of geometric progression. Oh, you mean, how do we get -- oh, that's the next one, yes.

Q. Let's start with the high dose.

How do we determine what dose we're going to give these animals in this experiment?

A. So if you're going to do a study for two years, you don't want to blow it. You know, if you choose a dose that's too high, you could kill the

animals in the first -- kill the animals at 18 months. Let's say you do that. That would destroy your experiment.

So you have to make sure you choose a dose that is not going to destroy your experiment. And usually what's done is called to identify the maximum tolerated dose.

You take these animals, the same types of animals and you put them in a shorter experiment. The first experiment is usually two weeks. You expose them for two weeks, and you look to see if the stuff makes them sick at varying levels. And it's a wide range of levels in the two-week study.

And then from that information, you design a new study with fewer doses, maybe as many as five, but that one is now 90 days, three months. And you run it for three months. And after three months, then you examine it very, very carefully to see if you see any type of toxicity occurring in three months that wouldn't be related to cancer. So you don't want to see them losing weight. You don't want to see them having hind legs that don't work because they're having neurological problems. All kinds of things like that you look for very carefully.

Then from those doses, you choose the highest

you can choose that doesn't seem to be harming the animal in 90 days, and that becomes the high dose in the study.

Q. Why are you trying to get to the highest tolerated dose? What's the purpose behind that?

A. So ultimately when you're doing these studies, your interest is human health, not the health of rodents. And we don't want to see human populations -- the standard in regulation in environmental exposures is a probability to the population of one in a million or one in 100,000.

That means if we allow this chemical into the environment, we might see as many as one person out of every million get a cancer from it, but that's it. And that's how they would set the exposure limit.

The problem is I can't study a million rodents. That study is just too big. It's been tried with 40,000 rodents, and even that was too big.

And so what you do is, the belief is, as you increase the exposure, you increase the probability of seeing cancer. And so you want to use the highest exposure you can get so you have the highest probability of seeing cancer. And then if you see it, you have to somehow now extrapolate back down to lower exposures to figure out what goes on at lower exposures.

But you want to, at least for safety assessment for testing to see if it can cause cancer, you want to go as high as you possibly can.

Q. All right. We're going to talk about that a little bit later in more detail about specific studies.

All right. So that gets us to the high dose.

How do we figure out the other two dose groups, the low-dose and the mid-dose groups?

A. Well, that was a good bit of my Ph.D. thesis, is digging that up. And most people use a sort of geometric progression. Here you can think of this as factors of three in this case. You start at the MTD, and then one-third of that, and then one-third of that again, which would be one-ninth but we have 10 fingers so we always go down to one-tenth instead. And this is the classic type of experiment we're looking at here in these studies.

Many of the MTD studies are more like a half and a fourth instead of a third and a tenth, but that's the type of progression you use.

Q. And in these studies that relate to glyphosate that we're going to be talking about in a minute, do most of them use the one-tenth, one-third MTD model?

A. Something along that line. We have some modifications. Some of them go one-fifth,

one-twentieth. But basically it's in that range.

Q. Why do you have these groups then? Why do you have lower dose groups? What's the idea behind that?

A. Two reasons. One is you want to -- you not only want to see a positive response at some dose, but you'd like to see climbing response if at all possible so you can see it increases as the exposure increases. That strengthens your belief that what you're seeing is really due to the exposure.

But also, as I mentioned before, you want to get down into that low exposure range, and by having three points on a curve you can at least draw a line and try to make a guess at where the low exposure safe point would be.

And so that's why it's done.

Q. Have you ever heard the expression "dose makes the poison"?

A. Oh, yes.

Q. What does that refer to in toxicology?

A. It's the belief that you can make anything toxic if you give a high enough dose. And that's probably conceivable. But that doesn't really apply here. Because you're using the preliminary studies to rule out any other forms of toxicity, that's not what's happening here.

These cancers are arising because of the compounds, not -- certainly it is dose-related. You wouldn't detect it in 50 animals if it wasn't a strong response. But nonetheless, it's -- this is not at the level of it being toxic simply because it's toxic. You try to rule that out.

Q. Would it be appropriate from a scientific perspective for somebody who's been studying this issue for 40 years to say, well, hold on, that high dose is way too high for what human experience so we should ignore it?

A. That would be an inappropriate -- just saying that and not looking at the rest of the science, that would be very inappropriate.

Q. Why?

A. Well, because it's -- there's literature out there that supports the fact that when you see cancer in rodents, you're likely to see cancer in humans. And so you can't just ignore that literature and say, well, but we want to do the studies at the level of human experience, and, okay, if you see it positive at the level of human experience in one of these 50-animal studies, that means 10 percent of the human population is probably getting cancer from it. That's an unacceptable risk.

So you can't study it at those levels. You have to go higher.

Q. Okay. So how old are these mice when they start in this study?

A. They start at six weeks old. That's just when they've been weaned from their mothers so they're no longer nursing at that point. Most places buy their animals from a place that specifically grows these animals. And that's a good age at which they can be shipped, five to six weeks.

And so by the time you get them, acclimate them, and start them on the study, they're six weeks old.

Q. And are they all essentially the same age when they start?

A. Oh, yes. The places where these are grown, the colonies, they -- when you tell them we're going to do a study, they start to build up their colony and they get them synchronized so the females are all giving birth at approximately the same time so that when they get the animals, they're all going to be the same age.

Q. Are these animals healthy at the time they start the experiment?

A. Yes.

Q. And then these experiments run for mice how

long?

A. 18 months to two years depending on how they design the study.

Q. And then after two years or 18 months, depending on the study, what do you look for?

A. You look to see if animals have tumors and how many animals have tumors and sometimes how many tumors those animals have. Because sometimes you get multiple tumors in the same animals. And you count them up and you evaluate that information.

Q. Now you say count them up. What does that actually involve? I mean, is it just, hey, I see a tumor, that's one? Or is there something more to it?

A. There's much more to it than that.

When an animal dies or it is sacrificed, killed intentionally, a full autopsy is run on the animal. It's called a necropsy for an animal, so I might use that word later. And all the tissue is examined to see if there are any lumps that look like tumors. And then the pathologist goes in and takes slides of all of those lumps, looks at them under a microscope to decide if it really is a cancer or not a cancer.

In addition, there's protocols in place that certain slides are taken in every tissue. So the lung

will be removed and a certain cut will be made in the lung in one direction and another cut in another direction. Those will be put on slides. And a pathologist will look at that entire slide to see if there are microscopic tumors in the lungs that he didn't see from the big tumors.

And that's true for 40 tissues, with 40-plus tissues in the animals, for every single animal.

Q. Now when they go through and they look for these tumors, do they just add up the number of tumors or do they look at it by cancer or organ site?

A. They look at it by cancer, by organ site, by type of cancer, by stage of cancer. Yes, all sorts of possibilities are looked at.

Q. And up here in the presentation, we have some circles that are drawn. And what are those supposed to be reflecting?

A. They're just reflecting the fact that you would see some animals with a tumor, in this case a specific type of tumor, and some animals not.

So here you've had in the control group there's one animal out of the 50 that had this particular tumor. In the low-dose group, there's one out of 50 that had this tumor. Mid-dose, two out of 50, et cetera. Correct.

Q. All right. And then I'd like to -- and then, okay, so at this point when you've done all the pathology, you've fed them for up to two years, you've studied them in this laboratory experiment, what happens next?

A. Well, now you have to analyze the data. Now you have to do a full-fledged evaluation of what this data is telling you.

Just because I see one, one, two, five doesn't mean it's beyond chance. Could that have arisen by chance?

So the first thing you do is ask yourself: Is this a chance observation? And that's where statistics comes in. Statistics has tools and methods for addressing that question directly.

Q. All right. So I want to use a real example in this case. I want to talk about the Wood study from 2009. We're going to talk about it more in detail when we go through all of the studies, but I want to talk about the results of malignant lymphoma in CD-1 mice. Okay?

A. Okay.

Q. And what does this chart represent?

A. So this is the finding for that tumor in this study. This study used that dosing one-tenth of the

MTD, one-third of the MTD, and the MTD, and saw no tumors in the control, one in the low-dose group, two animals with tumors in the mid-dose group, and five in the high-dose group.

So that's exactly the counts that came out of the evaluation of the pathology in those studies.

Q. Let's make sure I get this. So this is -- there's four groups; is that right?

A. Uh-huh.

Q. And in the control group, they're not exposed to any glyphosate?

A. That is correct.

Q. Okay. And in the low-, mid-, and high-dose groups, they're exposed at increasing levels of glyphosate?

A. That is correct.

Q. And this is for the duration of their lives?

A. In this case it was an 18-month study.

Q. So these mice weren't even fully old at that point?

A. No, not CD-1 mice. They were the equivalent of 50, 55 years old in humans.

Q. And this tumor that we're talking about is specifically malignant lymphoma. What does that mean?

A. Malignant lymphoma is it's a lymphoma, it can

form anywhere in the body so they're not organ-specific, although you note what organ you found it in, but they're not organ-specific.

And it's malignant meaning that it's very invasive into the tissue. In CD-1 mice these are very lethal tumors. And once you get one, you're likely to have many because they metastasize to other parts of the body.

So this is a very serious finding in this type of study.

Q. Now this might be obvious to some of us who live and breathe this, but lymphoma, is that the same thing as non-Hodgkin's lymphoma generally?

A. So there are similarities between malignant lymphoma in the male CD-1 mice and the non-Hodgkin's lymphoma in humans. They're both B-cell origin. Well, much of NHL is B-cell origin in humans. But the malignant lymphoma, as far as I understand it, is B-cell origin in the CD-1 mice. There are papers that discuss the closeness of the mouse malignant lymphoma to NHL and talk about the specific subtypes and how they relate to each other.

And, again, mice are used as the model of choice, not CD-1 mice because they rarely get malignant lymphomas, but in other mice as a model of choice for

therapeutic discovery.

Q. Now, the lymphoma in mice or the lymphoma in humans, are those both blood cancers?

A. Yes.

Q. Are they both cancers that involve mutations that originate in the bones?

A. Probably.

Q. All right. So we take these results and we put them on a graph like this; is that right?

A. That's typical, yes.

Q. And then I see right here we have this line that we've drawn, dose response or trend. Please explain to the jury what this is referring to. And how do you assess this result?

A. So, first a little bit about this graph.

Q. Sure.

A. The Y access, the one that goes up and down, tells you how many tumors were found in that group. Since all the groups have 50, then that's okay, but if one of the groups had 100, that would be a bad chart. You'd want the percentage instead of the exact number. But the exact number is fine here.

And the height of the bar tells you what the number was. So you can see the low-dose group has one tumor, et cetera.

The trend, or the dose response, is sort of the general direction that this is going as dosing increases. So as you can see in this picture, there's a trajectory up, upward in this.

And so one way in which statisticians test this is to test is that line you see right there, does it have a slope of zero? A slope of zero is a flat line, it doesn't go anywhere. And as the slope gets bigger, the line is going up.

And so if it's big enough that it can't possibly include zero by chance, then that says it's a statistically significant increased trend. That's a good test to use here because it is, in statistical parlance, the most powerful test, the strongest test.

Q. And what is the significance of the fact that in this actual study of mice, looking at those mice exposed to glyphosate, that we have this trend for malignant lymphoma; what does that show you as a scientist?

A. In this case, the probability of that trend being equal to zero was extremely small by I think it was below 1 percent. And that tells me that this is not due to chance and that glyphosate is indeed inducing these tumors in this experiment.

Q. And by "these tumors," you're talking about

the lymphoma?

A. The malignant lymphomas.

Q. Now we just went through one finding from malignant lymphoma in one study. How many of these types of analysis are done in just one study?

A. Oh, gee, potentially hundreds. But in practical -- in practical means, 25 to 30, give or take.

Q. And is that because you're looking at different types of tumors and organ sites?

A. Right. You typically are looking at 40 different organ sites, and some organs can have as many as three or four different types of tumors. And so you have to look at all of that information.

But I don't need to do a statistical analysis if it's zero across the board. I don't bother.

And statistics has its limitations. So I also know that if I don't see more than two animals with tumors in all the groups, there's no way statistics is going to find anything with this and so you just don't bother. You only do the things where you have enough information to do an analysis.

Q. All right. Now you have, to my understanding, gone through all the studies for Roundup -- for glyphosate; is that right?

A. That is correct.

Q. And how many total studies, these long-term rodent studies, have been done generally on glyphosate?

A. I think the number is 23. It keeps changing on me because I keep finding new studies that surprise me. But I believe the number at this point is 23.

Q. And of those 23 studies, how many of them, in your view, are worth looking at because they're scientifically reliable?

A. 13.

Q. All right. And have you -- and were those in mice and rats?

A. Yes.

Q. All right. So if you turn to your binder there, let's take a look at the -- well, actually, before I do that, I want to talk about -- before we get to the actual results of these studies, I want to talk a little bit about how we evaluate them as to cancer.

Please turn to Exhibit 940 in your binder.

Are you there?

A. Yes.

Q. What is Exhibit 940?

A. This is the current guidelines for carcinogen risk assessment for the U.S. Environmental Protection Agency. This is the book of how they do cancer risk assessment.

Q. Are you familiar with these guidelines?

A. Yes, I am.

Q. Why?

A. At one point I was involved in the writing of these guidelines for EPA. And then at another point I reviewed them before they were finally accepted and used.

Q. So you actually helped write the guidelines for assessing cancer for the EPA?

A. Yes.

MR. WISNER: At this time, Your Honor, permission to publish the document.

MR. ISMAIL: Your Honor, as long as it's going to apply for our questioning as well, that we can publish the EPA documents, I don't have an objection. But with that understanding, then we're fine, but --

MR. WISNER: Shouldn't be a problem, Your Honor.

THE COURT: All right. You may publish.

MR. WISNER: Thank you, Your Honor.

(Exhibit published.)

BY MR. WISNER:

Q. All right. So we're on a document, this is Exhibit 940.

All right, Doctor, do you see that on your

screen?

A. Yes, I do.

Q. Okay. And it says "Guidelines for Carcinogen Risk Assessment." Right? Do you see that?

A. Yes, I do.

Q. I'm going to turn to page -- well, it's 48 in the document, but it's actually -- well, it's 48 on the bottom right, but it's -- in the actual document it's 2. -- it's dash 1. It's the document --

A. I've got it.

Q. You've got it?

A. I've got it.

Q. Okay. So page 48 down here and it's 2-21 there. Do you see that? Okay.

And as we turn to the next -- at the bottom of that page actually, it says right there, it says:

> In general, observation of tumors under different circumstances lend support to the significance -- lends support to the significance of the findings for animal carcinogenicity. Significance is generally increased by the observation of more of the factors listed below. For a factor such as malignancy, the severity of the observed pathology can also affect the

significance. The following observations add significance to the tumor findings.

What is this referring to?

A. The list of things they're about to give you that add strength to a conclusion that the compound is really causing cancer in the animals.

So it's like you're building up -- you're building a bridge, and these are the pillars that go under the bridge. You don't necessarily have to have all of them, but the more of them you have, the stronger your bridge is and the stronger your belief is that this is causing cancer in the rodents.

Q. Would it be fair to say these are the guidelines that we should apply when looking at animal carcinogenicity studies?

A. Yes.

Q. So we turn to the next page. We have a list here of these -- we have a list here of those guidelines. Do you see that? That's not big enough.

A. Yes, I do.

Q. Okay. I want to quickly go through what they are and ask you why those are considerations in assessing animal studies.

The first one says uncommon tumor types. What does that refer to?

A. That refers to tumors you've either never seen before or very, very seldom see in these studies. If you're like the National Toxicology Program where you're constantly doing these types of studies, every time you do a study you have a control population, you have a bunch of animals which aren't exposed.

And so you keep track of those. You figure out how often they get cancer. And you know what a typical animal without exposures cancer patterns look like. And all of a sudden now after doing 50 studies, you come across one study and you've got this cancer you've never seen before. It's a unique cancer. It's in the lung, let's say, or wherever, but it's unique and you've never seen it before. That's now biologically significant. Whether statistics tells you anything or not, that's a very important finding because you've never seen it before.

And the only difference between that animal and the control animals is the chemical. So you have to assume the chemical caused that uncommon tumor. That's why they're very important.

Q. And in your time doing these studies in your career, have you ever had an instance where there was such an uncommon tumor that that finding alone caused concern?

A. Multiple times, yes. Multiple times.

Q. Okay. Tumors at multiple sites, what does that refer to?

A. So this is really more applying to humans than it is to the animal studies themselves, but I guess it applies to animal studies.

So if I just see liver tumors in my one study, then, okay, that's great. It's going to cause you liver cancer. That's important. But if you see liver tumors, lung tumors, increase in stomach tumors, an increase in colon tumors, all in the same animals from the same exposures, that's really telling me this is a bad flier and it's a stronger finding. So that's what that means.

When I apply this to humans, from the animal study to the human, if I see tumors at multiple sites in the animal studies, it raises concern about humans because it's so profligate in causing the cancers in rats and mice, it probably would do the same in humans.

Q. All right. We have here tumors by more than one route of administration. What's that referring to?

A. Well, sometimes the route -- so the route of administration is the way in which the animals get the chemical. And sometimes you have to force-feed the animals, sometimes you have them breathing a mist from the chemical, and sometimes you feed it to them.

If I feed it to them and they get liver cancer, and I put it in a mist and they breathe it in and they get lung cancer, that tells me something about this chemical that I didn't know before, that it's directing, acting on the point where it really comes into the body.

And so seeing more than one route of administration yielding tumors is going to strengthen your bridge. It's going to make it look stronger.

Q. It says here tumors in multiple species, strains, or both sexes. What does that refer to?

A. Well, again, your interest is humans, not the rodents. And if all I see it is in one mouse strain under one condition, et cetera, from one lab, from one exposure, that's less important than I see it in both rats and mice or I see it in mouse strain A and mouse strain B, then it's greater concern to human population because now we know it's not just one type of mammal system, but two or three or four mammalian systems.

Q. Is that also referred to as replication?

A. Not exactly. Replication would be doing exactly the same study in exactly the same way in another lab. This is more of broadening your scientific base for your decision.

Q. Okay. We have progression of lesions from

preneoplastic to benign to malignant. What does that refer to?

A. So that refers to being able to see a pattern where some of the animals have -- don't have a cancer, but they've got inflammation in the tissue that looks bad. And you also see in that same tissue some animals that have not a cancer, but the growth of a nodule that's just before it becomes malignant, a premalignant or preneoplastic lesion. And then you have some animals with a true cancer or real malignancy in there. And so when you see that progression and you see dose response on that progression, it strengthens your finding.

But that's very hard -- that one is difficult to apply because you're not looking at the animals constantly in time. So I can't look at an animal and say, oh, he's got a premalignant lesion now, let's follow him. Oh, now he's got the benign lesion, let's follow him. We can't do that because you have to look inside the animal, and we don't do surgery on the animals to see if they're getting tumors.

So this one seldom can be honestly applied.

Q. All right. There's a few more here. I don't want to talk about all of these, I want to get going here, but a couple of these are pretty interesting.

Unusual -- well, reduced latency of neoplastic

lesions, actually. What does that refer to?

A. You don't see more animals with the tumor, but they get it faster.

Q. So, for example, if you have a shorter term study where you're ending the rodent's life at like 55 and you're still seeing cancer notwithstanding that short period of time, how does that relate to that issue?

A. That would be one area. The other way you see that is with tumors you can actually see. Mammary tumors, breast tumors on the rodents, those are lumps and bumps that you can see on the outside. Skin tumors. So you can see those.

Q. All right. Unusual magnitude of tumor response, what does that refer to?

A. Huge. Every animal in the group got a tumor. And the treatment group, then the controls didn't have any. You know, it's huge.

Q. Last one here. Dose-related increases, what's that refer to?

A. Again, as you increase the dose, you clearly see an increase in the tumor response.

Q. Great.

MR. WISNER: Your Honor, I'm going to do one quick thing, and then it's probably a good time to take

a break for the morning.

THE COURT: It is.

MR. WISNER: Just before we do it because you'll see how it plays out.

Q. Now, I understand, Doctor, you've actually put all of these tumors onto a chart; is that right?

A. That is correct.

Q. Specifically for the glyphosate data?

A. That is correct.

Q. All right. And if you turn in your binder to Exhibits 103 and 101, are those -- those charts that you've put together?

A. Yes.

MR. WISNER: Okay. Your Honor, permission to quickly publish?

MR. ISMAIL: Your Honor, I do have an issue with one of them that I'd like to raise. Perhaps we can do that during the break.

THE COURT: That's fine.

MR. WISNER: All right. Let me just show -- is it the mouse one?

MR. ISMAIL: Yes.

MR. MILLER: Can I show the rat one for now?

MR. ISMAIL: Certainly.

(Exhibit published.)

BY MR. WISNER:

Q. Okay. I wanted to show it to you before the break, Doctor, because I want the jury to sort of understand what it is before.

So this is a chart. And at the top here we have a study, Lankas 1981. Do you see that?

A. Yes, I do.

Q. What's that refer to?

A. That's just the main author of the study and the year in which it was performed or reported.

Q. And we have one, two, three, four, five, six, seven columns. Do you see that?

A. Yes.

Q. So there are seven rat studies; is that right?

A. That is correct.

Q. And then under studies, we have these color boxes. What do those reflect?

A. Different tumors.

Q. Okay. So, for example, we have one here that talks about testicular interstitial cell tumors. Do you see that?

A. Yes.

Q. And that is on here because there's a finding that in the data you see related to that tumor type?

A. Correct.

Q. Okay. Now I see here that there's this trend dose -- I'll call it out. Trend dose M/F. Do you see that?

A. Yes.

Q. What does that refer to?

A. "Trend" refers to doing a statistical evaluation of whether that slope is greater than zero that I talked about earlier. "Dose" is testing whether each individual dose is different than control.

So instead of looking at the trend, you can just test the low-dose group against the control group, mid-dose group against the control group, et cetera. And so that's a different type of statistical test.

"M" and "F" are male or female. It depends where the tumors would be found. Since this is testicular, it's in the males.

Q. Okay. And we set it up so that you could actually draw on here what the results were for each one of these; is that right?

A. Correct.

MR. WISNER: What I'd like to do, Your Honor, is we could take a break and he could actually fill this chart out so we don't have to do it -- it takes a while. So we could do it over break and then we could talk about it.

THE COURT: Okay. You can explain it to me because I'm not entirely clear what we're talking about.

MR. WISNER: Sure.

THE COURT: Yes, we're going to take a 15-minute break, ladies and gentlemen. We'll be back at 20 after the hour.

(Jury excused for recess.)

(Proceedings continued in open court out of the presence of the jury:)

THE COURT: Okay. So two things.

I want to talk about your objection.

But when you were saying "fill out," tell me what you --

MR. WISNER: Yeah, so the way we've done this in all the other trials, instead of having him circle the trend and the dose and the M for each box, it takes about 20 minutes for him to do that through testimony. So I'm going to have him fill it out now and then we'll go over what's already been filled in. It's what we did in *Hardeman* and it saved some time.

MR. ISMAIL: Your Honor, people have started leaving the courtroom. Can you just remind people in the gallery to please watch conversations in the hallways.

THE COURT: I'm sorry. Yes, I did say I would

do that.

During the course of this trial, if you're attending, please don't talk about anything that you've heard in the courtroom outside, particularly if you are near any of the jurors. I would really appreciate it if you would confine your conversations to the courtroom or after you leave the courthouse altogether. Because you may run into them in all kinds of places.

I'm going to ask at the end of the day that the audience remain in the courtroom for a few minutes to allow the jurors to actually leave the courthouse. So if you would just be mindful of your conversation, that would be at the lobby, the coffee cart, anytime you're in the courtroom, please limit your conversation or just don't talk about the case at all. Thank you.

MR. ISMAIL: Thank you, Your Honor.

THE COURT: All right. I appreciate that.

MR. WISNER: Can we have --

THE COURT: You can step down.

I'm still not sure what I'm looking at, but go ahead. If he's going to do it anyway, and you don't have an objection to this one, I think that's fine. I just wanted to know what your objection was to the second chart.

MR. WISNER: I think it's the actual chart

that's the problem, not him marking it up.

MR. ISMAIL: So to address my objection.

THE COURT: Yes.

MR. ISMAIL: So, and I think Mr. Wisner knows what the issue is.

So Exhibit 101, Your Honor, is the mice studies tumor chart.

Can we excuse Dr. Portier from the discussion?

THE COURT: Sure.

MR. ISMAIL: Thank you.

(Witness exited the courtroom.)

MR. ISMAIL: So Dr. Portier has prepared several different expert reports. He's testified several times, as this Court is aware. There is a new study here that was -- that has not been part of his previous analyses, including as recently as when he testified in February -- I'm sorry -- yes, February, in Australia so --

THE COURT: So it's not part of his expert report; is that what you're saying?

MR. WISNER: Again, factually untrue. He talked about this very study in *Hardeman*. It was played to the jury. It was not an issue. And they cross-examined him about this very study in Australia. So that's not true that it's not disclosed.

The study they're referring to is the middle one here, Takahashi, Your Honor. It's the one from 1999. It wasn't known about until the Zhang publication came out, gosh, three weeks, four weeks ago, just before he testified in Australia.

We supplemented his reliance material, we told them about it. They had a chance to cross-examine him about it. And this is what the data shows in regards to that study. So it's just in another study that he's located. And they crossed him about it. So I don't think this is improper whatsoever.

MR. ISMAIL: So, Your Honor, his expert reports have very detailed statistical analyses, which studies he -- sorry -- which tumors he finds significant and why. He describes 12 rodent studies, five of them being the mouse studies. Takahashi is not one of them. It's a study from 1999.

THE COURT: Well, wait a minute. I just want to know whether or not it was part of his deposition testimony. I mean, when you took his deposition, did you guys talk about this study in his deposition? And did he submit a supplemental report opining about this particular study?

MR. ISMAIL: He has not issued a supplemental report. I believe in the *Hardeman* case, he said in the

Zhang study there's one that I didn't even know about, which is described therein. He doesn't provide any supplement either when that Zhang publication identified the study for Dr. Portier or since that describes his analysis of this particular study. That's the nature of our objection, Your Honor.

MR. WISNER: So, to be clear, he testified, "These are the results of the Takahashi study. I just learned about them in the Zhang article."

They cross-examined him about that study. They asked about, you know, what --

THE COURT: Are you talking about the trial? Or his deposition in preparation for this case? When you say "cross," I'm not sure if you're talking about in connection with his expert testimony here or in *Hardeman*. Maybe I'm unclear about that.

MR. WISNER: Let me clarify. There was no deposition taken in the Pilliod case of Dr. Portier.

THE COURT: Okay.

MR. WISNER: So all of his depositions that have been taken many times have occurred in the context of this general litigation.

THE COURT: Okay.

MR. WISNER: There was a deposition taken in Australia. It lasted three days. It was ultimately

played to the jury in *Hardeman*. The first day was my direct, the next day was their cross. And then we did some rebuttal.

THE COURT: Was Takahashi the subject of any of that?

MR. WISNER: Yes.

THE COURT: Okay.

MR. ISMAIL: Your Honor, to be clear, that was because of Dr. Portier's illness, that was his trial preservation. That was not a discovery deposition through which we could explore in a deposition format the reliance on this study and his analysis thereof. It was referenced briefly on direct examination in the trial examination.

We don't have -- and that was a month ago. If he had an analysis of that study that he could have provided us so that we could know why these are the particular tumors of interest to him, how he went about doing it statistically to analyze it, none of that is described in his several expert reports.

So to the extent this was something that was late-breaking news -- I would note it's a 20-year-old study -- to Dr. Portier, he's had ample time to actually provide the disclosure necessary that would allow us to do it rather than where we are today.

MR. WISNER: To be clear, this is a new study. No one knew about it until the Zhang publication came out. He tracked it down. He found it buried in a regulatory document later to find out what other information he could find about it.

But this was new information. The EPA didn't even look at this study because they didn't know about it.

THE COURT: I guess what I'm wondering is the opportunity to -- the parties have had an opportunity to cross-examine him and discuss this particular study, his opinions --

MR. WISNER: Yeah.

THE COURT: -- and whatever the scope of his opinions are, I guess in *Hardeman* is where it would have occurred.

MR. WISNER: That's correct. And that deposition was not just a trial deposition. They deposed him for nearly five hours and only played about an hour and a half of it to the jury. They asked questions about things he'd never seen. They did the ham sandwich stuff. It was a deposition. And that was the agreement, that they would be allowed to cross-examine him.

THE COURT: Okay. I'm not -- I don't know

what that -- what was in that deposition. To the extent that he was deposed on this in Australia, he can testify to it here. Not beyond that. But to the confines of whatever was discussed in Australia.

And I don't know how much he knew at that point, if he knew anything, or, you know, how broad his testimony or knowledge was, but that's what he can say here is what he said there.

So to the extent that the defendants have had an opportunity to cross-examine him and talk about his opinions, if he has any about the events, those are the confines of what he can say here.

MR. EVANS: And, Your Honor, briefly on the rebuttal point, you heard earlier Mr. Wisner said that this was raised by Dr. Reeves in January. I have the transcript in the *Johnson* trial. This issue with respect to feasibility was specifically addressed by our expert there. He's had nine months to supplement a report to do a proper disclosure, and it's not been done. And I just think it's fundamentally improper to say rebuttal when this very issue was raised in the *Johnson* trial.

And I'm happy to read the transcript. This is Dr. Foster on direct examination:

"Q. And it's been suggested that

Monsanto should have done long-term carcinogenicity testing in formulated product. In your opinion, would that have been scientifically feasible?

"A. I've not seen it done with any other pesticide, and I would anticipate that it would be very difficult to carry out, primarily because as you start increasing the concentration, you're getting into that high dose of a thousand milligrams per kilogram, you're increasing all the surfactants that are present in it. So things like soaps, that would have a very adverse effect upon the GI lining of the gut. The animals just wouldn't be able to survive.

"Q. So they'd be having toxic effects from the surfactants before you could tell anything about the carcinogenicity of the compound; is that your opinion?

"A. My opinion is that the data would be derived from them would not be interpretable."

Now that was raised in the *Johnson* case. So if they want this witness to address that, they need to do it the proper way which is to disclose it. And it's just not rebuttal at all when we raised the issue nine months ago. Completely anticipatable. It's not rebuttal.

MR. WISNER: Respectfully, Your Honor, that opinion that they just read to the Court was not disclosed by Dr. Foster, their expert at the time. And we actually thought about calling Dr. Portier back, but again he was out of the country and we decided we didn't need to deal with it.

Now that I know they're doing this, I'm seeking to have him offer an opinion in rebuttal to this very point now that I know that they're presenting it.

Dr. Foster was not on their witness list. He's not coming. So I didn't think it was an issue. And then they designated the testimony affirmatively of these specific witnesses, and that changed the dynamic. And that's why I'd like to have him offer his rebuttal opinion.

THE COURT: So, first of all, I've ruled.

MR. WISNER: Okay.

THE COURT: Second, to the extent that you guys come in here and "it's a brand-new day" and "I

never knew," "I have no idea," this is trial number three. These things are all out there.

So to the extent that the parties have known about this, have met and conferred or not met and conferred but thought about it in another trial, I mean, you're all blurring the lines. And I'm not throwing any shade on anyone in particular, but you're all blurring the lines in terms of relitigating what happened in *Hardeman* or relitigating what's happening in *Johnson* or who knew what which constitutes sufficient notice. I'm going to have a very limited amount of patience for all of that. Because this isn't anybody's first rodeo.

And I agree, there's lots of rules and regulations regarding how one prepares for trial and how you present your evidence at trial. But you can't decide you're going to blur some lines and not others or come in one day with "I was just so shocked," when in fact a year ago you all knew about it.

You know, you can sort of replay the battles if you want. But you also can't come in and expect me to adhere to the bright-line rules when nobody else is playing by them.

So I'm just going to ask everybody to have some respect for the fact that this is a separate trial. However, you know, from the very beginning, including

motions in limine, it's all about what was done in *Hardeman* or what was done -- I guess not in *Hardeman* but really what was done in *Johnson*. And you're citing all the things you did as authority for the proposition that, "Gee, I should be able to do it this time."

So I'm losing a little patience with that. It's early in the game, but just be aware that you really need to figure out which rules you're going to follow and not follow. But I'm taking notes.

Thank you.

MR. ISMAIL: Thank you, Your Honor.

THE COURT: Everybody needs to clear the courtroom so they can take a break. Thanks.

(Recess taken at 11:15 a.m.)

(Proceedings resumed in open court in the presence of the jury at 11:37 a.m.:)

THE COURT: You may resume, Mr. Wisner. We're going to go for about an hour and then we're going to take lunch.

MR. WISNER: Yes, Your Honor.

THE COURT: And you may resume, understanding that you remain under oath, Dr. Portier.

THE WITNESS: Yes, Your Honor.

BY MR. WISNER:

Q. All right. Dr. Portier, did you have a good

break?

A. Yes.

Q. I put you to work; right?

A. Correct.

Q. Okay.

MR. WISNER: Your Honor, can you see?

THE COURT: I can.

Okay. I don't know if defendant's counsel can see, or perhaps you need to move so they can also see.

MR. BROWN: Excuse me. What would be a good spot, Your Honor?

MR. ISMAIL: Let's see -- I can tuck in over there in the corner if that's okay with the Court.

MR. WISNER: No problem.

THE COURT: So whoever needs to stand over there to see.

And just to say, jurors, if you have questions, I notice you have a couple, if you would just sort of let the Court Attendant know that you have prepared a question and she'll come and quietly get it from you. And use your notebooks. So there may be points at which Madam Court Attendant may walk over and grab something quietly.

I think we're good.

MR. WISNER: I'm in the way now.

Q. All right. Doctor, during the break we had you fill out -- this is the rat chart; is that right, Exhibit 103?

A. Correct.

Q. Okay. And can you just briefly --

MR. WISNER: Well, actually, Your Honor, permission for him to come down and just walk us through what he did?

THE COURT: Sure. You know what, I think I have a pointer because I think it's going to be very difficult for him to do that without standing in the line of sight of the jurors. So let's see if we have a pointer.

Anybody have a pointer?

TECH PERSONNEL: I do.

THE COURT: I know we have one. I'll see if I can have Onesha get it. I think that makes sense.

MR. WISNER: Sure.

THE COURT: And if you need to stand up over here to do that, Dr. Portier, you don't have to be seated.

THE WITNESS: I prefer to stand.

THE COURT: You're free to do that.

BY MR. WISNER:

Q. And maybe I can kind of lead you through it a

little bit --

A. Certainly.

Q. -- so we can do this cleaner.

So let's start up at the top. We have all these studies. Why, sir, are these three in gray and those ones in white?

A. There we go. I'm going to blind the audience. I'm very sorry.

These are studies that were done in rats, not mice. Strictly the rat studies. There are seven of them.

Four of those studies, the first four right here, were done in what are called Sprague-Dawley rats, it's a specific strain of rats. It's quite commonly used in cancer studies. And these three were in Wistar rats, a different strain of rat. And I can tell you about them if you ever want to know more about the two different strains, but they're just two strains of rats.

Q. Well, we talked earlier about different strains, sexes, species. When you're talking about strains, are you referring to the different strains here?

A. Correct.

Q. Okay.

A. The dark-colored names are the Wistar rats,

and the light-colored ones are the Sprague-Dawley.

Q. Now, I notice that the years are in ascending order. So Lankas is 1981. And Enemoto is 1997. And then Suresh is 1996 to 2009. What does that reflect?

A. That's the year in which the results were reported to some regulatory agency somewhere. It's difficult for me to collect this information. Some of it is proprietary and so it's not in the public domain for me to be able to evaluate it. Some of it was done by Monsanto. They have given me all of that information. But some of this was done by other companies that wish to sell glyphosate and get it approved in other countries. And so I don't have all of the information.

But to the best of my knowledge, that's the year in which it was reported to some regulatory authority.

Q. Now, you've listed seven on this chart. Earlier you mentioned that there were over 20. Why have you listed these seven of the rat studies?

A. These are the ones that are most clearly done. They're done under guidelines that are accepted worldwide. I had the most information on these.

There were several other studies that were either too small to include in here or they used doses

that were so small, you expected to see nothing and that's what you saw. And so I just didn't bother to put that one up there. There was just one like that. Or they had other flaws.

Q. And in your review of the EPA documents and these other regulators that you've reviewed, is there a general consensus that these are the seven rat studies at issue?

A. Yes, pretty much. With the exception of some minor differences between different regulatory authorities, they all looked at these seven studies.

Q. Okay. All right. So let's sort of walk through what you've done here. So let's start off with the Lankas study from 1981. You've circled "trend" and have three pluses. What does that mean?

A. I forget to put the little thing at the bottom.

So that's one type of tumor. It's an interstitial cell tumor in the testicle in the rats. And they saw a dose-related increase in that. So that's why it says "trend." The statistical trend test was positive for that assay, meaning that it's not likely to have arisen by chance.

The three pluses means that the probability of it arising by chance is above -- is below one in 100,

below 1 percent. In this case I think it was four out of a thousand chance that this arose by chance. So that's what the three pluses mean.

"Dose" means --

Q. And, Doctor, can I have you just quickly come down and just write that key on the board? Because I know you normally do that, and I want to --

A. I'll just write all three of them down.

Plus plus plus promulgated less than 0.01.

Plus plus 0.01 less than promulgated equals 0.05.

And plus is 0.05 less than P, less than equal to 0.1.

So this is one in 100. This is between one and 100. And five in 100 and one in 20. And this is between one in 20 and one in 10.

That's the chances of it being random. So that gives you some idea of the statistics.

Q. Okay. Great.

So just tell me if this is correct way of characterizing this. This is to the 99th percentile competence level?

A. There's no confidence limits.

Q. Sorry. Let me say it again. 99 percent p-value.

A. Yeah, this is -- it's .01.

Q. Okay.

A. It's below .01.

Q. So that's 99 percent. That's 95 percent. And that's 90?

A. I don't know what you're saying 99 percent of what?

Q. Okay. Fair enough.

I just was trying to get the numbering down.

A. There's a one -- there's a less than 1 percent chance that the testes interstitial cell tumors arose at random --

Q. Got you.

A. -- that's what this means.

Q. Let me ask it another way. Is another way of saying this highly significant?

A. Yes.

Q. Significant?

A. Yes.

Q. Marginally significant?

A. Yes.

Q. Okay, great.

All right. So we have a finding here and we have the dose circled. What does that mean?

A. That means at least one of the exposure groups

by itself was significantly different from the controls, meaning the probability that that occurring by random chance is less than one in 20. That's what that means.

Q. Okay. And then you circled "M." What does that mean?

A. This is in male rats. It's testicular tumors, it's clearly in male rats.

Q. All right. And so if we use that metric, the pluses and circles, does that apply to all the other tumors that you've marked up here?

A. That is correct.

Q. All right. And I want to ask you a few just general questions.

The first one is: I see that there's repetition so thyroid C-cell, thyroid C-cell. Do you see that?

A. Yes.

Q. What, if any, significance is there to that?

A. You're seeing the same tumor in multiple studies. And if you remember the pillars of the bridge we were talking about with the EPA guidelines, this is one of the things that makes you more concerned about this being a carcinogen.

Q. And we have down here "pancreatic islet cell tumors"; do you see that?

A. Pancreatic islet cell tumors.

Q. Islet cell tumors, thank you.

And we see, again, it's happening in the dose at two pluses and males. What is the significance of that repetition?

A. Again, it's the same thing. You're seeing it twice in two of the four studies in Sprague-Dawley rats. It raises concern.

Q. And one that we see in four of these -- we see skin keratoacanthoma; do you see that?

A. Skin keratoacanthoma.

Q. Okay. We those in Atkinson, Enemoto, and then over here in Wood; right?

A. Correct.

Q. And what is the significance of seeing the same tumor in men in a trend in different strains?

A. Again, that strengthens the finding that this is probably something of great concern because it's being repeated. It strengthens the finding that it's a real finding. Even though each case has very little probability of being due to random chance, the fact that you've got it in four of the seven studies is even smaller random chance that that just occurs by chance. So it's a real finding. The skin keratoacanthoma is a real finding in these studies.

Q. Now, we're talking about lymphoma here.

A. Yes.

Q. What's the relevance of finding all these other types of tumors insofar as understanding the carcinogenicity of glyphosate?

A. There's no guarantee that you'll see the same tumor in rodents, rats or mice, that you would see in humans. That's no guarantee.

Different species, I can give a rat benzene, which is a known human carcinogen, causes leukemia in humans. I can give a rat benzene and it won't get leukemia. But if I give it to a mouse, it gets leukemia. So there are differences between the species and how they respond. But as a general rule in public health, if you see tumors in any of the animal species at any site, it raises concern for humans.

Q. Now you keep saying tumors. Is tumors cancer?

A. Tumors are cancer. Yes. I'm sorry. These are -- there's malignant cancers and benign cancers. These are benign cancers usually, which means they don't really invade the tissue and kill the animal.

Q. But "these are," what are you referring to?

A. The skin keratoacanthoma.

Q. Just these ones?

A. Just those.

Q. Not all of them are benign?

A. No. The rest are all malignant tumors.

Q. Okay. All right. And I didn't mean to interrupt. You were saying something. I just wanted to clarify.

A. Well, they can become malignant. These do in some cases become malignant tumors. But it's fairly rare. And I didn't see any indication that any of these were malignant cancers.

Q. By "these"?

A. The skin keratoacanthomas.

Q. Okay. But we're looking at all of them.

A. Correct, sorry.

Q. That's malignant?

A. That's malignant, absolutely.

Q. That's malignant?

A. Yes.

Q. Okay. So everything else is malignant expect for the pink ones?

A. Correct.

Q. Okay. And so I guess my question is: Have you ever heard of something called oncogenicity?

A. Yes.

Q. What is oncogenicity?

A. That's the ability of something to cause

cancer. If chemical X is oncogenic, that means it causes cancer.

Q. And what does this data tell you about the oncogenicity of glyphosate?

A. That it is oncogenic in rats. It can cause cancer in rats.

Q. Okay. And one of the things I want to clarify is so some of these have the word "tumors" on them. All right. So the pancreatic islet...?

A. Cell. Islet cell.

Q. Islet cell tumor. That says tumor; do you see that?

A. Correct.

Q. And then some of them have carcinomas; do you see that?

A. Correct.

Q. What's the difference between a tumor and a carcinoma?

A. This is nomenclature, this is the way they name things generally. Basal cell tumors refers to multiple types of carcinomas in the basal cells.

Kidney carcinoma adenoma is the exact correct term. Basal cell tumors is just a label for a grouping that is normally used for basal cells.

Q. What about like lymphoma, does that manifest

as tumors or how does that relate to the tumor nomenclature?

A. Lymphomas are malignant tumors.

Q. All right. Well, let's go to the mice studies.

This is Exhibit 101. We have here a very similar looking chart. We have one -- six studies. And again there's one that's in gray. Do you see that?

A. That's correct.

Q. What does that reflect?

A. That reflects the fact that the Kumar study is in a different mouse strain. That's in Swiss Webster mice, and the rest are in CD-1 mice.

Q. Okay. And just I noticed here and I think there was one reference to it on the other chart as well, but it says right here "limited for Atkinson"?

A. Correct.

Q. What does that mean?

A. They didn't do -- so remember I told you in every animal they take the organs out and look at it, take slides and look at it with the pathology under the microscope.

In Atkinson, they didn't do that. They tried to save money. And to be fair, there were a number of groups including the National Toxicology Program which

were trying to do the same thing. They would only look at the controls and look at the high-dose group. And they'd look at all the tissues, and if they saw something different, then they'd go and look at all the tissues like that in the other dose groups.

So you didn't have to do the pathology on every single tumor, every single tissue, and every single animal.

Eventually everybody abandoned that because it didn't work well. We were missing a lot of tumors. But that was limited in the sense that they didn't look at all the animals.

Q. And notwithstanding the fact that they didn't look at all the animals, the fact that you have findings, what is the significance of that?

A. It doesn't really make a big difference if you have findings. Having not seen findings, it would make a difference because I'd be worried that the limited pathology left us missing something important. But since we found things, it probably had no impact.

Q. Let's actually compare that to one of the previous studies, the rats study. See Suresh here --

A. Correct.

Q. -- 1996, that's limited.

A. Correct.

Q. And it doesn't have any findings.

A. That's correct.

Q. What, if anything, do we make of that?

A. We apply less weight to the fact that Suresh has no findings because of the fact that they didn't look at all the animals.

Q. Okay. All right. So going back to the mice, we have the same symbols here. Are these -- does the same chart apply to here?

A. Yes, the same symbols apply to this chart.

Q. Okay. And I want to look at -- the first one I want to look at is these kidney carcinomas or adenomas; do you see that?

A. Correct.

Q. We have that Knezevich and Hogan from 1983; correct? What other studies do we see that sort of tumor popping up?

A. The Sugimoto study here, 1997, the Takahashi study in 1999, and the Kumar study in 2001 all showed kidney carcinomas or adenomas increased in probability of showing up.

Q. Now, I want to explore that just quickly. I mean, how rare of a tumor is a kidney carcinoma or adenoma in mice?

A. We have to break the mice up a little bit here

to look at that question. So this separate strain right here, Kumar, the Swiss Webster mouse, I don't know how rare it is in that particular mouse. I really didn't look it up. So I can't answer the question.

These are CD-1 mice, all of them, but there's a difference here. These two studies were done for 24 months. Maybe we should put a "24" down here at the bottom. And these three studies were done for 18 months.

Now that's a difference. You're looking here in 18-month studies at something equivalent to 50-, 55-year-old person, and in the 24 months you're looking at something similar to a 65-, 70-year-old person.

So if you think about your understanding of cancer, the fact that people get kidney tumors, it increases as people get older, the chances of getting it.

So when I compare whether this is a rare tumor or not, it's going to depend. At 24 months, it might not be so rare of a tumor. But at 18 months, it may be very rare.

So my comments will then go to both of them.

So at 24 months, this is a fairly rare tumor. It occurs at a rate of about three in every thousand, I think, animals or four in every thousand animals. I'd

have to check my notes to be precisely accurate on that.

At 18 months in these two studies --

Q. Can we back up there. You said three or four out of every thousand animals. Well, how many tumors would you expect to see in 100 in one of the dose groups?

A. You wouldn't expect to see very many, maybe two at most in any -- if you add them all up in all the groups, you'd expect to see maybe two.

Q. And, for example, in the Knezevich and Hogan study, how many of those tumors did they see in just 50 male mice?

A. I'd have to check my notes to be exactly accurate, but I believe it's five.

Q. And how many in the high-dose group, just that one group?

A. I believe that was two adenomas and one carcinoma, or two carcinomas and one adenoma. But it was three.

Q. Three tumors?

A. Yes.

Q. So what is the probability from a statistical perspective of seeing three tumors, these rare tumors in mice who were exposed to glyphosate like that?

A. So that was calculated here. And we used -- I

used historical control information to be able to do that calculation. And that's less than one in a hundred. Less than 1 percent chance that what you see occurred by chance randomly.

Q. And we see this replicated in Sugimoto and Takahashi?

A. Correct.

Q. And these are shorter studies?

A. And tumors are extremely rare there.

Q. So the likelihood of seeing this tumor in the glyphosate-exposed mice is even smaller?

A. Correct, it's even smaller.

Q. And then you see obviously a result in Kumar.

A. Which is also an 18-month study.

Q. Okay. What does it tell you seeing all these rare tumors consistently appearing in the rodents -- or the mice exposed to glyphosate?

A. Glyphosate causes kidney adenomas and carcinomas in CD-1 mice.

Q. All right. Now I want to talk about the one that's probably most important one. Let's talk about lymphoma.

So in Knezevich and Hogan, we have this is spleen composite lymphosarcoma. What is that?

A. So that's the oldest study on the board.

That's 1983. And the next study is 1993.

Now, like anything else, pathology changes over time. They change the way they think about diseases. It's not static.

Back in 1983, they had this thing called composite lymphosarcoma. This is in the spleen. And that became my understanding in looking at the history of this is the composite lymphosarcoma became what's called a malignant lymphoma.

So this is the same tumor seen in the spleen but with a different nomenclature because it changed over time.

Q. And these studies go back to 1983, so what is that, 35 years?

A. 36.

Q. 36 years. Biostatistician. Thank you.

And we go here from '83, and we go through each one of these studies and there's a result for lymphoma.

A. That's correct.

Q. What does that tell you about glyphosate causing lymphoma?

A. That it's very consistent finding. It's clearly causing malignant lymphoma in CD-1 mice. I only have the one Swiss Webster mouse study, but it looks

like it just causes lymphoma in mice as a general rule.

Q. And you've looked at a lot of different substances and the rodent data associated with them. Have you ever seen a substance have such consistent results for lymphoma?

A. For lymphomas?

Q. Yeah.

A. I -- so, first, there are very few substances that have had this much study to it.

Q. Yeah.

A. And so with that caveat, the answer is no. But most of the studies I've looked at are one mouse study and one rat study for chemicals. Very seldom do they do this many.

Q. And so just to be clear, these mice that were studied in Wood are -- I mean, how many years apart are those?

A. Years apart from what?

Q. From Knezevich and Hogan, how many years have gone by?

A. 26, I think.

Q. All right. 26 years. And during that time, has the way we look at -- let me strike that because we kind of covered that already.

But -- well, I'll just move on.

Now, Doctor, I want to talk to you about what this -- well, before we move on, I see some other ones here. Right? We have hemangiosarcomas, hemangiomas; do you see that?

A. Yes, I do.

Q. And then we have this category of multiple malignant tumor/neoplasms; do you see that?

A. Yes.

Q. What does that refer to, those yellow ones?

A. That's a category that's sometimes used in these studies. They look to see how many animals had a malignant tumor regardless of the common. And then they look at the probability that that arises by chance. And so you can do a statistical analysis of that. And that's what these two things are. And they're both significant in those two studies.

Q. Okay. I want to go back to -- you can take your seat, sir.

I want to go back to the EPA guidelines that we were talking about a minute ago.

MR. WISNER: I believe that's Exhibit 940, Your Honor, permission to publish?

THE COURT: Yes. You don't have to ask. I've already given you permission.

MR. WISNER: Thank you, Your Honor.

(Exhibit published.)

BY MR. WISNER:

Q. So we were looking at this part earlier, and we were specifically looking at these characteristics that we use to assess carcinogenicity data.

Do you recall that?

A. Yes, I do.

Q. And now that we have sort of the data summarized in these tables, I want to go through some of these characteristics. All right?

A. Okay.

Q. So uncommon tumor types. Do we see that in this data?

A. Yes, we do.

Q. Where?

A. Kidney carcinomas and adenomas in the mice, hemangiosarcomas in the mice, and there's one more and it's eluding me at the moment. But, yes.

Q. We see it.

Now, I notice in the mice we had all these kidney carcinomas and adenomas. And I see one right here in the rats. What's the significance of that?

A. That's tumors across two species that match.

Q. Okay. So for this one, we see it, the uncommon tumor types?

A. Correct.

Q. What about tumors at multiple sites, do we have that?

A. Yes. Quite a bit. You can see every one of these studies that has multiple boxes under it has tumors at different sites in the same study.

Q. And does that strengthen the evidence about whether or not glyphosate causes cancer?

A. Yes.

Q. Tumors by more than one route of administration, do we have that here?

A. No.

Q. Okay. Why not?

A. Because all of these are feeding studies. Technically they're getting -- they're not just eating it, but they get it on their skin and other things, but it's one route of administration, it's through feeding.

Q. Now I understand that there has been at least one other study where they did it on the skin; is that right?

A. That's correct.

Q. We're going to talk about that study later. Would that be a different route of administration?

A. Yes, it would.

Q. All right. So tumors -- the next one is

tumors in multiple species, strains, or both sexes. Do you see that?

A. Correct. Check on all three of those.

Q. Yeah. Okay. So species, where do we have that?

A. Well, you can see, there are rats, tumors. There's mice, tumors, clearly you have two species with tumors in the multiple species. You even have the same tumors in multiple species.

Q. All right. What about strains?

A. Yes. Again, you have multiple strains here. Malignant lymphomas across them, skin keratoacanthomas in the multiple studies, et cetera.

Q. Do we have them in both sexes?

A. Yes, you do. The thyroid C-cell adenomas and carcinomas. Wherever those are in the mouse studies, I think they're in the mouse studies, those are in males and females. And there was one more that was in males and females. Where is that? Nope, I don't see it. It must be in the rats.

Q. Well, what about lymphomas? We have it in the females in Knezevich and Hogan.

A. That's true.

Q. And the males afterwards.

A. In the males afterwards, that's correct.

Q. Is there anyone with those double circles --

A. Actually, that's an error, by the way. I'm sorry, I have to check my own work here. The Takahashi study, that's in females, not males.

Q. Oh.

A. I'm pretty sure that was females. I'll check my notes, but I'm pretty sure that was females.

Q. All right. I'm going to circle the females. Okay?

A. Okay.

Q. So I guess in lymphoma then, we do have it in both sexes?

A. Correct.

Q. All right. Any other double circles? I think you might have mentioned it already, but there was one where there was both. Is that pituitary?

A. Yeah, that's it. Pituitary adenomas and carcinomas.

Q. Okay, great.

A. And thyroid follicular cell -- thyroid C-cell carcinomas and adenomas.

Q. Right here?

A. Yeah.

Q. All right. So progression of lesions from preneoplastic to benign to malignant, do we see that

here?

A. You have some of that for some of the studies for some of the tumor sites. The hepatocellular adenomas and carcinomas in the Brammer study showed progression.

Q. This one right here?

A. Yep, that one right there.

And what was the other one? Oh, I have it in front of me here. I can look at it for myself.

That may be -- oh, the mammary gland carcinomas or adenomas in the Wood study I believe also. But, again, I'd have to check to be absolutely certain.

Q. But you did see this progression in the data?

A. Correct. For some of them. You didn't see it for others.

Q. Okay. So I'm going to do a small check mark.

A. That works.

Q. All right. Reduced latency of neoplastic lesions, do we see that in the data?

A. Technically you do because you've got the 18-month studies and the 24-month studies in the mice and you're seeing the same tumors in the 18- to 24-months, and you're seeing them at 18 months which means they're appearing very early.

Q. And I just want to kind of parse that a little

bit. So these two were 24-month studies?

A. Correct.

Q. And these ones were all 18?

A. Correct.

Q. And what is the significance, if any, of seeing lymphoma in a 24-month study and in an 18-month study?

A. It's occurring fairly early in these animals as a function of exposure to glyphosate.

Q. All right. What about metastases?

A. Metastases. When a cancer grows in your body, sometimes some of those cells detach, they get picked up by the blood and they carry through the body and end up somewhere else and grow as a cancer there. Those are called metastases.

We generally don't use those in looking at cancer risk assessment. We're interested in causing that first cancer. We know cancers metastasize, but we generally don't bother to count and look at the metastases very closely. So I haven't looked at it here. But clearly there were some.

Q. Okay. What about we don't have an unusual magnitude of tumor response; right?

A. No, I wouldn't characterize any of these as an unusual magnitude of tumor response.

Q. To be clear, do you believe there is a tumor response, it's just not an unusual, sort of off-the-wall response?

A. I do believe there is a tumor response. And one could argue that the two cases where you were looking at the -- what were they called? Multiple malignant tumors or neoplasms --

Q. These ones?

A. -- that is looking at the question of did you have a lot of malignancy in these animals because you're looking at how many of the animals got anything at all. So there's a little bit of evidence for that in those two findings. But the individual tumors are not unusual magnitudes.

Q. What about the proportion of malignant tumors?

A. No.

Q. Not an issue here?

A. No, I don't see that as being a huge issue here.

Q. All right. What about dose response?

A. Clearly, you've got dose-related response here.

Q. All right. And let me just make sure I fully understand these factors.

Do you have to have these factors to confirm

your results?

A. Oh, no, no. These are just things that strengthen your overall findings. You could have none of these occurring and still call it a positive finding in the animal studies.

Q. What about the fact that we have one, two, three, four, five, six, seven, eight of them occurring?

A. It just simply strengthens the overall conclusion that glyphosate can cause cancer in rats and mice.

Q. And the last paragraph here, it states:

> In these cancer guidelines, tumors observed in animals are generally assumed to indicate that an agent may produce tumors in humans.

What does that mean?

A. Exactly what it says. From the perspective of both the scientific community and just good public health, if you see something causing cancer in mammals in a very carefully controlled experiment, you should be a little bit worried about it and you should investigate it more carefully. So it should be assumed that it could produce tumors in humans.

Q. And in your scientific opinion, seeing all these tumors and all these characteristics we just

discussed in all these different studies, do you have any doubt about whether or not glyphosate induces tumors in animals?

A. I don't have any doubt whatsoever that glyphosate induces tumors in animals.

Q. Now, have you heard of something called concordance?

A. Yes, I have.

Q. What is that?

A. Well, concordance would mean -- it's the question you asked me earlier: What is the significance of kidney tumors in the rat to NHL in humans?

So concordance would address that question. It may be that every time people have found something in humans that causes NHL, they've seen kidney tumors in rats. As odd as that may seem, that may be what the evidence says.

So that's what the question of concordance is addressing. Which tumors do you see in animals also appear in humans indicate what's going to happen in humans, let's put it that way.

Q. And just generally speaking, when it comes to seeing lymphoma in rodents, historically have we also seen lymphoma in humans?

A. Yes, we have. I just published a paper on

that. And I had looked at that, that paper was written four years ago, but it took us a long time to come to agreement on the paper.

But, yes. And I looked at lymphomas specifically to explain to my colleagues a problem we were having with the paper in 2014. So I had that information with me and I looked at it. And, yes, there is concordance between seeing lymphomas in mice and seeing lymphatic cancer in humans.

Q. All right. So far we've been talking about the animal studies as it relates to glyphosate; right?

A. Correct.

Q. In a second I want to talk -- well, I want to transition to Roundup or formulated Roundup.

What is your understanding of the difference between those two?

A. Oh, well, glyphosate is a pure chemical. It's a single molecule. It has a particular structure in form.

Roundup has multiple molecules of different types in it. There's things in there to help it cling to the cell. There's things -- to the plant. There's things in there to help it get inside the cells of the plant. There are other things in there that I don't necessarily know what they're there for. But it's a

mixture of chemicals and the glyphosate. So it's a formulation.

Q. And having reviewed the literature on glyphosate and Roundup, have there been any valid studies done on formulated Roundup to see if Roundup, as opposed to just glyphosate, can cause tumors?

A. Do you mean valid, long-term animal cancer studies?

Q. That's correct.

A. No.

Q. In your opinion, would conducting a long-term animal cancer study on Roundup be helpful for understanding its cancer risk?

A. On one of the specific formulations, yes, it would.

Q. Why would that be helpful?

A. It could -- well, let me rethink that question. Would it be helpful to me at this point in terms of looking at what I'm looking at? It could be. It could be.

If it was done in the mouse and it -- I could look and see how much production of malignant lymphoma would occur in the mouse, and I could compare that to what I saw for the pure glyphosate and get a feel for whether there's a change in that risk once you use the

other chemicals at the same time. Because they might affect the overall risk of getting the cancer.

Q. Now, you have looked at sort of mechanistic studies that look at both glyphosate formulations; right?

A. Yes, I have.

Q. And generally speaking, do you have an opinion about whether, based on those cell studies, glyphosate or Roundup is more toxic?

A. For cancer?

Q. Exactly.

A. For cancer. There's an indication there that it might be more -- of more likely to cause cancer than Roundup alone. There is an indication in that literature.

Q. You mean glyphosate more or Roundup more than glyphosate?

A. Roundup more than glyphosate.

Q. Okay. And just from a feasibility perspective, do you think it would be possible to conduct one of these long-term studies on Roundup?

A. Yes.

Q. How is that possible?

A. It's just yet another chemical to administer to the animals. You might have a problem getting the

same amount of glyphosate in the animal that you got in these studies because the pure chemical, I can put it on food, but once I get to the formulated chemical, there's all these other chemicals in there and it can overwhelm the food. So you don't want to put too much in the food that it tastes so bad they'll never eat it or it gives them stomachaches and things like that. So you may technically have some problems getting the glyphosate dose up high enough. But you could certainly do a study. There's no doubt there.

Q. And in -- what are some of the ways you could do that, just practically speaking?

A. Well, you could certainly do it in feed. There's no doubt about it. You may have some problems. You know, sometimes chemicals aren't palatable, they don't taste well. And I don't know how bad Roundup would taste if you were eating it and you were a mouse or a rat.

But you can overcome that. You can gavage the animals, which means you would take the Roundup, mix it with corn oil, and then there's a tube that you put it directly into their stomachs. We've done those studies before. They're fine. They're hard to do, but you can do it. There's ways to do that.

Q. What about inhalation?

A. You could. It would be a little bit technically more difficult because you've got to cause the glyphosate to -- the Roundup to volatilize into the air so the animal could breathe it in. But you could technically do it. Or you could paint it on skin.

Q. Let's talk about that. Has there been a study where they tried painting Roundup as opposed to just glyphosate on rodent skin?

A. I don't think there's been a Roundup study on rodent skin.

Q. The George study?

A. I thought that was glyphosate, pure.

Q. Let's take a look at it.

A. Okay. Good idea.

Q. Why don't you take a look in your binder. It's Exhibit 1768.

(Witness reviewing document.)

THE WITNESS: And I stand corrected.

BY MR. WISNER:

Q. Okay. Do you have the George study in front of you?

A. Yes, I do.

Q. Is that a fair and accurate copy of that study?

A. Yes, it is.

Q. Something that you relied upon?

A. Yes, it is.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection.

THE COURT: I'm sorry. No objection?

MR. ISMAIL: Correct. Sorry.

THE COURT: Yes, you may.

(Exhibit published.)

BY MR. WISNER:

Q. All right. So we're looking here at this study called the George study. The title of it is "Studies on glyphosate-induced carcinogenicity in mouse skin: A proteomic approach." Do you see that?

A. Yes.

Q. And it's authored by Dr. George and her colleagues?

A. Correct.

Q. Okay. Good.

Now, if you look at the second page under the "Materials and Methods" section. Do you see that, sir?

A. Yes.

Q. And it says:

> The commercial formulation of the herbicide Roundup original glyphosate

41 percent POEA 15 percent.

Monsanto Company, St. Louis, Missouri, was used.

Do you see that?

A. Yes.

Q. So this actually was a study that used Roundup?

A. Correct.

Q. And it says right here "POEA." Do you know what that is?

A. No. It's a chemical.

Q. Do you know what it's used for?

A. No.

Q. Okay. All right. And if we look here in the study -- well, please describe to the jury what this study did.

A. Okay. So it's like doing a cancer study before. You got groups of mice. In this case, it's only 20 mice in each group. I believe these are SENCAR mice. No. These are Swiss albino mice. Okay. And they randomize them to the groups just like you do in a cancer study.

Now instead of feeding the glyphosate to them, they paint the glyphosate on the skin of the animal.

Q. Doctor, we're talking about Roundup.

A. Roundup. I'm sorry. I'm sorry. Roundup. It is Roundup. You are absolutely correct. So they paint the Roundup on the skin of the animal.

Now this is an interesting study in the sense that this is called -- this is a two-chemical study. So what they do is -- well, a little bit on the mechanism of cancer first, if that's okay.

Q. Sure. Would you like to use your diagram?

A. That would be useful.

Q. Okay. I believe that's in your binder. It's exhibit -- it's Exhibit 108.

MR. WISNER: May I publish, Your Honor?

MR. ISMAIL: No objection, Your Honor.

THE COURT: Permitted.

MR. WISNER: I'm going to pop it up on the screen.

(Exhibit published.)

BY MR. WISNER:

Q. All right.

A. So this is a little cartoon I use to try to explain how cancer is formed from exposure to chemicals.

So you have a bunch of normal cells being happy, doing their normal thing all through your life. Your cells are constantly getting DNA damage. That's a normal reaction to living in a modern world. Your cells

are getting DNA damage all the time. And cells have machinery that cleans up that DNA damage, takes it out of the way, gets rid of it, and the cell goes on being happy.

Sometimes the DNA damage isn't repaired, and the cell replicates. And when the cell replicates, if you remember your high school biology, the DNA duplicates and then the cell splits apart and you get two new cells with the same DNA in it.

When the DNA duplicates, if that damage is not repaired, it's fixed in that DNA. So the cell no longer knows it needs to repair that site. That is a mutation. That's what happens when a mutation occurs.

If you get enough mutations in the cell of specific types, it loses growth control and it becomes a cancer. That's what a cancer is.

So what you see here is normal cells. They can become damaged. They can be repaired.

Let me see if I can do this here. Oh, sorry, audience. They can't see it. Oh, yes, they can.

Q. That was me.

A. Okay.

It can be repaired. If it's not repaired and the cells replicate, duplicate, it gets fixed as a mutated cell. If you get a lot of mutations, you have a

cancer.

Chemicals can come in and attack this whole process at different points. The two most interesting points for cancer that people spend a lot of time looking at are producing DNA damage and are changing cellular replication or changing repair rates in some sense.

So if a chemical causes mutation, they call that initiation. It starts the events of cancer in the body. If instead of doing that, the chemical comes in and changes DNA repair or cellular turnover in some way that this DNA damage that's routinely happening in your cells doesn't get repaired, then that's promoting a cancer to occur in your body. That's called a promoter.

So the study we're looking at is what's called an initiation promotion study. We can go back to the study now.

Q. All right.

A. So what you do in these studies is you take a chemical that you know attacks DNA and you put it on the skin of the animal. And then you take the chemical you're not sure about how it works, and after you put this initiator on and waited a little while, you start putting that on the skin of the animal and see if you get skin tumors. And if you do, then that new chemical

is promoting the DNA damage that was caused by the initiator.

Then you flip the tumor. So now you have your chemical and you put it on the animal and you wait a little while. And then you get a chemical that you know promotes carcinogenesis, something you know works that way, and you put it on the skin and look to see if you get skin tumors. And if you do, then your chemical now is known as an initiator of cancer.

So that study we're looking at here is a complicated design because it's got other chemicals involved. So it's got a group with no chemicals. It's got a group with just the known initiator. It's got a group with -- well, here it's got a group with no chemical, a group with only glyphosate, no initiation, no promotion, they just put the Roundup on the back of the animal. A group with Roundup. And then TPA which is a promoter. A group with Roundup and TPA, but I believe it's --

Q. Thrice a week as opposed to single.

A. Yeah, it's more Roundup.

Then you've got a group with just DMBA. And a group with just TPA, the initiator and the promoter. And then a group with DMBA followed by glyphosate, which is the initiator followed by the promoter -- by the

potential promoter.

Q. Let me talk about that group 8 right there.

A. And its Roundup, not glyphosate. Sorry. They used the word "glyphosate" in the paper quite a bit.

Q. All right. So in group 8, just to be clear, we have these mice, they're given a known cancer initiator; right?

A. Yes.

Q. And then they're followed for -- they're given Roundup painted on their skin how many times a week?

A. Three times per week for 30 weeks or so.

Q. And then at the end of that, what do they look for?

A. They look for tumors on the backs of the animal. Specifically in this case, they're looking for persistent little polyps on the back. These are called skin papillomas, and that's specifically what they're looking for here.

Q. So we look at the Table 1 from the study. We have that group 8 here at the bottom. Do you see that?

A. Yes, I do.

Q. And we have the percentage of animals with tumors; do you see that column?

A. Yes, I do.

Q. And in group 3, 100 percent of the animals had

tumors; do you see that?

A. Correct.

Q. Is that an expected result?

A. Yes, that is, because you're using a known initiator and a known promoter, you expect to see that result. And it's put in here as what's called a positive control.

So this tells me if this didn't occur, then this experiment was flawed, that there's a problem with it. But because it occurred with that, that says they did the experiment correctly. That's all it is.

Q. And then we have here the only other results with tumors appears to be group 8; do you see that?

A. Correct.

Q. And it says 40 percent of the animals who had the initiator and then Roundup applied for this period of the study had tumors.

A. That is correct.

Q. What is the significance of that? What does that tell you?

A. That Roundup, in this formulation, promoted the tumors that were initiated by DMBA.

Q. Now if we go to the beginning of the paper and we go to the abstract right here, it says carcinogenicity study revealed that glyphosate has

promoter -- has promoting activity; do you see that?

A. Yes.

Q. Is that what you were saying?

A. Correct. But it's -- they can't conclude it's glyphosate here. They can only conclude it's the Roundup form that was used.

Q. That's right. And I guess this was done back in 2010; right?

A. Yes.

Q. To the best of your knowledge, has -- other than these independent scientists, has anyone tried to do another Roundup study to see if it's a promoter or initiator?

A. Not that I'm aware of.

Q. Now one thing that I just want to make sure I understand here. It says "a proteomic approach." Do you see that?

A. Yes.

Q. What does that mean?

A. Now you're getting much more complicated.

They also took skin from the backs of the animals, and through the magic of modern science -- I'm not going to explain proteomics, it will take forever -- they were able to look at proteins, the major chemical groups in the cells of the skin, and they looked for

changes in those proteins as they exposed the animals to the Roundup. And they're comparing the level of those proteins in the control animals to the level of the proteins in the treated animals.

And then what you do is look to see which proteins have been changed. And based on that and historical knowledge of what proteins are used for what and what things change protein levels, you can start grouping it into groups of mechanism that tell you what you think happened here in terms of this particular cancer finding.

Q. So in the study, there's a Table 3, and it says: Fold changes of differentially expressed proteins get altered by glyphosate, TPA, DMBA with respect to untreated group.

Do you see that?

A. Yes.

Q. And it has these changes. As simply as you can, explain what these numbers are saying with regards to glyphosate or I guess Roundup, TPA and DMBA?

A. So remember in this case the glyphosate was looking like a promoter. So we really want to compare it to the TPA column. We would expect to see it looking like TPA for the proteins.

And you see that across the board it pretty

much looks like TPA. So what it's telling you here is that it seems to have the same mechanism of action as TPA in its ability to cause promotion.

Q. And I guess my question is: Is this something that you relied upon in assessing the sort of mechanisms through which Roundup could cause cancer, is you're saying it doesn't behave like something we know that causes cancer?

A. Yes.

THE COURT: So this might be a good time to take our lunch break.

So ladies and gentlemen, we're going to take a one-hour lunch break. I would ask the members of the gallery to please wait in the courtroom for five minutes before you leave.

If you leave the building, please come back in enough time to get settled so that we can get started.

THE WITNESS: Your Honor, would you like me to be seated when we come back in court?

THE COURT: No. We'll worry about that when they've left.

THE WITNESS: Okay.

(Jury excused for lunch recess.)

(Proceedings continued in open court out of the presence of the jury:)

THE COURT: So if you would just wait a few minutes before going out, I'd appreciate that.

And you can step down. And your counsel will tell you when to come up and be seated before I come out.

MR. WISNER: Do you want him before you're coming out or after coming out?

THE COURT: No. No. Before. He can be seated. When we all say we're ready, he can sit down.

(Luncheon recess was taken at 12:35 p.m.)

AFTERNOON SESSION 1:34 p.m.

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

THE COURT: Mr. Wisner, you may continue.

MR. WISNER: Thank you, Your Honor.

BY MR. WISNER:

Q. All right. Good afternoon, Doctor.

A. Good afternoon.

Q. Did you have a good lunch?

A. Yes, I did.

Q. Good.

We were, just before the break, we were talking specifically about the rodent studies.

MR. WISNER: And if we could turn the document on.

BY MR. WISNER:

Q. So far we've been talking about this first pillar, the animal studies. And we are going to talk about epidemiology in a second, but I want to turn the to the first one, mechanism studies.

Okay?

A. Okay.

Q. What do you mean by "mechanism studies"?

A. They are studies that tell you something about the potential ways in which glyphosate is causing cancer.

Q. And are there established mechanisms of action through which a chemical can cause cancer?

A. Yes.

Q. How many established methods are there?

A. Well, it's going to depend on who you talk to. But talking to me, I would say ten.

Q. Okay.

A. We just published a paper on it.

Q. To understand what these characteristics are, for these ten that you're talking about, does the substance have to be all ten of them to cause cancer, or just one of them?

A. None of them even. Because there's no guarantee we understand all the ways in which chemicals

cause cancer.

But seeing a mechanism strengthens the finding that this is probably causing cancer. Not seeing one doesn't do anything.

Q. And have you looked at the possible mechanisms through which Roundup or glyphosate could cause cancer?

A. Yes.

Q. And what are the mechanisms you've identified, that you have investigated?

A. In detail, there are two. Glyphosate causing DNA damage and glyphosate affecting the oxygen -- the movement of free oxygen radicals in the cell.

Q. And the first one, DNA damage, is that something called genotoxicity?

A. That's correct.

Q. I would like to talk to you first about genotoxicity, and then we can move on to the oxidation thing.

In your binder -- it's actually not in your binder. I have a copy right here.

MR. WISNER: Permission, Your Honor, to approach?

THE COURT: Sure.

BY MR. WISNER:

Q. Doctor, I'm handing you Exhibit 113.

Do you recognize this document?

A. I'm sorry, what was the question?

Q. Do you recognize this document?

A. Yes, I do.

Q. And what is it?

A. It's a cartoon, sort of showing you the different ways in which DNA can be damaged.

Q. All right. Would using this diagram aid you in explaining genotoxicity to the jury?

A. Sure.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection, Your Honor.

THE COURT: Yes.

BY MR. WISNER:

Q. So we're looking at this diagram here. Let's start off with genotoxicity, genetic damage.

A. Okay.

Q. What do you mean by "genetic damage"?

A. Basically, you change the inherent structure -- the inherent sequence of the DNA in some way, shape, or form.

Q. What does that mean?

A. Well, DNA is a long, long strand. And it's like reading a magnetic tape -- let's do it that way,

the simplest way.

You go up the strand to read it, and that decides what proteins get produced and how they get produced and how much and where and everything else to do the running of the cell effectively.

If you go in and clip that tape somehow, then you get a misread, something -- in music, you'll hear a blip on your earphones. Here, you change the protein expression in the cell, and that can change the way in which the cell works.

Q. Well, how does genetic damage relate to cancer?

A. Many of the cancers that we look at have mutations in the cells of the cancer itself.

So when you look at normal tissue in some way, and then you pull one of their cancer cells, the sequence of DNA is not the same in the two of them. And the cancerous cells have a changed DNA sequence.

Q. How, then -- so we have up here, for example, single strand breaks.

What does that refer to?

A. DNA is a molecule that basically has, sort of, two bands on the side. And all of the structures in between that hold the bands together, and it goes up like a ladder. Simplest way to think about it.

You can break one-half of that. So you think of a ladder, you just crack one side of the ladder and break it. That's a single-strand break. A double-strand break would be that you break both sides of the ladder.

The simplest way to think about this is with ionizing radiation. So that's like radiation from nuclear bombs and stuff. That radiation, when it hits your body, passes clean through your body. But as it's passing through your body, even though it's very, very small, it's hitting cells.

It causes single-strand breaks when it hits the DNA on one side; and if it hits the DNA just right, boom, it cuts both strands of DNA and you get a double-strand break.

So that's the simplest way to understand that type of DNA damage.

Q. All right. So we have -- so we just referred to that single-strand break.

What is this mismatch right here?

A. That has to do with the way the DNA was repaired. When you get damage to DNA, the repair machinery in the damage looks at the other strand of DNA. So you have two, and it looks to the other side and says, oh, you need this molecule here. You've got

this molecule, it's the wrong one; let's get rid of it and replace it with this other molecule.

Sometimes it gets that wrong and it says, you need this molecule. But it's wrong, so it's matched the wrong repair. So you get mismatched repair; it's not the right molecule there.

Q. All right. And then we have over here another one, intrastrand crosslink.

What does that refer to?

A. It's another DNA repair problem where you've now broken the rungs of the ladder that connect the ladder together. One of those rungs has bent over and filled in the hole on the other side, and the other two are just sitting out.

So you have a repair that tried to repair across, but instead went right up the same leg of the ladder.

Q. And when you have a substance that does this sort of damage to DNA, how does that, itself -- how does that lead to cancer?

A. Well, when the cell begins to replicate, it takes that ladder with the DNA and splits it apart.

So it takes half of the rungs of the ladder this way, half that way. If they're not the correct sequence of rungs, it's going to replicate that.

There's machinery that goes in and just builds the new ladder all over again for the new cell. And when it builds that new ladder, if one of those half-rungs is wrong, it's going to get the wrong ladder.

So things like the crosslink that you're looking at there lead to the wrong ladder. That's a mutation.

If that mutation is in a critical gene that, say, controls cellular communication -- cells talk to each other back and forth. And part of that talking to each other back and forth is to control growth of the tissue.

So the cells know when we need another cell. They talk to each other and go we need a new cell here, so it replicates.

If, for example, the mutation says, we're no longer going to talk to you, and we're going to replicate just because we want to replicate, those cells produce over and over and over again and that becomes a cancer. So it's the mutation aspect that's important.

Q. And on this diagram, we have a section called "Micronucleus."

A. Correct.

Q. What is a micronucleus?

A. It's a little piece of material -- nuclear

material, DNA -- that gets chopped off during the repair process. And it's an indication that DNA damage has occurred. It, in and of itself, is not a bad thing. But it being there tells you that this cell has been damaged. That's what it is.

Q. All right. Now, when it comes to looking at genotoxicity or any other mechanistic data, are you familiar with something called an in vivo study?

A. Yes.

Q. And that's V-I-V-O, right?

A. V-I-V-O.

Q. What is an in vivo study?

A. It comes from the Latin, I think, vivum, which is body. It's in the body.

So these are studies that are done in living, full, functional organisms; humans, rats, mice. That would be an in vivo study.

Q. And what's the other type of study called?

A. In vitro. And I don't know what that derives from in Latin, but that's in the cell.

So those are studies where you take cells, and you put them in a beaker or a little petri dish or something, and you put the chemical on it. That's an in vitro study.

Q. And when it comes to looking at data, do you

generally prefer in vivo or in vitro data?

A. That's going to depend. I have a hierarchy I work with.

Q. Okay.

A. So the most important information I'm going to look at will be things that have been observed in human beings. It's the species I'm interested in.

There are some things you can do to people who have accidental exposure or side exposure or something -- occupational exposure. And you can look for DNA damage in them and stuff like that. That's the best.

Barring I can't get that, then I want to look at human cells in the petri dish and whole animal studies. Because the human cells are humans. They're the right cells. But it's a petri dish; it's not exactly a functioning animal. And the animals are a full, functioning system, but they're not humans.

So I like to have both, and I look at both of them to get a feel for what's going on.

And then there's in vitro cells from the animals and all kinds of in vitro studies from non-animals: Bacteria, and things like that. And in vitro studies in animals who we wouldn't normally look at: Fish, frogs, things like -- in vivo studies in

fish and frogs. These are to look at the ecological impact of the compound. But they can also tell you something about DNA damage.

Q. So sticking with Roundup first, are there in vivo human studies, studies of Roundup genetic damage in living people?

A. Yes.

Q. Are there glyphosate in vivo human studies?

A. No. Because there are no studies of pure compound in people.

Q. And is that because people don't spray pure glyphosate; they spray Roundup?

A. That's correct. And also because we don't do laboratory studies on people.

Q. And then when we go from in vivo and go to the petri dish studies.

Are there studies that looked at human cells in a petri dish?

A. Yes.

Q. And to see whether or not, when you apply glyphosate or Roundup, does it cause genetic damage?

A. Yes.

Q. And in those studies, have they used both Roundup, or formulations, as well as glyphosate?

A. Yes.

Q. And you've looked at all of these studies, I assume?

A. Yes. I think so. It's a broad literature.

Q. When you draw like the circle around it and we look at all the studies -- whether it be in humans, animals, bacteria, hairy -- what is it? Hairy armadillos?

A. Oh, yes. The hairy armadillos from Peru, yes.

Q. How many studies are we talking about?

A. I don't know. Near a hundred, maybe a little more than a hundred studies.

Q. Okay. So let's start off with the human ones.

Have any researchers ever gone out and looked at people who were exposed to Roundup to see if they had genetic damage?

A. Yes.

Q. Who did that?

A. Paz-y-Mino in Ecuador; and a guy named Bolognesi in -- I believe it was Peru? Guatemala? In Central America somewhere.

Q. Let's talk about the Paz-y-Mino. If you turn to your binder, Exhibit 1826.

Is that a copy of the Paz-y-Mino study?

A. He did two studies. That is one of the two studies.

Q. Okay, great. And it just occurred to me that I don't know if I'm saying his name right.

Is it or --

A. I wouldn't really know.

Q. Okay.

MR. WISNER: Permission to publish, Your Honor.

MR. EVANS: No objection.

THE COURT: Okay.

MR. WISNER: So we're looking here at the study.

BY MR. WISNER:

Q. What year was this study, Doctor?

A. It looks like it's published in 2011.

Q. Let's talk about the first one.

What was the first study looking at, sir?

A. They were looking at people who lived in regions of Ecuador near where the government was spraying for illegal drugs. And they -- and then people who lived much further away.

And the spraying, I believe it was for illegal drugs. The spraying for illegal drugs was using Roundup to deal with that.

And the hypothesis was that the people who lived near these fields would get some degree of

exposure to the Roundup, and people who lived far away would not; and if we compared their blood and the DNA in their blood, you could look to see if they had DNA damage.

Q. And when they went out and looked at these people who had actually been sprayed with Roundup, what did they find?

A. They found that the people who lived near the areas that were being sprayed had high DNA damage compared to the people who lived further away, in areas that weren't sprayed.

Q. You said there was another study by Dr. Bolognesi, right?

A. Correct.

Q. How was that study different than the Paz-y-Mino study?

A. So the Bolognesi study was done in a different place. I'm not sure which country. There are a lot of studies here.

That study, they knew -- that study has five communities that they're looking at. One community is a farming community that does organic farming. So there's no pesticides, theoretically, in that community.

The other four communities, if I'm remembering correctly, three of them are in areas that are sprayed

for illegal drugs, getting rid of illegal drugs. And one of them is in an area where they grow sugar cane, and they spray with Roundup to dry out the sugar cane before they harvest it.

And so the -- in that case, they're expecting the people who are exposed -- live near there to get exposed. And people in the organic didn't get exposed.

But this had a better study design, because they also knew when the exposure was going to occur. They knew when the spraying would occur.

So roughly five days before the spraying, they went in and took blood from people. And then five days to two weeks after the spraying, they went in and took blood from the same people again. So now they're comparing blood from before spraying to after spraying to see if there was DNA damage.

And they did find DNA damage.

Q. Compared to who?

A. Compared to themselves.

Q. Okay.

A. You're your own control. I've given blood before they sprayed, I've given blood after.

Q. What about relative to the organic farming communities?

A. They were elevated relative to the organic

farming community, as well.

Q. You said three of those communities were from aerial spraying; is that right?

A. Correct.

Q. And then one was from a farming community for sugar; is that right?

A. Yes.

Q. Based on your understanding of exposures in these types of studies, which community would you expect to have the highest level of exposure?

A. Oh, definitely the sugar cane farming community. Because I would expect that some of those people were going in the field to help harvest the sugar cane, or to steal sugar cane for their own use. So you would expect them to have a little higher exposure; and, indeed, they did.

Q. Have you ever helped harvest sugar cane?

A. I've managed to go in and maybe borrow some sugar cane when I was growing up in south Louisiana.

Q. Now, what did the genetic damage show for these higher-exposed individuals in the sugar cane community?

A. It just showed clearly that there were DNA damages going on. I would have to look at the paper to see the exact way they measured it. I think it was the

common assay. So you're looking at fragments of DNA that exist after -- before repair.

Q. Now, what did these studies show relative to timing of exposure to the DNA damage?

A. In the sense that -- oh.

Q. How soon after spraying did they observe the genetic damage?

A. It was quickly after spraying. And then when they looked further down the line -- I think it was six months later -- in some communities, it was back to normal. In others, it was still somewhat abnormal, but it was definitely reduced.

Q. Now, in the concept of understanding the mechanism through which a chemical can cause cancer, what, if any, is the significance of the fact that, immediately after spraying, you have genetic damage, but then it goes away over time?

A. It's expected. This is normal behavior. We're talking about human blood; we're talking about lymphocytes. You're talking about -- well, just blood in general. And it doesn't stay around forever.

It's produced, it's used. And as it gets old, your body cleans it out and gets rid of the -- everything there. So the cells are what are called terminally differentiated; they're going to die and be

removed.

So just through that rate, you would see it disappear. The DNA damage would go away.

Q. But, Doctor, if we're talking about individuals who are exposed weekly, and you're showing immediate genetic damage, what does that tell you in the context of understanding the mechanism?

A. Well, in that situation, they would constantly be getting more damage over and over again. So you get damage, you would be repairing it. You would get a new damage, you would be repairing that. It would be a constant cycle.

Q. And when you do that, you repeatedly damage the cells over and over and over again.

Does that increase the risk of having a mutation or having cancer?

A. It clearly indicates the risk of a mutation, which then, in itself, increases the risk of cancer.

Q. So that was the in vivo data for genotoxicity. Let's talk about the in vitro data, specifically in human cells.

Have you reviewed that data?

A. Yes, I have.

Q. I understand you prepared some charts highlighting -- going over those data; is that right?

A. Yes, I have.

Q. If you look in your binder, it's Exhibits 102 and 104.

Are those the blank charts that you've put together?

A. Yes.

MR. WISNER: Your Honor, permission to publish Exhibits 102 and 104.

THE COURT: Mr. Ismail?

MR. ISMAIL: No objection, Your Honor.

THE COURT: Permission to publish.

MR. WISNER: Thank you, Your Honor.

MR. ISMAIL: May I, Your Honor?

THE COURT: Oh, yes, go ahead.

BY MR. WISNER:

Q. We have here a blow-up of Exhibit 102.

Dr. Portier, please walk me through what this chart is showing.

A. So under the column marked "Study," that's the name of the study, the authors of the study, and the year in which it was published -- or at least the first author; there are multiple authors, usually.

Some of the studies did pure glyphosate, some looked at a formulation, and some looked at both.

So if it didn't study pure glyphosate, you'll

see no data in that column that says glyphosate. If it didn't study the formulation, you'll see no data in the column that's marked "Formulation."

Q. So just to clarify, I see Bolognesi from 1997. Is that the same study we were talking about?

A. No, but it's the same Bolognesi.

Q. It's the same researcher, but a different study?

A. A different publication, yes.

Q. This is in vitro; so in a petri dish, right?

A. Yes.

Q. And the Bolognesi study a moment ago, that was humans?

A. The people were exposed, and then you had to do the analysis using petri dishes and other things. But yes, the people were exposed. Here, the cells are exposed directly.

Q. Let me make sure I understand. For example, in Vigfusson and Vyse, they did not study glyphosate, but they did study a formulation?

A. Correct.

Q. And they're studying specifically whether or not a formulation here is genotoxic?

A. That is correct.

Q. And this is in human cells?

A. That is correct.

Q. So I have this key, "positive" and "negative." Let's start off with the first one.

Was this one positive or negative?

A. It was positive. And there, by "positive," I mean that you're looking at a result that is unlikely to have occurred by chance, after looking at the statistical test and looking at the way in which the results are presented and how they did the study.

It's a valid study, and it appears not to have occurred by chance.

Q. And by "it occurred," what does "it" refer to?

A. The genotoxicity that's being measured. There are many different ways to measure genotoxicity. I haven't specified the methods in each of these cases; it would get too complicated.

Q. So this one showed a significant genotoxic effect of a formulated glyphosate product?

A. Correct.

Q. All right. What about Bolognesi, 1997?

A. It was positive for glyphosate and positive for the formulation.

Q. All right. What about Lioi in 1998?

A. It's positive for the glyphosate.

Q. Lukken?

A. It's positive for glyphosate.

That's an interesting study, because it's a two-chemical study. They also used hydrogen peroxide, so that one has a little more complicated interpretation.

Q. Fair enough.

But bottom line, it showed genotoxicity?

A. Yes.

Q. All right. Monroy, 2005?

A. They looked at two different cell types. I guess I have to be clear here.

There are different types of human cells being used here. And like we do with the animal studies, toxicologists love to have something you can repeat.

So what we've done is created these human cell lines from a particular human. And we've replicated the cells, break them into little patches, freeze them, and share them.

So we all try to keep the same cells so that, again, if I do something in my lab, and you do something in your lab, hopefully we get the same answer because we're using the exact same cells.

And so most of those cell lines are called immortal cell lines because they never die. You just grow up a whole bunch of new ones, and you can do more

studies. This one, the Monroy paper, they used two different immortal cell lines.

So that's plus in both of them. It's positive for both cell lines that they looked at.

To clarify the picture here, Lukken is also an immortal cell line. But the first three -- the Vigfusson, Bolognesi, and Lioi -- they went to people, got blood from people, took lymphocytes from the blood, put them in a petri dish, cultured them, and exposed those lymphocytes to glyphosate or the formulation.

So those are cells from living, breathing humans. And they can differ from human to human, but they culture them and then do the study once. I can't ever replicate that because I won't have the exact same human. So it's a slightly different study.

Q. You said they look at lymphocytes?

A. Correct.

Q. How many of these ones looked at lymphocytes, the ones we've gone through so far?

A. The first three.

Q. Lymphocytes, that's where lymphoma happens, right?

A. Yes.

Q. Let's go to Gasnier, 2009.

A. That one, they report as positive. I have

some questions about it. They didn't report their findings very clearly. I was uncomfortable with their positive, so I put a question mark on that one.

Q. So the authors say it's positive, but you're saying not so fast?

A. I'm saying not so fast.

Q. So I'm going to put a positive in parentheses, and then a question mark.

Does that work?

A. That works.

Q. Manas, 2009?

A. That one was positive for glyphosate. And both of those were immortal cell lines.

Q. Both of these two?

A. Yeah.

Q. Were any of them in lymphocytes?

A. No.

Q. Mladinic, 2009?

A. There are two of those, and hopefully they're both on there.

Q. Yes.

A. The first one was in lymphocytes in healthy humans. It was positive.

The second one did a different end point, also in humans. I couldn't tell if it was the same humans,

and they took the blood and published two papers because they used two methods.

But that one, on the second and third and fourth method, because they had three in that one, it was also positive.

Q. So three pluses?

A. No, just one. It's positive in lymphocytes, they're just reporting it different ways. Those may be the same humans in both of those studies.

Q. Gotcha. Was that also lymphocytes?

A. Yes. That was lymphocytes. It could be the same lymphocytes.

Q. What about Koller, 2012?

A. That's positive for both glyphosate and formulations. And that is immortal cells, not lymphocytes.

Q. Alvarez Moya, 2014?

A. That is positive for glyphosate, and that is lymphocytes.

And we missed one study, I'm sorry.

Q. Which one did we miss?

A. So we have Manas there in 2009?

Q. Yes.

A. Manas did two different studies. We have to report slightly differently, because they did a study in

lymphocytes.

So one study was immortal cells, that one was positive. They did a study in lymphocytes, humans, and that one was negative.

Q. So there's a negative study?

A. Yes.

Q. Does that accurately reflect that? And I guess I should put lymphocytes here, as well?

A. But the lymphocytes only applies to the minus.

Q. Okay. We'll just remember that.

Let's look at the next chart, because I know there's a lot more data here. Exhibit 104.

Doctor, these are a little more recent studies?

A. Yes, a little more recent than the last ones.

Q. And it looks like they did them from 2017 to 2018, right?

A. Right.

Q. What about Townsend, 2017?

A. That is positive, and that is immortal cell lines.

Q. The next one?

A. Yep. Luo, that's an immortal cell line. They list it as positive; I'm not convinced. I think the study was probably negative, but again I would put a

question mark on it.

Q. So they reported it as positive, but you're not so sure about it?

A. That's correct.

Q. What about the next one?

A. Kwiatkowska. That one is positive. And that one is human blood. They didn't specify whether it's lymphocytes or some other aspect of human blood. Or at least my notes don't say that. I can just say human blood.

Q. Just to be clear, lymphoma is a cancer of the blood?

A. It's lymph cells, usually in the blood, yes.

Q. The next one?

A. Kasuba. That's immortal cell lines, positive.

Q. Wozniak?

A. Again, that one is blood. This time, it's red blood cells from humans, and it's positive in both cases.

The interesting thing there is that they used fairly low exposures. Many of these studies used low exposures and high exposures, and only the high exposures were showing positives. This one study had range; even down in the low doses, they were seeing positive findings.

Q. What about -- what's the relevance of seeing genetic damage, even at low doses, in human blood cells?

A. Well, the lower dose you can go, the better. Because that's more relevant to the human situation. Some of these doses are higher than you would see in humans.

Q. Santovito?

A. That's lymphocytes in humans, and that was also positive. And another one that had very low doses.

Q. And that was glyphosate?

A. Yes.

Q. De Almeida?

A. De Almeida did immortal cell lines. And they were asking a slightly different question, but they still were looking for genetic damage.

They had cells that respond to estrogen. Estrogen is a hormone; and some cells in the body respond to estrogen, some don't.

So they were using one of these immortal cell lines that responds to estrogen, another one that doesn't respond to estrogen, and another one that's a breast cancer cell line that responds to certain types of estrogen.

There, they saw no effect on the estrogen-responsive cells, so that was negative. And

this is the same on both sides. And it was positive for the estrogen non-responsive and positive for the MCF7.

So it's negative, positive, positive; three different cell lines.

And then the last one, Anifandis was done in human sperm, and that one was negative.

Q. All right. If we sort of look at this human in vitro data combined -- all these results in human lymphocytes and blood -- in your opinion, does this evidence show that glyphosate and Roundup are genotoxic in human lymphocytes?

A. Yes.

Q. I want to talk to you about another aspect of genetic damage.

Remember earlier, we talked about micronuclei?

A. Yes.

Q. Has there been any sort of meta-analysis or study that looks at the emergence of micronuclei following exposure to glyphosate and Roundup?

A. So a micronucleus test -- looking for micronuclei -- is one of the tests that's generally required by most governments to allow a pesticide to be used in commerce. So it's a very common assay, and many people have done micronucleus assays and submitted them to the government.

In addition, there have been some done in the literature, so there are also micronucleus assays there. There has been a group that tried to pull all of this evidence together and do one big evaluation of the micronucleus data that's available in the literature.

Q. All right. If you turn to Exhibit 2116 in your binder.

Is that the study that you're talking about?

A. Ghisi, yes.

Q. Is this the study you relied upon in forming your opinions?

A. In part. I read all of the studies, as well as looking at their overall analysis.

But yes, it's part of what I looked at.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection.

THE COURT: Permission granted.

BY MR. WISNER:

Q. All right. So we're looking at this article. It's titled: "Does Exposure to Glyphosate Lead to an Increase in the Micronuclei Frequency? A Systematic and Meta-Analytic Review" by Drs. Ghisi, De Oliveira, and Prioli.

Do you see that, sir?

A. Yes, I do.

Q. Can you explain what these researchers are trying to do in this study.

A. Every time you do a study, you measure something. In this case, they're measuring micronucleus. So everyone is doing the same basic kind of study.

And when I have five or six studies of the same kind, one thing I would like to do is figure a way to bring that evidence together in one picture and ask myself, given all of this, is it chance or is it real that this happened?

So I'm going to combine -- I'm going to analyze the analysis. I'm going to take what other people have found and pull them all together and do one big analysis.

That's what they're trying to do here, is ask with one big statistical test: Does it appear that glyphosate increases micronucleus frequency?

Q. Okay. So if we look on the document, starting on Table 1 -- I'll just pull up a little bit of it -- it starts listing studies; do you see that?

A. Yes, I do.

Q. What is this listing, exactly?

A. It's similar to what we've done before.

They're looking at a reference.

So let's look at the first one. It's a study by Poletta, et al. in 2011. The study is in crocodiles. So they give you the species, which is C. latirostris or crocodylia. Is this a standard test system that people use? No.

And then they have all these other things they put in there in terms of the results, like what was the dose used? Were there micronuclei there? Were there not? Et cetera. What's the variance around the result? Et cetera.

Q. If you look at this chart, there are a lot of different studies referenced. It goes on to the next page. You have the Bolognesi study from '97 there.

Do you see all that, sir?

A. Yes, I do.

Q. All right. And if you go to the next page, there's this diagram right here that popped up on the screen.

Please explain to the jury what this diagram is showing.

A. Okay. So for each of these studies, for each of the doses, you get a micronucleus count. And then you get sort of an idea how much range there is around the probability that it is due to change.

So you might have a number like seven is the measure of how many micronuclei they have; and the reasonable range around that seven is, say, three to 12, okay?

And so that's what's called a 95 percent confidence limit. We're 95 percent sure that the mean reaction falls in this range. The mean number of -- the average count of micronuclei from this experiment somehow falls within this range.

So what you're looking at here is a forest plot. The Y-axis is meaningless. All you want to do is look at the X-axis.

If the estimate is 0, that means there were no micronuclei. And so this cell wasn't changed.

If the number is less than 0, that means there are fewer micronuclei than there were in control.

So you're always comparing against the control group here. So 0 means there was no difference from control, negative means I actually had fewer micronuclei to control, positive means I had more micronuclei to control.

Each little line there has a little plus in the middle. I don't know if you can see that. But there's a little plus. That was the best estimate of what was seen in that experiment at that dose.

And then that line that goes out from it, that's the 95 percent confidence region, where I think it could have been. And then you've got each experiment listed there going up the chart.

Q. All right. And so if we look in here, there is something that says "grand mean."

Can you see that, sort of?

A. Yes.

Q. And it's hard to see. But you see right there, that little spot?

A. Yes.

Q. What's that referring to?

A. So when you do -- now, this plot is showing each individual result. And then what you're doing in your meta-analysis is, you're bringing all those results together to ask the one question: What is the general tendency here?

That grand mean is the answer to that question. That is the general tendency.

Q. All right. And if you look at the actual full-on -- the full chart, the grand mean is to the right of the line, right?

A. That's correct. And so is its 95 percent confidence region. So that says, basically, this is positive for micronuclei, and it's not due to chance.

Q. Let's look at some of the other charts they give in here. For example, they break it down in Chart A between different animals.

Do you see that?

A. Yes.

Q. So we have the grand mean on here still.

Is that the same one as before?

A. Yes.

Q. And we have 0 over here; is that right?

A. Correct.

Q. And so anything to the right of that line is positive for causing micronuclei?

A. That's correct.

Q. Okay. And if you break it down to different animals, for mice, it's higher than the grand mean.

Do you see that?

A. Yes.

Q. But, for example, for fish, it's lower?

A. Correct.

Q. What does that tell you, looking at this sort of data?

A. Well, remember, there are not that many studies in fish and crocodiles and amphibians. So it has less weight, in my mind, than the others. But there were a lot of mouse studies.

So what that tells me is, with the mice, it's positive in the mice.

Q. Okay. And then we have another Chart B. This is looking at non-mammals and mammals.

Do you see that?

A. Yes.

Q. What is the significance of seeing the mammalian data being farther to the right than the non-mammalian data?

A. Again, it suggests that mammals are more susceptible to DNA damage from exposure to this massive collection of exposures than are the non-mammals. Because not all these studies used exactly the same exposures.

Q. We have another one here. It looks like different types of exposure.

Do you see that?

A. Yes.

Q. And we have, over here, the grand mean.

Is that the same grand mean?

A. Yes, it is.

Q. And then we have "Spraying."

Do you see that?

A. Yes.

Q. And that's to the right of the line.

Is that right?

A. That's correct.

Q. Whereas if you look at "Oral," that one is actually to the left of the line?

A. Just a little bit. The mean is a little bit to the left of the line.

Q. What does that tell you?

A. Well, when you look at all the studies of exposure to glyphosate via the oral route, it doesn't appear to show, in that collection of data, that there's an effect on micronuclei.

Q. What did it tell you about spraying, though?

A. Well, again, the spraying is a real collection of all kinds of animals. But it tells you that, in the spraying situation, it appears there is micronuclei being formed.

Q. All right. And then just a few more here. We're almost done.

We have it broken up by gender.

Do you see that?

A. Yes.

Q. And is this for humans, or is this for all species?

A. I would have to look at the bottom of the table. I don't think it's humans. But I'm not sure if

it's all mammals or not.

Q. Okay.

A. Okay. So this is all males or all females, regardless of species.

So this just says that, in general, males are more susceptible than females, given the data that they're looking at here.

Q. Okay. And then finally, on the last page here, there's one that looks at Roundup and glyphosate.

Do you see that?

A. Yes.

Q. And Roundup is farther to the right than glyphosate.

What does that suggest?

A. That would suggest that Roundup is more effective and efficient at causing DNA damage than is glyphosate pure.

Q. Almost done here. Last chart.

We have difference between peer-reviewed data and non-peer-reviewed data.

Do you see that?

A. Yes.

Q. What is peer-reviewed data?

A. Data that appears in the publicly-available literature, things I would normally look at. Articles

in Science magazine or the Journal of the American Cancer Institute -- National Cancer Institute or something like that.

The non-peer-reviewed is studies that are done specifically by the industry to support the registration of a chemical. That's the general rule for non-peer-reviewed. And those are more difficult for somebody like me to look at, but nonetheless, they're out there sometimes.

Q. And what does it mean, the fact that the studies that have been subjected to the peer review process -- publicly available -- are farther to the right than the ones that haven't been?

A. Well, there are all kinds of things that could suggest. The obvious one is, it appears that the peer-reviewed literature more commonly shows a positive effect for micronuclei than does the regulated industry literature.

But that could be due to real difference.

That could be due to the peer-reviewed literature using Roundup more often that glyphosate, because Roundup appears to be more toxic than glyphosate.

That could be due to what they call publication bias, where journalists like to publish

positive results and not negative results.

That one is less likely to explain all of it, given the bulk of the data there.

And there may be other explanations. The most likely for the big difference there is Roundup versus glyphosate and some -- a little bit of publication bias.

Q. And Doctor, having -- we went through the genotoxicity data in these charts, and we've gone through this meta-analysis looking at micronuclei frequency.

My first question to you is: How strong is the evidence that, in fact, Roundup and glyphosate are genotoxic?

A. I will make it very clear: Glyphosate is genotoxic, Roundup is genotoxic.

Q. And which one appears to be more genotoxic?

A. Roundup appears to be more genotoxic than glyphosate.

Q. We talked about there being two mechanisms that you looked at.

One was genotoxicity, right?

A. Correct.

Q. And the other one was what?

A. Oxidative stress.

Q. What is oxidative stress?

A. Well, the term "stress" is clear. It just means that in some way, the cell is being stressed; it's having a bad day. Just like cells have random DNA damage all the time and get repaired, cells generate free oxygen radicals. These are -- you know, oxygen -- I don't remember all my chemistry all the time.

Oxygen in the air appears as O2. Two molecules of oxygen bound together. When you break them apart, you have a single oxygen molecule; that is a free oxygen radical. That thing doesn't like to be by itself, so it will bind to hydrogen to form water. It will bind to anything it can find to bind to.

So your cells use that oxygen as an energy source. They're constantly breaking it, using it to bind something else, and doing things in the cell. It's one of the main sources of energy in the cell.

So you constantly have free oxygen radicals running around in your cells. They're very dangerous, because they can bind to DNA. And if they bind to DNA, they can damage DNA just like we saw for genotoxicity. And you don't want that happening. And they have machinery to try to keep that from happening.

But if you change the biochemistry of the cell so that there's more free oxygen radicals than usual, then you have more of this oxygen running around and

binding to all kinds of things. You have a higher chance of getting it to bind to DNA and a higher chance of genotoxicity, and you're back down that same road to getting cancer from genetic damage.

Q. Have you heard the term antioxidant?

A. Of course. That's a good term to use here.

Q. How does that relate?

A. An antioxidant is intended to take free radicals and pull them out of the system.

Q. This mechanism, through the generation -- an imbalance in oxygen particles, is that a known or recognized mechanism through which something can cause cancer?

A. Oh, yes.

Q. And have you looked at the data on oxidative stress for glyphosate and Roundup?

A. Yes, I have.

Q. Now, previously we talked about genotoxicity data for in vivo; so living, human beings.

Does that data exist for oxidative stress?

A. No. No one has ever done an oxidative stress study.

Q. So we don't have them in living humans.

Do we have oxidative stress data in human cells, in vitro?

A. Yes, we do.

Q. Have you looked at that data?

A. Yes, I have.

Q. Did you prepare a chart for that one, too?

A. I believe I did.

Q. Take a look at Exhibit 100.

Is that a copy of that chart, sir?

A. Yes.

Q. Okay, great.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection.

THE COURT: You may publish it.

BY MR. WISNER:

Q. All right, Doctor.

Can you see it?

A. Yes.

Q. All right. Same deal as before.

A. It's the same basic layout. The study name, and then whether it was done in glyphosate, alone, or as a formulation.

Q. All right.

A. And positive or negative.

Q. Great. So let's run through it.

Start with the first one. Gehin, 2005.

A. It was positive for both glyphosate and for the Roundup. And that is immortal cells, not human lymphocytes. It's still human, but it's immortal cells.

Q. Mladinic?

A. That's the same study we saw earlier. And that is positive, and that's lymphocytes in humans.

Q. So to be clear, this -- in this Mladinic study, they looked at genetic damage specifically?

A. Correct. And they looked at oxidative stress. They did both.

Q. So the same authors for 2009?

A. Yes.

Q. And it was positive?

A. That was positive.

Q. And that was lymphocytes?

A. Correct.

Q. The next one?

A. Elie-Caille, I guess. That's a mortalized cell line, so it's not lymphocytes. They said it was positive.

But the assay they used there is -- there's a way to get cells to glow. And then you can run them through a tube, and there's something that looks for the glow and it counts the cells. And if the cell is glowing, it's got oxidative stress; and if it's not

glowing, it doesn't.

So it's a very fast way to do it, but it has some limitations in the interpretation. And I think, in this case, those limitations were significant enough that I'm going to put a question mark on that. Or inadequate, one or the other.

Q. So again, they reported as positive?

A. They reported as positive.

Q. But you're not sure about it?

A. No.

Q. What about the next one?

A. George and Shukla.

Exactly the same thing. They report as positive, but they use the same assay. And they didn't care as much about how they were doing it as they should have. I've got a question mark on that one, too.

Q. All right. What about Chaufan?

A. That was clearly negative for glyphosate and positive for the Roundup.

Q. What's the significance for that; that it would be negative for the technical, but positive for the formulation?

A. It could be any number of reasons why it's negative. Sample size is too small, they're using a different technique than anybody else, and it doesn't

show up positive. It may be a true negative; that really, it doesn't happen.

The fact that you see it in the Roundup -- if this is the only study I had, and no other study, then the obvious interpretation is that it's not the glyphosate.

Q. Gotcha.

A. But, of course, there's more than this one study.

Q. Sure.

Is this result consistent with what we've talked about previously, about Roundup being more toxic than glyphosate?

A. Yes, it is.

Q. All right. The next one, 2014?

A. Coalova. That one is positive. And all of these are immortal cell lines. I'll tell you if it's a human blood or not.

Q. Okay. We saw this name last time.

A. I'm not even going to try --

Q. 2012.

A. I'm not going to try the name. That's in red blood cells. And that one was positive.

Q. What about Luo?

A. That one is also positive, but it's an

immortalized cell line, not human blood.

Q. Last two?

A. Kasuba. Here, we've got the opposite.

Kasuba said, we didn't see anything. Yet when I look at what Kasuba did and the data they have and the analysis they have, I'm going to call that positive. So we're in disagreement; the author and myself are in disagreement.

Q. So I'm going to put the negative in parentheses, and the plus reflecting your opinion.

A. Okay. And then Wozniak, 2018. It's positive for both. They also used the fluorescence assay, but they did other things.

One of the problems with the fluorescence assay they took care of. That's a much better-done study, much better-reported. So even though they used fluorescence, I'm going to agree with their positives on that study.

Q. And looking at the data here -- the human cell data -- what does this tell you, sir?

A. That it's obvious that glyphosate and Roundup both induce oxidative stress in the cells.

Q. And specifically human cells?

A. In human cells.

Q. Great.

A. Oh, and Wozniak was blood, by the way. Human blood from volunteers. Not from volunteers; they got it from the blood bank in Poland.

Q. Okay. You mention that there's these immortal cell lines that are used in different studies across researchers.

Did they come from some person at some point?

A. Yes. Usually they come from a cancer cell in a person at some point.

Q. What does that mean?

A. Well, it's -- normal cells don't become immortal. You can't immortalize them very easily.

But cancer cells, because they've lost growth control, they don't communicate as well as others. So it's easier to put them in a petri dish and get them to grow permanently for you and stay alive in batches for very long periods of time.

So most of them arise from things like a hepatoma that somebody had at some point. There's a Hep G2 that comes from a liver tumor that somebody had at some point, et cetera.

Q. But the ones that look at lymphocytes in blood, that's coming from --

A. Normal, healthy blood.

Q. All right. Let's go back to that document

camera.

We're kind of marching through the three pillars here. We've done the animal studies, and we just got through the mechanism studies.

Before I move on from mechanism studies, there are also data in animals and bacteria and stuff like that, right?

A. Yes. Every single chemical that, pretty much, is on the market today has been tested in what's called the Ames assay.

And the Ames assay looks for -- it takes salmonella, and it's a special salmonella; it won't grow in a petri dish because it has a mutation in it. And they expose that salmonella to the chemical. And if the chemical damages the DNA enough, the salmonella gets a mutation in the mutation that repairs, and the cells can then replicate. And you get little colonies of bacteria growing in the petri dish.

So that's called the Ames test. And they do that for everything. And so it's the simplest, cheapest, easiest way to look for DNA damage.

In this case -- I don't know -- there were 40 of them. There's tons of people who did Ames assays. They're all negative. There are one or two positives in there, but uniformly, they are negative. So it doesn't,

in that assay.

But there's also things people have done in fish and crocodiles and whatever that show positive findings.

Q. So to be clear, when you're weighing this cell data, and you're looking at human cells or even living humans, and you're comparing that to salmonella, which one is more convincing in understanding whether or not something causes cancer in humans?

A. Well, to me, it's clear that the human cells take priority over the cells in the Ames assay. Absolutely no doubt about it.

Q. All right. Let's move on to the third pillar here, epidemiology.

Now, Dr. Portier, our next witness -- our first witness next week is Dr. Ritz.

Do you know Dr. Ritz?

A. Yes.

Q. What does she do for a living?

A. She's an epidemiologist.

Q. So we're going to have Dr. Ritz here explaining to the jury the epidemiology data in significant detail, so I don't want to spend too much time on it with you today.

But I do want to ask you if you looked at the

epidemiology data as it relates to Roundup.

A. Yes, I have.

Q. And when we talk about epidemiology data, are we talking about glyphosate or are we talking about Roundup or formulated products?

A. We're talking about formulated product, no doubt about it.

Q. Why?

A. Because humans -- well, if there were an occupational study in a factory where they make glyphosate, then you might have a human population with pure exposure to pure glyphosate. But beyond that, when it leaves the factory, it gets formulated.

And when it gets to farmers and you in your backyard, it's, of course, mixed with other chemicals. So it's always the formulation you're looking at in human populations.

Q. And I understand that you've reviewed the data, and you've prepared a chart sort of summarizing some of the data you reviewed; is that right?

A. Yes, I have that.

Q. If you look at Exhibit 105, is that one of the charts that you've put together?

A. Yes, that is one of the charts I put together.

MR. WISNER: Permission to publish,

Your Honor.

MR. ISMAIL: No objection, Your Honor.

THE COURT: Permission granted.

BY MR. WISNER:

Q. I'm going to view this on multiple levels. I'm going to show it on the screen, and I also have a blow-up of it.

MR. WISNER: Your Honor, can I have Dr. Portier walk down and, for one minute, explain what these things mean.

THE COURT: Sure. Unless there's an objection.

MR. ISMAIL: No, Your Honor.

THE COURT: Go ahead, Dr. Portier.

THE WITNESS: We are looking at the same thing we saw before. This is the study that I'm looking at, and some of the studies have multiple evaluations in them. So I've included some of the multiple evaluations, and I'll explain those as I go through.

What we're looking at here in these epi studies is the risk ratio.

So this is the ratio of risk of people exposed, and the risk of people who were not exposed. If that ratio is 1, then there's no difference and there's no effect.

If the ratio is above 1, then there is a difference and it's bad for the people. They're getting the disease, they're getting the NHL.

If it's below 1, they're actually protected from the NHL.

There's a 95 percent confidence bound. So there's a lower value and an upper value. Remember that there's these areas, confidence regions, where you're pretty confident that it's going to fall in this area.

And then there's the same chart we saw before; this is a forest plot. So let's look at this first one. Andreotti, the relative risk is 1.12. That's what this is on the lower right. And then the lower bound is .83. That's the edge of this little whisker coming out of the box. And the 1.51 is the upper edge of the risk. So you're seeing the same sort of thing we saw before.

BY MR. WISNER:

Q. Thank you, Doctor. I just want to quickly ask what these three categories are.

The first ones are red. Do you see that?

A. Yes. The red studies, those are cohort studies.

Epidemiologists do different types of studies. A cohort study is where you identify a group of people who you think might be exposed to this, and you follow

them for a long period of time. And you look to see if those who are using the product, in this case, get cancer; and those who don't use the product get less cancer.

So the risk ratio you're calculating is from evaluating those who were exposed against those who were not.

Q. And what do the blue ones reflect?

A. Those are case control studies.

So in a cohort study, you're looking at risk in people who are exposed. In a case control study, you're looking at exposure in people who have the disease.

Here, you're doing something very different. I take a bunch of people who have non-Hodgkin's lymphoma and a bunch of people who don't, but sort of look like them. So I match them on age or I match them on race or I match them on sex, gender. But I get, kind of, the same looking people.

And then I ask them questions: Did you ever use Roundup or glyphosate or anything like that? And they answer yes or no. And then what I'm looking at is the risk ratio of the ones with the disease who said yes to the ones without the disease who said yes.

Are you more likely to have been exposed if

you got the disease than if you didn't have the disease?

So it's looking at the exposure end point rather than the disease end point.

Q. And when you have a point like this point right here, is that the same thing as these points in the cohort studies?

A. Yes. The risk ratio is the same general concept, although the mathematics are slightly different.

Q. And if it's to the right of the line, does that indicate an increased risk?

A. It indicates an increased rate of exposure to glyphosate if you had NHL.

Q. But it's read the same way as the cohort study?

A. Correct.

Q. And then these green ones, what do those reflect?

A. Remember, we just looked at the Ghisi study, which took a bunch of studies and pulled it together to get the grand mean and things like that. That's what they did here in these studies.

These studies take the other studies from above, bring them together in one analysis, and then give you a mean and that confidence region around that

one analysis.

And this column that says "Included," where I have, for the first one, Schinasi and Leon, B, D, F, I, K, M. If you look at the studies, I have them labeled A, B, C, D, et cetera.

So Schinasi and Leon took studies B, D, F, I, K, M, combined them, and did an analysis. And that's what they got in the overall analysis.

Q. I just want to ask you one sort of global question: What, if anything, is the significance of most of these being to the right of the blue line?

A. Well, let's start with a simple analogy first. If I have a coin, and I flip the coin in the air, you would expect that half of the time, I would get heads; and half of the time, I would get tails. It's a fair coin, so that's what you would expect.

Here, when we look at this, this risk ratio can lie above 1 or below 1. All right?

Now, if risk ratio is truly 1, if the truth is that there is no difference -- that glyphosate does not cause NHL -- then by random chance, it's like flipping a coin. By random chance, some of them are going to be the dot to the right; some of them are going to be the dot to the left.

But in this case, that's not what happens.

Virtually all of the dots are to the right. So that tells me, simply enough, that this is an unlikely picture in the case that this was a true risk ratio of 1. It's very unlikely.

And I can actually calculate that probability, if I have to, but it's very unlikely.

Q. How do you go about calculating that probability? I'm actually kind of curious about that.

A. It's the same way as the coin flips. If I flip a coin and I get heads, and I flip it again and get heads the second time, the chance of getting heads is one-half; 50 percent heads, 50 percent tails.

The chance of getting two heads in a row is one-half times one-half, because they're independent events, so it's one-fourth. And if you think about it, that makes sense. Because you can get heads/heads, heads/tails, tails/heads, and tails/tails in two flips of the coin, right? So there are only four outcomes that are possible, and one of the four is heads/heads.

So if I flip it three times, and I get heads a third time, it's one-half times one-half times one-half, which is one-eighth. Because you have eight outcomes, and that's only one of the possible eight outcomes.

So if I have -- I don't know how many there are there. But if you just look at "never exposure" in

these studies that you're looking at here, then you have -- I believe it's six studies that are positive, all above or equal to 1, and that's one-half times one-half times one-half times one-half times one-half times one-half, which is 1/64, which is 3 percent probability that that would occur.

So that's a simple way of analyzing the data. In statistical parlance, it's a sine test. But it's a valid way to look at the data.

Q. And in this statistical probability test, you actually discuss that in your report, right?

A. Correct.

Q. So I'm going to do a quick probability check with you, okay?

We talked about two-headed coins. One-half, one-half, you multiply them and get one-fourth; is that right?

A. Correct.

Q. What if we had a more rare event. One in 150, okay? And then another event --

MR. ISMAIL: Your Honor, I'm going to object to this line of questioning as not being a disclosed opinion. It can be heard on at sidebar.

THE COURT: Okay.

(Sidebar discussion not reported.)

MR. WISNER: May I proceed, Your Honor?

MR. ISMAIL: I assume the question is withdrawn, Your Honor?

THE COURT: Yes.

MR. WISNER: I don't think I got the question out.

THE COURT: I'm sorry.

Is your objection withdrawn?

MR. ISMAIL: No.

THE COURT: Okay. I am not going to rule on the objection. I'm going to let Mr. Wisner go ahead and finish asking -- complete his question, and then you can then state your objection.

MR. ISMAIL: Thank you, Your Honor.

BY MR. WISNER:

Q. So if we have two independent events, one is 150 and the other is another 150, would the proper way to calculate that probability be to multiple these two?

THE COURT: You can answer that.

THE WITNESS: As long as the events are considered independent of each other, yes, the appropriate approach would be to multiply those two things together.

MR. WISNER: Thank you.

THE COURT: The objection is overruled.

MR. ISMAIL: Yes.

THE COURT: He's permitted to ask that question.

MR. ISMAIL: Yes.

THE COURT: All right.

So if you're transitioning to something else, Mr. Wisner, this might be a good time for our afternoon break.

MR. WISNER: Sounds good.

THE COURT: All right. Fifteen minutes.

(Recess taken at 2:54 p.m.)

(Proceedings resumed at 3:09 p.m.)

(Proceedings held outside the presence of the jury.)

MR. ISMAIL: Your Honor, I was talking with Mr. Wisner. Rather than interrupt the flow when the jury comes out, we want to raise an issue now, outside of the presence of the jury. And it relates to Counsel's binder, Tab 112.

And our objection -- Your Honor, this issue, these data, these charts have never been disclosed by Dr. Portier. It's not been the subject of prior testimony. I understand it's an issue that Mr. Wisner did in his opening and I did in my opening, using this concept of discussing general causation. But this is not the witness to do it through because he's never

disclosed his opinions on this issue at all, never used this dataset in any of his prior testimony or his reports.

So we don't believe it's proper for Dr. Portier to -- for them to go through these demonstratives and ask him any questions about them, frankly.

THE COURT: Okay.

MR. WISNER: The first graph is from their opening. And so I literally took what they've showed the jury, and I'm going to ask them, is this a misleading graph? And he's going to say yes.

Because if you look at it, Your Honor, the NHL rates are scaled from 0 to 100.

THE COURT: He's offering an opinion --

MR. WISNER: About something that they've shown for the first time. None of their experts say any of this. This is all just them, in opening, saying something factually to the jury. And they showed this graph.

He's not going to say that he relies on this data, that he thinks it's credible or anything. He's simply going to say that the way this graph is scaled hides what the data is actually showing.

If you look at the first graph, Your Honor,

the orange is new NHL cases -- this is what they showed the jury -- and it's scaled from 0 to 100, even though it doesn't ever go above 21.

If you turn to the next page, it's the exact same graph, just properly scaled.

THE COURT: Okay.

MR. WISNER: That's all I'm going to show. That's it.

THE COURT: I don't think it's a question of what you're going to show; I think it's a question of what he's going to say and whether or not it's appropriate for him to say that.

MR. WISNER: Sure. I'm going to ask two questions. I'm going to ask: Is this a misleading way to present the data? He will say yes. He's prepared hundreds of graphs over his career that shows that.

And then the second thing is, I'm going to say: Sir, have you looked, is there any published study whatsoever suggesting that this rise in NHL and rise in glyphosate use, from a population perspective, has there been any study to show it's been related? And he will say no. It's never been done because it's not valid science. He hasn't opined about this because it's not science; it's just putting up two lines.

THE COURT: But having a scientist opine about

something that's not science or nothing he's ever disclosed or said before because Defendants are using whatever graph that they've created, it doesn't give him a platform to continue as an expert to opine about it. Not that he's never opined on that topic before, he's just winging it, but now here we have this document.

I don't know who put this together or what it's based on. And I'm not sure that Doctor -- part of my concern isn't so much that he may have an opinion about the underlying data, but does he know all the data -- the information that the defendants presented so that he can actually opine about it?

What is the underlying data? Does he know what it is? If he actually says that, what is he speaking to?

MR. WISNER: So --

THE COURT: You may have to cross-examine Defendants' witnesses and discredit them to say, how did you come up with this? You know, what is this saying? And then once you've got your basis for whatever this represents, have at it.

But just to have him look at something he's not familiar with, he has no idea what the underlying data is or basis is, and simply say -- it's wrong.

MR. WISNER: Fair enough.

Two things. My colleague just pointed out to me that he actually has disclosed this opinion in his report. It's right here. It's in one of the regulatory submissions. It's talking about exposure to glyphosate and it says:

"This is entirely speculative," talking about this very data. "And based upon an ecological assessment, glyphosate use has decreased dramatically over time, and not upon actual data pertaining to the studies at hand. Nor does it fully account for the full time since first exposure of the studies done with earlier" --

THE COURT: Has he disclosed these opinions in this case for --

MR. WISNER: Yes.

THE COURT: This testimony?

MR. WISNER: Yes.

THE COURT: And is it in his report?

MR. WISNER: Yes.

THE COURT: Is this his report?

MR. WISNER: Yes.

MR. ISMAIL: Absolutely not, Your Honor. What Counsel is referring to is a completely different issue than what we're talking about here.

This issue --

THE COURT: Let me just say this: Whatever the contours are of his opinion, he can offer his opinion if it's in his report. Don't use this graph, because I can't mediate that dispute right now to say, does this represent everything in the EPA report? It's 3:15.

So I can't break it down right now and ask you to present me, is the data the same? If it was the same, then it would be okay; if it's not the same, it wouldn't be okay.

Go ahead and he can disclose whatever opinions he has already indicated he would testify about. If that's included in his report, he's free to testify about it.

But opining about something the defendants used in their opening and say it's wrong without any foundational information about what it's based on, what it represents, specifically this, no.

MR. WISNER: Okay. I mean --

THE COURT: He can talk about the subject matter if it's part of his expert opinion that he's disclosed in his report. I don't have a problem with that.

MR. WISNER: All right. We can do this with

another witness. We have more witnesses who can talk about this. I'll just ask generally about ecological data right now.

THE COURT: Whatever is in his report is fine. I don't have a problem with that. I'm not preventing him from fully expressing his stated opinion.

But when you start having him opine about something that there's no foundation for -- I mean, if it was his document or his demonstrative, I wouldn't have a problem with it. But it isn't.

So since there's no foundation laid for his opining about something that somebody else created, and can't in the next ten minutes or next hour, and he hasn't done that, and there hasn't been some dialogue or at least a deposition or something, I can't permit that.

MR. WISNER: Well, here is my concern. And then we can move on from this.

But they are presenting a scientific theory that none of their experts have actually given, okay? They haven't said -- they didn't show that chart. What they showed the jury is a concoction made by Counsel.

And it's based on data. We know where it's from, and that's how we generated this reproduction of it. We didn't actually get a copy of theirs, but we know where it comes from. And we know that it's

fallacious and not properly scientific. Every one of my experts can explain why this kind of analysis is flawed -- every single one of them -- because it's across-the-board wrong.

How can we possibly refute lawyer-created science with our experts if we're not allowed --

THE COURT: We probably should have had this conversation before opening if there was an objective to the demonstratives.

MR. WISNER: We filed a motion in limine about it, and you overruled it.

MR. ISMAIL: You are right. Your Honor denied their motion in limine on this precise issue. We talked about the sourcing of it and what witness through which it would be presented.

THE COURT: Is it going to be presented through a witness?

MR. ISMAIL: In our case?

THE COURT: Yes.

MR. ISMAIL: Yes.

MR. WISNER: Who?

THE COURT: So can't you cross --

MR. WISNER: I didn't know that.

Your Honor, may I ask Counsel which witness is going to be --

MR. ISMAIL: We've already talked about this in the context of the motion in limine.

THE COURT: It would have to be Motion in Limine Number 65. It's fine. Really.

Reorient me to exactly what the motion is, and then I can --

MR. ISMAIL: I wasn't commenting to the Court; I was commenting to Mr. Wisner.

This was through Dr. Levine. She references this data in her report. It was the subject of a motion. The Court denied it.

My objection to Dr. Portier is that he's got neither the dataset nor these graphs.

THE COURT: So the MIL on Dr. Levine is this information; is that right?

MR. ISMAIL: They had a separate MIL on the topic, not just on Dr. Levine. And that was denied.

THE COURT: But if she's going to speak to it, then you can cross-examine her on the underlying data and/or basis for her opinion or the theory that Defendants are advancing.

MR. WISNER: Sure.

THE COURT: And I think that's perfectly fine.

MR. WISNER: Sure. Again, I'm just trying to have my expert explain why that's not scientifically

correct. Because I'm going to ask her, and she's going to say it's great, even though it's not true.

THE COURT: I'm simply saying that this demonstrative is the issue. If he's already opined about that subject matter, he can talk about it. I'm not trying to prevent him from that.

MR. WISNER: No problem.

THE COURT: Okay. We can bring the jury out.

MR. WISNER: Yes.

MR. ISMAIL: Thank you, Your Honor.

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

THE COURT: Mr. Wisner, you can continue.

MR. WISNER: Thank you, Your Honor.

BY MR. WISNER:

Q. Now, Dr. Portier, we've covered the animal studies and mechanism studies. And we briefly touched on epidemiology; again, we'll have another witness get into that later.

But I want to talk to you about another type of data. Are you familiar with something called ecological data?

A. Yes.

Q. What is ecological data?

A. It depends in which context you're talking

about it. In the context of epidemiology, which I think is where you want me to look, ecological data is where you're comparing -- so we saw epidemiology data that was cohort studies, where you're following one person over a long period of time.

Then we saw epidemiology data, where people have to sort of remember what they did in the past.

There's a third type of epidemiology data. Where I have a whole bunch of towns, and I can look at the rates of something in these towns, and I can look at a whole bunch of other towns and the rates of disease in those towns. And you compare the disease rates in the towns to some exposure in the towns. But it's not at the level of the person; it's sort of at the level of a large collection of people, a community of some sort.

Q. What if you look at the entire country?

A. That would clearly be an ecological study across the entire United States.

Q. So why don't we just do that? Why won't we just look at glyphosate use over the last 40 years, look at NHL rates over the last 40 years, and see if they're both going on?

Wouldn't that prove the story for us?

A. No, it would not prove the story.

The classic example of a failure to prove that

story is births in Europe.

Q. Sorry, what?

A. Births of children in Europe.

Q. Okay.

A. If you look at the rate at which children are born per population in Europe, and you compare it -- back in the 1980s and '70s and '60s -- and you compare it to the number of storks in Europe, the bird, they line up very well.

And so it's obvious you would conclude from that, because you're getting less storks -- the birth rate was going down. Because you're getting less storks, you have less babies delivered, because you don't have the storks to deliver the baby. But, of course, that's a nonsense association.

So one has to look carefully at that type of data. In this case of NHL and glyphosate, first and foremost, there are other causes of NHL. So you have to make sure they're not changing.

If you're going to compare this rate and try to attribute it all to glyphosate, you have to make sure that the other things are not going down or going up or whatever. They can affect the pattern, and, of course, the pattern would be wrong. That's the biggest problem.

The other problem is that you might not have a

reason to connect them. In this case, I think you do have a reason to connect them. But there are other things that can happen.

Q. To the best of your knowledge, having reviewed the public literature, and as much of the nonpublic literature that you've seen, has any scientist ever tried to do that -- look at the rate of NHL, look at the rate of glyphosate -- and make a connection?

A. I think yes. I would have to go back and look carefully. I may have it confused with cell phones.

Q. Okay.

A. But if you look at -- what's the name of the -- Hardell. If you look at -- Hardell, I think, did something looking at that question in Sweden. But I can't be absolutely certain.

Q. Okay.

A. I would have to look at that. But other than that, I haven't seen anything.

Q. Now, if you were to look back at that data for the last 40 years, have there been things changing in our society that might affect the NHL rate?

MR. ISMAIL: Objection, Your Honor. For the things we previously discussed. Undisclosed. Outside of expertise.

THE COURT: Overruled.

You can answer.

THE WITNESS: Well, the -- some of the other causes of NHL that I'm aware of would be HIV infection; and, obviously, in the last 40 years, that has changed dramatically in the United States. The other one was -- that's enough, I think.

Oh, Hepatitis B and C virus rates in the United States have tended to go up, and there is an association there, as well.

Q. Okay. So if you're going to do that kind of analysis, is it your opinion that you should adjust and consider all these other possible influences?

A. At least, yes.

Q. Notwithstanding this ecological data, we have looked at studies that specifically look at glyphosate and Roundup, right?

A. Correct.

Q. And we kind of started off your examination this morning -- after your background -- talking about how you got involved in this. And we talked about IARC.

Do you remember that?

A. Yes.

Q. And we kind of started there and said that IARC actually looked at these three pillars, as well, right?

A. Correct.

Q. What did IARC find with regard to the animal studies?

A. IARC has a guidance that tells the working group exactly how to label things based upon the science they see.

So for animal studies, they classified the animal evidence as "sufficient," which means that they believe there is enough evidence there to say that glyphosate caused cancer in the animals in the studies they looked at.

Q. Is there a category higher than "sufficient"?

A. No.

Q. So that's the highest category?

A. That's the highest category of association, yes.

Q. All right. What about -- let's go in the order in which we covered it -- mechanism?

A. Mechanism, they concluded that the evidence supporting genotoxicity was strong and that the evidence supporting oxidative stress was strong, and all other mechanisms were weak or no data.

Q. All right. Is there a higher category than "strong"?

A. No.

Q. Okay. And then the epidemiology.

Did they look at epidemiology?

A. Yes, they did.

Q. What category did they give it?

A. There, they categorized it as "limited evidence of carcinogenicity in humans."

What that means is that there is an apparent association between, in this case, NHL and exposure to Roundup.

It could be causal -- so you're not worrying about the stork and the births issues, which really can't be causal. It could potentially be causal. But the studies have weaknesses, have concerns about them, such that you can't rule out what's called chance, bias, or confounding. I can define those, but it might be better if your epidemiologist spends time on that.

But they can't rule out chance, confounding, and bias. So it falls into that "limited" category.

Q. And since IARC -- "limited," how high is that in the ranking? Is it the lowest? The middle? The highest? Where is it?

A. It's the second highest.

Q. Okay. So above "limited" would be "sufficient"?

A. Correct. Just like in the rodents.

Q. Okay. So IARC concluded, the highest category for animal studies, the highest category for mechanism studies, and the second-highest for epidemiology; is that right?

A. That is correct.

Q. Now, since IARC -- well, when did IARC do its analysis? When did they come to their conclusion?

A. March 2015, I believe.

Q. So that's been four years; is that right?

A. Correct.

Q. Almost to the date.

In the last four years, have there been new epidemiology studies?

A. Yes, there have.

Q. I want to talk about one of those, very briefly. This was that forest summary we showed the jury a minute ago.

And down here at the bottom, it says Zhang 2019. And up here, it says: "Derived from Zhang Table 7."

Do you see that?

A. Yes.

Q. What is the Zhang study?

A. Zhang did a meta-analysis. So Zhang took all of the existing epidemiology and set up rules for which

studies she would include in her meta-analysis, and then she analyzed the analysis of the studies. She pulled them together to look at one picture.

Q. What did they conclude?

A. They concluded that there was clearly an association.

Q. And that's reflected here at the very bottom, that green box?

A. Yes. They have two lines there. One line is including -- so the agricultural health study, which is one of the cohort studies where you're following people over time. Well, you stop after awhile, and you look at your population and see, do any of them have cancer? And what have they been exposed to? And when you stop, you publish a paper.

The first paper was published by De Roos in 2005, and the second was published by Andreotti in 2018. There's some controversy between the two studies, as to which one is good and which one is not, and problems with the Andreotti study.

So what Zhang did was, the first line, they included the Andreotti study in their meta-analysis; and then the second line, they took out the Andreotti study, put the De Roos study in and redid the analysis. And it made no difference, was their basic finding. It didn't

matter. You still got the same positive finding in the meta-analysis.

Q. So this meta analysis, which includes both versions of the AHS study, it's still positive in the meta-analysis?

A. That's correct.

Q. Was this study, the Zhang article, was that available to IARC when it assessed the epidemiology?

A. No.

Q. I want to shift gears a little bit now and talk specifically about the EPA.

Have you, sir, reviewed -- well, let's back up a little bit.

Since IARC, has the EPA issued any opinions or position papers about glyphosate and carcinogenicity?

A. Yes, they have.

Q. And have you had a chance to review that?

A. They've released two position papers. The first one, I sent them formal comments to. The second one, I have not. But I've read both of them.

Q. You sent comments.

What do you mean? What did you do?

A. Well, when the EPA releases a position paper like this, they release it for public comment. And so you're welcome to send them comments, and they will take

your comments and decide whether to include them or not in their overall evaluation.

So I sent public comments to them. They had a public meeting, where they brought in scientists to review their draft. And my comments went to that meeting, as well.

The current version is still a draft; they have not finalized it. It's just a second draft.

Q. Did it take some time preparing those comments to send to the EPA?

A. Oh, yes. It took a tremendous amount of time. It's a very detailed document. And my comments were to paragraphs and to certain lines and very specific comments about what they did, where they did it and how they did it.

Q. Were you being paid by Counsel or anybody to prepare those comments?

A. No.

Q. So you were doing it on your own time?

A. Yes.

Q. Why would you do that? Why would you spend so much time doing that?

A. Because my entire career has been linked around how to evaluate scientific literature to come to conclusion about cancer.

I've been involved in developing guidelines for many different countries, many different places. I want to see these types of things done right.

And here, I was looking at a document where, in my opinion, they were violating many of the guidances that were put in place to make sure they don't violate these guidances and don't go off and do things different every time they do a different chemical.

And so most of my comments were pointing out, you're violating this guidelines, you're violating that guideline, you're not supposed to do it this way, you forgot to do this. There's a number of problems in there.

And I felt it was important enough that I speak out. Because many times, scientists are too busy to actually get involved in this. Whereas I was semi-retired; I had the time, I had the interest, I had the knowledge because I had read the literature. I decided I needed to do something about this.

Q. And when you prepared those comments and you submitted them to the EPA, did any lawyers review them before you submitted them?

A. Oh, no.

Q. They're just yours?

A. They're just my comments.

Q. All right. Let's turn to the EPA report. Turn to Exhibit 3036 in your binders.

Is that a copy of the most recent interim analysis by the EPA?

A. 3063?

Q. No, 3036.

A. Mine is 3063, but the document says 3036. The tab is wrong.

MR. WISNER: Sorry, there must have been an inversion of the 36 and the 63.

THE COURT: Yes. It says 3036, and it's under Tab 3063. So I think we're all on the same page.

MR. WISNER: Apologize for the mistake. Late night.

THE COURT: No problem.

BY MR. WISNER:

Q. Is this a fair and accurate copy of the issue paper that is the most recent version of what the EPA is saying?

A. It looks like it, yes.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection, Your Honor.

THE COURT: All right. Permission granted.

MR. WISNER: Thank you, Your Honor.

BY MR. WISNER:

Q. All right, Doctor. You know, we spent a lot of time today talking about animal studies, so I want to focus on the animal study analysis that the EPA did.

And to do that, actually, I want to put up our mouse board to sort of help us -- is this the mice? No, this is the rat -- help guide our discussion.

Can you see?

A. Yes.

Q. Okay. I think I wiped off a plus mark there, but we'll fix that later.

So we're in the EPA document here, and I want to first turn to the section discussing the mice studies.

Let's start with -- well, let's actually start at the beginning. Let's start on page 12 of this document.

And here, we have a little background about why this document is being done. It says right here:

"Currently, glyphosate is undergoing registration review, a program where all registered pesticides are reviewed at least every 15 years, as mandated by the Federal Insecticide, Fungicide, and Rodenticide Act, FIFRA. The initial docket opening for

glyphosate occurred in 2009, with the publication of the human health scoping document and preliminary work plan."

Do you see that?

A. Yes, I do.

Q. What are you referring to? What is this 15-year re-registration issue?

A. You don't get a free run when you get a pesticide approved in the United States. After a period of time -- and it can be less than 15 years -- but at the least, 15 years, by law, it has to be reapproved by EPA.

And then by regulatory edict, EPA requires that a new review of all the literature be done when they do a re-registration.

Q. All right. And this document, who was it prepared for?

A. That's a good question. I guess it's prepared for the assistant administrator for toxics at EPA, because that would be the authority that would agree to re-registration or not.

Q. Okay. Turn to page 19.

There's a section here that says "Organization of the Document."

Do you see that?

A. Yes, I do.

Q. And it says:

"Although there are studies available on glyphosate-based pesticide formulations, the agency is soliciting advice from the FIFRA SAP on this evaluation of human carcinogenic potential for the active ingredient glyphosate only at this time."

Do you see that?

A. Yes, I do.

Q. First, was it your understanding that this document was to focus on glyphosate, and not glyphosate-based formulations?

A. That's right. The EPA interprets FIFRA as requiring them to have the main ingredient of a pesticide tested, but not all the other stuff. So that's why they're asking only about this.

Q. And it says "the FIFRA SAP" right there.

Do you see that?

A. Yes.

Q. What is the FIFRA SAP?

A. SAP stands for Science Advisory Panel. It consists of seven permanent members, and then they augment the permanent members with other scientists to help them when they're looking at each new topic.

So the permanent members sit for four or five years on the SAP. And they might look at this chemical this week, another chemical the next week. They might look at the design of cancer studies the week after that, to see if it should be altered in their guidance, things like that.

And then they get extra help from other people when they do that. I sat on the Science Advisory Panel for five years.

Q. That was my next question: Are the people who are brought in to participate in the SAP, are they independent scientists?

A. Yes. They are independent of EPA, independent of their institutions. They are there as scientists providing their opinion, as scientists, to the EPA.

Q. Let's move on to the mouse data.

Before I do that, actually, we talked about this briefly earlier. Turn to page 145 of this document, Section 7. It's on the screen, too, if you want to look at it there. But if you prefer paper, no problem.

There's a section that says "Collaborative Research Plan for Glyphosate and Glyphosate Formulations."

Do you see that?

A. Yes, I do.

Q. It says right here:

"As previously mentioned, some have believed that glyphosate formulations may be more toxic than glyphosate alone. Glyphosate has been studied in a multitude of studies, and there are studies that have been conducted on numerous formulations that contain glyphosate. However, there are relatively few research projects that have attempted to directly compare glyphosate and the formulations in the same experimental design."

Do you see that, sir?

A. I see it.

Q. So when we talk about experimental design, like here, we have all these long-term studies on glyphosate; there hasn't been a similar long-term study on Roundup?

A. That is correct.

Q. And what is your understanding of what the EPA's -- strike that.

Is it your understanding that the EPA is suggesting here that it needs to be studied?

A. I think they're leading to discuss the fact that they are partnering with the NTP to do some

studies. I don't know if that says that we need to do them.

Clearly, the NTP thinks they need to do it because the NTP will be paying for it. If I understand how NTP and others work -- and I'm pretty sure I do -- they will be paying for it. But that's, I think, what they're leading to.

Q. You understand how they work because you essentially used to run the rodent programs?

A. I ran the whole NTP for six years.

Q. All right. Let's go to the rodent studies. That's what we spent a lot of time with today. That starts on page 85.

Starting on page 85, it's the EPA's assessment of the mouse studies.

Do you see that?

A. Yes, I do.

Q. It's mice carcinogenicity studies with glyphosate.

Do you see that?

A. Yes.

Q. Now, I notice that the first study here is Reyna and Gordon, 1973.

Do you see that?

A. Yes.

Q. That's not on your chart here?

A. No.

Q. All right. Well, if you go to page 156 of the document, there's actually a -- it's actually discussion of a study. You see it says: "Reyna and Gordon, 1973, 18-month carcinogenic study in Swiss white mice, IBT number 8569."

Do you see that?

A. Correct.

Q. It says: "Prepared by Industrial Biotest Laboratories."

Do you see that?

A. Yes.

Q. Are you familiar with Industrial Biotest Laboratories?

A. Yes, I am.

Q. Are you familiar with what occurred in the 1970s related to IBT?

A. Yes, I am.

Q. What happened?

A. They -- they -- when you do studies as a contract lab for anybody, you have to keep records of all kinds of things.

There was an audit of the laboratory. They went in, and the records did not match the reports.

There was litigation. It was criminal fraud. They were making up some of the reports they were sending in.

EPA, FDA all sent in teams to look at what was going on there. And found 75 percent of their studies that they had recently done as being unreliable and unacceptable.

Q. Was that scientific fraud?

A. Some of it was. People went to jail for it.

Q. Specifically, this study right here -- Reyna and Gordon, 1973 -- was that study deemed invalid by the EPA back in the 1980s?

A. Yes, it was. That's why I have not included it.

Q. Well, let's look at what the EPA did. Back on page 85, there's a discussion here of Reyna and Gordon from 1973.

Do you see that?

A. Correct.

Q. And it discusses the results. It says:

"There were no treatment-related increases in tumor incidences observed in the study."

Do you see that?

A. Yes, I do.

Q. Does the EPA ever disclose, well, this study was actually invalid?

A. No, they do not.

Q. In your work in government for 35 years, would you prepare a report for the public talking about the safety of a product relying on a study that you knew was invalid?

A. I might mention the study, but then I would clearly end by saying, this study is invalid and not used in our overall risk assessment.

Q. And if we actually go a couple of pages in, there's a chart here, Table 4.20, on page 91. And it mentions the study.

Do you see this?

A. Yes.

Q. And it gives the result on the right and says:

"There were no tumors identified for evaluation."

Do you see that?

A. Correct, yes.

Q. And just putting things in perspective, we have looked at other mice studies, and there's tumors in all of them.

Is that consistent with that study having, in fact, been invalid?

A. No, it's not.

Q. Is it consistent with that study having been

invalid?

A. I don't understand the question.

Q. Bad question. I'll move on.

So let's go back to the analysis. And let's talk about Knezevich & Hogan.

Do you see that?

A. Yes.

Q. Now, on your chart here, you've identified two different tumors. You identified this older form of lymphoma and these kidney tumors.

Do you see that?

A. Yes.

Q. Does the EPA identify the lymphoma?

A. No, they do not.

Q. Do they -- okay.

Do they discuss the kidney tumors?

A. Yes, they do.

Q. So if we go back to that chart we were looking at, it's a nice little summary, if you walk through it here. Let me call out its reasoning.

It says:

"No statistical significance in trend or pairwise comparisons, including the mid and high doses which approached or exceeded 1,000 milligrams/kilograms per day. Incidents of

adenomas within historical range for performing laboratory."

Do you see that?

A. Yes.

Q. Let's start off with "no statistical significance in trend or pairwise comparisons."

What is that referring to?

A. That's referring to the fact that they do not see a p-value for the trend test of less than 0.05. The actual value for that trend test was 0.064 in that situation, if I'm remembering correctly. It's very close to that number anyway.

So they break the studies into: Yes, it was positive; no, it was not, at that 5 percent point.

Pairwise comparisons means it's the same thing, none of the individual dose groups was significantly different than the controls.

Q. Okay. Having helped write the standards for doing these types of studies, is that an appropriate approach to assessing tumors in a mouse study?

A. It's a typical approach. It's an inappropriate approach, but it is a typical approach.

There's recently been a publication in Science -- I think it was Science -- that was signed by 800 statisticians saying, stop using statistical

significance; start looking at the values of your trends or whatever test you're looking at and the p-values and interpret the data. Don't try to make it yes or no.

And so my opinion, this type of approach is a yes or no approach, and it's too simplistic for the data we're looking at.

Especially when you see a p-value in this study of .064, and I have another study of kidney tumors with a p-value of .04; they're both going in the same direction. So when I combine them, I get a strong picture of the fact that I'm really seeing kidney tumors being formed in these studies, it just didn't meet this 5 percent standard in one study, and it did in the other. But the trend is clear in both of them.

Q. I see here that you have three pluses.

Do you see that?

A. Correct.

Q. You have three pluses.

What do those three pluses refer to?

A. That refers to the last sentence they have up there. They partially dismiss this finding because the tumor rates that they see are in the range of the historical controls.

Now, I mentioned controls this morning. When I do multiple studies in the same lab over and over

again, I always have a control group. I keep those control animals all the time, and then I learn something about what the pattern looks like in unexposed animals. Sometimes you need that information, especially in the case of rare tumors. This is a rare tumor.

And the definition of a rare tumor is that it occurs at a rate of less than 1 percent in your historical controls. That's the standard that EPA uses, that's the standard that NPT uses, that's the standard that's common to call it a rare tumor.

In that case, you go to historical controls and you look to see if it makes sense that this is biologically important because of the controls.

But what the guidelines say very clearly is, don't use historical control range as a way of excluding results. Instead, you use a proper statistical test to look for the impacts with the historical control information also in there.

That is what I did in looking at the three pluses you see there. And when I include the historical control dataset that is available for this particular study, I get a p-value of less than 1 in 100.

Q. And just to -- I'm turning to the EPA guidelines. We've looked at them a couple times today. It's Exhibit 140.

And here on page 48 of the document, this is the EPA guidelines. It says:

"Generally speaking, statistically significant increases in tumors should not be discounted simply because their incident rates in the treated groups are within the range of historical controls."

Is that what you're referring to?

A. Yes.

Q. Why? Why is it a problem to see a tumor and say, you know what, it's within the range, it's cool?

A. It's like the flipping of the coin. Let's go back to the flipping of the coin example.

Even if I have a perfectly fair coin, there's a chance that I -- if I flip it enough, I'm going to get seven heads. There's a chance that if I flip it seven times, I get seven heads.

If I do that experiment a million times -- so I flip it seven times, record it, flip it seven times, record it, do that a million times -- the chances that I get seven heads is 100 percent. I'm guaranteed to get it.

So the problem with the range of historical controls is that the number of historical controls you have changes the range.

So if I only have five historical control groups -- and that might be 0 out of 50, 0 out of 50, 1 out of 50, 1 out of 50, 0 out of 50 -- then my range is 0 to 2 percent; 0 out of 50 to 1 out of 50.

If I have ten groups, I might pick up a 2 out of 50 just by random chance. So now my range is 0 to 4 percent.

If I have 100 such groups, I might pick up a 5 or 6 out of 50 by random chance, so now my range is 0 to 12 percent.

But in that 100 studies, I might have 95 that are 0 out of 50, two that are 1 out of 50, two that are 2 out of 50, and one that is 6 out of 50.

So the range doesn't actually tell me how rare the tumor is. The actual entire set of numbers tells me how rare the tumor is, and you have to look at all of them.

Q. Who are you to tell us or tell the EPA how they're supposed to do it?

A. In terms of analyzing data from animal cancer studies?

Q. Yeah.

A. I've published all over on that question. It's my forte. It's what I've done. In the first ten years of my entire career, that is what I did.

Q. These are the EPA guidelines right here, right?

A. Correct.

Q. How are you qualified to say whether or not they are following those guidelines?

A. First, I helped them write it. I reviewed it. This part about historical controls was driven by a paper by Joe Haseman at the National Toxicology Program, who worked for me at the time that this was being done. And I fully agree with what he wrote about historical controls. There's a long history to use of historical controls and the proper way to do it.

Q. Let's go back to the chart that we were going through. And I just want to be clear: I'm going to put onto your chart here -- I'll write in red so we know the red is mine, okay?

"EPA missed." And we established they missed this one, right?

A. Correct.

Q. So let's go to the next study, Atkinson study.

Did the Atkinson study observe all of these lymphomas and hemangiosarcomas?

A. Yes, it had both of them.

Q. So they caught both of them?

A. EPA?

Q. Yes.

A. No. EPA did not consider the lymphomas.

Q. So they missed the lymphoma again?

A. Yeah.

Q. Did they discuss the hemangiosarcomas?

A. Yes, they did.

Q. Let's look at what they said about them:

"Statistically significant trend for hemangiosarcomas that were only observed in 4 out of 45 high-dose male mice. Increased incidence was not statistically significant from the concurrent controls at all doses, including the highest dose tested, which is approximately 1,000 milligrams/kilograms per day."

Correct.

Q. How is the EPA dismissing that result there?

A. It's there. It's in science jargon, but they're giving it to you right there.

They're saying, yes, we saw a trend. But we didn't see a pairwise comparison that was positive, so we're going to disregard the trend.

Whereas the guidelines say, if you see a trend or pairwise positives, you should treat it as an observed effect to be worried about. So that's the

first part of their tossing it out.

The second part is the high dose. They're arguing that the dose was too high. But the guidelines have a clear definition of what constitutes too high of a dose. And that definition does not include 1,000 milligrams per kilogram per day as a fixed number; it has to do with the amount of the chemical that is in the feed of the animal. It can't exceed a certain point, or it can't exceed the maximum tolerated dose, which is defined by doing the 90-day study.

Q. Okay.

A. All in their guidelines.

Q. So going back to the guidelines. Now we're at page 46 of the guidelines.

And you said:

"By convention, for both tests, a statistically significant comparison is one for which p is less than .05, that the increased incidence is due to chance."

And then it says:

"Significance in either kind of test is sufficient to reject the hypothesis that chance accounts for the result."

Do you see that?

A. Yes.

Q. What does that mean, and how does that relate to what you just said?

A. That's exactly what they have now violated. By saying we're going to disregard the trend because we don't see the pairwise comparisons, they've violated that guidance in their own guidelines.

Q. And these are the guidelines you helped write?

A. Correct.

Q. Okay. Let's go back to the EPA document.

Let's move on to Wood. On this chart, the next one is Wood.

A. Okay.

Q. But we have Sugimoto. Well, Sugimoto is on the next page. We'll go in sequence.

Here, we have Sugimoto. Let's go through these. Which ones of these tumors in Sugimoto did the epidemiologist miss?

A. It's easier to go the other way. The only one they saw was the hemangiomas in the females. That's the darker green.

Q. Okay. So everything else, they missed?

A. Correct.

Q. So they missed the lymphomas, kidneys, hemangiosarcomas, multiple malignant tumors, and gland adenomas.

Is that right?

A. To be fair, in their first draft, they had the multiple malignant tumors in there; in the second one, it was removed.

Q. Okay. Well --

A. So they knew about it. They purposely did not put it in the second draft.

Q. Okay. So -- all right.

So what did they say about the hemangioma? I think you have that up here. It says:

"Statistically significant trend for hemangiomas in female mice following adjustment for multiple comparisons with incidents of 0/48 in controls, 0/47 at the low dose, 2/45 at the mid dose and 5/45 at the high dose. Increased incidence at high dose statistically significant following adjustment for multiple comparisons. Highest dose tested was more than four times the limit dose."

So how did they get rid of that one?

A. It's the last point in that paragraph, that the highest dose tested was more than four times what they're calling the limit dose. But they're misinterpreting the limit dose.

The limit dose is in some of their guidelines, and the limit dose says that you don't need to exceed

1,000 milligrams per kilogram per day in an animal cancer study. It doesn't say you must not exceed; it says you don't need to.

So a company that is comfortable that the exposure in humans is going to be so much less than that doesn't need to go higher than 1,000 milligrams per kilogram per day. But that's before you do the study.

Once you do the study, then the only way to get rid of an exposure and say it's not -- it shouldn't be counted, is in the guidelines, and that is if it exceeded the maximum tolerated dose. And there are definitions for what it means to exceed the maximum tolerated dose.

So they've inappropriately thrown out a dose group. And by doing that, they say, there's nothing there.

Q. All right. Let's go to Wood. We actually talked about Wood earlier today when we did our rodent tutorial.

Do you recall that, Dr. Portier?

A. Yes.

Q. The ones with the line.

So let's see what they said here.

So again, did they observe all of these -- oh, let's talk about Takahashi.

Do they mention Takahashi at all in the EPA report?

A. No, they didn't. Which was surprising once I realized what Takahashi was. Because the Takahashi report is mentioned in the report by the Joint Meeting on Pesticide Residues of the World Health Organization. They're very clear in mentioning it.

And EPA's report was written well after JMPR -- they were there for the JMPR meeting. So it should have been in there, but it's not.

Q. Okay. So they missed another lymphoma and kidney finding?

A. That's correct.

Q. All right. So now we're in Wood.

Did the EPA observe all these three tumors that you observed?

A. They did discuss them in the first draft, but in the second draft they again dropped the multiple malignant tumors neoplasms discussion and only talked about the lung adenosarcomas and the malignant lymphomas.

Q. Okay. All right. The malignant lymphomas. That's the one we showed the jury in that PowerPoint, remember?

A. Correct.

Q. So how did -- and from what I understand here, there's a positive trend analysis?

A. Correct.

Q. There's a positive dose analysis?

A. Correct.

Q. So it's got both?

A. Yes, it does.

Q. So what did the EPA do here?

It says:

"Statistically significant trend for malignant lymphoma with incidences of 0/44 in controls, 1/46 at the low dose, 2/48 at the mid dose, and 5/45 at the high dose. No statistically significant pairwise results following adjustment for multiple comparisons, including the highest dose tested, which was approaching 1,000 milligrams per kilograms per day. All observed incidences within historical control range for performing laboratory."

So let me break that down. Was this one less than a thousand?

A. Yes, it was less than a thousand.

Q. So it doesn't even have the high dose issue, it doesn't have the statistical significance issue. It looks like the last thing it says is they were within range of historical controls.

A. That's correct.

Q. Is that even correct?

A. We've been through that. That is an incorrect way of looking at this study. If we go back to the guidelines, you'll see that there's something else in there. And it says, "The concurrent control group is always the best group to compare to."

The only time you should use this -- it doesn't say this, but this is going to be my interpretation. The only time you use historical controls is when you have one of two problems.

The first problem is a rare tumor. You don't know what to do with it. You see two weird tumors, you don't know what to do with it; you have to go look at the historical guidance to get you some guidance.

The second place you have it is when you see a very specific pattern of dose response. I have the control response, and it's down low, and then all of the treated groups are up here, and they're about the same.

So the problem there is that by random chance, we might have gotten a control group that just dropped. We were unlucky. It's a very low control.

And so you go to the historical controls to make sure that's not the case and to guide you in deciding, well, I see a positive result, but I'm going

to throw it away because of that low dose.

But if you see a result that is a clear dose response, highly statistically significant, you don't white it out by looking at the range of the historical controls. That violates the guidelines and good science practice.

Q. So looking at the guidelines, is this the sentence you're talking about:

"The standard for determining statistical significance of tumor incidence comes from a comparison of tumors in dosed animals with those in concurrent control animals"?

A. Correct. There's another sentence, I believe further down.

Well, that's good enough.

Q. Okay.

A. But there's something even more blatant in the guidelines.

Q. You probably know them better than me since you helped write them.

Let's focus on this for a second, though, this malignant lymphoma. If you go back to the actual results reported by the EPA, they say that -- here, they say the numbers.

They say the highest dose was 4/45.

Do you see that?

A. 5/45.

Q. Sorry, 5/45.

What is that percentage amount, ballpark?

A. 11 percent, 12 percent.

Q. Okay. And have you actually went and looked at the historic rate of CD1 mice and how often they get lymphoma in 18-month studies?

A. Yes, I did.

Q. What's the rate, on average?

A. That's in my notes. I would have to go look in my notes. It's fairly low.

Q. About 2 percent?

A. About 2 percent. It's not a rare tumor. It's not 1 percent. But it's fairly low, about 2 percent. If I remember correctly, in the dataset that I had for historical controls, I had 26 historical control groups of 18 months.

And I believe about half of them were zero, and the rest were 1s and 2s. It's a low rate.

Q. So let me get this straight. You have a highly significant trend. You have a significant dose. It's less than a thousand milligrams. And it's, what, like 4 or 5 times the average historical control rate.

Do you think it's appropriate to disregard

this lymphoma finding?

A. No, not at all.

Q. All right. How did they go about dealing with the lung?

A. So for the lung tumors, these are adenosarcomas. And you would -- in the progression -- pathological progression of disease, you have lung inflammation, lung adenomas, lung adenosarcomas, okay.

And so they were looking at the rates for lung adenomas and looking for the rates for lung adenosarcomas, and they wanted to see increases in both of those in order to say that the adenosarcomas were real. And so, because they didn't see that, they said there were no premalignant lesions, and so they disregarded this finding because there were no premalignant lesions.

Q. Is that appropriate, in your opinion?

A. It can be appropriate. But one has to, again, look at that fact that you're not actually seeing the progression. So you have to think this through carefully.

Because you can have a chemical that takes premalignant lesions and turns them into malignant lesions. And if you do that for all the premalignant lesions, then you have only the adenosarcomas, and you

don't have any premalignant lesions because you converted them all into carcinomas.

And so you can have some that it's 50/50, in which case you would see both increase. You could have a case where it's only at the highest exposure that you can make that turn, so then you would have some weird pattern showing up.

The thing is you really have to think through that, and they did not here. They just simply said they don't see it, that's it.

Q. All right. So going through this chart, we have the IBT study at the top. We have three of these studies that you mentioned. There is this other study, this Pavkov and Turnier study.

Do you see that?

A. Yes.

Q. Did you review that?

A. I did.

And I found that interesting that they would include that study. It was -- which study is it again?

Q. Sorry.

A. Okay. That's -- that's not pure glyphosate.

So they start by saying, we want to look at pure glyphosate, and then they don't look at pure glyphosate in this particular study.

Now, it's not a formulation; it's something else. I don't know what it is because Pavkov and Turnier didn't explain exactly what they were looking at.

Q. So this study is looking --

A. At something.

Q. Yeah. Not specifically glyphosate and not a formulated product?

A. Not one they told us about.

Q. Okay.

A. It's poorly reported. It's from the literature, so it is one of the publicly available studies, but it's extremely poorly reported.

It's not clear that they did histopathology on any of the tumors that they looked at. I just don't think it's a worthwhile study to include.

Q. All right. I don't see on here the Kumar study.

A. That's correct.

Q. Do you know why they didn't consider the Kumar study?

A. The EPA rejected the entire Kumar study because there was speculation that the Kumar study had a murine leukemia virus in the colony.

What does that mean? There's a virus that

mice can get in laboratories, and it causes them to get blood tumors.

And it can mess up your study if, for example, your high exposed group has the disease, and your low exposure groups don't. So you think you're getting the increase in risk from the disease, but you're actually getting it from the virus. So laboratories routinely check their animals for virus.

Q. Now, if you go to page 70 in the document, it talks about the Kumar study. It says:

A carcinogenicity study in Swiss albino mice, Kumar 2001. This study was not included due to the presence of a viral infection within the colony, which confounded the interpretation of the study findings."

Do you see that?

A. I do see that, yes.

Q. And if you actually look at the citation there, it says 14, there actually is a citation there, and that's to the Greim article.

Do you see that from 2015?

A. Yes, I do.

Q. Let's take a look at the Greim article. It's actually not in your binder. I have a hard copy right here.

MR. WISNER: Permission to approach, Your Honor.

THE COURT: Yes.

BY MR. WISNER:

Q. I've handed you Exhibit 1246. Is this a copy of the Greim article?

A. Yes, it is.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection.

THE COURT: Permission granted.

MR. WISNER: All right.

BY MR. WISNER:

Q. So if you look here, this is the article that the EPA is referring to. And you can see here it's written by Helmut Greim, David Saltmiras, Volker Mostert and Christian Strupp.

Do you see that?

A. Yes.

Q. If you look down here, Dr. Saltmiras is actually an employee of Monsanto.

Do you see that?

A. Yes, I see it.

Q. I'll call it out so everyone can see it.

So this article was actually, in part, written

by them, right?

A. By one of their employees.

Q. Sorry, fair enough. I'm not suggesting that they wrote the article. I'm sorry. I meant by Monsanto.

So if we go to -- I believe it's page 17, let's go to it.

If you see right here, referring to study 13, the Feinchemie and Schwebda, 2001.

Do you see that?

A. Correct.

Q. If you look at what the epidemiologist said, they specifically refer to that same study, right?

A. Yes.

Q. So if you go back to --

A. That's the name of the company that paid for the study as compared to the guy who reported it.

Q. If we go back to what they report, they start talking about the 18-month study in Swiss albino mice.

And if we turn to the next page, it states:

"This study was rated Klimisch 2 for reliability."

I'll stop right there. What is Klimisch 2?

A. Klimisch is a scoring system used by the European regulators more than anybody else to score

individual studies as to their overall quality.

Klimisch 1 is a perfect study, very high quality. A Klimisch 2 has blemishes.

Q. So it's not even like it's a bad study, just blemishes?

A. Yeah. It's a little less reliable because it's Klimisch 2.

Q. It says here:

"This study was rated Klimisch 2 for reliability based on speculation of a viral infection within the colony discussed below."

Do you see that?

A. Yes.

Q. Is that why you said "speculation" earlier?

A. Yes.

Q. Now, have you looked at the mortality rates for the mice in this study?

A. Yes, they were normal.

Q. What about their weight?

A. There was no difference across the entire study in weight in these animals.

Q. Was there any indication in the study whatsoever that these mice actually had a viral infection?

A. None whatsoever in my opinion. But I didn't

get to see the whole study. But EFSA did, the European Food Safety Agency.

Q. And did they comment on whether or not there was a viral infection?

A. Yes, they did.

Q. All right. Let's turn your attention -- it's in your binder at this point. It would be Exhibit 2115.

A. Okay.

Q. Are you there, sir?

A. Yes, I am.

Q. And is this that document that was produced by EFSA or ECHA related to glyphosate?

A. This was produced by the European Chemical Agency for glyphosate, and they also commented on this issue.

MR. WISNER: Permission to publish, Your Honor.

MR. ISMAIL: No objection, Your Honor.

THE COURT: Granted.

BY MR. WISNER:

Q. So we're looking here at this document. It's this report. And as you can see, it's about glyphosate, and it's by the -- who was it by, Doctor?

A. The draft is done by the Federal Institute for Occupational Safety and Health of the Federal Republic

of Germany.

It's intended to be read by the Risk Assessment Committee of the European Chemical Agency. This is the science as it's being presented by the German government.

Q. Fair to say that they're a rapporteur?

A. Correct.

Q. And is it fair to say that the BFR or this agency in Germany, they prepare the hard science, and then the EU either approves it or doesn't?

A. They comment on it and make a decision based upon the science. They don't really approve or disapprove of the science review.

In this case, ECHA didn't alter the document. They simply took the document, read it and made a decision.

Q. If you look down here, it discusses -- it says:

"During a teleconference on the carcinogenicity of glyphosate held by EFSA, it was mentioned by a U.S. EPA observer that the Kumar 2001 study had been excluded from the U.S. EPA evaluation due to the occurrence of viral infection that could influence survival as well as tumor incidences, especially those of lymphomas. However, in the study report

itself, there was no evidence of health deterioration due to suspected viral infection, and thus the actual basis of EPA's decision is not known."

Do you see that?

A. Yes.

Q. Has the EPA ever actually explained where they got this from?

A. No. Not to my satisfaction, other than that one reference to Greim, who doesn't explain where he got it from.

Q. Okay. And then do you know who that EPA observer was there in that conference call?

A. Yes. I read some of the memos that dealt with that. And the EPA observer to that conference call was a guy named Rowland.

Q. Jess Rowland?

A. Yes.

Q. And I just want to draw your attention -- well, we're almost getting close to the end of the day, so I'll move on.

So if we go back to this chart here. Because of that viral infection that we have -- well, because of this viral infection, the EPA didn't consider lymphomas, kidney tumors or hemangiomas; is that right?

A. Correct.

Q. So take a step back, Doctor. And it's hard to do it because you have listed all the tumors here. But if you take a step back, and you just remove all the tumors that have red Xs on them; so, essentially, all the lymphomas except for Wood, these two would remain. There would be very few tumors left.

If you were to do that, would this tell you a different story about whether or not glyphosate is inducing tumors in animals?

A. Absolutely. I would see only one, two, three, four, five tumors on there. And that's all I would see. So I have studies with no tumors whatsoever. I have no matching tumors across studies; it's only this tumor in this study, this tumor in that study. So there's no continuity across the dataset. It would give me a completely different picture.

Q. But when you look at the whole tumor set, and you see lymphoma in every single study, what kind of picture does that paint?

A. A very, very different picture. It's a picture that says this is consistent. It's happening over and over again. It's a very solid finding, you should pay attention to it.

Q. So going back to this EPA report, the one that

we've been talking about for about 45 minutes here.

If you're a teacher, and you're going to give them a grade for how they did on this, what grade do you give them?

A. An F. I would fail them.

Q. All right.

Now, this paper was written by the scientific advisory panel, right?

A. It was reviewed by them. Again, I think it's written for internal decision-making at EPA, in essence. But the science advisory panel did look at it.

Q. Okay. And you've reviewed the scientific advisory panel's opinions of what the EPA did?

A. Yes, I did.

Q. All right. Turn to Exhibit 1083. We're coming close to the end, Your Honor.

THE COURT: I'm sorry, 10?

MR. WISNER: Exhibit 1083.

THE COURT: 1083.

THE WITNESS: Okay.

BY MR. WISNER:

Q. Is that a copy of the scientific advisory report?

A. Yes, it is.

MR. WISNER: Permission to publish,

Your Honor.

MR. ISMAIL: Your Honor ruled on portions of this document. I don't know which ones Counsel is planning on referring to.

THE COURT: If this is the motion that I considered in connection with your request --

MR. ISMAIL: Yes, Your Honor.

THE COURT: I approved the executive summary for the most part. And I may have looked at couple of other pages, but the entire document shouldn't be published.

MR. WISNER: Fair enough.

THE COURT: Just stick to that, and then we can discuss it further later.

MR. WISNER: I think we actually filed a motion for this one.

It doesn't make a difference, all right.

BY MR. WISNER:

Q. So, Doctor, I'm looking at this document right here. It's dated March 16, 2017.

Do you see that?

A. Yes, I do.

Q. And it says:

"Transmission of meeting minutes and final report of the December 13-16, 2016 FIFRA SAP

meeting."

Do you see that?

A. Yes, I do.

Q. I just want to draw your attention to page 18.

Sorry. I'm on the wrong page. Page 18 of the document; page 20 of the bottom. That's what I was getting mixed up on.

Just to give the jury a quick understanding, if you see right here, they say, "Overall, the panel concluded."

Do you see that?

A. Yes, I do.

Q. And then elsewhere, it, "Says some panel members."

Do you see that?

A. Yes, I do.

Q. What does it mean when they say "Overall, the panel concluded" versus "some panel members"?

A. If you say, "Overall the panel concluded," if it's the same way when I was there, that's a consensus, pretty much everybody said yeah. They don't take votes.

So as chairman, you're saying, does everybody -- does anybody disagree, and nobody raises their hand, so you move on.

Q. So it says right here:

"Overall, the panel concluded that the EPA evaluation does not appear to follow the EPA 2005 cancer guidelines in several ways, notably for use of historical control data and statistical testing requirements."

Did I read that right?

A. You did.

Q. Is that what you've been talking about here, in part, the way the EPA was not following the cancer guidelines?

A. Yes.

Q. And if you just turn to the front of this, you'll see who participated on this scientific advisory panel.

And we see right here -- I'll find it -- on the last page of the authors list, you have someone by the name of Dr. Zhang.

Do you see that?

A. Yes, I do.

Q. Is this the same Dr. Zhang that published the meta analysis showing a compelling link between Roundup exposure and NHL?

A. Yes, it is.

Q. And do you agree with Dr. Zhang?

A. In her analysis?

Q. Yeah.

A. Yeah. Except I have concerns about using the data from the Andreotti paper.

But other than that, yes, I agree that they did a good job. It's a very nice paper.

Q. And, in fact, two of the other authors on that paper were also members of the scientific advisory panel?

A. That is my understanding.

MR. WISNER: Thank you, Your Honor.

At this time, this would be a great time to end for the day.

THE COURT: All right, thank you.

All right, ladies and gentlemen, we are going to break for the day. Please remember not to discuss anything that you have heard today in the courtroom with each other, with your families, with anyone. Do not communicate about it.

It's important now that we're hearing evidence to wait until all the evidence from both the plaintiffs and defendants have been heard before you start discussing and reaching any decision about any issues that are presented here.

So have a good evening. I will see you here tomorrow. We will start promptly at 9:00 a.m. Thank

you.

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

THE COURT: I'm not sure I mentioned specifically [redacted] name this morning as the juror we excused, but I wanted to establish for the record that she will no longer be participating in the case due to illness.

The other thing is I have to leave at 3:30 promptly tomorrow so I will try to create a little extra time.

I would like to start right at 9:00, but I do have to walk out promptly at 3:30. So I will not have any opportunity to chat with you.

I don't know if there's anything we need to wrap up, put on the record before we finish up. I don't know if this is a good time or bad time to talk about videos or when you intend to use them.

How much more time do you have with Dr. Portier?

MR. WISNER: About 15 minutes.

MR. ISMAIL: Your Honor, I do have a couple of items to mention.

First of all, Your Honor ruled on a motion in limine regarding IBT, that there would be no reference

to criminal indictments or jail.

THE COURT: It was that we don't tie them to whoever the employee was who was working for Monsanto at the time but then later on became -- went back to Monsanto. I've not heard any inferences that it was on behalf of Monsanto.

MR. ISMAIL: Fair enough. That was the one that -- the motion in limine intended wasn't completed, and I was perhaps inferring from --

THE COURT: Yeah. I remember I said it orally. I haven't amended the order yet, but that's the sum total of the --

MR. ISMAIL: Fair enough, Your Honor.

Then with respect to the discussion we had this morning about the Takahashi paper. You know, the representation was made by Counsel that this was vetted during the witness' testimony in Hardeman.

And we went back and looked at it. And quite clearly, Dr. Portier testifies -- and I can provide the Court a copy -- that he only relied on 12 studies.

And he specifically says:

"The last one is an animal bioassay that I just found that looks like it's well-conducted, but it's really poorly documented, so I can't include it because I really don't know everything about it, so

it's not included here," referring to the Takahashi paper.

So the suggestion that he disclosed this study, his analysis of it, and was then allowed to be examined by Monsanto's lawyers at that examination is just untrue.

Counsel does ask him: "Was there a lymphoma finding?" And he says, "Yes." To which our co-counsel objects that it's an undisclosed opinion, and that's it.

So all this other stuff that they went into with this Takahashi paper wasn't subject to disclosure or examination. And by the witness' own admission, at the time of that examination, he hadn't analyzed the underlying paper, and so it couldn't have been fodder for probing questioning.

THE COURT: So let me ask you this: I thought part of your response was that when he took a deposition in Australia he discussed that.

MR. WISNER: That's correct. And he's pulling out select quotes. I haven't had a chance to go through the transcript. There's three whole days of it. I think he's only looking at the first two days. The third day we talked some more. But I'll find the quotes for you.

THE COURT: "We" as in the plaintiff's counsel

in Hardeman?

MR. WISNER: It was actually me, I was there, but yes.

THE COURT: Oh, okay. I didn't realize that.

MR. WISNER: I actually took Dr. Portier's testimony in Hardeman.

MR. ISMAIL: So I am referring to that Australia testimony. I am referring to all three days of -- Takahashi doesn't appear in the transcript once. Not once. His tumor chart, the same things they've been walking through, had five mouse studies.

His testimony was how many are you relying on? He says 12. He references this last one that was flagged in the Zhang paper but says in his testimony, "I haven't had a chance to review it."

Counsel asks him on day three, "Was there a lymphoma finding there?" He says, "Yes." We, of course, object as an undisclosed opinion, since we have no idea what he was talking about. And that was it.

THE COURT: Was your objection sustained or overruled?

MR. WISNER: It was a deposition.

THE COURT: I'm sorry, you're getting me a little bit confused with the Hardeman trial and the --

Okay. So he appeared by video, you're right.

MR. ISMAIL: To the extent the suggestion was that this paper was disclosed, his opinions were vetted, that's not consistent with the transcript that we've had a chance to review this afternoon, Your Honor.

We would ask that his opinions in reference to Takahashi be struck as undisclosed, and that's the relief we're asking for at this time.

THE COURT: Well, I'm going to have to give that some thought.

MR. WISNER: I just have to look at the transcripts. There was a lot of discussion with the lymphoma findings, and Takahashi was included in those. There was lots of testimony that was given about it, even if the word "Takahashi" wasn't said. Mostly because I was afraid to mispronounce it, probably. But there's lot of visual parts about it.

The second thing, I just want to clarify, this was a deposition, right, it just was also videotaped and played at trial. But after it was videotaped, we negotiated page and line, it was a full song and dance.

So it wasn't like a live video presentation where they would be afraid to ask questions. They could ask anything they wanted to and have it cut out later, which is what they did. Most of their cross-examination was not played for the jury, they didn't even designate

it, it was just deposition.

But I need to go through the transcripts, and I can show you the portions where I believe this lymphoma finding was discussed. And, frankly, that's what he discussed today. He didn't really offer much beyond that.

THE COURT: Well, I'll give you an opportunity to go over the transcript. We'll continue this conversation tomorrow.

MR. MILLER: Your Honor, unrelated to that, if the Court has time this evening or tomorrow morning, we filed for a temporary restraining order against Monsanto. We do have a reservation number.

It's a narrow ask. We think everyone ought to be trying this case to the jury in the box. No one should be talking to the jury in the hallway, and no one should be trying to talk to the jury through the Wall Street Journal or the internet.

If you go on the internet now, there is a -- and you want to Google up what's going up in the Roundup trial -- because there are bloggers that blog every day -- the first thing you get is a paid ad by Monsanto telling you everything they want you to hear about their studies and about a federal judge's ruling that they falsely claim precludes them from warning that Roundup

causes non-Hodgkin's lymphoma.

So it's a narrow ask. We simply want them to stop advertising until the trial is over because it's designed to reach this jury. And that's -- we could run ads in Google too --

THE COURT: I mean, if you're suggesting that I actually have the ability to order Monsanto in St. Louis, their executives, to cease advertising, you're treading pretty hard on the First Amendment. Just without having seen any of it.

MR. BRADY: Your Honor, I wrote the brief. We did not ask for that at all. In fact, we specifically excluded any ask that they be prevented from advertising. That's really not the whole point of this.

I wrote it, and Mike has been busy doing many things with this trial. All we asked is that they stop running pieces like they ran in the Wall Street Journal saying that it's 40 years of safe product and science, and the science all backs up its safety.

They're speaking directly to the issues that this jury is going to be asked to decide at the end in these ads, Your Honor, that show up at the beginning of any Google attempt to find anything out about this trial, and then these print ads they put in the Wall Street Journal.

They can advertise for their product until the cows come home. They ran 1300 pieces during March Madness last weekend on the Monsanto Family of products. Let them do that all day long. We're not trying to ask you to impinge on their First Amendment rights.

But they should not be running pages that are specifically designed to influence this jury in this courthouse in this courtroom in this trial that speak directly to the safety and the science that's being asked of this jury to decide, Your Honor. It's a very limited motion, and you'll see it speaks just to that issue.

THE COURT: Okay. I haven't seen it yet. I know you are filing it. And Defendant hasn't had an opportunity -- there's really no point in me trying to comment on the specifics of it.

MR. BRADY: Please read it.

The other thing, Your Honor, we made all the changes you asked for in the --

THE COURT: I know that you were involved in the videos. I didn't ask for specific changes. I just wanted to get a copy of it. After I saw it, I was concerned -- and that's a kind word -- with what I thought was conveyed in those videos.

I read the case. I don't thing it really had

much of anything to do with a civil case in terms of what the standards might be for animation. And certainly, I don't think has anything to do with my exercise of discretion and what I think the jury should or should not see.

Having said that, I don't know whether what you really want to do is go through the videos. Yes, my immediate reaction was we will take all the words out for sure. And some of it, I thought, was not so much a video of someone's opinion but, in fact, evidence itself, which was really worrying to me.

Because I think the minute the video -- you're looking at the video, and you're really not necessarily hearing, I think, what the expert is going to say. Because the video itself has its own story, and that concerns me.

MR. BRADY: Your Honor, this is exactly what the California Supreme Court en banc addressed in People vs. Duenas.

THE COURT: I went over it several times. It was, essentially, the Court didn't commit reversible error by including it, not setting a standard for animation or for video in connection with expert testimony in a civil case.

MR. BRADY: Your Honor, pages 23 to 27, they

addressed every single objection that was made by the defense in that criminal murder prosecution. And they very carefully went through all the arguments concerning whether it was speculative, whether it was cumulative, whether it had an air of scientific certainty, whether it was prejudicial.

The Supreme Court did an exhaustive analysis of those particular -- of all the objections you could ever make from a legal standpoint to that animation. And the Court, at the end, said that if the animation generally shows the expert's opinion and is just a demonstrative aid -- in that case, Your Honor, they showed literally the CSIs version of how the whole crime was carried out. These are much more general. They are --

THE COURT: I'll tell you, I can't tell you my reaction to that video, how strong it was, in terms of the creeping blue on the skin and the vapors and all that other stuff. Because I really felt you were telling a story with that video, and it did not seem to me at all to be, essentially, a live version of what we were seeing, you know, with something actually replicating what the document would be saying.

Because I can't imagine what the document would be saying along with some of that video.

MR. BRADY: Your Honor, that's been changed. But that's not even the one we're talking about. The one that we wanted to do with Dr. Portier today was the mechanistic animation, which you had no problem with.

I think it's different. This is the one that just the cells and how the cells begin to replicate after there's been DNA damage. There's almost no text. All it did was show the two mechanisms. It just said literally genotoxicity and oxidative stress. We removed all the other words on there.

THE COURT: I would have gone over it with you this morning, and I would have commented on it and said yay or nay, but I didn't have an opportunity.

MR. BRADY: Can we look at it tomorrow morning when everyone is fresh real quick? It will take you two minutes, and then you can decide whether or not Dr. Portier -- before he heads back to Australia -- can speak to this.

THE COURT: We'll see how much time we have. We'll be here at 8:30 tomorrow morning. I will look at that one only tomorrow morning. We'll talk about it. But I'm not making any promises.

MR. BRADY: It's very narrow, Your Honor. Thank you.

THE COURT: Thank you. I'll address all these

things you're telling me about, but I don't really have any documentation. To be continued tomorrow morning.

Thank you. I'll see you tomorrow morning.

(Proceedings adjourned at 4:43 p.m.)

State of California)
)
County of Alameda)

We, Kelly L. Shainline and Lori Stokes, Court Reporters at the Superior Court of California, County of Alameda, do hereby certify:

That we were present at the time of the above proceedings;

That we took down in machine shorthand notes all proceedings had and testimony given;

That we thereafter transcribed said shorthand notes with the aid of a computer;

That the above and foregoing is a full, true, and correct transcription of said shorthand notes, and a full, true and correct transcript of all proceedings had and testimony taken;

That we are not a party to the action or related to a party or counsel;

That we have no financial or other interest in the outcome of the action.

Dated: April 2, 2019

Kelly L. Shainline
CSR No. 13476, CRR

Lori Stokes
CSR No. 12732, RPR