SUPERIOR COURT OF THE STATE OF CALIFORNIA

COUNTY OF SAN FRANCISCO

DEWAYNE JOHNSON,

Plaintiff,

vs. Case No. CGC-16-550128

MONSANTO COMPANY, et al.,

Defendants.

_____/

Proceedings held on Monday, July 16, 2018, Volume 10, Morning Session, before the Honorable Suzanne R. Bolanos, at 9:17 a.m.

REPORTED BY:

LESLIE ROCKWOOD ROSAS, RPR, CSR 3462

Job No. 2965312A

Pages 2172 - 2279

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EXHIBITS ADMITTED

(None.)

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Volume 10

Morning Session

San Francisco, California

Department 504

Judge Suzanne Ramos Bolanos

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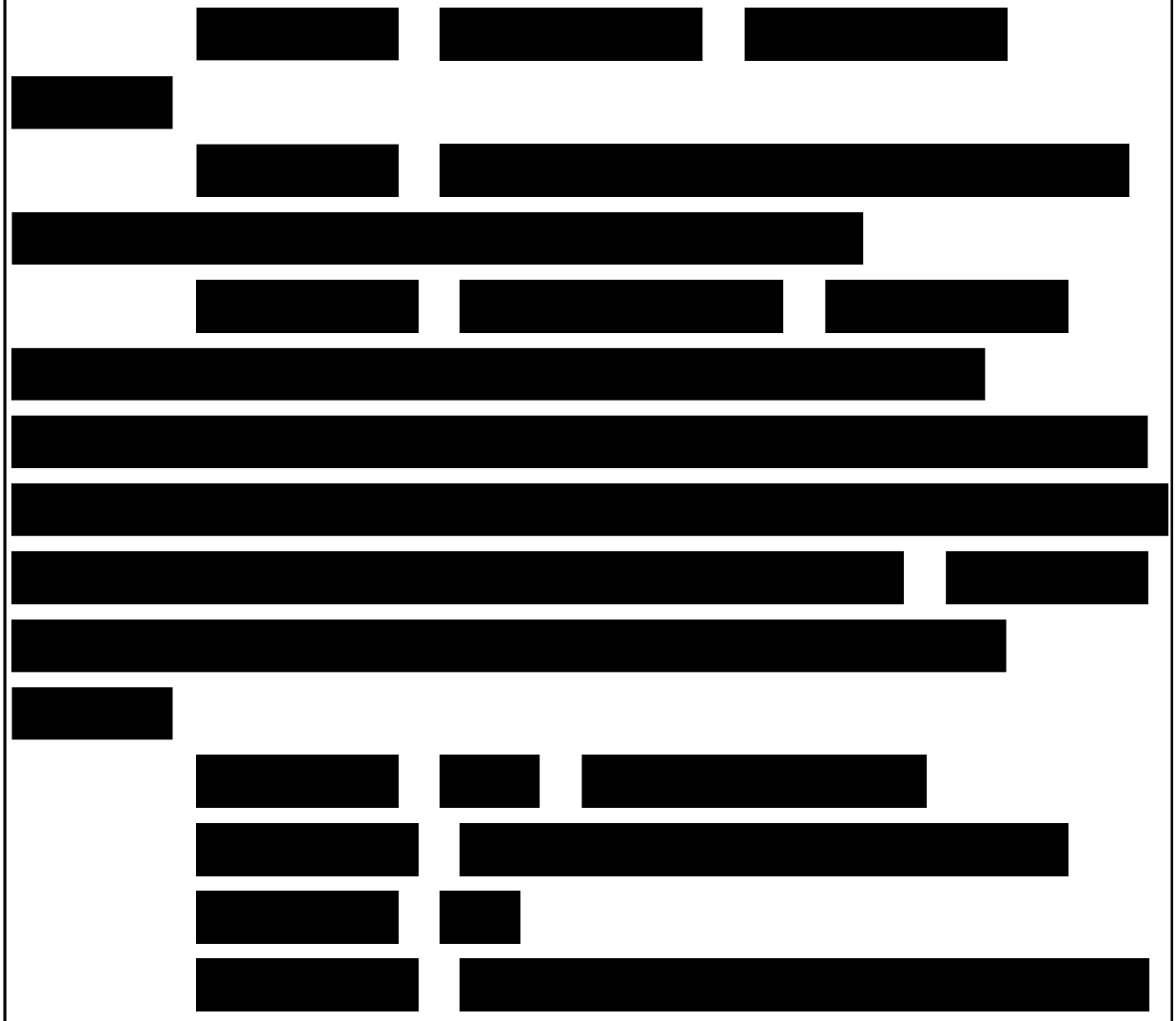

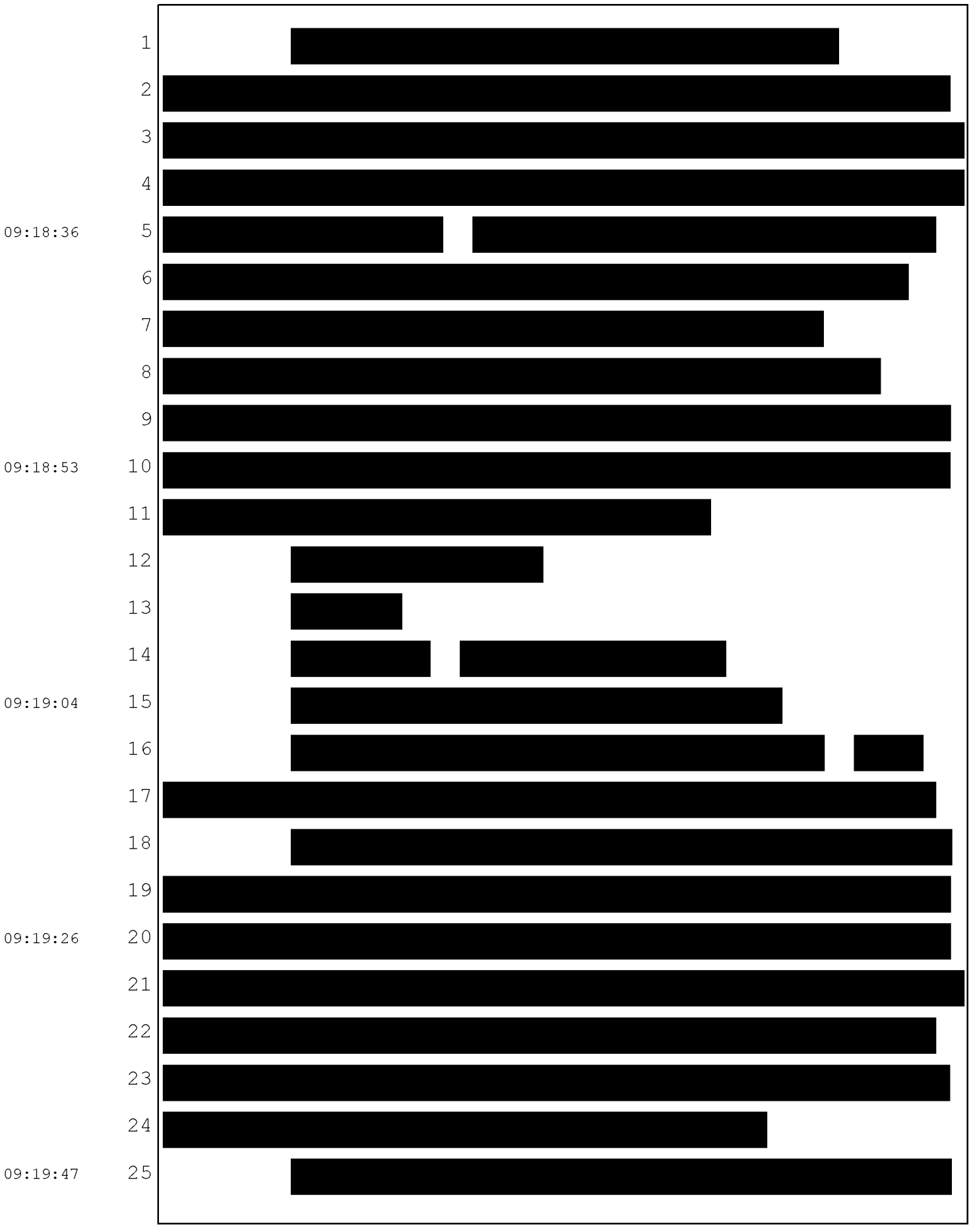
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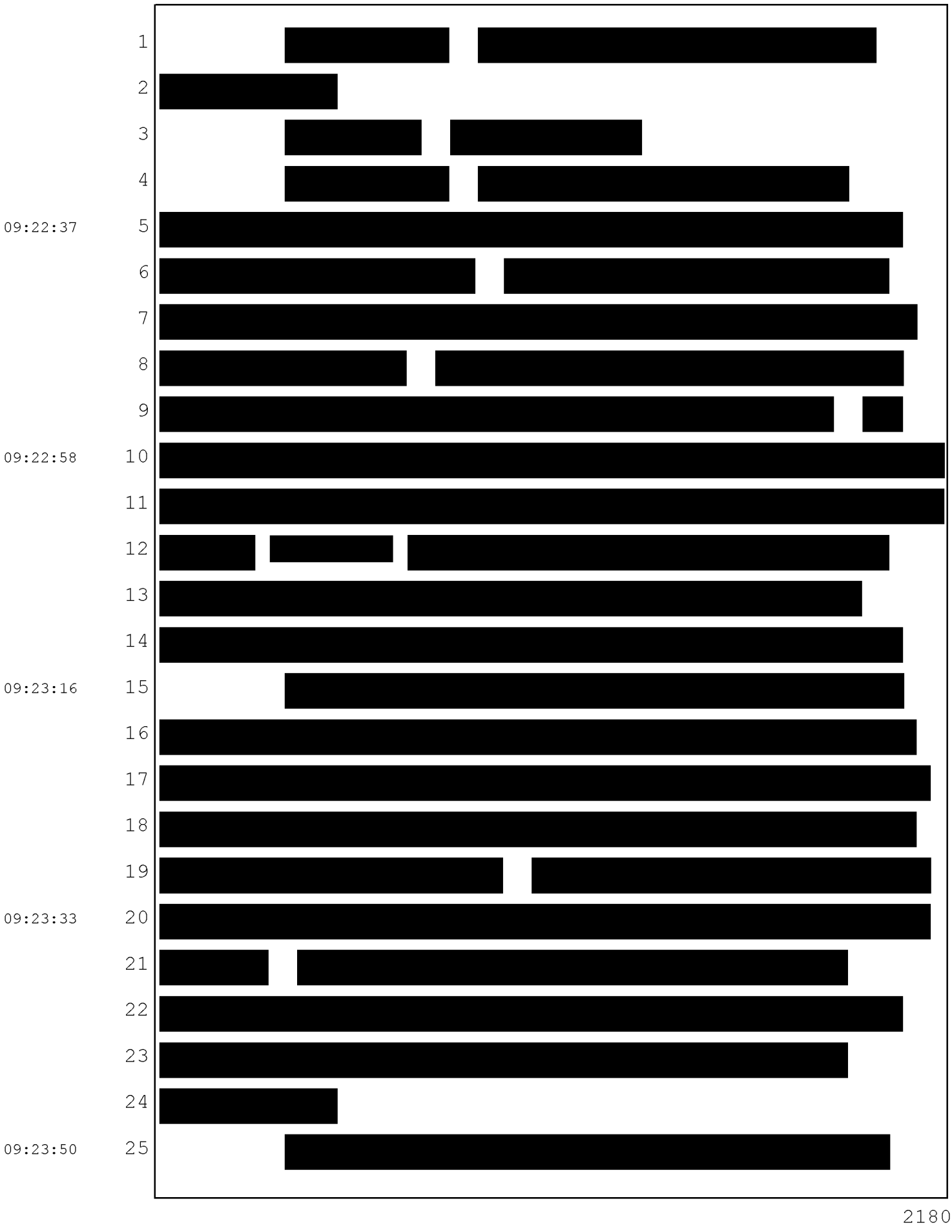
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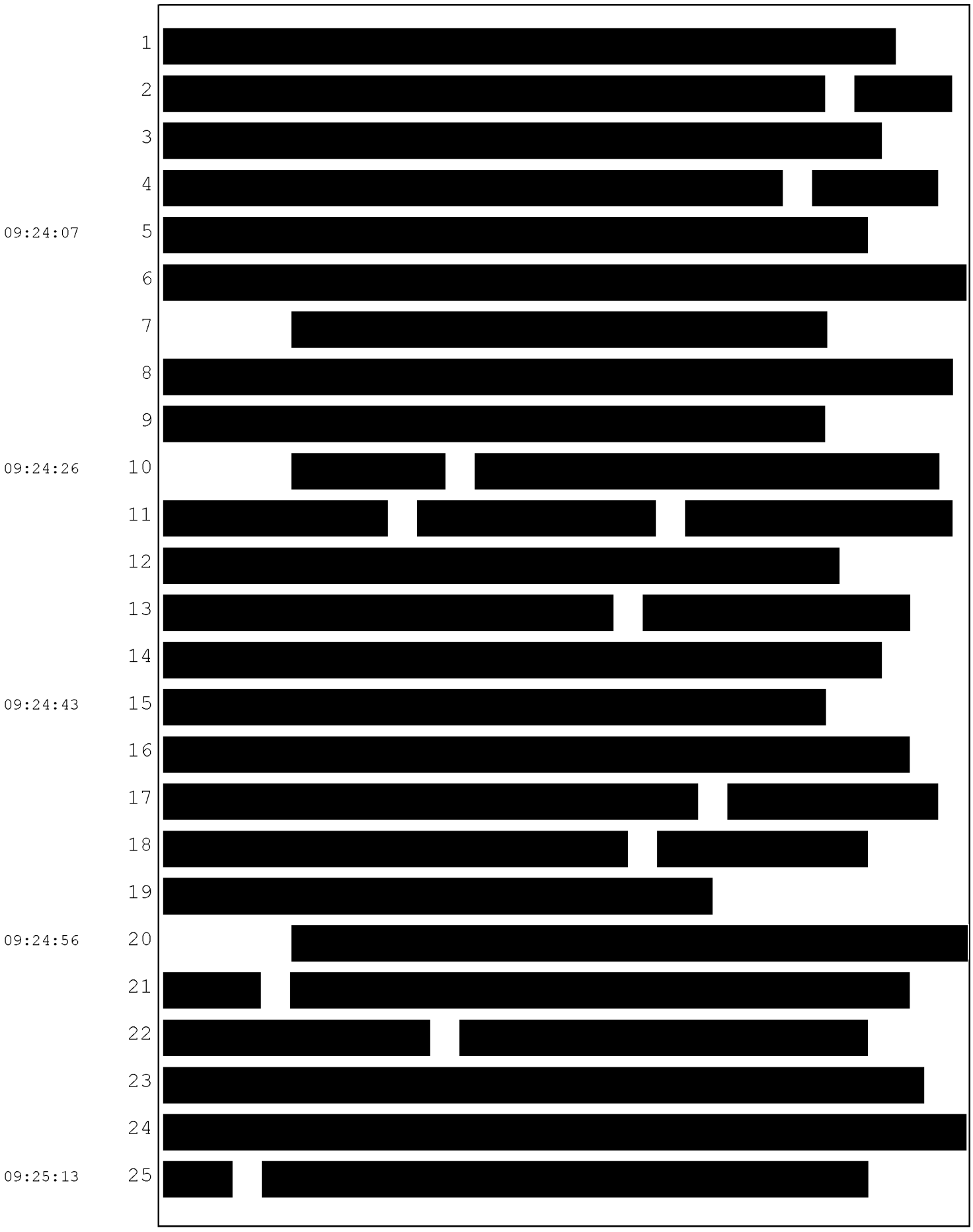
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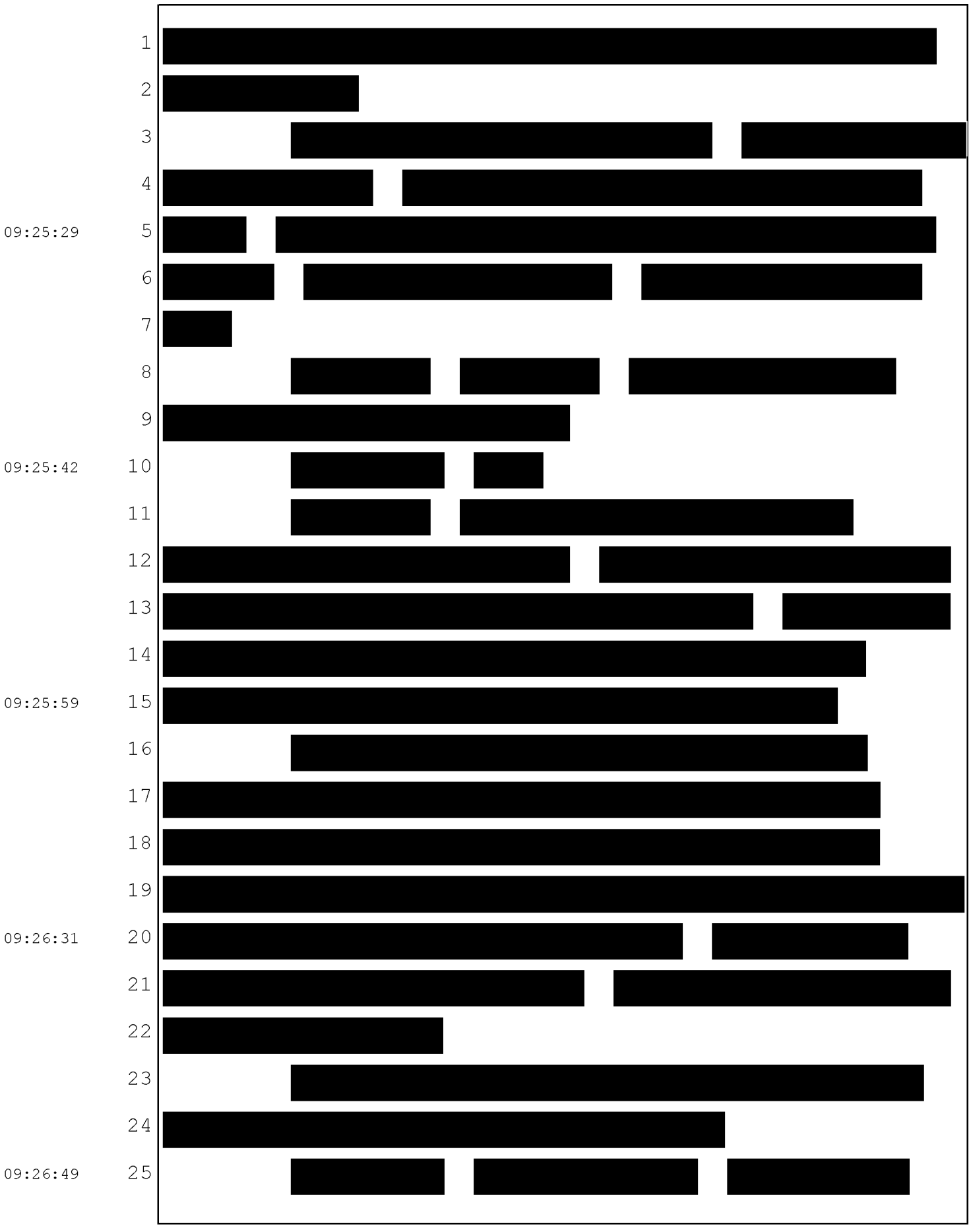
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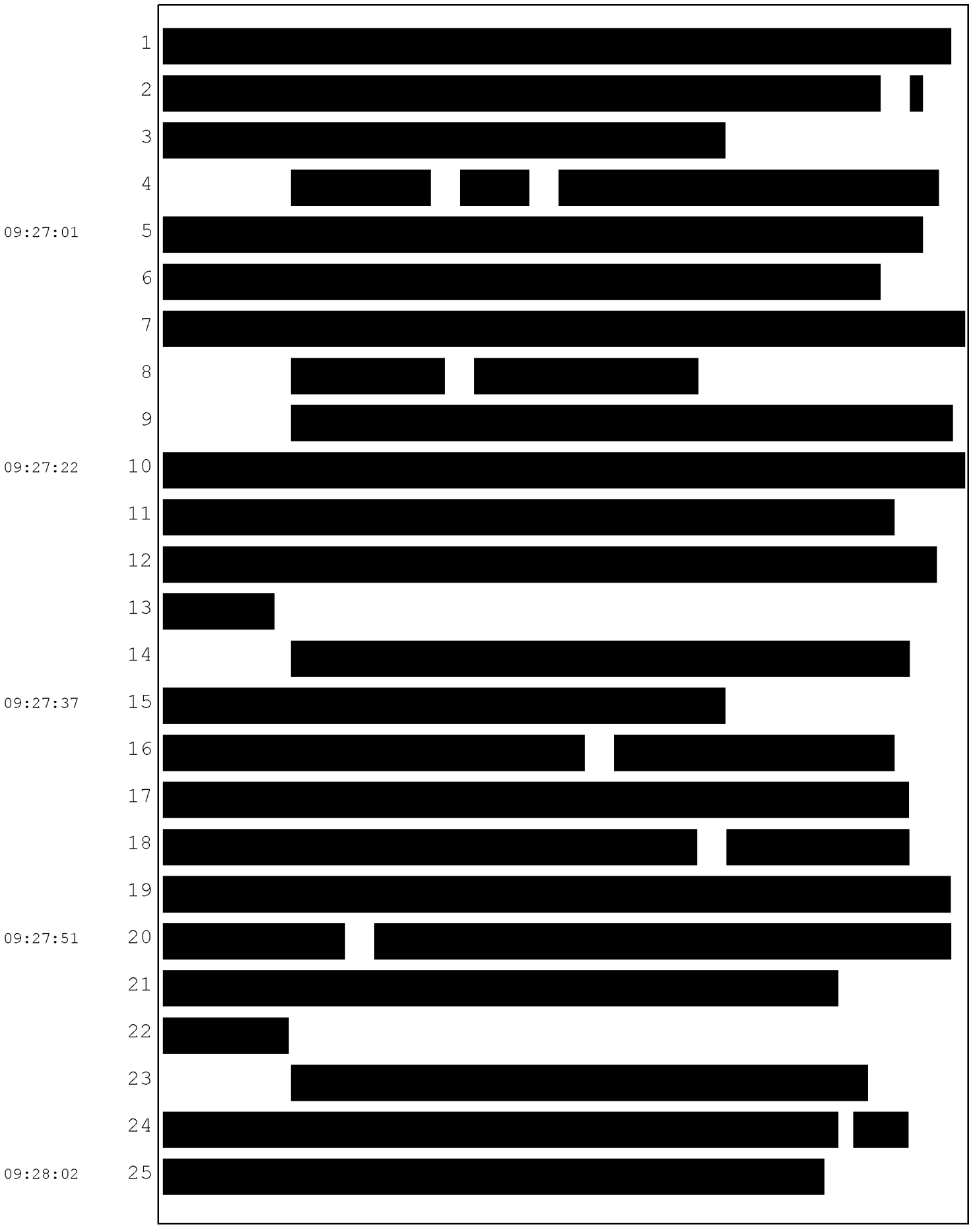
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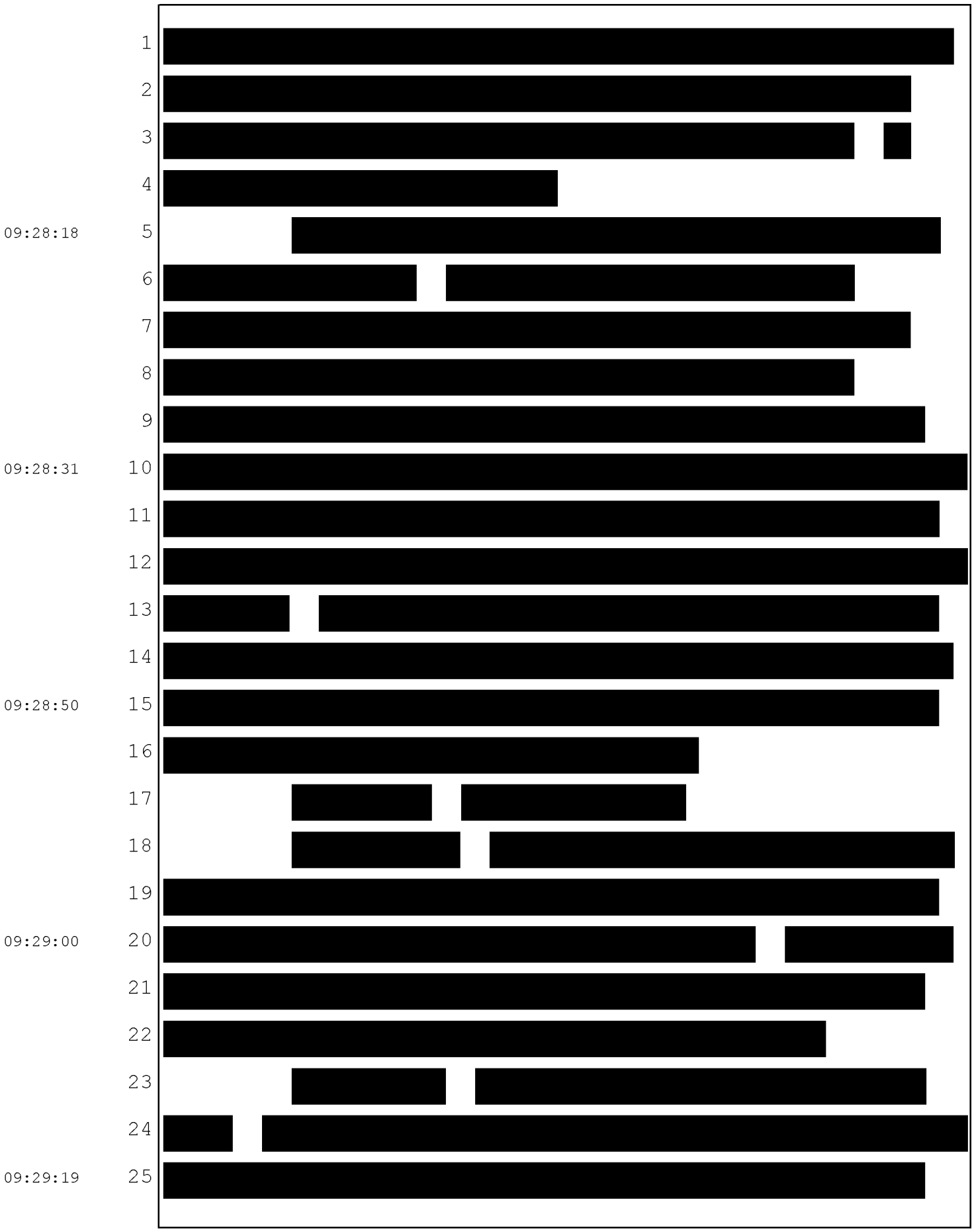
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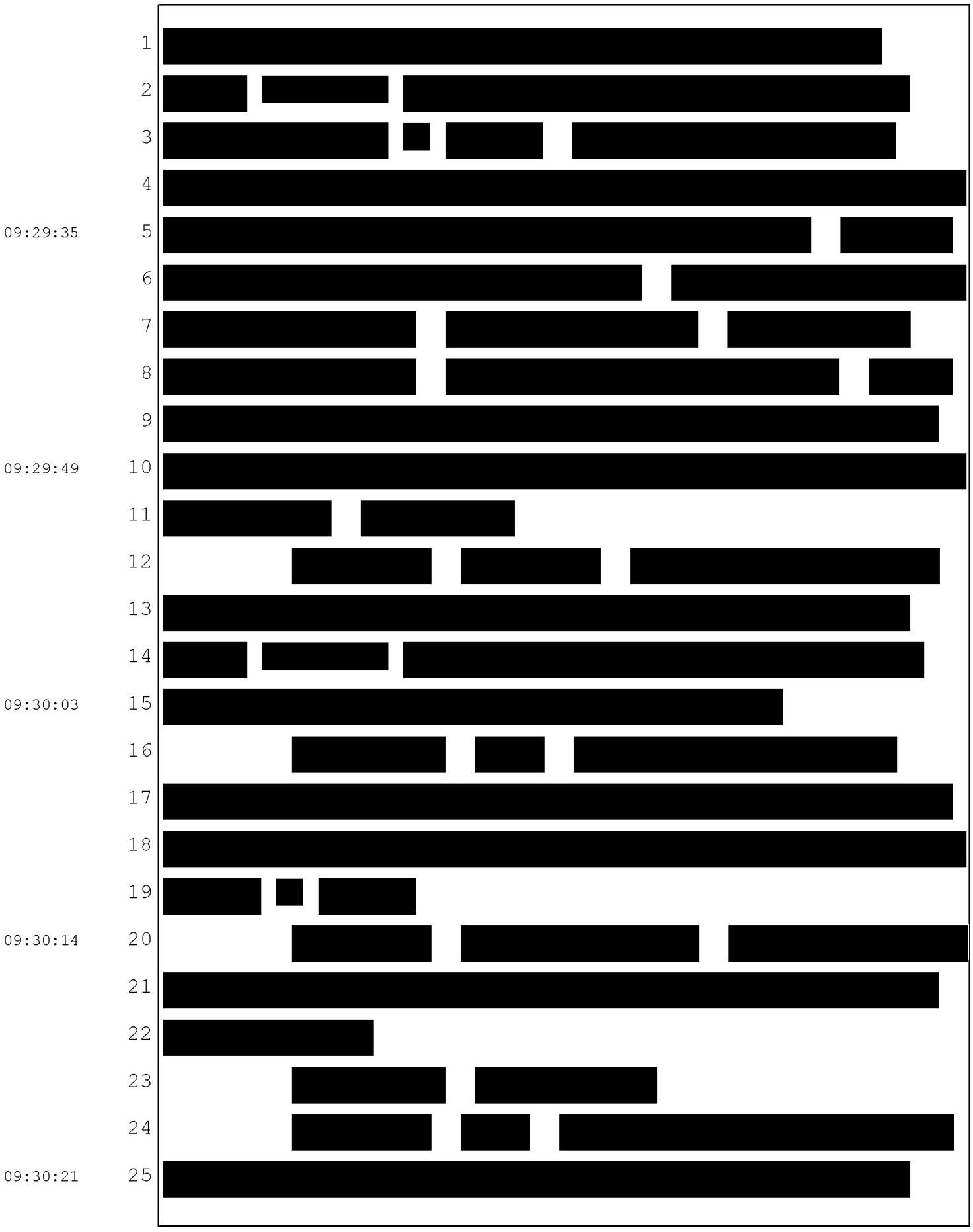
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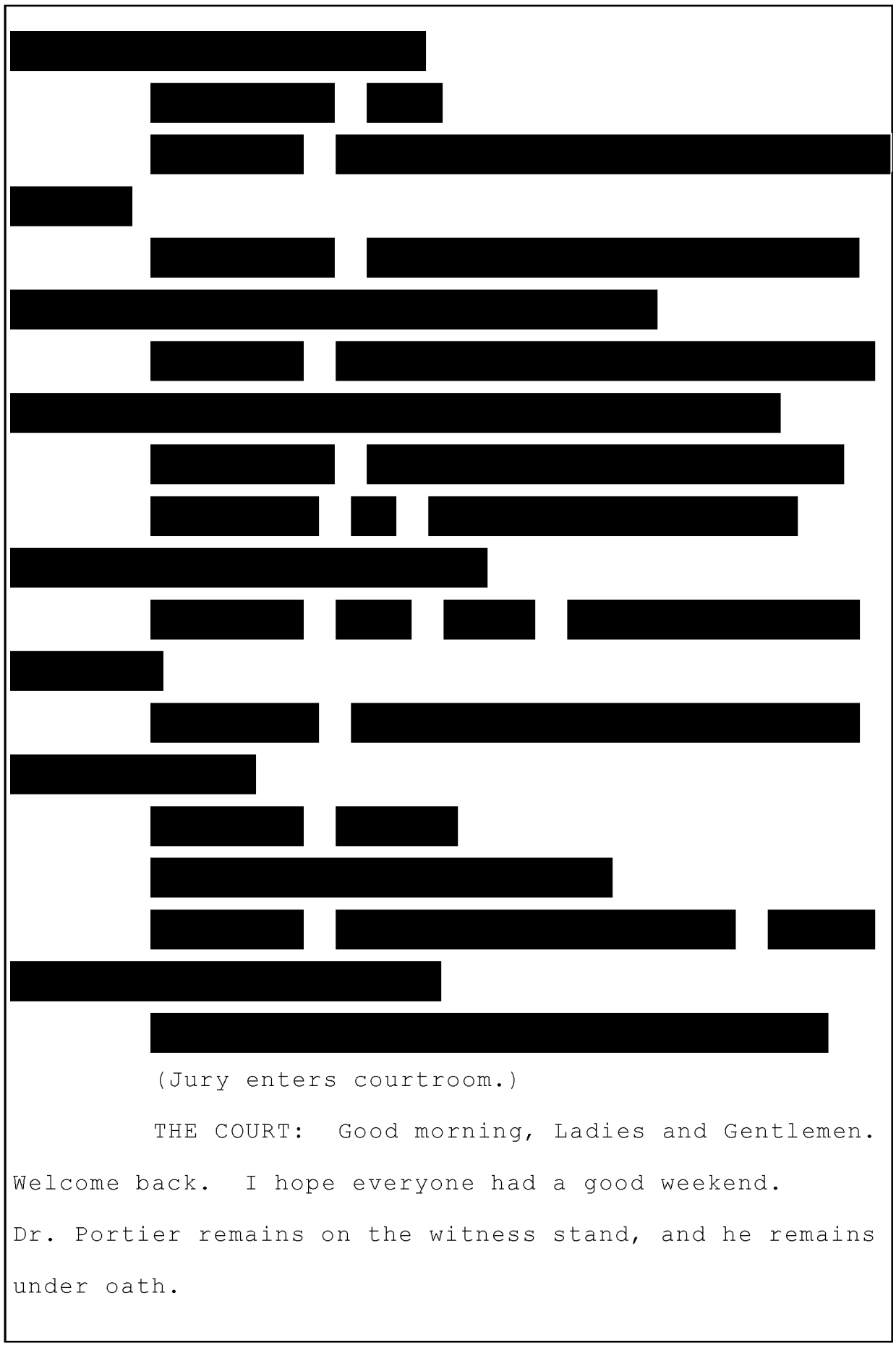


(Jury enters courtroom.)

THE COURT: Good morning, Ladies and Gentlemen. Welcome back. I hope everyone had a good weekend. Dr. Portier remains on the witness stand, and he remains under oath.

09:30:30
09:30:41
09:30:54
09:32:32
09:33:51

And, Mr. Griffis, when you're ready.

MR. LOMBARDI: Your Honor, there's a juror that had a question.

(Interruption in proceedings.)

09:34:01 MR. WISNER: Your Honor, may I approach with a cup of water for the witness?

THE COURT: Yes.

All right. Mr. Griffis, when you're ready, you may --

09:34:14 MR. GRIFFIS: I have one more binder for, your Honor.

THE COURT: Thank you.

MR. GRIFFIS: May it please the Court.

CROSS-EXAMINATION (Continued)

BY MR. GRIFFIS:

Q. Good morning, sir.

A. Good morning.

Q. On Friday we talked for a while about the issue

09:34:45 of multiple testing and the multiple testing problem that arises in the animal studies, but also in any kind of study, including mechanism studies, where there are many tests that are done and, therefore, the possibility of multiple false positives; right?

09:35:07 A. There's a possibility of false positives even

when there are few tests.

Q. And there are -- it's a bigger problem when there are more tests; right?

A. Usually, yes.

09:35:18 Q. And that's because a person who does not know about or correct for the multiple testing problem may say, "Oh, I've got a bunch of positives, the overall outcome must be positive or this must be a positive indicator," when, in fact, it might just be false

09:35:36 positives that you would expect, given the number of tests that were run; right?

A. It could be either, but there are two ways to address false positives, not just correcting the p-value.

Q. There are a bunch of tests that are done, and

09:35:50 these are done every day by lab scientists who run multiple tests like the false detection rate correction, the Bonferroni correction, the Holm-Bonferroni correction, the Sidak correction, et cetera; correct?

A. That's one of the ways in which this can be

09:36:09 done. The other way is the way the National Toxicology Program does, which is run all of the p-values at 05 or 01, depending on what they're looking at, and then consider whether these are false positives after the fact.

09:36:27 Q. Considering using biological criteria?

A. Well, both biological criteria and statistical criteria.

Q. Now, you mentioned biological and statistical. Those are the two main areas -- the two main disciplines that go into analyzing this sort of thing; right?

09:36:39

A. Well, that's the two main disciplines that go into -- into the development of any experimental study.

Q. Okay. And you're more on the statistics side. You've done a bunch of statistical analyses, you've been here talking about those statistical analyses, and you've been, in fact, consulting occasionally when you have a question with pathologists, like Dr. Weisenburger an expert witness for Plaintiffs, and people like that when they need a consult on the biology; right?

09:37:00

09:37:14

A. I've had exactly one consult on the biology, and that was that one email sent to Dr. Weisenburger. There are no others. And I would not characterize myself as a statistician -- solely as a statistician. I have credentials in toxicology. I have credentials in molecular biology. So I don't feel I need constantly to consult with anyone over the biology involvement in this particular case.

09:37:32

Q. You're fully qualified to comment on the biology in the case?

A. With the exception of complicated pathology and

09:37:42

in cases where I've said that's beyond my area of expertise.

Q. You didn't do a false detection rate correction or any of the other statistical corrections I discussed; right?

09:37:57

A. That's not true. I'll repeat it again. I did the same thing that is usually done by the National Toxicology Program, and that is to calculate the probability that these are all false positives or these are in a particular set of studies just to get a feel for whether they're false positives or not.

09:38:11

But the reason not to do a false positive correction is because you're looking for -- when you're doing an animal tox study, you're looking for patterns. You're not there definitively saying, "If I see a significant p-value, that is a real finding," which is what you're doing with a false positive correction.

09:38:28

Can I take a minute to explain this to the jury, as to what a false positive correction is?

Q. You're going to explain what a false detection rate correction is?

09:38:47

A. It's just one of the many false positive -- ways of correcting for false positives, like Bonferroni. So when you do an animal study and you do -- let's take a simple case and do 20 tests -- statistical tests on the

09:39:00

data and each one is tested at the .05 level. Well, that's 1 in 20. That's what .05 is or 5 percent, 1 in 20.

So on average, you would expect one positive finding just by chance. Doesn't mean you get it. You could get two or you could get none. You just -- it's a chance that you could get it. So one way to correct for that is called -- the simplest one is Bonferroni correction, but the false detection rate is another, and that is to change your p-value based upon how many tests you're going to do, so that when you see something at this level, you're more certain that it really is a positive finding.

Q. Those are the tests you did not run?

A. What?

Q. Those are the tests you did not run, false discovery rate, Bonferroni, et cetera; right?

A. Those are the corrections to the p-values I did not run. And the reason -- the reason it's not done by most people running toxicology studies is because they're not looking for the most definitive p-value. For example, I might run -- if I ran the 20 tests, then my correction would probably take my p-value down to, say, .03, let's say, just for sake of argument. I could get 4 positive -- 4 findings at .04, but I would exclude those,

because they're all above .03, when, in fact, if it's something that looks like, let's say, leukemias, lymphomas and lung tumors, all right, three of those four, well, it's already known in the literature that
09:40:51 leukemias, lymphomas and lung tumors are related to each other biologically, so you would miss that if you declared all of those to be not there.

You want -- you keep pushing that the p-value is a definitive "yes" or "no." It is not. It is a guidance
09:41:08 to tell you where the important findings are in the database.

Q. I want to talk to you about your patterns, your biological patterns in a moment, sir. But the answer about the false discovery rate, Bonferroni, et cetera,
09:41:23 you didn't go that route; right?

A. I definitely did not go that route.

Q. Now, you did, in your expert reports -- and we talked about the issue of multiple tests and how when you're looking at a whole bunch of animal studies, in
09:41:37 each one of which you could find -- potentially the pathologist could find tumors in all sorts of different organ systems. We looked at some of the charts very briefly and saw some of those lists of data.

When you have that, you have -- you're
09:41:53 essentially running multiple tests, and you compute it --

based on Dr. Haseman's guidance, which you decided to pursue in creating your expert report -- that there were over 500 potential tests that were run for purposes of doing the statistics; right?

09:42:11 A. I'd have to look at my expert report to give you the exact number.

Q. And both times that you did this -- you made some corrections at some point. We won't get into the weeds about the details of that. But both times, you

09:42:23 calculated how many overall positives you would expect by chance alone, and both times it was over 20; right?

A. Again, I'll repeat what I told you earlier. In the text, I pointed out that the correct way to evaluate false positive rate is by sex species group.

Q. Okay. Well --

A. And so I never had a single sex species group that I'm aware of that expected 20 tumors.

Q. Let me tell you why I'm asking you about all of them at once. We had boards put up that showed all of

09:42:59 them at once --

A. That was --

Q. -- and if you put the boards up with them all at once corresponding to the lines on your chart, sir, where you calculated what the numbers would be all at once,

09:43:07 both times you did that math, you came up with more than

20 that would be false positives; right?

A. The boards that were put up were color coded at the top by species and strain. So I'm not going to agree that we just threw them all up. There we had a board of rats broken up into Sprague-Dawley and Wistar, and we had a board with mice broken up into CD-1 and the single Swiss albino study.

09:43:27

My discussion -- the reason for putting all of the mouse on one picture is to look at the -- the agreement across the whole picture on various organs for various tumors.

09:43:47

Q. Okay. My point's just that -- and we're going to talk about your patterns in a moment. We are. But my point is just this: We had a little exchange where we put up a board -- and we put up both boards for you -- and you put some X's on them to show some of the ones that you were less persuaded by or that you didn't really feel were real persuasive on carcinogenicity, et cetera. We'll get to that. You didn't put 20 X's up, though; right?

09:44:00

A. I don't know how many X's I put up.

Q. Okay. So let's get to your pattern.

We've got five mouse studies and seven rat studies that you consider to be of high enough quality to consider for purposes of this analysis; right?

09:44:33

A. Correct.

Q. And you were telling us which ones you thought were more likely to be false positives and which ones you -- were more likely to be true. And because you're a statistician, you weren't saying, "Yes, yes, no, no, no." You were -- you were being more general than that. You were more confident in this one and less confident in this one. But that was the exercise we went through; correct?

09:44:50

09:45:03

A. No. The exercise was not just a statistical exercise. The exercise also dealt with the biology, as I pointed out. Noting that you saw patterns across the various studies where you had statistically significant and marginally significant findings that, when put together, point towards a much more strong finding than any statistics would pull out of that analysis.

09:45:19

Q. As an example, for the rats, the one finding that you considered to be persuasive and important -- you identified five total. The one for the rats was skin keratoacanthoma; right?

09:45:38

A. That is correct.

Q. And you pointed out to the jury that that was probably the strongest finding in the rat data, but they are benign tumors; right?

09:45:46

A. That is correct.

Q. And you said if you're looking for carcinogen, that technically these aren't carcinogenic findings; right?

A. Let me be clear. They're benign tumors that can, on occasion, become malignancies. But, yes.

09:45:55

Q. Okay.

A. An agency that was looking for carcinogenic potential of a substance would weight benign lesion findings lower than malignant lesion findings. But they'd still weigh them.

09:46:08

The guidelines clearly state that if you see -- for example, if you look at the European guidelines, if you see multiple benign findings, they're likely to call that a carcinogen.

Q. Let's look at the mice.

09:46:21

A. Okay.

Q. The four that you identified -- the four tumor types that you identified were hemangioma, hemangiosarcoma, lymphoma and then kidney carcinoma/adenoma; right?

09:46:33

A. Correct.

Q. Okay. And the others on the mouse chart, you told us -- you told us about various weaknesses. Like for lung, you know, carcinomas, you said the evidence was not strong enough that it pull you forward. The same

09:46:47

with harderian gland. You said you didn't use multiple malignant tumors to make your decision. That wouldn't typically be done, et cetera.

We're going to focus on the four that you wanted to focus the jury on; right?

MR. WISNER: I'm going to just object to -- the lawyer's testifying. There's, like, five sentences in there. I don't know if I disagree with all of them, but there's a lot in that question. It's cumulative.

I'm sorry. Compound.

THE COURT: Overruled. He may answer, if he understands the question.

THE WITNESS: I would love to hear the question again.

Q. BY MR. GRIFFIS: Sure.

You were talking about the strengths and weaknesses of the mouse studies. We also did the rat studies. And you identified the skin keratoacanthoma. And for the mouse, you found significant and told the jury that they should consider significant, hemangioma, hemangiosarcoma, lymphoma. And then kidney carcinoma/adenoma put together; right?

A. Yes.

Q. Okay. And you were pointing -- you were also pointing out at the same time various weaknesses in some

of the findings on that chart. Like, for example, for lung adenocarcinomas, you said the evidence wasn't strong enough that it would pull you forward; right?

A. Correct. Because there wasn't even -- there wasn't marginal findings even in the other studies.

Q. And you said the same for harderian gland. It wasn't strong enough that it would pull you forward?

A. Correct.

Q. Another example is you said there were several up there that said multiple malignant tumors. And you said you wouldn't use that to make your decision. That wouldn't typically be done; right?

A. Well, I'm not going to rule them all out. They're -- they're part of the evidence I'm looking at.

And I think you skipped the part where I said that the pituitary adenomas and carcinomas in a single study, because it was both sections, had to carry greater weight for that one study.

Q. Okay. Now I want to talk now about the five -- the four in mice that you identified. Let's start with malignant lymphoma.

That is in male mice, not females; right?

A. Correct.

Q. Okay. So for each one of those studies -- and I know the information was kind of on there, but it's not

like the column was divided in half. For every one of those, there were both male groups and female groups in the study. True?

A. I'm not sure I understand your question.

09:49:13 Q. Okay. When you do a rat study, like the medium dose group, there are 50 males, 50 females; right?

A. In -- we went through this. In any study typically for regulatory approval, you're looking at three exposure groups, one control group, 50 animals per

09:49:33 group, males and females.

Q. Okay. And the malignant lymphoma results are in the male groups and not the female groups?

A. That is correct.

Q. Okay. There were three CD-1 studies: Atkinson,

09:49:47 Sugimoto and Wood; and one Swiss albino, Kumar, that you considered significant with regard to malignant lymphoma; right?

A. I'd have to look in my expert report or the picture.

09:50:00 Q. I'll show you the picture (indicating).

A. I can see it. That's fine.

Q. Okay.

A. Now, when I did those circles, I had notes in front of me. Could I bring those notes back up?

09:50:45 Q. Yeah.

A. If I could find them.

THE WITNESS: Does anyone know what exhibit this was? Oh, 1020.

MR. GRIFFIS: 1020.

09:51:26 THE WITNESS: Thank you.

Q. BY MR. GRIFFIS: Do you have notes on your copy of 1020?

A. I've got notes on my copy, yes.

Okay.

09:51:35 Q. All right. So malignant lymphoma: Atkinson, Sugimoto, Wood, Kumar; right?

A. That's correct.

Q. And for all three, you circled male, male, male, male. Because it wasn't found in the females; right?

09:51:52 A. That is correct.

Q. Kumar -- the Kumar study here has been flagged by multiple regulators as suspect because of a virus problem in the study; right?

A. No regulator that I know of has owned up to that

09:52:10 fact in writing saying they clearly had a virus. Both EFSA and the EPA have backed off of that statement.

Q. Okay. Well, let's look at the OPP report, the Office of Pesticide Programs report, of EPA from 2016. You'll find it in your Regulatory Binder Number 2.

09:52:36 A. Let's have the most recent report, because

they've backed off on that statement. That's the point.

Q. They said it's not a virus?

A. They no longer state that there was a virus in the colony.

09:52:50 Q. Okay.

A. Because they couldn't -- they had no proof. They were challenged on it at their SAP meeting.

Q. Okay. Let's look at 2016 first, sir, on page 78.

09:53:02 A. Of this one (indicating)? The new one?

What's the --

Q. 2841.

A. Okay. Which folder is it in?

Q. It's Regulatory Binder Number 2, sir.

09:53:50 A. I have it.

Q. Page 70.

MR. WISNER: What exhibit number?

Is it 2481?

MR. GRIFFIS: Yes.

09:54:20 THE COURT: What page, Counsel?

MR. GRIFFIS: I'm sorry. We are on page 70, 7-0, of Exhibit 2841, which is in the regulatory binder.

MR. WISNER: Just for the record, it's 2481, not --

09:54:38 THE COURT: 2481, page 70.

Q. BY MR. GRIFFIS: Okay. You're there?

A. I am there.

Q. On page 70, they say at the top, sir, "These studies and justification for not including them in the analysis are listed below."

09:54:57

And you've read this and seen this many times; right?

A. Correct.

Q. You're very familiar with this report?

09:55:05

A. Yes.

Q. You've critiqued parts of it, too; right?

A. Including what we're about to discuss, I guess.

MR. GRIFFIS: I move the admission, your Honor, of 2481, so it may be displayed to the jury, and they can follow along.

09:55:18

THE COURT: You're moving the entire report into evidence --

MR. GRIFFIS: I am, your Honor.

THE COURT: -- or are you just asking to publish?

09:55:25

MR. GRIFFIS: Both. I'm asking to move it into evidence and to publish.

THE COURT: All right.

Any objection?

09:55:30

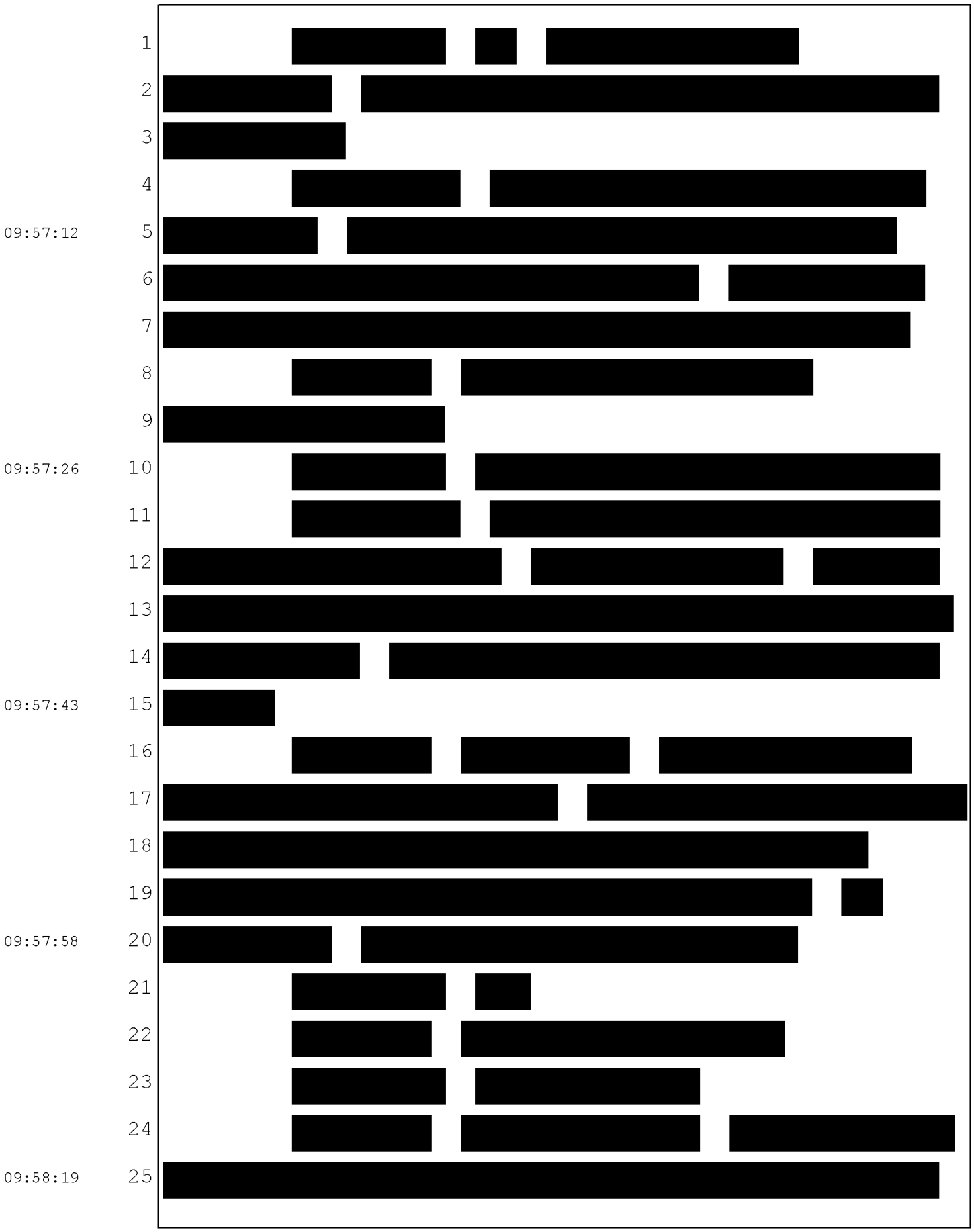
MR. WISNER: Yes, your Honor. It's hearsay.

THE COURT: Counsel, can you approach?

MR. GRIFFIS: Certainly.

(Sidebar.)

[redacted]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[redacted]

09:59:00 (End sidebar.)

THE COURT: So the objection's sustained.

You may continue, Mr. Griffis.

MR. GRIFFIS: Yes, your Honor. We will publish so the jury can follow along.

09:59:17 THE COURT: But the relevant portions of the document may be published to the jury.

MR. GRIFFIS: Thank you, your Honor.

Q. So this is Exhibit 2481, page 70. So on this page, the EPA in 2016, the Office of Pesticide Programs,

09:59:37 talks about a number of animal studies and the justification for not including them in the analysis; correct?

A. This is the 2016 document.

Q. Yes.

09:59:53 A. And for these four studies, this is their

justification.

Q. Okay. Now, let's look first at Number 4, "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"; correct?

A. That's what it says, correct.

Q. And they go on to talk about how malignant lymphomas were found in all those groups. But malignant lymphomas in mice are correlated with viruses in various ways; correct?

A. If we're going to read this, let's discuss it. They talk about malignant lymphomas. Then they talk about lymphomas. It's not the same as malignant lymphomas all the time. But they -- again, this is part of my comments to them.

The -- the reference they give breaking it down is not Swiss albino mice. So when they're talking about lymphomas being a common tumor in mice, they're not talking about CD-1 mice, and they're not talking about Swiss albino mice. They're predominantly talking about B63 F1 mice in the Brayton article. And they're generalizing in the Brayton article.

But if you look at the historical controls for the CD-1 mice -- I might not be right about the

Swiss albino. But if you look at the CD-1 mice, it's virtually 0 background.

Q. This is something that you've been very -- one of the many things you've been very critical about EPA about; right?

10:01:33

A. They're somewhat loose in both their references and their generalized statements, yes.

Q. Okay. So you have a disagreement with them, but this is what the EPA said in 2016 about why they considered Kumar not worthy of being included in the analysis; correct?

10:01:47

A. Yes, this is what they say.

Q. Yes, sir.

And while we're here, so we don't have to come back to this document later in the day, right above is another study they didn't consider in their analysis. And that's the George study, that tumor skin promotion study you talked about in your direct examination; right?

10:02:11

A. That's correct. But they wouldn't have included this study anyway.

10:02:29

Q. And they say, "Study deficiencies included: Small number, 20 of animals, tested only males and lack of phytopathological examination"; right?

A. That's what it says. But I will point out that EPA was doing an assessment of glyphosate only. So they

10:02:43

would not have included this study in their assessment anyway.

And as for their small numbers and testing only in males and lack of his pathological examination, they 10:02:57 obviously don't know the literature in skin painting studies, since almost all of them are 20 animals per group, predominantly done in male. And when you're just looking at pathalomas, you don't bother to do a phytopathological examination, which is what George did.

10:03:13 Q. That's another one you disagree with a lot.

A. And I will point out again this is not their final document.

Q. Yes, sir.

Let's put up 2486, because you didn't want me to 10:03:23 look at 2016, because you said we should go to the OPP 2017 report; right?

A. Correct.

Q. Okay. So let's do that. That's Exhibit 2486.

MR. GRIFFIS: And I move to publish page 70 of 10:03:36 that.

MR. WISNER: One second, your Honor. Let me take a look at it.

Your Honor, this isn't a document that Mr. Griffis is referring to.

10:04:03 THE COURT: 2486, page 70?

MR. WISNER: That's right. Dr. Portier referred to the paper that was issued after the SAP. This is still pre-SAP. It actually says it right there, issue paper (indicating).

10:04:21 MR. GRIFFIS: Mr. Griffis, are you asking for 2486, page 70?

MR. WISNER: Am I on the right document?

MR. GRIFFIS: I am. December 12th, 2017.

THE COURT: All right. Well, is there any

10:04:35 objection to publishing 2486, page 70?

MR. WISNER: Notwithstanding our previous objections, your Honor, no objection.

MR. GRIFFIS: Let's show the first page of it first, the cover page, so we have it identified for everyone.

This is a document from -- titled "Revised Glyphosate Issue Paper: Evaluation of Carcinogenic Potential."

Q. So this whole thing is about carcinogenesis;

10:05:05 right, sir?

A. Yes.

Q. Okay. EPA's Office of Pesticide Programs -- that's the OPP; right?

A. That's correct.

10:05:11 Q. December 12th, 2017?

A. That is correct.

Q. Okay. And page 70, we can be brief on page 70, because paragraph 4 and paragraph 3 -- paragraph 4 being about Kumar and 3 about George are the same; right, as in the 2016 paper?

10:05:28

A. Yes, it's the same.

Q. Okay. Sir, I have a blue sheet put on this one, so it would be easy to find.

Will you pull that out, please, and turn to 3185? Tell me when you're there, please.

10:06:18

A. Okay.

Q. Okay. So it's just a bar chart showing some of the data from the CD-1 mouse studies, i.e., Knezevich, Atkinson, Sugimoto and Wood.

Do you see that, sir?

10:07:05

A. It's showing something. I'm not sure what it is.

Q. It's showing the maximum dose from those studies, the names of the studies, the dates of the studies, the duration of the studies and the incidence of lymphoma reported in the male mice in those studies. Does that look correct to you, based on your notes?

10:07:16

A. There's no denominator in the incidence counts. These are just numbers. I'd have to see what the denominators are, but, yes, this is what you've just

10:07:33

described.

Q. Okay. I mean, those are the -- those are the counts from which you calculate the scores. Like, the number and the --

10:07:45 A. Well, you have to use the denominator -- you have to use a number of animals in the dose group as well as the number of animals with the tumor.

Q. Okay. It was 50, 50, 50, 50 for all of these; right?

10:07:55 A. I'm not certain. I'd have to look at my notes. It varied from study to study.

Q. Okay. Are there any numbers here that aren't consistent with your notes?

A. I'd have to look at my notes to make sure your

10:08:08 numbers are correct.

MR. GRIFFIS: Move to publish Number 3185, the demonstrative exhibit, so that I may question Dr. Portier about it.

THE COURT: Any objection?

10:08:21 MR. WISNER: Yes, your Honor. It's not his demonstrative. He just said he doesn't know if these numbers are correct. I -- I don't know how we can do that.

THE COURT: All right. Counsel, can you

10:08:32 approach, please?

MR. GRIFFIS: Yes.

(Sidebar.)

[redacted]

[redacted]

(End sidebar.)

Q. BY MR. GRIFFIS: Okay, Dr. Portier, please consult your notes and see if any of the numbers on that chart are incorrect, in your opinion.

10:10:10

A. Could I have a pencil so I can make notes?

MR. WISNER: Does a pen work?

THE WITNESS: A pen would work, yes. Thank you.

MR. WISNER: (Indicating.)

10:11:32

THE WITNESS: Okay. Yes, the number's correct.

THE COURT: All right. Very well then. With that confirmation, the Exhibit 3186 may be --

MR. GRIFFIS: 3185, I believe.

THE COURT: All right. So 3185 may be published.

10:11:47

Q. BY MR. GRIFFIS: Okay. Now that the jury can see it, sir, this is -- we have the Knezevich, Atkinson, Sugimoto and Wood studies, which are the four studies in CD-1 mice; correct?

10:12:08

A. Correct.

Q. Okay. There's a little bit of information about the date and duration of the studies under here, and then the high-dose groups are shown in the studies; correct?

A. Correct.

10:12:19

Q. Okay. So, for example, the Knezevich study, the

high-dose group was dosed at 4,945 milligrams per kilogram per day; Sugimoto, 4,348; Atkinson, 988; Wood, 810; correct?

A. Correct, except for the Knezevich & Hogan, there was a disagreement on what that dose was between EFSA and EPA. EFSA put it at 4,841 minor difference.

Q. Okay. That would be like there instead of --

A. Almost the same.

Q. Okay. Nothing turns on the exact numbers in my questioning, sir.

Now, there is a red line there that says OECD dose limit of 1,000 milligrams. If there's a treatment period for animals -- this is under the OECD guidelines, which you've talked about. You haven't talked about the specific one, I think, but you've talked about the OECD guidelines in general.

If there is a treatment period of less than 14 days, then the OECD has a dose limit of 2000 milligrams per kilogram per day, and for longer studies, like all these, you have 1,000 milligrams per kilogram per day; right?

A. I don't think they call it a dose limit, but we could bring up the OECD guidelines, if you wish. This is a -- first to begin with, OECD guidelines change over the years.

Q. Right. These didn't violate them at the time --

A. Knezevich & Hogan, they were all done in agreement with the OECD guidelines when they were done. Recently OECD has said if it appears that there is not likely to be any carcinogenic finding below 1,000 milligrams per kilogram per day, even though that is not the maximum tolerated dose, companies could use that dose limit, or whatever they called it, in their bioassays. But it's not a hard, set limit.

10:14:08

10:14:27

Q. The idea of having an OECD limit is that higher doses are much less relevant to human experiments; right? Humans are absolutely never going to be exposed to levels like that?

A. I'd have to look at the OECD guidelines and see what justification they used.

10:14:45

Q. Okay. I'm going to approach and hand this to you. Take a look at the front cover first. This is an August 31, 2015 guidance document on revisions to OECD genetic toxicology test guidelines.

10:16:16

A. Okay. All right.

Q. Okay. And is this applicable to animal studies, sir?

A. This is not their carcinogen guidelines.

Q. Okay.

10:16:33

A. So this is genetic toxicology. This is relevant

to tests for DNA damage.

Q. Okay. Let me come back to this then.

Tell me again what the OECD guideline for animal carcinogenicity of 1,000 is.

10:16:51 A. I have that in my notes. Would you like me to find the reference?

Q. Yeah, sure.

A. My references, my full set of references are not in here.

10:18:00 Q. Okay. Well, I don't want to hold us up. It's not a big deal, sir.

A. It has a number 245, OECD guideline 245 or something like that.

Q. Okay. When you give a dose that is worrisome

10:18:13 about cytotoxicity, will you please tell the jury what that means?

A. The dose can kill cells.

Q. Okay. And what is the problem with giving doses high enough to cause cytotoxicity when you're looking for

10:18:30 genotoxicity, when you're trying to assess whether a substance is capable of causing DNA damage?

A. So we're going away from the animal bioassay discussion now back towards the mechanistic proposal?

Q. We are, yes.

10:18:41 A. Okay. So in a petri dish, when you dose the

cells that are just in a little dish, if the dose is too high, it will kill the cells and you won't be able to see DNA damage.

If it's only killing some cells, the killing -- the damaging -- the cytotoxicity, the cell killing, there's a lesser version of that that can damage DNA. And so you can get a false reading if there's cytotoxicity. And the same holds true for oxidative stress.

10:18:57

10:19:15

Q. Okay. The Knezevich and Sugimoto studies, the two older studies, have doses much, much higher than Atkinson and Wood; correct? Over four times higher?

A. Well, the studies are in order of year. So Sugimoto is not that much older. In fact, it's quite a bit younger than the Atkinson study, but it has higher dose.

10:19:39

Q. And in Sugimoto, with this dose, the trend that you observed is due to the high-dose group; correct? 2206?

10:19:56

A. Probably, yes, but I -- yeah, I'm sure it is.

Q. Okay.

A. But the two and two there I think are extremely close. The control dose was -- this is Sugimoto; right?

Q. That's right.

10:20:13

A. Right here. So the control dose was zero, of

course. The next dose was 165. The dose after that was 838, and the high dose was 4348.

So the fact that the two twos are very close to each other is what -- it also contributes to the trend static here.

10:20:35

Q. Okay. And there's a little bit of strange dosing going on at the bottom end, too?

A. The distance between the control and the lowest dose is substantially smaller by an order of, what, 10, 20? It's 20 times closer to control than it is to the high dose. So it's -- in essence in a statistical analysis, it's almost a control.

10:20:48

Q. Lymphoma is a common spontaneously occurring neoplasm in mice; right?

A. Not in all mice. The historical control rate in the 18-month studies was .26 percent. So that's like three tumors in every thousand animals.

10:21:04

Q. Would you turn to 2320 in your regulatory binder 1, please.

And these are PCHA ECHA findings from 2017; correct?

10:21:33

A. Yes, that's what it looks like.

Q. Okay.

MR. GRIFFIS: Ask for permission to publish this to the jury so they can follow along.

10:21:54

THE COURT: I'm sorry, is this Exhibit 2320?

MR. GRIFFIS: It is 2320 in regulatory binder 1, the ECHA PCHA report from that year.

THE COURT: All right. Any objection to publication?

10:22:12

MR. WISNER: Your Honor, could we have a quick sidebar about something?

THE COURT: Yes.

(Sidebar.)

10:22:31

[redacted]

10:22:45

[redacted]

10:22:54

[redacted]

10:23:08

[redacted]

(End sidebar.)

THE COURT: All right. You may continue, Mr. Griffis.

Q. BY MR. GRIFFIS: Yes. Page 38, sir. Are you there?

10:23:32

A. Yes, I am.

Q. Okay. Now, this is where ECHA is going through various tumor types and discussing their reasons for their ultimate conclusion that glyphosate is not a human carcinogen. And at the bottom of 38, they're talking specifically about malignant lymphoma; right?

10:23:45

A. Correct.

Q. And the first sentence is: "In mice lymphoma is a common spontaneously occurring neoplasm." Right?

10:23:59

A. That is what it says.

Q. Let's go over to 41. This is where they are summing up. Are you there?

A. Yes.

Q. Okay. Top of the page, they say: "The biological and human relevance of the findings" -- and we're talking about mouse malignant lymphoma findings -- "is uncertain for the following reasons."

10:24:18

Correct?

A. That's what it says.

Q. Okay. "One, the maximum incidences are regarded

10:24:31

to be within the historical control range for the CD-1 mice although adequate historical control data were not available for all studies."

That's what they said; right?

10:24:46 A. That's what it says.

Q. Okay. And "maximum incidences" means the high numbers, like five in that one, six in that one, six in that one, five in that one. And when they say they're within the historical control ranges, they mean that when

10:25:03 control mice in multiple experiments over time were looked at, they had similar numbers; correct?

A. I'm sorry, could you say that again?

Q. Yes, sir. I'm just asking what the sentence -- asking about what the sentence means. They say the

10:25:21 maximum incidences were regarded to be within the historical control range for the CD-1 mice.

And what that means is that these numbers are within the historical range of just spontaneously occurring malignant lymphomas in CD-1 mice, untreated

10:25:39 mice; right?

A. They give no references for that statement.

Q. It's what the sentence means, though; right?

A. I guess that's what it means, but they give no references. How can I judge the quality of the

10:25:54 statement?

Q. Two. I'm reading the second item. "The increases in malignant lymphoma incidences appear to be confined to the high dose groups in the CD-1 mice."

A. That's what it says.

10:26:07 Q. "Three: The incidence of malignant lymphomas is known to be related to the age of the animals."

And you were telling us yesterday that for many cancers, maybe all cancers, the older the animals get, the more cancers you would expect; right?

10:26:20 A. Correct.

Q. "However, significant associations between exposure to glyphosate and induction of malignant lymphomas were not observed in the 24-month studies. Furthermore, there was no reduction in overall survival 10:26:33 in the exposed groups."

Correct?

A. That's what it says.

Q. And: "Four, no parallel increases were observed in female CD-1 mice."

10:26:43 And we talked earlier about how we're seeing this in males and not in females; right?

A. No, I don't remember us talking about -- well, yeah.

Q. You had identified --

A. In males, correct.

Q. But one would expect to see it in females just by chance alone; right?

A. No.

Q. Because females are more likely to develop spontaneous malignant lymphomas than male mice; right?

A. I would have to look at a historical control data set to answer that question.

Q. Okay. Let's talk about hemangioma now. You can take this slide down.

A. I will repeat again that this is without reference, except the last one, which you didn't read the reference.

Q. Would you like to?

A. I'll be happy to read the whole -- the whole thing through.

Q. After where I stopped reading. It says Son and Gopinath, 2004 ASB2015-2533. That's the reference?

A. That's the reference. It says: "Is known that female CD-1 mice are usually more prone to develop spontaneous malignant lymphoma than male mice," giving that reference. "The lymphoma incidences were generally higher in females than in males, but no glyphosate variant increases were seen in female CD-1 mice."

Now even though tumors increase with age, your ability to detect them may not increase with age. A

small increase that you see at 18 months could easily disappear at 24 months because all of the sudden you've got a lot more spontaneous tumors.

And so the noise gets bigger and the statistical p-values are harder to see a significant increase. In other words, a fivefold increase from zero to five -- take the Wood example. From zero to five in Wood at 18 months, at 24 months, even if it's still the same numbers, it could be 10 to 15, which is harder to pick up statistically.

The absolute climb is not -- is not the only thing that drives a statistical test.

Q. Okay, sir. I want to talk about the other three tumors that you identified that we haven't talked about yet today.

First let's talk about hemangiomas.

A. Okay.

Q. And you reported that in -- you detected that with your statistical analyses that you ran in the Greim data and gathering information from EPA, EFSA, ECHA, et cetera, in female mice and not in the males; correct?

A. The hemangiomas, that is correct.

Q. That's a benign tumor; right?

A. Again, it's -- it's the same as the other one. It's typically benign. And I don't know of it's

malignant counterpart. So from my knowledge, it's a benign tumor.

Q. Okay. It's those little red -- I mean, in humans, you can see it most easily in those little red dots you can get on your skin, and as you get older, they can appear overnight for no apparent reason, and your dermatologist says don't worry about it?

A. I don't know.

Q. I have two right there (indicating.)

And you said that this showed -- this was in Sugimoto. One study was in the Sugimoto study; right?

A. No. Atkinson also.

Q. Okay.

A. No, that's a hemangiosarcoma. I'm sorry. I'm looking at the wrong one. Yeah, Sugimoto.

Q. Okay. One study, and it's the Sugimoto study; right?

A. Correct.

Q. And that's one of the studies that's a relatively high-dose study compared to the others here; right?

A. Yes.

Q. Okay. Let's move on to hemangiosarcoma. This is a very common tumor in -- first of all, this is in the male mice, not in the females; right?

A. I'm sorry, I'm still trying to make sure that we didn't make a mistake up there with the one hemangioma.

Q. Oh, there it is in Kumar. We did, sir. Here it is in Kumar.

10:31:06 A. Right.

Q. So it's in the Swiss Albino Kumar study. That's the one we were talking about earlier that -- EPA said in the OPP report, there was a virus, and that's why they weren't looking at it?

10:31:21 A. That's what they said.

Q. Okay. And ECHA did look at it in its 2017 evaluation, and it reached the ultimate conclusion that there aren't any significant patterns here that point towards carcinogenicity; right?

10:31:34 A. I would have to read their report to see exactly what they said.

Q. Okay. It's what we were just looking at. They were considering Kumar in that group, do you know? Do you remember?

10:31:45 A. Again, probably, but I'd want to look.

Q. Okay. The hemangiosarcoma then, Atkinson, Sugimoto and Kumar. And Atkinson is the study that you described as limited; right?

A. Correct, because of the way they did the

10:32:03 pathology.

Q. Right. Sugimoto is the high dose?

A. But let me be clear.

Q. Yes.

A. That doesn't pertain here because hemangiosarcomas are -- are tumors that you find by inspection. And so the fact that they didn't do histopathology on every animal isn't affected by that because they did histopathology on every blood tumor they found.

10:32:28 And so the denominator there is all the animals as compared to some of the other cases where the denominator is much smaller because they look at all the animals.

Q. Okay.

10:32:38 A. So it doesn't affect this particular finding.

Q. Okay. Does it affect that one?

A. No. Same issue.

Q. It's not limited for purposes of this chart?

A. Correct.

10:32:46 Q. Okay. Sugimoto is a relatively high-dose one and Kumar is the one that EPA considers to be a virus issue; correct? But was considered again by ECHA and others and found by them not to be a problem?

A. It was two -- that was a very compound question.

10:33:07 Q. Okay. The regulators we've talked about, like

ECHA and EFSA and BfR and EPA, disagree with your evaluation of these studies; right?

A. Let me think. Have I ever gotten anything back from them that said they disagree with me? Certainly they reached a different conclusion, if that is the question you're asking.

10:33:24

Q. All right. And I showed you -- the few things that I showed you, I don't think you agreed with anything we put up on the screen; right?

10:33:34

A. That's, again, too broad of a question.

Q. Okay. Let's talk about kidney adenoma carcinoma.

A. Okay.

Q. Again, male mice, not females; right?

10:33:51

A. Correct.

Q. Okay. And adenomas are benign. They can transform but don't necessarily transform into carcinomas, which are not benign; right?

A. True, but unlike an angioma, if I were running a two-year bioassay and I saw kidney adenomas, I would almost certainly consider that a malignant finding because they're -- they're almost certain to go onto carcinomas at some point.

10:34:08

Q. Adenomas are more -- kidney adenomas are more alarming than hemangiomas are?

10:34:20

A. Correct.

Q. Okay. For you this was a -- well, let's skip that.

For all of the tumors in the mouse chart and the rat chart, the original researchers didn't find these to be compound related for various reasons; correct?

10:34:40

A. I don't know because I do not have the reports of the original researchers. Those are proprietary and have not been presented to me. I know that from ECHA's -- from EFSA's response to me, I know some of them were actually found. I don't know why they weren't included.

10:34:58

Q. The George study, sir, this is -- we discussed it briefly because we were looking at what OPP said about not considering it, and this is the one where skin papillomas appeared in the study, and you said they are a benign tumor, and you said that you're interpreting it as a papilloma finding and you're using it to give an indication of some of the mechanistic underpinnings of this particular chemical. That's right?

10:35:16

10:35:36

A. Again, very compounded sentence.

The George study is of a glyphosate formulation, and I'm interpreting it as saying that glyphosate can act as a promoter.

Q. Okay. And you said it's a papilloma finding,

10:35:58

not a carcinoma finding; right?

A. That's correct because they didn't do any histopathology to determine if there were skin carcinomas in that. But since carcinomas arrive from papillomas, regardless of what the bump was, there was a papilloma there at one time.

Q. Start with 3183 -- or wait a minute. Slide 316. This is slide 3183 in your binder, sir. Slide 316 for us, I believe.

You know, we talked on Friday about your being very critical of the scientific evaluations, methodologies of a number of national and international agencies; right, sir?

A. I don't believe I criticized the methodology of JMPR.

Q. Yes, sir. I want to get to JMPR. I do want to ask you about that.

A. That's the only international one.

Q. Okay.

A. The two I have criticized are the EFSA and EPA reviews. I think I sent criticism to ECHA as well.

Q. And by "criticism," I don't just mean -- I mean in your heart. I mean you sent them a letter. Let's put it up.

You said EPA was so amazingly wrong, EFSA was

astonishing and so amazingly wrong, their analysis was totally illogical. ECHA got one -- you pointed out one thing they got right. I forget what it was, but they got one thing right.

10:38:13 A. Historical control usage.

Q. But otherwise, it was kind of the same as the other ones.

BfR was basically the same as ECHA and EFSA in their wrongheadedness about this carcinogenicity issue,

10:38:25 and JMPR is what I want to ask you about. That's Joint Agency of World Health Organization and the UN Food and Agricultural Organization, and you said that they were focused on -- tell me if I don't have this quite right. They're focused on food exposure so it may be that if I

10:38:42 showed you their exact wording in the conclusion, you might agree with them on glyphosate not being a human carcinogen via that route of food exposure.

Is that what you said?

A. I don't know if I said that, but I certainly

10:38:55 wanted to see the exact wording. But was there a question on this picture here?

Q. You've answered them.

A. I didn't answer all of them. I'm not sure you've --

10:39:05 Q. Well, for BfR --

A. -- gave me an opportunity to answer a question on this. You just stated -- you criticized them and then threw it up there, but you have yet to ask me what my criticism is.

10:39:18 Q. No, I didn't ask you that.

A. Okay.

Q. I said -- because we went over that at some length on Friday.

A. No, we didn't.

10:39:23 Q. You have got a greater length than that?

A. No, but I would like to summarize the five points that make them all wrong in the way they did their evaluation.

Q. EPA you said is so amazingly wrong. EFSA, ECHA, 10:39:38 BfR, you're pretty much lumping them together, with the exception of ECHA doing the historical controls right; correct?

A. Correct.

Q. Go ahead and tell us your five.

10:39:48 A. Improper use of historical controls. They -- we just read it. Within the range of historical controls. We read that statement for EPA. That's just an incorrect way of analyzing the data, and that's about 30 percent of the tumors that they discarded because of that incorrect assumption.

In fact, even if you look at the OECD guidelines, the EPA guidelines, and the IARC guidelines, they all warn against using that approach, and yet it rejected 30 percent of the tumors.

10:40:19 Q. They are getting their own guidelines wrong, in your view?

A. It's absolutely clear they're getting their own guidelines -- they're not using them appropriately. I like the guidelines; they just aren't using them.

10:40:32 The second thing they require is that as the dose increases, the tumor incidence must increase or at least not go down. And so what they say is that -- the wording they use is that there's non-increasing dose response or not a clear dose response, I think is what

10:40:51 they used. That throws out a bunch of them.

The next one is no precursor lesions. Now, that doesn't pertain to malignant lymphomas, hemangiosarcomas, and hemangiomas because I know of no precursor lesions for those. But even then, there's good reason to believe

10:41:13 that doesn't have to happen or even if it's happening, you're not observing it. And I would -- that's a much more detailed difficult explanation.

The next one after that, fourth one.

Q. I promised Mr. Wisner I would get you home

10:41:36 today.

A. It's okay.

Q. Tell me if you want.

A. You've got the most important three. Thank you.

Q. Binder 3 of the -- regulatory binder 3 has the JMPR statement. You wanted to see that. Are you there?

10:41:48

A. Yes, I am.

Q. Okay. So this is the 2016 report of the JMPR, and on Friday you told me that you might possibly agree with them if you could see their exact conclusion. So on page 24 -- are you there?

10:42:36

A. Yes, I am.

Q. There's a -- there are two paragraphs, the first one starting "in view of the absence of carcinogenic potential."

10:42:51

Do you see that?

A. No.

Q. Okay. It's the third from the last paragraph in the top section on page 24. And I'm going by the page numbers of the original document, sir, in the upper left-hand corner.

10:43:09

A. Okay. "In view of the absence." I have it.

Q. Okay. "In view of the absence of carcinogenic potential in rodents at human relevant doses and the absence of genotoxicity by the oral route in mammals, and considering the epidemiological evidence from

10:43:23

occupational exposures, the meeting concluded that glyphosate is unlikely to pose a carcinogenic risk to humans via exposure from the diet."

A. Okay.

10:43:42 Q. Okay. So I read that correctly?

A. You read it correctly.

Q. Do you agree with it?

A. No. The absence of genotoxicity by oral route in mammals is incorrect.

10:43:53 Q. So you would add JMPR to the agencies you disagree with?

A. For a different reason.

Q. What's the different reason?

A. You asked me about this one paragraph, which is

10:44:03 all I'm commenting on, and the -- this one paragraph, they talk about the absence of genotoxicity by oral route in mammals, which I disagree with.

I haven't studied every other bit of their evaluation --

Q. Okay.

A. -- to be able to tell you if they mess up controls, et cetera.

Q. Fair enough. I mean, when we've been talking about EPA and ECHA and EFSA, BfR, you've had lots of

10:44:33 criticisms immediately, and you're intimately familiar

with the documents. That's not true for JMPR; is that fair to say?

A. That's correct. That's not true for JMPR.

Q. All right. Let's take a look, sir, at the -- at OPP's bottom line, the Office of Pesticide Programs at the EPA. That's Exhibit 2481.

10:44:53

A. Okay. Where am I looking?

Q. 01, page 140. You can put this up on the screen. 2481, page 140. And I just want to look at the first paragraph on the page.

10:45:39

MR. WISNER: Your Honor, objection. Violates the evidence code. But since it's already on the screen, just let him proceed.

THE COURT: Very well.

10:45:59

Q. BY MR. GRIFFIS: We do have a ruling on this.

"Overall there is not strong support for the suggestive evidence of carcinogenic potential cancer classification descriptor based on the weight of evidence, which includes the fact that even small non-statistically significant changes observed in animal carcinogenicity and epidemiological studies were contradicted by studies of equal or higher quality. The strongest support is for not likely to be carcinogenic to humans at the doses relevant to human health risk assessment for glyphosate."

10:46:14

10:46:32

And do you disagree with the Office of Pesticide Programs on that, sir?

A. Oh, yes, I do.

Q. Page 131. I'd like to call your attention to the bottom paragraph on that page.

"Overall there is remarkable consistency in the database for glyphosate across multiple lines of evidence. For NHL" -- which is non-Hodgkin's lymphoma -- "observed associations in epidemiological studies were non statistically significant and were of relatively small magnitude. Chance and/or bias cannot be excluded as an explanation for the observed associations."

Then they talk about all other cancer types, which is not what we're here for. Skip that.

"Across species strain and laboratory, tumor incidence was not increased at doses less than 500 milligrams per kilogram per day, except the testicular tumors, which were only seen in one study. Observed tumors were not reproduced in other studies, including those conducted using the same strain at similar or higher doses. The genotoxicity studies demonstrate that glyphosate is not directly mutagenic or genotoxic *in vivo*."

And you disagree with that statement by the Office of Pesticide Programs, sir?

A. Yes.

Q. Who is Dr. Jose Tarazona?

A. He is the -- I think the head of the Pesticide Unit at EFSA, I believe.

10:48:32 Q. Did I pronounce his name right?

A. Probably.

Q. I thought you'd know. This was a subject of -- and he and you wrote an article together.

A. No. We wrote separate articles to the same

10:48:49 weekly science magazine or monthly science magazine.

Q. It was like a little debate between the two of you?

A. Correct.

Q. And we talked about that on Friday. I guess you

10:48:59 didn't sit down together to do that. You just both sent it in -- sent in your suggestions?

A. That's correct.

Q. And we talked about -- we saw in the opening statement of Mr. Wisner and on your direct examination a

10:49:11 published article by you talking about the differences between EFSA's evaluation and IARC's evaluation; right? That's your August 2016 published article with --

A. The 96 -- 96 scientists article?

Q. Yeah, that one.

10:49:29 A. Yes.

Q. And Dr. Tarazona also wrote and published an article on the differences between IARC's assessment and EFSA's assessment; correct?

A. I don't know if that was the focus -- if he did, I don't know what article you're talking about.

Q. Okay. Take a look at 3039 in your blue binder.

(Interruption in proceedings.)

THE WITNESS: Okay.

Q. BY MR. GRIFFIS: This is an article in the Archives of Toxicology from April 2017 by Dr. Tarazona and a number of his colleagues at EFSA, the pesticides unit; correct?

A. That is correct.

Q. Okay. And one of the co-authors is from the BfR; right?

A. Yes.

Q. Okay. And the title is "Glyphosate Toxicity and Carcinogenicity, a Review of the Scientific Basis of the European Union's Assessment and Its Differences With IARC."

Right?

A. That's what it says.

Q. There's a table on the third page comparing IARC and EU's regulatory assessments -- well, their relative roles and the assessments that they made on glyphosate;

right?

A. There's a table. I read this article. I just didn't realize they spent any time at IARC.

Q. Okay. So you're familiar with the article?

10:51:28 A. I wrote a comment to the -- to the article.

MR. GRIFFIS: I move to publish this article.

MR. WISNER: No objection, your Honor.

THE COURT: Very well. This may be published.

Q. BY MR. GRIFFIS: Let's go to page 1 first.

10:51:42 THE COURT: And Mr. Griffis, before we get too deep into this article, we do need to take the morning recess at some point. Should we do that now?

MR. GRIFFIS: Sure, we can do it now.

THE COURT: Okay.

10:51:52 All right, Ladies and Gentlemen, we're going to take the morning recess now. We'll be in recess for 15 minutes and resume again at 5 after 11:00 on the wall clock. All right? Thank you.

(Recess.)

11:07:14 THE COURT: Welcome back, Ladies and Gentlemen.

Dr. Portier remains under oath, and, Mr. Griffis, when you're ready, you may proceed.

MR. GRIFFIS: Thank you, your Honor.

Q. So, Dr. Portier, we were starting to discuss the

11:07:26 article by Dr. Tarazona and his colleagues at EFSA and

BfR on glyphosate toxicity and carcinogenicity, and the EU's evaluation thereof; right?

A. We were reviewing this article, yes.

Q. I'd like to start out with the abstract, and the second sentence of the abstract starting, "Since glyphosate was introduced in 1974."

It reads: "Since glyphosate was introduced in 1974, all regulatory assessments have established that glyphosate has low hazard potential to mammals. However, the International Agency for Research on Cancer, IARC, concluded in March 2015 that it is probably carcinogenic. The IARC conclusion was not confirmed by the EU assessment or the recent joint WHO FAO evaluation," and that's a reference to JMPR; right, sir?

A. You're just talking about WHO?

Q. Right, WHO FAO.

A. That is the JMPR.

Q. Okay. "Both using additional evidence. Glyphosate is not the first topic of disagreement between IARC and regulatory evaluations, but has received greater attention."

Have I read that correctly?

A. Yes.

Q. And do you agree with it?

A. I don't know whether all regulatory assessments

have established that glyphosate has low hazard potential.

Q. Okay.

A. But the rest is just statement of fact.

11:09:15 Q. We don't know if he's referring beyond EFSA, ECHA, BfR and JMPR, but we've talked about all of those, and they certainly found that glyphosate has low hazard potential to mammals and concluded it wasn't a human carcinogen; right?

11:09:34 A. I don't know about low hazard potential to mammals.

Q. Okay.

A. That's the other endpoint that they evaluated. I have not spent time looking them over carefully.

11:09:44 Q. Oh, I see. So you're focused on the toxicity endpoints when you -- when you make that statement?

A. Low -- where's the statement again?

Q. Well, it says, "low hazard potential."

A. Low hazard potential there is not just cancer, 11:10:00 as far as I'm reading this. He's talking about everything.

Q. Okay. And just so that we know what you're talking about, this -- we're mostly interested in carcinogenicity in this case and not acute toxicity, but 11:10:11 there's all sorts of testing and evaluations that go on

about acute toxicity, like whether a substance causes eye irritation, whether it makes you sick to your stomach if you swallow it, whether it causes rashes if you get it on your skin, whether it makes mammals or humans acutely ill if they drink too much of it. That sort of thing; right?

A. And affecting immune system, affecting development, affecting reproduction.

Q. Right. So this next sentence I'm going to read is about long-term toxicity/carcinogenicity. It's one sentence down. "Use of" -- starting: "Use of different data sets, particularly on long-term toxicity/carcinogenicity in rodents, could partially explain the divergent views, but methodological differences in the evaluation of the available evidence have been identified."

And, sir, we've talked at some length about the difference in the data sets that the Working Group 112 came up with conclusions about two mouse studies, because it didn't have available to them even the Greim tables or didn't have available to them enough time to review the Greim tables that you spent more than six months reviewing, didn't have, certainly, individual animal data, whereas these regulators did have all of that; correct?

A. That's a correct statement of fact, yes.

Q. Okay. Turn to the third page of the article, please.

I'd like to look at the first column towards the bottom, starting with the sentence: "Regarding data sources." Let's get a little deeper into the data sources. "Regarding data sorts" --

A. I don't know where you are.

Q. Okay. It's the last two sentences of that long paragraph right there.

A. I've got it.

Q. "Regarding data sources, IARC assessments are primarily based on published evidence, i.e., scientific publications and regulatory assessments," and we saw that the other day when we were looking at the Monograph on the mouse studies. There was -- reference after reference was to EPA, was to the EPA violations; correct?

A. And one was to JMPR.

Q. Yes. So scientific publications, meaning things that are in the published literature and regulatory assessments.

"Industry-sponsored studies are used when reviewed and reported in regulatory evaluations, becoming a relevant secondary source for regulated agents such as pesticides."

And then he goes on to talk about what the EU

looks at. "Both scientific publications and mandatory industry-sponsored studies were primary sources in the EU evaluation."

And that's a correct description of the difference in data sources between the two; right?

A. Yeah, that's a pretty adequate description.

Q. Okay. Let's go to page 5, and I am under the section "Carcinogenicity in Animals" in the right-hand column, "Information Sources." And about -- on the second sentence it talks about, "Two additional published studies on glyphosate formulations," and it mentions the George study and a study Seralini. It says, "These were considered inadequate by IARC and EFSA for carcinogenicity assessments." And it cites EFSA 2012 and IARC 2015?

A. That's what it says, yes.

Q. And then the next -- and then we go on, "Consequently, industry-sponsored studies required by several jurisdictions worldwide have constituted the basis for the assessment of animal carcinogenicity by both IARC and EFSA. As expected for regulatory assessment, EFSA assessed the original study reports. According to their principles, IARC used unpublished studies based on secondary sources, i.e., the information on the studies as published by JMPR and the US EPA."

Just like you were telling us a few minutes ago; right?

A. Yes.

Q. Turn to page 6, I'm in the right-hand column, the second paragraph, first sentence. "Due to the large
11:15:09 number of studies, the assessment of chance results is particularly relevant."

Do you see that sentence?

A. Yes.

Q. And we've been talking, sir, about multiple
11:15:19 testing, and this sentence raises the issue that when you have 12 well-designed studies that are good enough for carcinogenicity assessment, rather than the regulators' required two, which you would normally have or often have when evaluating a substance, you have a bigger multiple
11:15:41 testing problem to overcome than you would with just two; right?

A. I'm sorry. That was a long question.

Q. Okay. We've talked about the notion that looking at animal studies that are looking at multiple
11:16:00 organ systems involve many, many tests, and you could get false positives in any or most of those tests, and that needs to be addressed in some fashion; right?

A. Yes, correct.

Q. Okay. Well, that problem gets bigger when you
11:16:14 have a whole bunch of animal studies instead of just two;

right?

A. No. The problem is the same. No matter how many studies you have, the problem is the same. It doesn't get bigger. It's just you'll have more tumor sites that arise, and some of those are more likely to be false positives.

Q. You'll have more false positives in your group that you're looking at and trying to assess; correct?

A. You are likely to have more false positives.

Q. Statisticians always say that.

A. You never know whether you have false positives or not.

Q. Statisticians always say that, because it's conceivable that we could have all those studies and just one positive or zero positive, although it's really, really, really, really unlikely. Generally speaking, the more studies you have, the more false positives you have to deal with. That's accurate; right?

A. That is accurate.

Q. Okay. Turn to page 9, please. And again, right-hand column, starting with the first sentence in the second paragraph. We're talking about animal studies here, sir. "Excessive toxicity, for instance toxicity at doses exceeding the maximum tolerated dose, can cause effects such as cell death, necrosis with associated

regenerative hyperplasia, which in turn can lead to tumor development as a secondary effect, unrelated to the intrinsic potential of the substance itself to cause tumors at lower and less toxic doses."

11:17:52 Did I read that right?

A. Yes, you did.

Q. Okay. And that's talking about the general principle that we discussed briefly earlier, that when doses get too high, you can lose the ability to detect

11:18:06 what the chemical is actually doing to genes, because you're starting to get gross cellular damage that is generating tumors by ways that are not of concern in carcinogenicity assessment; is that right?

A. Not -- not really. That's not what this is

11:18:25 saying, because it has nothing to do with the genetics. You can kill cells without damaging the genetics.

Q. Yes.

A. This is a theoretical statement.

Q. Right. You could induce tumors with a substance

11:18:40 that only irritates cells, has no carcinogenicity, doesn't damage DNA, doesn't cause oxidative stress, doesn't do -- doesn't by any mechanism cause cancer, there are multiple tests that shows it doesn't cause cancer in humans or animals, but if you put enough of it

11:19:01 on a group of cells or injected it into peritoneal, into

someone's abdomen cavity, you can do enough damage to cells and cause enough irritation that they become acutely ill and/or develop cancer because of that gross insult to their body; right?

11:19:15 A. It's theoretically possible. It does not always work that way. There's no guarantee, and you would know it, because you would see the hyperplasias in the tissues, and so you would see an increase in hyperplasias at doses that were producing that type of effect.

11:19:34 Q. You'd see sick animals; right?

A. Not necessarily, but you'd see -- hyperplasias is the cell tissue looks inflamed, like you would get with lymph nodes growing bigger in your neck or something like that.

11:19:55 Q. Let's go down a little farther. "It has been suggested."

"It has been suggested that almost all chemicals, including those non-genotoxic and without structural alerts for carcinogenicity" -- I should have

11:20:09 just read that instead of tried that last sentence I told you -- "would produce statistically significant trends if testing at or above the maximum tolerated dose in a sufficient large number of animals."

Would you agree with that, sir?

11:20:34 A. I agree that Gaylor said that and suggested it.

I don't agree with the suggestion.

Q. You don't agree with Gaylor on that?

A. I don't agree with Gaylor -- Dave Gaylor that this is going to happen commonly, which is, I think, the tone of the sentence.

Q. Okay. Let's keep reading.

"Significant trends for tumor induction" -- and now we're talking about the actual glyphosate results. "Significant trends for tumor induction were observed in two mouse studies, but only at very high doses, well above the proposed top dose for carcinogenicity studies, OECD 2012, of 1,000 milligrams per kilogram body weight per day. Clear indications of toxicity were observed at these high dose, such as reduced body weight, pathological changes in the bladder and liver and other toxic signs. Consequently, the tumor induction trends were considered confounding effects due to excessive toxicity."

Did I read that correctly?

A. That's what it says.

Q. Okay.

A. That's not what it says in the EFSA document, but that's what he wrote here.

Q. On page 11, sir, we have a large table that I will not go into in detail, as we haven't got enough

time. "Comments on IARC assessment," is the right-hand column, and I'd like to look at Point E, which is question, "Whether responses are in single or both sexes," and that is something that investigators consider when evaluating animal carcinogenicity studies; right? It's the criteria?

11:22:20

A. If you see a tumor significantly increased, biologically increased in two sexes in the same study, it adds strength to the finding.

11:22:35

Q. And the comments on the IARC assessment with regard to that issue, whether responses are in single or both sexes, says, "All trends were significant only in one sex, but no sex-mediated mode of action is discussed."

11:22:49

What's a sex-mediated mode of action?

A. For things we understand, there are tumors that arise that are related to specific hormones. For example, you would -- or specific tissue types. You could see testicular tumors in males, but, of course, never in females. And in humans, women get breast cancer much more readily than males do, even though they both have breasts, and that has to do with hormonal levels, but, of course, there are many, many examples of chemicals that only cause tumors in one sex, and there are no sexually-related issues that anyone understands

11:23:11

11:23:28

why it happens.

Q. Okay. Do you have any explanation for why tumors show up in -- primarily in males and not in females in the tumors that you've flagged for

11:23:49 significant?

A. Let me think them through for a minute.

There could be some explanations in terms of the spontaneous rates in these controls, but if you're looking for a mechanistic consideration that would say

11:24:07 why females didn't get malignant lymphomas and males did, I don't have that explanation.

Q. Okay. The tumors that you identified are not like testicular or breast cancer, et cetera, in that they are ones you considered important and that they are

11:24:21 sex-linked; correct?

A. There's no indication of a sex-linked mechanism within these tumors that I've looked at.

Q. Okay. Last item in this article, sir, is on page 18, the last page of text.

11:24:38 It's the last paragraph in the middle, starting, "From a health assessment perspective."

Do you see that?

A. Page 18.

Q. Yes, right there (indicating).

11:24:54 A. On the right-hand side.

Q. Yes.

"From a health assessment perspective, the IARC EFSA scientific divergence is at lower dose levels that are in reality of limited, if any, relevance. The toxicological reference cited proposed by EFSA provide a margin of protection of about four orders of magnitude for the trends in tumor induction and genotoxic damage at toxic levels reported by IARC. Those effects are expected only in concomitance with other signs of toxicity and at exposure levels orders of magnitude higher than the toxicological reference values recommended by EFSA.

Do you have any disagreement with that, sir?

A. I'm a bit confused by what he's stating here, since by European law he's not talking about carcinogenicity here. He's talking about something else. In European law, if a pesticide is a hazard for cancer, you don't calculate risk. It's banned.

Q. Okay.

A. And here he's talking about risk and order of magnitude safety. He can't be talking about cancer.

Q. Okay. Genotoxicity. You talked about your agreement with Working Group 112's conclusion that there is strong evidence of glyphosate and glyphosate-based substances being genotoxic and having oxidative stress;

correct?

A. Correct.

Q. Now, there are ten mechanisms of carcinogenesis that IARC considers to be relevant; right?

11:26:46 A. No. IARC -- there's a list of ten characteristics of carcinogenesis that are the ways in which chemicals can start tumors, but those ten are for categorization, and there are others probably that just didn't get -- didn't get captured in those ten

11:27:09 categories.

Q. Okay. Well, there's a document that you and others have worked on that -- the ten key characteristics of carcinogenesis. Is that --

A. Correct. We were trying to find groupings of

11:27:21 mechanistic data that would allow us easier to review it.

Q. Okay. And those are the ten that Working Group 112 applied to its analysis and other Working Groups applied to their analysis. They said, "Let's look at the mechanistic data and see what we find in those ten

11:27:38 categories"; right?

A. Correct. Although, to be fair, genotoxicity, oxidative stress have always been categories for the last 25 years.

Q. Okay. Sure. You didn't make up these ten?

11:27:49 A. The other -- no.

Q. You just wrote an article and listed the ten and said this is what we're going to look at?

A. Correct.

Q. Okay. And two of them were what Working Group 112 considered to be significant, genotoxicity and oxidative stress?

11:27:58

A. Correct.

Q. Not, for example, immune mediation?

A. No data.

Q. No data; right?

11:28:07

So genotoxic doesn't mean mutagenic; right?

A. No, it does not.

Q. Okay. And mutagenic would mean causing cell mutations; right?

A. On -- yes. Mutagenic would mean that you had a -- you have DNA, DNA has a sequence, and mutation would mean that the sequence of DNA and every other cell in the body is different than this one, so that's a mutation.

11:28:20

Q. And that's one of the steps in cancer, that something leads to a mutation and needs to be the right kind of mutation, because we have all sorts of mutations in our bodies all the time, but if it's the kind of mutation that makes a cell not take itself out of commission after a while but continue to be immortal or to reproduce rapidly and create many more of itself,

11:28:41

11:28:57

then ultimately that can lead to trouble if the body's many defensive mechanisms against that sort of thing don't work; right?

A. Boy, there was a lot there.

11:29:11 Q. Was I right anyway?

A. I'm not sure. I know you're not totally correct.

Q. Okay. How so?

A. A mutation is generally necessary for the

11:29:22 formation of a cancer, at least that's been our theory for the last 50 years. However, there now is an epigenetic literature out there, so that means outside of genetic material, that has been arguing certain tumors may actually arise by turning on a gene using other

11:29:45 things than the gene itself, but the sequence is there, that are only there early in life and should be turned off later in life, and inappropriately get turned on, and that leads to growth that shouldn't be there, and it gets a tumor, so --

Q. Okay.

A. -- but typically, most people think it's -- the mutation theory is most of the cancers out there.

Q. Right. Is epigenetic one of the ten key characteristics?

11:30:08 A. Yes, it is.

Q. And IARC didn't find that --

A. No data.

Q. -- in glyphosate; right? No data.

We've talked about cytotoxicity and how just irritating the cell, damaging the cell, insults to the cell, can cause genetic damage that's unrelated to genotoxicity, which is a separate concept; right?

11:30:18

A. It can cause the tissue to be inflamed, yes, and that's independent of the DNA damage.

Q. And what you're doing when you're looking for key characteristics, when you're looking for mechanisms, is finding ways in which this substance might do something to cells that might lead to cancer; is that fair?

11:30:43

A. But you're looking at the consistency and strength of that literature in supporting a cancer finding.

Q. Right. It could not alone show that something causes cancer?

11:30:56

A. That is correct.

Q. And that's because --

11:31:06

A. At least my understanding to date. That would be my opinion.

Q. Okay. And that's because you're just seeing, oh, here's a pathway by which that might work. We don't

11:31:16

know if it does work that way, but we found a pathway by which it could do that?

A. In this case, yes.

Q. Okay. And cautioning damage to DNA, even if that's really something that happens with glyphosate, causing damage to DNA does not necessarily mean that that leads to mutation, does not necessarily mean that the mutations are of the right sort to cause immortal cells or rapidly dividing cells, does not necessarily mean that it leads to cancer. There are many steps left in the process; right?

A. Again, a lot there. DNA damage as it's measured in a laboratory is exactly what that is, it's a damage to DNA. You argued that that doesn't always lead to mutations. I might take offense at that statement, but it may not lead to critical mutations that are important for carcinogenesis. That is certainly true.

Q. Well -- I'm sorry.

A. No, I'm done.

Q. It's fair to say -- I mean, so that the jury gets some understanding of what's going on in our bodies and the repair mechanisms in our cells, we have DNA damage happening in us, you and I both do, maybe us more than others, and everyone else also right now; right?

A. You -- you consistently have damage to your DNA.

Q. A lot?

A. A lot.

Q. Thousands and thousands and thousands. All the time; right?

11:32:50 A. Well, it depends. It depends on the organ. It depends on a lot of different things. DNA damage in peripheral blood is -- you can measure it. It's there. But it's not directly measuring where you want it to measure.

11:33:09 You want DNA damage in bone marrow, because that's where the blood comes from. And once the blood is formed, it stays for a period of time and goes away.

Other tissues like liver, the tissues are always there. So damage there matters a lot, et cetera.

11:33:30 So it's -- there's a lot of it going on; some of it important, some of it not.

Q. Okay. And almost always when DNA damage happens -- and it happens because of age; it happens because of cosmic rays; it happens because we ate potato

11:33:47 chips instead of broccoli for breakfast. It happens for lots of reasons. Including lots of reasons we'll never know. It almost always, like 99.999 percent of the time, the organelle in the cell, whose job it is to fix, do their job and fix it; right?

11:34:04 A. Again, it depends upon the cell type. But there

is repair machinery. It's fairly efficient. You're asking me for more specifics about doing 99.999 percent. Mutations will definitely be occurring. Hopefully those mutations are in non-coding gene areas or things that
11:34:24 don't really matter in the genome, but you don't know. And I don't think anybody's measured it.

Q. Okay. Let's go back to Exhibit 2481 in your regulatory binder. I'll tell you which one. It's Regulatory Binder 2. It's the OPP 2016 record.

11:34:54 A. Which one is it? 24?

Q. 2481.

A. Okay.

Q. Turn to page 97, please.

MR. GRIFFIS: Permission to publish this page, your Honor?
11:35:45

THE COURT: Any objection?

MR. WISNER: Yes, your Honor.

THE COURT: Let's not --

Can you approach, please?

11:36:04 (Sidebar.)

[redacted]

11:36:16

[redacted]

(End sidebar.)

THE COURT: All right.

You may proceed.

Q. BY MR. GRIFFIS: I'm in the second paragraph. And at the end of the second paragraph, where it describes, under FIFRA -- and that's -- that's the organizing statute for the Office of the Pesticide Programs; correct?

A. Correct.

Q. Under FIFRA, OPP requires genotoxicity tests of the technical grade active ingredients for the registration of the -- sorry about that -- for the
11:37:52 regulation of both food and nonfood use pesticides.

So this is describing what OPP requires manufacturers of pesticides, which includes herbicides, to submit to them in order to get registered. Stuff they want to look at to review the products; correct?

11:38:12 A. That is correct.

Q. "The current genotoxicity test battery," and they give a citation to the -- to the statute. "The current genotoxicity test battery for pesticide registration consists of," and then we have a list.

11:38:33 And Number 1 on the list is something called a bacterial reverse mutation test. And the jury's heard you talk about that as the Ames test; correct?

A. Correct.

Q. Okay. The second one is *in vitro* mammalian
11:38:45 forward gene mutation and *in vitro* mammalian chromosomal aberration test; correct?

A. Correct.

Q. And three, *in vivo* test for micronucleus induction. And it further elaborates on that. Or *in*
11:39:01 *vivo* chromosomal aberration test, and it further

elaborates on what it wants there; correct?

A. Correct.

Q. So there are three categories of tests that are required by EPA to be submitted to it; correct?

11:39:17 A. This is for?

Q. For genotoxicity.

A. For submission of a new -- new agent that isn't in the environment already, that is correct.

Q. Okay. And EPA has received testing in all of

11:39:33 these categories from Monsanto and from multiple other companies that wished to market glyphosate-containing substances; correct?

A. As I said, I -- I don't know where all the data comes from, et cetera.

Q. Okay.

A. But there is -- there is data along these lines that is proprietary, that the regulatory agencies have that have been summarized in documents that I've been able to look at.

11:40:04 Q. You haven't paid much attention to who generates what, but --

A. Some cases, I don't even know.

Q. Okay. Go to page 100, please.

And we have a three-page table listing studies

11:40:24 that have been submitted to EPA. In the first category,

the Ames test; correct?

A. That is correct.

Q. 5.2 -- Table 5.2, sir, *in vitro* mammalian gene mutation assays. So we're following the categories that we just read. And this is the list of studies that EPA has received in that category; right?

11:40:49

A. Yes, it is.

Q. Okay. 5.3 and 5.4, as you continue flipping through pages 108 and 109 and 110, are the next category; right?

11:41:16

A. Well --

Q. More studies that EPA has?

A. 5.3 is chromosome abrasions. And 5.4 is micronuclei.

Q. Right. 5.5 and 5.6 are *in vivo* chromosomal aberration and micronuclei induction; correct?

11:41:28

A. Correct.

Q. The first was *in vitro*. This is *in vivo*?

A. That is correct.

Q. And then finally, 5.7 is assays for detecting primary DNA damage; right? Several pages of that table.

11:41:44

A. Yes. These are mostly literature studies, not regulatory studies.

Q. Okay. EPA certainly considers literature studies when they exist; right?

11:42:15

A. Yes.

Q. Okay. Turn to -- hang on -- your binder, 3181, please.

A. Different binder.

11:42:34 Q. The blue binder, sorry.

A. Okay.

Q. Okay. And what we have, sir, here is a demonstrative exhibit in the categories that we've just discussed. It's, sort of, a table. It's titled

11:42:55 "Glyphosate Studies Considered By EPA."

We have, along the left-hand column, Ames test, then *in vitro* mammalian gene mutation assays, the second category we just discussed. Then *in vitro* tests for chromosomal abnormalities and micronuclei induction in

11:43:21 mammals. And then *in vivo* tests for chromosomal aberration and micronuclei induction in mammals. And then the last thing we were just discussing from the tables, assays for detecting primary DNA damage.

And then in the table, we have little icons

11:43:38 showing the studies from the tables that we just looked at; right, sir?

A. Probably. I don't know if they were all here or whatever, but that's certainly a reasonable interpretation of this.

11:43:52 Q. Okay.

MR. GRIFFIS: I move to publish 3181, a demonstrative exhibit of EPA's glyphosate studies considered.

THE COURT: Any objection?

11:44:04 MR. WISNER: Yes, your Honor. Sidebar.

(Sidebar.)

[redacted]

11:44:58 [redacted]

[redacted]

11:45:30 [redacted]

11:45:45 [redacted]

11:46:02 [redacted]

[redacted]

(End sidebar.)

THE COURT: You may proceed.

Q. BY MR. GRIFFIS: Sir, we've made a deal. We can put up the chart, but you aren't validating the accuracy of chart. I hereby certify that.

So 3181. And what this shows, for your information -- and I'm not going back on my promise -- but you can look up any of these you want with the tables that we just did.

We took the tables from the OPP report in these various categories. And the categories are what I want to ask you about.

And for the studies that they listed, for each one we put up a little icon in the right spot in the chronology, so that we could get an idea how much material they looked at.

So the Ames test that corresponded to Table 5.1 from the OPP's 2016 report, sir, is talking about Ames test. Those are the mutagenicity assays that you told

the jury about on Friday. And these have historically been used to assess whether substances can cause mutations in cell lines that have been bred and designed over the years for that purpose; is that fair?

11:47:52 A. In -- in bacteria, not --

Q. Yes.

A. Not cell lines. It's a bacteria.

Q. I'm sorry. Okay. I knew I'd get it a little bit wrong.

11:48:03 These are bacteria that have been bred over the years for this purpose, because they're good at it; right?

A. There are a number of different salmonella substrains that have specific genetic mutations in them 11:48:19 that were not implanted but identified through selection that are used in this.

Q. Precisely what EPA and EFSA and ECHA and so on require in the area of mechanism studies can change over time, but EPA has consistently, over the decades, 11:48:41 required these Ames tests be done; correct?

A. Yeah. It's cheap. It's simple. And it allows a comparison across multiple chemicals over time.

Q. And these tests of mutagenicity for glyphosate are overwhelmingly negative; right?

11:48:57 A. Yes, they are overwhelmingly negative.

Q. Table 5.2 corresponds to *in vitro* mammalian gene mutation assays, which was table -- I'm sorry -- what do you need?

A. I'm trying to figure out what table you're looking.

Q. Oh.

A. I don't have that book open anymore.

Q. I'll tell you what page to go to. This is in the 2016 OPP report.

A. I've got it.

Q. Page 104. Yeah, the information on the blue line comes from Table 5.2.

These are *in vitro* mammalian gene mutation assays. And if you would just tell the jury in a sentence or two what an *in vitro* mammalian gene mutation assay is.

A. You're -- you're basically looking at the same thing. It's a -- it's a reverse mutation in a mammalian cell line that's been, in this case, transgenically altered.

Q. And you have Table 5.2 in front of you?

A. That's correct.

Q. You see the EPA reports the results as negative, negative, negative, negative for those four?

A. That's what EPA reports.

Q. Let's go to the red line, *in vitro* test for chromosome abnormalities for micronuclei induction in mammals.

A. I'm not certifying that EPA is correct on the negatives here, because I evaluated these same studies. And for example, I think the Chinese hamster ovary cell, I think that was one positive.

Q. Okay.

A. Okay. Where are we now?

Q. Tables 5.3 and 5.4. That's on pages 108 through 110.

A. Uh-huh.

Q. These are *in vitro* tests for chromosome aberrations in mammalian cells for Table 5.3 and *in vitro* tests for micronuclei induction in mammalian cells for 5.4; correct?

A. Correct.

Q. And these are mostly negative for the Table 5.3 and fairly mixed for 5.4; is that right?

A. The EPA's decision?

Q. EPA's description.

A. Give me a minute to look through.

Q. Yes.

A. Yes, it's fairly mixed.

Q. Okay. Tables 5.5 and 5.6 correspond to the

orange line -- so we were just talking about *in vitro* tests, and now we're talking about *in vivo* tests -- for chromosomal aberrations, Table 5.5, and micronuclei induction, Table 5.6, in mammals; right?

11:51:55 A. Correct.

Q. The *in vivo* tests for chromosomal aberrations in mammals were all negative; right, as reported by EPA?

A. That is correct.

Q. And Table 5.6, there are a couple positives, but

11:52:22 almost all negatives; correct? Three positives. They're almost all negatives; correct?

A. Give me a minute.

Q. Sure.

A. They list a few positives, and all the rest are

11:52:47 negative.

Q. Okay. Table 5.7 corresponds to the purple row that starts on page 122.

A. Yes.

Q. And these are a number of -- there are some

11:53:13 negative, some positive. And those are the only two categories, negative and positive.

A. They're virtually all positive. I think there's three negatives in that -- in that batch.

Q. The first two, the first one's negative, the

11:53:37 second one is negative in kidney, positive in liver. The

last three are negative?

A. Yes. Last two are negative. Three? Three. Okay. And so there may be four negatives in this, five.

Q. And do you see "test end point" in the left-hand column, sir?

11:53:58

A. Yes.

Q. Sister chromatid -- oh, I'm sorry. I'm on page 124. They seem to be sorted by test end point.

A. Correct.

11:54:12

Q. And there are four sister chromatid exchanges; right?

A. Four studies of sister chromatid exchange in human -- three are human lymphocytes, one is cow.

Q. And sister chromatid exchanges, since I believe 2014, are no longer required by the regulators because how to interpret them is questionable; is that right?

11:54:32

A. I've heard that. I haven't read -- I haven't read the document.

Q. Okay. Now, sir, one could take this chart and compare it to the studies considered by Working Group 112 and remove the things that Working Group 112 didn't look at. Would you be able to do that by looking at this?

11:54:52

A. It would take several hours.

Q. Okay. We'll skip that, then.

MR. GRIFFIS: I can carry on with the next

11:55:10

chart, or we can break five minutes early for lunch, your Honor.

THE COURT: All right. Why don't we recess now.

Ladies and Gentlemen, we're going to break now for the lunch recess. Please remember: Do not discuss the case with anyone. Please do not do any research. And we will see you again at 1:30.

And, Counsel, could you please remain?

(Jury leaves courtroom.)

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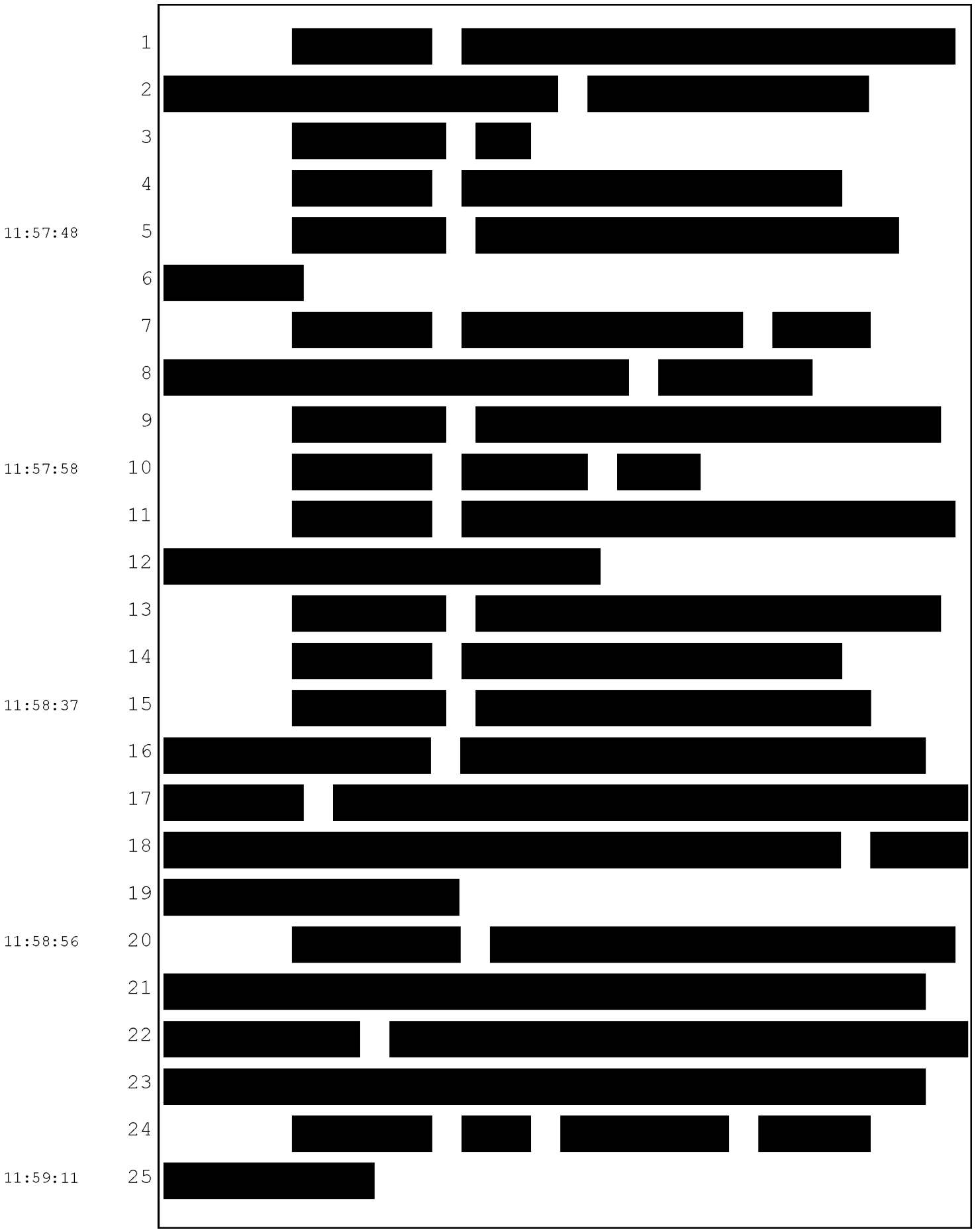
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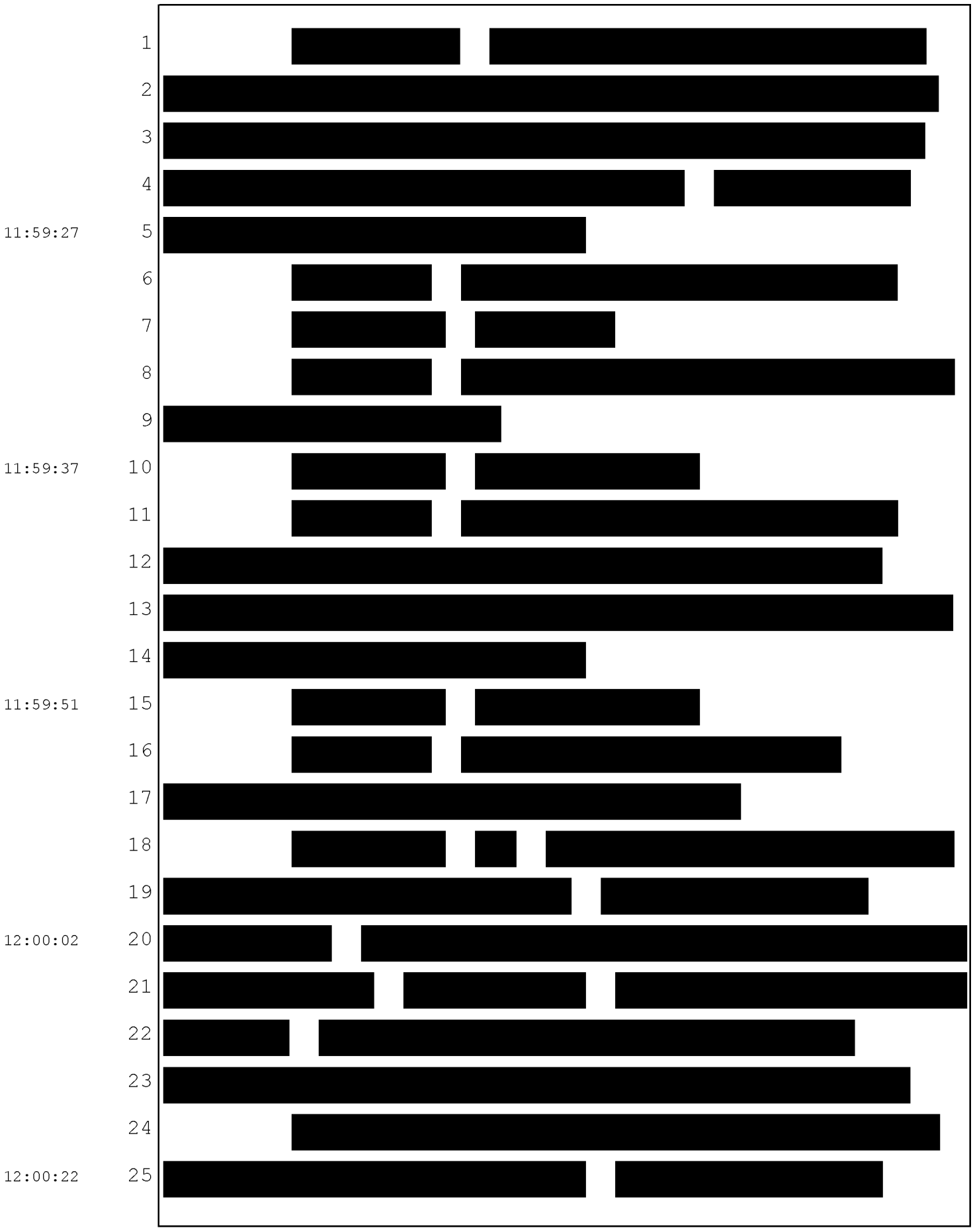
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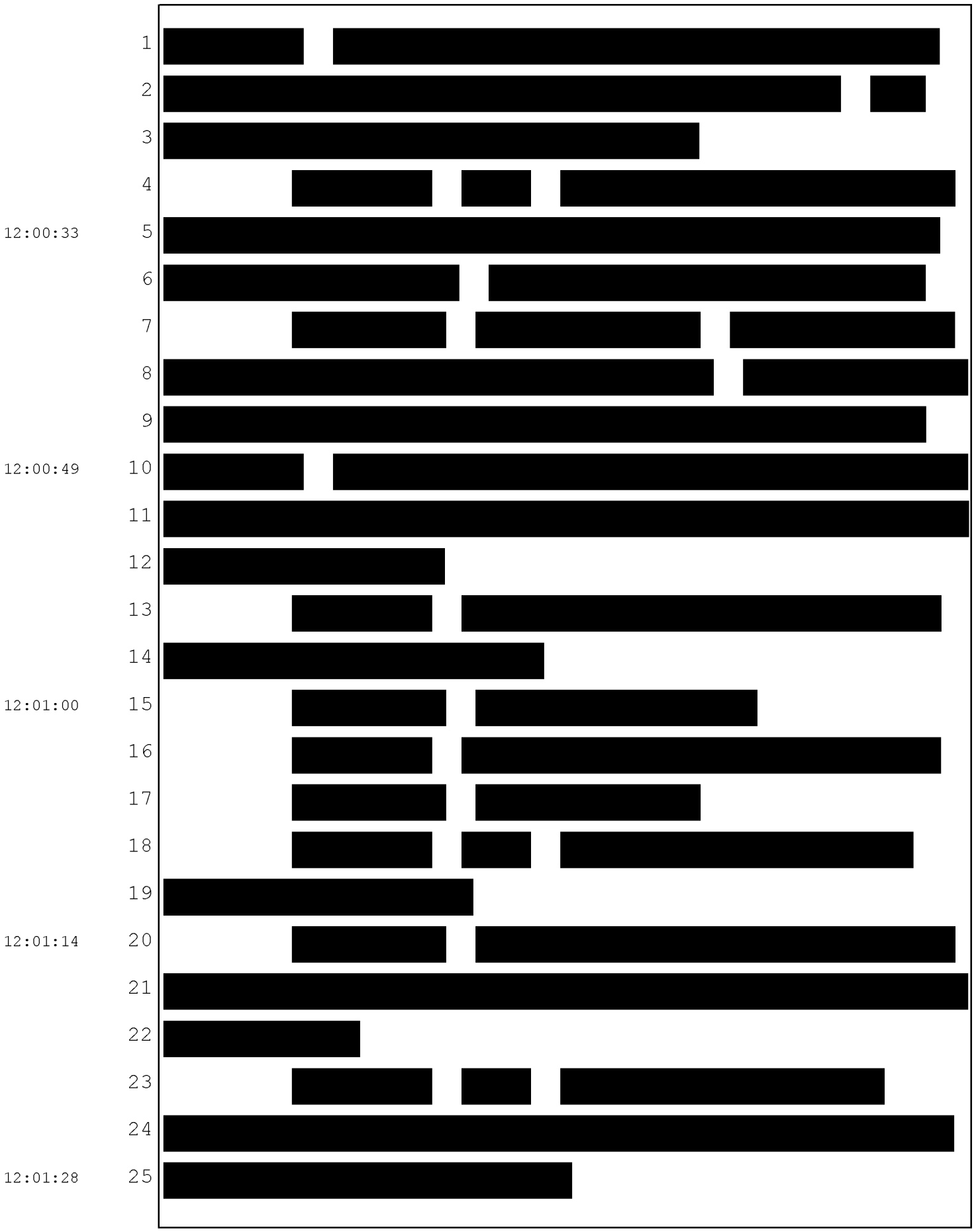
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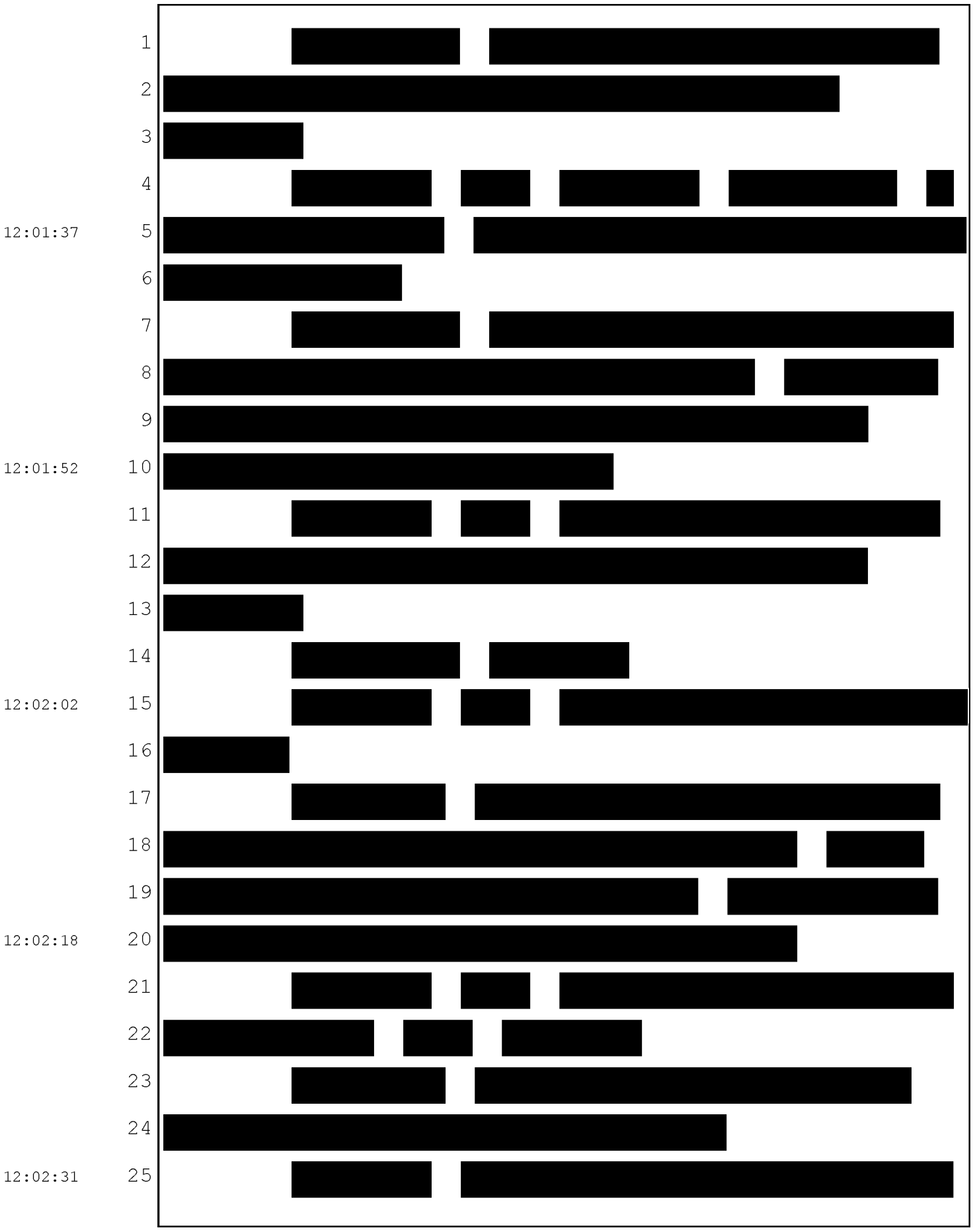
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(Time Noted: 12:03 p.m.)

REPORTER'S CERTIFICATE

I certify that the proceedings in the within-titled cause were taken at the time and place herein named; that the proceedings were reported by me, a duly Certified Shorthand Reporter of the State of California authorized to administer oaths and affirmations, and said proceedings were thereafter transcribed into typewriting.

I further certify that I am not of counsel or Attorney for either or any of the parties to said Proceedings, not in any way interested in the outcome of the cause named in said proceedings.

IN WITNESS WHEREOF, I have hereunto set my hand: July 16th, 2018.

<%signature%>
Leslie Rockwood Rosas
Certified Shorthand Reporter
State of California
Certificate No. 3462