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UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE PATENT TRIAL AND APPEAL BOARD

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FORTE BIOSCIENCES, INC.,
Petitioner,

vs.

No. PGR2023-00014
Patent No. 11,278,505

UNIVERSITY OF
MASSACHUSETTS,

Patent Owner.

_____/

DEPOSITION OF ABUL K. ABBAS, M.D.

September 13, 2023

REPORTED BY:

EARLY K. LANGLEY RMR, RSA, B.A.

CSR NO. 3537

JOB NO: YOM 6083960

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I N D E X

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RE CROSS-EXAMINATION BY MR. FELDSTEIN	103

E X H I B I T S

EXHIBIT NO.	(Previously marked exhibits not listed.)	PAGE
Exhibit 2002	Excerpts from the Ninth Edition of Cellular and Molecular Immunology by Dr. Abbas	57
Exhibit 2003	2003 Forte Biosciences press release, SEC files, August 1, 2023	75
Exhibit 2004	United States Securities and Exchange Commission submission, Forte Biosciences as the submitter	78

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DEPOSITION OF ABUL K. ABBAS, M.D.

BE IT REMEMBERED, that pursuant to Notice, and on September 13, 2023, commencing at the hour of 9:12 a.m., in the offices of Wilson Sonsini Goodrich & Rosati, 1 Market Plaza, Spear Tower, Suite 3300, San Francisco, California, before me, EARLY LANGLEY, a Certified Shorthand Reporter, State of California, personally appeared ABUL K. ABBAS, M.D., produced as a witness in said action, and being by me first duly sworn, was thereupon examined as a witness in said cause.

---000---

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P R O C E E D I N G S

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ABUL K. ABBAS, M.D.

sworn as a witness

testified as follows:

CROSS-EXAMINATION BY MR. FELDSTEIN:

Q. Good morning, Dr. Abbas. I'm Mark Feldstein, on behalf of the patent owner in this case. Nice to meet you.

A. Nice to meet you.

Q. I see you have some documents with you. Can you tell me what you have?

A. This is the patent, my declaration, and then the exhibits, which are mostly references.

Q. Okay. You have all the exhibits that are cited in your declaration in your binder?

A. I believe so.

Q. And so the patent, just for the record, is Exhibit 1001?

A. It's 1001.

Q. And your declaration is 1002?

A. And my declaration is 1002.

Q. Great. And I assume that those are unmarked copies, everything you have there?

Page 5

1 A. That's correct.

2 Q. Perfect. Besides what you have before you and
3 anything that's cited in your declaration, did you rely
4 on any other information to form your opinions in this
5 case?

6 A. I don't recall.

7 Q. You can't recall any other information you
8 relied on?

9 A. That's correct.

10 Q. Did you review any other information besides
11 the documents that are cited in your declaration,
12 Exhibit 1002, in forming your opinions on this case?

13 A. I don't recall.

14 Q. You don't recall considering any other
15 documents besides what's actually cited in your
16 declaration; correct?

17 A. That's correct.

18 Q. Okay. I'm going to start in your declaration,
19 Doctor, with your obviousness analysis. I think it's
20 paragraph 94, Exhibit 1002.

21 A. Which page are you looking at?

22 Q. I'm sorry. I was doing it by paragraph number.
23 It's also page number 44 at the bottom.

24 A. Yeah, got it.

25 Q. And in paragraph 94, you indicate your opinion

Page 6

1 that the claims of the '505 patent are obvious in view
2 of the disclosures of Xing and Harris; correct?

3 A. Correct.

4 Q. Can you turn to Xing in your binder? It's
5 Exhibit 1007.

6 A. Yeah, got it.

7 Q. Can you explain the C3H/HeJ model that's
8 described in Xing?

9 A. It's -- it's an inbred strain of mouse that
10 develops spontaneous hair loss as it ages.

11 Q. Were you familiar with this model prior to your
12 work on this case?

13 A. I was familiar with the C3H/HeJ mouse but not
14 in the context of hair loss.

15 Q. Okay. It says -- on the right-hand side of the
16 first page of Xing, second full paragraph, do you see
17 there is a paragraph that begins "We determine"?

18 A. "To determine."

19 Q. Oh, excuse me. "To determine." You're right.
20 I don't have my glasses on.

21 And you can read the whole sentence, but I want
22 to ask you about the part that says "spontaneously
23 develops alopecia and recapitulates many pathogenic
24 features of human AA."

25 What's your understanding of what features of

Page 7

1 human alopecia areata the C3H/HeJ mouse model
2 recapitulates?

3 MR. MILLS: Objection to form.

4 THE WITNESS: I actually don't have an opinion
5 because that's not part of my declaration and I did not
6 look up the reference.

7 BY MR. FELDSTEIN:

8 Q. So you don't have an understanding of what
9 features of human alopecia areata the H3- -- excuse
10 me -- the C- --

11 A. C3H/HeJ, yeah.

12 Q. Yeah, I'm going to just restart my question so
13 it's clear on the record.

14 So you don't have an understanding of what
15 features of human alopecia areata the C3H/HeJ mouse
16 model recapitulates?

17 A. That's correct.

18 MR. MILLS: Objection to form.

19 THE WITNESS: Oh.

20 MR. MILLS: I just want to give an early
21 reminder. So there were a couple of times where he was
22 talking, Dr. Abbas, and you started speaking.

23 THE WITNESS: Oh, it will be difficult.

24 MR. MILLS: So please just make sure only one
25 person speaks at a time for the court reporter.

Page 8

1 THE WITNESS: Okay.

2 BY MR. FELDSTEIN:

3 Q. Do you have a background on what's important
4 for an animal model in terms of what features of a
5 human disease it should recapitulate?

6 MR. MILLS: Objection to form.

7 THE WITNESS: Some human diseases but not
8 alopecia.

9 BY MR. FELDSTEIN

10 Q. What human diseases?

11 A. The one that I have the most knowledge of is
12 lupus, but that's not relevant to the -- to my
13 declaration.

14 Q. And what -- is there an animal model of lupus?

15 A. Yes.

16 Q. Is there like a gold standard animal model?

17 A. Gold standard in that a lot of people use it.

18 Q. Does it have a name?

19 A. (NZB X NZW)F1 mouse.

20 Q. And what features of lupus in a human does that
21 model recapitulate?

22 A. The mouse makes anti-DNA antibodies, which is
23 characteristic of lupus, and develops kidney disease,
24 which is also characteristic of lupus.

25 Q. Anything else?

Page 9

1 A. I don't recall.

2 Q. In the same -- well, the -- in what context
3 were you familiar with the C3H/HeJ mouse model?

4 A. It's a strain of mouse that we used in my lab
5 just as a source of lymphocytes. I mean -- but that
6 was -- that was decades ago, and I don't really
7 remember the specifics.

8 Q. So you weren't using it as a mouse model of
9 disease; you were using it just as a source for
10 lymphocytes?

11 A. Correct.

12 Q. Are you familiar with the C3H/HeJ mouse model
13 being used as a model for any diseases besides alopecia
14 areata?

15 A. No.

16 Q. Have you seen the C3H/HeJ model used as a model
17 for vitiligo?

18 MR. MILLS: Objection to form.

19 BY MR. FELDSTEIN:

20 Q. I'm sorry. Can you answer the question?

21 Have you seen the C3H/HeJ mouse model used as a
22 model for vitiligo?

23 MR. MILLS: Objection to form.

24 THE WITNESS: I don't recall.

25 BY MR. FELDSTEIN:

1 Q. Do you know if the C3H/HeJ mice can develop
2 vitiligo?

3 MR. MILLS: Same objection.

4 THE WITNESS: I don't know.

5 BY MR. FELDSTEIN:

6 Q. And so I guess you don't know if the C3H/HeJ
7 mouse model has ever been reported to develop vitiligo;
8 is that correct?

9 MR. MILLS: Objection to form.

10 THE WITNESS: I don't know.

11 BY MR. FELDSTEIN:

12 Q. In that same paragraph that we were looking at
13 on page 1 of Xing, the first sentence says, "To
14 determine the contribution of CD8 NKG2D+ T cells."

15 Do you see that?

16 A. Yes.

17 Q. What are CD8+NKG2D+ T cells?

18 A. CD8 is a marker for cytotoxic T cells. So most
19 CD8+ T cells are cytotoxic. Cytotoxic. Killer T
20 cells, that's a more casual way of saying it. And
21 NKG2D is a marker originally described on NK cells, but
22 it is also expressed on some killer T cells.

23 Q. And the NKG2D marker is a marker for what?

24 A. Natural killer cells was how it was originally
25 described, but it is also expressed on some cytotoxic

Page 11

1 T cells.

2 Q. So some cytotoxic T cells express NKG2D, and
3 some do not?

4 A. As far as I know, yes.

5 Q. Other than expressing -- it's a protein it
6 expresses; is that right?

7 A. It's a protein it expresses.

8 Q. Other than the difference in that it expresses
9 the protein, are there any differences in the
10 properties or functions of NKG2D+ T cells as compared
11 to CD8 T cells generally?

12 MR. MILLS: Objection to form. Foundation.

13 THE WITNESS: I don't know the answer.

14 BY MR. FELDSTEIN:

15 Q. Other than expressing the protein, are you
16 aware of any differences between NKG2D+ T cells and
17 CD8+ T cells generally?

18 MR. MILLS: Objection to form. Foundation.

19 THE WITNESS: I don't know the answer.

20 BY MR. FELDSTEIN:

21 Q. From Xing, Exhibit 1007, do you have an
22 understanding of what's reported with respect to the
23 differences, if any, between administering a JAK
24 inhibitor versus administering an antibody to IL-15
25 receptor?

1 MR. MILLS: Objection to form.

2 THE WITNESS: From what I can recall, there is
3 no direct comparison between the two in -- in this
4 reference.

5 BY MR. FELDSTEIN:

6 Q. You're not aware of any differences in what's
7 reported between administering JAK inhibitor
8 versus administering an antibody to IL-15 receptor?

9 MR. MILLS: Objection to form.

10 THE WITNESS: I'm not aware of differences.

11 BY MR. FELDSTEIN:

12 Q. Do you have an understanding from Xing, Exhibit
13 1007, whether the antibody to IL-15 receptor that they
14 administered was able to reverse alopecia areata?

15 A. I don't think they did the -- from what I
16 recall, they didn't look at antibody -- reversal by
17 antibody. They looked at reversal by the JAK
18 inhibitor.

19 Q. And you're talking the mouse model or human?

20 A. In this mouse model.

21 Q. So to your recollection, Xing doesn't report
22 anything with respect to whether its antibody to the
23 IL-15 receptor was able to reverse alopecia areata in
24 mice?

25 MR. MILLS: Objection to form.

Page 13

1 THE WITNESS: From what I remember, I can --
2 from what I can recall from the paper, the antibody was
3 used for prevention in the paper --

4 BY MR. FELDSTEIN:

5 Q. Okay.

6 A. -- and the JAK inhibitors were used for
7 reversal, from what I can recall. I can look at it
8 more carefully, but I think that is correct.

9 Q. If you turn to page 4 of Exhibit 1007, the Xing
10 reference, in the right-hand column in the middle, it's
11 discussing Figure 2D through F and G through I.

12 Do you see that?

13 A. On page 4, yeah.

14 Q. And for Figure 2G through I, it's referring to
15 the antibody blocking the IL-15 receptor; correct?

16 A. Yeah.

17 Q. And the last sentence of the paragraph on
18 page 4 of Exhibit 1007, it says:

19 "Notably, none of these blocking
20 antibodies given alone was able to reverse
21 established AA (data not shown)."

22 Do you have an understanding what that means?

23 A. According to data not shown -- it's a statement
24 that they made. The statement says that the antibody
25 did not reverse established disease in this model of

Page 14

1 alopecia.

2 Q. And that's in contrast to the JAK inhibitor,
3 which did reverse established AA in the mouse model;
4 correct?

5 A. From what I can tell it did, yes.

6 Q. Do you have an understanding of why the JAK
7 inhibitor was able to reverse established AA but the
8 antibody to IL-15 was not?

9 A. No. No comment on that.

10 Q. You'd agree that an antibody to the IL-15
11 receptor is not interchangeable in terms of its effects
12 with the JAK inhibitor in the Xing mouse model;
13 correct?

14 MR. MILLS: Objection to form. Foundation.

15 THE WITNESS: From what I can tell from what
16 they have stated. But I really don't have anything
17 more to say about that.

18 BY MR. FELDSTEIN:

19 Q. So as far as you know, that's the case?
20 They're not interchangeable; correct?

21 MR. MILLS: Objection to form. Foundation.

22 THE WITNESS: From their statement, that's what
23 it seems.

24 BY MR. FELDSTEIN:

25 Q. Can you turn to page 6 of Xing.

1 The left-hand column, do you see the paragraph
2 that begins, "Taken together"?

3 A. Paragraph --

4 Q. The last paragraph.

5 A. "Taken together," last paragraph.

6 Q. It says:

7 "Taken together, our data suggests that
8 CD8+NKG2D+ T cells promote AA pathogenesis,
9 acting as a cytolytic effector responsible for
10 autoimmune attack of the hair follicles."

11 A. Yeah.

12 Q. Beyond what's written in that statement, do you
13 have an understanding of how Xing's data suggests that
14 NK+ -- excuse me -- CD8+NKG2D+ T cells promote AA
15 pathogenesis?

16 MR. MILLS: Objection to form.

17 THE WITNESS: According to their statement,
18 these CD8 T cells kill cells in the hair follicle.

19 BY MR. FELDSTEIN:

20 Q. And so it's these specific T cells, the
21 CD8+NKG2D+ T cells, that are killing cells in the hair
22 follicle; correct?

23 A. That's the claim in this paper, yes, from what
24 I understand.

25 Q. As opposed to just generic, if that's the

Page 16

1 appropriate term, CD8+ T cells; correct?

2 MR. MILLS: Objection to form.

3 THE WITNESS: Yeah, I don't think that's been
4 tested. From what I can tell, they've -- they've shown
5 that this subset of CD8+ T cells can kill the hair
6 follicle.

7 BY MR. FELDSTEIN:

8 Q. And you can't necessarily extrapolate the
9 effect of the NKG2D+ T cells to the effects of CD8+ T
10 cells generally; correct?

11 MR. MILLS: Objection to form. Foundation.

12 THE WITNESS: You know, I -- no comment.

13 BY MR. FELDSTEIN:

14 Q. You don't have an opinion on whether --

15 A. I don't have an opinion.

16 MR. MILLS: Doctor, just -- we're getting close
17 to where it -- it was like you were going to talk over
18 him. So just give him a moment, make sure he finishes
19 his question before you start.

20 THE WITNESS: Okay.

21 MR. MILLS: Thank you.

22 BY MR. FELDSTEIN:

23 Q. In the next sentence of that paragraph, it
24 says:

25 "We postulate that IFN-gamma produced by

Page 17

1 CD8 T cells leads to the collapse of immune
2 privilege in the hair follicle..."

3 Do you see that?

4 A. Yes.

5 Q. The CD8 T cells that they are referring to
6 there are the NKG2D+ T cells? Is that your
7 understanding?

8 MR. MILLS: Objection to form.

9 THE WITNESS: I think that's the implication,
10 but it's not explicitly stated.

11 BY MR. FELDSTEIN:

12 Q. Do you have an understanding beyond what's
13 written here about why IFN-gamma produced by these CD8
14 T cells leads to the collapse of the immune privilege
15 in the hair follicle?

16 A. I have no opinion.

17 Q. And I know, because I've read your book, that
18 you write about immune privilege. What's your
19 understanding of immune privilege of the hair follicle?

20 A. Immune privilege broadly refers to protection
21 from attack by immune cells.

22 Q. And hair follicles have different immune
23 privilege than the skin, for example?

24 A. I don't have an opinion.

25 Q. You don't know?

1 A. I just don't know.

2 Q. The sentence that I was reading continues:

3 "Inducing further production of IL-15 and
4 a feed-forward loop that promotes type 1
5 cellular autoimmunity."

6 Do you see that?

7 A. Yes.

8 Q. What is type 1 cellular autoimmunity?

9 A. When we say "type 1 immunity," we mean immunity
10 involving Th1 cells, which make gamma interferon also,
11 and CD8 killer T cells. Th1 cells and CD8 killer.
12 That's called type 1 immune response. And in the
13 autoimmunity context, it refers to -- that's called
14 type 1 autoimmunity.

15 Parenthetically, it's not a widely used term,
16 "type 1 autoimmunity," but that's what I think they are
17 referring to.

18 So they're basically saying that it's
19 autoimmunity caused by CD8 T cells that may involve
20 other T cell populations that have similar functions.
21 For example, they make gamma interferon.

22 Q. And what's the feed-forward loop that promotes
23 the type 1 cellular autoimmunity?

24 A. When we talk about feed-forward, what we are
25 saying is, for example, that one cytokine then

Page 19

1 stimulates the production of another.

2 So in this particular case, what they're
3 postulating, I believe, is that gamma interferon
4 enhances the production of IL-15, which then enhances
5 the production of gamma interferon. So it's
6 feed-forward amplification.

7 Q. And that amplification is what leads to the
8 collapse of the hair follicle immune privilege?

9 A. That's what they're postulating.

10 Q. To your knowledge, prior to Xing, Exhibit 1007,
11 was there an understanding of a feed-forward loop
12 leading to the collapse of the hair follicle immune
13 privilege?

14 A. I have no opinion.

15 Q. You don't know?

16 A. I just don't know.

17 Q. As of April 2017, are you aware of any
18 analogous feed-forward loop reported for vitiligo?

19 A. I don't know.

20 Q. In your report, Exhibit 1002, you describe at
21 various places how one would or wouldn't be able to
22 distinguish whether an antibody to CD122 -- whether
23 it's affecting the IL-2 or IL-15 pathways; is that
24 right?

25 A. Well, let me just look at what I said about

1 that, because...

2 Where did I say that?

3 Q. Let me try to start with just a factual
4 question. Is it right that -- well, what is CD122?

5 A. It's the beta chain of the trimeric IL-2
6 receptor, and it's also used by IL-15. So it's a
7 common, shared beta chain for the IL-2 receptor and
8 IL-15 receptor.

9 Q. And if you administer an antibody to CD122 and
10 you see a biological effect, how would you know whether
11 the effect is mediated through IL-2 or IL-15 or both?

12 MR. MILLS: Objection to form. Foundation.
13 Calls for speculation.

14 THE WITNESS: I don't know how to answer this
15 question because, you know, partly, you look at the
16 effect, and you compare it to previously demonstrated
17 effects of specifically getting rid of 15 or 2. So if
18 you get an effect that looks like what has been
19 demonstrated, then you say that's what we are seeing.

20 Partly, you can do studies to specifically
21 ask -- mostly in vitro studies -- whether the antibody
22 is blocking signaling by IL-2 or blocking signaling by
23 15 or both.

24 MR. FELDSTEIN: Thank you.

25 I want to turn you to two places at once.

Page 21

1 Jess, can you hand out copies of Exhibit 1031.

2 THE WITNESS: 10- --

3 BY MR. FELDSTEIN:

4 Q. Yeah, I'd like to turn you to, in your report,
5 paragraph 50 on page 20 and Exhibit 1031, which you
6 cite on that page in the paragraph.

7 A. Page 50, 5-0?

8 Q. Paragraph 50 of your report. I'm sorry. I
9 keep giving paragraph numbers.

10 A. Okay. I'll keep that in mind. I keep thinking
11 page numbers because I'm not used to numbered
12 paragraphs.

13 Q. So paragraph 50 of your report, in the first
14 sentence, you cite, among other things, Exhibit 1031;
15 correct?

16 It's just a foundational question, if you can
17 answer --

18 A. Yeah.

19 Q. -- that. Is that correct?

20 A. Yeah.

21 Q. Okay.

22 MR. MILLS: Sorry. Were you going to give him
23 a separate copy if you want him to look at -- you want
24 him to look at both at once or...

25 BY MR. FELDSTEIN:

Page 22

1 Q. Would you like a separate copy, Doctor, of
2 Exhibit 1031?

3 A. No, no. I can go back and forth. It's okay.

4 MR. FELDSTEIN: Then we won't.

5 BY MR. FELDSTEIN:

6 Q. If you need separate copies of anything at any
7 time, we have them. Just let us know.

8 A. Okay.

9 Q. Okay?

10 So just to refresh, paragraph 50 of your
11 report, Exhibit 1002, the first sentence, you're citing
12 Exhibit 1031; correct?

13 A. Yes.

14 Q. And in paragraph 50, you say:

15 "It was known that the damage to
16 melanocytes in vitiligo and the hair follicles
17 in AA are caused, at least in part, by
18 autoreactive CD8+ T cells, including
19 tissue-resident memory T cells, Trm."

20 Correct?

21 A. Yes, that's what it says.

22 Q. What is an autoreactive CD8+ T cell?

23 A. In this case, it would be a CD8+ T cell that
24 reacts against self and, specifically, would be reacts
25 against self-antigens in the hair follicle or the

Page 23

1 melanocyte, depending on which disease you are looking
2 at.

3 Q. So autoreactive, you end up with a reaction
4 between two CD8+ T cells?

5 A. No, no, no.

6 Q. No?

7 A. In an individual, a CD8+ T cell reacts against
8 that individual's own tissue. "Auto" means self-
9 reactive.

10 Q. I see.

11 And the autoreactive -- Exhibit 1031 is also
12 specific to the NKG2D+ T cells; correct?

13 A. Yes. That's what is described, yes.

14 Q. And so Exhibit 1031 isn't saying that all
15 autoreactive CD8+ T cells have a role in either
16 vitiligo or alopecia areata; correct?

17 MR. MILLS: Objection to form. Foundation.

18 THE WITNESS: From what I can tell, this one
19 does -- focuses on the NKG2D+ CD8 T cells.

20 BY MR. FELDSTEIN:

21 Q. It's not saying that all autoreactive CD8+
22 T cells are -- cause damage to either melanocytes in
23 vitiligo or hair follicles in AA; correct?

24 MR. MILLS: Objection to form. Foundation.

25 THE WITNESS: I'm actually not sure, because it

Page 24

1 also says that CD8 T cells that express CD56 can cause
2 the lesions.

3 BY MR. FELDSTEIN:

4 Q. Where are you reading?

5 A. Look -- look at the exhibit, the reference.
6 Look at page 727.

7 Q. 727?

8 A. Yeah.

9 Q. Okay.

10 A. It says, "Cells that express" -- under Number
11 2, the second paragraph -- "Cells that express CD56 or
12 NKG2D are found near human scalp hair follicles."

13 And in the -- in the figure, Figure 1, they
14 talk about "enriched for NKG2D+ and CD56+ cells."

15 What they haven't done -- so it's impossible to
16 say from these data -- is to take CD8 cells that
17 express CD56 and do not express NKG2D. They have not
18 tested those.

19 So they say you have CD8 cells that can express
20 one or the other around the hair follicle, but they
21 test populations that seem to express both. So it's
22 not really possible to conclude from this whether it's
23 only NKG2D+ or also CD56+.

24 Q. Okay. So you're saying it's one or both of
25 CD56+ and NKG2D+ that Exhibit 1031 is linking to either

Page 25

1 melanocyte destruction or hair follicle attack; right?

2 MR. MILLS: Objection to form.

3 THE WITNESS: That's correct.

4 BY MR. FELDSTEIN:

5 Q. Exhibit 1031 isn't saying that all CD8+ T cells
6 damage melanocytes or hair follicles; correct?

7 MR. MILLS: Objection to form. Foundation.

8 THE WITNESS: I don't recall it saying that.

9 BY MR. FELDSTEIN:

10 Q. Can you turn to page 733 of Exhibit 1031. It's
11 the eighth page of the exhibit also.

12 A. Yeah.

13 Q. There's -- on the left-hand side, the second
14 paragraph above "Conclusions" begins, "The key role."

15 Do you see that?

16 The second paragraph above --

17 A. "The key role," yeah.

18 Q. "The key role of NKG2D+ cells (including
19 NK and CD+ T cells) is increasingly
20 recognized not only in AA pathobiology,
21 but also in type 1 diabetes mellitus, multiple
22 sclerosis, rheumatoid arthritis, and Crohn's
23 disease?"

24 Do you see that?

25 A. That's the statement they've made, correct.

Page 26

1 Q. Beyond what's written there, do you have any
2 knowledge or understanding of the role of NKG2D+ cells
3 in diabetes, MS, rheumatoid arthritis, or Crohn's
4 disease?

5 A. No, I don't.

6 Q. Do you have any knowledge of the role, if any,
7 of NKG2D+ cells in vitiligo?

8 MR. MILLS: Objection to form.

9 THE WITNESS: I do not.

10 BY MR. FELDSTEIN:

11 Q. Can you turn on that same page to the first
12 full paragraph on the right. It says, "Lessons
13 learned."

14 A. Uh-huh.

15 Q. "Lessons learned from pre-clinical AA research
16 on how to prevent the experimental induction
17 of hair loss lesions and how to stimulate hair
18 regrowth in established disease promises to be
19 relevant for the management of related
20 medically important -- medically more
21 important but less easily accessible...."

22 Well, okay. I'm not going to read the
23 whole paragraph.

24 A. I got it.

25 Q. Can you read the paragraph "Lessons learned"?

Page 27

1 A. Yes.

2 Q. Okay. What's your understanding of what's
3 being disclosed there?

4 MR. MILLS: Objection to form.

5 THE WITNESS: This is their speculation that
6 you can take the lessons from these alopecia areata
7 models that they've used and perhaps extend them to
8 other autoimmune diseases.

9 BY MR. FELDSTEIN:

10 Q. And do you have an understanding of the basis
11 of their, as you say, speculation that you could
12 extrapolate to other autoimmune diseases?

13 MR. MILLS: Objection to form.

14 THE WITNESS: No, I don't have any
15 understanding of what -- what their thinking is, no.

16 BY MR. FELDSTEIN:

17 Q. And do you have any understanding of what
18 information one would need to be able to extrapolate
19 from pre-clinical AA research to other autoimmune
20 diseases?

21 MR. MILLS: Objection to form. Foundation.

22 THE WITNESS: No opinion on that.

23 BY MR. FELDSTEIN:

24 Q. And do you have an understanding of why
25 Exhibit 1031, in the other autoimmune diseases that it

1 lists, does not include vitiligo?

2 A. I have no opinion.

3 Q. And I'm sorry if I have asked you some version
4 of this before, but do you have an understanding of
5 how, if at all, NKG2D+ cells are linked to diabetes?

6 MR. MILLS: Objection to form.

7 THE WITNESS: No.

8 BY MR. FELDSTEIN:

9 Q. Do you have an understanding of how, if at all,
10 NKG2D+ cells are linked to multiple sclerosis?

11 MR. MILLS: Objection to form.

12 THE WITNESS: No. And you're referring to
13 NKG2D+ CD8 cells.

14 BY MR. FELDSTEIN:

15 Q. Yes -- well --

16 A. I just want to make that point because NKG2D is
17 expressed on natural killer cells. That's not what you
18 are referring to.

19 Q. Well, maybe we should clarify a little bit
20 again.

21 On page 733 of Exhibit 1031, we were looking at
22 that paragraph, "key role."

23 A. Yeah.

24 Q. And it said:

25 "The key role of NKG2D+ cells (including

Page 29

1 NK and CD+ T cells)...."

2 Do you see that?

3 A. Oh, yeah. Yeah.

4 Q. So in the con- -- if we're discussing the
5 context in Exhibit 1031, should we be discussing NKG2D+
6 NK or T cells?

7 MR. MILLS: Objection to form. Foundation.

8 THE WITNESS: We could be. The data they've
9 provided is only for CD8 T cells.

10 BY MR. FELDSTEIN:

11 Q. Okay.

12 A. But they haven't -- that doesn't rule out the
13 role for NKG2D+ natural killer cells.

14 Q. Okay. Well, let me go back and try to make my
15 question better, hopefully.

16 Do you have an understanding of how, if at all,
17 NKG2D+ cells, either NK cells or T cells --

18 A. Uh-huh.

19 Q. -- CD8 T cells are linked to multiple
20 sclerosis?

21 A. No.

22 Q. Do you have an understanding how, if at all,
23 NKG2D+ cells, whether NK or CD8 T cells, are linked to
24 rheumatoid arthritis?

25 A. No.

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1 MR. MILLS: Objection to form.

2 BY MR. FELDSTEIN:

3 Q. Do you have an understanding how, if at all,
4 NKG2D+ cells, whether NK or CD8+, are linked to Crohn's
5 disease?

6 MR. MILLS: Objection to form.

7 THE WITNESS: No.

8 BY MR. FELDSTEIN:

9 Q. Do you have an understanding of how, if at all,
10 NKG2D cells, whether NK or CD8+ T cells, are linked to
11 vitiligo?

12 MR. MILLS: Objection to form.

13 THE WITNESS: No.

14 BY MR. FELDSTEIN:

15 Q. Can we turn now to Exhibit 1006, the Harris
16 mouse model paper.

17 A. Yeah.

18 Q. What's your general understanding of
19 Exhibit 1000- -- excuse me. What's your general
20 understanding of what's disclosed in Exhibit 1006?

21 A. Come again? Can you just restate that?

22 Q. What is your general understanding of what's
23 disclosed in Exhibit 1006?

24 A. A mouse model of vitiligo in which T cells
25 specific for a melanocyte antigen are injected into

Page 31

1 mice that are induced to express large numbers of
2 melanocytes, and those T cells are activated in the
3 mouse by injecting a virus that produces the melanocyte
4 antigen, and then the loss of pigmentation is followed.

5 That's the simple description of the model.

6 Q. In the "Abstract," a little bit farther than
7 halfway down, it refers to "melanocyte-specific CD8+
8 T cells."

9 Do you see that?

10 A. Correct.

11 Q. What does that mean, "melanocyte-specific CD8+
12 T cells"?

13 A. Well, in their model, they have used transgenic
14 technology to express T cell receptors specific for
15 this melanocyte antigen, Pmel, in a mouse. And so lots
16 of T cells in that mouse become melanocyte-specific
17 because you force expression of one antigen receptor.
18 Right?

19 Q. And "Pmel," you're referring the abbreviation
20 P-m-e-l?

21 A. Correct. It's the antigen. It's the
22 melanocyte antigen.

23 Q. And is that melanocyte antigen Pmel unique to
24 the mouse model, or is that shared with humans?

25 MR. MILLS: Objection to form.

1 THE WITNESS: I don't know the answer.

2 BY MR. FELDSTEIN:

3 Q. In the same place we're looking, in the
4 "Abstract," it refers to the "melanocyte-specific CD8+
5 T cells," and then it says, "which recapitulates the
6 human condition."

7 Do you see that?

8 A. Yes.

9 Q. What's your understanding of how the mouse
10 model, as described in Exhibit 1006, recapitulates the
11 human condition of vitiligo?

12 A. In my opinion, it does not.

13 Q. Are there any features of the human condition,
14 to your understanding, that it does recapitulate?

15 A. That it does recapitulate?

16 Q. Correct.

17 A. Loss of pigmentation, the end result, but it
18 does not recapitulate how that was achieved.

19 Q. So other than loss of pigmentation, in your
20 understanding, the Harris mouse model in Exhibit 1006
21 doesn't recapitulate any other features of human
22 vitiligo?

23 A. It involves CD8 cells, but the CD8 cells are
24 completely different from what you would expect to see
25 in the human.

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1 Q. What do you mean, that these CD8 T cells in the
2 Harris mouse model, Exhibit 1006, "are completely
3 different from what you would expect to see in the
4 human"?

5 A. In this mouse model, they express a single
6 antigen receptor in the CD8 cells, so these CD8 cells
7 are all, or mostly -- I would say mostly, but maybe
8 all -- specific for the melanocyte antigen.

9 In humans, CD8 cells recognize lots of
10 different things -- viruses, et cetera -- and a very
11 small fraction could conceivably recognize the
12 melanocyte antigen.

13 There is no relationship between those
14 situations.

15 Q. Why, in your view, is that difference
16 significant?

17 A. Because in the mouse model, you are looking at
18 immune attack by -- I'm going to come up with a number.
19 They don't describe the number. But supposing
20 50 percent of the T cells are specific for the
21 melanocyte antigen, because you have forced expression
22 of a single antigen receptor. Correct?

23 In the human, one in a million T cells might be
24 specific for the -- a melanocyte antigen.

25 That's the difference you are looking at;

1 50 percent of the immune system attacking melanocytes
2 versus one in a million immune cells attacking --
3 potentially attacking melanocytes.

4 There is no comparison between the two.

5 Q. And so if you wanted to be able to make a
6 comparison between one system and another system, you
7 need to have some equivalence in the type of CD8
8 T cells involved; is that correct?

9 MR. MILLS: Objection. Form. Foundation.

10 THE WITNESS: Types of T cells, but even more
11 importantly, the number of T cells.

12 The second reason why there is no equivalence
13 with the human situation is that after they transfer
14 the T cells into the mouse, they then activate them
15 systemically by giving a virus expressing the
16 melanocyte antigen. Correct?

17 The rVV Pmel, that's a vaccinia virus producing
18 the melanocyte antigen. They inject that into the
19 mouse to activate the T cells they've administered to
20 the mouse.

21 Well, you never see vaccinia virus expressing a
22 melanocyte antigen in a human being.

23 I just want to be sure you understand the point
24 I'm trying to make.

25 The mouse model is taking a single population

1 of T cells, all of which or almost all of which express
2 the receptor for the melanocyte antigen, parking those
3 T cells in a mouse, and then hitting them with the
4 melanocyte antigen in the form of a virus. Right?

5 That's the model, as set up.

6 In the human situation, very few T cells can
7 recognize the melanocyte antigen, and they never see a
8 virus expressing that antigen.

9 BY MR. FELDSTEIN:

10 Q. So in terms of the types of T cells, you're
11 referring to what antigen that they're able to bind to?

12 What do you mean by "types of T cells"? Let me
13 ask that.

14 A. I didn't say "types of T cells." I'm referring
15 to the number of T cells specific for the melanocyte
16 antigen.

17 Q. Earlier, at least as transcribed, I was asking
18 you about -- when you are comparing one system to
19 another, I had asked if there was -- there needed to be
20 some equivalence on the type of CD8 T cells involved.

21 Do you remember me asking you something along
22 those lines?

23 A. Yeah. And if I remember correctly -- you can
24 re-read it -- I said, not just the types, the numbers.

25 Q. Yes, you did.

1 And my question now is, what did you mean by
2 "types of T cells"?

3 A. Oh. Yeah, I should take that back.

4 The types of T cells may both be CD8 T cells,
5 so there may be equivalent types of T cells.

6 But the numbers and how they are activated are
7 completely different in the mouse model versus what you
8 would expect in a human with vitiligo.

9 Q. And in terms of types, do they have to be
10 similar in terms of the antigen they recognize?

11 A. They could be, but I don't know the
12 specificities -- the antigen-recognition specificities
13 of all the T cells that cause vitiligo in humans.

14 Q. Okay.

15 A. So just to explain more clearly, now in the
16 mouse model, you have T cells recognizing one
17 melanocyte antigen. In the patient with vitiligo, you
18 may have T cells recognizing N number, an unknown
19 number of melanocyte antigens. I don't know the
20 answer.

21 So that's what I mean by the difference.

22 Q. So one type is specific to a single melanocyte
23 antigen, and the rest in human may be specific or not
24 specific to other melanocyte antigens?

25 A. Numerous. And I don't know if we know the

1 number.

2 Q. And without knowing that, it's improper, in
3 your view, to try to extrapolate from a mouse model to
4 human?

5 MR. MILLS: Objection to form.

6 THE WITNESS: It's improper to extrapolate from
7 this mouse model to the human situation -- to the human
8 disease.

9 BY MR. FELDSTEIN:

10 Q. So in terms of what's common between the mouse
11 model of vitiligo in Exhibit 1006 and human disease,
12 you had told me that they both involve loss of
13 pigmentation, they both involve CD8+ T cells.

14 Is there anything else that's common, to your
15 understanding, between the mouse model of vitiligo in
16 Exhibit 1006 and the human vitiligo condition?

17 A. Nothing that I can think of.

18 MR. MILLS: We have been going for about an
19 hour. Are we ready for a break?

20 MR. FELDSTEIN: I could use another five or so
21 minutes to finish the document. Is that okay with
22 everybody?

23 THE WITNESS: Okay with me.

24 BY MR. FELDSTEIN:

25 Q. Okay. To your understanding, are there any

1 common histologies between the Harris mouse model
2 vitiligo and the human vitiligo disease?

3 A. I don't know.

4 Q. Is that something you'd know from your skill,
5 to interpret comparing -- comparative histologies?

6 A. I could interpret, but I have no experience in
7 looking at biopsies of vitiligo from people.

8 Q. What are chemokines, c-h-e-m-o-k-i-n-e-s?

9 A. They are a subset of cytokines. Cytokines
10 are -- you know, the broad term is not a good term, is
11 "interleukin."

12 Cytokine -- the chemokines are a subset of
13 cytokines that chemically attract cells towards
14 themselves. That process of attraction is called
15 chemotaxis, moving towards a chemical gradient. And so
16 the term "chemokine" is a contraction of "chemotaxis,"
17 "cytokine." "Chemokine."

18 Q. To your understanding, are there any common
19 chemokines between the Harris mouse model, Exhibit
20 1006, and human vitiligo?

21 A. I don't know. I don't know about what
22 chemokines, if any, have been described in human
23 vitiligo.

24 Q. Can you turn to page 1872 of Exhibit 1006.

25 A. Yeah.

1 Q. Do you see on the bottom right the heading
2 "Depigmentation" --

3 A. Yeah.

4 Q. -- "critically depends"?

5 Do you see that?

6 A. Yes.

7 Q. And then I will do my best to read the first
8 sentence, but you can just read it yourself, too.

9 "Consistent with human disease, IFN-gamma
10 expression was elevated with the depigmenting
11 skin of mice with vitiligo and was expressed
12 from autoreactive CD8+ T cells."

13 A. Yes.

14 Q. Do you see that?

15 A. Yes.

16 Q. Do you have any basis to agree or disagree that
17 this is common with human disease?

18 A. No. I don't know the data from human disease.

19 I do want to add one more thing, which you
20 didn't ask me about. But from what I recall -- and I'm
21 flipping through this paper now to refresh my memory --
22 there is no data on human disease gamma interferon
23 production included in this publication.

24 Q. And you're referring to Exhibit 1006; correct?

25 A. The Harris paper, yeah. I don't see any data.

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1 Q. And so maybe this actually goes to your point.
2 If you could turn to page 1873?

3 A. Yeah.

4 Q. The beginning of the first full paragraph on
5 the left, it says, "The expression of IFN-gamma"?

6 A. Yeah.

7 Q. It says:

8 "Expression of IFN-gamma correlates with
9 depigmentation in both human vitiligo and in
10 this mouse model."

11 A. Correct.

12 Q. You're not aware of whether that's accurate or
13 not; correct?

14 A. I'm not aware of that, yeah. That's the point.

15 Q. And on that same page, the beginning of the
16 last paragraph on the right, it says:

17 "IFN-gamma has widely variable effects on
18 the immune response."

19 Do you see that? And then it continues.

20 A. Correct.

21 Q. Is that a fair statement of the art, that
22 "IFN-gamma has widely variable effects on immune
23 response"?

24 A. It's a fair statement except for one phrase,
25 "direct target cell cytotoxicity."

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1 I don't know if interferon-gamma has been
2 implicated in killing cells. I simply don't know.

3 Q. And are you aware of any differences, any
4 variability on the effects of IFN-gamma in the hair
5 follicle versus in the skin with respect to CD8+
6 T cells?

7 A. I'm not aware. I just don't know.

8 MR. FELDSTEIN: Okay. Why don't we take a
9 break.

10 (Recess taken.)

11 BY MR. FELDSTEIN:

12 Q. Doctor, still with Exhibit 1006, the Harris
13 mouse model --

14 A. Uh-huh.

15 Q. -- are you aware of other models of vitiligo
16 prior to April 2017?

17 MR. MILLS: Objection to form.

18 THE WITNESS: I am not -- I'm just thinking
19 aloud. I'm not aware of other mouse models of
20 vitiligo.

21 BY MR. FELDSTEIN:

22 Q. Are you aware of other uses or reliance on the
23 Exhibit 1006 mouse model prior to April 2017 besides
24 what's actually reported in Exhibit 1006?

25 A. No.

1 Q. Are you aware of how people skilled in the art
2 view the Harris mouse model, Exhibit 1006, in terms of
3 its relevance to human vitiligo?

4 MR. MILLS: Objection to form.

5 THE WITNESS: I can tell you my opinion, but I
6 can't tell you other people's views.

7 BY MR. FELDSTEIN:

8 Q. One thing of background. I kind of completely
9 skipped background. We were talking about -- off the
10 record about the most accurate way to refer to your
11 medical degree.

12 A. Yes.

13 Q. That's not my question. But my question is,
14 are you a treating physician?

15 A. No.

16 Q. So do you treat vitiligo?

17 A. No.

18 Q. Have you treated vitiligo?

19 A. No.

20 Q. Do you treat alopecia areata?

21 A. No.

22 Q. Have you treated alopecia areata?

23 A. No.

24 Q. Have you treated any immune diseases --

25 A. No.

1 Q. -- autoimmune diseases?

2 A. No. My clinical training, just to be clear, is
3 in pathology. So we look at and analyze tissues. We
4 don't actually treat patients.

5 Q. Can you turn in your declaration, Exhibit 1002,
6 back where we started at paragraph 94. Paragraph 94,
7 obviousness analysis. And can you just refamiliarize
8 yourself, Doctor, with your testimony in paragraphs 94
9 through 97. It's the two pages of your report.

10 A. Okay.

11 Q. And we had talked a lot in terms of saying
12 about the NKG2D+ cells that are discussed there;
13 correct?

14 A. Yeah.

15 MR. MILLS: Objection to form.

16 BY MR. FELDSTEIN:

17 Q. And we talked about the Pmel CD8 cells that are
18 in the Harris model, Exhibit 1006; correct?

19 A. Yes.

20 Q. In paragraphs 94 through 97, are you trying to
21 convey that Harris and Xing have the same CD8+ T cells?

22 A. No.

23 Q. They don't have the same CD8+ T cells; correct?

24 MR. MILLS: Objection to form.

25 THE WITNESS: No.

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1 BY MR. FELDSTEIN:

2 Q. And I'm sorry. It's a terrible question,
3 because I don't know if "no" agrees with me or not.

4 But you agree with me that Harris and Xing do
5 not have the same CD8 T cells; correct?

6 MR. MILLS: Objection to form.

7 THE WITNESS: Can I explain that a little bit?
8 I mean --

9 BY MR. FELDSTEIN:

10 Q. Sure.

11 A. -- my response.

12 Q. Sure.

13 A. Harris, in his model, is studying T cells of
14 one specificity. They all have the antigen receptor
15 for Pmel.

16 Xing is studying autoreactive CD8 T cells that
17 cause alopecia. He does not know the specificity.
18 It's unlikely to be Pmel-specific because that's not
19 what it is in the hair follicle.

20 So in terms of specificity -- I just want to be
21 clear about this. In terms of specificity, they are
22 looking at different T cells; however, they may both be
23 looking at CD8 T cells.

24 Q. CD8 T cells of different specificity?

25 A. Correct.

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1 Q. Different types of CD8 T cells?

2 A. Well, I don't know what you mean by "type." In
3 the field, we tend to be more precise than that. The
4 correct way to say it is what you just said; they are
5 looking at CD8 T cells of different specificities.

6 Q. Can we turn to Exhibit 1027.

7 A. '27.

8 Q. The first author is Cheuk, C-h-e-u-k.

9 A. "Cheuk."

10 Q. "Cheuk"?

11 A. I don't know how he pronounces his name. Yes.

12 Q. The great thing is, no matter how we pronounce
13 it, our friendly court reporter is going to type it the
14 same. So I can say "Cheuk," and you can say "Cheuk."

15 You're familiar with Exhibit 1027, the Cheuk
16 reference?

17 A. I've read it.

18 Q. And can you explain the relationship, if any,
19 between the CD49a marker that's described and the
20 NKG2D+ cells addressed in Xing or the Pmel cells
21 addressed in Harris?

22 A. I have no idea, no.

23 Q. Is it correct that Exhibit 1027 is reporting
24 that CD49a+ CD8 T cells accumulate in vitiligo lesions?

25 A. That's correct.

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1 Q. To your understanding, Cheuk is not identifying
2 melanocyte destruction as being caused by the CD49a+
3 CD8+ T cells?

4 A. From what I recall, that's correct. He
5 describes that population, but he does not say that it
6 is causing vitiligo, from what I recall.

7 Q. It's just a correlation, not a causation?

8 A. That's a good way of saying it.

9 Q. Okay. Can you turn to -- I don't know the page
10 numbers -- the second page of the document,
11 Exhibit 1027. It's page 287 of the article.

12 A. Page 287 in the article, yeah.

13 Q. There is a single full paragraph on the
14 right-hand side. Do you see that?

15 A. Starting with?

16 Q. "Following."

17 A. "Following." Okay.

18 Q. And then the final sentence says:

19 "Thus, if Trm cells drive these decreases,
20 then vitiligo and psoriasis would be expected
21 to involve the contribution of functionally
22 different subsets."

23 Do you see that?

24 A. I see the statement, yes.

25 Q. In context of Exhibit 1027, what do you

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1 understand that to mean?

2 A. What he's suggesting is that within that
3 population of Trm cells, some are more involved in
4 vitiligo and others are more involved in psoriasis.
5 That's the suggestion in that statement.

6 Q. Is "functionally different" merely a reference
7 to what disease that they're involved in?

8 A. No. "Functionally different" is a reference to
9 what they do.

10 And they go on in the paper to say that in
11 psoriasis, the T cells make IL-17, one of the
12 cytokines, and in vitiligo, they make gamma interferon.
13 So that's the functional difference they are leading up
14 to.

15 Q. And what drives CD8 T cells to have such
16 different functions in terms of what they produce?

17 A. I have no opinion at this point.

18 Q. Is that beyond science as of today to answer
19 that?

20 A. It's very speculative, so I really don't want
21 to express an opinion.

22 Q. Okay. In the next paragraph on page 287, the
23 very last line begins, "Nonetheless, phenotypic markers
24 of Trm cell subsets with specialized effector functions
25 have not been established."

1 Do you see that?

2 A. Correct.

3 Q. Can you explain your understanding of what that
4 means?

5 A. When people talk about phenotypic markers, they
6 typically refer to cell surface proteins that can be
7 detected by staining with various anti-CD antibodies.

8 And what he's saying is that there are no cell
9 surface proteins that readily distinguish subsets of
10 Trm cells.

11 Q. So here, "subsets" is being used to describe to
12 what antigens the Trm cells are specific to?

13 A. No, it's not referring to specificity. It's
14 referring -- typically, when you talk about subsets,
15 you refer to functionally distinct populations.

16 So if you want to state it more precisely, what
17 he's saying is that there is no CD marker that will
18 tell you which Trm cell is making IL-17 and which Trm
19 cell is making IFN-gamma. You cannot correlate it with
20 cell surface markers.

21 Q. Can you turn to Exhibit 1026, 1026 in your
22 binder. The first author is Becker.

23 A. Yes.

24 Q. Are you familiar with Becker Exhibit 1026?

25 A. I'm familiar with the paper, yes.

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1 Q. And feel free to refresh your recollection, but
2 is it correct that Becker Exhibit 1026 is not
3 demonstrating that a CD122 antibody reversed
4 depigmentation in vitiligo lesions?

5 MR. MILLS: Objection to form.

6 THE WITNESS: That is correct. From I recall,
7 this paper has nothing to do with vitiligo.

8 BY MR. FELDSTEIN:

9 Q. Is it correct, to your knowledge, that prior to
10 April 2017, CD122 antibodies had not been shown to
11 reverse depigmentation in vitiligo lesions?

12 MR. MILLS: Objection to form.

13 THE WITNESS: To my knowledge, they had not
14 been shown to reverse depigmentation, that is correct,
15 to my knowledge.

16 MR. FELDSTEIN: Can we hand out Exhibit 1023,
17 please?

18 THE WITNESS: '23?

19 BY MR. FELDSTEIN:

20 Q. 1023. Agarwal.

21 Are you familiar with Exhibit 1023.

22 A. Not well. I read it when I was working on the
23 declaration, but I have not refreshed my memory with
24 this one.

25 Q. Okay. Well, feel free to refresh yourself.

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1 My question is, is Exhibit 1023 -- is the focus
2 on targeting IFN-gamma signaling as a treatment
3 strategy for vitiligo?

4 A. That's what the "Abstract" says, that they want
5 to target IFN-gamma signaling.

6 Q. And what do you understand that to mean,
7 targeting IFN-gamma signaling?

8 A. My understanding is to block or reduce
9 IFN-gamma.

10 Q. And in terms of blocking signaling, what does
11 that entail?

12 MR. MILLS: Objection to form.

13 THE WITNESS: Blocking signaling would be
14 blocking the downstream actions of IFN-gamma.

15 BY MR. FELDSTEIN:

16 Q. For example, either by an antibody to IFN-gamma
17 or to a receptor for IFN-gamma?

18 MR. MILLS: Objection to form.

19 THE WITNESS: Both of those would block
20 signaling by IFN-gamma, or an inhibitor of, say, the
21 JAK-STAT pathway.

22 MR. FELDSTEIN: All capitals, J-A-K dash
23 S-T-A-T.

24 BY MR. FELDSTEIN:

25 Q. To your knowledge, through April 2017, in the

1 treatment of vitiligo, was the focus on targeting
2 IFN-gamma signaling?

3 MR. MILLS: Objection to form.

4 THE WITNESS: I don't know.

5 BY MR. FELDSTEIN:

6 Q. Are you aware of any experiments prior to
7 April 2017 describing the use of targeting IL-15
8 signaling as a treatment of vitiligo?

9 MR. MILLS: Objection to form.

10 THE WITNESS: I am not.

11 BY MR. FELDSTEIN:

12 Q. Can we go back to the Xing paper, Exhibit 1007.

13 Short of the step where they actually
14 administer it to humans, can you describe for me the
15 methodology where they identified -- by which they
16 identified a JAK inhibitor as a treatment for alopecia
17 areata? The research methodologies, in particular.

18 A. From what I recall, they first showed that in
19 this spontaneous mouse model of alopecia, that
20 CD8+NKG2D+ cells accumulate in the skin. Then they
21 went on to show that blocking IFN-gamma or IL-2 or
22 CD122 prevents development of the disease.

23 And that led them to ask, "If I look at
24 inhibitors of JAK-STAT signaling downstream of those
25 cytokines, will that have a beneficial effect?" And

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1 that's how they got to the JAK-STAT inhibitors -- JAK
2 inhibitors.

3 Q. In terms of the research methodology, first
4 they found an accumulation of CD8 cell expressing a
5 certain protein; correct?

6 A. Correct.

7 Q. In terms of a research methodology for looking
8 at cytokine-dependent diseases, is that a standard sort
9 of first step to try to elucidate the disease
10 mechanism?

11 A. It's often what we do first. We ask what's in
12 the lesion. Could be an antibody. Could be antibody
13 deposits, like in lupus kidneys. Could be T cells.
14 Could be CD4 T cells or CD8 T cells. So we first ask
15 what's in the lesion.

16 Q. And it could be an interleukin itself?

17 A. Well, the interleukin has to be produced by
18 someone. Right? So we first ask what's in the lesion.
19 If it's a T cell, then we try to isolate those T cells
20 and ask what are they making, which would be an
21 interleukin, for example. And then we use blockers of
22 that interleukin to see whether it will prevent or
23 ameliorate the disease.

24 Q. If it blocks or ameliorates -- let me back up.
25 If the blockers of the interleukin, for

1 example, prevent or ameliorate the disease, what does
2 one conclude about the mechanism?

3 MR. MILLS: Objection to form. Foundation.

4 THE WITNESS: My conclusion would be that that
5 interleukin plays a role in the pathogenesis, in
6 causing the disease.

7 BY MR. FELDSTEIN:

8 Q. And that blocking that interleukin could then
9 play a role in preventing or ameliorating the disease?

10 MR. MILLS: Objection to form.

11 THE WITNESS: It could.

12 BY MR. FELDSTEIN:

13 Q. Can we turn to Exhibit 1030. It's a reference
14 by Hechinger.

15 Are you familiar with Exhibit 1030, Doctor?

16 A. I looked at it when I was writing the report.
17 I must say I have not re-examined it recently.

18 Q. Okay. So as always, take your time if you need
19 to refresh yourself.

20 A. Yeah.

21 Q. My question is a similar one: If you could
22 describe generally the methodology that's reported in
23 Hechinger for what they report as the therapeutic
24 activity of gamma chain cytokine inhibition in GvHD?
25 How do they determine that mechanistically?

1 A. It's a complicated paper.

2 Okay. What they've shown or -- and I'm mainly
3 basing it on the "Abstract," not going over all the
4 data figures.

5 What they've shown is that in this model of
6 acute graft-versus-host disease, many different
7 cytokines -- levels of many different cytokines are
8 elevated. I suspect they looked at serum -- yes -- in
9 the -- oh, in patients, not just in the model. In
10 patients with acute graft-versus-host disease, the
11 levels of many cytokines are elevated.

12 Several of those cytokines -- actually, all the
13 ones that they've listed in the "Abstract" share the
14 common gamma chain, CD132, so they hypothesize that "If
15 we block the common gamma chain, we may block the
16 actions of all the cytokines that are elevated." And
17 that was the rationale for this project.

18 Q. And what did they conclude?

19 A. And they concluded that it does block -- the
20 antibody against CD132 does block all those cytokines.
21 And then they go on to show that mainly, it's blocking
22 cytokines coming from CD8 T cells.

23 Q. And so in terms of the first step you
24 mentioned, finding elevated cytokines --

25 A. Correct.

1 Q. -- is that a standard research approach, or is
2 it something that they newly developed in Hechinger?

3 A. It's not a standard approach, from my
4 knowledge. The reason why it's not a standard approach
5 is it's very difficult to measure cytokines in the
6 serum, get reliable assays. So most people wouldn't do
7 this. But they must have the tools for doing that.

8 Q. Assuming you have the tools, is that a
9 reasonable first step, in your view, to look for what
10 cytokines are elevated in association with the disease
11 condition?

12 A. If you could do it reliably, yes. But I don't
13 think you can do it reliably.

14 Q. Okay. And then they conclude that CD122 --

15 A. '132.

16 Q. CD132. They conclude that CD132 is a
17 therapeutic target for GvHD?

18 A. That's their conclusion.

19 MR. FELDSTEIN: I'm going to mark -- can we
20 mark, Jess, a new document?

21 We're going to mark a document you don't have
22 but you're intimately familiar with. We're going to
23 mark as Exhibit 2002 excerpts from the Ninth Edition of
24 Cellular and Molecular Immunology by Dr. Abbas.

25 So let's mark it first as Exhibit 2002.

1 (Whereupon, Exhibit 2002 was marked for
2 identification.)

3 MR. MILLS: So I'll register an objection at
4 this time on the basis that it's an excerpt. So under
5 the federal rules, does not appear to be a complete
6 document.

7 And I'll also register an objection to the
8 extent that any statements here are asserted for the
9 truth of the matter, there's not foundation to admit
10 them as such.

11 And moreover, the date of the document appears
12 to be post-critical date.

13 BY MR. FELDSTEIN:

14 Q. To the best of your knowledge, Doctor, is the
15 information contained in Exhibit 2002 accurate as of
16 April 2017?

17 A. Well, it's accurate as of the date -- yes.
18 This is a 2018 copyright. I didn't remember. So yes.

19 Q. Okay. And just for the record also -- it's a
20 little silly, but can you just identify what
21 Exhibit 2002 is?

22 A. It's excerpts from the textbook in which I'm
23 the first author, called Cellular and Molecular
24 Immunology, Ninth Edition.

25 Q. Can you turn to Chapter 19, page 430.

1 A. 430.

2 Q. It is excerpts of the book, but it's the
3 complete Chapter 19, among other complete chapters.

4 A. Yeah.

5 Q. On page 430, you're discussing pathogenesis and
6 therapeutic strategies for selected immunological
7 diseases; correct?

8 A. Uh-huh.

9 Q. And on 430, in particular, you're discussing
10 systemic lupus erythematosus; correct?

11 A. Correct.

12 Q. And we'll just call it lupus?

13 A. I'm sorry?

14 Q. Can we just call it lupus?

15 A. Call it lupus.

16 Q. And that is one of your specialties; is that
17 correct?

18 A. No.

19 Q. You had used the C3H mouse model for --

20 A. No, (NZB X NZW) mouse model for lupus.

21 I interrupted you, but I think that's what you
22 were asking.

23 Q. Sure.

24 On the right-hand side of page of 430, you have
25 a heading "New therapies for systemic lupus

1 erythematosus"; correct?

2 A. Correct.

3 Q. And you refer to clinical trials that are
4 underway to test the efficacy of antibodies against
5 IFN-alpha or its receptor in the disease.

6 Do you see that?

7 A. Correct.

8 Q. Do you have an understanding of what was known
9 prior to April 2017 as to why antibodies against
10 IFN-alpha or its receptor were thought to have
11 potential to treat human lupus?

12 MR. MILLS: Objection to form and scope.

13 THE WITNESS: I can tell you the basis for
14 this. It's not relevant to my declaration.

15 BY MR. FELDSTEIN:

16 Q. Okay.

17 A. But many -- several investigators had shown by
18 then that IFN-alpha, which is different from
19 IFN-gamma -- IFN-alpha production was elevated in lupus
20 patients and immune cells from lupus patients made
21 exaggerated -- or excessive amounts of IFN-alpha. And
22 so that was the basis for the clinical trial.

23 Since the time the book was -- this edition was
24 written, IFN-alpha antibodies have been approved for
25 the treatment of lupus. So it's now an approved

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1 therapy.

2 Q. And in terms of what you were explaining, that
3 investigators had found elevated levels of IFN-alpha in
4 lupus patients, that's similar, at least conceptually,
5 to what we saw in Exhibit 1030, looking to elevated
6 cytokines there as a disease marker; is that correct?

7 MR. MILLS: Objection to form and scope.

8 THE WITNESS: Conceptually, yes, but the
9 technology used to detect increased production of
10 IFN-alpha was completely different. It was all based
11 on gene expression and transcripts, not on measuring
12 IFN-alpha in the serum of patients.

13 BY MR. FELDSTEIN:

14 Q. But in a simplified manner, what's common to
15 both Exhibit 1030 and what you're describing for lupus
16 is that they both started by looking at elevated
17 cytokines?

18 MR. MILLS: Objection to form and scope.

19 THE WITNESS: In principle, yes, elevated
20 production or action of cytokines.

21 BY MR. FELDSTEIN:

22 Q. Besides GvHD and lupus, prior to April 2017,
23 had others, to your knowledge, looked for elevated
24 cytokines associated with the disease -- with a disease
25 as a way to elucidate the disease mechanism?

1 MR. MILLS: Objection to form and scope.

2 THE WITNESS: Many examples. The most -- the
3 most dramatic is rheumatoid arthritis, where elevated
4 levels of TNF were found.

5 And subsequently -- I don't remember the exact
6 date, but I believe it was well before 2017 -- elevated
7 production of IL-17 in psoriasis lesions. That
8 directly led to the clinical trial of anti-IL-17 in
9 psoriasis, which is extremely successful.

10 BY MR. FELDSTEIN:

11 Q. So at a simplistic level, what you are
12 describing is finding elevated cytokines associated
13 with the disease and then using blockers of that
14 cytokine signaling as a methodology to treat the
15 disease; is that correct?

16 MR. MILLS: Objection to form. Scope.
17 Incomplete hypothetical.

18 THE WITNESS: Yes.

19 BY MR. FELDSTEIN:

20 Q. And is that a fairly standard, at a high level,
21 methodology for researching cytokine-dependent
22 diseases?

23 MR. MILLS: Objection to form, scope, and
24 incomplete hypothetical.

25 THE WITNESS: To my knowledge, yes.

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1 BY MR. FELDSTEIN:

2 Q. So we make good use of your textbook, if you
3 could turn a little farther, to page 432. It's also --
4 still part of Chapter 19.

5 In the bottom left, you're addressing new
6 therapies for rheumatoid arthritis.

7 A. Yes.

8 Q. Do you see that?

9 A. Yes. Yes.

10 Q. And you say, consistent with what you said a
11 minute ago, that chief among these new therapies are
12 antagonists of TNF?

13 A. Yes.

14 Q. And just to clarify the record, what does it
15 mean to be an antagonist of TNF?

16 A. Blocker.

17 Q. And so it's either a blocker of TNF or the TNF
18 receptor?

19 A. In this case, they use blockers of TNF only,
20 not against the receptor.

21 Q. Why is that?

22 A. It becomes complicated. There are two
23 receptors, and it's much easier to block the cytokine.
24 There's only one cytokine.

25 Q. And at a high level, was the research that led

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1 to the antagonism of TNF alpha for rheumatoid arthritis
2 based on finding that TNF -- excuse me; not TNF alpha,
3 just TNF -- was elevated in rheumatoid arthritis and
4 then testing what happens when you inhibit TNF
5 signaling?

6 MR. MILLS: Objection to form, scope,
7 incomplete hypothetical.

8 THE WITNESS: As it so happens in this one
9 disease, no. It was based on an animal model in which
10 a group in London showed that if you block TNF, you
11 ameliorate the disease, in the mouse model.

12 BY MR. FELDSTEIN:

13 Q. And that was without previously determining
14 that TNF was associated with rheumatoid arthritis?

15 A. Well, increased levels of TNF had been
16 described in the synovial fluid of patients with
17 rheumatoid arthritis. But there were increased levels
18 of multiple cytokines, and that group chose to focus on
19 TNF.

20 Q. So they went into -- the London group went into
21 an animal model without -- without having first
22 determined that TNF was a unique marker of rheumatoid
23 arthritis? Am I understanding correctly?

24 A. That is correct. It was not a unique marker.

25 Q. But the animal model determined that antagonism

1 of TNF was --

2 A. Effective.

3 Q. Effective.

4 MR. MILLS: Let him finish the question.

5 THE WITNESS: Sorry. My fault.

6 MR. MILLS: So my objection to the question:
7 Form, scope, incomplete hypothetical.

8 BY MR. FELDSTEIN:

9 Q. And then continuing on, the next column on page
10 432, you say, "Blockade of cytokines other than TNF has
11 been effective." And then it continues.

12 Do you see that?

13 A. Correct.

14 MR. MILLS: Objection to form and scope.

15 BY MR. FELDSTEIN:

16 Q. Can you explain how -- well, what was known by
17 April 2017 about blockade of other cytokines other than
18 TNF being effective for the treatment of rheumatoid
19 arthritis?

20 MR. MILLS: Objection to form and scope.

21 THE WITNESS: Just what's stated there, that
22 blocking IL-16 (sic) by blocking the -- with an
23 antibody against the receptor or blocking IL-1 with a
24 different drug -- it's not an antibody; it's an
25 inhibitor -- all had some beneficial effect in RA.

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1 BY MR. FELDSTEIN:

2 Q. And so was it known that IL-6 was elevated in
3 rheumatoid arthritis disease?

4 MR. MILLS: Objection to form and scope.

5 THE WITNESS: I don't know the answer. I do
6 know that it was known that IL-1 was elevated. I don't
7 know the answer about IL-6.

8 BY MR. FELDSTEIN:

9 Q. Okay. Can put the book aside.

10 Can you turn to Exhibit 1022.

11 You're familiar with Exhibit 1022, Doctor?

12 A. Yes.

13 Q. Can we turn to the "Conclusion" of the paper.

14 Well, let me just ask. What, generally, do you
15 understand Exhibit 1022 to be about?

16 A. It's a review-type article. It's not primary
17 data. It's a review article, or what they call a
18 commentary, on the potential of the IL-15/IL-15
19 receptor system blockade in auto- -- in inflammatory
20 diseases.

21 Q. And in the "Conclusion" on page 176 of the
22 article --

23 A. Yeah.

24 Q. -- it begins:

25 "Our emerging understanding of the

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1 IL-15/IL-15 receptor system is providing the
2 scientific basis for the development of
3 rational approaches for the IL-15 targeted
4 treatment of autoimmune diseases," and then it
5 continues.

6 Do you see that?

7 A. Correct.

8 Q. Can you explain what that means:

9 "Scientific basis for the development of
10 rational approaches for the IL-15 targeted
11 treatment of autoimmune diseases".

12 A. Well, "scientific basis" means that there is a
13 basis -- there is a reason for considering IL-15 as a
14 target based on experimentation or other analysis. So
15 that's what we refer to as the "scientific basis."

16 And the "rational" means that those approaches
17 provide enough information that you can rationally
18 contemplate coming up with a therapy.

19 Q. And so the scientific basis would be finding
20 that, for example, a disease is associated with
21 elevated IL-15?

22 MR. MILLS: Objection to form, scope, and
23 incomplete hypothetical.

24 THE WITNESS: That's part of it, or blocking
25 experiments in reliable animal models would be another

1 part of it. That's how these things are analyzed. So
2 finding it elevated in patients, in mouse models, then
3 blocking it in mouse models, those are all the pieces
4 we consider.

5 BY MR. FELDSTEIN:

6 Q. And then the next step of the rational design;
7 how does that come in?

8 A. Well, the next step would be to block that
9 cytokine pathway in an animal model, and that's your
10 proof of concept -- your pre-clinical proof of concept.
11 And then from there, you would consider going to
12 clinical trials.

13 Q. So is that a fairly standard approach, that you
14 have a scientific basis followed by rational design
15 followed by proof of concept followed by clinical
16 trials?

17 MR. MILLS: Objection to form, scope,
18 incomplete hypothetical.

19 THE WITNESS: It's -- in principle, that's the
20 standard approach, yeah.

21 BY MR. FELDSTEIN:

22 Q. For, essentially, any disease, there's going to
23 be some uncertainty going from the proof of concept in
24 an animal model, for example, to human clinical trials;
25 correct?

1 MR. MILLS: Objection to form, scope,
2 incomplete hypothetical.

3 THE WITNESS: Yes, to my knowledge.

4 BY MR. FELDSTEIN:

5 Q. Now, Exhibit 1022 is from 2004. Do you see
6 that? It is dated 2004 above the "Abstract."

7 A. Yeah.

8 Q. Do you have an understanding of how, if at all,
9 targeting the IL-15/IL-15R system further developed
10 through April 2017?

11 A. Not a detailed understanding. I do know -- and
12 there's another article by Tom Waldmann in this set of
13 references -- that they made an anti-IL-15 -- they had
14 their own anti-CD122 antibody. I don't know what
15 diseases they tested it in.

16 I think his initial interest was to test it in
17 leukemias derived from natural killer cells. In the
18 clinical world, they're called large granular
19 lymphocytic leukemias, LGL leukemias. He was very
20 interested in doing that, but I don't know how far the
21 clinical trials went. I just don't know the data.

22 Q. Okay. Can we turn to Exhibit 1015, reference
23 by Jabri.

24 You're familiar with Exhibit 1015, Doctor?

25 A. I had read it. I have not refreshed my memory.

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1 This is another one of these review articles.

2 Q. What is your general understanding of what's
3 addressed in Exhibit 1015?

4 A. I think this is the -- the broad issue is the
5 biology of Interleukin-15. And Bana suggests that it's
6 involved in what she calls a danger signal which is
7 telling the immune system to react and, in this
8 particular case, telling T cells to react.

9 Q. Can you turn to the second page of the article,
10 page 772, Figure 1.

11 A. Yeah.

12 Q. Can you explain what's depicted on the top
13 right corner with the green blob above the red circle?

14 A. Come again? This transpresentation thing?

15 Q. Correct.

16 A. This is the idea that has developed with IL-15
17 that a cell that expresses the alpha chain of the
18 receptor binds the cytokine and presents it to the
19 T cell or the NK cell that expresses the betagamma
20 complex.

21 This is an unusual model of cytokine activity,
22 this trans- -- one cell is -- presenting the cytokine
23 to another cell is very unusual.

24 Q. It's unusual among cytokines, or it's unusual
25 for IL-15?

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1 A. It's unusual among cytokines.

2 Q. It's common -- the transpresentation is common
3 for IL-15; correct?

4 A. That's correct. That's what is believed, yes,
5 that's correct.

6 Q. And in the transpresentation for IL-15, is it
7 typical that it's the IL-15 receptor alpha on one side
8 and the beta and gamma portions on the other side?

9 A. That's the theory, yes.

10 Q. And so to inhibit the transpresentation, you
11 could do something to antagonize IL-15 directly; is
12 that correct?

13 A. That's correct.

14 Q. You could do something to antagonize IL-15R
15 beta; correct?

16 A. That's correct. But because, as it says here,
17 it's shared by IL-2, which is usually not
18 transpresented but the cell expresses the trimeric
19 receptor in IL-2, that would block IL-2 also.

20 Q. Just focusing on IL-15 specifically, one way to
21 block the IL-15 transpresentation is to antagonize the
22 IL-15 receptor beta portion?

23 A. Theoretically, yes.

24 Q. And a third way to antagonize the -- a third
25 way to inhibit the transpresentation for IL-15 is to

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1 antagonize the gamma portion; correct?

2 A. Also correct.

3 Q. And any of those would inhibit the
4 transpresentation that's shown; correct?

5 A. Theoretically, yes.

6 MR. MILLS: If you are moving on to another
7 document, do you want to take a break?

8 MR. FELDSTEIN: That sounds fine.

9 (Recess taken.)

10 BY MR. FELDSTEIN:

11 Q. As of April 2017, are you aware of how many
12 species there were that were known to antagonize IL-15
13 signaling?

14 MR. MILLS: Objection to form.

15 THE WITNESS: No.

16 BY MR. FELDSTEIN:

17 Q. A ballpark?

18 A. Of course, I -- I'm just trying to think. Not
19 really -- I know about CD122 blockers, but I am not
20 sure if alpha chain blockers or cytokine blockers had
21 been developed by then. I don't know.

22 Q. You hadn't looked into that in the context of
23 your report in this case; correct?

24 A. No, I had not.

25 Q. About how many CD122 blockers were there known

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1 as of April 2017?

2 A. Well, I'm aware of three published. One is the
3 one in the patent. One is the one developed by Tom
4 Waldmann's lab. And I think the one in the Xing paper
5 is different from those two, but I cannot be a hundred
6 percent sure. I think it's different.

7 Q. Okay.

8 A. But there may have been many more because these
9 were commercially available by then, the antibodies --
10 at least against the mouse, CD122 were commercially
11 available. So there may be more.

12 Q. Okay. Can we look at Exhibit 1011.

13 A. That's the patent?

14 Q. It's the Tso patent, T-s-o.

15 A. Yes, yes.

16 Q. Exhibit 1011 is an example of antibodies known
17 as of April 2017 that block IL-15 signaling?

18 MR. MILLS: Objection to form.

19 THE WITNESS: It's one antibody.

20 BY MR. FELDSTEIN:

21 Q. Exhibit 1011, is that what you are looking at?

22 A. Exhibit 1011 is the patent.

23 Q. 1001 is the patent. Exhibit 1011 is what I am
24 referring to.

25 A. Oh, I'm sorry. I'm sorry.

1 Yes.

2 Q. So let me start again now that you're --

3 A. Yes.

4 Q. -- in Exhibit 1011. Are you familiar with
5 Exhibit 1011?

6 A. I read it.

7 Q. And Exhibit 1011 discloses an antibody known as
8 of April 2017 for antagonizing IL-15 signaling; is that
9 correct?

10 MR. MILLS: Objection to form.

11 THE WITNESS: That's correct.

12 BY MR. FELDSTEIN:

13 Q. Can you turn to column 4 in the patent. Are
14 you familiar with column numbers?

15 A. Got it.

16 Q. The line numbers in the middle, line 25.

17 A. Yeah.

18 Q. It's the paragraph that begins:

19 "The invention further provides methods of
20 suppressing an undesired immune response."

21 Do you see that?

22 A. Yes.

23 Q. And among the diseases listed are sclerosis,
24 rheumatoid arthritis, lupus, diabetes, psoriasis,
25 celiac disease, and uveitis; right?

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1 A. Correct.

2 Q. What would have been the basis of concluding
3 that an antagonistic of IL-15 signaling would treat
4 those diseases short of actual testing it in humans?

5 MR. MILLS: Objection to form, foundation,
6 scope.

7 THE WITNESS: I have no idea. No idea what
8 they base that on.

9 BY MR. FELDSTEIN:

10 Q. Was it common as of 2017 that if you created an
11 antagonist for a given pathway, you would reasonably
12 expect it to work where antagonism of that pathway had
13 shown efficacy in a proven concept model?

14 MR. MILLS: Objection to form, foundation,
15 incomplete hypothetical.

16 THE WITNESS: It depends on the reliability of
17 the proof of concept model, obviously.

18 BY MR. FELDSTEIN:

19 Q. Was it common -- to your knowledge, was it
20 common as of April 2017 to expect that species that
21 would antagonize IL-15 signaling would be useful in
22 diseases -- autoimmune diseases that had been
23 identified as having an IL-15 mechanism?

24 MR. MILLS: Objection to form, foundation,
25 incomplete hypothetical.

1 THE WITNESS: It's reasonable, but it depends
2 on how the mechanism was identified.

3 BY MR. FELDSTEIN:

4 Q. Is that different today than in April 2017,
5 about how people rely on proof of concepts to infer
6 treatment activity for an antagonist?

7 MR. MILLS: Objection. Form, foundation,
8 incomplete hypothetical, outside the scope.

9 THE WITNESS: As far as I know, the principles
10 have not changed.

11 MR. FELDSTEIN: Okay. I'm going to mark
12 Exhibit 2003.

13 THE WITNESS: 2003?

14 MR. FELDSTEIN: It's a new exhibit we are going
15 to mark.

16 THE WITNESS: 2003 is...

17 MR. FELDSTEIN: We're going to mark it.

18 THE WITNESS: Oh, got it.

19 MR. FELDSTEIN: Mark this 2003, please.

20 (Whereupon, Exhibit 2003 was marked for
21 identification.)

22 MR. FELDSTEIN: So let me just identify it, and
23 then feel free to do what you've got to do.

24 We have marked Exhibit 2003, a Forte
25 Biosciences press release from SEC files dated -- I'm

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1 sorry -- August 1, 2023.

2 Go ahead.

3 MR. MILLS: So I object to the document as
4 lacking foundation, lacking a demonstration of
5 authenticity, as being outside the scope of Dr. Abbas's
6 testimony.

7 BY MR. FELDSTEIN:

8 Q. Dr. Abbas, Forte Biosciences is the company for
9 whom your attorney is working; is that correct?

10 A. That -- I've seen that, yes.

11 Q. Yeah. And are you familiar with Forte
12 Biosciences other than what you have written in your
13 report?

14 A. No.

15 Q. Are you familiar with -- do you see there's an
16 underlined heading in the middle of the page "FB-102
17 R&D Updates"?

18 A. Yeah, I see the heading.

19 Q. And it says, "FB-102 targets CD122, a subunit
20 of the IL-2 and the IL-15 receptors."

21 Do you see that?

22 MR. MILLS: I'm going to object to the
23 question. Outside the scope. This is not a document
24 that Dr. Abbas has relied on. It's not a document he's
25 testified on. And it seems like maybe what you are

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1 doing here is trying to read an irrelevant document
2 into the deposition transcript. So I don't believe
3 this is a basis for any of Dr. Abbas's testimony.

4 BY MR. FELDSTEIN:

5 Q. Dr. Abbas, are you familiar with FB-102?

6 MR. MILLS: Objection to scope.

7 THE WITNESS: No.

8 BY MR. FELDSTEIN:

9 Q. Do you see at the bottom it says:

10 "Published pre-clinical PoC" -- which I
11 assume is proof of concept -- "data also
12 supports the therapeutic potential of
13 anti-CD122 in multiple autoimmune diseases
14 anti-CD122 including vitiligo, celiac disease,
15 alopecia areata, type 1 diabetes, and solid
16 organ transplant."

17 Do you see that?

18 MR. MILLS: Objection. Outside the scope.

19 THE WITNESS: I see the statement.

20 BY MR. FELDSTEIN:

21 Q. Are you aware of any pre-clinical proof of
22 concept data to show that anti-CD122 has therapeutic
23 potential in organ transplant?

24 MR. MILLS: Objection. This is outside the
25 scope.

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1 THE WITNESS: I'm not aware of that.

2 BY MR. FELDSTEIN:

3 Q. Are you aware of any pre-clinical point
4 of care -- proof of concept data to show that
5 anti-CD122 would have therapeutic potential in
6 vitiligo?

7 MR. MILLS: Objection. Questions about this --
8 if you're asking about this document, the document is
9 outside the scope.

10 THE WITNESS: Only what was in the patent.

11 MR. FELDSTEIN: Going to mark the next
12 Exhibit 2004.

13 (Whereupon, Exhibit 2004 was marked for
14 identification.)

15 BY MR. FELDSTEIN:

16 Q. Doctor, you have been handed Exhibit 2004,
17 which I will identify as a submission to the United
18 States Securities and Exchange Commission with, on the
19 third page of the document -- or naming Forte
20 Biosciences as the submitter on the first page.

21 MR. MILLS: So I'm objecting to Exhibit 2004.
22 Like 2003, these are documents that are outside the
23 scope of Dr. Abbas's declaration. These are documents
24 that postdate the critical date. They're irrelevant to
25 the proceedings. And their use in this deposition

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1 appears to be an improper attempt to launder the
2 exhibits into the record, and we reserve the right to
3 move to strike this line of questioning as well as
4 these exhibits.

5 BY MR. FELDSTEIN:

6 Q. Doctor, can you move -- turn to the slide that
7 has a number 24 in very small print in the bottom right
8 corner. The slide -- if you go into it, there are
9 slides, and they have small print numbers in the bottom
10 right corner.

11 A. In my copy, I don't see any numbers at the
12 bottom. I don't know which one you're referring to.
13 Just hold it up and show it to me.

14 PB-something Overview?

15 Q. "FB-102 Overview."

16 A. Oh.

17 Q. It's maybe about ten physical pages in from the
18 back.

19 A. "FB-102 Overview." There is no number here.

20 Q. Very small print in the bottom right --

21 A. Oh, my gosh.

22 Q. -- in light blue. Right?

23 A. It's in light blue. I see it.

24 Q. Okay. So we are on the slide that is titled
25 "FB-102 Overview" with the very small "24" in light

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1 blue; correct?

2 MR. MILLS: So I renew my objection to this
3 exhibit and this line of questioning.

4 BY MR. FELDSTEIN:

5 Q. Doctor, you're on the same page as me?

6 A. Yes.

7 Q. Do you see the second bullet from the bottom?

8 MR. MILLS: Object to the question as outside
9 the scope.

10 THE WITNESS: I see the bullet.

11 BY MR. FELDSTEIN:

12 Q. "Mechanisms of FB-102 also show activity in
13 vitiligo and alopecia areata."

14 Do you see that?

15 MR. MILLS: Renew my objections to this exhibit
16 and line of questioning.

17 THE WITNESS: I can see that in the document,
18 yeah.

19 BY MR. FELDSTEIN:

20 Q. As of today, what is your understanding of the
21 knowledge of what mechanisms would attribute activity
22 in vitiligo to an anti-CD122 antibody?

23 MR. MILLS: Objection. This is outside the
24 scope.

25 THE WITNESS: Yeah, I have no comment on that.

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1 BY MR. FELDSTEIN:

2 Q. Anything besides the patent, to your knowledge?

3 MR. MILLS: Objection. This is outside the
4 scope and irrelevant.

5 THE WITNESS: Yeah, I prefer not to comment on
6 that.

7 BY MR. FELDSTEIN:

8 Q. You don't have knowledge of any mechanistic
9 analyses of anti-CD122 for vitiligo beyond what is in
10 the patent; is that correct?

11 MR. MILLS: Objection that questions about this
12 document are outside the scope and irrelevant.

13 THE WITNESS: Yeah, I do not have any personal
14 knowledge.

15 BY MR. FELDSTEIN:

16 Q. Okay. You can put that aside, Doctor.

17 Are you familiar with any small molecules -- if
18 I say "small molecules," we distinguish them from
19 antibodies; is that fair?

20 A. Correct.

21 Q. Okay. Are you familiar with any small molecule
22 antagonists of IL-15 signaling that were known prior to
23 April 2017?

24 MR. MILLS: Objection to form.

25 THE WITNESS: No. I mean, they may be there,

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1 you know, these JAK inhibitors, but I don't know which
2 ones specifically block IL-15 signaling.

3 BY MR. FELDSTEIN:

4 Q. That's not something you --

5 (Clarification requested by Reporter.)

6 MR. FELDSTEIN: I'll ask the question again.

7 Okay?

8 BY MR. FELDSTEIN:

9 Q. That's not something you investigated in
10 preparation for your report, Exhibit 1001; correct?

11 MR. MILLS: Objection to form.

12 THE WITNESS: That is correct.

13 BY MR. FELDSTEIN:

14 Q. What is your understanding of RNA technology to
15 antagonize IL-15 signaling -- let me actually withdraw
16 the question.

17 What is your understanding of RNA technology to
18 antagonize cytokine signaling generally?

19 MR. MILLS: Objection to form.

20 THE WITNESS: Not -- by "RNA technology," I
21 just want to be clear, you're referring to RNA
22 interference, short hairpin or short interfering RNA?
23 Because the other technology is antisense RNA. That's
24 totally different.

25 BY MR. FELDSTEIN:

1 Q. Well, let's do two buckets. Okay? So the
2 first bucket is what? Small interfering?

3 A. Small interfering or small hairpin. Those are
4 used interchangeably.

5 Q. And the other version is antisense?

6 A. Yes. And that's not mentioned in the patent.

7 Q. Okay.

8 A. So there are two types of RNA technology --

9 Q. Thank you.

10 A. -- and I just wanted to be clear you're
11 referring to the first one.

12 Q. Let me be specific in my question. I
13 appreciate your help.

14 As of April 2017, what is your understanding of
15 small interfering RNA technology to -- what is your
16 understanding of the knowledge in the art as of
17 April 2017 with respect to small interfering RNA to
18 antagonize cytokine signaling?

19 MR. MILLS: Objection to form.

20 THE WITNESS: I don't know. I have no
21 knowledge about that.

22 BY MR. FELDSTEIN:

23 Q. Is that something you investigated for your
24 work in this case and your declaration, Exhibit 1001?

25 MR. MILLS: Objection to form.

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1 THE WITNESS: No.

2 BY MR. FELDSTEIN:

3 Q. Are you aware of what small interfering RNA
4 species there were as of April 2017 to antagonize IL-15
5 signaling specifically?

6 MR. MILLS: Objection to form. Incomplete
7 hypothetical.

8 THE WITNESS: I am not aware of any.

9 BY MR. FELDSTEIN:

10 Q. And is that something you investigated for your
11 work in this case and your declaration, Exhibit 1001?

12 A. No.

13 MR. MILLS: Objection to form.

14 THE WITNESS: Sorry. I interrupted you.

15 BY MR. FELDSTEIN:

16 Q. Can you turn to Exhibit 1017.

17 A. '17.

18 Q. You're familiar with Exhibit 1017, Doctor?

19 A. I read it when I was preparing the declaration.
20 I have not re-read it.

21 Q. Feel free to refamiliarize yourself with the
22 document, but my question for you is whether you agree
23 that Exhibit 1017 does not demonstrate that inhibition
24 of an IL-15 cellular response treats vitiligo.

25 MR. MILLS: Objection to form.

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1 THE WITNESS: From what I can tell, that is
2 correct. It does not deal with vitiligo.

3 BY MR. FELDSTEIN:

4 Q. Going back a little bit to antagonists of IL-15
5 signaling, can you explain your understanding of what a
6 peptide inhibitor is in the context of antagonizing
7 cytokine signaling?

8 A. I don't know anything about peptide inhibitors
9 and their ability to antagonize -- I have not seen any
10 data on peptide inhibitors blocking cytokine signaling.

11 Q. Is that something you investigated for your
12 work in this case and your declaration, Exhibit 1001?

13 A. No.

14 MR. FELDSTEIN: Doctor, that's all the
15 questions we have right now.

16 MR. MILLS: You're done with questioning?

17 MR. FELDSTEIN: Yes.

18 MR. MILLS: Okay. Let's take a break.

19 (Recess taken.)

20 DIRECT EXAMINATION BY MR. MILLS:

21 Q. Dr. Abbas, do you recall being asked questions
22 by counsel about NKG2D+ CD8+ T cells that were
23 monitored in the Xing reference?

24 A. Yes.

25 Q. And do you remember being asked questions about

1 the Harris mouse model in the prior art, the Harris
2 reference?

3 A. Yes.

4 Q. What impact, if anything, does Xing's
5 monitoring of NKG2D+ expression have on your opinions
6 about the predicted effect of administering the CD122
7 antibody in Xing to Harris's mouse model?

8 MR. FELDSTEIN: Objection. Beyond the scope of
9 the cross. Form.

10 THE WITNESS: In my opinion, no impact, and the
11 reason -- I should explain why I say that.

12 NKG2D is used as a marker for activated CD8
13 T cells, the killers, the active killers. My
14 expectation is that the way the mouse model in the
15 patent is set up, a lot of the CD8 T cells will be
16 activated.

17 I couldn't find any data for NKG2D staining.
18 Maybe they all express NKG2D. Maybe a fraction of them
19 does. I simply don't know from the data provided.

20 So in my opinion, the CD122 antibody kills --
21 what Xing shows is that they kill -- it kills activated
22 T cells in the skin of the mouse. And I think in the
23 Harris model, they will be activated T cells in the
24 skin of the mouse.

25 So I expect the antibody will kill the

1 activated T cells in the Harris model. That's my
2 opinion.

3 BY MR. MILLS:

4 Q. When you refer to the antibody in Xing, what
5 antibody are you talking about?

6 A. Oh, it's -- I can't remember the name. It's
7 something -- TM-beta 1. TM-beta 1.

8 Q. And you were talking about your expectation.
9 What's the time frame for that expectation? Is that
10 the expectation that you would have had before the
11 critical date in 2017 or not?

12 MR. FELDSTEIN: Objection. Beyond the scope.
13 Form. Leading.

14 THE WITNESS: Yes. Because the paper was
15 published before 2017, the Xing paper.

16 BY MR. MILLS:

17 Q. And when you talk about the expectation before
18 the critical date in 2017, is that just your
19 expectation, or is it or is it not the expectation of a
20 person of ordinary skill in the art before the critical
21 date in 2017?

22 MR. FELDSTEIN: Objection. Beyond the scope of
23 the cross. Form.

24 THE WITNESS: My expectation is that a person
25 with average skill would understand that.

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1 BY MR. MILLS:

2 Q. Do you recall being asked questions about the
3 T cells in the Harris model having Pmel specificity?

4 A. Yes.

5 Q. What impact, if anything, does the Pmel
6 specificity of the T cells in Harris's mouse model have
7 on your expectation regarding the effects of
8 administering Xing's TM-beta 1 anti-CD122 antibody to
9 Harris's mouse model?

10 MR. FELDSTEIN: Objection. Beyond the scope of
11 the cross. Form.

12 THE WITNESS: The specificity of the T cells
13 should make no difference to whether the anti-CD122
14 antibody will work on those T cells or not.

15 BY MR. MILLS:

16 Q. And is that the expectation as of a
17 particular -- is that your expectation as of -- before
18 the critical date in 2017?

19 MR. FELDSTEIN: Same objections.

20 THE WITNESS: Yes.

21 BY MR. MILLS:

22 Q. And is that a private expectation that you have
23 or is it an expectation that a person of ordinary skill
24 in the art before the critical date in 2017 would
25 share?

1 MR. FELDSTEIN: Same objections.

2 THE WITNESS: A person of ordinary skill should
3 have that same expectation.

4 BY MR. MILLS:

5 Q. Do you recall being asked questions about the
6 Cheuk reference monitoring CD49a expression in T cells?

7 A. Yes. Yes.

8 Q. What impact, if any, does Cheuk's monitoring of
9 CD49a expression have on your expectation regarding the
10 impact of administering Xing's TM-beta 1 anti-CD122
11 antibody to Harris's mouse model?

12 MR. FELDSTEIN: Objection. Beyond the scope of
13 the cross. Form.

14 THE WITNESS: I don't think it should have any
15 impact.

16 BY MR. MILLS:

17 Q. Why is that?

18 A. Because --

19 MR. FELDSTEIN: Same objections.

20 THE WITNESS: Sorry.

21 Because CD49a is also an activation marker of
22 T cells; in this case, CD8 T cells. So I would expect
23 that it would be expressed on activated CD8 T cells in
24 both models.

25 BY MR. MILLS:

1 Q. Do you remember being asked questions about the
2 experiments in the '505 patent?

3 A. Yeah, the -- the experiments -- the major
4 experiments presented in the '505 patent deal with the
5 transgenic mouse model.

6 Q. The transgenic mouse model in the '505 patent,
7 does it bear any relationship to the Harris mouse model
8 in the prior art Harris reference?

9 A. That -- it's the same model we're talking
10 about, yeah.

11 Q. So the mouse model that's used in the '505
12 patent, in your opinion, from the perspective as an
13 immunologist, is that mouse model representative of
14 spontaneous or natural vitiligo?

15 MR. FELDSTEIN: Objection. Beyond the scope of
16 cross.

17 THE WITNESS: And in my opinion, it is not.
18 And there are numerous reasons, which I should explain.

19 First, in the mouse model, you have T cells of
20 a single specificity. In people, you have T cells of
21 unknown specificity.

22 Second, in the mouse model, the endogenous
23 immune cells have been significantly eliminated. I
24 don't know what fraction, but the majority have been
25 eliminated by prior irradiation. Obviously, in

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1 patients, you don't get rid of the immune cells.

2 Third, the T cells in the mouse model following
3 transfer are reactivated systemically by giving a virus
4 expressing the antigen. Well, you never have viruses
5 expressing cell tissue at skin antigens in a person.

6 So in every respect that I can think of, other
7 than the fact that you end up with vitiligo, these are
8 very different.

9 Now, I should address the question of what are
10 mouse models like this useful for.

11 My own lab used mouse models like this for
12 decades. We had an antigen. Sometimes we expressed
13 the antigen as a transgene. We had a T cell receptor
14 that recognized the antigen. We took T cells
15 expressing that receptor and gave them to a mouse
16 expressing the antigen, very similar to what is being
17 done here.

18 And we treated these models as extremely
19 valuable for asking fundamental mechanistic questions:
20 How does a T cell respond to an antigen? How does it
21 respond to an antigen in a tissue versus in
22 circulation?

23 But we never claimed -- literally, decades of
24 this. We never claimed that these models were
25 representative of a human disease. They're

1 representative of a transgenic mouse.

2 BY MR. MILLS:

3 Q. So I want to ask a follow-up question.

4 So when I asked you about the specificity of
5 the Pmels in the Harris model, you said that would have
6 no impact on your expectation that Xing's specific
7 antibody would eliminate those T cells.

8 But when I asked you about whether the vitiligo
9 in Harris was representative of spontaneous or natural
10 vitiligo, you mentioned the specificity.

11 So can you explain why you're talking about --
12 you're saying it's important for one but not the other?

13 MR. FELDSTEIN: Objection. Beyond the scope.
14 Leading. Form.

15 THE WITNESS: When I talked about specificity,
16 I was really referring to the fact that in the mouse
17 model, all the T cells are specific for one antigen.
18 In people, they obviously are not.

19 But they're all CD8 T cells, and all CD8
20 T cells -- or the majority of CD8 memory cells are
21 dependent on IL-15. So it doesn't matter what their
22 specificity is. If you block IL-15, you will get rid
23 of a lot of the memory CD8 cells.

24 BY MR. MILLS:

25 Q. And why is it relevant -- I understand you to

1 be saying that it's relevant to the Harris model that
2 you are getting rid of the CD8+ T cells. Why is that
3 relevant to the Harris mouse model?

4 MR. FELDSTEIN: Objection. Beyond the scope.
5 Form. Leading.

6 BY MR. MILLS:

7 Q. If it is?

8 MR. FELDSTEIN: Same objections.

9 THE WITNESS: It's relevant because in the
10 Harris mouse model, the only T cells that are present
11 are the CD8 T cells. That's what's being put into the
12 mouse. So if you get rid of them, you'll cure the
13 disease. To me, this is a -- this is a completely
14 predictable result.

15 BY MR. MILLS:

16 Q. I want to ask you about the antibody that's
17 used in the '505 patent. Do you recall how that
18 antibody is referred to?

19 MR. FELDSTEIN: Objection. Beyond the scope.

20 THE WITNESS: The name of the antibody is,
21 like, ChMBC7 or something like that.

22 BY MR. MILLS:

23 Q. Other than the ChMBC7 antibody, the specific
24 one that's used in the '505 patent, is there any other
25 type of IL-15 signalling inhibition that is

1 demonstrated in the '505 patent?

2 MR. FELDSTEIN: Objection. Beyond the scope.
3 Form.

4 THE WITNESS: None that I saw.

5 BY MR. MILLS:

6 Q. And do you recall being asked questions by
7 counsel about your silencing RNA, small molecules,
8 peptides, et cetera?

9 A. Yes.

10 Q. You recall being asked questions about your
11 investigation, if any, into those different categories
12 of inhibitors?

13 A. Yes.

14 Q. So my first question is, is the ChMBC7 antibody
15 that's used in the '505 patent -- is it representative
16 of using RNA to inhibit IL-15 signaling?

17 MR. FELDSTEIN: Objection. Beyond the scope.
18 Form.

19 THE WITNESS: Not from what I can tell.

20 BY MR. MILLS:

21 Q. Why not?

22 MR. FELDSTEIN: Objection. Beyond the scope.
23 Form.

24 THE WITNESS: Because they work in completely
25 different ways. The antibody blocks the receptor. The

1 interfering RNA blocks the expression of the receptor.
2 We have no idea whether they'll have the same effect or
3 not.

4 BY MR. MILLS:

5 Q. The ChMBC7 antibody used in the '505 patent, is
6 it representative of using a small molecule to inhibit
7 IL-15 signaling?

8 MR. FELDSTEIN: Objection. Beyond the scope.
9 Form.

10 THE WITNESS: No.

11 BY MR. MILLS:

12 Q. Why not?

13 MR. FELDSTEIN: Same objections.

14 THE WITNESS: Because the small molecules, at
15 least the ones described here, inhibit downstream
16 signaling pathways from IL-15. Many of those pathways,
17 by the way, overlap with signaling by other receptors.
18 The antibody blocks the IL-15 receptor beta chain only.

19 BY MR. MILLS:

20 Q. Is the ChMBC7 antibody that's used in the '505
21 patent representative of using a peptide inhibitor to
22 inhibit IL-15 signaling?

23 MR. FELDSTEIN: Objection. Beyond the scope.
24 Form.

25 THE WITNESS: Not as far as I can tell.

1 BY MR. MILLS:

2 Q. Why not?

3 MR. FELDSTEIN: Same objections.

4 THE WITNESS: Because we don't know where the
5 peptide inhibitor will be -- will work, and I can't
6 relate it to how the antibody works.

7 BY MR. MILLS:

8 Q. Is the ChMBC7 antibody used in the '505 patent
9 representative of using an anti-IL-15 antibody to
10 inhibit IL-15 signaling?

11 MR. FELDSTEIN: Objection. Beyond the scope.
12 Form.

13 THE WITNESS: No.

14 BY MR. MILLS:

15 Q. Why not?

16 A. Because the antibody blocks one component of
17 the receptor, which, by the way, is shared with IL-2
18 also. The anticytokine specifically blocks that
19 cytokine.

20 Q. Is the ChMBC7 antibody used in the '505 patent
21 representative of using an IL-15 alpha antibody to
22 block IL-15 signaling?

23 MR. FELDSTEIN: Objection. Beyond the scope.
24 Form.

25 THE WITNESS: No. Because the IL-15 receptor

1 alpha blocking will only affect IL-15 and the beta
2 chain blocking, as used in this, can affect IL-2
3 signaling also.

4 BY MR. MILLS:

5 Q. Is the ChMBC7 Fc-diminished murine antibody
6 used in the '505 patent representative of all IL-15R
7 beta antibodies that could be used to block IL-15
8 signaling?

9 MR. FELDSTEIN: Objection. Beyond the scope.
10 Form.

11 THE WITNESS: Not necessarily, because it's
12 going to depend on where the antibody binds and what
13 the affinity of binding is and, possibly, unknown
14 factors.

15 BY MR. MILLS:

16 Q. Is the ChMBC7 antibody used in the '505 patent
17 representative of using an IL-15R gamma antibody to
18 inhibit IL-15 signaling?

19 MR. FELDSTEIN: Objection. Beyond the scope.
20 Form.

21 THE WITNESS: Definitely not, because the gamma
22 chain -- common gamma chain blocking will block
23 responses to six or seven cytokines and the ChMBC7
24 antibody will block only cytokines that bind to -- that
25 use the beta chain.

1 BY MR. MILLS:

2 Q. You testified earlier, a moment ago, about --
3 that in the Harris mouse model, the CD8+ T cells are
4 causing the lesions and so getting rid of those T cells
5 is predictable to get rid of the lesions. Do you
6 recall that?

7 A. Yes.

8 Q. Do you recall being asked by counsel questions
9 about using interferon-gamma for treating alopecia and
10 vitiligo?

11 MR. FELDSTEIN: Objection. Form.

12 THE WITNESS: Yes. I remember questions, yes.

13 BY MR. MILLS:

14 Q. Specifically, you were asked about whether the
15 Xing reference tells you that blocking interferon-gamma
16 could be a method of treating alopecia. Do you recall
17 that?

18 A. Correct. Yes.

19 Q. So your testimony about that, does that
20 undermine the testimony that you just gave us that for
21 the Harris mouse model of vitiligo, it would be
22 reasonably predicted that administering the Xing
23 antibody would reverse the lesions?

24 MR. FELDSTEIN: Objection. Beyond the scope.
25 Form. Leading.

1 THE WITNESS: No.

2 BY MR. MILLS:

3 Q. Why not?

4 MR. FELDSTEIN: Same objections.

5 THE WITNESS: Because blocking gamma interferon
6 is completely different from getting rid of the CD8
7 T cells. So I don't think that -- I think comparing
8 the two is very much apples and oranges.

9 BY MR. MILLS:

10 Q. With regard to the Harris mouse model, why is
11 it that you are focused on getting rid of the CD8+
12 T cells?

13 MR. FELDSTEIN: Objection. Beyond the scope.
14 Form. Leading.

15 THE WITNESS: Because they're the only T cells
16 in the mouse and they have to be the cause of the
17 disease.

18 BY MR. MILLS:

19 Q. Do you recall being asked questions by counsel
20 about other entities using a CD132 antibody to treat
21 GVHD?

22 A. Yes. We talked about one paper in which the
23 gamma chain antibody was used to treat graft-versus-
24 host disease.

25 Q. So if it were correct that a CD132 antibody had

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1 been used successfully to treat graft-versus-host
2 disease, does that impact your testimony about the --
3 about whether the patent's use of the ChMBC7 antibody
4 would be expected to successfully treat spontaneous or
5 natural vitiligo?

6 MR. FELDSTEIN: Objection. Beyond the scope.
7 Form.

8 THE WITNESS: No.

9 BY MR. MILLS:

10 Q. Why not?

11 A. Apples and --

12 MR. FELDSTEIN: Objection --

13 (Clarification requested and record read by
14 Reporter.)

15 THE WITNESS: No impact, in my opinion.

16 BY MR. MILLS:

17 Q. And then I asked, why not?

18 MR. FELDSTEIN: Objection. Beyond the scope
19 and form.

20 THE WITNESS: And the answer is that using
21 CD132 antibodies to treat GvH has nothing to do with
22 getting rid of CD8 T cells in this mouse model.

23 BY MR. MILLS:

24 Q. And why are you focused on getting rid of CD8+
25 T cells in the patent's mouse model?

1 MR. FELDSTEIN: Objection. Beyond the scope.
2 Form. Asked and answered.

3 THE WITNESS: Because, again, those are the
4 only T cells that are present in the mouse and they
5 have to be causing the disease.

6 BY MR. MILLS:

7 Q. Do you recall being asked questions by counsel
8 about the use of TNF to treat rheumatoid arthritis?

9 A. Yes.

10 Q. So if it were correct that a TNF antibody had
11 been used to successfully treat rheumatoid arthritis,
12 does that impact your testimony about whether the
13 patent's use of the ChMBC7 antibody would successfully
14 treat spontaneous or natural vitiligo?

15 MR. FELDSTEIN: Objection. Beyond the scope.
16 Form. Leading.

17 THE WITNESS: No impact. They're unrelated.

18 BY MR. MILLS:

19 Q. Why not?

20 MR. FELDSTEIN: Same objections.

21 THE WITNESS: Because TNF blocking in
22 rheumatoid arthritis has nothing to do with getting rid
23 of CD8 T cells in this mouse model.

24 BY MR. MILLS:

25 Q. And why are you focused on getting rid of CD8+

1 T cells in this mouse model?

2 MR. FELDSTEIN: Objection. Beyond the scope.
3 Form. Asked and answered.

4 THE WITNESS: Because those are the only
5 T cells present in the mouse model and must be causing
6 the lesions.

7 BY MR. MILLS:

8 Q. Before the critical date in 2017, was a person
9 of ordinary skill in the art aware that the CD8+
10 T cells were the only T cells present in the mouse
11 model and must be causing the lesions?

12 MR. FELDSTEIN: Objection. Beyond the scope.
13 Form.

14 THE WITNESS: In my opinion, yes, a person of
15 ordinary skill should be aware of that.

16 BY MR. MILLS:

17 Q. What would a person of ordinary skill in the
18 art have predicted before the critical date in 2017
19 would be the impact of administering the TM-beta 1
20 antibody from Xing to the Harris mouse model?

21 MR. FELDSTEIN: Same objections.

22 THE WITNESS: Since we know the antibody gets
23 rid of CD8 T cells from the skin -- the memory CD8
24 T cells from the skin, the expectation would be that it
25 works in the -- in the Harris mouse model.

1 BY MR. MILLS:

2 Q. And when you say, "it works in the Harris mouse
3 model," what do you mean?

4 MR. FELDSTEIN: Same objections.

5 THE WITNESS: It gets rid of the T cells and
6 alleviates the lesions.

7 BY MR. MILLS:

8 Q. And is the alleviation of the lesions going to
9 be transient or durable or something different?

10 MR. FELDSTEIN: Objection. Beyond the scope
11 and form.

12 Go ahead.

13 THE WITNESS: It's actually a complicated
14 situation that I have got to explain.

15 From what I can tell in the mouse model, the
16 T cells were given only once, they were not repeatedly
17 administered to the mouse, which means if you get rid
18 of the memory T cells, there's nothing to come back.

19 So then you would predict that that has a
20 long-term effect --

21 MR. MILLS: Thank you --

22 THE WITNESS: -- but that's a reflection of the
23 model more than anything else.

24 MR. MILLS: Thank you. No further questions.

25 RECROSS-EXAMINATION BY MR. FELDSTEIN:

1 Q. Doctor, is it fair to say that during the
2 break, you discussed the testimony you just presented
3 with your counsel?

4 A. Yes. Mr. Mills told me the questions he was --
5 he was going to ask, and I told him how I was going to
6 respond.

7 MR. FELDSTEIN: Okay. No questions. Thank
8 you.

9 MR. MILLS: Thank you.

10 The deposition is over. The witness reserves
11 to right to review the transcript and sign.

12 (Whereupon, the deposition was
13 concluded at 1:20 p.m.)
14
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25

1 STATE OF CALIFORNIA

2
3 REPORTER'S CERTIFICATE

4
5 I, EARLY LANGLEY, a Shorthand Reporter, State
6 of California, do hereby certify:

7 That ABUL K. ABBAS, M.D., in the foregoing
8 deposition named, was present and by me sworn as a
9 witness in the above-entitled action at the time and
10 place therein specified;

11 That said deposition was taken before me at
12 said time and place, and was taken down in shorthand by
13 me, a Certified Shorthand Reporter of the State of
14 California, and was thereafter transcribed into
15 typewriting, and that the foregoing transcript
16 constitutes a full, true and correct report of said
17 deposition and of the proceedings that took place;

18 That before completion of the proceedings,
19 review of the transcript was requested.

20 IN WITNESS WHEREOF, I have hereunder subscribed
21 my hand this September 15, 2023

22 

23
24 EARLY LANGLEY, CSR NO. 3537

25 State of California

1 Jad Mills

2 Jmills@wsgr.com

3 September 15, 2023

4 RE: Forte Biosciences, Inc. v. University Of Massachusetts

5 9/13/2023, Abul Abbas, M.D. (#6083960)

6 The above-referenced transcript is available for
7 review.

8 Within the applicable timeframe, the witness should
9 read the testimony to verify its accuracy. If there are
10 any changes, the witness should note those with the
11 reason, on the attached Errata Sheet.

12 The witness should sign the Acknowledgment of
13 Deponent and Errata and return to the deposing attorney.
14 Copies should be sent to all counsel, and to Veritext at
15 clientservices-yom@veritext.com.

16
17 Return completed errata within 30 days from
18 receipt of testimony.

19 If the witness fails to do so within the time
20 allotted, the transcript may be used as if signed.

21
22 Yours,

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24
25

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Forte Biosciences, Inc. v. University Of Massachusetts
Abul Abbas, M.D. (#6083960)

E R R A T A S H E E T

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Abul Abbas, M.D. Date

1 Forte Biosciences, Inc. v. University Of Massachusetts
2 Abul Abbas, M.D. (#6083960)

3 ACKNOWLEDGEMENT OF DEPONENT

4 I, Abul Abbas , M.D., do hereby declare that I
5 have read the foregoing transcript, I have made any
6 corrections, additions, or changes I deemed necessary as
7 noted above to be appended hereto, and that the same is
8 a true, correct and complete transcript of the testimony
9 given by me.

10
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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS

COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

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