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

)
FORESIGHT DIAGNOSTICS)
INC.,)
) Case No.
Petitioner,) IPR2023-00545
) U.S. Patent No.
V.) 11,299,783
)
PERSONALIS, INC.,)
)
Patent Owner.)
-----)

ZOOM DEPOSITION OF HENRY MORRICE FURNEAUX
Hartford, Connecticut
Friday, January 26, 2024
Volume I

Reported by:
LORI M. BARKLEY
CSR No. 6426
Job No. 6412559
PAGES 1 - 213

1 UNITED STATES PATENT AND TRADEMARK OFFICE
2 BEFORE THE PATENT TRIAL AND APPEAL BOARD
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5 _____)
6 FORESIGHT DIAGNOSTICS)
7 INC.,)
8)
9 Petitioner,)
10) Case No.
11 V.) IPR2023-00545
12) U.S. Patent No.
13 PERSONALIS, INC.) 11,299,783
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15 Patent Owner.)
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Deposition of HENRY MORRICE FURNEAUX, Volume I,
taken on behalf of PETITIONER, at Hartford, Connecticut,
beginning at 7:10 a.m. and ending at 2:34 p.m. on
Friday, January 26, 2024, before LORI M. BARKLEY,
Certified Shorthand Reporter No. 6426.

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I N D E X

WITNESS

HENRY MORRICE FURNEAUX

PAGE

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EXHIBITS

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INSTRUCTION NOT TO ANSWER

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1 Hartford, Connecticut; Friday, January 26, 2024

2 7:10 a.m. Pacific time

3
4
5 HENRY MORRICE FURNEAUX,
6 having been administered an oath, was examined and
7 testified as follows:

8
9 EXAMINATION

10
11 BY MS. SELLERS:

12 Q. Good morning, Dr. Furneaux. How are you today?

13 A. Well, I'm pretty good. I'm well recovered, I
14 hope, from the COVID experience.

15 I should let you know that one of the post-COVID
16 sequela that I've noticed before is this excessive
17 saliva, which tends sometimes to distort my speech.

18 So I'm going to try and speak even more slowly
19 than I usually do. But, you know, we'll see how it
20 goes.

21 Q. Great. It's good to meet you. I'm glad you're
22 feeling better. I'm Abby Sellers. And I'm from Irell &
23 Manella and I represent Foresight Diagnostics.

24 And with me today we also have Andy Krause who
25 you've met before from Irell & Manella, who also

1 represents Foresight Diagnostics.

2 A. Very good.

3 Q. Do you understand today that you're here in
4 connection with IPR 20230046, which relates to US Patent
5 Number 10,450,611?

6 A. I do. Let me actuate just inform you I have my
7 declaration in front of me right now in paper format in
8 this white binder.

9 Yeah, that's correct, '545 and '546; is that
10 correct?

11 Q. Correct, and the '611 and '783 patents?

12 A. That is correct, yes.

13 Q. And are you okay today if we refer to those as
14 '611 and '783 patents?

15 A. Sure, absolutely.

16 Q. All right, perfect.

17 A. I'm just wondering about your Zoom etiquette. I
18 still have bits and pieces floating around here.

19 Should I place my phone down.

20 Q. If you could place that over to the side. We'll
21 get to that in one second.

22 Do you understand you're under oath and this
23 oath carries the same obligations as if you were in
24 court?

25 A. I do.

1 Q. And Lori already touched on this, but she will
2 be writing down your answers today and my questions, and
3 providing them afterwards in a booklet.

4 While it's difficult because we're on Zoom and
5 it can be easy it to talk over each other. For Lori,
6 please try to refrain from speaking over one another.

7 We'll try our best today but it obviously is a
8 little more difficult in this format.

9 And you've been deposed before, correct?

10 A. That's correct. Actually by your firm in the
11 last go around, the last two cases.

12 Q. Correct. And so you've been deposed by our
13 firm. And how many times other than in connection with
14 the Personalis IPR have you been deposed before?

15 A. I think this present one may well be my fifth.
16 Either fourth or fifth, I can't recall.

17 Q. Today I'll be asking you a series of questions
18 as you know. If at any point, you don't understand a
19 question, please let me know.

20 If you don't say anything, I'll assume you
21 understand the question. And if you need a break at any
22 time, just let me know and we can go off the record.

23 A. Sure.

24 Q. Is there any reason today that you can't give
25 your best and complete testimony?

1 A. Not that I'm aware of.

2 Q. So other than the window for Zoom, do you have
3 anything else opened on your computer currently?

4 A. Let me take a little look. Maybe the best thing
5 to do is I'll close things as I go along and I'll let
6 you know what's going on. I moved into forced quit.

7 Acrobat is up but, you know, it keeps tabs on
8 everything you've read. I think I'll force quit that
9 for you.

10 Q. Okay.

11 A. We will certainly, I'm sure, be opening it up
12 very soon. But I don't think the old documents come up.

13 I'm leaving mail opened. I see I've got Excel
14 open and I'll quit that. I've got Microsoft Word open;
15 I'll quit that.

16 I have Notes opened, quit. I have Preview
17 opened; I'll do a forced quit. Safari, should I just
18 leave that on just in case we wander into that domain.

19 Q. I don't think so.

20 A. I don't think I'll need a browser. But you
21 never know. It's up to you.

22 Q. I don't think we'll need that. We just need you
23 to download the exhibits sent over. Nate, can you
24 forward over to Dr. Furneaux, I think it just got sent
25 to you.

1 MR. LUMAN: I did just forward it.

2 BY MS. SELLERS:

3 Q. I think we can close out your e-mail as well
4 after you open that.

5 A. Here's the link for today's exhibits, that's
6 what I've received. Should I just click on that?

7 Q. Correct.

8 A. This link has been deemed malicious and cannot
9 be accessed.

10 MR. LUMAN: I received the same note. I just
11 forwarded it to my help desk to see if they will let me
12 access it but I actually received same note.

13 THE REPORTER: Can we go off the record while we
14 deal with this or do you want this on the record?

15 MS. SELLERS: We can go off the record.

16

17 (Whereupon a discussion was held off
18 the record.)

19

20 BY MS. SELLERS:

21 Q. So the only thing you have opened on your
22 computer is your e-mail; is that correct?

23 A. Hold on. I'm just noticing Safari is still
24 opened. Let me just quit that.

25 All I've got left now is mail, Zoom, and Finder,

1 of course. Let me just clean out my screen a wee bit.

2 Q. On your desk, you said you put your phone on the
3 side --

4 A. I put my phone to my side. Let me just show
5 you, here we go, it's upside down, my glasses are here
6 just in case.

7 And I have a note pad and I put that over on my
8 far office over there. And the other binder I alluded
9 to is sitting below me here, you can't quite see it.

10 I'll probably shuffle that backwards and
11 forwards.

12 Q. All right, perfect. So you have one binder that
13 has your declaration, and then other with the exhibits
14 that Nate said you had?

15 A. Yeah, I would prefer if you lead the
16 conversation by sharing whatever is relevant on your
17 screen.

18 I may want to go the paper versions just to get
19 those extra contexts I need. We can work off the screen
20 predominantly.

21 THE REPORTER: I'm making you co-host right now.

22 MS. SELLERS: Thank you.

23 Q. Dr. Furneaux, how did you prepare for your
24 deposition today?

25 A. Let's see, yeah, it is interesting. We did a

1 very preliminary prep session, I think, it must be two
2 or three weeks ago. Unfortunately, then I came down
3 with COVID, and one of the bizarre things about COVID is
4 I have almost no recollection of that meeting
5 whatsoever, unfortunately.

6 So I guess this week starting, must have been
7 Monday or Tuesday, we had a short session.

8 And yesterday or the day before, we had two or
9 three hours I would say going over -- primarily
10 reminding myself what I said in the declaration.

11 Q. So you had two sessions, one on Wednesday and
12 one yesterday, correct?

13 A. Those were the main sessions, yeah.

14 Q. And you had other sessions outside of those?

15 A. Yes, I think we had a short session on
16 Tuesday -- no, must have been Monday.

17 Q. And you said "we." Is we -- is we just Mr.
18 Lumen or was there someone else present as well?

19 A. It was Nate. And occasionally Steve would chime
20 in only very sporadically as I recall. No offense,
21 Steve.

22 Q. So no one else there other than Mr. Lumen and
23 Mr. Moore and yourself?

24 A. And myself, yes.

25 Q. And what materials did you review to prepare for

1 your deposition today?

2 A. Primarily it was the declaration, and obviously
3 on occasion I reacquainted myself with the prior art
4 papers primarily. That's the publications that were
5 cited in support of Dr. Q's declaration.

6 Q. When you say Dr. Q, just for the record you mean
7 Dr. Quackenbush, correct?

8 A. I apologize for shortening his name. But in the
9 interest of brevity, I have been referring to him as Dr.
10 Q.

11 Q. That is fine, I wanted to make sure that was
12 clear. Did you discuss your deposition today with
13 anyone other than Mr. Lumen and Mr. Moore?

14 A. No.

15 Q. So let's talk a little bit about your
16 declaration itself. Did you write the entire
17 declaration yourself?

18 A. Predominantly. As you well know, these are
19 collaborative efforts. The main thing the legal team
20 does from my viewpoint is translate the legal questions
21 into scientific questions or at least that I can
22 appreciate are scientific cases and that allows me to
23 opine on the scientific issue.

24 And then, of course, the opposite process, where
25 my opinion is then translated back into legalese. And

1 then again, of course, I check to see that the
2 scientific content has not been changed in anyway,
3 unintentionally changed.

4 And so that process goes backwards and forwards.
5 I view it as a collaborative effort.

6 Q. So you cite some additional documents that Dr.
7 Quackenbush didn't cite in his deposition such as
8 articles from the Foresight founders.

9 Did you provide those? Or was that something
10 that counsel provided to you?

11 A. I -- well, I can't recall exactly.

12 Some, I suggested. I'm not sure if they made
13 the final evaluation. Some the legal team suggested.
14 It's a mixture.

15 Q. Did anyone other than you and the legal team,
16 when we refer to the legal team are you referring to Mr.
17 Luman?

18 A. Primarily. I don't know who else is involved
19 behind the scenes. I'm sure Nate has some folks who
20 work with him. But I have no idea who that is.

21 My primary interaction is with Nate.

22 Q. And anyone other than you and Mr. Luman revise
23 or edit your declaration?

24 A. No.

25 Q. How much time did you spend preparing your

1 declaration for these IPRs?

2 A. Preparing this declaration, yeah. It's somewhat
3 difficult -- you may well be aware of this, there's a
4 barrage of cases in this domain.

5 I would guess its probably my typical thing, I
6 would say 50 to 70 hours in terms of preparing for the
7 declaration.

8 But again it's a wee bit complicated because
9 within what time, we were also doing some work on the
10 first two cases. But I think that's a good estimate.

11 Q. So you agree it's important not to rush your
12 judgment or your opinion when drafting these
13 declarations?

14 A. Yeah, in fact it is my habit to do a first go
15 around, what I call a shallow evaluation. And then come
16 back a week or so later, and do a more in-depth analysis
17 and then again leave it and come back.

18 That's primarily an academic habit one has in
19 writing papers.

20 Q. So you agree it's important to consider all the
21 evidence before forming your opinion, correct?

22 A. Oh, absolutely.

23 MR. LUMAN: Objection, foundation.

24 THE WITNESS: Can you give me the question
25 again, sorry, I got lost there.

1 BY MS. SELLERS:

2 Q. Do you agree it's important to consider all the
3 evidence cited by Foresight before forming your opinions
4 in this declaration?

5 A. It's always appropriate to review all the
6 evidence one is provided with. It's difficult to review
7 if there's evidence that has not been provided.

8 Q. And so did you review all of the documents cited
9 in Dr. Quackenbush's declaration?

10 A. Well, I think one has to be quantitative about
11 that. Some of the evidence that was cited, and, again,
12 I think primarily about the papers that were, I think
13 what's called the prior art, that I did review in I
14 would say significant depth.

15 Many of the other assertions, the evidence
16 behind the assertions that Dr. Q made, perhaps I did a
17 more shallow read.

18 So in general, there would be varying extents to
19 which I would review the evidence.

20 Q. So when you say a shallow read, describe what
21 you mean by that.

22 A. A shallow read in my mind is a quick review of
23 the evidence, and a quick decision as to whether this is
24 critical evidence that should be reviewed in much
25 greater depth.

1 Or in fact it's a somewhat content piece of
2 evidence that's merely relaying one piece of information
3 and doesn't require an in-depth analysis of a paper in
4 terms of how the data was made, how the experiments were
5 carried out, what were the controls and so forth.

6 It's that kind of more detailed level that I did
7 bring to the prior art papers and particular discussion.

8 Q. Do you agree it's important to follow correct
9 legal principles when forming your opinion?

10 A. Well, I don't think I can agree or disagree with
11 that because I'm not a legal expert. In fact, I would
12 probably say I'm pretty well oblivious of any legal
13 implications.

14 As I mentioned before, the job of Nate's legal
15 team is to present the issues to me in such a format
16 where I can look at primarily the science.

17 And when I have then given opinion an on the
18 science, I understand it has legal repercussions. But
19 I'm not a legal expert.

20 Q. When you're forming your opinions, do you
21 consider the legal principles?

22 A. No.

23 Q. All right, and --

24 A. To the extent that one can, of course. I'm
25 aware that people win or lose cases obviously.

1 Q. Do you agree that if you fail to consider all
2 the relevant elements, that you might have reached the
3 wrong conclusion in your declaration?

4 MR. LUMAN: Objection, form, lack of foundation.

5 THE WITNESS: Well, I think you're asking me for
6 a general opinion about the nature of forming an
7 opinion.

8 And there are circumstances where indeed if one
9 has not considered all the available evidence to the
10 best of one's ability, of course. And again, I would
11 point out that one of the salient features of this
12 particular discussion has been the unfortunate lack of
13 evidence.

14 And that makes it very difficult to agree with
15 an assertion when there is no evidence supporting an
16 assertion.

17 BY MS. SELLERS:

18 Q. Okay. And then how would you characterize the
19 level of effort that was required to prepare your
20 declaration?

21 A. Well, I think it's always useful to relate this
22 to academic research life. In some ways, I would view
23 the exercise as reviewing the prior art papers much in
24 the same way that I would prepare something that's
25 called an academia a journal club.

1 This is where you look for a paper that you
2 think may be of interest to a group. Then you sit down,
3 then you read the paper and you look at the data and
4 then in a one-hour session, you present that data.

5 And that's typically varies obviously depending
6 on the paper but sometimes takes four or five hours of
7 preparation.

8 And then of course the preparation takes an
9 hour. And in my mind that's the closest I can think of
10 in terms of the endeavors in this particular arena.

11 Q. All right. And sitting here today, are there
12 any corrections or changes that you would like to make
13 to your declaration?

14 A. Not that I'm aware, although, you know, I
15 wouldn't be surprised as we go along, we might see some
16 typos, that would not be shocking to me.

17 Q. So nothing short of maybe an occasional typo --

18 A. That's what I would anticipate.

19 Q. All right. Dr. Furneaux, so is TP53 a tumor
20 driver gene?

21 A. So I guess in the old school we call that P53.

22 And I think I would ask you what your definition
23 of driver is before I can give an opinion.

24 Q. What would you consider to be a tumor driver
25 gene?

1 A. People have describes driver genes primarily in
2 the context of contrasting them with another -- well,
3 let's go back to the beginning.

4 And let's stress, immediately, I'm not quite
5 recalling your question. But was your question about
6 the gene?

7 Q. Yes, is TP53 a tumor driver gene?

8 A. Well, you'll forgive me for being pedantic. I
9 used to teach in this area.

10 And, of course, the P53 gene is neither a driver
11 or a passenger. I think what you're referring to is
12 should there be a mutation in P53 that would alter its
13 function, then that altered function, one may test to
14 see whether that, in fact, causes cells to become
15 transformed.

16 And if that mutated variety results, and if you
17 introduce it into a cell that is not transformed, this
18 is an experiment now I'm relating --

19 If one introduced that into a cell that is not
20 transformed. And the introduction of that mutant
21 variety of P53 gene -- and let me just point out
22 immediately there are many, many different varieties of
23 mutations in P53.

24 But for the sake of brevity, let's assume this
25 is a mutation that in fact does alter its function. And

1 to make the argument in its simplest form, one in fact
2 that abrogates its function, it's normal function.

3 In fact, let me add a little bit to that: One
4 that normally abrogates its function but in fact makes
5 it do something different.

6 Then if this were a so-called driver mutation,
7 one would expect that the result of that experiment
8 would be that those untransformed cells would then turn
9 into cancer cells, which you could test by a number of
10 methods, perhaps their unlimited growth, their ability
11 to do unusual things that normal cells don't do.

12 Should all of that pan out, I would be probably
13 convinced, having seen the data, that in fact this
14 mutation in P53 was driving carcinogenesis.

15 Q. So in the field, are there known mutations in
16 the P53 gene that do drive cancer pathogenesis?

17 A. There certainly are. There's certainly lots of
18 examples of mutations in P53, some of which have a
19 limited effect, and some of which have a dramatic
20 effect.

21 Q. All right. And then are there known mutations
22 of the P53 gene that are associated with inherited forms
23 of cancer?

24 A. Absolutely. The classic is call Li-Fraumeni
25 syndrome in which the unfortunate individual inherits

1 the mutation in P53, which renders them unfortunately
2 much more susceptible to the onset of cancer.

3 Q. All right. And then are there known mutations
4 in the BRCA2 gene that are known to drive cancer or
5 drive the pathogenesis of cancer?

6 A. The BRCA2 gene was first identified in terms of
7 being identified as a gene whose mutation is inherited.

8 So it's a slightly different story from P53 in
9 that it first became known as the inherited mutation
10 that increases susceptibility to cancer, which it does
11 incredibly to an incredible extent.

12 I think it increases the chances of breast
13 ovarian cancer 40 fold, which is a huge increase. As is
14 typical in science and medicine, people thought, oh,
15 well, it's an inherited mutation.

16 But in actual fact, if one looks, one can also
17 find somatic mutations in BRCA2, which also then, of
18 course, contribute to carcinogenesis.

19 And, of course, the key difference here is
20 between inherited and somatic, a somatic mutation is
21 occurring post-zygotic phase.

22 So somatic mutations, it's important to point
23 out at this juncture are those that are occurring
24 post-zygotic.

25 Unfortunately these individuals typically

1 through some environmental carcinogen has altered the
2 base in that particular gene with rather unfortunate
3 results.

4 Q. All right. Just to confirm that I understand,
5 the BRCA2 gene -- mutations in the BRCA2 gene are
6 associated with inherited forms of cancer, correct?

7 A. There are inherited forms of cancer that in fact
8 are related and indeed perhaps even caused by a mutation
9 in the gene that has been inherited either from mom or
10 dad.

11 There are also somatic mutations in BRCA2. Just
12 to clear up. Because there was an idea early on that
13 the only mutations one finds in BRCA are inherited.

14 That turns out to not to be the case. It's one
15 of those unfortunate assumptions that folks sometimes
16 make.

17 Q. And these somatic mutations in the BRCA2 genes,
18 would you refer to these as cancer or tumor driver
19 genes?

20 A. Well, I think as I tried to point out with P53,
21 you can have a mutation in a gene and it depends on the
22 location of that mutation.

23 There may be some mutations, alterations, that
24 do not significantly increase the potential for
25 carcinogenesis, or, in the experiment I outlined, in

1 other words, one would take that gene under the
2 requisite experimental conditions and find it only had a
3 very, very low effect, if any, upon creating cancer or
4 transformed cells.

5 And there well be other mutations in BRCA2, for
6 example, that have in fact a severe effect.

7 It all depends on the context. One can't make a
8 general statement about mutations in a gene without
9 doing the experiment.

10 Q. Are there known mutations in the -- known
11 somatic mutations in the BRCA2 genes, from the BRCA2
12 gene, sorry, that drive tumor genesis --

13 A. I would have to look it -- I would have to
14 acquaint myself with the literature. Sitting here, I
15 don't -- I'm not recalling a very good experiment in
16 which the transformation ability of mutant BRCA2 -- and
17 remember there's a BRCA1, so there's a little bit of
18 ponderance in my mind here -- I'm not thinking of a
19 fantastic experiment that shows that that was extremely
20 tumorigenic.

21 There is a problem in general in showing that a
22 gene causes cancer that stretches back to the early days
23 when one needed not one change, but two changes in
24 genes.

25 And I think you'll appreciate it also depends

1 upon the model system that you're using. Typically back
2 in the old days, people were using cells that were
3 basically just one step away from being cancer cells.

4 So it didn't take that much to push them over
5 the edge. It was then appreciated that perhaps that's
6 not perhaps the most rigorous test. Perhaps you want to
7 use cells that are quite far away from being cancer
8 cells, and therefore if a mutation on the gene causes
9 them to become cancer cells, that's somewhat more
10 impressive, and one would perhaps think is more
11 important.

12 There's a lot of variability in those studies.
13 In terms of your specific question, I'd have it to look
14 at the specific mutation and the specific paper and the
15 experiments that were done.

16 Q. And then are you familiar with the concept of
17 the priority date for patents?

18 A. Yeah, I mean, I'm familiar with -- well, my
19 understanding is this, if the publication occurs before
20 that priority date, it's in the game, sports analogy.

21 I also understand there can be a lot of
22 conversations about what that priority date might be.
23 I've certainly seen that that's an area of controversy.

24 Q. You're aware that for these two patents we're
25 talking about today, we're all assuming that the

1 priority date is in 2016, correct?

2 A. That's not a -- I'm not holding that in my mind.

3 But what I'm quietly confident is that the prior
4 art that I have reviewed, is, if I recall, is not going
5 to disappear.

6 In other words, there was no wasted effort.

7 Q. Are you comfortable if we acknowledge that today
8 when we're talking about what was known in the field, we
9 use prior to 2016 or prior as the timeframe?

10 A. Yeah, I'm pretty comfortable with that.

11 Q. Okay, perfect.

12 So around -- in 2016 or prior, what was the
13 longest read length that was available with commercially
14 available sequencers?

15 A. When you say around 2016, what does that mean?

16 Q. Let me rephrase that. That was a bad phrasing.

17 In 2016, what was the longest possible read
18 length available using commercially available
19 sequencers?

20 A. Well, I think -- let me just point out
21 immediately that my function here is to view evidence,
22 not to be a compendium of the top ten hits in DNA
23 sequencing machines, if you allow that expression.

24 And so what I would be interested in is not what
25 the machine says on the outside. But actually how it

1 performed.

2 So were you to be interested in this area, I
3 would be happy to look at publications which actually
4 measured the read length in a sequencing experiment.
5 And I would be able to look at that and give you an
6 opinion.

7 Q. All right. Do you have perhaps a ballpark
8 answer today, are you looking at thousands of bases,
9 hundreds of bases?

10 A. I think, you know, in these kinds of
11 conversations, I tend not to give a general viewpoint.

12 It's been my experience that when one attempts
13 to give a general viewpoint in good faith, and I
14 understand your function in these proceedings, that
15 often that general viewpoint is then attached to a
16 particular context, which then of course turns out to be
17 somewhat misleading.

18 So I'm happy to discuss particular context of
19 which I can look at the experimental evidence and what
20 the investigators did. And I can happily give you an
21 opinion on that.

22 Q. So you didn't review any papers on the available
23 read lengths of sequencers in 2016 before issuing your
24 opinion in this case?

25 A. What I did do when I issued my opinion on

1 various assertions made by Dr. Q is I looked at the
2 relevant paper that was the evidence for such
3 assertions.

4 And indeed of course the read length that the
5 investigators had hoped to use or did use, these are two
6 quite different things, was relevant to my inquiry.

7 Q. Just to circle back and get an answer, then you
8 did not review any papers or any evidence of what the
9 longest possible read length was in commercially
10 available sequencers in 2016, correct?

11 MR. LUMAN: Asked and answered.

12 THE WITNESS: I think I've already gave somewhat
13 of a lengthy answer to your first inquiry. Let me be
14 more specific.

15 I do not see how a general review of the top ten
16 hits of DNA sequencing machines would help me in my
17 evaluation of a specific context.

18 And what I'm interested in is the context that
19 provides evidence for the assertions that Dr. Q has
20 made. And I should perhaps remind you that my function
21 in this was primarily to review the evidence that
22 supports or, in fact as it turns out, does not support
23 the assertions made by Dr. Q.

24 This has been my primary focus.

25 ///

1 BY MS. SELLERS:

2 Q. So can you tell me a little bit about what you
3 would consider a homogenous sample?

4 A. I'd definitely need a little bit more context
5 for that.

6 Q. All right. So if you -- what is a -- if we took
7 a tumor biopsy and we said it was a homogenous sample,
8 what would that mean to you?

9 A. I think what would mean to me is if you
10 described it as thus, you were not being entirely
11 accurate in your subscription.

12 Q. Can you tell me why you would think that?

13 A. Because you're not telling me what the
14 homogeneity is related to.

15 Q. So when you define homogenous, it needs to be
16 related to a specific type of cell?

17 A. Well, you're jumping ahead. Homogenous could be
18 applied as a concept to many entities. One could talk
19 about homogenous with respect to cells, one could talk
20 about homogenous with respect to sand composition.

21 Homogenous is a general term. One needs to know
22 what one is talking about.

23 Q. So if we take a biopsy of a breast cancer tumor.
24 And we say that it is a homogenous tumor sample,
25 does that imply to you that the sample is entirely made

1 up of tumor cells?

2 A. You're creating a somewhat unusual scenario
3 here. So "we" would be, I presume, a surgeon.

4 Q. Correct.

5 A. And Mrs. X has a tumor they suspect, at least
6 Mrs. X has an unusual mass, I'd say. And the surgeon
7 goes in, takes a sample, and then the question would be
8 what is the composition of that sample.

9 And I'm assuming here that you're primarily
10 interested in the composition as that relates to cells,
11 not a composition that relates to molecules or hormones
12 or small molecules or whatever.

13 But with respect to cells; am I correct?

14 Q. Correct.

15 A. So before making any pronouncement, before the
16 surgeon can make any pronouncements, one needs to look
17 at exactly what it is. How might one do that.

18 I know that I shouldn't ask you questions. But
19 I hope you'll understand, I have to sort of understand
20 what your question is.

21 So when you pronounce a sample as being
22 homogenous, one can't do that until one does the
23 experiment or the analysis.

24 Q. And what analysis would one perform to determine
25 whether a sample is homogenous?

1 A. Well, there are literally hundreds of ways.

2 Back in the old days, of course, one would take
3 that sample, depending on its size and composition.

4 But even to the eye, the old-time surgeons would
5 take a sample and look at it and go, oh, may well be,
6 for example, that this is not a tumor, they go, oh, my
7 god, this is a cyst and there's a whole pile of fat
8 around it.

9 Oh, gee, one would describe that as not being
10 homogenous, what I just described to you. On the other
11 hand, they might say in their experience and only by
12 their experience, oh, this looks like tumor issue.

13 They would be completely unable to pronounce as
14 to whether what was homogenous, heterogenous, or any
15 kind of genous.

16 What they would have to do, of course, is use
17 the first line technique, which would be to cut a slice
18 through that material, stain it, and look under a
19 microscope.

20 That would then be passed along to the next
21 expert, the pathologist, who might be able to say here
22 are tumor cells in my opinion, here are normal cells in
23 my opinion.

24 That, of course, is why pathologists do what
25 they do. But that evidence could then be further

1 validated by looking at particular markers, more
2 advanced techniques, one may even go and look at DNA
3 sequence.

4 There's a whole pile of things one could do
5 before one could make a pronouncement that the material
6 is of whatever homogenous, heterogenous state.

7 Q. Let's talk about heterogenous samples. You
8 would refer to a tissue sample that has both tumor and
9 regular cells as a heterogenous sample, correct?

10 A. Again, what we have to be careful about is what
11 facet of heterogenous or what the entity that we're
12 describing is heterogenous or homogenous might be.

13 If we're simply looking at whether the
14 "generosity," if you like, interesting new word, pertains
15 to cells in general, then one might describe a tissue
16 that has normal cells and tumor cells as being
17 heterogenous because the heterogeneity that we're
18 referring to simply relates to the state of cells.

19 Now, within that, there's a sub-heterogeneity
20 one may be interested in looking at.

21 In fact are all the tumor cells the same? We're
22 leaving behind now the normal cells, we're just looking
23 at tumor cells and saying, oh, is there a tumor cell
24 heterogeneity.

25 So to clarify completely, the first example I

1 talked about was a cell heterogeneity or not. The next
2 phase is do we have a tumor cell heterogeneity.

3 Now in the real world, this requires a skilled
4 pathologist now, a cancer pathologist if you'd like, who
5 perhaps in his experience or his or her experience can
6 say oh, yes, this is a tumor cell type A, we can see
7 that very clearly just from looking at the shape of the
8 nuclei, looking at the intensity of things in the
9 cytoplasm and so forth.

10 And this cell over here, oh, my goodness, that's
11 a tumor cell C, that's quite a different beast. Looks
12 to be like maybe, I don't know, you know, 10 percent of
13 one, 90 percent of the other, but, hey, that's a
14 mixture. So that would be heterogenous.

15 Now, of course, the task keeps on going because
16 it may well be that that thing, that tumor cell C, that
17 the pathologist saw and maybe we're way back in, you
18 know, the 1980s or something.

19 Know we've realized, oh, there's two different
20 forms of type C, there's a type CA and a type CB.

21 So there we have heterogeneity even within the
22 tumor cell type C cells. And so it would go on.

23 Q. So for the purposes of today's deposition and
24 we're referring to a tumor and we refer to the tumor
25 having cell heterogeneity, can we accept that that is

1 there's different types of cells, cancer and non-cancer,
2 within the tumor?

3 MR. LUMAN: Objection. Incomplete hypothetical.
4 Lacks foundation.

5 THE WITNESS: I would direct you again -- I'm
6 trying to be very careful about indicating the context
7 of our discussion.

8 So what I'd hoped to do there was illustrate the
9 difference between -- if one talks about cell
10 heterogeneity, one would be talking about the difference
11 in population between normal cells and what one thinks,
12 hopes, passes, determines, opines on, would be cancer
13 cells.

14 In contrast, we might talk about cancer cell
15 heterogeneity or tumor cell heterogeneity, where now
16 we're no longer talking about normal cells -- although
17 in the real world, who knows, something we thought was a
18 normal cell, looks like a normal cell, walks like a
19 normal cell, actually turns out to be a tumor cell when
20 we look in a more detailed fashion.

21 So what I'm trying to say here is that the
22 context is critical. One can't make general aphorisms
23 about the state of heterogeneity.

24 BY MS. SELLERS:

25 Q. So if we have a sample that has cell

1 heterogeneity, that has tumor cells and non-tumor cells
2 and you want to determine a specific mutation within the
3 tumor cells, is it beneficial to have higher read depths
4 than if it was homogenous sample with just the tumor
5 cell?

6 A. Okay, let me -- read that question back to me
7 again.

8 Q. If we have a sample that has cell
9 heterogeneity --

10 A. How do we know that?

11 Q. It's been determined prior to this. So they've
12 done some tests and we've determined it has cancer cells
13 and normal cells?

14 A. So we already know and so what you are seeking
15 to do is confirm.

16 Q. What we're seeking to do is to confirm the
17 prevalence or presence of different somatic mutations in
18 the cancer cells.

19 A. So we're on a confirmatory exercise?

20 Q. Correct.

21 A. Do we know if they were normal cells or tumor
22 cells in the first place?

23 Q. Sorry, let's stick to what we know here, which
24 is just that we know currently that the sample has both
25 tumor and normal cell line within it?

1 A. I sort of hoped to point out to you in the
2 discussion that sometimes there can be surprises. There
3 could be cells that look like normal cells and they turn
4 out to be tumor cells.

5 So you're setting up a scenario which is sort of
6 like a Catch 22. We already know there's a mixture and
7 now you're asking me how do we confirm there's a mixture
8 but we already know there's a mixture apparently.

9 Q. That's okay, we're not going to fight about the
10 hypothetical.

11 Let's keep going down this line, which is we
12 know there's both, we're in a confirmatory exercise,
13 that's fine.

14 But if you did want to confirm or identify
15 specific somatic mutations within that tumor cell, would
16 you want a higher read depth than in the sample were
17 homogenous?

18 A. Well, so let's term rather a different way. If
19 one wants to look at the status of DNA sequence, any
20 sample, it's an enormous advantage when we put it that
21 way, if the sample is essentially pure, essentially
22 clonal, that would be a remarkable advantage to your
23 endeavors.

24 If it is not, then I would put it to you that
25 the job gets more difficult. And the solution to that

1 job -- there are many factors that are involved in
2 solving that problem.

3 Q. And now what are some of the solutions that can
4 solve that problem?

5 MR. LUMAN: Incomplete hypothetical.

6 BY MS. SELLERS:

7 Q. Sorry, what are some of the solutions that can
8 increase the accuracy of sequencing when you have a
9 heterogenous sample?

10 MR. LUMAN: Incomplete hypothetical.

11 THE WITNESS: Perhaps if you could ask the
12 question again.

13 BY MS. SELLERS:

14 Q. So you opine that there are many factors that
15 could solve the problem of not having a pure sample when
16 doing DNA sequencing.

17 What are some of those factors that help solve
18 the problem?

19 A. I think you meant techniques when you said
20 problem.

21 Q. Yes, sorry.

22 A. Well, you know, the first go round a very simple
23 solution is to obtain a purer population. And when I
24 say purer, of course, I mean the best of one's ability,
25 99 percent, 95 percent.

1 That would be the first and simplest solution to
2 that problem.

3 Q. And what would be some other solutions to that
4 problem?

5 A. Again, we're interested in obtaining what --
6 what's our objective here.

7 Q. Sequencing that tumor.

8 A. Just sequencing?

9 Q. Um-hmm.

10 A. If our objective is just to sequence, we would
11 go ahead and sequence.

12 Q. But if we were trying to determine a specific --
13 if there's a specific point mutation, there's a single
14 --

15 (Reporter clarification.)

16 -- single nucleotide variant?

17 A. Okay, so our objective -- I'm still a little
18 confused about our objective.

19 Our objective is to find a variant for the first
20 time or merely to confirm since it seems to me you have
21 some notion that it's there already.

22 Q. Find it for the first time.

23 A. Find to the first time, okay. What would be the
24 factors that we would bear in mind? And in your
25 scenario here, you have authority about what the

1 prevalence of what this variant might be.

2 Q. But that is unknown, correct? If we've not
3 sequenced it before, we would not know the prevalence?

4 A. We would not know the prevalence, okay, all
5 right. And your method of determining such a mutation
6 is DNA sequencing?

7 Q. Correct.

8 A. Because there are other ways of identifying a
9 mutation, correct.

10 Q. Correct.

11 A. I'm going into professor mode. I apologize.

12 For example, one could determine that there is
13 an alteration in a tumor protein by looking at protein
14 structure. One doesn't necessary have to go to DNA
15 sequencing.

16 Q. But if we are doing DNA sequencing here, what
17 factors would you consider?

18 A. We've decided we're going to go down the DNA
19 sequencing route?

20 Q. Yes.

21 A. And we are completely oblivious as to whether
22 such a mutation exists?

23 Q. Correct.

24 A. And we have no inkling of what gene might harbor
25 such mutation.

1 Q. We can say -- let's say we're looking at the P53
2 gene just for -- to make this hypothetical easier.

3 A. Well, yeah, because I'm going to need a little
4 bit more context. What year are we in now?

5 Q. We're in 2016.

6 A. Oh, we're in 2016 and we've decided we're going
7 to look and see in a tumor sample whether there's a
8 mutation in P53.

9 Q. Correct, for a heterogeneity sample.

10 A. Pure thought experiment, right? And, of course,
11 if we decided that, I think it would be fair to say that
12 we might have an idea where we should find such a thing
13 within P53 in 2016.

14 We might be aware of where to find such a thing
15 in 2016.

16 Q. Let's get back to the original question, which
17 was which factors would you consider when sequencing
18 because the tumor is heterogeneous?

19 MR. LUMAN: Objection, incomplete hypothetical.

20 THE WITNESS: And I'm happy to go along with
21 this interesting, fascinating thought exercise, but if
22 we are going to think about it in anyway whatsoever it
23 does behoove us to think about timeframe in which we are
24 discussing this.

25 If this was 1983, we might be having an entirely

1 conversation about P53.

2 BY MS. SELLERS:

3 Q. Well, let's assume for today we're talking about
4 2016, as we discussed earlier. So in 2016, if we are
5 going to do sequence a heterogeneous tumor sample to
6 determine whether there is a transformation in the P53
7 gene?

8 A. Correct, and so --

9 Q. What -- sorry, Dr. Furneaux, not finished with
10 that question: What factors would you consider?

11 A. So we're in 2016, okay, we've elected to look at
12 a transformation in P53 -- you know, an interesting way
13 of presenting this sort of discussion is let's imagine a
14 student in my lab came to me and said, hey, I just got a
15 tumor sample from Joe, the surgeon.

16 I want to go look now into 2016 and see whether
17 it has a mutation in P53. I think my response would be
18 in 2016, why would you want to do that, that's already
19 been done.

20 You pretty much have an extremely high chance of
21 finding such a mutation since they're very prevalent.

22 While we're on that conversation, where exactly
23 are you going to look in P53 because if you're
24 suggesting we should do this by sequencing, although one
25 could do it simply by immunochemistry.

1 But if we're going to do it by sequencing, it
2 seems prudent to think about where you should look would
3 be the first thing and, as it turns out, P53 in 2016 you
4 know pretty well where to look if you're hoping to find
5 a prevalent mutation, perhaps one might decide to say,
6 oh, let's look at the region between exons four and
7 five, because that's pretty much a hot spot, so we're
8 going to have a high chance of finding something there.

9 Q. All right. So let's discuss this maybe a little
10 more generality.

11 If you have a heterogeneous sample like that has
12 both cancer and non-cancer cells and we know this, does
13 higher sequencing depth reduce errors when sequencing
14 such samples?

15 MR. LUMAN: Incomplete hypothetical.

16 THE WITNESS: I think -- there's a prevalent
17 problem here that I would like to alert you to is that
18 we continue to talk in generalities rather than
19 specifics.

20 My role in this discussion is to comment about
21 specifics, and to comment upon particular data that I've
22 reviewed in the prior art.

23 I'm well aware that when one starts talking
24 about generalities, and I'm cautioning now, that I may
25 well say something that actually could be interpreted in

1 an entirely different way in a different context.

2 And so I'm really reluctant to discuss such
3 generalities. And already you've construed a rather
4 strange generality where you're now inferring that there
5 would be something different about performing DNA
6 sequencing on a tumor sample versus a normal sample.

7 BY MS. SELLERS:

8 Q. Would you say that -- are there any different on
9 how you would perform the sequencing on a homogenous
10 tumor sample versus a heterogenous tumor sample with
11 cancer and noncancer cells?

12 A. Again, we persist talking about generalities.
13 In any conversation about any experimental strategy, one
14 has to be fully aware of what the objective is. And
15 whether one may or may not succeed in that objective.
16 And whether one may alter or change our strategy to be
17 successful in that objective.

18 Until one is talking about a specific scenario,
19 I could not comment on what the requisite thing to do
20 might be.

21 I've tried endlessly to obtain from you
22 information about the particular scenario you're
23 interested in. But there are still missing factors in
24 your description.

25 Q. So let's switch topics a little. Would you

1 consider an assay that costs \$400 per sample to be
2 prohibitively costly?

3 MR. LUMAN: Incomplete hypothetical.

4 BY MS. SELLERS:

5 Q. Would you consider an assay that's designed to
6 determine specific mutations in a suspected tumor to be
7 prohibitively costly?

8 A. I think the best people to ask for that would be
9 the folks who do insurance, the attending physician, and
10 the patient.

11 Q. Well you work in a lab. In the laboratory
12 setting, would \$400 an assay for an experiment to
13 determine whether a specific cell has a given
14 transformation, would that be prohibitively costly?

15 A. Well, just to clarify, I run a lab. I run an
16 independent laboratory that, in fact, transcends many,
17 many fields and is capable of doing things outside of
18 government funding.

19 So my perspective is likely to be a little
20 different, okay, in that respect.

21 As in all things, one has to measure what one
22 gains from a procedure versus what it costs. And the
23 piece that's missing from your description is what would
24 one gain from this.

25 Q. All right, so if the gain was significant, the

1 \$400 cost could be justified?

2 A. I don't know what the gain is. I would have to
3 evaluate whether that gain was indeed significant.

4 Q. Let's say the gain would allow you to determine
5 whether a specific therapy would be beneficial for a
6 patient based on their specific mutation, would that
7 \$400 cost be --

8 A. You know, I'm happy to take your word for what
9 you're saying. And I understand your endeavors.

10 One would need a little bit more detail than
11 that, right. If one said, oh, the gain is that the --
12 let's imagine, in fact, I am the very patient, the gain
13 is that you live for another 31 days, gee, I don't know,
14 tough call.

15 Q. All right, well, would you consider \$400 for a
16 sample of whole genome sequencing to be cost
17 prohibitive?

18 A. Again, it depends on the objective. So, for
19 example, what springs to mind immediately is -- and I
20 don't know what the dollar cost was, but it did seem
21 that the endeavor carried out by Ley, et al, would seem
22 to be at first glance, I don't know the numbers.

23 But back of the envelope, that would seem to be
24 considerably more than \$400. And when one looked at the
25 success of that venture, it would seem to be extremely

1 meager in all respects.

2 Q. So how would you define the term orthogonal
3 technologies?

4 A. Wow, that's a chemistry term. You'd have to
5 explain that to me.

6 Q. All right, well, let's take a look at your
7 deposition, which I believe you have in front of you.

8 Let's find that paragraph. If you can go to
9 paragraph 116, on pages 67 through 68.

10 Can you see that?

11 A. I think you're referring to paragraph 116, I can
12 see it on your screen, yeah, absolutely.

13 Q. So this is declaration 2169, which has just been
14 introduced. And it has been previously marked as
15 Exhibit 2169.

16 (Exhibit 2169 was marked for identification by
17 the court reporter and is attached hereto.)

18 BY MS. SELLERS:

19 Q. Dr. Furneaux, let's take a look at this last
20 sentence here in this paragraph. You'll see it says,
21 "Dr. Quackenbush's colleagues at the Dana Farber Cancer
22 Institute wrote that PCR and capture probe arrays are
23 'orthogonal technologies.'"

24 How would you define what "orthogonal
25 technologies" means here?

1 A. First of all let me point out that orthogonal
2 technologies is in inverted commas --

3 (Reporter clarification.)

4 -- Orthogonal technologies is in -- oh,
5 quotation marks, I think is American English. We call
6 them inverted commas, sorry.

7 Q. So how would you define what "orthogonal
8 technologies" means in this paragraph. It is in your
9 declaration.

10 A. Yes, it is in my declaration. And I know that
11 many of our colleagues in the science world are fond of
12 these buzz phrases.

13 But again this is a generality, okay. I may
14 even be whimsical here. I think we can certainly
15 discuss at length the differences between the
16 preliminaries chain reaction and capture probe arrays --

17 Q. That's not my question here. My question is how
18 do you define "orthogonal technologies"?

19 A. Can we go and see what the dictionary says about
20 orthogonal?

21 Q. Well, I think -- you included this term in your
22 declaration. So do you have an understanding of what it
23 means?

24 A. You'll notice I put it in inverted commas, in
25 quotes.

1 Q. I understand that. So is your testimony that
2 you don't have an understanding of what term "orthogonal
3 technologies"?

4 A. My testimony is that it's a phrase that's
5 bandied about without any real context.

6 Q. So your testimony is that this phrase doesn't
7 have any significance; is that correct?

8 A. What I'm saying here is that, which has been my
9 observation, is that sometimes folks in fields talk
10 about things and use buzz words when they'd be better
11 employed talking about the real differences between
12 things.

13 Q. So what did you intend when you included when
14 you included this phrase in quotes in your declaration?

15 A. What I think I'm intimating here is my
16 skepticism of the understanding of the field, and its
17 inability to relate specific differences and to
18 certainly put them into a basket of orthogonal.

19 It's one of the most abused words I can think of
20 in contemporary scientific terms.

21 Q. So in your view, the term doesn't really carry
22 much meaning?

23 A. Well, in my view, what it actually does convey
24 is there's a little bit of let's say less rigorous
25 thinking when one is thinking about techniques.

1 MR. LUMAN: Abby, we've been going for about an
2 hour, do you want to take a break soon?

3 MS. SELLERS: Yeah, this is now a good time.

4 MR. LUMAN: So off the record at 8:14.

5

6 (Recess taken.)

7

8 MR. LUMAN: Back on the record at 8:30.

9 BY MS. SELLERS:

10 Q. Welcome back, Dr. Furneaux.

11 A. Thank you.

12 Q. So approximately how many bases is the human
13 exome?

14 A. The human exome?

15 Q. Yes.

16 A. By that you would mean the total of exomes.

17 Q. No, the number of bases.

18 A. Yeah, I know but I'm trying to figure out what
19 you mean by exome.

20 Q. Oh, correct, yes, the exome?

21 A. Of protein encoding segments?

22 Q. Just entire exome.

23 A. Well, there are differences. Obviously one
24 could in -- and what timeframe are we at now?

25 Q. 2016.

1 A. 2016. Okay, even in 2016, there certainly was a
2 movement to regard noncoding segments as being exonic.

3 So I think that's a little open to debate.

4 Q. So let's include noncoding segments, coding and
5 noncoding, how many bases were known in the human exome
6 in 2016?

7 A. Well, when you say known, what does that mean?

8 Q. It has varied a little bit throughout the years,
9 correct, the number has fluctuated slightly?

10 A. The number that one attributes to what one would
11 regard as the exome --

12 Q. Right.

13 A. -- has varied?

14 Q. That's my understanding. In 2016, how many
15 numbers would a person of ordinary skill in the art had
16 considered to be in a human exome?

17 A. Again, I'm deigning to give you general numbers
18 about general things. I'm happy to comment on what a
19 particular researcher and co-author of a particular
20 paper thought it was at the time because that's where
21 it's relevant.

22 Q. Well, your job here is to answer questions.

23 So my question is what would a person of
24 ordinary skill in the art have considered the
25 approximate number of bases in the human exome in 2016.

1 Do you have an answer to that?

2 A. The answer I have for you is a person of skill
3 in the art upon reading a paper which is of interest to
4 that person in terms of his endeavors, would review what
5 the authors thought was the number.

6 We are in the -- it's a matter of fact, we're in
7 the sort of post-Google era where one doesn't keep in
8 one's mind content numbers like that, which, as we've
9 already discussed change according to the
10 interpretation, and also most importantly according to
11 the methods that one might be conceiving of.

12 Q. Would you say that 30 million bases is a fair
13 approximation of the size of the human exome?

14 A. Well, I can give you -- let me give you a back
15 of the envelope flavor. And again these are
16 generalities.

17 But if one is thinking about exomes in the
18 traditional format, as in the things that are the bits
19 and pieces of genes that encode protein, I think people
20 tend to believe that such a content is between 1 and 2
21 percent of the genome.

22 So again there may well be lots of discussions
23 about the size of the genome. I'm going to refer to the
24 actual size, which is more than likely 3.2 billion
25 bases.

1 So if one takes 1 percent of 3.2 billion, I
2 guess that takes us to what, 30 million.

3 Q. Right, right around 32 million.

4 A. Yeah, sure. But, as I said, these are all very
5 back-of-the-envelope generalities.

6 Q. Correct, let's assume that the human exome is 30
7 million bases.

8 If a person of ordinary skill in the art wanted
9 to sequence a person's exome, how many bases would need
10 to be read in order to do that?

11 MR. LUMAN: Incomplete hypothetical.

12 THE WITNESS: I'm not sure this was the question
13 you intended. But what I heard was if there are 32
14 million bases, which we have agreed upon just as a back
15 of the envelope, how many would you have to sequence and
16 the answer would be 32 million.

17 BY MS. SELLERS:

18 Q. Okay, if we wanted to -- if a person of ordinary
19 skill in the art wanted to get an accurate understanding
20 of the structure of the exome, how many bases
21 approximately would have to sequence?

22 A. And, again, let me point out we're talking about
23 generalities here. And we haven't even -- are we
24 talking about the human genome?

25 Q. Correct, the human genome.

1 A. We're talking about the human genome.

2 And so, obviously, there's all sorts of
3 specifics, okay. What I would suggest to you -- and I'm
4 neither agreeing nor disagreeing with this notion, but
5 the industry standard, and let me put this in quotation
6 marks, the industry standard that folks in the field
7 have sort of gravitated towards, is if you attempt --
8 and the key word is attempt -- to sequence each base 30
9 times.

10 And let's imagine for a second you might be
11 successful in that endeavor, although lots of
12 considerations have to be given to that, then the
13 industry feels that's probably going to do a good job,
14 would probably be the most generous statement I could
15 make.

16 Q. All right. So the 32 million bases we want to
17 sequence each 30 times, we would have around 9.6
18 gigabases sequenced, correct?

19 A. Yeah. I understand the 39, I think that's the
20 reference I gave you but whatever.

21 Q. And you, Dr. Furneaux, do you believe that that
22 30 base number is accurate -- sorry, the 38 times
23 sequencing each base?

24 A. I do understand your question, it's okay.

25 Let me, again, repeat these are generalities

1 we're talking about. If I'm interested in evaluating
2 this, I have to see the context.

3 And I, as many people in the field are well
4 aware, from the very, very beginning way back in the
5 '80s, not every base is equally sequenced, not all bases
6 are equal, sad but true.

7 So as ever one has to. And again reinforcing
8 what I said before about somewhat little bit of a
9 confusion about exactly what the exome is, one would
10 have to look at the detail.

11 Q. If you were going to sequence a human exome,
12 would you try to sequence each base 30 times or would
13 you choose a higher number?

14 A. If I were going to sequence a human exome, I
15 would have to first consider my objective.

16 Is it critical that I actually know accurately
17 every single base? For many applications, this is not
18 necessarily the case.

19 Q. So let's say it is critical that you do know
20 every single base --

21 A. Then I would pursue the sequencing until I was
22 satisfied that that was the case.

23 Q. Is there an initial amount that you would start
24 with? Would you start to try with doing 30 reads per
25 base, 35, 40?

1 A. I think you're asking very general questions,
2 which really don't have any relevance to the protocol.

3 We are in what timeframe?

4 Q. 2016.

5 A. There's a variety of techniques one could use.
6 And so perhaps the first thought would be what
7 particular technique one might use.

8 Q. So you don't have any inclination as to whether
9 you would sequence each base a number of times?

10 (Speaking Simultaneously.)

11 A. You continue to ask me very general questions
12 without providing details with which I could evaluate
13 the strategy that you are hoping I may indicate.

14 Q. If you were to use an Illumina sequencer to
15 sequence bases, does that change any of your analysis?

16 A. Again, it depends upon my objective. If the
17 objective is purely to obtain an accurate sequence, that
18 might be a good place to start, sure.

19 It would probably depend whether we had such a
20 machine, it would probably depend on if we can access
21 one.

22 Q. All right and then --

23 A. It would be very nice to be in a building where
24 there's like 100 machines stacked up in a room, that
25 would be fantastic. Doesn't happen often.

1 Q. Have you ever sequenced over 20 gigabases on a
2 single sample?

3 A. No. Of DNA?

4 Q. Correct.

5 A. No.

6 Q. What about of RNA?

7 A. Doing that right now.

8 Q. Okay. For what purpose?

9 MR. LUMAN: I would caution the witness not to
10 reveal any third-party confidential information.

11 THE WITNESS: I'm not sure what you know about
12 my background. But I have a small company that does
13 quite interesting research and the interest in the
14 research that I do, of course, is privileged
15 information.

16 BY MS. SELLERS:

17 Q. Okay. And then what's the greatest number of
18 bases of DNA you've ever sequenced for a single sample?

19 A. In my own hands?

20 Q. Yes?

21 A. It would a mixture of hands -- my graduate
22 student at the time, Anika Szabo, I think we're now
23 talking, '84-'85, unknown to us, we were actually
24 pioneers in what's actually become to be known by the
25 cognoscenti tiled sequencing.

1 (Reporter clarification.)

2 -- which is in other words, we sequenced using
3 Sanger sequencing, and having obtained that sequence, we
4 then synthesize a new oligo, which acted as a new
5 primer, we extend it along.

6 I think Anika had the world record in we
7 proceeded about 20 bases a shot.

8 However, the advantage of all that was that
9 having completed the sequence of the HUD gene. And when
10 I say gene, we're talking about from beyond --

11 (Reporter clarification.)

12 -- we're talking a little bit beyond the
13 five-prime end of the messenger RNA all the way down to
14 the 3-prime end, if I recall, which, if I recall, is
15 something like 2,000, 2.5, 1.5 or 2.5 thousand bases.

16 Just as a matter of interest, that sequence has
17 remain unchallenged throughout now, where are we '84, 40
18 years.

19 Q. So I didn't hear you super well in the middle of
20 that. Did you say 45 gigabases was the highest amount
21 of DNA you ever sequenced?

22 A. Oh, no, no, no. We're in 1984, okay, so what I
23 said was, I think it's either 1,500 bases, 1.5K or 2.5K.

24 Q. And is that the length of the gene you're
25 referencing or the total number of bases sequenced using

1 the sequencer?

2 A. That would be -- well, that gets a little bit
3 more complicated to work out.

4 As I said, our strategy was to synthesize and do
5 Sanger, which would do on a good day about a 150, 200
6 bases.

7 And as I said then, using that sequence we now
8 made a new primer and could now get the sequence of the
9 rest of the cDNA that we were using.

10 So some bases would be sequenced more than once,
11 difficult to give you a calculation on that.

12 Q. Do you have degree in economics, Dr. Furneaux?

13 A. I don't think so, no. But I have an empirical
14 experience of money and life.

15 Q. Do you consider yourself to be an economist?

16 A. I consider myself to be -- due to my advance in
17 years, wise in the ways of the world in money.

18 But I couldn't say any more than that.

19 Q. Do you have experience working on clinical
20 trials?

21 A. Yeah.

22 Q. Okay, can you tell me a little bit about that
23 experience?

24 A. Well, I mean, there's a number of different
25 experiences.

1 Gee. The one that springs to mind -- I don't
2 know if this is privileged information or not. Why
3 don't I just talk in general 'cause that's what we've
4 been doing and I'll try to leave out names and places.

5 If I'm recalling correctly there was a little
6 bit of controversy in this trial. Anyway, the bottom
7 line was that these were patients who might well benefit
8 from being treated with the anti-VEGF antibody.

9 I won't give you the trade name.

10 And so my role in this, which I prompted, was to
11 evaluate perhaps whether a given patient, whether we
12 could prospectively determine whether that patient may
13 benefit from such a regimen.

14 I think that's best to leave it there. But let
15 me just try to fill it in a little bit. By obviously
16 measuring let's say suspected biomolecules of relevance.

17 Q. Then do you consider yourself an expert on
18 clinical trials?

19 A. Well, I would actually consider myself as
20 someone who has been in on let's describe it as the
21 evolution of molecular strategies and how they integrate
22 into clinical trials.

23 I was certainly one of the first at Sloan
24 Kettering to directly interact with clinicians and try
25 and bridge that significant gap between a clinician who

1 wants to know what to do with a patient and the basic
2 scientist who's happy to go do things, you know, in six
3 months and not worry about real world implications.

4 I'm being a little harsh there. But I think
5 that was the scenario way back when I first got into
6 this arena.

7 Q. So in your declaration, Dr. Furneaux, you cite
8 an article from The Scientist that discusses Personalis'
9 NeXT Personal assay.

10 Do you recall the article?

11 A. Yeah, I mean, I would have to stress to you
12 again that I probably did a shallow reading of that,
13 such a journal. I have a recollection.

14 MS. SELLERS: Let's pull it up here. All right.
15 So this is previously introduced Exhibit 2001.

16 (Exhibit 2001 was marked for identification by
17 the court reporter and is attached hereto.)

18 BY MS. SELLERS:

19 Q. So let's take a look at it?

20 A. If you don't mind, I'll see if I can pull it
21 out. What is it called?

22 Q. I can share my screen with you.

23 MR. LUMAN: That's not an exhibit that's been
24 provided to Dr. Furneaux.

25 ///

1 BY MS. SELLERS:

2 Q. Are you okay with just view it here on the --

3 A. Sure, that's perfectly fine. I was wondering
4 because -- never mind. What are we looking at?

5 Q. So here is this article --

6 A. Where is in this article coming from, what
7 journal is this?

8 Q. It is from The Scientist and it discusses
9 Personalis's NeXT Personal assay. You'll see here the
10 article mentions the NeXT assay to identify 1,800
11 single-nucleotide variants.

12 Do you see that?

13 A. Yeah.

14 Q. Why do you think that this article mentions
15 Personalis' or the NeXT assay's ability to track up to
16 1,800 variants?

17 MR. LUMAN: Lacks foundation. Calls for
18 speculation.

19 THE WITNESS: You'd have to ask the authors of
20 this article.

21 BY MS. SELLERS:

22 Q. Do you think there's anything significant about
23 these 1,800 variants?

24 THE WITNESS: What I think is it says -- let me
25 just track here, "performs whole genome sequencing to

1 identify up to 1,800 single-nucleotide variants."

2 Sounds like an admirable endeavor to me.

3 BY MS. SELLERS:

4 Q. You think tracking 1,800 -- tracking 1,800
5 single-nucleotide variants in an assay is a unique
6 aspect of the NeXT Personal assay?

7 A. I couldn't comment about on that. This is -- I
8 would have to say The Scientist, this is somewhat one
9 expects -- you see these statements, I would probably
10 like to see the actual data that supports this and the
11 relevance of this and why this number and so forth
12 before I could offer any opinion.

13 Q. All right. Well, if we can pull back up your
14 declaration, which is Exhibit 2169. If we go to
15 paragraph 157.

16 A. 157.

17 Q. So you cite this as evidence of praise in the
18 industry for the NeXT Personalis assay, correct?

19 A. Yes, I think I'm merely pointing out here that
20 it's received attention. What I'm saying is, I mean,
21 the topic here is industry praise.

22 So I think, yes, this is evidence that it has
23 been praised in the industry as in being featured in
24 this particular magazine, I would hasten to add, not a
25 journal.

1 And certainly it appears to be in the top ten
2 innovations. So this is all very good, and this would
3 qualify for industry praise.

4 I don't think the article provides any
5 information about exactly the nature of the assay and
6 why it would be innovative. Someone has placed it in
7 the top ten. I think that's all that says.

8 Q. Would you say it would be more difficult for an
9 assay to track 1,800 variants, rather than say 500
10 variants?

11 MR. LUMAN: Incomplete hypothetical.

12 THE WITNESS: Yeah, again, we're always straying
13 into these generalities. It would very much depend upon
14 what these variants are and many, many, many other
15 factors, which if you provide to me, I'd be happy to sit
16 down for an hour and figure out the answer to that.

17 Not the answer. To give you an opinion.

18 BY MS. SELLERS:

19 Q. So you, Dr. Furneaux, couldn't comment on
20 whether NeXT Personalis's Personal assay is really
21 praiseworthy without more information than what's given
22 here?

23 A. No, you're distorting what I said. What the
24 objective of this section, as I understand it, is to
25 establish whether Personalis has received industry

1 praise.

2 There are two scenarios. Personalis assay may
3 have received no attention whatsoever. And the other
4 possibility is it finds itself in the top ten
5 innovations of 2022 according to The Scientist magazine,
6 which I presume in this discussion we're saying is
7 industry, which I think is a reasonable thing.

8 And so it has received praise --

9 Q. Perfect. Sorry.

10 So what I'm asking you, Dr. Furneaux, is if you
11 yourself would need more information than what's given
12 here in this article in order to determine whether you
13 believe the NeXT Personalis assay is praise worthy?

14 A. Yes, I think it's no secret that one of the
15 challenges in doing this particular endeavor has been
16 lack of information that allows me to make a clear
17 opinion.

18 And I'm referring primarily to Dr. Q's
19 declaration. In this particular arena, I have not been
20 charged with the responsibility or the desire to look
21 carefully at the raw data that Personalis produces.

22 Clearly I understand why. I am also in the
23 field in a general sense and, although, of course, I
24 would obey all confidentiality restriction and so forth,
25 it would not seem wise for me to Personalis to tell me

1 exactly what they're doing and for me to actually review
2 their raw data.

3 I would not expect that and I would not ask for
4 that.

5 Q. Okay, so if there was another assay on the
6 market other than Personalis' NeXT assay, that only had
7 the capability to sequence up to 500 variants, would
8 there be a reason for at a clinician to choose the NeXT
9 Personalis assay over that 500 variant assay?

10 A. Again, we continue to talk about generalities.
11 A clinician, again, this I know very well, is
12 instinctively skeptical of any assay until they see it
13 working.

14 The proof of the pudding is in the eating. And
15 so what one might suspect if one really wanted to know
16 the answer to that question is, one would expect to see
17 a review in which people have performed differing assays
18 and therefore evaluated it in a metric that the
19 clinician is interested in: Does it detect cancer, does
20 it predict how one may treat that cancer, does it
21 actually work?

22 These would be the kinds of reviews that I think
23 a clinician would look at in determining the choice of a
24 particular strategy.

25 I have not reviewed that area. That's not been

1 the scope of my work for this particular presentation.

2 Q. So for your declaration, you didn't review, for
3 example, whether the Personalis NeXT assay works for its
4 intended purpose?

5 A. Again, let me emphasize, I have actually not
6 considered those aspects and looked at the Personalis
7 assay in detail.

8 That would require me looking very, very clearly
9 at the raw data, the techniques, evaluating them, asking
10 for information way beyond either what I desire or in
11 fact what they would probably like to for relate to me.

12 My focus, again, let me emphasize again, has
13 been to review the assertions made by Dr. Q. This is my
14 role in these proceedings, is to look at his assertions,
15 to look at the evidence that he has cited, and do
16 evaluate that and give my opinion on that.

17 Q. You consider yourself an expert on current
18 diagnostic methods in the cancer field?

19 A. Well, I'm not quite sure what such an expert
20 might be. I have certainly through family history and
21 associations, I certainly am perhaps more knowledgeable
22 than most about strategies that may be relevant.

23 But, again, this revolves around many, many
24 factors we have not discussed today.

25 Q. Were you aware of the NeXT Personalis assay

1 before you were asked to join these proceedings?

2 A. If I recall, when I was first contacted for at
3 least these two cases or the cases before -- I guess
4 we're back in which January last year, I guess.

5 I was aware of Personalis in a sort of a loose
6 sense. Obviously what I do every morning is take a
7 little look at my Twitter feed, see what's going on.

8 I knew that they had released an assay of some
9 kind. And as in all these things, you kind of wait and
10 see what happens, is it all going to work out or not.

11 And that was pretty much the extent of my
12 interest and knowledge.

13 MS. SELLERS: So let's take a look, I'm going to
14 introduce for the record Exhibit 1001.

15 (Exhibit 1001 was marked for identification by
16 the court reporter and is attached hereto.)

17 BY MS. SELLERS:

18 Q. Do you have a copy of that?

19 A. Can you tell me what it is and that might help.
20 Is that the patent.

21 Q. Yes.

22 A. I have a copy here, I'm happy to look at your
23 screen though.

24 Q. This is Exhibit 1001 from IPR 546.

25 A. Which is the '611; is that correct?

1 Q. Correct.

2 Can you see is the patent, Dr. Furneaux?

3 A. Yes, I can.

4 Q. And you've reviewed the '611 patent, correct?

5 A. Yes, I've reviewed it. It's generally my
6 experience that the parts of specification and claims
7 are the key pieces. But I have reviewed it, yes.

8 Q. Correct. So have you reviewed the
9 specifications and claims in their entirety?

10 A. To the best of ability, yeah, I've reviewed it.

11 Q. So let's take a look at a specific portion of
12 the specification, which is -- sorry, it's not going to
13 let me.

14 A. Right-hand side or the left-hand side?

15 Q. The right-hand side.

16 A. Okay, sure.

17 Q. So it states (as read):

18

19 In some embodiments, the subject
20 has cancer or is suspected of having
21 cancer, and wherein at least one
22 biological sample includes a tissue
23 sample or a blood sample from the
24 subject.

25

1 Correct?

2 A. That's what it says.

3 Q. So if we go back to your declaration for a
4 moment, paragraph 166, which I will pull up for you.

5 A. Thank you. 166, all right.

6 Q. So you state here, you see your declaration on
7 my screen?

8 A. Yeah, I can.

9 Q. So you state (as read):

10

11 The claimed method requires
12 sequencing nucleic acid molecules from
13 at least one biological sample, which
14 can be a tissue sample, blood sample,
15 or tumor sample.

16

17 Correct?

18 A. I see that.

19 Q. And you cite to column 8, line 63 to 65 of the
20 '611 patent, correct?

21 A. That's what it says there. I hope that's
22 correct.

23 Q. I'm going to pull these up side by side so we
24 can see them quickly together.

25 Can you see both of these on my screen?

1 A. Yeah, I do.

2 Q. Perfect. And do you see that lines 63 to 65 are
3 the section that we just read together?

4 A. Yeah, that's gratifying that it's correct.

5 Q. And did you state in your declaration that "the
6 at least one biological sample can be a tissue sample,
7 blood sample or tumor sample" because that's what's
8 stated in the specification on line 63 or 65?

9 A. Yes, I mean yes. I'm just taking another look
10 at it, not exactly the same wording is it, and, but it's
11 representative, yes.

12 Q. So if one were to say that the "at least one
13 biological sample" as referred to in the '611 patent
14 wasn't a blood sample. Or couldn't be a blood sample,
15 would you agree with them?

16 A. You have to ask me that question again.

17 Q. If someone were to say that the "at least one
18 biological sample" referenced in the '611 patent, could
19 not be a blood sample, would you agree with them?

20 A. Well, you know, I would need the context of
21 that. What the specification here says is that it can
22 be tissue, blood or tumor.

23 So I would agree with you if someone says, no,
24 it cannot be blood, then that would be contrary to the
25 information that's in the specification here. Or

1 contrary to the I would say advice.

2 Let's see what this said. Requires. Yeah, I
3 would regard this as advice. And if someone said, no,
4 you cannot use blood sample as the one biological
5 sample, that would contravene the advice given here.

6 Q. And you would agree it's not correct to
7 contravene the advice given in a specification?

8 A. Well, yeah, I think we're straying into legal
9 grounds here. And in my view, in a scientific view,
10 they're saying one can use tissue, blood or tumor
11 tissue, that what would be my expectation.

12 Q. All right. So if one were to give an example of
13 what at least one biological sample could be in the
14 specification, you would want to use that when defining
15 what at least one biological sample means?

16 A. Well, again, it depends on the particular
17 argument that we're revolving around here.

18 But as a generality, there's an instruction here
19 that one biological sample at least, let me put it that
20 way, can be a tissue sample, can be a blood sample or a
21 tumor tissue.

22 Q. If you had -- apart from this specification that
23 said widget A can be B, C, or D, you would say it's
24 incorrect to exclude D from that list?

25 A. Well, given the positive information you just

1 outlined, without any other information that may
2 contravene that we just discussed, that would be a
3 logical conclusion.

4 Q. Thank you. And then lastly, you agree it's
5 really important or it's important to consider the
6 content in the specification when interpreting claims in
7 a patent, correct?

8 A. Sorry, you went in and out there. I didn't hear
9 that.

10 Q. It's important to consider the specification --
11 start that one over.

12 It's important to consider the disclosures in
13 the specification when interpreting the claims of a
14 patent, correct?

15 A. Yeah, I think we're moving into believable
16 considerations. But there is an example I can think of
17 in the present declaration.

18 I'm blanking on which particular patent it might
19 be. But I'm given to understand that one can take
20 elements from the specification where appropriate and
21 use those to further define or use those to produce a
22 more sample informative definition, for example, where
23 one --

24 (Technology discussion.)

25 THE WITNESS: We could have had even perhaps end

1 there. Let me just add a different piece.

2 In my endeavors, what I'm most interested in is
3 whether that combination can give me a test to apply to
4 the notions that are asserted.

5 BY MS. SELLERS:

6 Q. So were you familiar with Foresight Diagnostics
7 prior to Personalis approaching you about these IPRs?

8 A. Gee, I remember Derek telling me that the
9 opposition was Foresight. And I remember thinking
10 vaguely. Again it's a name that flashes across my
11 Twitter feed and stuff is going on.

12 It is my habit to perceive these kinds of things
13 and then wait until something, you know, quite
14 definitive that I can latch onto occurs.

15 As you well know, this is a very busy area where
16 every company, I think, is suing another company and
17 companies are coming up and disappearing, being taken
18 over.

19 If you blink, what you just read about is
20 already being done by another company. You know, it's a
21 very complicated and fast-moving arena.

22 So I pay a certain level of attention but I
23 don't do a big depth analysis.

24 Q. Did you know Jake Chabon, one of Foresight's
25 founders prior to this --

1 A. I would sincerely doubt it. If he's been at any
2 science meetings that I've been at, he'd probably know
3 who I am.

4 Q. What about Ash Alizadeh?

5 A. No. And let me just make a little point here.

6 DNA sequencing has evolved significantly since I
7 personally was working at the bench. And to a large
8 extent -- and this is not meant to be a disparative
9 comment but I would say the majority of the field has
10 now been populated by bioinformatics experts and perhaps
11 more engineering disciplines.

12 And there's been much more focus on that end
13 rather than the actual biochemical techniques that are
14 involved. Just a throw away point.

15 So to answer your question, no, I probably
16 wouldn't know who these folks are because I think they
17 are probably more likely in that bioinformatic sphere.

18 Q. Are you aware of Maximilian Diehn?

19 A. No.

20 Q. All right.

21 A. I mean, not before. Maybe I saw the name.

22 Q. And then David Kurtz, do you know -- were you
23 aware of already or did you know of David Kurtz?

24 A. No, there's a general thing in science that you
25 don't tend to -- I wouldn't say want to know people.

1 But you know people by what they do, not who they are.

2 Q. So let's go back to your declaration, which is
3 Exhibit 2169. And we'll go to paragraph 91, I will pull
4 that up for you.

5 Do you see that here?

6 A. I do.

7 Q. So you cite a few articles by the Foresight
8 founders in this paragraph, correct?

9 A. Yeah.

10 Q. Have you read these articles before you worked
11 on these IPRs?

12 A. No.

13 Q. Where did you get these articles from?

14 A. I think these particular articles were brought
15 to my attention by the legal team.

16 Q. Okay.

17 A. It seemed to me as a somewhat obvious areas
18 since these are the -- in fact, I don't think I've seen
19 it before.

20 But these are actually the oppositions' company
21 owners and these are their papers.

22 Q. And then what was your thought process when
23 deciding to include these in your declaration?

24 A. Thought process. Well, in general, it's quite
25 possible, and I think quite likely, that if one examines

1 these, they're quite potentially going to say things
2 that would be not supportive of some of the assertions
3 in Dr. Q's declaration.

4 Q. Was -- did counsel's decision to give you these
5 articles influence your decision to put them in your
6 declaration?

7 A. As in all cases materials provided to me,
8 there's a discussion about what it may or may not do in
9 terms of being inserted in my declaration.

10 And I take a review of that. And say, yeah, you
11 know, it helps. Sometimes there are strong points,
12 sometimes there are not so strong points.

13 And, you know, there's a level of judgment of
14 how much you put it in here.

15 Just by way of explanation, the way I approach
16 these exercises is very much the way that I review
17 academic papers and folks' grants back in the old days.

18 And the sort of modus operandi there is to look
19 for fatal flaws. If one looks for fatal flaws and one
20 accumulates a certain level of uncovered fatal flaws,
21 one feels one's job is done.

22 So I think it's important to point out that my
23 declaration does not include all the fatal flaws that
24 perhaps could be uncovered in Dr. Q's assertions but
25 merely some.

1 Q. So let's talk about the '611 and '783 patent
2 disclosure. If I represent to you that they have the
3 same specification, do you accept that?

4 A. You blanked out on the very first part of that.

5 Q. Do you understand that the '611 and '738 patents
6 have the same specification?

7 A. You mean, are they similar?

8 Q. Yes.

9 A. In a broad sense, yes. I mean, in terms of
10 specification, yeah.

11 Q. Correct.

12 A. My understanding is that's why they're being
13 grouped together in this discussion in the first place.

14 Q. Yes.

15 A. It's a decision by someone else not by me.

16 Q. If I generally represent to you that they have
17 an almost identical specification, would you accept
18 that?

19 A. I'm happy to take your word for it. I haven't
20 walked through it and analyzed each, you know, dotting
21 the -- well, crossing the T, dotting the I.

22 That be my general perception. But, you know, I
23 always live to be surprised.

24 Q. Let's just discuss the '611 patent, it discusses
25 performing whole genome or whole exome sequencing on a

1 tissue, blood or plasma sample, correct?

2 A. Yeah, if you don't mind, I'm going to pull up
3 for myself, I think, we pulled together a nice little
4 sort of comparison of the two.

5 Do you think that would be useful for my --

6 Q. We're talking about the specifications here, not
7 necessarily the claims. I don't think we need to look
8 at that.

9 But in general the patent discloses performing
10 whole exome or whole genome sequencing on a tissue,
11 blood or plasma sample from a patient, correct?

12 A. Yeah, I don't want to be pedantic. But I hope
13 you will understand we're now on something like the
14 fifth patent.

15 And so I do need a little mental refreshment now
16 and again. If you could just direct me to that passage
17 that you're talking about, I'd be grateful.

18 Not to denigrate what you're saying at all. I
19 totally understand what you're saying. But I just want
20 to make sure we are talking about the same thing.

21 Q. Yes. I guess do you know in a general sense
22 having reviewed these patents whether they do generally
23 disclose performing whole exome genome sequencing on a
24 tissue, blood sample or plasma sample from a patient?

25 A. Well, again, that's a very generic statement.

1 I'd much be -- much happier if I were looking at
2 the abstract of the patent before I said something that
3 may actually conflict with what the patent says,
4 unintentionally, of course.

5 Q. We'll come back to this in a little bit then.

6 So let's go ahead and start discussing some of
7 the references that are cited here. You reviewed the
8 Ley reference, correct?

9 A. Oh, yeah, yeah, yes, very happy about that.

10 Q. So in Ley's experiment, it requires the tumor
11 sample contain a blast count greater than 80 percent,
12 correct?

13 A. Well, again I don't want to be pedantic. But
14 when you say "required," that's a little bit of a
15 strange expression.

16 Q. Sorry, can you repeat that?

17 A. You said it "required" a certain blast count. I
18 wonder if you could you clarify for me who required
19 that, what requires that, why is it a requirement.

20 Q. So in order for a subject to have been enrolled
21 in Ley's experiment, the samples taken had to have a
22 greater than 80 blast count, correct?

23 A. Again, I would dispute your characterization of
24 that. It's my understanding that Ley on deciding that
25 they wanted to do whole genome analysis, brave endeavor,

1 selected this patient by virtue of the characteristics
2 of this particular patient.

3 One of which was that, in fact, the percentage
4 of tumor cells is extraordinarily high,
5 extraordinarily -- let me say perhaps not through the
6 lens of a leukemia world. But it is certainly high
7 versus all the other typical solid tumor worlds that one
8 could think of.

9 MS. SELLERS: Let me introduce previously marked
10 Exhibit 1042 to the record.

11 (Exhibit 1042 was marked for identification by
12 the court reporter and is attached hereto.)

13 BY MS. SELLERS:

14 Q. Dr. Furneaux, I think I sent this to you. But I
15 can pull it up as well. It is the supplementary
16 materials or information from the Ley reference.

17 A. It's Ley supplement, sure. I'm not sure I have
18 that.

19 Q. I believe we e-mailed it to you. But I also
20 pulled it up right here?

21 A. All right.

22 MR. LUMAN: There's a copy in your binder.

23 THE WITNESS: Do we have a number on that,
24 exhibit number?

25 MR. LUMAN: Should be 1042.

1 THE WITNESS: I've got it here on paper.

2 BY MS. SELLERS:

3 Q. So you see here at the top of the page, it says
4 "rationale for selection of an AML genome for
5 sequencing," correct?

6 A. Yeah, I think that's pretty much what I just
7 said. They decided to select, they're calling it a
8 specific genome, for the reasons elicited underneath.

9 Q. If you look here it says, "one of the criteria
10 for selection was M1 AML required high sample purities,
11 e.g., a blast count of greater than 80 percent,"
12 correct?

13 A. Yes.

14 Q. So Ley required that the tumor sample to contain
15 a blast count of greater 80 percent; is that fair?

16 A. Yeah, I think we need to finish the sentence:
17 "To eliminate the need to further purify the malignant
18 cells."

19 Q. So a blast count greater than 80 percent means
20 that 80 percent or more of the cells in the sample were
21 cancer cells, correct?

22 A. Well, we have to be careful. I think that's a
23 reasonable thought. And, of course, this is by visual
24 inspection.

25 It doesn't say here how that was determined.

1 But I think we can go with the fact that a reasonable
2 pathologist looked at this and concluded that in fact it
3 was greater than 80 percent.

4 As we will see if we go through the paper, this
5 would appear to be correct.

6 Q. Then the initial tumor sample that Ley used in
7 the experiment, that contained 100 percent myeloblast?

8 A. Right. I think you're correct. I think there
9 was a statement to that effect in the paper.

10 We can go do that if you want, we can double
11 check. But I suspect you are correct in your statement.

12 Q. All right. And so that means that that sample
13 essentially had 100 percent tumor cells; is that
14 correct?

15 A. That would be your expectation, yes.

16 Q. So within what sample, could there have been
17 intratumor heterogeneity?

18 A. There may very well be.

19 Q. And can you define what we would mean by
20 intratumor heterogeneity?

21 A. Well, you've introduced the phrase.

22 Intertumor, so let me just check what you mean
23 by that is could there be more than one variety of tumor
24 cell, albeit when the pathologist looks in the scope and
25 says, hey, it's 100 percent.

1 But could there be 5 percent of that 100 percent
2 whose phenotype is subtly different.

3 I think that's your question.

4 Q. Yes. Perfect, thank you.

5 A. Absolutely could be. And in fact I think
6 there's evidence in Ley that may well be the case.

7 Q. When were these samples taken from the patient
8 in Ley; do you recall?

9 A. I think that's a difficult one. Let's call her
10 Mrs. X was deceased when they started these studies.

11 Q. Correct. But the patient was alive and
12 receiving treatment for cancer when they took these
13 samples, correct?

14 A. I totally agree. I don't think the time was
15 identified, especially vis-à-vis treatment strategies or
16 whatever. I don't recall that.

17 Please feel free to correct me. But like we
18 took the sample day X and the next day we did this, I
19 don't think that was clear.

20 Q. But we can agree that the patient was alive when
21 the samples were taken?

22 A. I would certainly hope so. It's my
23 understanding that those samples were banked. I guess
24 amongst all the sort of possible to-ing and fro-ing, I
25 would imagine that she was, you know, checked for the

1 extent of blast cells on at least one occasion, that
2 would make a lot of sense to me. I don't know why you
3 may necessarily bank the sample and what those
4 conditions were.

5 These are all things that are not clarified,
6 primarily, I think, because the authors really were not
7 primarily interested in that aspect.

8 Q. So to avoid any doubt, let's take a look at the
9 supplemental materials, Exhibit 1042.

10 And look at the highlighted portion tab, which
11 specifies that the specimens of bone marrow, peripheral
12 blood and skin were collected and cryopreserved for
13 genomic studies.

14 And Dr. Furneaux, when you read this and the
15 context around it, it's clear that the patient was alive
16 when they collected these?

17 A. Oh, yeah, I'm not disputing that. I didn't mean
18 to give you that impression.

19 What I'm saying is obviously when someone is
20 deceased, it does make life a little difficult going
21 back and trying to figure things out.

22 That's all I'm merely saying.

23 Q. All right. Take a look at a paragraph in your
24 declaration, we'll go to paragraph 96. And I will pull
25 that up for you.

1 In this, you state that Ley's assay was only
2 able to detect allele frequencies at approximately 3
3 percent for the skin sample, correct?

4 A. I'm sorry, I'm seeing something different here.
5 Am I at the right place? (Reading inaudibly) -- at
6 approximately 3 percent for the skin sample, yes.

7 Now what was your question?

8 Q. I wanted to make sure we were on the same page
9 that you see that.

10 How did you come to that conclusion?

11 A. Well, can we look at the actual data?

12 Q. We can pull up the Ley reference itself.

13 A. It might be fig. 3, the legendary fig. 3.

14 MS. SELLERS: Introducing premarked exhibit
15 1012.

16 (Exhibit 1012 was marked for identification by
17 the court reporter and is attached hereto.)

18 BY MS. SELLERS:

19 Q. Do you see my screen?

20 A. I do. If you can make it bigger? Thank you.

21 I'm looking at that phrase in my declaration.
22 And I think that can be improved upon. I think it's a
23 little too cryptic.

24 Was that your question, as to the crypticness of
25 that statement?

1 Q. So how would you improve upon that statement?

2 A. Well, this is in essence is Ley and what they're
3 interested here is to determine actually the percentage
4 of tumor cells.

5 And they're determining the percentage of tumor
6 cells by using the knowledge that they've already
7 established that there are, in fact, variant alleles in
8 this population. And they're using that information
9 that they already have.

10 And the objective here is to determine, well,
11 you know, what percentage of these cells are tumor.

12 Now, it's important to point out that what
13 they're using here are two controls. They have
14 determined -- they have utilized the information that
15 they already know in that this individual actually
16 inherited a homozygous mutation.

17 So what they're doing here is giving you in
18 essence an internal control. This patient unfortunately
19 or well this patient has in fact a homozygous mutation.

20 What that means is there's a mutation in both
21 alleles, the allele this individual got from the mom and
22 in the allele that this individual for from her dad.

23 Now, what that predicts, then, is that you know
24 that if you look at the percentage of such cells in this
25 population, it should be 100 percent because both

1 alleles are mutated.

2 And, again, let me stress these are inherited
3 mutation. And, again, Mrs. X has inherited a mutation
4 in BRCA2, but only in one allele, maybe dad's or mom's,
5 we would not know in this analysis.

6 And that's what one would expect then is only 50
7 percent of the cell would be, in fact, redolent of this.

8 And, in fact, that's the case, so that's a nice
9 internal control. And, of course, that status would
10 pertain to what we call here the primary tumor, what
11 they call the relapsed tumor, and what they call the
12 skin, all good.

13 Now, the problem starts coming in determining
14 how sensitive this assay is. And the problem is
15 manifested in the yellow bars, which are the skin, which
16 are one would have to say, hesitate to use the word, but
17 ridiculously high.

18 For example, the FLT3, which is not a single
19 point mutation, it's actually, if I recall, an insertion
20 or deletion, is ridiculously high. And even the most
21 modest one, which maybe is EB12, I think, is still
22 coming in something like 5 percent.

23 So within my expertise of evaluating the
24 sensitivity of an assay, one has immediate problems
25 here.

1 The background in the assay, which is
2 understandable to some extent, they ascribe this
3 background to the fact that the normal skin sample will
4 wind up having leukemia cells, tumor cells, and that's
5 sort of understandable, although one may be critical of
6 the investigative approach here.

7 But what that does, is it makes life very
8 difficult to enable a sensitive assay.

9 And so I think the statement I gave is a little
10 too cryptic. I'm not recalling what the authors
11 themselves said is the sensitivity.

12 But clearly, simply put, you would be hard
13 pressed to make a conclusion given that what you're
14 looking at is the red and green bars, that's what you're
15 really interested in, and which, of course, as one might
16 expect most of these come in at 50 percent, because they
17 do assure us that these are heterozygous mutations.

18 So one would expect 50 percent, one would expect
19 the green material, which is the relapsed tumor, which
20 has less cells, to go down a wee bit and it is kind of
21 what you expect, although there's a bit of bouncing
22 around.

23 But the real problem here is you have such a
24 high background that this is going to preclude you from
25 determining the sensitivity of the assay with any

1 precision.

2 And what I think I did, if I recall correctly,
3 and it's probably a generous estimation, is I sort of
4 averaged the background there and said, okay, well gee,
5 you know, if it was less than that, the average of all
6 those yellow lines then, hey, you stand no chance at all
7 to make an assay.

8 Q. So your conclusion of this 3 percent came from
9 measuring the variant frequencies in the skin sample?

10 A. Yeah, if I recall correctly, the first problem
11 with an assay like this is the sensitivity is dictated
12 by the unfortunate high level that one sees in what
13 ostensibly one regards as the normal sample.

14 That's a real complication for such an assay.

15 And so if my memory serves me correct, I merely
16 looked at this diagram and figured, oh, it's probably
17 about 3 percent.

18 It may be worse than that to be fair. And I'm a
19 little concerned about my one sentence statement there.
20 I think it probably should have been less cryptic than
21 it is.

22 I think that crypticness is reflected in my
23 dismissiveness of rather poor data like this.

24 Q. So isn't it possible that Ley's skin sample just
25 didn't have tumor variant or tumor cells at a frequency

1 of 3 percent, is that possible?

2 A. That's certainly possible and, in some cases, up
3 to 15 percent. We're talking about very much
4 biochemistry here and the nature of assays and
5 sensitivity.

6 What's a little alarming here is, yeah, one
7 could say, sure, the background is leukemic cells.
8 Sure, that's the first possibility.

9 But it's a little surprising then if that
10 background is not constant. And what's a little
11 surprising, for example, if you look at FLT3 and I'm
12 going by memory but I think that was something -- coming
13 in at something like 15 percent.

14 And I wouldn't make too much obvious sense given
15 that in the actual tumor samples it's a guy who's less
16 than everybody else. That's a little perplexing.

17 So when I reviewed data like this if I was
18 referring this for a grant or a paper, I would be
19 thinking this is not very reliable data.

20 And as I is said before, it must have been a
21 very generous day when I wrote that sentence about the 3
22 percent. Can we just see what the author said because
23 I'm not recalling what the author said was the
24 sensitivity.

25 Q. I have one more question for you.

1 A. Sorry, I don't think I answered your question.
2 Let me apologize.

3 What you were saying is can't we just say, hey,
4 that was the background?

5 But just so you can understand, let's imagine
6 instead of this particular scenario, let's imagine we
7 have the same background.

8 And now we have samples coming in at let's just
9 say something like 1 percent, then we're going be faced
10 with a difference between with the NP1 -- with the FLT3,
11 for example, it's going for be 15 versus 16, right, oh,
12 it's getting very difficult to discern, right.

13 So I hope you now see the difficulty of having a
14 high background, how that severely crimps your
15 sensitivity.

16 Q. Well, isn't it possible that Ley could say have
17 defeated allele frequencies below 3 percent, that just
18 the skin sample by itself had an average of the 3
19 percent of the variant mutations?

20 A. Yeah, but the objectives here is it to determine
21 the percentage of tumor cells in the tumor samples,
22 that's the objective. Having high background
23 compromised that objective.

24 Q. I'm just asking because you discussed the
25 sensitivity. So the sensitivity could be less than 3

1 percent, correct, if the skin samples did, in fact, have
2 less than --

3 A. We have no way of knowing.

4 Q. Right.

5 A. Sorry, I just want to finish that thought.

6 Unfortunately in the sort of biochemistry review
7 world, this is just not good data. What one would do is
8 say please go and do this again and do certain things
9 that we can suggest so that we can get a much better
10 view of what you're saying.

11 MS. SELLERS: So we've been going for about an
12 hour and 15 minutes. So I think it's time to take
13 another break.

14 What works with you Nate, Dr. Furneaux.

15 THE WITNESS: Is this lunch we're talking about?

16 MR. LUMAN: Let's go off the record at 9:41.

17

18 (Whereupon at the hour of 9:41 a.m., a
19 luncheon recess was taken. The
20 deposition was resumed at 10:33 a.m.,
21 the same persons being present.)

22

23 MS. SELLERS: Back on the record.

24 MR. Luman: Back on the record at 10:33 Pacific.

25 ///

1 BY MS. SELLERS:

2 Q. So --

3 A. I wondered if it's possible to go back to that
4 figure 3. Since I realized thinking about it over lunch
5 I was being maybe a little over cryptic.

6 And I'm not sure of your background -- what's
7 your background. I don't want to denigrate your
8 background and education but it's important I know if a
9 Ley person or have some idea of some --

10 Q. Trained in biochemistry.

11 A. All right, well, in that case, I think I should
12 be less cryptic. If we can look at figure 3.

13 Q. We can discuss that a little bit later. Let's
14 move on to a different point right now.

15 A. I'm probably going to forget it as we go on.

16 MR. Luman: The witness has asked to clarify an
17 answer. I think we should let him clarify the answer.

18 MS. SELLERS: Let's pull it back up.

19
20 (Whereupon a discussion was held off
21 the record.)
22

23 THE WITNESS: In terms of my crypticness, I
24 think you will know exactly what I mean, Abby.

25 So imagine you were a graduate student, you've

1 done this experiment, you come to me and I'm reviewing
2 it, okay.

3 The purpose of the skin sample in a very general
4 sense is to convince one that the assay's working. You
5 expect nothing and if you get nothing, you say, great,
6 the assay's working.

7 Now, when you see such ominous signals and the
8 skin normal, then one thinks of possibilities. And I
9 think you will actually entirely understand, the
10 theoreticians of the world, those who don't do
11 experiments might be thinking, oh, well it's probably
12 tumor cells in the skin.

13 The more skeptical empirically derived folks
14 like myself would say that's one possibility. And the
15 other possibility is that the assay is misleading us.

16 For some crazy reason, could be lots of things,
17 hey, it's actually misleading us, it's reporting that
18 there is a mutation but there actually isn't.

19 And I'm sure you know some of the simplest
20 notions of how this could occur, not denigrating
21 graduate students.

22 But, gee, you know, somebody actually managed to
23 get some of the primary tumor sample into the skin
24 sample.

25 Has that ever been done before? Of course, it

1 has. So I hope you understand why I'm skeptical about
2 this particular data and whether in truly in fact this
3 yellow material that you see here is in fact redolent of
4 leukemia cells.

5 And the other aspect that's worth stressing to
6 you is if someone had gathered the skin cell with an
7 intentionality of doing this experiment, they would be
8 well aware of this potential complication.

9 They would probably do very simple things like
10 perfuse the skin, treat it in some way, perhaps even
11 instead of taking the whole skin, take an area that has
12 been deemed to be normal, all sorts of things you would
13 do.

14 And so something I also want to relate is when
15 the skin sample was gathered, it strongly suggests to me
16 there was very little intentionality about the ultimate
17 use of this sample.

18 In other words, it had nothing gathered for the
19 express purpose we're looking at. It was probably
20 gathered generically with no intentionality.

21 BY MS. SELLERS:

22 Q. All right, thanks for the clarification. So if
23 we look at the description of figure 3, it does note
24 specifically that the normal skin sample was
25 contaminated with leukemia cells, correct?

1 A. That's correct, Abby. And you and I both know
2 that that's a conclusion and we're wondering what's the
3 evidence.

4 One looked at the skin and saw leukemic cells.
5 Or did one surmise that that must be the case because of
6 the perhaps erroneous interpretation of the results.

7 It's ambiguous is what I'm saying. To be fair
8 to the authors, this was not the objective of the
9 authors and I'm holding them up to a higher level of
10 scrutiny only in terms of the assertions that your side
11 is making.

12 Q. So let's turn to Exhibit 1001, which is the '611
13 patent. And I just wanted to look at one of the claims
14 quickly, claim one.

15 Do you see that here on my screen?

16 A. I do. Sorry, we're in the patent now, right.

17 Q. Correct. The '611 patent.

18 A. I don't think I need to see that independently,
19 I think this will surmise.

20 Q. I've highlighted a limitation here which says,
21 "subjecting said sequencing library to a coverage of 175
22 kilobases or less."

23 What do you interpret that to mean?

24 A. Well, what I did interpret that to mean is one
25 is going to look -- in other words, we're going to look

1 at 175 kilobases or less.

2 In other words, the extent of our looking at
3 things.

4 Q. So the number of bases within the gene you're
5 looking at would be 175 kilobases or less?

6 A. Well, I don't know if 175 kilobases within a
7 gene is probably a little unlikely. But I think my
8 interpretation of this is this is the extent of the
9 segments of the genome that we're going to look at, the
10 extent.

11 Q. So let's go look back at Exhibit 1012, back to
12 Ley. And we can look at this flow chart together in
13 figure 2.

14 Do you recall this flowchart?

15 A. Absolutely, yeah.

16 Q. So in this flow, Ley discloses how they filtered
17 from approximately 3.8 million -- approximately 3.8
18 million single-nucleotide-variant mutations that were
19 identified in the tumor down to the 8 validated somatic
20 mutations, correct?

21 A. That's correct, yes.

22 Q. All right. And so would you agree that Ley
23 filtered out all single-nucleotide variants in non-genic
24 and intronic regions?

25 A. Well, let me look at this more carefully. Ley

1 elected only to study single-nucleic variants in coding
2 sequences. I think that's the sixth lines down in the
3 flow diagram. That's what they elected to do.

4 Q. So you would agree that Ley elected to only
5 focus on single-nucleotide variants in exons?

6 A. Well, in coding sequence because remember exon
7 is ambiguous, there may be exons in noncoding genes.
8 But, you know, I would agree with your parlance, they
9 are looking at coding sequence.

10 Q. And so Ley narrowed this down to the 8 validated
11 somatic mutations, and at that point, Ley would have
12 known the exact exon and location in the human genome of
13 these eight mutations, correct?

14 MR. Luman: Calls for speculation.

15 THE WITNESS: Yeah, one would hope so. I don't
16 recall seeing evidence to that end.

17 If I recall correctly, these were validated,
18 confirmed in the first instance by Signer sequencing of
19 a PCR amplicon, if I recall, with unknown primers.

20 So in some senses, that might indeed -- one
21 would hope that it might truly identify an exon within
22 which the accommodations reside, provided there's no
23 complications and reiterated sequences.

24 And these are tumor cells, things jumping
25 around, you know. I think that's the hope there.

1 BY MS. SELLERS:

2 Q. But all of these mutations would have been
3 located within coding regions of exons, correct?

4 A. Well, we can double check that. Let's look at
5 the table where they describe the mutations at the
6 protein level.

7 Q. So you can't tell just from figure 2?

8 A. Well, you know there's harm in looking for more
9 evidence. You're referring to the H, starting at CD H24
10 and ending with EB12, correct.

11 Q. Um-hum.

12 A. Yeah, in each case here it appears there's an
13 amino acid with a number that has been changed. So that
14 does imply that what we're looking at here is a mutation
15 within a coding region, as they said, and it seems to be
16 ascribed to a particular amino acid whose number I
17 presume is coming from the -- well, is coming from the
18 interpretation of the referenced genome at that time,
19 I'm sure.

20 Q. So you can confirm that Ley would have known the
21 location of the specific exon and the base number of
22 each somatic mutation?

23 A. So what you are confirming and what I am seeing
24 is what Ley asserts. What's lacking here is the
25 evidence for that assertion.

1 It would have been useful to know what that
2 Signer sequence was, the right sequence.

3 Q. All right. So let's turn back to your
4 declaration for a minute. And if we go to paragraph 69,
5 and this is Exhibit 2169.

6 A. 69, okay, I see it.

7 Q. So you state that, "to identify the definition
8 in the context of the patent which means to establish
9 the identity of;" is that correct?

10 A. Yes, yes, I think it's always worth while saying
11 identify genetic variants but, yeah, we're looking at
12 the definition here of identify.

13 Q. Okay. And is this still your opinion today?

14 A. Very much so.

15 Q. So in this, you cite to the American Heritage
16 Dictionary, which is Exhibit 2170, which I don't think
17 we sent you yet today. But are you okay if I pull it up
18 on my screen?

19 A. Absolutely. I entirely trust your judgement
20 here.

21 MS. SELLERS: So this is previously marked
22 Exhibit 2170.

23 (Exhibit 2170 was marked for identification by
24 the court reporter and is attached hereto.)

25 ///

1 BY MS. SELLERS:

2 Q. So here we have the dictionary definition and
3 here is identifies very tiny in the top corner for us?

4 A. That's fine. Perfect.

5 Q. So where did you get this dictionary definition
6 from?

7 A. Well, I think it might be quite obvious that I'm
8 from Scotland and American English wouldn't be my
9 expertise.

10 And so the legal team indicated to me that this
11 is a typical resource they use when they think I should
12 be acquainted with the plain usage.

13 And so they presented me with this set here.
14 And my job was obviously to say okay, which one of these
15 most resembles or is in fact consistent with what I
16 would regard as identified.

17 Q. So the dictionary entry itself was provided by
18 counsel, correct?

19 A. Correct, absolutely.

20 Q. And did you consider any other dictionaries or
21 dictionary entries when --

22 A. No, I mean I, as I said, you know, there are
23 differences between British English and American
24 English. And I'm trusting the legal team to use this as
25 a source. I think it's quite bone fide as I understand.

1 Q. So this is from the American Heritage
2 Dictionary. Do you use that often in the field of
3 biochemistry?

4 A. Well, I think it's somewhat rare that I would
5 use a dictionary in biochemistry. I was fortunate to be
6 trained by the giants in the field and most of the words
7 that I became familiar with are actually based in
8 Yiddish.

9 So very rare I would consult the dictionary.

10 Q. Do you believe that the first dictionary
11 definition here -- the first dictionary definition,
12 which is "to establish the identity of" is a
13 comprehensive definition of identify?

14 A. Yes, I do, and if I can suggest you probably
15 already know that I'm very empirically based, I looked
16 at this and thought number one is the one, yeah.

17 Then I said to myself, well, how can I test
18 this. Suppose I directed a student to go and do some
19 sequencing and identify a mutation.

20 What would they come back and say to me if it
21 was successful? They would say, aha, I established the
22 identity of the allele T in the sample, okay, cool.

23 I don't think they would say, hey, I've
24 ascertained the origin and nature of definitive
25 characteristics of allele T.

1 I've determined the taxonomic classification
2 of --

3 (Reporter clarification.)

4 -- I've determined the taxonomic classification
5 of -- I think I'd be wondering really there. And I have
6 considered identical or united -- doesn't make any
7 sense.

8 I have associated affiliated myself closely with
9 a person or group, that would be very worrisome -- so
10 the one that fits the test is number 1.

11 Q. So in this context when you said my grad
12 students, hey, identified this variant -- that was when
13 they were discovering it, I guess, for the first time,
14 correct?

15 A. Yeah, I mean, I don't remember what objective I
16 gave out there. It could be for the first time and, in
17 fact, more than likely would be in that circumstance.

18 Q. So they are going to verify --

19 A. No, I mean, I might say that I go say, hey, go
20 and see if you can identify such and such.

21 Q. Okay.

22 A. What's important here is not the nature of the
23 objective per se but their response, and I think you
24 would agree that anything other than response number 1
25 would not make any sense.

1 Q. So could identify have and any meanings than to
2 establish the identity of?

3 A. I think we've already established that's that
4 what I think it means.

5 Q. Do you agree that the broadest possible
6 definition or could it encompass more than just to
7 establish the identity of?

8 A. Well, I think we're looking at five
9 possibilities, which would be a broad interpretation.
10 And I've identified number 1 as being the one I think is
11 the most relevant and most accurate.

12 Q. So outside of this dictionary definition, in
13 your own experience, would you classify identify as to
14 establish the identity of or would you say it is
15 typically construed broader than that?

16 A. I would say in -- again, this is a general
17 question you're asking me, I don't know what the context
18 of this is.

19 But if someone said to me I've identified
20 something, I would think, oh, you've established the
21 identity of it.

22 Q. So let's look at the actual claim language here
23 a little bit. And what I'm going to do is substitute
24 your definition back into the claims.

25 So here we have E, which says, "subjecting such

1 sequencing library to sequencing at a coverage of 175
2 kilobases or less."

3 And then I'm going to substitute in your words
4 here, "to establish the identity of the presence or
5 absence of at least a subset of said genetic variants."

6 How do you establish the identity of the absence
7 of something?

8 A. How do you identify the absence of something?

9 Q. How do you establish the identity of the absence
10 of something?

11 A. Yes, so we have to see this in the context, that
12 these are identifying genetic variants. So presence or
13 absence is related to whether, for example, the genetic
14 variant might be an A or a T.

15 If it's an A, then T would be absent, and if
16 it's a T, then A would be absent.

17 Q. So what you're saying then is there's two
18 possibility for each location and what you're saying is
19 you're always either going to have a presence or an
20 absence?

21 A. What I'm saying is only one example. There are
22 all sorts of possibilities. There may be a second sense
23 where you have an A in some reference point and you may
24 have a T or a C or a G, it depends on the circumstance.

25 But obviously, if you had something other than

1 the A, the A would be representing absence, and the T
2 would be representing presence.

3 Q. Okay. So just to be clear, your testimony is
4 that to establish the absence of something -- to
5 establish the identity of the absence, you have to
6 establish the identity of what's present?

7 MR. Luman: Mischaracterizes his testimony.

8 THE WITNESS: Yeah, I think we may be talking at
9 cross purposes here.

10 Presence and absence in the context of genetic
11 variants is in the most straightforward thinking that an
12 A is present and then an A is absent.

13 Yet it is fulfilled by a different base, a T.
14 I'm giving the simplest example of a single base change.

15 BY MS. SELLERS:

16 Q. Okay, so in Ley, it was dealing with
17 heterozygous somatic mutations, correct?

18 A. Well, can we look at the section you're
19 referring to?

20 Q. Let's leave the reference out of this for a
21 second. We were talking about earlier how Ley
22 identified the eight heterozygous genetic mutations,
23 correct?

24 A. I would agree with that, yes.

25 Q. And then you describe this in your deposition as

1 the validation step of the process where Ley takes each
2 of the samples and sequences them and tallies up how
3 many times each base is read, correct?

4 A. No there was a step before that.

5 Q. I'm just talking about the validation step.
6 Do you recall that step?

7 A. There were two validation steps.

8 Q. So the first validation step was the
9 amplification through PCR and then the sequencing,
10 correct?

11 A. By Sanger, S-A-N-G-E-R.

12 Q. So in this pool of samples that Ley is reading,
13 for each specific base pair that it's looking at, there
14 would be two different nucleotides, right?

15 For example, there could an A on one side and a
16 C on the other; is that correct?

17 A. Well, I think we have to be a little bit more
18 precise than that. Let's call it dad's allele and mom's
19 allele.

20 Let's assume A is the reference base, just for
21 the sake of argument. So you would have an A on mom's
22 side and a T on dad's side, if it was dad's side -- if
23 it was dad's allele, A-L-L-E-L-E, that had suffered the
24 somatic mutation.

25 Q. Okay, but in each of these cases, you have two

1 different bases that Ley would be sequencing, correct,
2 at the given location?

3 A. Well, yeah, we know what those bases are in Mr.
4 Ley, just FYI.

5 Q. I guess we do know what the codons change for --

6 A. No we know amino acid --

7 Q. Excuse me --

8 (Speaking Simultaneously.)

9 THE WITNESS: You are well aware that the
10 genetic code is degenerative, correct?

11 BY MS. SELLERS:

12 Q. Correct.

13 A. We have no information as to what actual base
14 was changed here.

15 Q. Okay. So when Ley is sequencing these DNA
16 samples, the tumor, the skin, and the recurrent tumor,
17 each one of those that it's sequencing, it's not going
18 to know for each read whether you have the mutated
19 version or the nonmutated version, correct, until that
20 whole strand of sequence?

21 MR. Luman: Objection, form.

22 THE WITNESS: I think you might want to try that
23 question again in a more direct way.

24 BY MS. SELLERS:

25 Q. So what I'm asking is that -- what you're saying

1 is we have a heterogeneous mutation, right? So we have
2 one normal copy, one non-normal copy?

3 A. Sure, I mean, for the sake of argument and I'm
4 sure you will understand this. Let's just say it's an A
5 and a T. We don't know what it is. But just for the
6 sake of argument so we can have something to talk
7 around.

8 Q. Okay, perfect. That is acceptable to me. So
9 let's say at the given location of the somatic mutation
10 we have an A and a T.

11 When that -- when the sequencer is sequencing
12 that specific location, you're not going to know for a
13 given strand, a single strand of DNA, whether that's an
14 A or a T, right, until the sequencer says A or T,
15 correct?

16 A. I would dispute that. You're very well aware
17 that there are As and Ts in there. What the figure 3
18 and the 454 sequencing is doing is simply figuring out
19 the number of such entities, which, of course, one
20 anticipates is going to be close to 50 percent.

21 None of this is going to be a surprise.

22 Q. Okay, but for a given read, you're not going to
23 know whether it's an A or a T until that read is done,
24 correct, and the key of that base is established?

25 A. Yes, I would disagree with your using the word

1 identity there. You're not going to confirm which read
2 has an A or a T until you look at the read, absolutely,
3 but the word there's confirm.

4 Q. All right --

5 A. -- on an average you're looking at one of two
6 reads. One's going to be an A and one's going to be a T
7 because it's at a 50-50. I know the data's like 45 and
8 55 but just allow me that for 50-50.

9 Q. Okay. All right and then so just to confirm, to
10 know whether there is an absence of a genetic variant,
11 you have to know in advance what to expect at that
12 particular position, correct?

13 A. And you certainly do know in advance. The
14 material has been sequenced first -- which I would
15 describe as the true identification phase. It's been
16 confirmed by Sanger sequencing.

17 Sadly, we don't know all the details. But as I
18 keep emphasizing, Ley didn't write this paper to fulfill
19 Dr. Q's expectations.

20 I'm sure that's a tragedy. Ley wrote that paper
21 for their own objectives. And perhaps the sequence as
22 revealed by Sanger was not remarkable.

23 But the sad thing is we don't know what that
24 was. But certainly that was a confirmatory step.

25 And as you can see in Ley, the beginning of the

1 paragraph is now a new quest: We would like to know
2 what percentage of this material is tumor cells.

3 It's an entirely new objective. We're going to
4 use what we know already, we know already there's As and
5 Ts in there.

6 And now all we want to know is how many As there
7 are and how many Ts there are.

8 To be clear this method would not work in the
9 first place, would not even be considered, unless one
10 was very sure that one knew that there were As and Ts.

11 Q. All right, so lets it talk a little bit about a
12 different part of your declaration.

13 If you can turn to paragraph 51.

14 (Reporter clarification.)

15 Okay, so let's turn to paragraph 51 of your
16 declaration.

17 A. I have it here and also on your screen.

18 Q. Do you see it on my screen?

19 A. Yeah, very well, thank you.

20 Q. So here you opine on the definition of "variable
21 portion." And this occurred between paragraphs 51 and
22 53 in your declaration.

23 So how did you come up with this definition of
24 variable portion?

25 A. And fixed portion.

1 Q. Let's just talk about variable portion for right
2 now.

3 A. Okay we'll talk about the variable portion. I
4 think we talked about this earlier this morning.

5 The definition -- or in my world, I would regard
6 it as the test, which is what you see in paragraph 53,
7 required relating to both the claims and this extra
8 piece that came from the specification.

9 And I'm reliably unsure that this is appropriate
10 to do. And that's how I came up with it.

11 Q. I'm sorry, did you say you were reliably unsure?

12 A. I've been reliably informed by the legal team
13 that it is appropriate to combine in this fashion
14 material that one sees in the specification of the
15 patent with the claims to, in my world, come up with a
16 test in essence, which is what you see in paragraph 53.

17 Q. So to use your language, you came up with this
18 test by using the example from the specification,
19 correct?

20 A. Yeah, I mean what you see in 53, I would regard
21 as in the legal world you would perhaps call this a
22 definition, which if I recall correctly, one not given
23 before in legal proceedings.

24 And so, yeah, this was basically the test with
25 which I used to examine any questions.

1 Q. And you included this example because in your
2 view it's inappropriate to exclude examples from the
3 specification from your test, correct?

4 A. I'd have to have that question again.

5 Q. So in your view, it would have been
6 inappropriate to exclude this example of variable
7 portion and fixed portion from the specification from
8 your test, correct?

9 A. I think that's a double negative. What I'm
10 saying is, I think the combination of the material in
11 the specification combined with the claim and gives me
12 something that I can use to test a notion wherein
13 someone might say this is variable, someone might say
14 this is fixed.

15 I will use this to determine what the meaning
16 may be.

17 Q. Did counsel direct you to this portion of the
18 specification?

19 MR. Luman: Objection, you asked him what did
20 counsel tell you.

21 That's privileged. I instruct him not to
22 answer.

23 BY MS. SELLERS:

24 Q. Did you get your definition from counsel?

25 MR. Luman: Same instruction.

1 BY MS. SELLERS:

2 Q. So how did you come to find this portion of the
3 specification?

4 A. By reading the patent.

5 Q. And why did you choose this specific portion
6 over others?

7 A. Well, you know, let me just digress for a minute
8 or two.

9 This is, I would say, a very innovative aspect
10 of this particular patent. They are broaching into
11 somewhat uncharted waters in terms of legalese and
12 definitions, and even in terms of science.

13 And so at first glance when I looked through the
14 patent, this triggered significant conversations, which
15 I'm not going to relate in detail, but as you might
16 guess, this was a significant talking point as I
17 grappled to find something I could use as a test that
18 fulfills both the legal implications and the scientific
19 implications.

20 And the best fit I could find was combining the
21 claims with in extra piece of information.

22 Q. I want to direct you to a section of the
23 specification. Can you see my screen?

24 A. Yeah.

25 Q. So this is from the '611 patent, which is

1 Exhibit 1001. And I want you to read this paragraph
2 right here.

3 A. (As read):

4
5 In some embodiments, the variable
6 portion of the oligo-directed genomic
7 content corresponds to variants that
8 may be responsible for the Mendelian
9 phenotype of a proband.

10 And the fixed portion corresponds
11 to one or more of, A, additional
12 genetic content not related to the
13 Mendelian condition of the proband, or
14 B, pharmacogenomics, or C, genetic
15 sample ID by a fixed panel of variants
16 or a fixed panel of phenotype-related
17 variants such as gender, blood type, or
18 D, variants common in the population
19 and used by B allele methods to detect
20 structural variation.

21
22 Q. My apologies, this is the wrong one. I wanted
23 you to read right above it, I apologize.

24 A. I can read more.

25 (As read):

1
2 In some embodiments, the variable
3 portion of the oligo-directed genomic
4 content corresponds to potential
5 neoantigen-causing variants of the
6 individual.

7 And the fixed portion corresponds
8 to one or more of, A, cancer driver
9 genes, B, genes involved in the
10 pharmacogenomics of cancer drugs, genes
11 involved in Mendelian immunological
12 diseases, genes related to inherited
13 forms of cancer, genes associated with
14 tumor escape from a targeted or immune
15 cancer therapy, HLA typing, variants
16 common in the population and used by B
17 allele methods to detect structural
18 variation.

19
20 Q. All right. Thank you. My first question is
21 does your definition of variable portion include
22 potential neoantigen-causing variants of the individual?

23 A. Yeah, I think it's important at this point to
24 point out that the claim pertains to the sequence of
25 what's called here the probe.

1 Q. Correct.

2 A. So this content here refers to allele directed
3 genomic content. It's a little ambiguous as to what
4 that is talking about. Are we talking about --

5 Q. I'm sorry --

6 A. -- the genome or the probe.

7 Now as I say, I sympathize with the authors
8 because they are trying to legally characterize quite a
9 new and somewhat complex concept.

10 Q. But in your definition would variable portion
11 include neoantigen-causing variants of the individual?

12 A. So neoantigen-causing variant is clearly -- that
13 would be a mutation, let's just say, in the human
14 genome, right.

15 Whereas the claim language in this recitation
16 are related to the sequence of the probe that is being
17 used, which may or more likely may not be, in fact,
18 encompassed in the actual variant.

19 Q. So are you saying that the probes have no
20 relation to the genomic content that they're contacting?

21 A. No, you're talking about here neoantigen-causing
22 variants, that implies that that would be, in fact, the
23 mutation itself.

24 And the probe that is being used in these
25 endeavors does not necessarily have to cover that

1 particular base.

2 Q. So let's go to the next portion, which is the
3 fixed portion.

4 Would you agree, consider these seven examples
5 of fixed portions to be included in your definition of
6 fixed portion?

7 A. Okay, so in my definition the fixed portion, the
8 most typical example of that would be are -- those would
9 be, for example, standard PCR primers that have been
10 used in the past to ascertain perhaps mutations in
11 cancer driver genes, that's entirely possible, genes
12 involved in the pharmacogenomics of cancer drugs, that's
13 plausible and possible, genes involved in Mendelian
14 immunological diseases, yes, I would think so.

15 Genes involved in inherited forms of cancer,
16 could be. Genes associated with tumor escape from a
17 targeted or immune cancer therapy, possible.

18 HLA typing, possible. Variants in the
19 population and used by B allele methods to detect, all
20 possible.

21 I guess what's missing from our conversation
22 here is one of the key distinguishing tests.

23 Q. Sorry, were you -- did you complete that --

24 A. I was wondering if you would ask me, that's all.

25 Q. So my question to you is did you consider this

1 passage when you determined your definition of variable
2 and fixed portions?

3 A. Yeah, I mean, I read it. I didn't think it was
4 adding much to the efficiency of my text. It's a very
5 generic thing.

6 Q. And do you agree that your definition should
7 cover the variants or the specific variants that were
8 listed in that?

9 A. Yeah, I guess we never really clarified any of
10 the key points in my definition. So one point that we
11 might want to clarify is that the probe sequence that is
12 used, if it was a -- could be called a variable entity,
13 would be derived from a sequence analysis of the
14 individual.

15 Whereas the -- coming around the other way, the
16 fixed guy would not be relying upon such information.
17 And in fact such information may be even garnered from
18 the reference database, probably has been used quite
19 extensively by many investigators, probably is
20 commercially available as such, and would not be
21 suspected to be different between individuals.

22 Nor in fact would its use probably be impinged
23 by the prospect of different individuals.

24 Q. All right. So that passage we were looking at
25 earlier in the specification, which I can pull back up,

1 would you agree that it was discussing these probes that
2 are based off genetic sequence?

3 A. Yeah, it's a very generic thing. And the thing
4 that is missing from that whole passage would be the
5 very experiment I just indicated whereupon one knows
6 whether the probe has been derived from sequenced
7 information from the individual or not.

8 That whole passage is silent on that very
9 critical test.

10 Q. Let's see if we can take a look and determine
11 that this passage is discussing the genetic variants
12 that are derived from an individual sequencing.

13 So I'm going to pull this back up.

14 Can you see my gene?

15 A. I see your screen. I don't see anything
16 highlighted.

17 Q. The passage currently is in here: It says in
18 some embodiments, see that?

19 A. Yes, I see that.

20 Q. And this entire paragraph is talking about, "in
21 some embodiments, the oligo-directed genomic content of
22 the second assay."

23 Do you see that?

24 A. Um-hmm.

25 Q. This paragraph gives many examples of what that

1 oligo-directed content of the second assay could be; is
2 that correct?

3 A. Yes, as I said before, that paragraph is
4 ambiguous. The authors are doing their best. But it
5 appears to be talking about the genomic sequence
6 primarily, not the sequence of the probe.

7 Q. Okay, so let's see if we can figure out what the
8 second assay is. So if we come up to -- here we go,
9 let's look at this paragraph right here.

10 A. So just to clarify, we're not looking at claims
11 here.

12 Q. No, we're looking at the specification.

13 A. We're looking at the specifications. Some of
14 which can be useful, some of which might not be useful
15 in this particular regard.

16 Q. Correct. Let's look at this paragraph right
17 here.

18 Let me know when you've had a chance to read it.

19 A. I'm sorry. I mean, I skimmed it quickly.

20 Q. Not a problem. So would you agree the second
21 assay in this case comprises nucleic acid probes that
22 are designed from the personalized genetic sequencing
23 that was done earlier on in this example?

24 A. Let me just see what this says.

25 "The first assay" -- so section B you're

1 referring to primarily.

2 (As read):

3
4 Specifying the design of the second
5 assay, in particular the sequences of
6 multiple -- from A, using a first assay
7 design to sequence the nucleic acid
8 derived from individual person,
9 determining multiple characteristics of
10 that person or their sample from that
11 data, using the genetic characteristics
12 from, B, to specify the design of a
13 second assay, and, in particular, the
14 sequences of multiple additional
15 nucleic acid molecules be used in that
16 second assay.

17 Synthesizing the additional nucleic
18 acid molecules on at least one array,
19 using the synthesized nucleic acids to
20 perform a second assay on one or more
21 samples in the same individual and/or
22 from individuals in their family.

23
24 Okay, that's a very general statement.
25 What was your question again?

1 Q. My question is in this second assay that's
2 described here, do you agree they are using nucleic acid
3 probes?

4 A. Would I agree they're using nucleic acid probes?

5 Q. Yes, in the second assay.

6 A. I think one could debate that. It's a little
7 ambiguous.

8 Some folks regard probes as being in liquid
9 medium, some folks say if a probe is sitting on a array
10 -- I mean if a sequence is sitting an array it can be
11 called a probe, and there is always a little bit of
12 debate about that.

13 Q. You've opined what a probe is in terms of
14 patent, correct, you've given opinions as to what's
15 considered a probe?

16 A. I have? Can you direct me to that?

17 Q. Well, you've defined certain things in the prior
18 art as probes or not probes, correct?

19 A. Can you direct me to where I say that in my
20 declaration?

21 Q. Well, that is a part of the claims, correct?

22 A. No, I know, I want to see the precise words I
23 used.

24 Q. That's not -- stay focused on what we're doing
25 right now, which is looking at this. Would you consider

1 what they're doing right now, in this paragraph we're
2 looking at to be a probe within the words of the claim?

3 A. They're not doing anything. They're talking
4 about a generalized statement on the method.

5 Q. Okay. Let's move on from that.

6 So let's go back to your declaration where you
7 talk about these fixed and variable portions.

8 A. Very good.

9 Q. And applying your definition, would you consider
10 a sequencer adapter such as those used with an Illumina
11 sequencer to be a fixed portion?

12 MR. Luman: Incomplete hypothetical.

13 THE WITNESS: Are we looking at paragraph 53?

14 BY MS. SELLERS:

15 Q. Correct, the definition that you applied in
16 this: Would you consider a sequencer adapter such as
17 those used in an Illumina sequencer to be a fixed
18 portion?

19 A. So let me think about that before I give -- for
20 a minute or two.

21 So what you're referring to is the sequences
22 that are typically appended let's say to the ends of
23 fragments to enable sequencing in a, for example, a
24 Illumina machine?

25 Q. Correct.

1 A. Is that what you're referring to?

2 Q. Correct, that is what I'm referring to.

3 A. Your question to me is would I call that a
4 variable or a fixed entity?

5 Q. Correct.

6 A. Okay. In the context, of course, that most of
7 our discussion has been about sequences derived from the
8 human genome.

9 Q. Correct.

10 A. So this sequence is not derived from the human
11 genome. So it would be outside my current purview,
12 outside the scope of my opinionating.

13 Q. I don't think it's outside the scope. So
14 looking at your definition of fixed portion, which you
15 said should be construed, "as which does not change from
16 one subject to another" and "is independent of the
17 results of sequencing data."

18 A. Yes, but you alighted upon an example that is
19 somewhat puzzling as to why one would be considering
20 whether it's variable or fixed in the first place since
21 its utility is clearly not related to the function that
22 we're ascribing to the variable and fixed portions.

23 Q. Well, let's look at here and we'll think about
24 this, I'm asking you --

25 (Speaking simultaneously.)

1 THE WITNESS: The sequence of an adapter, which
2 is not a sequence that one finds typically in the human
3 genome, is irrelevant to this conversation.

4 BY MS. SELLERS:

5 Q. Again, you are familiar with the concept of
6 these adapters that we are discussing, correct?

7 A. Totally. Doesn't even matter what the function
8 is. What I'm saying is the sequence is something quite
9 distinct from what we would find in the human genome and
10 therefore it would be my opinion is outside of our
11 discussion.

12 Q. Okay, well, I would ask, you does one of the
13 sequencing adapters, does it change from one subject to
14 another and is independent of the sequencing results?

15 MR. Luman: Incomplete hypothetical.

16 BY MS. SELLERS:

17 Q. Does an Illumina sequencing adapter change from
18 one subject to another?

19 A. That's -- and forgive me for saying this, a
20 rather ridiculous question.

21 Q. Can you answer it?

22 A. So I would need a little bit more information.

23 It may well be. There are all sorts of
24 different flavors of sequencing adapters. So an
25 individual, it might turn out their DNA has been

1 fragmented, an adapter has been attached to it, perhaps
2 even one of quite esoteric flavors.

3 And you might have another individual who, in
4 fact, has undergone cleavage of their DNA, and a
5 different adapter has attached to their DNA.

6 So what relevance does this have?

7 Q. Dr. Furneaux, your job here today is to answer
8 our questions and the question is --

9 A. What I'm saying is we need to know the context.

10 There will be some occasions where that adapter
11 would change from one individual; other instances where
12 it would not change. It's ambiguous.

13 Q. All right, and then so let's say you have a bar
14 code for use in demultiplexing. Is that a fixed or
15 variable portion?

16 MR. Luman: Incomplete hypothetical.

17 THE WITNESS: Again, I would direct you to the
18 same answer. There are many different flavors of bar
19 codes. Individual Mr. X might undergo covalent addition
20 of such a bar code to his DNA, and Mr. Jones might
21 undergo a different one.

22 So it would be very difficult to apply my test
23 because the test subject is not something that one
24 should be seriously considering.

25 ///

1 BY MS. SELLERS:

2 Q. So would you consider PCR primers designed for a
3 specific patient to be a fixed or variable portion?

4 A. I would have to look at the context again. And
5 I'm happy to comment on PCR primers that have been used
6 and we can take a little look at the information that
7 surrounds them to make that deliberation.

8 Because -- let me tell you in advance, if we
9 don't even know the sequence of the primer nor how it is
10 designed makes that task extraordinarily difficult.

11 Q. So let's talk a little bit more about Ley. So
12 Ley identified ten somatic mutations and two germline
13 mutations, correct?

14 A. Yeah, eight single nucleotides are two inserts
15 or deletions -- I can't recall, and certainly two
16 inherited mutations, yes.

17 Q. And then Ley used PCR and Roche plus 454
18 sequencing to sequence the primary tumor, the skin
19 sample and the relapsed tumor sample, correct?

20 A. You're quite correct. I mean, we could look at
21 it, that's always helpful. But Ley indeed performed a
22 PCR amplification with unknown primers, performed Sanger
23 sequencing on that, confirmed the nature of the
24 alterations, let's say, because ^ (inaudible) and then
25 took that material and performed 454 pyrophosphate.

1 Sorry it's a lab joke, pyrophosphate,
2 P-Y-R-O-phosphate. I had a post doc from the southern
3 states, who liked to say pyro.

4 Anyway so the 454 and, yes, again, no surprise,
5 confirmed that there were in fact two different -- at
6 least two different alleles.

7 Before we got on to that, I think it's worth
8 broaching the notion that I suspect this does not
9 conform to claim -- I'm working by memory here, is it
10 one B or one C, I'm not sure which patent we're in now.

11 I wonder if you could bring that up, one B or
12 one C in either patent. And we can take a little quick
13 look at it because it might forestall our whole
14 conversation.

15 Q. Let's focus the questions that are being asked
16 because right now there is no question opened.

17 So let's move on --

18 A. I just thought, you know, because I do respect
19 your style, Abby, I thought I'd just let you know I
20 don't think fig 3 in 454 fulfills at least two
21 limitations.

22 Q. Okay, let's talk a little bit more about Ley in
23 general, which is -- Ley -- you admit Ley said uses PCR
24 to generate amplicons which are then pyrosequenced,
25 correct?

1 A. First by Sanger, and second by pyrosequencing,
2 yeah.

3 Q. Okay. And Ley used these primers to create
4 amplicons of the exons with the mutant alleles, correct?

5 A. That's what we're given to understand. It would
6 be nice to have some actual information and data, but,
7 yes, that's the speculation here.

8 Q. All right. And Ley was interested in capturing
9 the amplicons with both the wild type and the -- both
10 the mutant alleles and the wild type alleles, correct?

11 A. I think one could summarize that. As a said,
12 none of this was a surprise. So it really boils down to
13 what the objective was.

14 And the objective in the experiment I think
15 we're discussing, to determine the percentage of tumor
16 cells versus normal cells. That was their objective.

17 Q. Correct and to do that, you would need to
18 generate amplicons and sequence amplicons that have both
19 the mutant allele and the wild type, correct?

20 A. You would need to use the information you
21 already know that there are different alleles and now
22 the job merely is to quantitate them.

23 Q. So given that Ley wanted to sequence both the
24 mutant alleles and the wild type alleles, Ley would have
25 used a primer that paired with the nucleotide sequence

1 upstream of the given mutation, correct?

2 A. One would hope so, yeah.

3 Q. Is there a way that Ley could have PCR'd these
4 mutant and wild type alleles without using a primer that
5 paired upstream?

6 A. You mean are there different ways of
7 establishing the percentage of each allele other than
8 sequencing? Or other than using primers at the end, I
9 think is what you're saying.

10 Q. Saying if we know Ley PCR'd, we know Ley
11 used PCR --

12 A. There are other PCR methods. I mean, the
13 classic one that springs to mind is you can devise a
14 oligo that may or may not anneal to the mutation that
15 you're anticipating.

16 But I do not think this is -- that's the case in
17 the present analysis.

18 Q. So given the information that we know from Ley,
19 Ley would have used a primer that would have paired
20 upstream of the desired mutation?

21 A. And one downstream.

22 Q. Correct.

23 A. I think that is a very -- that would be a very
24 reasonable supposition, that's what probably I would
25 expect. Be nice to see it, mind you.

1 Q. Okay, let's turn to --

2 A. Because as you well know, Abby, we all live for
3 surprises, right? They may have done that PCR reaction
4 and, goodness grief, the size of the product is nothing
5 like what they thought it was going to be.

6 Q. Let's turn to paragraph 201 and 202 of your
7 declaration. Do you see my screen here?

8 A. I do.

9 Q. So in these two paragraphs, you state, "the
10 mutation in the BRCA2 gene identified by Ley could have
11 changed the AAT codon to a CAT codon or a CAC codon,"
12 correct?

13 A. Yeah, you're down in 202, sorry -- so you're
14 talking about the amino acid histidine is coded either
15 by a CAT or a CAC.

16 Q. Correct.

17 A. All right. Yeah, so it's asparagine in the
18 reference database, right. And we're on BRCA2, yeah, I
19 think I'm up to speed with the context.

20 Q. Okay, so what you are stating is the codon
21 changes from AAT to either CAT and/or CAC, correct?

22 A. Yeah, and let me just say in the first instance
23 these are inherited mutations.

24 Q. Okay. Regardless -- so I wanted to clarify,
25 regardless of whether the mutation resulted in the CAT

1 or CAC codon, the middle base is always going to be at
2 adenine, correct, for this mutation?

3 A. Well, I'm just double checking. Did I describe
4 this as a mutation. Yes, this is a polymorphism, right?

5 Q. Um-hmm.

6 A. So one doesn't want to describe it as mutation.
7 It's a polymorphism this poor lady has inherited.

8 Q. Let's rephrase that. So regardless of which
9 polymorphism this patient had, the codon sequence is
10 going to be CAT or CAC?

11 A. That would be our conventional understanding.
12 We all live for surprises but I think that's our
13 expectation.

14 Q. And the middle base then of that codon is always
15 adenine, correct?

16 A. Yes, that would be correct.

17 Q. Are you doing good, Dr. Furneaux, or do you need
18 a break?

19 A. Oh, I'm fine. Sorry. I want to apologize, I'm
20 chewing on these butterscotch things that help my saliva
21 production -- so don't be distressed by my weird sounds
22 of me eating the butterscotch.

23 MS. SELLERS: No, that's okay. We've just been
24 going an hour and I wanted to check and make sure --

25 THE REPORTER: I could use a break.

1 THE WITNESS: It's been fun, I'm enjoying this.

2 MS. SELLERS: Let's take this off the record.

3 MR. Luman: Off the record at 11:39.

4

5 (Recess taken.)

6

7 MR. Luman: Back on the record at 11:52.

8 BY MS. SELLERS:

9 Q. So Dr. Furneaux, in 2016, it was known that one
10 benefit of determining the genetic mutations of a given
11 patient's tumor is that it can potentially help guide
12 therapies selected for that patient, correct?

13 A. By 2016, I think that was well established. One
14 could say that the benefit for the patient may be have
15 been exaggerated.

16 I was certainly involved in the very early
17 applications of the mutations in EFJR that led to
18 specific therapeutic agents way back in either -- 2001,
19 2002, somewhere there.

20 So as in all things, you know, there's no
21 guarantee of any benefit, very much depends on the
22 context.

23 Q. So then you would agree that there are mutations
24 that counsel against certain therapies?

25 A. Against therapies? No, I think that's a little

1 bit more of a difficult area. You know, it's somewhat
2 easier in science to prove that something is than it
3 ain't, right.

4 So I would sort of counsel you there that one
5 may make an observation that may suggest that X may not
6 be a good idea. But one is unaware of the fact of the
7 mutation in a different gene over here that in fact is
8 good in that sense, good in commas.

9 And so I think to say something ain't useful,
10 you certainly need more than one mutant allele would be
11 my guess but interesting.

12 Q. There were theories in the field in 2016 that
13 certain mutations counsel against certain therapies,
14 correct?

15 A. Well, that's a very general statement. I'm sure
16 there were. One would have to look at the evidence
17 surrounding such theories.

18 Q. Do you know if these theories were in practice
19 in 2016?

20 A. That's a difficult question for answer. I don't
21 know what therapy you're talking about or theory.

22 Q. Do you know if this principle was in practice,
23 the idea that certain genetic mutations counsel against
24 certain therapies?

25 A. In general -- in medicine in general I'm aware

1 of fairly well established phenomena where
2 African-Americans with a particular variant, if I
3 recall, are very sensitive to blood pressure
4 medications, am I correct? Yeah, working by memory but
5 that was pretty well established, yeah.

6 Q. But for treating cancer, was it an established
7 practice -- strike that.

8 In which 2016, was it an established practice to
9 refrain from prescribing certain treatments for certain
10 types of cancers that have particular genetic mutations?

11 A. Well, you know, I would quibble over what
12 established practice means. Were there ideas that there
13 were mutations that may suggest against X and had that
14 actually been proven by experiment, I'm open to
15 reviewing that data.

16 Just as a matter of interest in terms of the
17 declaration from Dr. Q that I reviewed, there was scant
18 evidence to suggest that such an alteration may
19 adversely or suggest that one should not carryout a
20 certain therapy, extraordinarily absent and I guess most
21 generously weak evidence.

22 Q. Do you agree that in 2016, one of the
23 overarching goals of genomic cancer research was to
24 learn how to better treat cancer?

25 A. To learn how to better treat cancer has been in

1 demand throughout my entire professional career.

2 Q. Cytarabine is a chemotherapy drug, correct?

3 A. You are correct.

4 Q. And is it used to treat acute myeloid leukemia?

5 A. Can be indeed. You're better asking a
6 clinician, of course, but, yes, that's my understanding.

7 Q. Are you familiar with theories that certain
8 mutations may cause Cytarabine resistance?

9 A. We both know why you're laughing, Abby. But,
10 yes, let me just cut to the chase. I did actually read
11 with great interest Dr. Q's statements and some of the
12 stuff in the supplementary of Ley.

13 And let me see if I remember this correctly
14 without looking in my deposition. I'm not impugning Dr.
15 Q, I'm sure he's a fine person.

16 He appears to get confused between genes there.
17 There was a story and I can only say a story on one gene
18 suffering a mutation and there was some evidence that I
19 actually tracked down and looked at and turned out to be
20 very poor evidence that that gene would potentially and
21 only potentially inform a physician not to use the drug
22 you mentioned.

23 Now sadly, Dr. Q talked about gene A and
24 actually the data was related to gene B. They both had
25 the same initial Social Security number except they were

1 different genes.

2 That's not quite correct. They wouldn't have
3 the same Social Security number. They both had the same
4 initial -- they were members of a multigene family and,
5 of course, sometimes if one's a theoretician makes an
6 assumption that's all members of the family act the same
7 way. That's not usually the case.

8 Q. Do you recall Ley suggesting that its whole
9 genome sequencing technique should be applied to other
10 patients with AML tumors?

11 A. You would have to direct me to what was said in
12 the paper.

13 Q. All right. Let's go to Ley, this has already
14 been provided as Exhibit 1012. And I will pull it up
15 for you, Dr. Furneaux, but we're going to page 71.

16 Can you see my screen near?

17 A. Good, great you blew it up a bit.

18 Q. So it says (as read):
19

20 Furthermore until hundreds or
21 perhaps thousands of normal genomes and
22 other AML tumors are sequenced, the
23 contextual relevance of the mutations
24 found in this genome will be unknown.
25

1 A. I think that's probably what I just said in our
2 last conversation.

3 Q. So do you agree then that Ley is suggesting that
4 we wouldn't need to sequence additional AML genomes?

5 A. I think particularly with respect to the
6 phenomenon that we were talking about of -- that it
7 would negatively --

8 Sorry, that the observation would suggest that
9 one should not carry out a particular therapy, I think
10 that most certainly would need many more analyses other
11 than eight mutations. Ten to be fair.

12 Q. But as a general statement, do you agree that
13 Ley's suggesting that additional patients AML genomes
14 need to be sequenced?

15 It's a yes or no question.

16 A. I wouldn't interpret it that way. I would say
17 what they're basically saying is, hey, we haven't done
18 enough to make a clear conclusion that would be of
19 interest to a clinician. We should go sequence more
20 tumors, is how I would interpret that statement.

21 Q. All right, so let's take a look at the Newman
22 reference here. So Newman discloses a technique known
23 as CAPP sequencing, correct, or CAPP-Seq?

24 A. Really help if you brought up the paper.

25 There's been so many papers in these cases. If

1 you could just bring up the paper that would be better.

2 MS. SELLERS: Introducing previously stamped
3 Exhibit 1043.

4 (Exhibit 1043 was marked for identification by
5 the court reporter and is attached hereto.)

6 BY MS. SELLERS:

7 Q. Do you see that?

8 A. Yes, I do see the paper.

9 Q. And so to repeat my question, Newman discloses a
10 technique known as CAPP sequencing or CAPP-Seq; is that
11 correct?

12 A. That would be correct, yeah, that's what the
13 abstract says.

14 Q. Before applying the CAPP sequencing technique,
15 Newman discloses performing initial sequencing either on
16 a tissue sample or a cell-free DNA, correct?

17 A. Let's see, where are we here. You'd have to
18 show me in the paper where they describe that.

19 Q. All right. Here we go, let's look right here.

20 A. Yeah, I mean the actual data though.

21 Q. Let's look at this paragraph right here. So it
22 states (as read):

23

24 That CAPP Seq combines hybrid
25 infinity capture of hundreds of genomic

1 regions with deep sequencing and a
2 specialized bioinformatics workflow.

3 With a priori knowledge of tumor
4 genotypes from sequencing of tumor
5 biopsies, this method achieved a
6 detection limit of 0.02 percent in
7 cfDNA.

8
9 So is that correct? Did I read that --

10 A. Yes, that is quite correct.

11 Q. So what they are stating here is that prior to
12 performing the CAPP sequencing technique on cfDNA they
13 did sequencing on a tumor biopsy, correct?

14 A. Yeah, they had been selected by affinity
15 capture.

16 Q. Perfect. So then this CAPP sequencing technique
17 can be used with genetic sequencing information derived
18 from a tumor biopsy sample, correct?

19 A. It certainly could, that's what they're saying
20 they're going to do.

21 Q. Okay, you mentioned earlier that Newman uses --
22 use the exact term that you had -- did you say capture
23 probes --

24 A. Did I say that, where did I say that?

25 Q. I'm trying to see -- I know you used a specific

1 term here and I want to see if it's in the rough
2 transcript.

3 A. You mean what I just said, I think I said
4 affinity capture.

5 Q. Affinity capture, there we go. But --

6 A. Reading from their paper.

7 Q. Okay. So Newman's CAPP sequencing unique uses
8 selector/capture probes to sequence the specific
9 targeted mutations, correct?

10 A. Well, again, I'm looking at just at this
11 paragraph. But what they've done, I would figure, is by
12 hybrid affinity, I'm assuming they've used RNA sequences
13 to select for particular regions they think are of
14 interest, and subjected them to deep sequencing and
15 whatever specialized bioinformatics work flow maybe.

16 I recall this paper -- yeah, I have to say after
17 the first paragraph of the results, I became very
18 skeptical of these authors. And I have to say I didn't
19 perhaps pursue this in significant depth.

20 Q. Let's talk a little bit about capture probe/
21 selector design. When a person of ordinary skill in the
22 art is designing a set of capture probes to detect
23 mutations in tumor DNA, you first want to consider what
24 regions of the DNA you want to capture, correct?

25 A. I think that's one thought that one might have.

1 Sounds reasonable.

2 Q. And then increasing the number of regions of DNA
3 that are targeted for capture could potentially increase
4 the number of locations where mutations could be
5 detected, correct?

6 A. Well, you know, again, that's a very general
7 statement. You know, I'm sort of reminded by the old
8 thing about where does a drunk look for his lost keys,
9 that's underneath the porch light.

10 So you know, (inaudible) thinking we should look
11 here. But as a matter of fact, one should be looking
12 over there.

13 Q. But potentially adding additional areas that are
14 targeted for capture could allow the assay to detect
15 additional mutations. Is that --

16 A. It could and it could not, you know. Obviously
17 if one's a theoretician, one assumes there's going to be
18 even a linear correlation between such a number and
19 one's success. But you've got to do the experiment.

20 Q. All right. So increasing the number of
21 mutations that can be detected in a given assay, can
22 have clinical benefits, correct?

23 A. I think I have to repeat what I said: It could
24 and it could not. You've got to do the experiment. And
25 there's loads of variables on what you said there, I

1 don't know what the clinical outcome is, I don't know
2 what we're measuring here, all sorts of possibilities,
3 maybe, maybe not.

4 Q. All right, so if you want to increase the number
5 of regions targeted for capture using capture probes, in
6 general you would have to increase the number of capture
7 probes used, correct?

8 MR. Luman: Incomplete hypothetical.

9 THE WITNESS: Well, yeah, I mean, you know,
10 there's a general theme here where you're asking very
11 general questions of which there may be several
12 different strategies.

13 One could increase the number of segments that
14 one hybridizes to (inaudible) or one could elect to
15 catch a larger portion.

16 I think it really depends upon -- to a large
17 extent, this will start depending on if you have some
18 knowledge about the nature of the tumor and some
19 knowledge about the existing known variants.

20 All this stuff has to be put together before one
21 could think about strategic planning.

22 BY MS. SELLERS:

23 Q. And so increasing the number of capture probes
24 in a single assay could potentially reduce the accuracy
25 of the assay, correct?

1 A. Well, again, we're talking in very general
2 terms. To give you an opinion, I would have to look at
3 an experiment someone has done towards this end and
4 review the data.

5 Q. Is it possible that increasing the number of
6 capture probes in assay could --

7 A. I think as you well know, Abby, in biochemistry
8 anything is possible. But the real thing is what did
9 happen.

10 Q. Is it fair to say when designing a set of
11 capture probes for an assay that's directed towards
12 detecting a specific mutation or specific mutations in a
13 tissue sample, that a person of ordinary skill in the
14 art would consider that there are benefits of increasing
15 the number of capture probes, and then there are also
16 potential disadvantages of increasing the number of
17 capture probes?

18 MR. Luman: Objection, form.

19 THE WITNESS: Well, I think that's in essence
20 you said what I've just been saying. There could
21 benefits, there could be disadvantages. Everything's
22 going to depend on the context.

23 BY MS. SELLERS:

24 Q. All right. So let's take a look really quickly
25 at a passage from Newman.

1 A. Back to Newman.

2 Q. Back to Newman. All right. Can you see that?

3 A. Yes. This is what they're calling their
4 selector thing.

5 Q. If you could just go ahead and read this
6 paragraph to yourself and let me know when you're done.

7 A. Sorry, when you said that before I thought I was
8 supposed to read aloud.

9 Q. I think I did ask you to read aloud.

10 A. Oh, you did, okay.

11 Well, yes, I've read that sentence.

12 Q. Can you read actually the entire paragraph.

13 A. Sure. Well I've read the paragraph.

14 Let me just say as an aside that my -- this
15 further confirms my feelings that these investigators
16 aren't entirely clear about what they're doing.

17 Q. Okay. So let me ask you some specific questions
18 about the selector designs that are disclosed. They
19 disclose three different selector designs in this
20 paragraph, correct?

21 A. Let's see. I do apologize, when I figure people
22 don't know what they're talking about, I tend not to pay
23 much attention to the paragraph. Okay, there's the
24 first one, there's a final one. There's a final one of
25 203, then a bigger one of 300, is that what you're

1 referring to?

2 Q. Yes, so they have 175, a 203, and a 300 kilobase
3 selector design, correct?

4 A. That would be correct. All presumably for
5 utility in non-small-cell lung cancer.

6 Q. So this first selector design they discuss, the
7 175 kilobase, they state that one has the highest
8 sensitivity, correct?

9 A. Can you tell me where --

10 Q. The part --

11 A. The very first one, okay. Do they show that?

12 Q. Let me rephrase the question.

13 The 175 kilobase selector, which is the smallest
14 selector that they disclose, was designed to have the
15 highest sensitivity, correct?

16 A. They say it was developed to maximize analytical
17 sensitivity. I'm not quite sure what analytical
18 sensitivity is, they are not saying what they're
19 detecting here and I'd like to see the data that
20 supports that assertion.

21 Q. Is it fair to say that they're asserting that
22 the smallest selector design has the highest
23 sensitivity?

24 A. They are telling themselves that they've
25 developed something that has maximal analytical

1 sensitivity.

2 Q. And then so would higher sensitivity be an
3 advantage to a certain selector design?

4 A. I can't opine. I have no idea what they mean by
5 "analytical sensitivity."

6 Q. What does sensitivity typically mean in --

7 A. Sensitivity typically means the ability to
8 detect something.

9 Q. Okay, so can we assume that this means the
10 ability to detect something, analytical sensitivity.

11 A. We don't know what that "something" is, though.

12 Q. So it's designed to detect genetic mutations in
13 the non-small-cell lung cancer, correct?

14 A. The sensitivity could be referring to the
15 ability to detect tumor.

16 Q. Okay. But either way, what they're suggesting
17 is that no matter what the sensitivity is directed
18 towards, the smaller panel is the one that has the
19 highest sensitivity, correct?

20 A. Are they saying that, are they actually saying
21 that's a necessity or are they just remarking, we design
22 this guy and we think it's got a high level sensitivity
23 but it may be unrelated to the size of the panel.

24 Q. Well, what I'm asking you is, is what they're
25 saying is we have three panels, the smallest one is 175

1 kilobases and that one was designed at the maximum
2 sensitivity?

3 A. Well, yeah, what I'm saying to you is, are we
4 talking about a correlation or a causal association
5 here?

6 Q. Well, we're talking about correlation.

7 A. Okay, okay, so its meaningless.

8 Q. What I'm asking you and this is a yes or no
9 question, are they saying that their maximum sensitivity
10 assay is the 175 kilobase panel?

11 A. Yeah, the point I'm bringing up here is, for
12 example, the 175KB panel, for that to exert maximal
13 analytical sensitivity, is it necessary and sufficient
14 for it to be 175. And is it necessary and sufficient
15 for it to contain eight or is that merely a coincidence?

16 Q. So in general, sensitivity can -- the size of
17 selector panel can vary and sensitivity can vary. Is
18 that what you're trying to say, is that it's not
19 necessarily a linear relationship?

20 A. Well, I would be astonished if it was a linear
21 relationship. But I'm saying I have no evidence here to
22 base my opinion upon.

23 All I see here is a statement and these factors
24 may be inexorably connected, if they are necessary and
25 sufficient for such maximal analytical sensitivity, but

1 on the other hand, they may well not be. There's no way
2 of knowing.

3 Q. Okay, let's move on from this one. So the
4 paragraph goes on to state that they may increase the
5 size of the panel to accommodate for additional
6 mutations, correct?

7 A. So let me just see. Again, I apologize, I tend
8 to switch off as soon as I figure that I'm not being
9 given enough detail.

10 Where is that sentence exactly.

11 Q. They say to improve --

12 A. Oh, yes, "to improve coverage for nonsmokers
13 whose tumors generally have lower mutation burdens," I
14 wonder where they heard that?

15 Maybe they heard that in the deli on the way to
16 the lab. Who knows.

17 This is diminishing their credibility immensely,
18 even more than their rather tragic experiment they
19 presented in the first paragraph.

20 However, sorry, do you have a question?

21 Q. Yes. So the paragraph goes on and it states
22 that they designed an increased sized panel and they
23 used that to target additional mutations, correct? So
24 they increased the size of the panel to increase the
25 number of mutations, correct?

1 A. They decided to include their ability to detect
2 mutations in ALK, ROS1, RET. So what I'm reading here
3 is they decided for whatever reason, their new super
4 duper selector, mark 2, to put in an ability to detect
5 presumably fusions in ALK, ROS1, and RET.

6 Not actually saying what those fusions are,
7 though. Would be nice to know.

8 Q. And then they increase the size of the panel
9 again to include complete exon coverage; is that
10 correct?

11 A. Yeah, that would be the super, super duper,
12 the --

13 Q. All right. So this paragraph, would you say
14 this is an example of what we were discussing prior,
15 which is that there are advantages and disadvantages to
16 increasing selector size and decreasing selector size?

17 A. Yeah, I think that we were still talking about
18 very general things. Presumably what I'd be looking for
19 here that would make me believe these statements are
20 worthwhile entertaining, would be some evidence that
21 the -- these -- the sensitivity, which is presumably
22 their incident, changes in some way.

23 Q. All right. So let's go ahead and take a look at
24 another reference you cited in your declaration.

25 I don't think which is one we sent but I will

1 pull it up on here. And it is a paper authored by
2 Dudley. So let's pull that up here.

3 A. Dudley? Yeah, if you could pull it and up that
4 would ring a bell probably. The first authors does not
5 ring a bell.

6 MS. SELLERS: So introducing previously marked
7 Exhibit 2166. Then I will share my screen with this,
8 Dr. Furneaux.

9 THE WITNESS: Thank you. I have it here also
10 in -- oh, I've got a different one. Oh, I got it.

11 (Exhibit 2166 was marked for identification by
12 the court reporter and is attached hereto.)

13 BY MS. SELLERS:

14 Q. All right. So do you recall Dudley disclosing
15 potential advantages and disadvantages to using hybrid
16 capture probes?

17 A. Yeah, if you could direct me in my declaration
18 where I talk about this because I don't want to say
19 that -- anything without referring to that.

20 Q. Yeah, so this one I'm not asking specifically
21 about what you said in your declaration. I'm just
22 asking about what Dudley disclosed in general.

23 If you want to take a look at it, it's these two
24 paragraphs here starting with "an additional"?

25 A. I should read the hybrid capture starting

1 paragraph, oh, starting at "an additional"?

2 Q. Um-hmm, and you can read it down.

3 A. Yeah, I mean I gave it a quick read. I think
4 I'm okay depending on the question, of course.

5 Q. My question is just would you agree that Dudley
6 discloses that there are advantages and disadvantages to
7 the use of hybrid capture probes?

8 A. Well, you know, I hesitate before saying this
9 but this paragraph reveals that Dudley isn't perhaps the
10 most reliable source in some senses.

11 Q. Well, reading Dudley, it states, would you agree
12 that are both advantages and disadvantages to the use of
13 capture probes?

14 A. Again, in a general conversation, of course,
15 there are advantages and disadvantages of anything.

16 And the critique I would have here is certainly
17 one challenge and he's quite correct, he or she, I don't
18 know, one challenge with the hybrid capture approach is
19 that artifactual mutations may arise.

20 And in fact this group ascribes such artifactual
21 mutations to having to incubate the DNA at high
22 temperature, which, of course, can encourage chemical
23 mutagenesis, it's a little bit ironic, right.

24 Sadly the experiments that they came up with to
25 address this particular phenomena were so poorly

1 executed in the paper we just looked at that one does
2 have reservations about the general credibility of this
3 group.

4 Although, of course, the statements they make
5 one has to evaluate in a legal sense.

6 Q. All right. So let's talk a little bit about
7 genetic mutations in breast cancer. We touched on this
8 a little.

9 But were there certain exons in 2016 in the P53
10 gene that were known to have poly (inaudible) mutations
11 in breast cancer?

12 A. Yes, as I indicated before, we all have Google
13 brains now. We used to carry this information at hand.

14 I would be very confident that that what's
15 called the P53 hotspot was known well before 2016. And
16 I'm actually thinking to myself, myself and Uve Schlegel
17 when we were among the first to identify P53 mutations
18 in DNA from paraffin-embedded sections, which is back in
19 '83-'84, somewhere about there, I would suspect we were
20 probably looking at mutations from the alleged hotspot
21 even back then.

22 Q. Can you explain a little bit what is the P53
23 hotspot?

24 A. Well, of course, the hotspot may well change as
25 the years go on.

1 But it's like everything else, someone finds a
2 mutation. And then someone finds a mutation pretty much
3 next door and they go, oh, this is where we should look.

4 Again, this is the drunk looking for their keys
5 under the lights. And so one always has to be a little
6 careful, of course, it may well be it's called a hotspot
7 because everyone looks there and that's the only place
8 they look.

9 It wouldn't be the first time in science that
10 that's occurred. But I think there's a general
11 consensus -- I'm thinking of Carol Prives' work and
12 Vogelstein's work -- I'm very confident there's a
13 website somewhere that identifies every single P53
14 mutation since the beginning of time and where they are.

15 I don't have that at hand.

16 Q. Yes, don't mean to put you on the spot here --

17 A. -- Exon 4 to 5 spring to mind as I'm sitting
18 here.

19 Q. So if a person of ordinary skill in the art was
20 trying to look for commonly mutated genes in the P55
21 region, it would be safe to say that they may look at
22 the hotspot, correct?

23 A. Yeah, I mean, while we're on that subject, could
24 you remind me what we're thinking a person of ordinary
25 skill in the art might be.

1 Q. Yeah, I think you, let's see, you included it in
2 your declaration.

3 A. Oh, I acknowledge that's what the board and the
4 powers that be came up with. I'm not sure I agreed with
5 it. I think I said I would work with that definition.

6 Q. Correct, so that is the definition we are
7 working under today.

8 A. Just to put it on record, I've never met such a
9 person apart from myself, a few of those, but they're
10 very, very rare.

11 Q. There's your definition.

12 A. So I guess to expand on what I'm saying is, this
13 looks like a remarkably gifted individual.

14 Q. Okay, well --

15 A. Is that -- my simple question is, is that who
16 we're talking about when you ask me that question?

17 Q. Correct. So I'll ask that again, which is, if a
18 person of ordinary skill in the art was trying to
19 identify mutations in a given patient in the P53 gene,
20 would they --

21 A. In what year?

22 Q. In 2016.

23 A. Okay. I think I just indicated we did that back
24 in '83 or '82, '84. So this legendary person in 2016
25 has been challenged with the objective to find P53

1 mutations in a sample from a breast cancer patient?

2 Q. So we're saying this person of ordinary skill in
3 the art knows a person with breast cancer, they're
4 trying to determine what mutations they have, would they
5 look to the hotspot in particular for potential
6 mutations?

7 A. Ah. They might, right, and they might also say
8 to themselves, gee, everyone's looked here. I think it
9 would be really smart to look here and also elsewhere.

10 But 2016, I think that would be quite advisable.
11 And knowing that, do we move beyond the coding regions
12 and say, oh, there might be mutations in the promoter,
13 there might be mutations in various regulatory elements
14 in DNA.

15 There might be some kind of association that we
16 can't see. In other words, there might be a silent,
17 what's been called a silent mutation.

18 Doesn't change the amino acid structure but
19 changes the sequence of the RNA, and thus changes its
20 regulation.

21 By 2016, I'd be hopeful that this incredibly
22 rare individual with such incredible talent would
23 certainly be thinking of all of these possibilities.

24 Q. And if a person of ordinary skill in the art
25 wanted to target the hotspot for sequencing, could they

1 use capture probes that would just target exons 4 and 5
2 and sequence that specific region of the genome?

3 A. Yeah, we're always talking about what one
4 individual may or may not do. Following from what I
5 just said, it would probably be more prudent to -- given
6 how well examined this gene has been in the past, it
7 would be very prudent to extend the analysis.

8 And perhaps, in fact, instead of targeting just
9 four or five, target the whole gene. And, of course, by
10 the whole gene, we're now into the hinterland of
11 regulatory sequences upstream, regulatory sequences
12 downstream, hither to unknown RNAs that are coming from
13 this particular region.

14 I would have thought a person of ordinary skill
15 in the art such as we have here, that's exactly what
16 they would be thinking of.

17 Q. Okay. So if you're a clinician or someone who's
18 trying to use this for diagnostic purposes and are
19 looking to discover some of -- whether it's well-known
20 mutations are present in a specific patient, could you
21 use capture probes to just sequence the hotspot in exons
22 four and five to detect mutations there?

23 A. Are you asking me what the best strategy might
24 be for that objective?

25 Q. I'm asking you if you could use that technique?

1 A. Anything is always possible. The question is
2 what would be the most reasonable approach.

3 For the experiment you're suggesting -- so in
4 other words, this is an individual who has been charged
5 with finding particular mutations, not looking to see
6 the possibilities that may exist, which is probably a
7 prudent thing to do in 2016, but to look for specific
8 ones.

9 The advice I would such an individual -- they
10 should know themselves -- probably the best thing to do
11 would be to do a PCR across the region of interest.

12 Q. Well, let's take a look at an additional
13 reference. Do you remember the Forsheew reference --

14 A. Yeah, absolutely.

15 Q. Okay, so let's look at that.

16 A. That's the Cambridge group, right, Carlos
17 Cardone -- no, that's a different guy.

18 MS. SELLERS: Introducing previously marked
19 Exhibit 1030.

20 (Exhibit 1030 was marked for identification by
21 the court reporter and is attached hereto.)

22 BY MS. SELLERS:

23 Q. I'll pull it up on the screen share for you, Dr.
24 Furneaux.

25 A. Oh, I love these papers. Yeah.

1 Q. So that is in front of. Do you recall Forshew
2 disclosing identifying deep sequencing technology?

3 A. Yes, I recall what he did was blitzkrieg the
4 region between five and six, which happened to be
5 actually two of the smallest exons in P53 with a tiled
6 array of PCR products.

7 Q. Do you recall Ley also disclosing the
8 application of its tag amplicon sequencing to a breast
9 cancer patient?

10 A. Ley?

11 Q. Sorry, Forshew.

12 A. Forshew, yeah? You mean the -- let's call her
13 Mrs. Y. If I recall somewhere at the end of the paper,
14 Mrs. Y, there was a little bit of very, very minimal
15 description of that.

16 Let me test my memory. They established ten
17 possible mutations of interest.

18 Q. Correct. All right.

19 And so if a person of ordinary skill in the art
20 wanted to expand on the tag amplicon sequencing assay
21 that Forshew disclosed here to look at mutations in the
22 P53 gene that are related to breast cancer, would they
23 be motivated to look to this hotspot that we've been
24 discussing?

25 A. That's a very general question. I think

1 the significant feature here is the motivation. Why
2 would an individual in the first place be motivated to
3 look at P53 in this context?

4 Q. P53 is a very common site of mutations, correct?

5 A. Um-hmm.

6 Q. And you discuss that P53 is a known cancer
7 driver gene location, correct?

8 MR. Luman: Mischaracterizes his testimony.

9 THE WITNESS: Can you say that again so I can
10 check?

11 BY MS. SELLERS:

12 Q. So you testified that the P53 -- the mutations
13 in the P53 region are known to be some cancer driving
14 mutations, correct?

15 A. Some more than others. Some mutations are
16 clearly driving, some not so much. Some not at all
17 actually.

18 Q. Okay. So if a person of ordinary skill in the
19 art did desire to look to see if any of these cancer
20 driving mutations were in this particular patient would
21 they be motivated look to the hotspot region?

22 A. I think -- I'm reading between the lines here,
23 what you're actually asking me is would a person of
24 skill in the art who's embarked on this particular
25 endeavor, then feel the he should indulge Dr. Q in his

1 assertion that one would gladly and willingly now
2 incorporate analysis of P53.

3 I think that's what you're asking me.

4 Q. I'm actually asking you the question that's on
5 the record, and I appreciate if you stuck to that.

6 So I'll ask it again: If a person of ordinary
7 skill in the art did look to the P53 gene and was
8 looking for potentially known cancer driver mutations,
9 would they be look to the hotspot region in particular?

10 A. Well, yes, but rewind a little bit. What I'm
11 questioning is why would a person of skill in the art --
12 let's imagine who's already done this whole genome
13 sequencing, has already identified these ten mutations
14 and is clearly in this experiment interested in the
15 objective of whether they may help track the patient's
16 progress because that's what we see there, albeit
17 carried out rather imprecisely, it would be astonishing
18 to me that that person of skill in the art turned and
19 said, oh, why don't we do this well, right, wouldn't
20 make any sense to me at all.

21 Q. Dr. Furneaux, I do ask that you please only
22 answer the question that is being asked. And if you
23 need clarification, you can ask for it but beyond
24 that --

25 A. Well, what I'm saying here is when we're

1 discussing motivation, we can't assume in the first
2 place that this person of skill in the art would have
3 the motivation to look at P53 in the first place.

4 That would be making an assumption.

5 MS. SELLERS: Move to strike as nonresponsive.

6 MR. Luman: That answer is quite responsive.

7 You've within asking about what the motivation was.

8 He's addressing that.

9 BY MS. SELLERS:

10 Q. The last question on the record was would they
11 look to the P53 region. It did not discuss motivation.

12 But we can leave that on the record. But I do
13 ask, Dr. Furneaux, that you please focus your answers on
14 the questions being asked?

15 A. I apologize, it is my habit, as you know, as I
16 indicated, my habit to find fatal flaws and communicate
17 those so we don't have to tarry long with irrelevant
18 conversation.

19 Q. Okay, so Dr. Furneaux, setting aside Forshew, if
20 you are sequencing the P53 gene and you want to identify
21 whether specific mutations are present, looking at fewer
22 regions could potentially allow your assay to have
23 better sensitivity, correct, for detecting those
24 mutations?

25 A. Yes, and, again, let me just emphasize that in

1 science one always has to consider if things work, which
2 many theoreticians always assume everything works.

3 And you always have to consider things don't
4 work. And certainly you wouldn't want to be a person
5 skilled in the art who is charged to find P53 mutations.

6 You come back to the lab and say oh, I didn't
7 find any. And then somebody can say, well, where did
8 you look.

9 I looked here. And they go oh, that's silly,
10 why didn't you look everywhere because, gee, it's 2016,
11 patient could be anywhere, what are you doing?

12 Q. Okay, well, in addition, looking at a few select
13 mutations could also allow your assay to have greater
14 accuracy, correct, rather than a larger pool of
15 mutations?

16 MR. Luman: Objection, incomplete hypothetical.

17 THE WITNESS: I think we're still confused about
18 the objective. I thought you said the objective was to
19 find mutations in P53.

20 BY MS. SELLERS:

21 Q. So we're trying to identify whether a particular
22 patient has known mutations. Whether a particular
23 patient has known mutations?

24 A. Okay, so in that exercise, I guess, you would
25 say, I wonder if they have mutation of amino acid 321.

1 Oh, yes or no. Oh, no, let's go look at in
2 mutation from amino acid 225. Yay or nay. Oh, no.

3 See what I'm getting at. Seems to be very
4 fruitless exercise.

5 Q. But what I am asking is an assay that targets
6 fewer mutations can potentially have a greater accuracy
7 than an assay that targets a larger number of mutations;
8 is that correct?

9 A. That's -- you know, I'm afraid to say that's a
10 overly simplistic generalization. There may be well
11 mutations that are resident in areas that are difficult
12 to sequence.

13 And so focusing on those actually is probably
14 not a good idea.

15 Q. So let's talk a little bit about Forsheo again.
16 Forsheo suggests that the results of its tag
17 amplicon sequence technique could be used to inform a
18 choice of therapy; is that correct?

19 A. Yes, that's what he says, yes.

20 Q. Okay. That is good. Then let's turn to page 7
21 here, and I wanted to direct you to this figure --

22 A. Can we blow that figure up a wee bit?

23 Q. Before that, I want to look at your declaration
24 actually.

25 A. Sure, absolutely. So do I.

1 Q. So let's look here at your declaration,
2 paragraph 146.

3 A. 146, thank you. Yes, okay.

4 Q. So you opined here in your declaration that it
5 would be indication of PD, or progressive disease state,
6 that motivated the commencement of the paclitaxel
7 treatment in the patient; is that correct?

8 A. Yeah, is it possible to see the figure at the
9 same time in a double panel? If you take the figure
10 from the paper and put it on the right-hand side, then
11 we're good.

12 Q. Okay, see that?

13 A. Could we blow up a wee bit. Perfect.

14 Q. All right. So let's look at figure 4D then in a
15 little more detail. So on the right here, this legend
16 shows the specific mutations that were being tracked in
17 the breast cancer patient; is that correct?

18 A. Yup, I think that's true.

19 Q. Could we call these the tracked mutations?

20 A. I think that works for me.

21 Q. So around day 275, do you agree that the allele
22 fraction of each tracked mutation was measured?

23 A. It appears to be, yes, and some are going down
24 and some are going up.

25 Q. And around day 425 was the next date that the

1 allele fractions were measured; is that correct?

2 A. After PD, correct.

3 Q. Yes, so this measurement at that time day 45 was
4 taken just after the CT scan that showed a progressive
5 disease state, correct?

6 A. Yeah, so is to make it clear -- it's my
7 understanding from reading this that the scan was done
8 before the assay was reported.

9 Q. Is progressive disease state equivalent to a
10 relapse?

11 A. You would have to ask a clinician. My
12 interpretation of this is that a scan has been done that
13 has major concerns. And prompts a different
14 treatment --

15 (Reporter clarification.)

16 -- and prompts either a different treatment or
17 continued treatment. But basically it indicates
18 treatment is advisable.

19 Q. So you would say that progressive disease state
20 is equated to a worsening disease state in a way?

21 A. What I think it equates to is the clinician says
22 things are not good, we've got to do something either
23 extend what we did before or do something new.

24 Q. And so the allele fraction of each tracked
25 mutation increased from day 275 to day 425; is that

1 correct?

2 A. Day 275 to day 425 of each one, I think I would
3 agree with that, yes.

4 Q. So a person looking at figure 4D, would they
5 recognize that Forsheew shows a correlation between an
6 increase in allele fraction of the tracked mutations and
7 a worsening of the patient's disease state?

8 A. A person would appreciate that there is a
9 correlation here but not necessarily a causation.

10 Q. All right. So let's actually pull up the '783
11 patent, which is Exhibit 101 from the 545 IPR.

12 I think that was the incorrect exhibit that was
13 shared via the share site so I will -- or the Exhibit
14 Share so I'm going to re-share that one.

15 For the record previously marked Exhibit 1001
16 and this is from 545 IPR. And I want to look at
17 claimant one, Dr. Furneaux, and I will pull it up for
18 you as well.

19 Do you see my screen, Dr. Furneaux?

20 A. I do.

21 Q. So I want to look at limitation D, if you want
22 to just read that to yourself.

23 A. Yeah, I've read it.

24 Q. Does this claim require that the genetic variant
25 are the only factor that informs the choice or change in

1 therapies?

2 MR. Luman: Objection, calls for a legal
3 conclusion.

4 THE WITNESS: Yeah, I would again stress I'm
5 very conscious of the fact that I'm not a legal expert
6 and it sounds to me like what I say here could be
7 conceived in a legal forma that I am not intending.

8 BY MS. SELLERS:

9 Q. Well, that is the question on the table. So can
10 you to the best of your ability answer?

11 A. No, what I'm saying is that's a legal question,
12 and I'm not skilled in answering legal questions.

13 Q. Well, the objection is an objection that you
14 still have is to answer over. So if you have an opinion
15 on this --

16 A. The opinion I have is that you're asking me a
17 question that I cannot readily see can be answered by an
18 experiment. And thus I place it in the category of a
19 legal question.

20 Q. You can read the claim. Does the claim say that
21 only the genetic variance needs to inform the choice of
22 therapy?

23 A. I will repeat my answer. My sense is that this
24 is leading to a possible legal interpretation that I do
25 not intend.

1 Q. I appreciate that. But I'm asking you if
2 wording in the claim, does it say only --

3 A. You're asking me. And I'm electing not to
4 respond to since this is out with my expertise.

5 Q. Can you point to any portion of the
6 specification of these patents that explains how to use
7 the result from the sequencing assay to help inform a
8 choice of therapy or change in therapy?

9 A. Sorry, what was the question.

10 Q. Can you point me to any portion of the patent
11 that explains how to use the results from the assay
12 that's claimed?

13 A. Well --

14 Q. -- help inform a choice of therapy or a change
15 in therapy?

16 A. Yeah, I don't think we need to be a legal expert
17 or a clinical expert to answer that. My expectation,
18 irrespective of what the patent might say is, in terms
19 of plain speaking, one would suspect that one would
20 inform a clinician of such results before the clinician
21 makes a decision.

22 That would be in my mind, the key feature of
23 such an endeavor.

24 Q. All right, so for assays where you're gaining an
25 understanding of the presence or actions of a genetic

1 variant, your understanding is that one should inform
2 the physician of those findings?

3 A. One should inform -- not merely inform the
4 physician, one should inform the physician prior to them
5 making a clinical care decision, which is clearly not
6 occurred in Forshew.

7 Q. All right. So let's take a look at supplemental
8 table 7 in Forshew. And I will pull that up.

9 Let's take recess and go off the record.

10 A. Can I just ask -- I don't want to curtail this,
11 I just need a little rough guess as to when we're going
12 to finish off so I can arrange dinner. Very rough
13 guess, nothing, I'm not holding anyone to anything.

14 It's 4 in my world.

15 Q. My guess is we should wrap up by 6 o'clock.

16

17 MR. Luman: We're off the record at 1 o'clock
18 Pacific.

19

20 (Recess taken.)

21

22 MR. Luman: Back on the record at 1:12.

23

24 BY MS. SELLERS:

25 Q. So, Dr. Forshew, let's go back and discuss --

1 A. Dr. Furneaux.

2 Q. Oh, so sorry. So Dr. Furneaux, let's go back
3 and discuss Forshew.

4 A. Right.

5 MS. SELLERS: I think we are discussing turning
6 to table 7 of the supplemental materials, which is
7 Exhibit 2167, which I will introduce. It's previously
8 marked Exhibit 2167.

9 (Exhibit 2167 was marked for identification by
10 the court reporter and is attached hereto.)

11 BY MS. SELLERS:

12 Q. So that should be in Exhibit Share now for
13 everyone. And I will pull it up on my screen for you,
14 Dr. Furneaux.

15 It is the supplemental materials for Forshew.

16 A. Yeah, I'm familiar with it.

17 Q. So looking at table 7, a person of ordinary
18 skill in the art would have known that the genes in
19 table 7 are associated with cancer, correct?

20 A. All of these genes, let me just, a person of
21 skill in the art would probably go check these in the
22 cosmic database if they were interested in doing that.

23 I guess we could do that. We're still in 2016?

24 Q. Correct.

25 A. We'd have to go back and see if we can do a

1 cosmic analysis around about 2016.

2 Q. Just looking at this table --

3 A. Well, the guys that stick out, PIC3CA sticks out
4 obviously. I wouldn't be able to tell you with any -- a
5 level of precision.

6 I wouldn't be surprised that they have been
7 implicated in cancer, their mutations therein, depending
8 on the nature of the mutation. That wouldn't be
9 surprising to me.

10 Q. So would a person of ordinary skill in the art
11 have known that they necessarily involved in breast
12 cancer?

13 A. It would really depend on the extent knowledge
14 at the time as I said in terms of the cosmic -- but a
15 person skilled in the art, if they were interested in
16 this, they would go to that database and see what had
17 been done already.

18 Q. So ForsheW was targeting these ten mutations of
19 interest with their targeted amplicon sequencing assay,
20 correct?

21 A. Yeah.

22 Q. Do you agree it would have been obvious to
23 increase the number of mutations tracked to 20?

24 A. Not necessarily. It depends on the objective
25 here, depends on what they're trying to achieve.

1 Q. So do you agree it would have been obvious to
2 increase the number to 50?

3 A. Well, what you're saying here is it obvious to
4 an individual that when you increase the number, the
5 number increases.

6 Q. It would have been obvious you could increase
7 the number to 50?

8 A. Well, if we go back to what these folks actually
9 did, these were the ones they obtained, one tacitly
10 assumes that these were the only ones that they found.

11 Or the only ones they thought may well be
12 instructive. There is remarkably little detail that
13 helps us understand why they selected this particular
14 set.

15 Were they the only ones, were they ones they
16 thought may perhaps be of some utility. Was it based on
17 a track record of known mutations in these genes or the
18 function of these genes. Whole bunch of considerations.

19 Q. All right, and do you agree that Forsheo teaches
20 that higher read depth and using additional replicates
21 allows for enhanced mutation detection without change to
22 protocols?

23 A. Well, I think that's again something that's
24 asserted. One would like to see the evidence for that.

25 The simple experiment one has in mind is if in

1 fact one runs a set of experiments in which these
2 variables are increased and decreased and one sees the
3 attendant increase and decrease in whatever one seeks to
4 measure.

5 Q. All right. Let's go back to your declaration,
6 Exhibit 2169, for a moment. And let's turn to paragraph
7 236 to 237.

8 A. Um-hmm.

9 Q. I will share the screen with you. Or if you
10 have it in front of you --

11 A. No, we're best doing it both ways so we're all
12 on the same page.

13 Q. Do you see my screen here?

14 A. Yes.

15 Q. So in this paragraph, you cite multiple
16 references to support your argument that a person of
17 ordinary skill in the art would not have been motivated
18 to add additional primers to Ley's assay; is that
19 correct?

20 A. Yeah, let me just make sure I have the context
21 here of what the assertion was. If my memory serves me
22 correct, I'm looking at this a little bit.

23 I think the assertion by Dr. Q was that a person
24 of ordinary skill in the art would be motivated to
25 modify Ley's assay by including more PCR primer probes

1 targeted to various regions.

2 Again, we're talking about motivation primarily
3 before we get into the details. I'm not at all clear
4 why a person of ordinary skill in the art would be
5 motivated to modify Ley's asset.

6 Why would someone do that?

7 Q. So did you review these reference that you cited
8 in entirety?

9 A. Which references would those be now?

10 Q. So, for example, you cite, Mamanova and Mertes,
11 did you review these in their entirety?

12 A. Well, I certainly reviewed them. In fact, I
13 reviewed them more carefully for the previous litigation
14 in which we were consumed with the differences between
15 PCR and capture probes, which was a subject in the
16 previous litigation.

17 I probably refreshed myself a little bit with
18 them. But as I said before, I think the primary thing
19 here is motivation.

20 Q. Let's take a look at paragraph 236. And I want
21 to read you a portion of this. So you quote Mertes here
22 as saying (as read):

23

24 The main downside of the method is
25 that it does not scale easily in any

1 format to enable the targeting of very
2 large genome subregions or many DNA
3 samples.

4
5 Did I quote you correctly?

6 A. Yeah, let me just -- I think we're talking about
7 PCR here, am I correct, yeah?

8 Q. Yes.

9 A. Doesn't scale easily, yeah. I think what we're
10 illuminating here is given the unlikely scenario where
11 an individual who is presumably working in Ley's group,
12 having been charged to find new things, would suddenly
13 decide to do PCR on things that perhaps were already
14 known. I think it's extremely unlikely.

15 Q. Ley assay is directed toward 12 specific
16 mutations, correct?

17 A. Yes -- which have arisen from a search, which in
18 their minds, they were hoping to find new things. That
19 was the hope in their minds, whether they did or not we
20 can discuss.

21 So it would seem very bizarre that a person of
22 skill in the art would decide to learn from that and
23 then go off in a completely different direction that in
24 fact Ley counseled against.

25 Ley says, oh, don't look in the regions where

1 you already know where to look. Let's try and look in
2 new places.

3 MS. SELLERS: Well, I want to look at Mertes,
4 this reference that you cited. It is Exhibit 1006.

5 Introducing previously marked Exhibit 1006. I
6 will put this up on the screen again.

7 (Exhibit 1006 was marked for identification by
8 the court reporter and is attached hereto.)

9 BY MS. SELLERS:

10 Q. Can you see this exhibit, Dr. Furneaux?

11 A. Yes.

12 Q. So I would like you to read starting with "to
13 increase" and all the way down to "single tube" for me.
14 You can read it to yourself. You don't need to read it
15 out loud.

16 A. Sure. Yeah, I think I'm at least ready to
17 answer preliminary questions.

18 Q. All right. So here Mertes discloses that's
19 there is software that can help design multiplex PCR
20 arrays, correct?

21 A. That's certainly true.

22 Q. And Mertes disclose that using this software
23 several dozen PCR primer pairs can be included in a
24 single reaction, correct?

25 A. That's possible.

1 Q. Does Mertes disclose that?

2 A. Mertes gives some helpful hints about what one
3 could do and might do. What one should do is a
4 different issue.

5 Q. Does Mertes also disclose that using this
6 software in running many reactions in parallel hundreds
7 of different DNA fragments can be amplified?

8 A. Anything is possible.

9 Q. Does Mertes say that?

10 A. Yes, Mertes is giving some very general advice.

11 Q. Okay. And lastly, does Mertes disclose that
12 using a microdroplet PCR technique up to 20,000 PCR
13 primer pairs can effectively be included in a single
14 tube?

15 A. Quite correct. That is what he is saying, yeah.

16 Q. Thank you. So let's go back to your
17 declaration, Exhibit 2169. You see this, Dr. Furneaux?

18 A. Yeah, I do, thanks. We were at 236B.

19 Q. Yeah.

20 A. Yes.

21 Q. Here you also cite Mamanova as evidence that a
22 person of ordinary skill in the art would not be
23 motivated to add additional PCR primers to the assay,
24 because Mamanova states (as read):
25

1 PCR is difficult to multiplex to
2 any useful degree, the simultaneous use
3 of many primer pairs can generate a
4 high level of nonspecific amplification
5 caused by interaction between the
6 primers and, moreover, amplicons can
7 fail to amplify.

8
9 Is that an accurate description of your
10 declaration?

11 A. That's what my declaration says, yeah.

12 Q. So let's turn to Mamanova. I'm going to go
13 ahead introduce previously marked Exhibit 1007.

14 (Exhibit 1007 was marked for identification by
15 the court reporter and is attached hereto.)

16 BY MS. SELLERS:

17 Q. Do you see that on your screen?

18 A. Yeah.

19 Q. Great. If you could take a moment and read this
20 paragraph for me to yourself starting "with the
21 RainStorm platform."

22 A. I think I'm good, Abby, for any --

23 Q. Perfect. So here Mamanova is disclosing (as
24 read):

25

1 That using microdroplet PCR using
2 the RainStorm platform can be used to
3 perform multiplex PCR up to 4,000
4 primer pairs.

5
6 Is that correct?

7 A. That's correct.

8 Q. So let's take a look at figure 2, right here.
9 If you want to take a moment to review that if you need
10 to familiarize yourself with that again?

11 A. I think I'm probably ready.

12 Q. So this figure is communicating that if you have
13 one sample, PCR is feasible for enriching 1,000 genes;
14 is that correct?

15 A. Feasible for -- I didn't catch the last piece
16 you said.

17 Q. Feasible for enriching 1,000 genes?

18 A. Are we talking about 1,000 reactions or 1,000
19 simultaneous multiplex reactions?

20 Q. So what I'm asking you is that -- is this figure
21 communicating that PCR is suitable for 1,000 --
22 enriching 1,000 genes from a single sample?

23 A. So where are we starting, point 5?

24 Q. So it looks like this line from zero is
25 extending back --

1 A. Oh, so we're talking about an extrapolation.
2 What's the question?

3 Q. Whether this figure is communicating that if you
4 have one sample PCR is feasible for enriching 1000
5 genes.

6 A. Yeah, this figure suffers from the sort of
7 general problem we've been discussing all day in that
8 it's generic. It's merely an attempt to illustrate
9 differences but obviously the context of the particular
10 objective is key here.

11 These are theoretical warblings.

12 Q. I'm asking you what this figure is
13 communicating. And is it communicating that if you have
14 one sample, PCR is feasible for enriching 1,000 genes?

15 A. What this is doing is its theorizing that that
16 may be the case.

17 Q. And is it also communicating that if you have
18 one sample, PCR is feasible for enriching the whole
19 exome of a human?

20 A. Exomes change in size.

21 Q. The human exome?

22 A. Within individuals, yeah.

23 Q. Well. If we look at the figure, indicates where
24 the human exome is, correct, approximately? So is it
25 communicating that if you one sample, PCR is feasible

1 for enriching the whole exome of a human?

2 A. This is a theoretical figure that I understand,
3 you know, and all parts of reviews there are pieces that
4 are useful and pieces that are not useful.

5 This is an attempt to somehow stratify all of
6 these techniques within a theoretical framework. This
7 may not bear any, any significance to a particular
8 problem within a particular context.

9 Q. Okay, so we're discussing again general theories
10 of PCR. And so is this communicating that PCR can be
11 used to enrich a single sample or sorry -- let me strike
12 that.

13 I'm asking that, in general, is this figure
14 communicating that if you have one sample, PCR is
15 feasible for enriching the entire exome of a human?

16 A. Again, these are theoretical projections.

17 Q. All right, so let's turn back to that paragraph
18 of your declaration again. Do you see that for me?

19 A. Yep.

20 Q. So if we go to 237, I think we looked at this
21 briefly a little earlier today.

22 You cite three papers here, correct?

23 A. Yeah.

24 Q. Okay. And is it fair to say that your
25 conclusion from these papers is that PCR has limited

1 multiplex ability?

2 A. Yeah, I would agree with you that tends to be
3 the bottom line here. And it's somewhat significant
4 that this is coming from the folks of Foresight.

5 Q. Okay, in your opinion, in your declaration, was
6 that because of this limited multiplex ability, you
7 believe a person of ordinary skill in the art would not
8 have been motivated to add additional primers to Ley's
9 assay?

10 A. Yeah, to be clear, the initial motivation is
11 entirely lacking. And in the extremely unlikely event
12 that an individual was motivated to add more -- in
13 essence to test more alleles, which is what we're doing
14 here, in that very, very, very unlikely event that
15 individual would then also think twice by thinking, oh
16 dear, I have a system that's working, we've spent a
17 remarkable time and effort getting these eight or ten
18 potentially useful, who knows, yet to be proven, and now
19 I'm going to throw it all in the wind by adding in a
20 whole bunch of things that are not really clearly
21 related to the objective.

22 So the acknowledged difficulties in multiplexing
23 PCR and either carrying lots of reactions all in the
24 same tube, would be yet another nail in the coffin of
25 somebody who foolishly embarked upon this exercise.

1 Q. So if you had an assay that was working, would
2 you argue there's a not a motivation to improve upon it?

3 A. I think an American phrase is if it ain't broke,
4 don't fix it.

5 Q. So let's talk a little bit about multiplexing
6 and PCR. When you're using PCR, you don't have to run
7 or put all of the primers in the same pool, correct?

8 A. You're absolutely correct.

9 Q. So we could do either -- you could do multiple
10 multiplex reactions, complex?

11 A. You could split them up into -- suppose you had
12 to do 50, you could do five batches of ten.

13 Q. All right.

14 A. You could do all sorts of things. The catch is
15 you don't know which combinations are going to be useful
16 and which combinations may actually be bad news.

17 I didn't say that scientifically accurate. In
18 other words, there may be some combination of primers
19 that are in fact inimical to them working, and some
20 that are not.

21 And you unfortunately would be totally unaware
22 of which possibilities would work. So you could spend a
23 lot of time, undue experimentation, figuring that out.

24 Q. There are software tools such as Primer 3, for
25 example, that are designed to help you select primer --

1 A. Again, I think I have to say what characterizes
2 our conversation and Dr. Q's declaration is the
3 difference between a theoretician and an empirical
4 person.

5 Yes, you know, I was present way back when PCR
6 first came in. And we all had fun designing primers.

7 And we all had equal fun in understanding that
8 sometimes bioinformatical predictions didn't come to
9 success.

10 So sure, could do, may not do. But clearly it's
11 something that would make you think twice.

12 Q. So then a person of ordinary skill in the art
13 would recognize that running multiple primer pools in
14 parallel, PCR pools in parallel, and spreading out the
15 primers between the pools, could reduce the risks of,
16 for example, nonspecific amplification, correct?

17 A. Could reduce and could increase. And like I
18 said, you're working blind. And once we start talking
19 about, you know, numbers beyond 100, the number of
20 permutations of those pools you're suggesting -- do we
21 combine one two three four or five or do we combine one,
22 six, eight, nine?

23 Starts to be an infinite number of permutations.

24 Q. So let's say we have 20 PCR primers that went to
25 use in a PCR assay. You could run 20 singleplex PCR

1 wells; is that true?

2 A. I think that's absolutely true.

3 Q. So let's take at a look at that Dudley really
4 quickly, which we looked at a little earlier, Exhibit
5 2166; you see this?

6 A. Yes.

7 Q. So I'm going read you a section of Dudley. It
8 size that (as read):

9
10 PCR based assays have limited
11 multiplexability. With qPCR and
12 digital PCR it's difficult to assay
13 more than one mutation in a single
14 reaction pool.

15 When assaying for multiple
16 mutations, therefore, the original
17 sample is typically split across
18 multiple separate reaction pools, a
19 requirement that may pose challenges
20 when cfDNA is limited.

21

22 Is that a correct reading of the article?

23 A. I'm not seeing where you are. Where are you in
24 the third paragraph or --

25 Q. Oh, apologies.

1 A. (Reading inaudibly.)

2 Again, it's a very general one context-related
3 statement.

4 Q. Let's focus on the last sentence. Essentially
5 what Dudley is saying is that doing singleplex with PCR
6 and cfDNA can be difficult because cfDNA samples can be
7 pretty limited in size; is that correct?

8 A. That's what he's saying. I'm not sure I would
9 agree necessarily until I see the context that is
10 applicable to our discussion.

11 Q. Okay, well, a cell-free tumor DNA is a type of
12 cfDNA, correct?

13 A. It's "cell-free tumor DNA is a type of cfDNA."

14 Q. And your answer was?

15 A. You have to remind me what the question was.

16 Q. Cell-free tumor DNA is a type of cell-free DNA?

17 A. Yes, tumor, one imagines when released, it is
18 DNA -- sorry, the one imagines one is released by a
19 tumor and it is devoid of a cellular structure yet still
20 associated with cellular proteins.

21 Q. And Ley did not PCR cell-free DNA, correct?

22 A. My recollection is Ley, well, which experiment
23 are you referring to, the Sanger sequencing or the 454.

24 Q. When Ley sequenced the original tumor, relapsed
25 tumor, sample tumor.

1 A. Oh, let's be careful. The relapse tumor was not
2 sequenced originally, as you said. It was not subject
3 to whole genome sequencing.

4 Q. Okay.

5 A. So we're referring to the skin and the, you
6 know, for want of a better word, let's just call it the
7 first sample, the first tumor sample.

8 Yeah, the whole genome sequencing was performed
9 on that. And so therefore what they were using there
10 was DNA extracted from tissue, which would be in this
11 particular case, the leukemic cells and skin tissue.

12 Q. And the -- great. So essentially this idea that
13 you can't split a reaction into singleplex assays here
14 by Dudley doesn't have as much implication for full
15 tumor samples than cfDNA samples, correct?

16 MR. Luman: Objection, mischaracterizes the
17 document.

18 THE WITNESS: All I see here is they're saying
19 it may pose challenges and there is no data to support
20 this statement.

21 So I don't think this is a particularly informed
22 statement. And I also would add I think your
23 interpretation is a little skewed.

24 BY MS. SELLERS:

25 Q. So let's go back to paragraph 239 of your

1 declaration. Do you see that here?

2 A. Yes, I have it right here.

3 Q. You cite an article by Ross; is that correct?

4 A. That's correct.

5 MS. SELLERS: So let's take a look at Ross,
6 which I will introduce. All right, introducing
7 previously marked Exhibit 1014.

8 (Exhibit 1014 was marked for identification by
9 the court reporter and is attached hereto.)

10 BY MS. SELLERS:

11 Q. I'm going to share this with you, Dr. Furneaux.
12 So on the top of page 537, which is this page,
13 it says (as read):

14

15 Experts in the cancer sequencing
16 field hold that as many as 75 percent
17 or more of cancer gene sequence
18 variations can be missed by a
19 hotspot-only analysis.

20

21 Is that correct reading of the
22 document?

23 A. You have read the document accurately, yes.

24 Q. So would you agree this statement is in
25 agreement with Ley's --

1 (Reporter clarification.)

2 -- statement that hypothesis-driven examination
3 of tumor genomes is inherently limited and will miss key
4 mutations?

5 A. Sorry, was there a question in there?

6 Q. So would you agree that statement here from Ross
7 is in agreement with Ley's statement that
8 hypothesis-driven examination of genomes is inherently
9 limited and will miss key mutations?

10 A. I think a more accurate reflection on that is
11 both statements are consistent with a old scientific
12 adage, that is if you just keeping looking in the same
13 place, you may not see things in places that you don't
14 think they should be.

15 Is the simple reflection. Back to the drunk
16 looking for his glasses under the light.

17 Q. All right, and --

18 A. Keys, not his glasses, keys.

19 Q. So if we continue on in this section from Ross,
20 it states (as read):

21

22 However, it is equally clear that
23 tens or hundreds of individual
24 traditional mutation analysis assays to
25 test for all of the possible important

1 mutations in a given tumor are not
2 practical or feasible given the amount
3 of tumor sample, time, and money it
4 would take to perform so many tests.

5
6 So do you see that?

7 A. That's what he says.

8 Q. So would you agree that Ross is suggesting that
9 it's not feasible to test for hundreds or thousands of
10 possible mutations in every patient given the time and
11 expense of doing so?

12 A. You know, that's his suggestion. I would argue
13 with the statement that it's clear. And if I were
14 writing or reviewing this, I'd probably say it's
15 possible that, it's worth considering that this might
16 not be the way to go.

17 But, as ever, you need to look at the context of
18 a particular situation.

19 Q. All right. So Ross finishes by stating that (as
20 read):

21
22 The potential generation of
23 actionable gene sequence abnormalities
24 and abnormalities of unknown clinical
25 importance, the successful NGF-based

1 clinical service will have a result
2 report that is focused on the
3 abnormalities that can lead directly to
4 the best therapy, direct a patient to
5 an ongoing clinical trial or result in
6 some other actionable response from the
7 treating oncologist.

8
9 Do you see that?

10 A. Um-hmm.

11 Q. All right. So would you agree that Ross is
12 implying that a phase should be designed to target a
13 limited number of individual and hypothesis-driven
14 potential mutations that have the best possibility of
15 aiding a patient's treatment?

16 A. What I think he's saying is something a little
17 bit more subtle. What he's actually saying is perhaps
18 heretofore many of the variants that have been cataloged
19 aren't necessarily actionable.

20 And what he's basically saying, which is quite
21 common sense is, if we had a scenario where the vast
22 majority of the abnormalities that we're observing are
23 actionable, that would be a good thing.

24 Q. All right. So let's say that a person of
25 ordinary skill in the art discovers a new mutation such

1 as a deletion of base number 890 in gene A.

2 Gene A has 16 exons, it's a 100,000 bases --

3 A. Do you have a diagram for this? That would
4 help.

5 Q. If you want to take a second to jot it down?

6 A. Absolutely. Okay, fire away?

7 Q. Let's start over from the beginning. Let's say
8 a person of ordinary skill in the art discovers a new
9 mutation.

10 A. This is the person of super duper ordinary
11 skill.

12 Q. The definition we've been applying today,
13 correct.

14 A. So Mrs. Super Duper is going to do this stuff.

15 Q. Yes, the definition that we looked at earlier in
16 your declaration. And this mutation is a deletion of
17 base 890 in gene A.

18 A. Okay, I've got gene A, a line here, when you say
19 base 890, where is number one?

20 Q. Let's say number one is the start of the first
21 exon.

22 A. The start of the first exon. Is that coding
23 exon or a noncoding exon?

24 Q. I don't think that matters for this
25 hypothetical.

1 A. Well, time will tell, right?

2 I'm drawing an exome. How thick should that
3 exome be?

4 Q. Let's just keep going here. And you shouldn't
5 need that detail. So gene A has 15 exons and spans 100
6 kilobases?

7 A. When you say spans, what are we talking about,
8 from number one again?

9 Q. Yes, from the first exon all the way to the end
10 of the last exon including all of the noncoding and
11 intronic regions.

12 A. Fifteen exons and we go from one residue or base
13 to -- what was it now?

14 Q. 100 thousand.

15 A. Okay, good number, 100,000.

16 Q. And this deletion at number 890 is at exon
17 number 2?

18 A. 890, help me out, is in the middle of that exon,
19 what are the borders of the exon.

20 Q. I think it's the middle.

21 A. 890 we're going to say is in the middle of exon
22 number 2.

23 Q. And this person of ordinary skill in the art now
24 wants to test additional patients to see if they also
25 have the same --

1 A. Sorry, we've got to role back. So this is a
2 deletion mutation.

3 Q. Correct. So deleted between where and where?
4 It's just base 809, let's say is deleted.

5 A. A single-based deletion?

6 Q. Correct.

7 A. Do we have a base for that so we can talk about
8 it.

9 Q. So we can also say it's a single-based
10 substitution if that's an easier one for us to discuss.

11 A. How about there was an A at 890, and there's no
12 longer an A at 890 because now you there's a T because
13 the A's gone.

14 Q. Sounds good?

15 A. Because T was at 891.

16 Q. Yeah. We're focused on a single-nucleotide
17 variant here?

18 A. Before you said a single-nucleotide deletion.

19 Q. Correct?

20 A. Somewhat rare, correct?

21 Q. Correct. But we're just looking here just for a
22 general kind of understanding. Just want to set the
23 stage.

24 So a person of ordinary skill in the art wants
25 to then see if additional patients have this mutation.

1 A. How does that person of ordinary skill in the
2 art determine this phenomenon in the first go round?

3 Q. Let's say they did whole exome sequencing and
4 compared it to a reference?

5 A. Well, let's instead of whole exome, how about
6 they just analyzed this gene.

7 (Reporter clarification.)

8 Q. We'll assume they did whole gene sequencing,
9 they sequenced just that one gene to determine that.

10 So now, let's get into in this hypothetical.
11 And so now they want to look at additional patients and
12 see if they also have this mutation.

13 A. Remember, the place we stalled here was I'm
14 somewhat anxious to know how they established this was a
15 single-based deletion.

16 Q. Let's move on to my question and we'll go from
17 there. So does the person need to perform whole exome
18 or whole genome sequencing on each of the individual
19 patients, each of the additional patients to determine
20 whether they have this mutation?

21 MR. Luman: Incomplete hypothetical.

22 THE WITNESS: Okay, notwithstanding that. But
23 so let me just get a little bit more detail here.

24 This DNA they sequenced here in first place is
25 coming from a tumor or --

1 BY MS. SELLERS:

2 Q. Correct, they're sequencing tumor DNA?

3 A. So that's important. So they sequenced tumor
4 DNA and ascertained this single-based deletion. And
5 they've checked that by running sequence of normal DNA.

6 Q. Correct.

7 A. Right?

8 Q. Correct.

9 A. And now they've said well we've assayed this
10 tumor DNA and now we're going to do what?

11 Q. Now, we want to see if in additional patients,
12 this mutation -- additional patients have the same type
13 of cancer if this mutation's also present?

14 A. Why would you do that?

15 Q. Let's go through the question --

16 A. In the imaginary world where this is important,
17 and one would desire to find this mutation in yet a new
18 patient with the same kind of tumor.

19 Q. Um-hmm.

20 A. Exactly the same kind of tumor?

21 Q. Correct.

22 A. How do we know that?

23 Q. We know that, we've established this -- do they
24 need to perform full genome or full exome sequencing
25 again on this additional patient to determine if they

1 have that mutation?

2 A. They don't need to do sequencing at all?

3 Q. Okay, do they need -- if they are already using
4 sequencing, do they need to sequence all of gene A?

5 A. I just said they don't need to do sequencing at
6 all.

7 Q. I'm saying they are. If they are doing
8 sequencing, do they need to sequence all of gene A?

9 A. You're asking me what to do. And I'm saying
10 what one would do, is one would notice that that
11 single-base deletion actually creates a new restriction
12 site and they decide to go that way.

13 Q. I'm saying if you are doing sequencing --

14 A. We're still in the imaginary world, right?

15 Q. Yes, and in this imaginary world, we're doing
16 sequencing --

17 A. And the person of skill in the art has not been
18 asked to leave my lab yet because they are doing silly,
19 silly things?

20 Q. No. Okay, so would you sequence all of gene A?

21 A. Okay, so if we want to check that in fact this
22 patient has a single-based deletion, as we have
23 presumably discovered in Mrs. A.

24 We're now looking at Mrs. B, and I was directing
25 this person of ordinary skill in the art, they would go

1 forth and come back with two possibilities: We have
2 found this deleted base in patient B, and the second
3 possibility which we're all going to remember, is we
4 haven't found it.

5 And at this point, I would say, yes, well you
6 are an idiot because for all we know this fascinating
7 bit of DNA might have other mutations in other regions.

8 Q. Okay. But would you need to sequence all of
9 gene A to look for that specific deletion?

10 A. Well, one of the things we keep falling into
11 here is you keep imposing as if it's very clear what the
12 motivation and the objective is.

13 I'm thinking, gee, Mrs. B has cancer and Mrs. B
14 holds our attention to the fullest extent, we would like
15 to tell Mrs. B, hey, you don't have mutation in gene A,
16 all is good in this imaginary world.

17 We would like to tell her, oh, you know we
18 looked at position 898 but hey that's okay.

19 What's Mrs. B going to say? Well, you did look
20 at the other bits, did you? Well, grrr, we didn't.

21 Q. Okay. Let's move on from this hypothetical.

22 A. I'm not sure what character I'm playing in this
23 imaginary world.

24 Q. Okay. So let's move on from this hypothetical.

25 A. We're not in Alice in Wonderland anymore? Where

1 are we now?

2 Q. Let's just again talk in which general about the
3 disclosure in the '611 patent.

4 A. Okay.

5 Q. So one of the disclosures is, as we've
6 discussed, this probe set that you can use, this
7 personalized probe set?

8 A. Where are we in the declaration now? I'm having
9 difficulty getting out of Alice in Wonderland.

10 Q. Let's just look at the claim here. Pulling up
11 Exhibit 1001.

12 A. Is that still the '611 or --

13 Q. Let's pull up the '789 or sorry the '783.

14 A. Very small font.

15 Q. Can you see my screen?

16 A. Yeah, thank you.

17 Q. So in claim 1, we end up synthesizing -- the
18 claim directs the person of ordinary skill in the art to
19 come up with a personalized probe set, correct, to
20 selectively enrich or amplify sequences?

21 A. Yeah, let me just reacquaint myself with this.
22 (Reading inaudibly.)

23 So the end of B is to generate a sequencing
24 library, am I correct? Have I read all this correctly?

25 Q. Yeah.

1 A. Okay.

2 Q. So would you agree that one of the goals of
3 creating this personalized probe set and doing a second
4 assay to create the sequencing library is to increase
5 the speed at which these additional samples can be
6 sequenced?

7 A. I haven't really thought about that in great
8 detail. Let me think about that.

9 Yeah, there's a little too much ambiguity to
10 give you -- because there's two possibilities here,
11 right, it says selectively enrich, which one would
12 imagine is done by hybridization or amplify.

13 Those are two possibilities. So in terms of
14 giving you an opinion on the speed would depend on which
15 of those roads one is going down.

16 Q. Okay, so if you were to selectively enrich,
17 would you argue that that could -- or would you agree
18 that one of the goals could be to increase the speed at
19 which you process additional samples?

20 A. Increase the speed over what scenario?

21 Q. So in the beginning, you do a larger genome
22 sequencing, correct? And select specific mutations then
23 to target with your probe; is that correct?

24 A. That is correct.

25 Q. All right, so then the goal here would be that

1 you can potentially increase the speed of additional
2 sample processing because your probes can be more
3 targeted and you don't need to do as wide a gene
4 sequencing; is that correct?

5 A. Well, I think we have to be more specific here.

6 If we are talking about enriching, for example,
7 hybridizing a probe to set desired portions of the human
8 genome, that does take time.

9 It resolves around temperature and time. This
10 is a chemical reaction. So, of course, you know, it
11 depends, of course, also on concentration that we're
12 dealing with.

13 But, what I'm getting to here is, we're still
14 seeking the comparison with the scenario that you are
15 implying takes less time or more time. What are
16 comparing here in terms of saving time, what was the
17 other option?

18 Q. Of resequencing the entire sample just as was
19 done in step A?

20 A. Well, the whole purpose of this regimen is to
21 avoid sequencing the whole genome again. That is the
22 driving force of this invention to come up with a
23 solution to this problem.

24 Q. So you would agree that the goal here is to
25 decrease the footprint of the second assay?

1 A. Footprint in the sense that we're using here --
2 in other words, there is -- if this is successful, there
3 will be less bases to sequence is the simple principle
4 here.

5 Q. Right.

6 A. You were asking me about time, which is an
7 entirely different consideration.

8 Q. Okay, so the goal here is to decrease the
9 footprint from step one to step -- from the first assay
10 to the second assay?

11 A. And to generate a sequencing library. I keep
12 emphasizing that because I do feel that this is
13 information I should intimate to you at some point that
14 may not be entirely clear in my deposition.

15 Q. Okay, well, let's say your first sequencing
16 assay, your footprint is let's say ten million bases.
17 And then in your second one, your footprint is --

18 A. In the first one, I think we're intuitively
19 understanding that is the whole genome --

20 (Speaking simultaneously.)

21 THE WITNESS: Let me go back and see what we're
22 doing here again. We're starting off obtaining -- da,
23 da, da, da, da, da -- are identified in sequence data or
24 least -- okay.

25 So the scenario that one assumes has taken place

1 here is that we have done a large-scale sequence effort,
2 that has identified useful areas of investigation.

3 BY MS. SELLERS:

4 Q. Correct. So are you comfortable then let's say
5 we're sequencing 30 million bases in this first part of
6 the assay or this first assay?

7 A. In step one, I would intuitively imagine we're
8 going much wider than that.

9 Q. Okay.

10 A. I would imagine in the context of what we are
11 discussing here although it doesn't explicitly say that.

12 What it says is identified in sequence data.
13 But clearly if you are looking for new things, you
14 should go as wide as possible.

15 And so therefore, for example, what Ley did
16 would be the sort of things one would imagine that would
17 be going on if one was pursuing this endeavor.

18 Q. Okay. And then so let's say your second assay,
19 you have a footprint of 500 kilobases. Do you still
20 think the goal of the assay here -- or the goal of the
21 procedure here was accomplished?

22 A. You're asking me hypothetically. I'd like to
23 see the evidence that supports that notion.

24 Q. Would you say that decreasing from 15 million
25 gigabases to 500 kilobases is furthering the purpose of

1 this assay, which is to decrease the footprint?

2 A. Well, the purpose of the assay is to identify
3 genetic variants.

4 Q. Correct, but earlier you said that one of the
5 benefits here is that from the first assay to the second
6 assay is that the footprint is drastically --

7 A. Sure, but, you know, things could go wrong in
8 life. You might pick the wrong segment to focus on, it
9 might be a mistake.

10 Your initial analysis said, hey, there's a
11 variant in let's just say section B. You go zoom in on
12 section B and horror or horrors, there's no variant in
13 there.

14 MR. Luman: We've been going for about an hour.
15 Are you going to take break soon?

16 MS. SELLERS: After we finish this line of
17 questioning. We can take a break.

18 MR. LUMAN: Absolutely.

19 BY MS. SELLERS:

20 Q. So you would agree then that there is no magic
21 number of what the footprint of the second assay needs
22 to be. Just the benefit comes from drastically reducing
23 the footprint of the second assay?

24 A. I would agree there's no magic in biochemistry.
25 Let me just say that first of all.

1 And again, we're talking about generalities.
2 General things. Kinds of things theoreticians like Dr.
3 Q talks about in a world where actually quite like Alice
4 in Wonderland things always work.

5 I represent a different world where things may
6 not work in the way you think. And thus generalities
7 become interesting but not relevant.

8 Q. So would you say, would you agree then, that the
9 goal of this assay or the goal of this disclosed assay
10 is accomplished assuming that the footprint is
11 drastically reduced in the second set of --

12 A. Repeat that question again?

13 Q. Would you agree that the purpose of this
14 disclosed two-step or two-part assay is accomplished
15 assuming that the footnote is drastically reduced from
16 the first assay to the second assay?

17 A. The objective of this exercise, which is to
18 identify genetic variance, is the accomplishment we are
19 seeking.

20 Q. Okay. I think we can go ahead and go off the
21 record here. Take a break.

22 MR. Luman: Off the record at 2:12 p.m.

23

24 (Recess taken.)

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MR. Luman: Back on the record at 5:25.

MS. SELLERS: So we will go ahead and pass the witness.

MR. Luman: Let's go off the record. Off the record at 2:25, and give me ten minutes.

(Recess taken.)

MR. LUMAN: Back on the record at 2:34, and we have no additional questions for Dr. Furneaux. So we can now go off the record at 2:34.

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I declare under penalty of perjury under the laws of the
State of California that the foregoing is true and
correct.

Executed on _____, 2024, at
_____, _____.

SIGNATURE OF WITNESS

1 STATE OF CALIFORNIA) ss.
2 COUNTY OF LOS ANGELES)
3

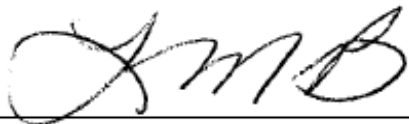
4 I, Lori M. Barkley, CSR No. 6426, do hereby
5 certify:

6 That the foregoing deposition proceedings were
7 taken before me at the time and place therein set forth
8 and were recorded stenographically by me, and were
9 thereafter transcribed under my direction and
10 supervision, and that the foregoing pages contain a
11 full, true and accurate record of all proceedings and
12 testimony to the best of my skill and ability.

13 I further certify that I am neither counsel for
14 any party to said action, nor am I related to any party
15 to said action, nor am I in any way interested in the
16 outcome thereof.

17 IN WITNESS WHEREOF, I have subscribed my name
18 this 30th day of January, 2024.

19
20

A handwritten signature in cursive script, appearing to read 'LMB', is written over a horizontal line.

21 LORI M. BARKLEY, CSR No. 6426
22
23
24
25

1 Abigail Sellers, Esq.

2 Asellers@irell.com

3 January 30, 2024

4 RE: FORESIGHT DIAGNOSTICS INC. vs. PERSONALIS, INC.

5 January 26, 2024, Henry Morrice Furneaux, Vol. I (JOB NO. 6412559)

6 The above-referenced transcript has been

7 completed by Veritext Legal Solutions and

8 review of the transcript is being handled as follows:

9 ___ Per CA State Code (CCP 2025.520 (a)-(e)) - Contact Veritext
10 to schedule a time to review the original transcript at
11 a Veritext office.

12 ___ Per CA State Code (CCP 2025.520 (a)-(e)) - Locked .PDF
13 Transcript - The witness should review the transcript and
14 make any necessary corrections on the errata pages included
15 below, notating the page and line number of the corrections.
16 The witness should then sign and date the errata and penalty
17 of perjury pages and return the completed pages to all
18 appearing counsel within the period of time determined at
19 the deposition or provided by the Code of Civil Procedure.

20 ___ Waiving the CA Code of Civil Procedure per Stipulation of
21 Counsel - Original transcript to be released for signature
22 as determined at the deposition.

23 ___ Signature Waived - Reading & Signature was waived at the
24 time of the deposition.

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1 ___ Federal R&S Requested (FRCP 30(e)(1)(B)) - Locked .PDF
2 Transcript - The witness should review the transcript and
3 make any necessary corrections on the errata pages included
4 below, notating the page and line number of the corrections.
5 The witness should then sign and date the errata and penalty
6 of perjury pages and return the completed pages to all
7 appearing counsel within the period of time determined at
8 the deposition or provided by the Federal Rules.
9 _x_ Federal R&S Not Requested - Reading & Signature was not
10 requested before the completion of the deposition.

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1 FORESIGHT DIAGNOSTICS INC. vs. PERSONALIS, INC.
2 Henry Morrice Furneaux, Volume I (JOB NO. 6412559)
3 E R R A T A S H E E T
4 PAGE_____ LINE_____ CHANGE_____
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6 REASON_____
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19 PAGE_____ LINE_____ CHANGE_____
20 _____
21 REASON_____
22 _____
23 _____
24 WITNESS _____ Date _____
25

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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.

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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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