

1 UNITED STATES PATENT AND TRADEMARK OFFICE

2
3 BEFORE THE PATENT TRIAL AND APPEAL BOARD

4
5 SAREPTA THERAPEUTICS, INC.,
6 Petitioner,

7 v.

8 THE TRUSTEES OF THE UNIVERSITY OF PENNSYLVANIA,
9 Patent Owner.

10 IPR2024-00580
11 Patent 11,680,274 B2

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14 Video Deposition of RICHARD JUDE SAMULSKI, Ph.D.
15 (Taken by the Petitioner)
16 Durham, North Carolina
17 Thursday, March 6, 2025

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22 Reported by: Marisa Munoz-Vourakis -
RMR, CRR and Notary Public

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Page 1

1 APPEARANCE OF COUNSEL: 2 For the Petitioner: 3 ANNE TOKER, ESQ. 4 ROBERT WILSON, ESQ.(By Zoom) 5 Quinn, Emanuel, Urquhart & Sullivan, LLP 6 555 Twin Dolphin Drive, 5th Floor 7 Redwood Shores, CA 94065 8 212-849-7000 9 annetoker@quinnemanuel.com 10 11 For REGENXBIO and the Deponent: 12 SUSAN E. MORRISON, ESQ. 13 CASEY M. KRANING, Ph.D., ESQ. 14 Fish & Richardson P.C. 15 222 Delaware Avenue, 17th Floor 16 Wilmington, DE, 19899 17 302-652-5070 18 morrison@fr.com 19 cmk@fr.com 20 21 Also Present: CARY MILLER, Ph.D. of REGENXBIO 22 Also Present by Zoom: LAURA CORUZZI of REGENXBIO 23 24 25 Page 2	1 INDEX 2 Examination of: Page 3 RICHARD JUDE SAMULSKI, Ph.D. 4 EXAMINATION BY MS. TOKER 7 5 EXAMINATION BY MS. MORRISON 270 6 FURTHER EXAMINATION BY MS. TOKER 289 7 DEPOSITION EXHIBITS 8 EXHIBIT NUMBER DESCRIPTION PAGE 9 Exhibit 1007 '772 Patent Application 40 10 Exhibit 1009 Nuclear targeting by 88 adeno-associated virus capsid 11 proteins and virions, by Stephen Snowdy 12 Exhibit 1057 7-1-2019 Statement: Pfizer 11 13 Phase 1b Clinical Trial Results 14 Exhibit 1058 Therapeutic Liabilities of In 33 Vivo Viral Vector Tropism: 15 Adeno-Associated Virus Vectors NMDAR1 Antisense, and Focal 16 Seizure Sensitivity, paper 17 Exhibit 1059 Cover Sheet for Filing 100 Provisional Patent Application 18 60-636126 19 Exhibit 1060 Single Amino Acid Changes Can 201 Influence Titer, Heparin 20 Binding, and Tissue Tropism in Different Adeno-Associated 21 Virus Serotypes article 22 Exhibit 1061 US Patent 7,892,809 233 23 Exhibit 1062 Phase 1 Gene Therapy for 250 Duchenne Muscular Dystrophy 24 Using a Translational Optimized AAV Vector article 25 Page 4
1 APPEARANCES (Continued): 2 For the University of Pennsylvania: 3 JULIE S. GOLDEMBERG, ESQ.(By Zoom) 4 Morgan Lewis Bockius LLP 5 2222 Market Street 6 Philadelphia, PA 19103-3007 7 215-963-5095 8 julie.goldemberg@morganlewis.com 9 10 Also Present: JOHN COOPER, Videographer 11 12 13 o0o 14 15 Video Deposition of DR. RICHARD JUDE 16 SAMULSKI, taken by the Petitioner, at Hilton Durham, 3800 17 Hillsborough Road, Durham, North Carolina, on the 6th day 18 of March, 2025 at 9:18 a.m., before Marisa Munoz-Vourakis, 19 Registered Merit Reporter, Certified Realtime Reporter 20 and Notary Public. 21 22 23 24 25 Page 3	1 2 DEPOSITION EXHIBITS(Continued) 3 4 Exhibit 2027 Declaration of Dr. Richard 25 Jude Samulski 5 Exhibit 2028 Dr. Samulski's CV 20 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 Page 5

1 PROCEEDINGS 2 THE VIDEOGRAPHER: Good morning. We 3 are going on the record at 9:18 a.m., on 4 Thursday March 6, 2025. 5 This is media unit one of the video 6 recorded deposition of Dr. Richard Jude 7 Samulski, taken in the matter of Sarepta 8 Therapeutics, Incorporated versus The 9 Trustees of the University of Pennsylvania, 10 Patent Number 11,680,274 B2, Petition Number 11 IPR2024-00580, being heard in the United 12 States Patent and Trademark Office before 13 the Patent Trial and Appeal Court. 14 We are at the Hilton Durham located at 15 3800 Hillsborough Road in Durham, North 16 Carolina. 17 Counsel will now state their 18 appearances and affiliations for the record 19 beginning with the noticing attorney. 20 MS. TOKER: I am Anne Toker 21 representing Sarepta in this proceeding, and 22 I'm from the law firm of Quinn Emanuel in 23 New York City. 24 MS. MORRISON: Susan Morrison of Fish 25 & Richardson on behalf of REGENXBIO and the Page 6	1 correct? 2 A. Correct. 3 Q. Have you ever been deposed before? 4 A. I have. 5 Q. In what kind of proceeding? 6 A. It was related to, shoot give me a second. 7 This is embarrassing. It was related to AAV gene 8 therapy, but I'm blanking on the specifics of what we 9 were there haggling over. 10 Q. Was it a patent proceeding? 11 A. Yeah. 12 Q. Okay. Is this the only time? 13 A. Yeah. I'm sorry, yes. 14 Q. Doctor, I will get to a few ground rules. 15 Okay. So you were deposed in a proceeding related to 16 AAV gene therapy? 17 A. Yes. 18 Q. Can you give me any more specifics about 19 that? 20 A. I'm trying to remember. It was related to 21 the -- for the life of me I'm blanking on it. This is 22 what happens when you're first time on TV and you're 23 anxious. 24 Oh, yes, it was a Novartis 25 self-complimentary technology challenged by Genzyme in Page 8
1 witness. And with me today are Casey 2 Kraning, also of Fish & Richardson, Cary 3 Miller from REGENXBIO and on video is Laura 4 Coruzzi of REGENXBIO, and also on video is 5 Julie Goldemberg, who is counsel for the 6 University of Pennsylvania. 7 MS. TOKER: And I will add that Robert 8 Wilson from Quinn Emanuel is also joining us 9 on video, also representing Sarepta. 10 Whereupon, RICHARD JUDE SAMULSKI, having 11 been first duly sworn, was examined 12 and testified as follows: 13 EXAMINATION BY COUNSEL FOR PETITIONER 14 BY MS. TOKER: 15 Q. Good morning, Dr. Samulski. 16 A. Hello. 17 Q. Would you please state your full name for 18 the record? 19 A. It's Richard Jude Samulski. 20 Q. And please state your residential address 21 for the record? 22 A. It's 201 Southwind Lane, Hillsborough, 23 North Carolina 27278. 24 Q. You understand that you are here to testify 25 on behalf of REGENXBIO and University of Pennsylvania, Page 7	1 the Barry Carter patent, where they claimed they had 2 developed self-complimentary technology. 3 Q. And you were an expert in that proceeding? 4 A. Yes. 5 Q. For which party? 6 A. Novartis. 7 Q. Did you testify in court? 8 A. No. We did a similar thing like this, and 9 I think after that, there was a consensus, and it was 10 resolved. 11 Q. And that's the only time you've been 12 deposed? 13 A. Um-hum. 14 Q. I'm just going to say a few ground rules 15 then just to help us get a clear record. 16 I think, as the court reporter mentioned 17 before we went on the record, you know, we should try 18 not to talk over each other, right? 19 A. Understood. 20 Q. Okay. So let me finish my question before 21 you start your answer, and I'll do my best to let you 22 finish your answer before I start my next question, 23 okay? 24 A. Yeah, understand. 25 Q. Yes, and you have to answer. Page 9

1 A. I got it, because she can't see it. 2 Q. You have to answer out loud. Understood? 3 A. Yes. 4 Q. Okay. And it's also because if you don't, 5 then, right, we won't get your answer written down as a 6 verbal answer to the questions, right? 7 A. Correct. 8 Q. Okay. If you answer a question, I will 9 assume that you have understood my question, okay? 10 A. Yes. 11 Q. All right. And if you need a break at any 12 time, feel free to ask for a break, but I would ask 13 that you answer a pending question before we take the 14 break, okay? 15 A. Understood. 16 Q. All right. Now, beginning in 2001 and 17 going up to the present, you've been involved in 18 various capacities with several different companies, 19 correct? 20 A. That's correct. 21 Q. I see you're wearing an AskBio fleece as we 22 speak here, is that right? 23 A. That's correct. 24 Q. All right. And in fact, from September of 25 2001 to March 2024, you were associated with AskBio, Page 10	1 by your company, AskBio, correct? 2 MS. MORRISON: Objection to form. 3 BY MS. TOKER: 4 Q. And, I'm sorry, Dr. Samulski, did you say 5 yes? 6 A. I'm assuming you're correct in that, yes. 7 Q. All right. And it's dated July 1 of 2019, 8 correct? 9 A. That's what it says, July 1, 2019, correct. 10 Q. And that's when you were chief scientific 11 officer of AskBio, right? 12 A. Correct. 13 Q. All right. And so this press release from 14 your company -- 15 MS. TOKER: Sorry, can we go off the 16 record for a second? 17 THE VIDEOGRAPHER: The time is 9:26. 18 We are off the record. 19 (Recess.) 20 THE VIDEOGRAPHER: The time is 9:26. 21 We're on the record. 22 BY MS. TOKER: 23 Q. This press release relates to some results 24 that Pfizer reported from a Phase 1b clinical trial 25 having to do with a gene therapy for Duchenne muscular Page 12
1 correct? 2 A. That's correct. 3 Q. All right. So you were the founder of 4 AskBio, right? 5 A. That's correct. 6 Q. Also known as Asklepios BioPharmaceutical, 7 Inc.? 8 A. Asklepios. 9 Q. Asklepios BioPharmaceutical, Inc.? 10 A. Correct. 11 Q. So you were the founder, and then you were 12 the president and also chief scientific officer, right? 13 A. Correct. 14 MS. TOKER: I'm going to ask the court 15 reporter to mark this document as 16 Exhibit 1057 please. 17 (The document referred to was marked 18 Deposition Exhibit Number 1057 for 19 identification.) 20 Q. Dr. Samulski, the court reporter has just 21 handed you what's been marked Exhibit 1057. Have you 22 seen this document before? 23 A. No. I'm aware of this, but not this 24 specific document. 25 Q. All right. This is a press release issued Page 11	1 dystrophy, correct? 2 A. That's correct. 3 Q. And if you could look down with me, please, 4 at the fourth paragraph. It says: We commend Pfizer 5 Solid, Sarepta Therapeutics and all other companies for 6 their work in finding curative therapies for 7 life-threatening diseases, such as DMD. Do you see 8 that? 9 A. Yes. 10 Q. So as of 2019, you commended Sarepta for 11 its work in finding curative theories for 12 life-threatening diseases such as DMD, right? 13 MS. TOKER: Objection to the form. 14 A. I think all we did was acknowledge all of 15 the groups that were working to try to provide a cure 16 for DMD. 17 Q. And of the groups that you mentioned, 18 Pfizer, Solid and Sarepta, only Sarepta in fact today 19 has an FDA approved marketed gene therapy for DMD, 20 correct? 21 A. That's correct. 22 Q. You, yourself, Dr. Samulski, have not 23 invented a therapy that has gotten FDA approval and 24 come to market, correct? 25 MS. MORRISON: Objection to the form. Page 13

1 A. Well, I think I disagree with that. 2 Q. How so? 3 A. Every drug that's been approved has got my 4 gene therapy, AAV terminal repeats in it. There will 5 be no drug that will be approved by the FDA without 6 carrying those elements, and therefore essentially 7 every drug that's approved, it's got a component that I 8 developed. 9 Q. Does AskBio have a gene therapy that's been 10 approved by the FDA? 11 A. Not that I'm aware of. 12 Q. You're currently the cofounder and chief 13 scientific officer of a company called M34, Inc., 14 correct? 15 A. That's correct. 16 Q. Where does the name M34 come from? 17 A. You want the long version or the short 18 version? 19 Q. Let's start with the short version. 20 A. Okay. There are 33 miracles that have been 21 performed in the Bible: Making blind people see; lame 22 people walk; rising people from the dead; and since we 23 achieved all of those through the technology of AAV 24 gene therapy, we thought if Christ would have been 25 around one more year, this would have been miracle 34. Page 14	1 this proceeding, correct? 2 A. Correct. 3 Q. Dr. Snowdy carried out his thesis work in 4 your laboratory at the University of North Carolina, 5 right? 6 A. Correct. 7 Q. You were his thesis adviser? 8 A. That's correct. 9 Q. What role does a thesis adviser play in a 10 graduate student's research work during graduate 11 schooling? 12 A. There are many different roles, depending 13 on the maturity and the development of the student. 14 Some of it is just monitoring their work. Some of it 15 is going to the bench and showing them how to do 16 experiments. 17 Q. So a thesis adviser supervises the graduate 18 students' research work? 19 A. Again, depending on different levels. 20 Sometimes it's holding the hand to make sure they know 21 how to run a sequencing gel, and sometimes it's telling 22 them how to outline a paper or manuscript. 23 Q. You supervised Dr. Snowdy's graduate work, 24 correct? 25 A. I did. Page 16
1 Q. Your company would have been miracle 34 of 2 Jesus Christ? 3 A. Um-hum. 4 Q. Yes? 5 A. Yes. 6 Q. What is M34 focusing on in its effort to 7 perform Jesus' 34th miracle? 8 A. It's called transdifferentiation. It's 9 where you take the parts of the brain that no longer 10 produce neurons and you turn on a switch to make cells 11 turn back into neurons, therefore getting rid of 12 dementia, Alzheimer's, Parkinson's and all neurological 13 disorders. 14 Q. Is this an AAV gene therapy approach to 15 curing all neurological disorders? 16 A. Correct. 17 Q. Now, we're going to be talking more later 18 today, Dr. Samulski, about a dissertation of a graduate 19 of yours Stephen Snowdy. You're familiar with Stephen 20 Snowdy, correct? 21 A. Yep, Steven Snowdy. 22 Q. Snowdy, yes? 23 A. Yes. 24 Q. All right. And you've given opinions on 25 Dr. Snowdy's dissertation as part of your opinions in Page 15	1 Q. And he worked on a project that you helped 2 him select, correct? 3 A. I did. 4 Q. You helped him design the project that he 5 worked on? 6 A. I helped guide him on that project, yep. 7 Q. You and Dr. Snowdy worked closely on the 8 experiments he carried out in your laboratory, right? 9 A. We did the same amount of attention as the 10 other students that were there. 11 Q. So you worked closely on the experiments 12 that he carried out in your laboratory, right, you both 13 did? 14 A. When you say closely, you have to help 15 define that, because Stephen was an ex-Seal Marine, and 16 he was much older than the other students. So he was 17 around 28-years old, when my other students were 21, 18 22. So, again, the attention to closeness was based on 19 maturity as well as scientific skills and things of 20 that nature. 21 Q. So Dr. Snowdy was older, and having been a 22 Marine, he needed less supervision than some of your 23 younger fresh-out-of-college type of graduate students, 24 fair? 25 A. That would be a fair assumption. Page 17

1 Q. Now, Dr. Snowdy worked in your laboratory 2 for several years, correct? 3 A. Yes. 4 Q. You would never allow a graduate student in 5 your lab to submit a thesis for approval by his or her 6 thesis committee that you thought did not meet the 7 requirements for a Ph.D., right? 8 A. It's not my job. It's the job of the 9 thesis committee. 10 Q. You're on the thesis committee, aren't you? 11 A. Right. So that the whole committee has to 12 make that decision. 13 Q. But you as the thesis adviser would not 14 bring work to a thesis committee, or allow a graduate 15 student to bring work to the committee that you felt 16 was not worthy of the grant of the Ph.D., right? 17 MS. MORRISON: Objection to the form. 18 A. So I can present to you Stephen Snowdy's 19 situation that would shed some light on the question 20 you're asking. 21 Q. My question was a general question. 22 A. And my answer is more specific. 23 Q. Well, can you answer my question first, 24 which is, would you allow a graduate student to submit 25 a thesis for approval by the committee that you thought Page 18	1 indicates that you and the whole rest of the 2 committee -- 3 A. Correct. 4 Q. Let me just finish my question. 5 A. Sorry. 6 Q. -- thought that the dissertation was worthy 7 of the grant of a Ph.D, correct? 8 A. That is the correct assumption. 9 Q. It's not an assumption. It's a statement, 10 right? It's correct to say that you and the rest of 11 the committee believed that the thesis was worthy of 12 being granted a Ph.D when you all signed off on it, 13 correct? 14 A. I would say that's correct. 15 Q. Now, let's take a look at your CV. I am 16 just going to ask the court reporter to hand you what 17 has already been marked Exhibit 2028 in this 18 proceeding. 19 (The document referred to was 20 previously marked Deposition Exhibit 21 Number 2028 for identification.) 22 Q. Dr. Samulski, the court reporter has just 23 handed you what has been marked Exhibit 2028 in this 24 proceeding. You're familiar with this document, right? 25 A. Yes. Page 20
1 did not meet the requirements for the Ph.D.? 2 MS. MORRISON: Objection to the form. 3 A. As I mentioned, before we get students at 4 different levels or capability, and sometimes they 5 perform Ph.D. theses that are acceptable, and 6 sometimes they perform thesis that are exceptional, and 7 Stephen's was acceptable. 8 Q. But worthy of a Ph.D., correct? 9 A. Yes. 10 Q. All right. Because you wouldn't -- you 11 wouldn't sign off on a thesis that you believed did not 12 meet the requirements for a Ph.D, right? 13 A. That's what I said. 14 Q. And you signed off on Stephen Snowdy's 15 dissertation, right? 16 A. We typically sign off. We typically 17 recommend that they fix edits and things before they 18 turn in their thesis and the following; and whether 19 they do or don't, we typically don't follow up. 20 Q. But the fact that your signature is on the 21 first page of his dissertation means that you thought 22 that it was worthy of being granted a Ph.D, correct? 23 A. Yeah, me and the other four colleagues. 24 Q. So the fact that all of those signatures 25 are on the first page of Dr. Snowdy's dissertation Page 19	1 Q. This is your CV, correct? 2 A. It changes periodically, so I would say 3 this is a version of my CV. 4 Q. This is the version we were provided by you 5 and your counsel as, you know, part of your expert 6 testimony in this case. So I assume that you provided 7 us with whatever was most up to date at the time, 8 correct? 9 A. Pretty much. 10 Q. Yeah? 11 A. Pretty much. 12 Q. All right. Now, on page 44 of your CV, 13 publication 216, do you see that? 14 A. Yes. 15 Q. That's a publication in which Dr. Snowdy, 16 Stephen Snowdy is a coauthor of yours, correct? 17 A. That's correct. 18 Q. All right. And on page 48 is a publication 19 260 is another publication on which Stephen Snowdy is a 20 coauthor, right? 21 MS. MORRISON: Did you say 260? 22 MS. TOKER: Sorry, 260, page 48. 23 MS. MORRISON: Thank you. 24 A. That's correct. 25 Q. And these papers were both published in Page 21

1 peer-reviewed journals, right? 2 A. That's correct. 3 Q. Now, in your opinions, you criticize a 4 portion of Dr. Snowdy's dissertation because that 5 particular portion was not published in a peer-reviewed 6 journal, right? 7 MS. MORRISON: Objection to the form. 8 BY MS. TOKER: 9 Q. Correct? 10 A. I'm not familiar with portion you're 11 referring to. 12 Q. Oh, so you're not aware of your opinion in 13 this case that -- 14 A. No, I'm just not aware of the portion 15 you're referring to. 16 Q. I'm just saying that you, in your opinions 17 in your declaration, criticized a portion of 18 Dr. Snowdy's dissertation, because you said it wasn't 19 published in a peer-reviewed journal. 20 MS. MORRISON: Objection to the form. 21 A. You would have to show it to me so I can 22 affirm that it's the same thing we're talking about. 23 Q. Okay. So you're not sure sitting here that 24 you -- 25 A. I'm just making it clear that if you're Page 22	1 details that they're written. 2 Q. Right. And you also feel they enhance your 3 credibility in the field, the sum total of the book 4 chapters you've written, right? 5 MS. MORRISON: Objection to the form. 6 A. Are you referring to a specific book 7 chapter, or the fact that I've written over 45 or 50 8 book chapters? 9 Q. Yeah, I mean, the fact that you come up 10 with that number and don't distinguish the ones that 11 are refereed from the ones that aren't refereed to just 12 that you think all of them are valuable and enhance 13 your credibility in the field, right? 14 MS. MORRISON: Objection to the form. 15 A. I leave that to the field. 16 Q. You haven't separated out the ones that are 17 peer reviewed from the ones that are not in this list, 18 correct? 19 MS. MORRISON: Objection to the form. 20 A. Again, it's not common practice on the CV 21 to make a distinction between the peer-reviewed book 22 chapter and those that aren't. Some of the higher 23 journals demand review before they accept them. 24 Q. And you list patents on your CV also right? 25 A. It's probably there. Yep, it says patents. Page 24
1 making an assumption, I want to make sure it's a 2 correct assumption. 3 Q. Okay. Let's look at page 55 of your CV. 4 On page 55, you listed a number of book 5 chapters, correct? You begin the list of a number of 6 book chapters, right? 7 A. Um-hum. Yes, correct. 8 Q. Book chapters aren't peer reviewed, are 9 they? 10 A. Some of them are. 11 Q. Well, you don't list these under refereed 12 publications, do you? 13 A. We typically keep book chapters in the 14 tenet of papers that are submitted to journals. 15 Q. So some of them are not in refereed 16 journals, right? 17 A. Some of them are not. 18 Q. All right. And yet you listed them here, 19 even though they are not all peer reviewed, correct? 20 A. It's because it's a CV. It says list all 21 of your contributions. 22 Q. Well, you feel that even your unrefereed 23 book chapters are contributions, right? 24 A. I think that you help people who may not 25 have the time to read the specific papers and the Page 23	1 Q. And patents aren't peer reviewed, are they? 2 A. Not by the academic community. 3 Q. Right. Have you ever chosen not to publish 4 some of your research but to patent the research? 5 A. I don't think I've ever done that. 6 Q. Have you ever patented the research first 7 before publishing? 8 A. Always do that. 9 Q. Okay. 10 A. Let me back up. 11 Always submit for patent before publishing. 12 Whether it gets patent or not, it doesn't impact a 13 publication. 14 Q. All right. I'm going to hand you, 15 Dr. Samulski, what's been marked Exhibit 2027 in this 16 proceeding. 17 (The document referred to was 18 previously marked Deposition Exhibit 19 Number 2027 for identification.) 20 Q. Actually, I'm going to ask the court 21 reporter to hand it to you. 22 Have you seen this before, Dr. Samulski? 23 A. Yes. 24 Q. This is a declaration that you submitted -- 25 A. Yes. Page 25

1 Q. Sorry, Dr. Samulski, let me just get my 2 whole question out before you answer. 3 A. Okay. 4 Q. This is a declaration that you submitted in 5 this proceeding, right? 6 A. That is correct. 7 Q. And that's your signature on the front 8 page? 9 A. That is correct. 10 Q. I'd like to talk a little bit about your 11 discussions of the term tropism in your declaration. 12 And I'd like to turn first to paragraph 38 of your 13 declaration, which is on page 22. 14 Now, let me know when you're there. 15 A. I'm there. 16 Q. Now, in paragraph 38, you state: Tropism 17 refers to the process of the AAV vector reaching a 18 particular tissue or cell type, i.e. the steps that 19 occur before the AAV reaches the cell or tissue. 20 Do you see that? 21 A. I do. 22 Q. All right. And you actually emphasize the 23 word before in this sentence, right? 24 A. I do. 25 Q. Okay. And in this same paragraph, you Page 26	1 to the steps that occur that allow an AAV vector to 2 reach and be taken up by the cell or tissue of 3 interest -- steps one and two in the Figure I describe 4 above; the intracellular steps." 5 A. Where are you at? 6 Q. I'm so sorry. Let's go back. I'm on page 7 44, and let's start one, two, three, the fourth line 8 down. Do you see it says: As I discuss in detail? 9 A. Correct. 10 Q. As I discuss in detail above in paragraph 11 30, tropism relates to the steps that occur that allow 12 an AAV vector to reach and be taken up by the cell or 13 tissue of interest -- steps one and two in the Figure I 14 describe above; the intracellular steps that follow 15 infectivity. Do you see that? 16 A. Um-hum. 17 MS. MORRISON: Objection to the form. 18 I think you missed a word in that last 19 sentence. It's fine, but I just wanted 20 to -- 21 MS. TOKER: Okay. Which word? 22 MS. MORRISON: It says steps that 23 follow affect infectivity. 24 MS. TOKER: Sorry. 25 Q. The intracellular steps that follow affect Page 28
1 contrast tropism with infectivity, which you define as 2 "the steps that occur after the AAV vector reaches the 3 cell or tissue of interest," right? 4 A. Correct. 5 Q. And you emphasize the word after in this 6 sentence, right? 7 A. Correct. 8 Q. And you go on to define the steps that 9 occur after the AAV reaches the cell or tissue of 10 interest as quote -- so this is now what's included in 11 infectivity and not in tropism: "Intake of the AAV 12 vector into the cell, translocation to the nucleus, and 13 expression of the gene of interest," right? 14 A. Correct. 15 Q. All right. So those are the steps that are 16 involved in infectivity but not in tropism, according 17 to you, right? 18 A. Correct. 19 Q. All right. Now, let's take a look at page 20 44 of your declaration, paragraph 73. Actually, 73 21 starts on the prior page 43, but I'm going to be asking 22 you about the part of paragraph 73 on page 44. 23 Let me know when you're at page 44. 24 A. I'm there. 25 Q. Okay. Now, here you say: "Tropism relates Page 27	1 infectivity. Everybody good? Okay, Dr. Samulski? 2 A. Okay. 3 Q. Now, let's -- okay. So when you say here 4 the Figure I describe above, you mean the figure from 5 paragraph 30, right? And we can take a look at 6 paragraph 30 on page 16. 7 A. Correct. 8 Q. And that's the figure there in paragraph 30 9 that you're referring to in paragraph 73 that we just 10 looked at, right? 11 A. Correct. 12 Q. If you remember what we just read, you said 13 that steps one and two in the figure in paragraph 30 14 are included in the term tropism, correct? 15 A. Correct. 16 Q. All right. Let's take a look at the figure 17 then on paragraph 30, okay? Yes? You have that? 18 A. Yeah. 19 Q. Step one is reaching and binding to the 20 cell surface, when the AAV reaches and binds to the 21 cell surface receptor, right? 22 A. Correct. 23 Q. Step two is when the AAV is taken up into 24 the cell and is in an early endosome, right? 25 A. The steps of initiating endocytosis. Page 29

1 Q. Right. So in that case, tropism includes 2 intake into the cell? 3 A. That's steps that initiate endocytosis. It 4 may never happen. 5 Q. Well, it's in the cell? 6 A. Technically it's not. 7 Q. Ah, so you're defining intake as something 8 other than -- 9 A. No, I'm making a very -- 10 Q. Please, Dr. Samulski, let me just finish my 11 question before -- 12 A. Okay. Sure. 13 Q. Okay. All right. Let's in fact -- let's 14 go back to paragraph 73. You say in paragraph 73 on 15 page 44 that you just read: Tropism relates to the 16 steps that occurred that allow an AAV vector to reach 17 and be taken up by the cell, right? 18 A. To reach and be taken up by the cell. 19 Q. Yeah, and that's what's showing in the 20 figure in paragraph 30, it reaches and it's taken up by 21 the cell, right? 22 A. With clarification? 23 Q. No. I'm asking you. I'm not asking you to 24 clarify. I'm asking you what it actually says? 25 A. Reach and taken up by the cell. Page 30	1 and cycle back out and never go any further in an 2 infectious process. It depends on whether you bind to 3 a cell, initiate endocytosis, and it allows it to 4 continue the steps of infectivity. 5 So it's a remedial picture, and it shows 6 that that step is not a complete step but an 7 intermediate step that can either go in or out or cycle 8 in and cycle out. 9 So the word allows is what you want to pay 10 attention to, not whether it's step one or step two. 11 And I'll be happy to go back through it and clarify it, 12 if you need further clarification. 13 Q. No, thank you. So your opinion now -- 14 A. No, my opinion is written as it's stated. 15 It's very clear. 16 Q. Dr. Samulski, I'm going to try not to 17 interrupt you, and I will ask you not to interrupt me. 18 A. I will continue to do the best I can. 19 Q. Thank you. I appreciate that. 20 So when you say in paragraph 38 that 21 infectivity involves intake, and that tropism refers to 22 the process of the steps that occur before the AAV 23 reaches the cell, you are somehow defining endocytosis 24 as something that happens before the AAV reaches the 25 cell, right? Page 32
1 Q. Right. So in your paragraph 73 definition, 2 tropism includes being taken up by the cell, right? 3 MS. MORRISON: Objection to the form. 4 A. Yes. 5 Q. And that's actually shown in the figure in 6 paragraph 30, right? 7 A. It's not shown in the figure. 8 Q. Well, you said it was. 9 A. It's initiating the step of endocytosis. 10 Q. You actually wrote here that allow -- in 11 paragraph 73: Tropism relates to the steps that allow 12 an AAV vector to reach and be taken up by the cell or 13 tissue of interest -- steps one and two. 14 So you define in paragraph 73 steps one and 15 two as reaching and being taken up by the cell, right? 16 MS. MORRISON: Objection to the form. 17 A. I will clarify my statement. Allows. 18 Allows -- 19 Q. First answer my question. 20 A. No. No. That is the question. You're 21 asking if it's taken up. I'm saying if it allows. 22 Allows is what defines whether it's taken up or not. 23 You can bind to a cell. You can initiate endocytosis 24 and not proceed inside the cell. You can bind to a 25 cell. You can initiate endocytosis. You can cycle in Page 31	1 MS. MORRISON: Objection to the form. 2 BY MS. TOKER: 3 Q. Right? 4 A. To one skilled in the art, this would not 5 be a problem. It may be an issue for someone not 6 skilled in the art. 7 Q. Okay. So it's your testimony that a person 8 would understand a step that occurs before the AAV 9 reaches the cell to be the first step in endocytosis? 10 A. I think I'm correct in that response. 11 Q. I'm just asking if that's your testimony? 12 A. I said I think I'm correct in that 13 response. 14 Q. Okay. Now, we can agree that you are 15 saying that tropism does not include steps that occur 16 inside the cell, say gene expression, right? 17 A. Correct. 18 MS. TOKER: I'm going to ask the court 19 reporter to mark this Exhibit 1058. 20 (The document referred to was marked 21 Deposition Exhibit Number 1058 for 22 identification.) 23 Q. Dr. Samulski, the court reporter has just 24 handed you what's been marked Exhibit 1058. 25 Have you seen this document before? Page 33

1 A. I have. 2 Q. All right. And this is a paper on which 3 you are an author, correct? 4 A. That's correct. 5 Q. Published in 2002, right? 6 A. That's correct. 7 Q. And the first author is someone named 8 Rebecca Haberman, correct? 9 A. That's correct. 10 Q. I'm going to refer to this paper as 11 Haberman, is that okay, as we go forward? 12 A. That's acceptable. 13 Q. Now, the title of this paper is Therapeutic 14 Liabilities of In Vivo Viral Vector Tropism: 15 Adeno-Associated Virus Vectors NMDAR1 Antisense, and 16 Focal Seizure Sensitivity, right? 17 A. Correct. 18 Q. For your scientific publications, you do 19 your best to ensure that everything you're publishing 20 is true and correct, right? 21 A. Correct. 22 Q. Just I'll note also Stephen Snowdy is also 23 an author on this paper, right? 24 A. Correct. 25 Q. And he was a graduate student in your lab Page 34	1 by transgene expression? 2 MS. MORRISON: Objection to the form. 3 A. Tropism can be determined by promotor 4 tropism if you're looking for expression. If you're 5 looking for delivery and binding and it could be there 6 but not technically expressed but still define the 7 tropism. 8 Q. So here you're using the word tropism to 9 refer -- 10 A. To promote it. I'm sorry. 11 Q. Let me just get it out, yeah. 12 Here you're using the word tropism to refer 13 to an effect that takes place in the nucleus, right, 14 gene expression? 15 A. So you want -- I want to make sure we're 16 defining tropism of a virus versus tropism of a 17 transgene. There's a difference. 18 Q. Okay. Let's just focus on what we say 19 here. What we say here is you use the word tropism to 20 refer to a function of the virus that takes place in 21 the nucleus? 22 MS. MORRISON: Objection to the form. 23 A. I use the word tropism here as a function 24 of the promotor that identifies an expression of the 25 transgene. Page 36
1 at the time that this was published, right? 2 A. I'm assuming he was still there. I don't 3 remember when he left the lab. 4 Q. Well, he got -- his Ph.D. dissertation is 5 dated 2003, and this is from 2002, right? 6 A. Yep. 7 Q. So he was presumably in your lab at the 8 time? 9 A. Presumably. Sometimes they leave and write 10 their thesis at their next location, job preference. 11 Q. And this is one of the peer-reviewed 12 publications in your CV, right? 13 A. Correct. 14 Q. Now, in the first paragraph under the 15 introduction that's in the left-hand column, you say: 16 AAV preferentially binds neuron in vivo, and this 17 physical property "combined with promotor tropisms," 18 contributes the differential patterns of transduction 19 across various brain structures, right? 20 A. That's correct. 21 Q. And by promotor tropisms, you mean here 22 differences in tropism produced by different promotors 23 regulating transgenes carried by AAV vectors, right? 24 A. That's correct. 25 Q. You're saying AAV tropism can be determined Page 35	1 Q. Yeah, expression of the transgene that 2 takes place, you would say, after the virus enters a 3 cell? 4 MS. MORRISON: Objection to the form. 5 A. After the promotor expresses the 6 transgene -- 7 Q. Well, after the virus enters the cell -- 8 MS. MORRISON: Wait, sorry, if you can 9 let him finish too, before you ask your next 10 question. 11 A. I will yield. Go ahead and ask your 12 question again. 13 Q. My question was, that expression of a 14 transgene is the function of the AAV that happens after 15 the AAV enters the cell? 16 MS. MORRISON: Objection to the form. 17 A. So I'm sorry, I was reading this. Can you 18 say that one more time? 19 Q. Yes. My question was, that expression of a 20 transgene is the function of the AAV that occurs after 21 the AAV enters the cell? 22 MS. MORRISON: Objection to the form. 23 BY MS. TOKER: 24 Q. Is a function, I'm sorry, is a function of 25 AAV that occurs after the AAV enters the cell, right? Page 37

1 MS. MORRISON: Same objection. 2 A. I'm still not following. 3 Q. Okay. So here you're talking about tropism 4 as being something that can involve a function of AAV 5 that happens after the AAV enters the cell, right? 6 MS. MORRISON: Objection to the form. 7 A. There's confusion in how you're wording it 8 to me. I can reword it in the way I think it's being 9 said, and we can go on from there. But I'm not 10 following the nuances of what you're asking. I'm 11 sorry. 12 Q. So in your declaration, you define tropism 13 as functions that occur before the virus reaches the 14 cell -- let me finish. And here in your paper you're 15 talking about tropism as a function that happens after 16 the virus reaches the cell. 17 MS. MORRISON: Objection to the form. 18 A. So the word tropism is being used 19 differently for expression than it is for the virus 20 infectivity. 21 Q. So when you defined tropism in your 22 declaration, you weren't trying to cover all the 23 meanings of tropism? 24 A. Correct. 25 Q. So there are other meanings of tropism Page 38	1 Q. Dr. Samulski, the court reporter has just 2 handed you what's been marked Exhibit 1007 in this 3 proceeding. You're familiar with this document, right? 4 A. I'm aware of this document. 5 Q. Okay. This is a United States Patent 6 Application Publication US 2003/0138772, right? 7 A. Sorry, I didn't follow the numbers, but I 8 assume that they're right. 9 Q. Okay. Just for purposes of our discussion, 10 I'm going to refer to this as the '772 publication, 11 okay? 12 A. Okay. 13 Q. Now, the '772 publication uses the term 14 tropism -- 15 MS. MORRISON: Dr. Samulski, don't 16 write on the document. 17 A. I was going to write '772 on there. 18 Q. The '772 publication uses the term tropism, 19 right? 20 A. I haven't seen it firsthand here, but I 21 will make an assumption you're correct. 22 Q. So when you formulated your opinions in 23 this case and gave a definition of the term tropism, 24 you did not consider the use of the term tropism in the 25 '772, correct? Page 40
1 beyond the meaning that you defined? 2 MS. MORRISON: Objection to the form. 3 A. There are other ways to define tropism 4 other than by the virus specifically binding to step 5 one and two. 6 Q. Okay. And in your paper here, you use 7 tropism in a different way? 8 A. We use tropism as based on the promotor 9 expression. 10 Q. Right. Okay. And in fact, this use -- let 11 me strike that. 12 This paper looked at differences in tropism 13 between two different promoter constructs that 14 regulated an AAV transgene, right? 15 A. Correct. 16 Q. And you know that basically means that each 17 construct had a different regulatory sequence that was 18 directing transcription of the transgene? 19 A. Correct. 20 Q. Okay. Let's take a look at another 21 exhibit. I am going to ask the court reporter to hand 22 you what has been marked Exhibit 1007. 23 (The document referred to was 24 previously marked Deposition Exhibit 25 Number 1007 for identification.) Page 39	1 MS. MORRISON: Objection to the form. 2 A. So I will respond to the word tropism used 3 in our community is used differently by the clinical 4 component of the viral infection, and it's used 5 differently by a virologist that's dealing with an 6 infectious step of a viral component, and it's used 7 differently by a molecular virologist or a threonine 8 therapist who refers to it as a way of nomenclature for 9 gene expression. 10 Q. Right. So I'll ask again. In formulating 11 your opinions in this case, and coming up with the 12 definition of the term tropism, you didn't consider how 13 the term was used in the '772 publication, correct? 14 MS. MORRISON: Objection to the form. 15 A. I would have to look at this specifically 16 to see what they said, to see if it's consistent with 17 mine. 18 Q. Right. But I'm asking you about what 19 happened before you formulated your opinion in this 20 case. And you just told me that you had not in fact 21 considered the definition or the use of the term 22 tropism in the '772 publication, correct? 23 MS. MORRISON: Objection to the form. 24 A. I'd have to read it to make sure. 25 Q. Well, I'm just asking you -- we will look Page 41

1 at it, Dr. Samulski. But if you could focus on my 2 question. I'm just asking you about what you did in 3 formulating your opinions in this case? We'll look at 4 the '772 in a minute. And you've told me here that you 5 didn't consider how tropism is used in the '772 6 publication -- 7 A. I don't think I said that. 8 Q. Well, have you considered it? 9 A. Again, I'd have to look through it again to 10 see if it's consistent with my comments of tropism. 11 Q. But sitting here, without looking at it 12 again, you don't know whether what -- 13 A. I don't remember. 14 Q. You don't remember whether the definition 15 that you gave in your declaration is consistent with 16 how it's used here, correct? 17 A. That would be correct. 18 Q. Okay. And it wasn't important to you in 19 coming up with your definition whether or not it was 20 consistent with the '772 publication, right? 21 MS. MORRISON: Objection to the form. 22 A. I don't know about your comment about 23 important. It would be someone skilled in the art in 24 the space would have formulated ideas of tropism based 25 on their training and experience. And as I mentioned a Page 42	1 here may be similar to my experience. 2 Q. It may be, and we'll look at that in a 3 minute, but that's not my question. My question isn't 4 whether coincidentally it's the same. My question is 5 that in formulating your definition coming up, you 6 know, with Dr. Samulski's definition of tropism and 7 writing that down and signing it, you did not consider 8 how the term is used in the '772 publication? 9 A. Oh, I think I would have. I don't see how 10 I couldn't. They're both related to viruses. 11 Q. Oh, so your definition -- you would have 12 changed your definition if the '772 -- 13 A. Not changed. You're asking the question 14 would I have considered it, and I'm answering -- you 15 asked me would I consider it because it's viral in 16 definition. 17 Q. But you wouldn't necessarily have changed 18 your definition. Your definition was based on your 19 experience, and if the '772 said something else would 20 still offer your definition, right? 21 MS. MORRISON: Objection to the form. 22 A. I think I would not have defined my 23 definition by their definition, but I would have 24 considered whatever they proposed from a virus 25 perspective, whether it was consistent or inconsistent Page 44
1 virologist has a different, slightly different 2 definition of tropism than does a clinical person 3 compared to a molecular virologist and a gene 4 therapist. 5 Q. And you formulated your definition based on 6 your experience in the field, right? 7 A. Based on my experience in the field. 8 Q. And you didn't formulate your definition 9 based on however the term is used in the '772 10 publication? 11 A. I'm sure I did not use it as my sole 12 definition of tropism. 13 Q. Right. Your definition was based on your 14 own experience, not how it was used in the '772, 15 correct? 16 MS. MORRISON: Objection to the form. 17 A. There's probably commonality in the two, 18 because they're talking about viruses. 19 Q. I'm just asking -- that may be, but my 20 question is a little different. My question is just 21 that your definition of tropism that you offered in 22 your declaration was based on your experience and not 23 based on anything about how the term was used in the 24 '772? 25 A. I caution you because how the term is used Page 43	1 with my definition. 2 Q. Okay. And then you would have offered your 3 definition based on your experience in this field and 4 your expertise in this field, right? 5 MS. MORRISON: Objection to the form. 6 A. Correct. 7 Q. So let's take a look at what the '772 8 publication actually says about tropism. 9 I would like to look, please, at example 9 10 on page 133. And I'm going to specify that when I'm 11 referring to page numbers, I'm referring to the little 12 page numbers at the bottom, which are the ones they say 13 Sarepta Exhibit 1007, page something. And those are 14 the numbers I'm referring to. And I'd like to look at 15 page 133, example 9. Let me know when you're there. 16 A. I'm there. 17 Q. Okay. And do you see under example 9 18 there's a heading that says tissue tropism studies? 19 A. I do see that. 20 Q. Okay. After this heading, tissue tropism 21 studies, the '772 publication describes, right, some of 22 these studies that are used as tissue tropism studies, 23 correct? 24 MS. MORRISON: Objection to the form. 25 A. Correct. Page 45

1 Q. And it describes these studies. So now I'm 2 looking at the paragraph that is indicated as bracket 3 0253, okay? Do you see that? You do? 4 A. I do. 5 Q. Okay. And then looking right around the 6 middle of the paragraph, and it talks about these 7 studies as gene transfer studies targeting three 8 different tissues; liver, lung and muscle to screen for 9 tissue tropism of a particular AAV construct. 10 Do you see that? 11 A. I do see that. 12 Q. Okay. Now, these studies were designed to 13 evaluate the tropism of different serotypes of AAV in 14 three different tissues: Liver, lung and muscle, 15 correct? 16 MS. MORRISON: Objection to the form. 17 A. I see that. 18 Q. And now if we look at the experiments there 19 that are shown, for example in Table 8, these studies 20 measured the amount of reporter protein produced in 21 each different type of tissue per mL of serum from the 22 animals infected with that AAV serum type, right? 23 A. That's correct. 24 Q. These tropism studies therefore mentioned 25 gene expression to evaluate tropism, right? Page 46	1 right? 2 A. So they are using tropism in another 3 manner. 4 Q. Yes, they are using tropism in a different 5 way from the way you use it in your declaration? 6 A. Correct. 7 Q. Okay. Now, let's also look at the 8 discussion here. It doesn't just discuss tropism, it 9 discusses transduction, right? It says, for example, 10 in paragraph 0257, which is in the right-hand column, 11 it says that: Interestingly, performance of clone 12 44.2, and you understand clone 44.2 to be AAVrh10, 13 right? 14 A. Um-hum, yes. 15 Q. Performance of clone 44.2 in liver and 16 muscle-directed gene transfer was also outstanding, 17 close to that of the best liver transducer, AAV8, and 18 the best transducer, AAV1, correct? 19 A. Correct. 20 Q. So here they're referencing these tissue 21 tropism studies and saying that they also reveal the 22 ability of these different serum types to transduce 23 these tissues, correct? 24 A. So be careful, not tissue tropism studies, 25 tissue transgene expression studies, that they call Page 48
1 A. That's correct. 2 Q. And your definitions of tropism that you 3 gave in your declaration exclude gene expression from 4 the term tropism, right? 5 A. From the term tropism. But to get to this 6 definition of tropism, you need term one and two that I 7 describe. 8 Q. To get to this, you actually have to 9 look -- 10 MS. MORRISON: Wait, sorry. 11 A. To get to this term of tropism of gene 12 expression, you have to achieve step one and two of my 13 definition of tropism. 14 Q. Right, but this gene -- 15 A. It's not possible to get from one to the 16 other. 17 Q. Okay. But this definition of tropism that 18 involves gene expression would be what you call 19 infectivity? 20 MS. MORRISON: Objection to the form. 21 A. No, it would not. 22 Q. Well, you exclude gene expression from the 23 definition of a tropism, right? 24 A. Yes. 25 Q. And these are gene expression studies, Page 47	1 tropism. 2 Q. Okay. So they call them tropism studies? 3 A. Correct. 4 Q. You call them something else, but they call 5 them tropism, right? 6 A. Correct. 7 Q. So we have your definition in your 8 declaration. We have the definition that here remains, 9 and the declaration that the '772 publication reads, 10 right? 11 MS. MORRISON: Objection to the form. 12 A. The correlation that you're drawing 13 together are not three different definitions. They're 14 all dependent on step one and two. So they are defined 15 by my definition of tropism. You can't get to 16 Haberman's or transduction tropisms without step one 17 and two. 18 Q. Right. But you expressly exclude the 19 interior steps, including gene expression, from your 20 definition of tropism? 21 A. I separate them, as you can have 22 infectivity that succeed or infectivity that will fail, 23 but that does not include the tropism for the cell 24 type. 25 Q. Right. So your definition doesn't include Page 49

1 gene expression, right? 2 MS. MORRISON: Objection to the form. 3 A. My definition says gene expression cannot 4 take place without my definition. But you can have the 5 failure of gene expression and still have tropism. 6 Q. Okay. 7 A. Does that make sense? Are you following 8 what I'm saying? 9 Q. Well, I'm just saying that -- 10 A. You can't open a door without pushing or 11 holding the handle, but the door can be open. If the 12 door is open, it had to go through opening the handle. 13 Q. Right. But I could define tropism as 14 opening the door and not entering the room? 15 A. No. You could say that. That's what I'm 16 saying. That's exactly what I'm saying. 17 Q. And these folks are saying no, tropism can 18 describe the stuff that happens in the room, right? 19 MS. MORRISON: Objection to the form. 20 A. They're using the word tropism with respect 21 to their assay, and their assay is measuring transgene 22 expression. 23 Q. Well, they and you in Haberman were using 24 it in reference to gene expression to an activity that 25 happens in the room, right? Page 50	1 A. Yes. 2 Q. And so you're arguing here that the Snowdy 3 dissertation relates to nuclear localization and 4 therefore in your definition it relations to 5 infectivity rather than tropism, right? 6 A. That's correct. 7 Q. All right. And this argument relies on the 8 definition of tropism that you've offered in your 9 declaration based on your experience and expertise, 10 correct? 11 MS. MORRISON: Objection to form. 12 A. That's correct. 13 Q. All right. Now, if you were to use a 14 different definition of tropism, right, that argument 15 would no longer be the case that nuclear localization 16 would be unrelated to tropism? 17 A. I don't think I'm following your question. 18 Q. Okay. So your argument is that 19 Dr. Snowdy's dissertation is unrelated to tropism, 20 right? 21 A. Correct. 22 Q. That depends on your definition of tropism, 23 that tropism stops at the door, right? 24 MS. MORRISON: Objection to the form. 25 A. No. I think in the case of tropism, for Page 52
1 MS. MORRISON: Objection to the form. 2 A. We were referencing it again to gene 3 expression, tropism gene expression. 4 Q. Right. Not just to opening the door, 5 right? 6 A. Not just to opening the door. 7 Q. Right. Okay. Now, you say with respect to 8 Dr. Snowdy's dissertation that -- I guess let's just 9 look at that. It will make it easier. 10 Let's actually go back to your declaration 11 first. 12 So I'd like to look at your declaration, 13 which is Exhibit 2027, and I'd like to look back to 14 paragraph 73, which you looked at before on page 43. 15 So I'm going to focus you on paragraph 73, 16 but this time the portion on paragraph 73 starting on 17 page 43, okay? Now, let me know when you're there. 18 A. I'm there. I think I'm there. 19 Q. Okay. So you're talking about Dr. Snowdy's 20 dissertation, and you're saying this in the first 21 sentence you're saying: Additionally, and perhaps more 22 importantly, this study is unrelated to improving or 23 changing tissue tropism, which is the sole motivation 24 Sarepta identifies or combining Snowdy with the '772 25 publication. Do you see that? Page 51	1 tissue tropism, we're not working with tissue, we're 2 working with a cell line. So we're only looking at the 3 mechanics that take place within a cell line, not an 4 organ, not an in vivo setting, and nothing to do with 5 whether or not you would describe tropism for that 6 virus to go to the brain or the liver or the muscle. 7 Q. Let's look at page 44, same paragraph. You 8 say: In my view, nuclear mobilization relates to 9 infectivity that is unrelated to the tissues-specific 10 tropism driving Sarepta's motivation to combine, right? 11 A. Correct. 12 Q. So you're classifying nuclear localization 13 is something that relates to infectivity, not tropism 14 here, right? 15 A. Correct. 16 Q. Right. But that depends on your special 17 definition of tropism as stopping when you enter -- you 18 know, stopping at the door of the cell, right? 19 MS. MORRISON: Objection to the form. 20 A. No. I think you're misreading my 21 definition. When you study cells, you're not studying 22 tissues. So tissue is where tropism is attached to a 23 virus' fingerprint, it's virology. Will it bind? And 24 will it be taken up in tissue. 25 In this case, we're looking at steps that Page 53

1 take place during the infectious process of the virus 2 to get from the cytoplasm to the nucleus. 3 Q. Yeah, but that's not what you say right 4 here. What you say here is nuclear localization 5 relates to infectivity, correct? 6 A. Correct. 7 Q. But that's your definition of infectivity, 8 right? 9 MS. MORRISON: Objection to the form. 10 A. I think my definition is pretty much what 11 the field builds its historical premise on. 12 Q. Yeah, but your field in your Haberman 13 paper, right, that paper talks about tropism as things 14 that affects functions in the nucleus, that's related 15 to functions in the nucleus? 16 A. No. I think you're wrong again. That 17 paper reflects expression as a way of defining tropism. 18 Q. Okay. And that's a function that takes 19 place inside the cell expression, right? 20 A. Inside of a tissue. 21 Q. So in your view -- okay. Let's move on. 22 A. In my view, there are multiple scenarios 23 how people use tropism. 24 The Polio virus tropism is it's 25 neurological, but the virus infects the cells that line Page 54	1 know, how the virus may later operate in the organism, 2 is what you're saying? And you've never used an in 3 vitro cell culture study to provide information about 4 how a serotype may work in an organism, is that your 5 testimony? 6 MS. MORRISON: Objection to the form. 7 A. What I am saying is that I do not use cell 8 culture to define tropism, because it's one cell type. 9 Q. That's not my question. 10 A. But that's my answer. 11 Q. But my question though, I'm going to ask it 12 again, is you agree that cell culture studies can 13 provide information that can predict how a serotype may 14 function in an organism? 15 MS. MORRISON: Objection to the form. 16 A. I disagree. 17 Q. You never use cell culture? 18 A. I do. 19 Q. What for? 20 A. Let me give you a clear example. I use 21 cell culture to show that AAV would not infect an 22 astrocyte on a petri dish. That would have said AAV 23 would not infect astrocytes. But when I went in vivo, 24 AAV infected astrocytes. So tissue cultures were in no 25 way predictable of what was happening in vivo. Page 56
1 the intestinal in order to get inside of the body. 2 That is not the tropism of the virus. It's the neuro 3 cells that it replicates and kills that defines its 4 clinical tropism. 5 So you have a cell type that's required to 6 get through the door in order to go to the next step 7 that allows it to have a tropism for its clinical 8 symptoms of killing neurons. 9 In each and every one of these studies, 10 people are defining variations of tropism by gene 11 expression, gene expression in tissue culture, gene 12 expression in cells, gene expression in animals. And 13 the tropism that I'm referring to always has to be 14 related to an animal, not a cell culture-based system, 15 because there's no distinction between am I choosing a 16 lung cell versus a liver cell? I only have one cell 17 population on the plate. And so I'm looking at steps 18 of infectivity, not tropism. 19 Q. You're not suggesting that cell culture 20 studies can't provide information that could be related 21 to tropism, right? 22 MS. MORRISON: Objection to the form. 23 A. I am saying definitively that cell culture 24 cannot define things that are related to tropism. 25 Q. It can't provide information about, you Page 55	1 Q. Why did you do the experiment with 2 astrocytes and cultures? 3 A. Because those were the cells that we had 4 available. 5 Q. Well, sometimes when you infect the cell 6 and culture it, it does speak to how it's going to 7 infect -- 8 A. And sometimes it doesn't. 9 Q. Fair enough. You have to test. But, but 10 you still carry out the cell culture experiment because 11 it gives you information that you will later test to 12 see if it's reflective of how the AAV will behave in 13 the organism? 14 MS. MORRISON: Objection to the form. 15 A. So I think I will help you better 16 understand why I use tissue culture. I use a cell type 17 that doesn't represent any organ in the body but simply 18 lets us look at the steps of infectivity. And those 19 cells are promiscuous for other reasons; and, 20 therefore, they allow us to study steps related to the 21 molecular infectivity steps. 22 Q. Okay. Let's shift gears a little bit and 23 talk about your opinions on single amino acid changes 24 as a way to change tropism as of the time around 2005. 25 Let's turn back to your declaration, Page 57

1 paragraph 53, which is page 32. Now, are you there? 2 A. I'm there. 3 Q. All right. You say in paragraph 53, you 4 say: At the outset, I note that the '772 publication 5 is silent as to using single amino acid substitutions 6 to alter tissue tropism. Do you see that? 7 A. Um-hum. 8 Q. Okay. Let's take a look back at the '772 9 publication, and I would like to turn, please, to page 10 113 and to look at paragraph 0072. Okay. Let me know 11 when you're at paragraph -- 12 A. I'm there. 13 Q. Okay. Now, the second sentence of 14 paragraph 0072 refers to the fact that AAV7 has an 15 unusual GTG start code on it. Do you see that? 16 A. I do. 17 Q. All right. Now, you're aware that the 18 normal start code on is ATG, correct? 19 A. ACG. 20 Q. ACG? 21 A. It's ATG followed by ACG followed by AUG. 22 Q. Okay. All right. So you're aware though 23 that the normal code on it is ATG, right? 24 A. It depends on which way you start. PP1 is 25 ATG. Page 58	1 to find you where this is. If you look down at sort of 2 the bottom half of the paragraph, there's a sentence 3 that says: Thus, the inventors anticipate. Do you see 4 that? 5 MS. MORRISON: Counsel, you're still 6 on paragraph 72 of Exhibit 1007? 7 MS. TOKER: Yeah, that's correct. 8 Q. Count up from the bottom, 1, 2, 3, 4, 5, 6, 9 7, 8, 9, 10, 11, 12, 13 lines from the bottom, sorry, 10 it says however, and then the next sentence says thus. 11 Let me know -- 12 A. Thus, you anticipate? 13 Q. Unfortunately, no line numbers to help. So 14 we're just counting here. Thus, the inventors 15 anticipate it says this is desirable, let's just say 16 the investors anticipate that it is desirable to alter 17 the start code on of the VP3 of other AAV serotypes to 18 contain this rare GTG start code. Do you see that? 19 A. I do see that. 20 Q. And we just agreed, I mean VP3 is one of 21 the AAV capsid proteins, right? 22 A. That is correct. 23 Q. So the inventors of the '772 publication 24 here are recommending that you might want to alter the 25 normal start code on a VP3, which is ATG to GTG, right? Page 60
1 Q. Right. 2 A. PP2 is ACG, and it's a weakened start code 3 for methionine ACG, and VP3 is an ATC or AUG, which is 4 a start code. And this one they're noting that there's 5 an unusual start code on GTG, which is a variant of 6 ACG, which is used by most of the other serotypes. 7 Q. Okay. So but most of the serotypes at VP3 8 have an ATG start code on it? 9 A. For the start of VP1? 10 Q. VP3. 11 A. VP3 have an ATG, correct. 12 Q. Right. And that's what this is talking 13 about. It's talking about that VP3 of AAV7 has a GTG 14 instead of an ATG, correct? 15 A. Okay. 16 Q. Agree? 17 A. From what you said, yes. 18 Q. All right. And we agree that ATG codes for 19 the amino acid methionine, right? 20 A. Correct. 21 Q. And we also agree that GTG codes for 22 different amino acid valine, right? 23 A. Correct. 24 Q. Okay. Now, the '772 publication here in 25 paragraph 0072 goes on to state, and I'm going to try Page 59	1 A. That's what they are suggesting. 2 Q. And a person of ordinary skill in the art 3 would also understand that this mutation to GTG would 4 be present in VP1 and VP2 proteins in that mutant 5 capsid, right? 6 A. That's correct. 7 Q. So the VP1 and VP2 proteins of this mutant 8 capsid would each have a single amino acid mutation at 9 that position where methionine is mutated to valine, 10 right? 11 MS. MORRISON: Objection to the form. 12 A. It would if you're comparing it to the 13 other serotypes. 14 Q. Correct. Correct. So what they're saying 15 is -- let me -- sorry, let me back up. I'm not sure I 16 understand what you mean. 17 My question is, they're recommending that 18 you might take a serotype other than AAV7 and make this 19 mutation, correct? 20 MS. MORRISON: Objection to the form. 21 A. They have an anomaly that's inconsistent 22 with the other serotypes. It seems to be distinct to 23 AV7, and they're making the proposal that you might be 24 able to do this to other serotypes. 25 Q. Right. And when you say doing this, you Page 61

1 mean mutating the ATG start code on a VP3 to GTG? 2 A. That's correct. 3 Q. All right. And if you made that mutation 4 in another serotype, let's pick your favorite serotype, 5 AAV2 let's just say, if you made that mutation in AAV2, 6 you would be changing that methionine at least in the 7 VP1 and VP2 proteins of AAV2 to valine, right? 8 A. Correct. 9 Q. Okay. And if that's the only mutation 10 made, that would be a single amino acid change, right? 11 A. For those two proteins. 12 Q. Well, it would be a single amino acid 13 change in the capsid, right -- 14 MS. MORRISON: Objection to the form. 15 BY MS. TOKER: 16 Q. -- of AAV2? 17 A. No, because it may not make -- allow you to 18 make VP3, because VP3 starts with an ATG. And if you 19 use a GTG, it may not make VP3. So you could have a 20 single amino acid change of VP1 and VP2 but never 21 express VP3. If you don't express VP3, you don't make 22 a virus. 23 Q. Right. But what we're reading here is what 24 the '772 publication inventors are recommending, which 25 is that you want to make that mutation because it might Page 62	1 hypothesis, I don't consider that a teaching. I 2 consider that a hypothesis. It has to be tested in 3 order to be proven if it's true or not. 4 Q. Okay. But it is suggested here, and you 5 didn't consider this as part of your opinion, right? 6 MS. MORRISON: Objection to the form. 7 A. I had no reason to consider it as part of 8 my -- 9 Q. Okay. So -- 10 A. It's a hypothesis I don't believe in. 11 Q. You don't agree with it, so you didn't 12 consider it as part of your opinion, right? 13 MS. MORRISON: Objection to the form. 14 A. I'll back up to you help me understand my 15 explanation. 16 Start code on start with ATG. There is a 17 cosec(sic) sequence that's typically surrounding an 18 ATG, which has nucleotides flanking on either side. 19 For it not to be an ATG, it is called a slippage or a 20 less preferred start site. 21 ACG is one of those preferred start sites, 22 that's how VP2 is made. It's a slippage mechanism. 23 GTG is not typically or usually explained 24 as a major start site for translation. 25 So for GTG to work in AAV7, there's no Page 64
1 be beneficial, right? 2 MS. MORRISON: Objection to the form. 3 A. I think they're hypothesizing an anomaly 4 that they see in 7 and making an assumption that it may 5 work in another serotype. 6 Q. Yeah, right, exactly. And if it "worked" 7 in another serotype, you would then have a capsid with 8 a single amino acid mutation at that GTG, ATG, right? 9 A. If if and buts were candy and nuts, we all 10 would have a Merry Christmas. It's a hypothetical. It 11 has no meaning. We don't know if that's going to work 12 or not. 13 Q. Right. But it's what we call a disclosure. 14 This is what they're suggesting. This is a teaching. 15 You may not agree with it, but this is what the '772 16 publication is suggesting? 17 A. It's suggesting. 18 Q. Right? It's teaching that -- 19 A. It's not teaching, it's suggesting. 20 Teaching means it works and you demonstrate it. 21 Q. Okay. So it's your understanding that 22 disclosures like this you're free to ignore them in 23 formulating your opinions in this case? 24 MS. MORRISON: Objection to the form. 25 A. As a researcher who looks at someone's Page 63	1 mechanism that's taking place. Moving that single 2 amino acid doesn't mean that you move over the 3 mechanism to get translation to take place, but it's 4 flanking sequences that are necessary and involved in 5 order for it to work. 6 So it's a hypothesis that is hypothesize, 7 how to get to the moon without using spaceships. You 8 can do that too. It's a hypothesis. 9 Q. Yeah, so this is just as irrelevant to your 10 opinion as a spaceship to the moon, right? 11 MS. MORRISON: Objection. 12 A. An hypothesis. 13 Q. Yeah, irrelevant for your opinion, correct? 14 MS. MORRISON: Objection to the form. 15 A. It's -- it's -- in my opinion, it's not 16 related to what we're discussing. 17 Q. Okay. Now -- and you didn't consider it as 18 part of your opinion? 19 MS. MORRISON: Objection to the form. 20 A. I looked at that and saw it as a 21 hypothesis. 22 Q. Right. So when you said that the '772 is 23 silent as to single amino acid mutations, you just 24 don't believe this, so you didn't include it in your 25 opinion, right? Page 65

1 MS. MORRISON: Objection, 2 mischaracterizes the declaration. 3 A. I'm not following your '772 description. 4 Q. All right. Let's finish looking at this 5 paragraph and what the inventors here are saying about 6 this single amino acid mutation. 7 They're saying, and I'm going to go back to 8 that sentence: Thus the inventors anticipate that it 9 is desirable to alter the start code on of the VP3 of 10 other AAV serotypes to contain this rare GTG start code 11 on in order to improve packaging efficiency, to alter 12 the varying structure and/or to alter location of 13 epitomes, e.g. neutralizing antibody epitomes of other 14 AAV serotypes. Do you see that? 15 A. No, I don't. 16 Q. Oh, I'm sorry. I had gone back to that 17 same sentence thus that we counted, we counted up to 18 before, so if we want to count it's 1, 2, 3, 4, 5, 6, 19 7, 8, 9, 10, 11, 11 up from the bottom, thus. 20 A. Thus? 21 Q. Yeah, you found it? 22 A. Yeah. 23 Q. So it says: Thus, the inventors 24 anticipate, you see all that, and we had read that 25 before, you know, that it may be desirable, right, to Page 66	1 ahead. 2 Q. Well, it depends, right? It depends on -- 3 A. It depends on -- 4 Q. -- on doing an experiment and finding out 5 whether or not in fact whether or not it's on the 6 surface, it could alter the surface, agreed? 7 A. It could potentially alter the surface. 8 Q. Mutations even that are not on the surface 9 can alter the surface of the capsid, you agree with 10 that, right, doctor? 11 A. It could potentially alter the surface. 12 Q. Of course. So it doesn't really matter, 13 for our purposes here, whether or not it's on the 14 surface or not, the point is the '772 publication is 15 suggesting that you might want to make this mutation, 16 because it might alter the surface, and particularly 17 alter the location of epitopes on the surface, right? 18 MS. MORRISON: Objection to the form. 19 A. That's what they're proposing. 20 Q. And even under your definition of tropism, 21 which stops at opening the door, altering an epitope on 22 the surface could affect whether or not the virus can 23 open the door, right? 24 MS. MORRISON: Objection to the form. 25 A. And based on the way you're describing it, Page 68
1 make this amino acid -- to make this change of ATG to 2 GTG, right? 3 A. Um-hum. 4 Q. And then they explain why you might want to 5 do it, and they say: In order to improve packaging 6 efficiency, to alter the variant structure and/or to 7 alter location of epitopes, e.g. neutralizing antibody 8 epitopes of other AAV serotypes. Do you see that? 9 A. I see that. 10 Q. So they're suggesting that making this 11 mutation, right, could have effects on things such as 12 the location of epitopes on the surface of the capsid, 13 correct? 14 A. That's what they're saying. 15 Q. Right. So they're saying here that a 16 single amino acid change could affect the location of 17 epitopes on the surface of the capsid, right? 18 MS. MORRISON: Objection to the form. 19 A. That's what they're saying. 20 Q. And you agree with me that altering the 21 location of epitopes on the surface of the capsids 22 could affect tropism, even under your definition, 23 right? 24 A. So on the surface the AAG or the GTG is not 25 on the surface. That's embedded in the virus, but go Page 67	1 that's correct. 2 Q. Okay. So, that's fine. 3 MS. MORRISON: Is this a good time for 4 a break? 5 MS. TOKER: Yes, sure. Let's take a 6 break. Ten minutes? 7 MS. MORRISON: Sure. 8 MS. TOKER: Go off. 9 THE VIDEOGRAPHER: The time is 10:38. 10 We're off the record. 11 (Recess.) 12 THE VIDEOGRAPHER: The time is 10:54. 13 We're on the record. 14 BY MS. TOKER: 15 Q. All right. Dr. Samulski, did you speak 16 with your counsel about the substance of the deposition 17 during the break? 18 A. No. 19 Q. Okay. Now, I'd like to turn back to your 20 declaration again, please, Exhibit 2027, and I would 21 like to look at page 39, please. Looking at paragraph 22 65. Let me know when you're there. 23 A. I'm there. 24 Q. Okay, thanks. Now, you argue in your 25 declaration, and we're looking at one paragraph where Page 69

1 you make this argument that a person of ordinary skill 2 in the art seeking to target delivery of a gene to 3 muscle would have started with AAV7 rather than 4 modifying AAVrh10, right? 5 A. That's correct. 6 Q. Now, you point -- let's see, I believe that 7 you point on the previous page in paragraph 63, you 8 look at Table 6. 9 A. Wait a minute, paragraph 63? 10 Q. 63 on the prior page, yeah, I'm sorry, page 11 38, so I'm going back. And I'm looking at paragraph 63 12 where you point to Table 6 in your discussion of the 13 performance of AAV7 in muscle, right? 14 A. Correct. 15 Q. Okay. And you say: These data show the 16 performance of AAV7 in muscle is approximately three 17 times better than the previously known best muscle 18 transducer in AAV1, right? 19 A. Correct. 20 Q. Now, in Table 6 that you produce right 21 there, in that same page 38, right, that's Table 6 from 22 the '772 publication, correct? 23 A. Correct. 24 Q. All right. Those experiments in Table 6 do 25 not include an experiment involving AAVrh10, right? Page 70	1 Q. And we had discussed Table 8 a little bit 2 before. Those are the tissue tropism studies of the 3 different serotypes in three different tissues, 4 correct, correct? 5 A. Correct. 6 Q. Measuring the expression of the marker 7 gene, right? 8 A. Correct. 9 Q. And this table does include experiments 10 that involve AAVrh10, correct? 11 A. But not rh7. 12 Q. Right, not AAV7, right? 13 A. Correct. 14 Q. All right. So this table involves AAVrh10, 15 and it also involves AAV1, right, but not AAV7, 16 correct? 17 A. Correct. 18 Q. So, therefore, to draw any comparison 19 between AAV7 and AAVrh10, based on the data presented 20 in the '772 publication, you would have to be looking 21 across experiments, because there is no single set of 22 experiments that actually compares AAVrh10 to AAV7 23 directly, right? 24 MS. MORRISON: Objection to the form. 25 A. Based on the way they present it, there's Page 72
1 A. That's correct. 2 Q. So there's no comparison in Table 6 between 3 AAV7 and AAVrh10, right? 4 A. That's correct. 5 Q. Let's look at Table 8 of the '772 6 publication, which is page 133. 7 A. Is this the patent? 8 Q. The '772 publication, it's a publication, 9 1007. 10 A. Okay. 11 Q. You're aware this is a patent publication, 12 Dr. Samulski? 13 A. Correct. 14 Q. Okay. If I refer to it as the '772, you'll 15 know? 16 A. I am now. I was going to write it on 17 there, but. 18 Q. Sorry. Your counsel said no. 19 A. What page are you on? 20 Q. And I am now page 133, which is the same 21 one we had looked at at one point for Example 9. 22 A. Yes. 23 Q. Okay. You see that in Example 9, there is 24 a Table 8, right? 25 A. Yes, Example 9, Table 8. Page 71	1 no example where they do them side by side. 2 Q. Right. And in your experience as a 3 scientist, you would agree that it's difficult to 4 compare the, you know, outcome of an experiment -- let 5 me rephrase that. 6 As a scientist, you would agree that when 7 you haven't tested two things together in the same 8 experiment, and instead you've tested one in one 9 experiment and another in another experiment, it's 10 difficult to draw conclusions, right? 11 MS. MORRISON: Objection to the form. 12 A. I would not draw that conclusion. I would 13 use AV8, which seems to be the gold standard in each 14 one of these as a barometer of how accurate are they 15 consistent between experiments. 16 Q. So in your opinion, you don't, in your own 17 practice, test things in the same experiments, under 18 the same conditions to determine how they perform 19 relative to one another, right? 20 MS. MORRISON: Objection to the form. 21 A. I do multiple experiments to confirm 22 things, but I don't necessarily have to have everything 23 in the same experiments. 24 Q. Would you prefer to have things in the same 25 experiment to compare them? Page 73

1 A. If I had a concern, I would have everything 2 in the same experiment. 3 Q. Okay. And in fact, if we look at paragraph 4 257, which is in the right-hand column. 5 A. Yes. 6 Q. It compares -- it compares the performance 7 of AAVrh10 with the performance of AAV8 and AAV1, 8 right? 9 A. Wait a minute. 10 Q. I'm sorry, take your time. 11 A. You're talking about 257? 12 Q. Yes. In the right-hand column of page 133. 13 A. Interestingly performance of clone 442? 14 Q. That paragraph. So, yeah, take a look at 15 that, and then let me know -- I can repeat my question 16 too, if it's helpful. 17 (Pause.) 18 A. So go ahead. I have read it. 19 Q. My question is, in this paragraph, the '772 20 publication compares the performance of AAVrh10, which 21 we've agreed is clone 44.2, right? 22 A. Okay. 23 Q. Sorry, I'll back up. It talks about clone 24 44.2, but we agree, right, that clone 44.2 is AAVrh10? 25 A. Based on this table. Page 74	1 experiments in Table 8, correct? 2 A. The 44.2 AAV8 and AAV1, correct. 3 Q. So it's reasonable to assume that paragraph 4 257 is referring to the data -- 5 MS. MORRISON: Objection to the form. 6 Q. -- correct? 7 A. Not the data in Table 10. 8 Q. It probably is referring to the data in 9 Tables 9 and 10, however, those only relate to lung, 10 right? 11 A. Okay. I didn't discern that as easily. 12 Q. Right. So paragraph 256 refers to the data 13 from Tables 9 and 10 in saying the data from both 14 experiments confirm the superb tropism of clone 44.2 in 15 lung-directed gene transfer, right? 16 MS. MORRISON: Objection to the form. 17 A. The data experiments confirms tropism 44.2 18 in the lung. 19 Q. I'm just trying to make sure we are, you 20 know, all on the same page. That's referring to Tables 21 9 and 10, correct? 22 MS. MORRISON: Objection to the form. 23 A. I'm lost in what are you referring to? The 24 reading from 256, as related to Tables 9 and 10, and 25 the reading from 257 is related to Table 8. Page 76
1 Q. Yeah, based on Table 8, it tells you that, 2 right? 3 A. Correct. 4 Q. Okay. So paragraph 257 is comparing the 5 performance of AAVrh10, right, to AAV8 and AAV1, 6 correct? 7 A. Correct. 8 Q. It doesn't mention AAV7, does it? 9 A. No. 10 Q. And in fact, it is drawing these 11 comparisons based on this Table 8, where the three were 12 directly compared, AAVrh10, AAV8 and AAV1, correct? 13 MS. MORRISON: Objection to the form. 14 A. I'm losing when you say they are drawing it 15 directly from 8 back over to the statement in 257? 16 Q. Well, what I was saying was that -- let me 17 rephrase that, okay. 18 The statement in 257 discusses at least 19 three serotypes, right: AAVrh10, AAV1 and AAV8, 20 correct? 21 A. Correct. 22 Q. It doesn't discuss AAV7, correct? 23 A. Correct. 24 Q. The three serotypes that it mentioned in 25 paragraph 257 were all tested in the same set of Page 75	1 Q. That was my question. I only involved 2 Tables 9 and 10 because you mentioned them, 3 Dr. Samulski. I'm trying to focus on paragraph 257 and 4 Table 8. But I just wanted to make sure we were on the 5 same page, because you mentioned 9 and 10. We can 6 ignore 9 and 10, if you don't mind for a moment, and 7 just look at 257 and Table 8. 8 A. Okay. I'm with you now. I read it. 9 Q. Okay. So my question from several 10 questions ago was that paragraph 257 is comparing 11 AAVrh10, AAV1 and AAV8, we agree on that, right? 12 A. Correct. 13 Q. And it is referring to the results shown in 14 Table 8, correct? 15 MS. MORRISON: Objection to the form. 16 A. That's the assumption. 17 Q. Right. And Table 8 tests all three of 18 those serotypes directly against one another, right? 19 A. That's correct. 20 Q. All right. And AAV7 is not mentioned in 21 paragraph 257, and it's not tested in Table 8, correct? 22 A. That's correct. 23 Q. So that for a person of ordinary skill to 24 try to draw a comparison directly between AAVrh10 and 25 AAV7, that person would have to look across experiments Page 77

1 in the '772, right? 2 MS. MORRISON: Objection to the form. 3 A. Because they were not in the same 4 experiment, you would end up comparing between 5 experiments. 6 Q. Right. And that's a comparison -- I'll 7 strike that. Okay. 8 Now, I'll shift gears. Let's take a look 9 back at your declaration, Dr. Samulski, and I would 10 like to look at page 15, paragraph 28. And let me know 11 when you're there. 12 A. I'm there. 13 Q. All right. Now, you say here that the kind 14 of modifications that I understand are being proposed 15 by petitioner here, targeting a single amino acid in 16 the AAV capsid to allegedly improve or change its 17 functionality, such as it's tissue targeting ability, 18 has not been reported in the literature by 2005. 19 Do you see that? 20 A. Yes. 21 Q. And you go on in fact to say that it wasn't 22 until years later that this kind of thing was done, and 23 you cite a 2019 paper, right? 24 A. And it gives an example, yes. 25 Q. All right. Now, the Snowdy dissertation Page 78	1 single amino acid in the AAV capsid to improve or 2 change functionality, right? 3 MS. MORRISON: Objection to the form. 4 A. So I am acknowledging that he chose 5 multiple amino acids and had to change them one by one 6 in order to define if a single amino acid was capable 7 of imparting a change. Some of them did not. Some of 8 them did, were positive, and some of them were 9 negative. 10 So it was a collection of studies, not just 11 choosing a single amino acid. That would have been 12 near impossible. 13 Q. So you're saying that Snowdy's results 14 don't provide information about single amino acid 15 changes because he tried several of them before he 16 found one that altered the function of the capsid? 17 MS. MORRISON: Wait, sorry, objection 18 to the form. And, counsel, I'm going to ask 19 you to stop raising your voice at the 20 witness. Ask your questions. He'll give 21 you answers. 22 BY MS. TOKER: 23 Q. I apologize, Dr. Samulski. I did not mean 24 to be rude. I apologize. 25 A. I'm not offended in any way. Page 80
1 shows that people of skilled in the art at the relevant 2 time were making single amino acid changes to the AAV 3 capsid that could improve or change functionality, 4 right? 5 A. In what way? 6 Q. Sorry? 7 A. I'm sorry, in what way? 8 Q. Oh, in what way? Oh, well, let's start, do 9 you agree that Snowdy, first of all, was making single 10 amino acid changes -- 11 A. Yes. 12 Q. Wait, let me just finish, sorry. 13 Do you agree that Snowdy was making single 14 amino acid changes to the AAV capsid? 15 A. Yes. 16 Q. All right. And do you agree that his 17 results showed that a single amino acid change in the 18 AAV capsid could improve or change functionality? 19 A. After he made multiple single amino acid 20 changes, he was able to deduce that some of them and 21 finally one of them looked like it was important to 22 impair change, correct. 23 Q. Was important to what? I'm sorry. 24 A. Impact and biological outcome of the virus. 25 Q. Okay. So you agree that Snowdy targeted a Page 79	1 Q. I'll try to -- 2 A. What I am saying is it's impossible to 3 choose a single amino acid, make that change and assume 4 it's going to have an impact on a viral function, that 5 you had to choose multiple amino acids and 6 systematically make changes. Sometimes experiments 7 were done where he made these changes and absolutely 8 nothing would happen, and other times after an 9 exhaustive study like this, you would come across one 10 that would impact a biological step. But it could have 11 been one in not letting the virus be stable and have 12 nothing to do with infectivity or tropism or any of the 13 other definitions that people typically use them for. 14 Q. I see. So your opinions about the 15 state-of-the-art have to do with whether or not a 16 person of ordinary skill at the time could look at a 17 whole linear sequence of 738 amino acids and say that's 18 the one that's going to change tropism, and you're 19 saying people couldn't do that at the time? 20 A. I'm saying absolutely could not do that. 21 Q. All right. Okay, so you -- 22 A. Still can't. 23 Q. Still can't, right. 24 A. Even with AI. 25 Q. Yeah, I agree. Separate discussion. Page 81

1 A. Wait a second, do you agree with that? 2 Q. Separate discussion for the break at the 3 end of the day. 4 A. Okay. 5 Q. But let's go back to the state-of-the-art. 6 So and so you are responding in this 7 proceeding to Dr. Schaffer's opinions, correct? 8 A. Yes, that's correct. 9 Q. Dr. David Schaffer? 10 A. Yes, that's correct. 11 Q. Do you know Dr. Schaffer? 12 A. I do know David. 13 Q. And what are your thoughts on David as the 14 member of the AAV research community? 15 A. David is a very talented and he has been a 16 significant and consistent contributor to our field. 17 And I think he has one of his AAV tropic viruses for 18 the eye in phase three clinical trials, if I'm not 19 mistaken, demonstrating the success. 20 Q. So Dr. Schaffer's opinion in this case is 21 that a person of ordinary skill in the art would have, 22 you know, combined with teachings of the '772 23 publication with Snowdy, right? You agree with that? 24 A. Go ahead, I'm listening. 25 Q. Let me just -- Page 82	1 MS. MORRISON: Objection to the form. 2 A. I think I'm understanding what you're 3 saying that -- you have to say it one more time so I 4 make sure. '772 is the publication of the patent? 5 Q. Yes. It's exhibit -- I mean, it's a patent 6 publication, correct. 7 A. Right, I'm just talking about the document 8 we're referring to. 9 Q. Correct. 10 A. And Snowdy's thesis. 11 Q. Correct. 12 A. Are the two that you're referring to? 13 Q. Well, I'm saying that -- 14 A. That David was referring to? 15 Q. Let me just finish. Yes. Yes. I'll start 16 over. 17 I'm just trying to ask you whether we're on 18 the same page in terms of the opinion of Dr. Schaffer 19 that you are responding to in this proceeding? 20 A. Okay. 21 Q. I'm not asking if you agree with his 22 opinion. I'm just saying he said X, and you're 23 responding to X at this point. Is that -- I'll 24 rephrase the question, but is that clear, generally? 25 A. Yeah, I think I follow you now. Page 84
1 A. I'm trying to figure out. 2 Q. Let -- 3 MS. MORRISON: Sorry, Ms. Toker, could 4 you just start that question over? 5 MS. TOKER: Okay. 6 MS. MORRISON: The transcript says 7 Dr. Samulski. 8 MS. TOKER: I don't think I said that, 9 but I'll try again. 10 Q. So Dr. Schaffer's opinion in this case that 11 you're responding to, right, is that a person of 12 ordinary skill in the art would have combined the 13 teachings of the '772 publication with Dr. Snowdy's 14 thesis, correct? That's his opinion that you're 15 responding to? I'm not asking that you agree with it. 16 I'm saying you are responding to that opinion? 17 A. Correct. 18 Q. And his opinion is that this person of 19 ordinary skill, looking at the teachings of both, would 20 have selected several amino acids to test to see if any 21 of them affected functions such as tropism, 22 transduction. Do we agree with that? 23 A. That would have been one approach. 24 Q. Do you agree that that's Dr. Schaffer's 25 opinion that you're responding to? Page 83	1 Q. Okay. So my question was that 2 Dr. Schaffer's opinion that you are responding to is 3 first part, a person would combine the teachings of the 4 '772 publication with Snowdy's dissertation, that 5 that's his opinion? 6 A. That's his opinion, correct. 7 Q. Okay. And that as, you know, following 8 from that part of the opinion, Dr. Schaffer, and I'm 9 not asking if you agree, I'm just saying Dr. Schaffer 10 says that based on the teachings of these two sources, 11 references, a person of ordinary skill in the art at 12 the time would have selected several amino acids to 13 mutate to see if they altered functions, such as liver 14 tropic? I'm not asking if you agree. I'm asking if 15 agree that that's his opinion that you're answering? 16 A. That's his premise, correct. 17 Q. Okay. So his opinion does not involve 18 saying that a person of ordinary skill would be able to 19 look at 738 amino acids of AAVrh10 and go if I mutate 20 that, I have a silver bullet for DMD, Duchenne muscular 21 dystrophy or anything else. You agree that's not what 22 Dr. Schaffer is saying? 23 MS. MORRISON: Objection to the form. 24 A. Based on the way you described it, that's 25 correct. Page 85

1 Q. Okay. And so but -- nonetheless, 2 Dr. Schaffer is identifying four amino acids of 3 interest in the AAVrh10 sequence that a person of 4 ordinary skill would mutate. Do you agree with that? 5 MS. MORRISON: Objection to the form. 6 A. That's the approach he's taking. 7 Q. And that's the approach now he took, right? 8 He identified in this case three amino acids of 9 interest, and he mutated those and tested their effect 10 on function, right? 11 MS. MORRISON: Objection to the form. 12 A. That's the approach Snowdy took with 13 failure with multiples of those not working -- 14 multiples of those targets not working. 15 Q. Science don't always work, does it? 16 A. No. 17 Q. And sometimes if you mutate three amino 18 acids and one of them works, it's a pretty good 19 percentage for an experiment, right? 20 MS. MORRISON: Objection to the form. 21 A. That would be an assumption. Most of the 22 time they don't work, and you're doing the same thing 23 over again. 24 Q. And you're thrilled when one of them works, 25 one of out three? Page 86	1 (The document referred to was 2 previously marked Deposition Exhibit 3 Number 1009 for identification.) 4 Q. Now, let's look at this is Exhibit 1009, 5 and I am going to be referring to the page numbers, the 6 little page numbers in the bottom right-hand corner, 7 that Sarepta Exhibit 1009 page. It's a little 8 confusing, because it has its own page numbers, but I'm 9 going to be referring to those little bottom right-hand 10 numbers, okay. 11 So I would like, please, to turn to page 12 125 in the little right-hand corner number 125, and 13 there's a heading on that page that says Section IV.B, 14 as in boy, Roman numeral IV.B, results. Let me know 15 when you're there, doctor. 16 A. I'm there. 17 Q. So, now he says here, Snowdy, that he chose 18 three cites to mutate in the AAV capsid, and he lists 19 them; serine 148, threonine 159 and serine 181, 20 correct? 21 A. Correct. 22 Q. And serine and threonine are 23 phosphorylatable amino acids, right? 24 A. Correct. 25 Q. And now Snowdy says that he chose these Page 88
1 MS. MORRISON: Objection to the form. 2 A. If you're lucky. 3 Q. If you're lucky, you get one out of three 4 to work right? Yes? 5 A. Correct. 6 Q. And in fact, what Snowdy did was he didn't 7 just get his experiment to work, he got an unexpected 8 result that he enhanced a function that he didn't 9 expect to enhance, right? 10 MS. MORRISON: Objection to the form. 11 A. That's the data suggests that, correct. 12 Q. Right. And so what he found when he 13 mutated a single amino acid, right, in an AAV2, he 14 found that he enhanced transduction efficiency with 15 that single amino acid change, correct? 16 A. That is an outcome from his study. 17 Q. I'm sorry? 18 A. That's an outcome from his study. He also 19 found ones that knocked down the ability to make the 20 virus. 21 Q. Let's focus on the -- let's actually look 22 at this, okay, because I want to make sure we're on the 23 same page. Let's take a look now at -- I'm going to 24 ask the court reporter to hand you, Dr. Samulski, what 25 has been marked Exhibit 1009. Page 87	1 three cites based on prior work in the field of nuclear 2 localization signals that related to a different virus, 3 SE40, correct? 4 A. Correct. 5 Q. And this prior work on this different virus 6 showed that phosphorylation of residues near a nuclear 7 localization site could affect nuclear localization, 8 right? 9 A. That's correct. 10 Q. And so based on this prior work with SE40, 11 Snowdy decided to mutate each of these three 12 phosphorylatable residues in the AAV capsid that we 13 just talked about, serine 148, threonine 159 and serine 14 181, right? 15 MS. MORRISON: Objection to the form. 16 A. Correct. 17 Q. And he decided to mutate each of these 18 three either to alanine, which is nonphosphorylatable 19 or to aspartic acid which simulates a constitutively 20 phosphorylatable residue, right? 21 A. Correct. 22 Q. As he talked about, he made a total 23 therefore of six mutants, correct? 24 A. Correct. 25 Q. And he tested the ability of each of these Page 89

1 six mutants to transduce HeLa cells, right? 2 A. Correct. 3 Q. Now, he says on the next page, which is 4 page 126, he says: We infected cultured HeLa cells 5 with 500 particles of cell of each of the six mutants 6 to establish whether the potential phosphorylation 7 sites are important in the virus's ability to deliver 8 its genome to the target cell during an infection, 9 right? 10 A. Correct. 11 Q. Here he refers to the target cell, which is 12 more broad than just to a HeLa cell, right? 13 A. He's referring to HeLa cells. Those are 14 the cells he analyzed. 15 Q. I know, but he's not just interested, is 16 he, in HeLa cells in this work? 17 A. It says in the sentence from the beginning: 18 Infected cultures of HeLa cells. 19 Q. Right, but he's looking at HeLa cells, with 20 the expectation that the results he finds could be more 21 broadly applicable to other cell types, right? 22 MS. MORRISON: Objection to the form. 23 A. I'm not sure if I would take that 24 conclusion home from that. I think he just -- and his 25 grammar wasn't writing HeLa cells twice. He was Page 90	1 MS. MORRISON: Objection to the form, 2 foundation. 3 A. I think we are moving back into that 4 generic cell line versus tissues. 5 Q. No, I wasn't asking about that. I was just 6 asking about the purpose of conducting experiments in 7 HeLa cells and suggesting that they would influence 8 other experiments in other tissues. 9 Do you disagree with that? 10 A. No. I was citing specific -- we did it in 11 HeLa cells, because HeLa cells were the most permissive 12 cell to study in a molecular step and be consistently 13 able to reproduce it. 14 Q. It's a good in vitro system to test nuclear 15 localization of AAV, right? 16 A. That pretty much is the default line. 17 Q. For at least AAV2? 18 A. For AAV2. 19 Q. But you're interested in nuclear 20 localization of AAV as a broader phenomenon than just 21 nuclear localization of HeLa cells, right? 22 MS. MORRISON: Objection to the form. 23 A. We're hoping to uncouple steps that are 24 involved in the infectious pathway of AAV2. 25 Q. For what purpose? Page 92
1 writing the ability to deliver genome to target cell 2 during an infection, target cell, HeLa cell. 3 Q. So you and Dr. Snowdy were very interested 4 in infection of HeLa cells? 5 A. For this step, correct. 6 Q. Well, you were using this to test nuclear 7 localization with the expectation it might be more 8 broadly applicable, right? 9 MS. MORRISON: Objection to the form, 10 and, again, counsel, please don't argue with 11 the witness. 12 A. We were studying a very specific step that 13 we could only confer that it worked in that cell type, 14 until we could test it in other cell types. It may be 15 unique to HeLa cells. 16 Q. It might be, but you would use your results 17 from HeLa cells to determine what further tests to do, 18 right? 19 MS. MORRISON: Objection to the form. 20 A. Well, he could do that. 21 Q. Would you do that? 22 A. I didn't do that. 23 Q. Okay. But testing in HeLa cells, depending 24 on the results, could influence future experiments in 25 other cells, right? Page 91	1 A. As illustrated in the cartoon, that this 2 may be an important step for AAV to get into a cell. 3 Q. Right. A cell other than a HeLa cell? 4 A. Other than a HeLa cell. 5 Q. Even a tissue like in vivo, an organ tissue 6 or, you know -- 7 A. We would make an assumption, yes. 8 Q. That these results could be informative? 9 A. They could be informative, but they may not 10 be related. 11 Q. They may not be? 12 A. Related. 13 Q. They may not. But they might be? 14 A. I'm going to back up on this, so there's no 15 confusion. 16 There are different mechanisms by which 17 cells enter the nucleus. Some of them are defined in 18 tissue culture but do not occur in tissue, if that 19 makes sense. And therefore what we were deciphering 20 were the steps that we can say worked in tissue 21 culture. We've been burnt enough where if we made this 22 assumption, it did the same thing, and it didn't work, 23 it would be simple like, okay, we study something 24 unique to this cell type and that's not explorable to 25 everything. It's restricted to this cell type. Page 93

1 Does that make sense? 2 Q. Yeah, at the time, did you know that the 3 phenomenon you were studying here was restricted to 4 HeLa cells? 5 A. We only did -- I'm sorry. 6 MS. MORRISON: Objection to the form. 7 Go ahead. 8 A. We only did HeLa cells. 9 Q. Right. But you didn't already know that 10 this was not translatable to other tissues? 11 A. We hadn't explored other tissues. 12 Q. Right. And in fact, you might have gone on 13 to explore other tissues, depending on, you know, your 14 interest and the results here, right? 15 MS. MORRISON: Objection to the form. 16 A. I think what you're asking similar to the 17 GTG hypothesis in the patent, we might have gone on to 18 test it and other AAVs, but probably wasn't going to 19 work. 20 Q. Okay. What part of this did you think 21 wasn't going to work? 22 A. I didn't have a hypothesis that it wasn't 23 going to work. We were never able to find a nuclear 24 localization signal by definition of nuclear 25 localization signal needs to be attached to a form Page 94	1 the three mutations, sorry, the three amino acids that 2 Dr. Snowdy mutated in this part of his dissertation, 3 and we just discussed that he mutated each of these 4 three cites to one of two amino acids, either alanine 5 or aspartic acid, correct? 6 A. Correct. 7 Q. And what he says is that -- I'm just 8 reading the last sentence now on page 126, he says: 9 The same number of particles containing S181A 10 mutation -- and just so we're all clear, that means 11 serine 181 mutated to alanine, correct? 12 A. Correct. 13 Q. The same number of particles contained in 14 S181A mutation infected far more cells than did the 15 wild-type, and the same number of particles containing 16 the S181D -- and to be clear, that means serine 181 17 mutated to aspartic acid, right? You have to say yes 18 or no. 19 A. Yes. 20 Q. Thank you. Contained the S181D mutation 21 infected almost no cells at all, right? 22 A. Correct. 23 Q. All right. And now you had mentioned is 24 that -- you had mentioned something about not being 25 able to form a capsid, correct? Page 96
1 protein and carried into the nucleus. We were in some 2 smoochy area where it was basic regions that had 3 charged amino acids that we were trying to link to 4 nuclear localization signals. 5 So any and all of our outcomes could have 6 been related to another aspect of AAV biology that was 7 unrelated to nuclear localization signals. 8 Q. Okay. Now I understand why we have not 9 been -- had a meeting of the minds. Yes, okay, let me 10 be very -- more specific then. I totally understand 11 now. 12 So what you're saying is the results that 13 you've got here didn't substantiate a hypothesis about 14 a nuclear localization signal that you had or that you 15 all had when Dr. Snowdy started this work, correct? 16 MS. MORRISON: Objection to the form. 17 Go ahead. 18 A. Yeah, we were not convinced it was a 19 nuclear localization signal, and therefore any 20 observations that we got left us with is it a nuclear 21 localization signal or something next to it that may be 22 impacting the virus. 23 Q. Okay. All right. 24 A. So our hypothesis was in flux. 25 Q. Understood, okay. So now let's go back to Page 95	1 MS. MORRISON: Objection to the form. 2 BY MS. TOKER: 3 Q. Right. Sorry, give me one second. 4 A. I think you want page 129, the last 5 paragraph, another possibility. 6 Q. 129, not yet. Let me just clarify one 7 thing first. Oh, I know what I wanted, I'm sorry, page 8 126, my apologies. Page 126, the very first sentence 9 said: All of the mutants were capable of forming virus 10 at normal titers, agree? 11 A. Agree. 12 Q. So all six of these mutants formed the 13 capsid and formed the virus, correct? 14 A. That's what it says, titer, yes. 15 Q. Okay. And the issue is just that he found 16 that this one single mutation, S181A, enhanced 17 efficiency of transduction, right? 18 MS. MORRISON: Objection to the form. 19 A. It's S184A(sic) when changed from serine to 20 alanine saw an increase in transduction. 21 Q. Correct. Agreed? Yes? 22 A. Agree. 23 Q. Now, this was surprising to Snowdy, because 24 he expected this mutation to lower, to reduce 25 transduction efficiency if S181 is important for Page 97

1 regulating the putative nuclear localization signal, 2 right? 3 A. So it was opposite observation of what was 4 expected from nuclear localization signals. 5 Q. Right. And in fact, he concludes, he says 6 on page -- and this may be where you had headed us 7 before on page 129, he says: It is premature. Let me 8 know if you're there before I start reading. I'm on 9 page 129, the first full paragraph. 10 A. 129? 11 Q. The first full paragraph that begins until 12 we establish? 13 A. Until we unequivocally. 14 Q. Until we established unequivocally that 15 basic region 3 of AAV2 is an NLS, meaning nuclear 16 localization signal, right? 17 A. Correct. 18 Q. It is premature to assign the cause of the 19 changes in transduction efficiency resulting from 20 mutations to S181 as interfering with the function of 21 an NLS, right? 22 A. Correct. 23 Q. And so a person of ordinary skill in the 24 art reading this disclosure would understand that 25 Snowdy's results suggest that another function, other Page 98	1 (The document referred to was marked 2 Deposition Exhibit Number 1059 for 3 identification.) 4 Q. Dr. Samulski, the court reporter just 5 handed you what has been marked Exhibit 1059. 6 Have you seen this document before? 7 A. Can I look at it for a second? 8 Q. Sure. I will just read in for the record 9 that this document is a provisional patent application 10 number 60-636126. 11 A. Okay. 12 Q. All right. I'll ask again, have you seen 13 this before, Dr. Samulski? 14 A. I think I have. 15 Q. And this is a provisional patent 16 application on which you are a named inventor, correct? 17 A. Correct. 18 Q. This patent application is titled chimeric 19 vectors, correct? 20 A. Correct. 21 Q. Now, for purposes of our discussion today, 22 and I apologize for throwing more numbers at you, I'm 23 going to refer to this as the '126 provisional, because 24 it's the last three digits 126 in the documents. Is 25 that okay? If you look at the number in the right-hand Page 100
1 than nuclear localization, was affected by the S181A 2 mutation, right? 3 MS. MORRISON: Objection to the form. 4 A. He was coming up with other hypothesis. 5 Q. Okay. I'm sorry, I didn't mean to cut you 6 off. Were you saying something? 7 A. Um-um. 8 Q. And so a person of ordinary skill in the 9 art reading this disclosure would further understand 10 that this hypothetical other function had been affected 11 by removal of a single putative phosphorylation site by 12 substituting a nonphosphorylatable residue in somehow 13 that enhanced AAV transduction, right? 14 MS. MORRISON: Objection to the form. 15 A. The data implied that. 16 Q. Okay. I just wanted to check before I 17 start in my next section. Could we go off the record? 18 THE VIDEOGRAPHER: The time is 11:40, 19 we're off the record. 20 (Recess.) 21 THE VIDEOGRAPHER: The time is 11:53, 22 we're on the record. 23 MS. TOKER: I am going to ask the 24 court reporter to mark this document 25 Exhibit 1059. Page 99	1 corner, it's a bar code, the last three digits are 126, 2 okay? 3 A. Yes. 4 Q. Feel free to ask me what I'm talking about 5 if you forget later on that it's the '126. It's 6 Exhibit 1059, and I'll also call it the '126. 7 Now, this provisional application was filed 8 on December 15 of 2004, correct? 9 A. That's what it says, yes. 10 Q. Now, one of the listed inventors on this 11 provisional is someone named Josh Grieger, correct? 12 A. Correct. 13 Q. And I think his name is actually misspelled 14 there G-R-E-I-G-E-R, when it's actually G-R-I-E-G-E-R, 15 right? 16 A. Correct. 17 Q. It's actually corrected later in the 18 document, but we understand that we're talking about 19 Josh Grieger, right? 20 A. Correct. 21 Q. And he was a graduate student in your lab, 22 correct? 23 A. Graduate student and a post doc. 24 Q. At the time that this was filed, he was a 25 graduate student, right? Page 101

1 A. That's correct. 2 Q. And he had about two years of experience in 3 your lab at the time that this '126 provisional was 4 filed in December of 2001? 5 A. I can't recall that. 6 Q. Let's take a quick look at your CV, okay, 7 just to remind you. Give me one second. Your CV was 8 marked Exhibit 2028. Do you have that? 9 A. Yep. 10 Q. Okay. And I think it's on page 19 of your 11 CV. Let me know when you're on page 19. 12 A. 19. 13 Q. Okay. So at the very first name in this 14 table it says Josh Grieger, and that's who we're 15 talking about, right? 16 A. That's correct. 17 Q. It says he was a pre doc from 2002 to 2006, 18 right? 19 A. Correct. 20 Q. So in 2004 he had about two years of 21 graduate work in your lab? 22 A. That's a correct assumption. 23 Q. So he -- just to be clear, he did not have 24 a Ph.D. yet at the time the work was carried out as of 25 December 15? Page 102	1 A. Yes. 2 Q. All right. And what does it mean by the 3 backbone of AAV2? 4 A. The original sequence that's been 5 identified for that serotype. 6 Q. For the capsid? 7 A. Correct. 8 Q. So the backbone is the original AAV2 9 sequence or capsid? 10 A. Correct. 11 Q. So so far what we've read is describing 12 substituting amino acids from the capsid of 1AAV 13 serotype, AAV1, into the capsid of another AAV 14 serotype, AAV2, right? 15 A. Correct. 16 Q. The next sentence says: In particular 17 embodiments, the chimeric viruses of the invention have 18 enhanced transduction capability e.g. transduction of 19 skeletal muscles, cardiac muscle, liver, retina, and/or 20 lung, et cetera, and/or enhanced levels of transgene 21 expression with earlier onset of expression. 22 Do you see that? 23 A. Yes. 24 Q. And so potentially some of these 25 embodiments here have enhanced transduction capability Page 104
1 A. Correct. 2 Q. December 15, 2004, okay. 3 And he actually subsequently went to work 4 for you at AskBio, is that right, Josh Grieger? 5 A. Josh left the lab, came back as a post doc, 6 joined Bamboo, went to Pfizer, left Pfizer, came back 7 and joined AskBio. 8 Q. Okay. Now, let's take a look then at 9 Exhibit 1059, the '126 provisional, and I'd like to 10 look at the section titled summary of the invention. 11 And there's no number on the page, but it is the first 12 page of the publication, so it's like the fourth page 13 of the document. There's something that says the 14 summary of the invention. You may have just passed it, 15 Dr. Samulski, summary of the invention. 16 Do you see that? 17 A. Yes. 18 Q. So we're looking, yeah, it's around line 32 19 on that page, the heading, summary of the invention, 20 and I'm going to be directing you to what he says under 21 that. It says: The inventors have identified the key 22 amino acids of AAV1 responsible for enhanced in vivo 23 transduction and selectively engineered these amino 24 acids into the backbone of AAV2 as well as other AAV. 25 Do you see that? Page 103	1 across different tissues, right? 2 A. Correct. 3 Q. Now, the next sentence says: With 4 respect -- and I'm still reading. I'm just reading 5 from the top of page two, and I'm at the end of line 6 two, okay? 7 A. I'm following. 8 Q. With respect to chimeric viruses based on 9 AAV2, these chimeras retain the desirable properties of 10 AAV2 e.g. ease of purification and known safety, while 11 exhibiting some of the advantageous properties of AAV1, 12 including enhanced transduction of skeletal muscle. 13 Do you see that? 14 A. Yes. 15 Q. So the patent -- I'm sorry, this 16 provisional patent application here is disclosing AAV2 17 mutants that have selected amino acids replaced with 18 amino acids from AAV1, right? 19 A. Correct. 20 Q. And these AAV2 mutants retained desirable 21 properties of AAV2 while taking on some of the 22 desirable properties of AAV1? 23 A. Correct. 24 Q. In particular, these mutants take on 25 enhanced ability of AAV1 to transduce skeletal muscle, Page 105

1 right? 2 A. Correct. 3 Q. Okay. And these particular amino acid 4 mutations preserve, as we said, certain desirable 5 properties of the parent, AAV, which in this case is 6 AAV2? 7 A. Correct. 8 Q. So what you and the other inventors are 9 doing here is that you wanted to improve muscle tropism 10 of AAV2 to make it more like AAV1 in that respect while 11 preserving other desirable properties of AAV2? 12 MS. MORRISON: Objection to the form 13 of the question. 14 A. We were increasing -- trying to improve 15 muscle tropic transduction. The byproduct was it 16 continued to retain attributes of AAV2. 17 Q. And those were desirable attributes that 18 you wanted to retain, such as ease of purification and 19 known safety, right? 20 A. Yeah, those were true. 21 Q. Sorry, you agree? 22 A. That's true. 23 Q. Now, you didn't decide here to work with 24 AAV1, which is better in muscle than AAV2, right? 25 MS. MORRISON: Objection to the form. Page 106	1 A. Most people would look at this as rational 2 design. 3 Q. And you were included as most people? 4 A. Yep. 5 Q. Now, I'd like to look at the section on 6 page five that is titled detailed description of the 7 invention. So this is around line 20 of page 5. 8 Let me know when you're there, 9 Dr. Samulski. 10 A. I'm there. 11 Q. Okay. Now, the second sentence under that 12 heading begins the inventors. Do you see that, the 13 inventors have? 14 A. Are we on page five? 15 Q. Yeah, I'm on page five and actually on 16 line -- 17 A. The inventors have discovered? 18 Q. Yeah, sort of the inventors have, yes? 19 A. Yes. 20 Q. Okay. And that sentence says: The 21 inventors have discovered that in some cases as little 22 as a single amino acid insertion or substitution from a 23 first parvovirus e.g. and AAV, into the capsid 24 structure of another parvovirus, e.g. and AAV, to 25 create a chimeric parvovirus is sufficient to confirm Page 108
1 A. At that time, AAV2 was the only virus that 2 the FDA would approve for going into patients. So it 3 was thought and decided that if we did it in 2, we 4 would have a better chance of the FDA accepting the 5 modified 2 versus a completely new AAV serotype. 6 Q. So sometimes there are reasons that you 7 would choose not to go with the serotype that's best in 8 a particular tissue, even if you're interested in 9 targeting that tissue, right? 10 A. There were other aspects that taken into 11 account, AAV1 was best for intramuscular injection but 12 not for systemic injection. AAV2 was better for 13 systemic injection but not for muscle. 14 So we deduced that if we could capture the 15 muscle aspect into a systemic virus, we would achieve a 16 better outcome. 17 Q. Right. And just because AAV1 had these 18 superior muscle transduction properties over AAV2, you 19 still chose, for other reasons, to modify AAV2 rather 20 than to work with AAV1, correct? 21 MS. MORRISON: Objection to the form. 22 Go ahead. 23 A. That is correct. 24 Q. Now, you would describe this approach as a 25 rational design approach, right? Page 107	1 one or more of the desirable properties of the first 2 parvovirus to the resulting chimeric parvovirus. 3 Do you see that? 4 A. Yes. 5 Q. Okay. So here the '126 provisional, 6 Exhibit 1059, discloses that a single amino acid 7 insertion or substitution from one AAV serotype into a 8 different AAV serotype is sufficient to confirm one or 9 more of the desirable properties of the first AAV onto 10 the second AAV, right? 11 A. That's correct. 12 Q. Okay. Let's look at page 11, the section 13 titled chimeric virus vectors. It's around line 27. 14 Let me know when you are there. 15 A. Chimeric viral vectors? 16 Q. Yeah, page 11, there's a bold heading 17 underlined. 18 A. The inventors have identified? 19 Q. Yes, exactly. 20 So I want to look at the last sentence on 21 the page, on page 11 under that heading that begins to 22 illustrate, okay? Do you see that? 23 A. Um-hum. 24 Q. It says: To illustrate, in the case of 25 AAV2, the inventors have discovered that with a single Page 109

1 amino acid insertion from AAV1, AAV2 can acquire the 2 enhanced transduction capability of AAV1 while 3 retaining the ease of purification, i.e., retains 4 ability to bind heparin, and the known safety features 5 of AAV2. Do you see that? 6 A. Yep. 7 Q. So here the '126 provisional is disclosing 8 that a single amino acid change, specifically inserting 9 a single amino acid from AAV1 to AAV2 confirmed the 10 superior transduction ability of AAV1 onto AAV2? 11 MS. MORRISON: Objection to the form. 12 BY MS. TOKER: 13 Q. Right? 14 A. That's what it says. 15 Q. And this single amino acid change also 16 preserved the favorable properties of AAV2, right? 17 MS. MORRISON: Objection to the form. 18 A. That's what it says. 19 Q. Okay. Now, I want to look a little bit at 20 how you determined which amino acids to substitute in 21 from AAV1 and AAV2 here, okay? 22 A. Okay. 23 Q. All right. So we're going to look at 24 beginning on line 9 of page 12, you know, the page we 25 had started, page 12, line 9, it's the first full Page 110	1 A. Correct. 2 Q. And that will show you regions or even 3 specific amino acid positions where the sequences might 4 be the same or might differ from each other, correct? 5 A. Correct. 6 Q. So this is a way of figuring out places 7 where the sequences differ that you might be interested 8 in making mutations from one sequence into the other, 9 correct? 10 A. Correct. 11 Q. Okay. Now, let's look at -- I'm going to 12 just skip around a little bit, I'm sorry. Let's look 13 at page 41. And on page 41, around lines 10 to 11, 14 this section is further discussion of the approach that 15 you took in this provisional to identify amino acids to 16 mutate, and then the effects of that. And it says, 17 just looking at line 10, the amino acid sequences of 18 AAV1 and AAV2 are 83 percent identical. 19 Do you see that? 20 A. Yes. 21 Q. All right. And let's see how many amino 22 acids that actually translates into, how many amino 23 acids are different, you know, that 17 percent between 24 AAV1 and AAV2. 25 So let's look at Figure 1A, which is Page 112
1 paragraph on page 12, and it begins in the present 2 studies. Do you see that? 3 A. Yes. 4 Q. It says that: In the present studies, the 5 inventors have compared the linear amino acid sequences 6 of two serotypes with high levels of skeletal muscle 7 transduction, AAV1 and AAV7, with two that are less 8 effective in transducing skeletal muscle, AAV2 and 9 AAV8. 10 Do you see that? 11 A. Yeah, did they mention rh10? 12 Q. No. No. It just says here, there's just 13 comparing -- 14 A. The best. 15 Q. Well, they're comparing AAV1, AAV7, AAV2 16 and AAV8, right? That's what it says there, correct? 17 A. Yeah, referring to the best. 18 Q. Sorry? 19 A. Most likely we used those because we refer 20 to the best. 21 Q. Okay. And you did a sequence alignment, 22 right? It says linear amino acid sequences. 23 A. Correct. 24 Q. That's a sequence alignment of all four 25 sequences, right? Page 111	1 towards the back, Dr. Samulski. I'm afraid you're just 2 going to have to page through it towards the end. It 3 doesn't have any page numbers, but there is a figure 4 called Figure 1A. Let me know when you found it. 5 A. I'm there. 6 Q. Again, it's a little bit hard to read, so 7 I'm not going to ask you to read all the stuff that's 8 blacked out, I promise. But the heading says 36 9 candidates based on triage and then (versus 83 from 10 AAV1 to 2 alignment). Do you see that? 11 A. I do see that. 12 Q. So they're saying here that the alignment 13 showed 83 positions at which the sequences deferred, 14 right? 15 MS. MORRISON: Objection to the form. 16 A. The alignment infers that 83 were different 17 between AAV1 and 2 by the alignment. 18 Q. Okay. And AAVrh10 and AAV8 are more 19 closely related than AAV1 and AAV2, right? 20 A. Say that again, rh10 and who? 21 Q. AAV8. 22 A. I think so. 23 Q. Yeah, because in fact those -- 24 A. 48 nucleotides. 25 Q. Sorry, let me finish my question, and then Page 113

1 the answer is good. 2 Right, AAVrh10 and AAV8 as you said differ 3 by 48 amino acids, correct? 4 A. That's correct. 5 Q. And that translates to be about 93 percent 6 identical, fair? 7 A. That's fair. 8 Q. So as we just said, AAV1 and AAV2 are less 9 closely related than AAVrh10 and AAV8, correct? 10 A. Correct. 11 Q. And then what this is saying, this heading 12 36 candidates based on triage, was that you used these 13 alignments to reduce the number of amino acids that you 14 were considering for mutagenesis from 83 down to 36 in 15 this step, correct? 16 MS. MORRISON: Objection -- I'm sorry, 17 objection to the form. Go ahead. 18 A. In this step. It shouldn't be remiss that 19 the crystal structure was also required -- 20 Q. We're getting to that. 21 A. -- to understand. And that when you say 22 candidates were eliminated, a lot of times those were 23 amino acids that were similar charge but not 24 hydrophobic versus hydrophilic. 25 So you begin to say those are the same Page 114	1 do that analysis. 2 Q. So other inventors on the provisional -- 3 A. Right. 4 Q. -- will analyze the crystal structures to 5 help narrow down the number of spots where you were 6 going to conduct mutagenesis? 7 A. Yeah, because I considered myself an 8 ordinary one skilled in the art, but I can't do the 9 crystal structure. She's an extraordinarily one that's 10 skilled in the art and was absolutely required to go 11 forward. 12 Q. Okay. So let's go back to page 12, which 13 discusses what we were just talking about. Turn back 14 to page 12 of this '126 provisional. 15 MS. MORRISON: Just to be clear, you 16 mean the page actually numbered 12? 17 MS. TOKER: Yeah, I do, because we 18 haven't numbered this yet. I mean, the 19 actual numbers on the bottom of the page, 20 not some other thing. 21 MS. MORRISON: Yes. 22 MS. TOKER: Thank you. 23 A. Okay. 24 Q. Yeah, just take your time, Dr. Samulski. 25 Let me know when you're there. Page 116
1 amino acids based on charge or hydrophobicity and 2 things like that and should not impair differences. 3 Q. Right, okay. So you analyzed the sequence 4 alignments, and, again, we're just talking about the 5 first step. I agree we'll get to the crystal structure 6 and other things. But just in terms of this step of 7 sequence alignment, okay, you analyzed the sequence 8 alignments, and out of the 83 places where AAV1 and 9 AAV2 differ, you selected 36 -- based on considerations 10 such as you mentioned of would it have been a 11 conservative substitution, for example, if you were to 12 make a change versus a nonconservative, correct? 13 A. Correct. 14 Q. Now, in fact, since we're right there, 15 let's just look at Figure 1B, which is right next door, 16 and it says exactly what you were just beginning to 17 tell us about, which is that it was then reduced to 12 18 candidates based on amino acid and crystal structure 19 analysis, correct? 20 A. Correct. 21 Q. So you used the crystal structure to reduce 22 further the number of positions that you decided you 23 would mutate here, correct? 24 A. Yeah, so let me clarify. Mavis used the 25 crystal structure. She was the only one qualified to Page 115	1 A. I'm there. 2 Q. So we're looking back at the numbered page 3 12, and I wanted to look at around lines 17 to 19, 4 where there's a sentence that begins this initial pool 5 of candidate positions. Do you see that? 6 A. Yep. 7 Q. This initial pool of candidate positions 8 was narrowed considerably based on the analysis of the 9 known crystal structure of AAV2, e.g. to identify amino 10 acid positions that are located in regions that are 11 likely to affect biologies of virus. 12 Do you see that? 13 A. Correct. 14 Q. And just to be clear, the known crystal 15 structure was the crystal structure published by first 16 authors, I'm going to spell the name because I 17 otherwise would probably butcher the pronunciation, but 18 X-I-E, correct? 19 A. Xie. Yes, Xie, Xie in Chapman's lab. 20 Q. Okay. And that's the crystal structure 21 that you're referencing here, correct? 22 A. Correct. 23 Q. Now, Dr. Schaffer outlined a similar 24 process in his declaration, doesn't he? 25 A. I'm assuming that -- I don't remember Page 117

1 exactly what he said. 2 Q. He says that a person of ordinary skill 3 would have aligned the sequence of AAV1 or AAV8 so -- 4 A. Correct. 5 Q. Let me just get it out. I appreciate it. 6 A. Sure. My bad. 7 Q. No. No. No. No worries. He says: A 8 person of ordinary skill would have aligned the 9 sequence, the linear sequence of AAV1 and AAV8 and 10 identified 48 positions where the two sequences differ, 11 right? 12 A. Correct. 13 Q. And he then proposes different ways that 14 the person of ordinary skill looking at those 48 15 differences might have narrowed down which positions to 16 mutate, right? 17 MS. MORRISON: Objection to the form. 18 A. I don't remember specifically what he 19 referred to. 20 Q. Okay. In one section he referred to the 21 X-I-E publication, Xie -- 22 A. Xie crystal structure. 23 Q. Crystal structure. And said he would have 24 looked at amino acids seen from that structure to be 25 relevant to the biology of viruses we see here? Page 118	1 site that Schaffer identifies as changing would have 2 been one that would have been triaged, because it's 3 redundant. There's no difference in the amino acids 4 that were chosen. And we relied on an expert with 5 crystal structure capability to guide us on what to do 6 next. And from that expertise, we continued to narrow 7 down to a set of five amino acids of which we 8 eventually found out we were wrong on four of them. 9 And of the one that we chose, it did not exist in the 10 AAV2. It actually was an insertion, which we would 11 have never been able to project an insertion versus 12 something that was already there. 13 So it was a tedious process of going 14 through a, one ordinary skilled in the art see 15 differences, one extraordinarily skilled in the art to 16 say these don't matter, these do matter, to ones that 17 after we did it, we were not correct. And she was not 18 correct in her five amino acid choices. That went into 19 a clinical trial based on the assumptions of 20 importance, only to be found out later that we took 21 probably the wrong capsid in, if that makes sense. 22 Q. Yeah, and we'll get to all that. We'll get 23 to all that. 24 A. We tediously found out it was just one, and 25 it wasn't an amino acid that we could line up and say Page 120
1 MS. MORRISON: Objection to the form. 2 BY MS. TOKER: 3 Q. You're not disagreeing with that? I mean, 4 not disagreeing with what he did but that he said. I'm 5 just asking if you agree that that's what he said? 6 A. That's what he said. 7 Q. Okay. And you here were looking at the 8 crystal structure, right, to narrow down which 9 positions to mutate, and you were doing that by looking 10 at the positions in the linear sequence that were in 11 relevant biological positions in terms of the 12 three-dimensional structure? I don't know if I said 13 that right. I'm going to try that again. 14 You were narrowing down -- you were 15 narrowing down the number of positions to mutate by 16 looking at the 36 spots you had identified based on the 17 linear sequences and looking to see which of those 18 positions in the three-dimensional crystal structure 19 appeared to be of most relevance to the biology of the 20 virus? 21 MS. MORRISON: Wait just a second. 22 Objection to the form. 23 A. We followed the process that was alignment, 24 83 were aligned. Of the 83, there was the triaging of 25 those that were conserved or redundant. In fact, the Page 119	1 here's the obvious one to pick, because it did not 2 exist in the AAV2 backbone. 3 Q. Right. We'll look at that. I want to look 4 at something you said. You said you wouldn't have made 5 the substitution that Dr. Schaffer identified because 6 it was redundant, right? 7 A. The amino acids, they were like a 8 difference in charge or difference in hydrophobicity or 9 bulky in groups. Those would have been the changes 10 that a crystallographer or someone saying oh, here's a 11 significant difference. This one might be important. 12 Q. Right. And that changes, to be clear, was 13 a change from serine to asparagine, right? 14 A. Correct. 15 Q. And you would say the same thing for a 16 threonine to asparagine, that would be redundant too, 17 right? 18 A. So I had threonine insertion to one that 19 did not exist. 20 Q. No -- I'm sorry, go ahead. Go ahead. 21 A. I'm confused if you were referring to our 22 insertion? 23 Q. I wasn't. I was just saying you were 24 saying serine to asparagine would be a redundant 25 substitution. I'm not talking about the threonine Page 121

1 assertion we'll get to that. 2 A. A redundant substitution. 3 Q. And similarly, threonine -- 4 MS. MORRISON: I'm sorry, I just want 5 to make sure that he's finishing. 6 A. Yeah, the -- the alike amino acids would 7 not have been targeted, because they were already 8 alike. 9 Q. And so you would not have targeted a 10 threonine to asparagine as a substitution, just like 11 you wouldn't have targeted a serine to asparagine? 12 A. So our serines to aspartic acids and 13 serines to alanine were done to specifically put no 14 charge or charge in there. 15 Q. Okay. Yeah, I was just going back to what 16 you said, that in this study, you didn't bother making 17 a substitution -- well, let's strike that. I think 18 that's fine. We'll get to it. We'll do it step wise, 19 okay. 20 So okay. Now, let's look back to page 12. 21 And on page 12 around line 22, there's a discussion 22 that begins of corresponding modifications. Do you see 23 that, it's sort of in the line? 24 A. Yes. 25 Q. Corresponding modifications can be -- I'm Page 122	1 line 27, where it says, it starts talking about a 2 selective amino acid change. Do you see that? It's at 3 the bottom of page 13, the last full paragraph, 4 according to the present invention? 5 A. Yes. 6 Q. A selective amino acid change -- it says, 7 sorry, now I've gone down to the last sentence in that 8 paragraph, it says: The selective amino acid change 9 results in the insertion and/or substitution and/or 10 deletion of less than about, and it's got a whole 11 string of numbers, but it ends with less than about 12 three contiguous amino acids, right? 13 A. Correct. 14 Q. So these amino acid changes that this '126 15 provisional is disclosing can be, in this instance it 16 says, as few as three contiguous amino acids, right? 17 A. That's what it says. 18 Q. All right. And the very next sentence 19 reduces it even further, right? It says: In 20 particular embodiments, only two contiguous amino acids 21 or even point mutations, i.e., one amino acid, are 22 inserted and/or substitute into and/or deleted from the 23 capsid. Do you see that? 24 A. Yes. 25 Q. And sort of jumping back to that paragraph Page 124
1 sorry, corresponding modifications can readily be 2 produced into other AAV based on the present disclosure 3 in view of the high level of skill in the art. A 4 corresponding modification can be an insertion and/or a 5 substitution and or a deletion. For example, a 6 modification that is an insertion in AAV2 may be a 7 substitution mutation in another AAV. 8 Do you see that? 9 A. Yes. 10 Q. So first of all, it does say that a person 11 of ordinary skill in the art would know how to 12 introduce these substitutions and insertions that are 13 being discussed here, right? 14 MS. MORRISON: Objection to the form, 15 to the extent it mischaracterizes the 16 document. 17 A. Based on the way we describe it, someone 18 could then follow up and do a substitution or a 19 deletion or an insertion. 20 Q. Right, okay. And a corresponding mutation 21 here doesn't really distinguish between an insertion or 22 substitution or a deletion. It could be any of those. 23 It could be a corresponding modification, correct? 24 A. A corresponding modification. 25 Q. All right. Now, let's look at page 13, Page 123	1 on page 13, this '126 provisional contrasts the work 2 that's being described here with previous work that 3 you've discussed, particularly of a paper by Hauck, 4 H-A-U-C-K, that involved domain swapping whole subunit 5 or large domain swaps between AAV serotypes, right? 6 A. You're pulling up a reference that we 7 referred to, or are you on a page? 8 Q. It's on the page right there, page 13, 9 sorry. It says: This approach is in contrast to 10 previous work with whole subunit or large domain swaps? 11 A. Yes. 12 Q. So you're contrasting this approach that 13 you all were taking in December 2004 with previous 14 approaches that involved large chunks of the AAV capsid 15 that were swapped, right? 16 A. We're contrasting that, right. 17 Q. You're saying we're not doing that. We're 18 not doing whole domain swaps. We, in 2004 here, are 19 doing much finer changes. We're doing even single 20 amino acid changes to change properties of AAV, right? 21 MS. MORRISON: Objection to the form. 22 A. So we're saying that in contrast to 23 previous work to swaps, one could approach this from 24 our strategy. 25 Q. And your strategy is even single amino acid Page 125

1 changes that could change the properties of AAV? 2 MS. MORRISON: Objection to the form. 3 A. Our strategy is reducing the 83 amino acids 4 down systematically to something that could be 5 potentially a single amino acid. 6 Q. Right. And, I'm sorry, go ahead. 7 A. As you said, it's not identifying a single 8 amino acid. It's a process of narrowing down which 9 requires, from my perspective of 50 years of this, an 10 expert who can do crystal structures and interpret 11 them, an expert that can do virology, and an expert 12 that can do molecular biology. I don't think that was 13 ordinary skill of the art in the majority of the 14 students that I trained, including myself. I wasn't 15 capable of carrying that out by myself. If it wasn't 16 an expert from the crystal capability to give us GPS 17 coordinates, we would have stayed forever with mutating 18 83 amino acids in combinations. Does that make sense? 19 Q. Yeah. There could be other ways though to 20 narrow down without using the crystal structure, right? 21 MS. MORRISON: Objection to the form. 22 A. None obvious that I'm aware of. I would 23 have used them, because then I didn't have to include a 24 collaborator that had a core expertise I didn't carry. 25 Q. Okay. Well, someone else might be able to Page 126	1 provisional. Now, you say that -- let's look at -- 2 let's go back to your declaration, actually, for a 3 moment. And let's look at page 14, paragraph 27. Let 4 me know when you're there. 5 A. I'm there. 6 Q. All right. You say that -- I'm looking at 7 the second sentence: By and large -- and I am sorry, 8 paragraph 27 begins with the phrase at the time, right? 9 A. Correct. 10 Q. And that's referring to in or around 2005, 11 at the time for the patent at issue here, correct? 12 A. Correct. 13 Q. Around 2005, you're saying: By and large, 14 mutagenesis and genetic engineering were not yet being 15 used for the goal of enhancing a specific AAV function 16 for clinical benefit, right? 17 A. Correct. 18 Q. But you were using it, right? 19 MS. MORRISON: Objection. Sorry, I 20 didn't mean to interrupt you. 21 BY MS. TOKER: 22 Q. You were using it in 2004? 23 MS. MORRISON: Objection. 24 MS. TOKER: I'm really sorry. Let me 25 finish my question. Page 128
1 use a different strategy to narrow down differences 2 between two serotypes to decide which amino acids to 3 test, right? 4 A. In 2004 and '5, I think we were the cutting 5 edge, because everybody else was doing fragment 6 swapping. 7 Q. Well, I mean, your student, Dr. Snowdy, 8 identified three amino acids to test without even doing 9 sequence alignment, right? 10 MS. MORRISON: Objection to the form. 11 A. I think Dr. Snowdy took NLS sequences and 12 tried to infer they were similar in AAV, which we were 13 not successful. 14 Q. Right. But he identified three amino acid 15 to test. He tested them, and found that one of the 16 three produced an enhancement of transduction, right? 17 MS. MORRISON: Objection to the form. 18 A. In that study, he stumbled into something 19 we didn't understand it, yeah. 20 Q. Sometimes science involves stumbling in, 21 but it's still a result, right? 22 MS. MORRISON: Objection to the form. 23 A. I think you build on fundamental 24 understandings. 25 Q. Okay. Let's keep going with the '126 Page 127	1 MS. MORRISON: I'm sorry. 2 BY MS. TOKER: 3 Q. You were using it in 2004? You were using 4 mutagenesis and genetic engineering for the goal of 5 enhancing a specific AAV function for clinical benefit, 6 right? 7 MS. MORRISON: Objection to the form. 8 A. I think what we were doing was still 9 fundamental basic research. Some of the outcomes were 10 some viruses were dead, some didn't make virus, some 11 were more infectious; and each and every one of those 12 things kept giving us a foundation of how to approach 13 what I think you were referring to, which is trying to 14 get viruses with specific "tropisms" for targeting 15 tissues of interest. 16 Q. Well, I'm just referring to what you're 17 referring to. You're saying that -- let's go on to the 18 next sentence. Maybe it will clarify it. One later, 19 actually. 20 For example, studies like the one by Hauck 21 that petitioner relies on explored how different 22 regions of the AAV capsid affected its ability to 23 infect muscle tissue. So you're -- 24 A. Where are you now? 25 Q. I'm sorry, I'm four lines up from the Page 129

1 bottom. 2 A. On what page? 3 Q. 14. 4 A. Okay. For example? 5 Q. Yeah. So you're saying here that a study 6 like Hauck was representative of the state-of-the-art 7 in 2005, right? 8 MS. MORRISON: Objection to the form. 9 A. It's for different regions of AAV capsid 10 affect its ability to affect muscle tissue. That was 11 one common practice, yeah. 12 Q. You're not really just saying it's one 13 common practice. You're saying that was what people in 14 the art were doing at the time, swapping the domains, 15 they weren't using mutagenesis and genetic engineering 16 for the goal of enhancing a specific AAV function for 17 clinical benefit, right? 18 MS. MORRISON: Objection to the form. 19 Go ahead. 20 A. So let me put it in historical perspective. 21 AAV2 is the only serotype that we had 22 sequence information on. In the beginning, it was the 23 first serotype, the one I cloned as a graduate student, 24 and that was the only candidate that was being utilized 25 for gene therapy. It was later determined that in Page 130	1 to it. 2 Q. Yeah, exactly. So that does suggest that 3 the work with AAV2 here was intended ultimately for the 4 clinic, right? 5 MS. MORRISON: Objection to the form. 6 A. It was the one we had the most 7 understanding of. 8 You have to realize, there was a paper that 9 described AAV1, but there was no molecular information 10 that it replicate the same way, that it packaged the 11 same way. We ended up doing those studies to confirm 12 that. 13 Q. Fair enough. I'm just saying that the goal 14 of the work in the '126 provisional was to enhance 15 muscle tropism of AAV2 with the idea that it could be 16 used ultimately in the clinic? 17 MS. MORRISON: Objection to the form. 18 A. I think the goal was to understand what 19 caused AAV1 to be preferred to muscle. The two that we 20 generated was used by our clinical colleagues to go 21 into the clinic. Eventually it was determined that was 22 not the virus that should have been used because we got 23 it wrong. 24 So you could split this into there was a 25 basic science approach to understand things and then Page 132
1 animals, there may be other serotypes that were 2 preferred over to. So anyone stilled in the art 3 quickly switched to other serotypes, like 1 for muscle 4 8 for liver, so forth and so on. 5 As a consequence of that, basic science was 6 trying to understand why is 1 different than 2 and 7 that's where we were coming in with basic science 8 approaches to genetically decipher what is responsible. 9 Until then, large domains were being swapped, and large 10 domains basically said something in this region of 11 interstate blah, blah, blah, causes your radios to go 12 out. 13 What we end up finding out was that 14 systematically taking that approach down to traditional 15 mutagenesis and genetics and crystal structures, 16 that -- it's this tower that's responsible for it. And 17 that was just a foundation that was constantly being 18 laid down. For clinical applications, everybody went 19 from AAV2 serotypes to natural serotypes that had 20 predilection to go into certain cell types, muscle, 21 liver, lung, whatever they were. 22 Q. You even told me before though with respect 23 to the '126 provisional, that you chose AAV2 because it 24 had been -- 25 A. It had a clinical safety approach attached Page 131	1 there were clinical approaches that people took just to 2 quickly try and see if they have success in humans. 3 Q. Okay. So your statement then in paragraph 4 27 of your declaration about the state-of-the-art in 5 2005, you're not saying that people weren't doing 6 single amino acid changes to affect properties of AAV, 7 you're just saying they weren't doing them and then 8 directly putting that into the clinic, is that what 9 you're saying? 10 MS. MORRISON: Objection to the form. 11 A. I get confused when she objects and you say 12 something. So I'm going to ask you to say that again? 13 Q. Sure. I'm going back to your sentence, it 14 says by and large mutagenesis and genetic engineering 15 were not you yet being used for the goal of enhancing a 16 specific AAV clinical function for clinical benefit? 17 A. That's correct. 18 Q. Okay. Let me just back up with one more 19 question. 20 Sometimes you have a goal to do something, 21 and it doesn't work out, right, but it was still your 22 goal, fair? 23 MS. MORRISON: Objection to form. 24 A. I think that's a common part of science. 25 Q. Okay. So you could have had a goal, in Page 133

1 fact you did bring one of the mutants from this 2 provisional, the 2.5 mutant, you brought -- that's 3 disclosed here, we'll get to that, you took that and 4 tried to take that into the clinic, right? 5 A. I did. 6 Q. Someone? 7 A. Someone took what we generated and then 8 take it into the clinic. 9 Q. And in fact, your company AskBio worked on 10 taking that into the clinic and helped with the 11 clinical trial? 12 A. The company helped with the clinical trial. 13 Q. So the company AskBio was involved in the 14 clinical trial of one of the mutants disclosed in this 15 provisional, correct? 16 A. That's correct. 17 Q. All right. And yet you're still saying to 18 me that the goal of making these mutants had nothing to 19 do with the clinic, even though your company took one 20 of the units and put it into the clinic? 21 MS. MORRISON: Objection, 22 argumentative. Go ahead, Dr. Samulski. 23 A. My goal was to sort out the biology of AAV. 24 There were other people's goals, which I found in the 25 structure, to take reagents and tried to turn them into Page 134	1 to a basic research fundamental foundational question. 2 MS. MORRISON: Objection to the form. 3 A. So specifically the safety was we had never 4 seen toxicity with AAV2 in animals. That's what that's 5 referring to. 6 Q. Well, toxicity in animals is irrelevant to 7 bringing a drug to market, right? 8 MS. MORRISON: Objection to the form. 9 A. I think one would argue that doing studies 10 in animals is a precursor for what you might expect in 11 humans. Our precursor was that AAV2 in animals did not 12 cause toxicity, but AAV2 was the first virus to show 13 toxicity. 14 So attributes, one would say safety based 15 on animal studies was not a predictor of what was 16 happening in the clinic. And therefore you continue to 17 look at attributes to determine what's responsible for 18 this, what's responsible for that. 19 Prior to that, people trying to make 20 capsids that were going into the clinic were looking 21 for efficiency, what you might refer to as tropism. 22 AAV8 was the second capsid to go into the clinic for 23 hemophilia after AAV2 showed toxicity. 24 Q. All right. So it's your testimony that 25 this '126 provisional does not disclose anything being Page 136
1 clinical delivery systems. 2 Q. But in the disclosures in the '126 3 provisional itself suggest that this work was being 4 undertaken with an eye to the clinic, because it 5 discusses maintaining favorable properties of AAV2, 6 such as purification needs and safety, right? 7 MS. MORRISON: Objection. Objection 8 to the form. 9 A. I would disagree, because AAV2 was the 10 first virus to show toxicity in humans, and we would 11 have preferred to take a capsid that did not show 12 toxicity. So while you can identify attributes that we 13 thought were favorable, and we could still produce it 14 and still purify it, there were other attributes that 15 were and still are liability to an AAV2 backbone. It's 16 the first capsid to ever show toxicity in humans. 17 Q. Fair enough. But you actually say that you 18 were trying to preserve purification and safety would 19 seem like clinical rather than basic research? 20 MS. MORRISON: Objection -- sorry. 21 BY MS. TOKER: 22 Q. I will start over again. 23 You say that you were trying to preserve in 24 AAV2 its ease of purification and known safety, which 25 sounds like things relevant to the clinic rather than Page 135	1 used for the goal of enhancing a specific AAV function 2 for clinical benefit, even though you and your company 3 took one of the mutants here and put it into patients, 4 right? 5 MS. MORRISON: Objection to the form 6 and argumentative. Go ahead. 7 A. I would say the company made a choice, 8 which I considered a risky choice, to take an unproven 9 capsid into the clinic, which ultimately failed. 10 Q. Okay. But to get back to then sort of the 11 state of the art, but you do agree that people at the 12 time were making single amino acid changes in AAV 13 capsid to affect function of the capsid, you and 14 others, right? 15 MS. MORRISON: Objection to the form. 16 A. What we were doing was a procedure we 17 referred to as wreck and check. Mutate something and 18 see what the phenotype was, systematically making 19 changes across the AAV genome. 20 Q. You considered the '126 provisional to 21 disclose a wreck and check approach? 22 A. Yeah, absolutely, because in the end we 23 narrowed it down to one amino acid. We couldn't have 24 done that without a wreck, check, wreck, check, wreck, 25 check. You had to do five, then you did four, then you Page 137

1 did four plus the fifth one missing, then you did 2 three, then you did three with the second and the third 3 one missing. So by the time you do that, that wreck 4 and check is an exponential how many variations are 5 there. 6 And then after we found out the threonine 7 was the primary amino acid that was responsible, we put 8 each of the 20 amino acids into that one location and 9 found out hey, guess what, Mother Nature was wrong, 10 threonine is not the best amino acid, it's glutamic 11 acid, it's phenylalanine, and it's aspartic acid. 12 So the exhaustive study of trying to take 13 what we understood from crystal structures and treated 14 the tropisms ended up being like a five or seven year 15 wreck and check study. 16 Q. So just to be clear, your definition of 17 wreck and check, you are looking to enhance function, 18 and you succeed in that, but you still call it a wreck 19 and check, because you're looking here to enhance 20 skeletal muscle transduction of AAV2, that's what 21 you're describing as a wreck and check strategy, 22 correct? 23 MS. MORRISON: Objection to the form 24 and argumentative. Go ahead. 25 A. No. I think you're looking at the wreck Page 138	1 voice at the witness. 2 A. It doesn't bother me. 3 Q. I apologize again. 4 A. You don't need to apol -- I think what we 5 missed in this communication, you have a propensity to 6 make an assumption I'm looking for something to be 7 better. I'm looking for an outcome that tells me a 8 biological activity resides in a domain. So it could 9 be it makes ten times more virus but it's not 10 infectious. So that's an important piece of 11 information for how do you increase production. But as 12 a consequence, you get a negative outcome. So it's not 13 always trying to get a positive result. 14 The wreck and check was what role do you 15 play, if any, and is it plus or minus in this 16 background. It could be like Snowdy saw. As soon as 17 he put in the 158 mutation, that was aspartic acid, it 18 went to a negative virus. It didn't transduce. It 19 made particles, but they were not infectious. That's a 20 wreck and check approach, trying to map domains or 21 amino acids that may be important. 22 Q. What he did in the study we talked about 23 was he chose specific residues, because they were 24 subject to a particular process called phosphorylation, 25 right? Page 140
1 and check the wrong way. 2 Wreck and check was just a laboratory word 3 for make every possible mutant one by one determine 4 what its phenotype is and continued to narrow it down 5 to find out who's the key players. 6 Q. But I'm talking about the strategy you used 7 in the '126 provisional, which is to make specific 8 mutations that you're hoping will enhance function, 9 why? Because you're taking the guidance of Mother 10 Nature, you're looking at another sequence that Mother 11 Nature invented that has a function that you want. You 12 consider that to be wreck and check? 13 A. Correct. 14 MS. MORRISON: And just objection to 15 that last question as argumentative. 16 BY MS. TOKER: 17 Q. Okay. Just so we're clear, then we know, 18 so in your declaration, when you say wreck and check, 19 you mean a procedure in which you align two sequences, 20 look for differences and substitute in amino acids from 21 one sequence to the other hoping to improve the 22 properties of one of the two sequences? 23 MS. MORRISON: And objection, again, 24 to form, and also argumentative. And I'll 25 just, again, ask counsel to stop raising her Page 139	1 A. He was looking at things that were next to 2 basic regions to see if -- how phosphorylation would 3 impact them. 4 Q. Right. And there were specific amino acids 5 that could be phosphorylated, right? 6 A. Could be phosphorylated. 7 Q. Not every amino acid can be phosphorylated, 8 right? 9 A. No, and it was still a wreck and check 10 approach. 11 Q. It seems like you're calling all 12 mutagenesis a wreck and check approach? 13 A. Because we don't know the outcome. It's a 14 complete unknown. 15 Q. Okay. So any process, in your view, that 16 where you don't know the outcome you consider wreck and 17 check? 18 A. Correct. 19 Q. Okay. So let's put aside the term wreck 20 and check then, and let's just talk about what was 21 happening in your '126 provisional. And what was 22 happening was you were using, and it says that 23 selective engineering. 24 A. Where are we? 25 Q. I'm looking at the summary of the Page 141

1 invention, back on page one. 2 A. The declaration? 3 Q. No, I'm sorry, the '126 provisional, which 4 is Exhibit 1059. 5 A. Okay. 6 Q. Yeah, it says that the inventors have 7 identified the key amino acids of AAV1 responsible for 8 enhanced in vivo transduction and selectively 9 engineered these amino acids into the backbone of AAV2 10 as well as other AAV, right? 11 A. Correct. 12 Q. Okay. That sounds a lot like a process 13 that you say wasn't going on in 2005. You say that 14 mutagenesis and genetic engineering were not being used 15 for the goal of enhancing a specific AAV function for 16 clinical benefit. And so the only difference here is 17 you're something what was going on in '126 first of all 18 wasn't being done for clinical benefit? 19 MS. MORRISON: And objection to the 20 form and argumentative. And, again, I'd ask 21 counsel to please stop raising her voice. 22 A. I think I am saying that correct. 23 Q. Okay. 24 A. I'm also saying this was not ordinary 25 people skilled in the art. These were extraordinary Page 142	1 Q. You don't say ordinary skill in the art. 2 You say by and large mutagenesis and genetic 3 engineering were not yet being used. You don't say 4 ordinary skill in the art. 5 A. I'm defining what's not being used. 6 So wind yourself back 20 years and sit in 7 an environment where someone discovered new serotypes 8 AAV8, AAV9, and they do studies on animals and says 9 this is the best for muscle. This is the best for 10 liver. The majority of the community ran into those 11 capsids and developed them to go into the clinic. 12 We were systematically -- I'm trying to 13 understand the biology, what was involved in an 14 infectious cycle, binding to the receptor, entering the 15 cell, trafficking to the nucleus, escaping in the 16 nuclear pore, un-coding, second strand synthesis, end 17 transcription and gene expression. We were doing that 18 in parallel. Others were simply just trying things in 19 the clinic. 20 If you remember Terry Flodds'(phonetic) 21 first clinical trial, there was no promoter in that 22 cassette. His gene was too big to carry a promotor. 23 The expression came from terminal repeats of AAV, which 24 no one knew they had transcription activity. 25 So there was an example of trying to treat Page 144
1 people skilled in the art, which was why we were the 2 first to be able to do this and the only ones to do it. 3 You can't find another publication during this time 4 that demonstrates these outcomes. 5 So we're looking at, in my opinion, someone 6 doing something out of the ordinary, cutting edge, to 7 find observations to bring back to the rest of the 8 community and approach to take. If we failed on all of 9 that, it would have never gotten published, and 10 everybody would have continued to be using large chunks 11 of capsid swapping in order to map and/or enhance 12 features of AV biology. 13 Q. Okay. So all the statements in your 14 declaration, where you're saying no one was doing this 15 at the time this selective engineering, this, you know, 16 substitution of single amino acids to enhance function, 17 you just mention no one except you was doing it? 18 A. Pretty much. 19 Q. Okay. But you didn't mention that you were 20 doing it. Your statements are much broader. You say 21 in your declaration no one was doing it. 22 A. I'm saying if you look at the literature, 23 look at the publications and look at what was being 24 done at that time, you won't find people ordinary 25 skilled in the art doing what we were doing. Page 143	1 a patient with a terminal disease, with a reagent that 2 doesn't even have a classical promotor to express the 3 therapeutic transgene. 4 There was a sense of desperateness to get 5 into the clinic, and then there was a community of 6 Mavis and others that were doing systematic molecular 7 virology on these systems. 8 Q. So you agree with me that you at the time 9 were doing single amino acids in the AAV capsid to 10 change properties of AAV? 11 A. Based on Mavis' instructions. Without her 12 instructions, we would have left with 83 amino acids 13 narrowed down to 36, going through what Nick and others 14 were doing, systematically changing them one by one 15 without any scanning mutants, which was another wreck 16 and check approach. 17 Q. Well, you don't have to do alanine 18 scanning, do you? You can substitute in mutations from 19 the other stereotype, which is what you say you were 20 doing here. You were looking -- 21 A. So -- 22 Q. Let me just finish my question. 23 You were looking to AAV1 as guidance for 24 which mutations to make in AAV2? 25 MS. MORRISON: Objection to the form. Page 145

1 A. So realize that it was only from the 2 alanine scanning mutants that we knew which regions to 3 start to look at. So you needed a roadmap before you 4 could actually focus. Or you were looking at Ad3 amino 5 acids to change between 1 and 2, and that was not a 6 feasible approach in any lab. 7 Q. Well, you said you narrowed it down from, 8 you know, the 83 to the 36 based on the sequence 9 alignments, right? 10 A. Sequence amino acids that were different, 11 but technically were still hydrophilic or hydrophobic 12 or phosphorylated and so forth. They were being 13 dismissed based on let's keep our fingers crossed and 14 hope those are not important. 15 Q. That's not an alanine scanning, right? 16 A. No. Prior to that, we were doing alanine 17 scanning mutants. Without Mavis' guidance, we were 18 doing alanine scanning mutants. 19 Q. Right. Certainly others in the field had 20 done alanine scanning mutants and published it before 21 2004, correct? 22 A. Yeah, that was the state of the art. 23 Q. Including single amino acid mutations -- 24 MS. MORRISON: Objection. 25 MS. TOKER: Counsel, I would Page 146	1 Q. Between serotypes? 2 A. Correct. 3 Q. You were doing single amino acid changes in 4 the hope of altering the function of the capsid, 5 correct? 6 MS. MORRISON: Objection to the form. 7 Go ahead. 8 A. We were -- we were guided to change amino 9 acids, not single amino acids, a group of amino acids. 10 A group of amino acids got us to regions 11 where we saw biological changes, and only after that, 12 and those studies were published, which a group took 13 into the clinic. We were systematically saying which 14 of the five are the most important, or are they all 15 important, and from that we ended up deducing that 16 guess what, we were wrong. There were not five amino 17 acids. There was one amino acid. And guess what, it 18 wasn't swapping one for another. It was actually an 19 insertion of something that did not exist. 20 So we would have never been able to guess 21 to put an insertion into something that didn't exist 22 before. And then added the one we inserted, we ended 23 up finding out how foolish we were, because we inserted 24 one that we thought was important because AAV1 carried 25 it. But, again, Mother Nature said no, no, no for Page 148
1 appreciate it if you could avoid 2 interrupting me. 3 MS. MORRISON: I'm really trying not 4 to. I apologize. 5 MS. TOKER: Okay. 6 Q. Others in the field, other than you, had 7 done single amino acid alanine scanning mutations, 8 right? 9 MS. MORRISON: Objection to the form. 10 A. Say it one more time? I know you're 11 objecting. 12 Q. Others in the field, besides you, before 13 2005, had done single amino acid alanine scanning 14 mutations in the AAV capsid, right? 15 A. They were doing that, correct. 16 Q. And had published that, right? 17 A. Correct. 18 Q. And you here were not doing alanine 19 scanning -- 20 A. This is the first time we transitioned, so 21 you're correct in your question. 22 Q. So in the '126 provisional, you were not 23 doing alanine scanning, and you were not swapping large 24 domains, correct? 25 A. Correct. Page 147	1 AAV2, it's not threonine, it's three other amino acids. 2 And if you would have tried to guess those three amino 3 acids into an insertion onto a capsid where you didn't 4 know that an insertion would make a difference, you 5 would have spun your head for many, many years trying 6 to decide which one do you pick and where do you go. 7 MS. MORRISON: I don't want to 8 interrupt you. If there's a place to break 9 in the next couple of minutes, that would be 10 great. I'm sorry. 11 MS. TOKER: I'll do one more and then 12 we can break. 13 Q. So in terms of your statement just now that 14 you had no idea how to insert this threonine, right, 15 that's not what you said in the '126 provisional, is 16 it? 17 A. I don't remember which part of the 18 provisional you're referring to. 19 Q. So first of all, let's just take a look at 20 Figure 7, which is again at the back, I'm sorry, 21 there's no identifying way to get there, other than 22 define Figure 7. I'm sorry the '126 provisional 23 towards the end there's all the figures, and it's 24 Figure 7. You have to pass all the really messy 25 looking ones and keep on going. Page 149

1 A. Seven? 2 Q. Yeah, it's actually -- 3 A. Sorry. 4 Q. That's okay, seven. And this is a figure, 5 and we can look at the legend, but I think you can see 6 that this is a figure that is a sequence alignment of a 7 portion of the capsid sequences of AAV, right? 8 A. Correct. 9 Q. From different serotypes, correct? 10 A. Correct. 11 Q. And the top two serotypes are AAV1 and 12 AAV2, right? 13 A. Correct. 14 Q. And we've been talking about the '126 15 provisional, as comparing AAV1 and AAV2 and looking at 16 differences and making changes in AAV2 to make it look 17 and act a little more like AAV1, right? 18 MS. MORRISON: Objection to the form. 19 A. Correct. 20 Q. All right. And so let's look at the 21 threonine that actually we're talking about, and we can 22 go back and trace through that this is in fact the 23 threonine, but for purposes of this discussion you see 24 in AAV1, there is a threonine around amino acid, it's 25 actually like right after 264 or so in this alignment. Page 150	1 in AAV1, where if you made it in AAV2, the two 2 sequences would align at that position, correct? 3 MS. MORRISON: Objection to the form. 4 A. Align better. 5 Q. Align better, right? 6 A. Correct. 7 Q. All right. So this is a single amino acid 8 insertion in AAV2 from AAV1, right? 9 MS. MORRISON: Objection to the form. 10 A. Correct. 11 Q. And these substitutions and insertions were 12 being done according to the '126 provisional to enhance 13 the muscle transduction of AAV2, right? 14 MS. MORRISON: Objection to the form. 15 A. It was a study to try to understand 16 important domains between AAV1 and AAV2 related to 17 muscle. 18 Q. This is not a whole domain. This is a 19 single amino acid, right? 20 MS. MORRISON: Sorry, objection to the 21 form. 22 A. It sits in a region. 23 Q. Okay. So this is a single amino acid 24 insertion from AAV1 to AAV2 to try to improve the 25 muscle transduction of the AAV2, right? Page 152
1 That this is a threonine that appears in AAV1 that does 2 not appear in AAV2, correct? 3 A. Correct. 4 Q. And you can see that the amino acids around 5 that threonine do align pretty well between the AAV1 6 and AAV2, correct? 7 MS. MORRISON: Objection to the form. 8 A. The alanine amino acid, which is next to 9 the serine, which would be next to the threonine does 10 not align, but other than that, the rest of them are 11 aligned in some degree. 12 Q. You in fact chose to mutate that alanine 13 because it doesn't align here, right? 14 A. I would have. 15 Q. You did in this '126, okay? Yes? We can 16 look, but do you have any reason to doubt that? 17 MS. MORRISON: Objection to the form. 18 A. That was our wreck and check approach. 19 Q. Okay. So, again, wreck and check is 20 looking at AAV1, seeing where the amino acid sequence 21 differs and making those mutations in AAV2 to look a 22 little more like the AAV1 sequence, right? 23 A. Correct. 24 Q. All right. And so this threonine is not 25 widely out of the blue. This threonine is an insertion Page 151	1 MS. MORRISON: Objection to the form. 2 A. Let me just explain it. This is the HI 3 loop, it's a loop, and so I would call it a domain, 4 because it's not partnered in the beta barrel sheet. 5 It's got a loop. So it's the loops and some of them 6 the sizes were different, and some of them the loops 7 are not the same size. 8 MS. MORRISON: Counsel, is it a good 9 time for lunch? 10 MS. TOKER: I'm going to ask one more 11 question. 12 MS. MORRISON: Okay. You said one 13 more question almost ten minutes ago. 14 MS. TOKER: I didn't, actually. I 15 said a couple more. 16 THE WITNESS: I'm okay. I want to 17 finish this. So there's clarity in what's 18 going on here, if you don't mind. 19 BY MS. TOKER: 20 Q. Thank you, Dr. Samulski, I appreciate that. 21 A. No problem. 22 Q. Let's just look also at page 4 of the '126, 23 and I promise I won't keep you much longer, and we can 24 get lunch. 25 A. Before we leave the Figure 7 -- Page 153

1 Q. Yeah. 2 A. I want to simply bring to your attention 3 the threonine and AAV1, the threonine and the AAV1. 4 Q. Yes, that's the one we were talking about, 5 right? 6 A. Right. When we put an end to AAV2, as I 7 mentioned to you, that was the only amino acid that 8 made a difference. And when we put in 20 amino acids, 9 there were three others. So based on that logic, I'm 10 just curious what would happen to AAV1 if you took the 11 threonine out? 12 Q. Good question. But not what I can explore 13 in this deposition right now. Maybe at the break or at 14 the end of the day. 15 A. Removing the threonine makes AAV1 the 16 strongest muscle capsid it's ever been. It's 17 completely contrary to everything we described and 18 discussed in here. 19 Q. That's interesting. But let's go back to 20 what you knew at the time in 2004, and that's on page 21 4. And I want to look at -- so this is -- I just want 22 to look at the legend to Figure 5, and we can look at 23 Figure 5, but I'm mostly interested in the legend, 24 which sighs the threonine insertion at position 265 is 25 responsible for the majority of the enhanced muscle Page 154	1 AFTERNOON SESSION 2 THE VIDEOGRAPHER: The time is 2:06. 3 We're on the record. 4 BY MS. TOKER: 5 Q. Welcome back, Dr. Samulski, from lunch. I 6 will let you maybe finish chewing your chocolate. 7 But I can ask you to look at a page while 8 you're chewing, so I'll do that, but I won't expect you 9 to answer yet. 10 So I want to look, again, at the '126 11 provisional Exhibit 1059. 12 A. Okay. 13 Q. Let me know -- you're all done? Okay. 14 A. I'm ready. 15 Q. Let me ask you, did you speak with your 16 counsel during the luncheon? 17 A. No. 18 Q. Not about the substance or anything about 19 this deposition? 20 A. No. No. No. 21 Q. Okay, good. Let's take a look at page 9 of 22 the '126 provisional, Exhibit 1059. You have a 23 definition of tropism here at the top of the page. Do 24 you see the first sentence, it says the term tropism as 25 used herein? Page 156
1 tropism of 2.5. Do you see that? 2 A. Yes. 3 Q. So that 2.5 is the mutant that we have been 4 talking about, that you or your company took to the 5 clinic, right, the 2.5, right? 6 A. Correct. 7 Q. And you're saying here that although there 8 were several mutations in the 2.5 -- in AAV2.5, the 9 '126 provisional says that that threonine insertion 10 that we just looked at in Figure 7 was responsible for 11 the majority of the enhanced muscle tropism of this 2.5 12 variant, correct? 13 A. Correct. 14 Q. Okay. So this single amino acid insertion 15 is what the '126 provisional is saying was responsible 16 for transferring the enhanced, or at least some of the 17 enhanced muscle tropism of AAV one into AAV2? 18 MS. MORRISON: Objection to the form. 19 A. Correct. 20 MS. TOKER: Okay. Lunch. 21 THE VIDEOGRAPHER: The time is 1:12. 22 We're off the record. 23 (Luncheon recess.) 24 (Continued on next page.) 25 Page 155	1 A. Um-hum. 2 Q. Okay. It says: The term tropism, as used 3 herein, refers to preferred entry of the virus into the 4 cell, optionally and preferably followed by expression, 5 e.g., transcription and optionally translation of 6 sequences carried by the viral genome in the cell, 7 e.g., for a recombinant virus, expression of the 8 heterologous nucleotide. Do you see that? 9 A. Yes. 10 Q. Okay. So that's page 9, lines 1 to 4, 11 right? 12 A. Correct. 13 Q. Okay. And so this definition of tropism is 14 also different from the one that you gave in your 15 declaration, in that it allows for inclusion in the 16 definition of expression, right, entry and then 17 optionally and preferably expression of the transgene 18 or the genes carried by the virus in the cell, right? 19 MS. MORRISON: Objection to the form. 20 A. This one still is accurate with respect to 21 the entry steps. It's the virus into the cell. 22 With respect to expression, you can control 23 tropism by having a muscle-specific promotor or a 24 neuronal-specific promotor, but that wouldn't have 25 anything to do with the viruses' tropism. Page 157

1 So in this setting, where it says optimally 2 preferably following expression, it has to be a 3 ubiquitous promotor, so it's not influencing the 4 tropism of where the virus is at play. 5 Does that make sense? 6 Q. I'm not entirely sure. I think my question 7 is a little simpler. It says: The term tropism here 8 can include expression of the viral genome in the cell, 9 right? 10 A. And I'm make a distinction between 11 translation and expression to that can be artificially 12 controlled by the promoter to imply a tropism, but it's 13 not -- imply a viral tropism, but it's not the virus 14 that's tropic, it's the promotor. 15 Does that make sense? 16 Q. Well, in your Haberman paper, you suggested 17 that the promotor did affect the tropism. 18 A. The promotor was influencing the tropism, 19 not the virus. 20 Q. Okay. So this definition, again, it may be 21 correct in your mind, but it's different from what you 22 said in your declaration, right? 23 MS. MORRISON: Objection to the form. 24 Go ahead. 25 A. No -- yeah, I would say no again, because Page 158	1 A. No. That's not correct. 2 Infectivity is different than expression. 3 Infectivity are the steps that carry out the virus 4 getting all the way to the step of un-coding and 5 transcription. That would be an infectious 6 infectivity. 7 Expression can be any byproduct of 8 transcription, microRNA, tissue-specific expression, 9 antisense, that wouldn't be related to the virus or the 10 tropism. That would be your transgene cassette. 11 So there should be a clear distinction 12 between the cargo and how the cargo defines tropism and 13 the virus and how the virus defines tropism. 14 In this case, tropism is dependent on the 15 virus, which is step one and two by an entry. Again, 16 it toggles over to the transgene, which is the cargo, 17 to define the rest of the tropism, that's being 18 referred to as tropism. 19 Q. You did in your declaration say that 20 expression is not part of tropism, right? 21 MS. MORRISON: Objection. 22 BY MS. TOKER: 23 Q. You said expression is part of infectivity, 24 right? 25 MS. MORRISON: Objection to the form. Page 160
1 if you read it refers to preferred entry into -- of the 2 virus into the cell. So that still requires the first 3 steps of virus binding and entry, which is my 4 definition of tropism. 5 After that entry, the rest of this can be 6 modified by promoters, translation and other elements 7 that are not related to the virus. 8 Q. In your declaration, you classified that as 9 infectivity, right? 10 A. Yeah, that would be infectivity. 11 Q. But here you don't classify it as 12 infectivity. You classify it as part of tropism? 13 MS. MORRISON: Objection to the form. 14 Go ahead. 15 A. Again, I don't -- it's making a distinction 16 here. The term Tropism, as used herein, it's not 17 referring to it as tropism, as I described it, but it's 18 modifying the tropism, as I described it. And that 19 tropism, as modified, is beginning to allow expression 20 and transcription and other activities of the virus 21 infection help define the tropism. 22 Q. In your declaration, you distinguish 23 tropism and infectivity, and you classified expression 24 as part of infectivity. Here you're including 25 expression in tropism. Page 159	1 Go ahead. 2 A. No. You might need to go back and look at 3 those, if you feel the need. 4 Q. Sure. We can turn to page 22 of your 5 declaration, paragraph 38, where you say in the middle 6 of the paragraph: Infectivity, on the other hand, 7 generally refers to the steps that occur after the AAV 8 vector reaches the cells of intake, i.e., intake of AAV 9 vector, into the cell translocation to nucleus and the 10 expression of the gene of interest, right? 11 A. Correct. 12 Q. So here you include expression of a gene of 13 interest under infectivity? 14 A. So I'm making a distinction. Expression, 15 if it's not driven by a specific promoter that 16 determines tropism. 17 Q. First of all, you don't say that in the 18 declaration, right? 19 A. No. It's understood that if you go into a 20 cell as a virus, the virus promoters would be 21 promiscuous in those cells. When you target the gene 22 therapy, you're now using transgene cassettes where 23 you're designing the promoters. They may or may not be 24 specific for the cell where the virus has tropism for. 25 So once again, the AAV that is being Page 161

1 described could go into muscle and brain at the same 2 time but only expressed in muscle because the tropism 3 and the promoter determines that. The only expression 4 you see is the expression in the cell that that 5 promoter is specific for. 6 So you have to be careful if you start 7 making generic comments about my definitions, and then 8 referring to the -- what are we in? 9 Q. The '126? 10 A. The '126. 11 Q. So you have a different definition here in 12 the '126 than you just defined in your declaration? 13 A. I think it says "the term tropism used 14 herein." So it's already making a clear distinction 15 that it's different. So you're right in saying it's 16 different than the one I'm declaring. 17 Q. Okay. Now, let's take a look at, still in 18 the '126 provisional that we've been looking at, let's 19 take a look, please, at page 18, and I'd like to look 20 around -- 21 A. Declaration or the -- 22 Q. Sorry, the '126 provisional, Exhibit 1059, 23 and I'm looking at page 18. 24 A. Okay. 25 Q. Okay. I wanted to look at the second Page 162	1 Q. I was looking at the paragraph right before 2 that that was talking about a mutation in AAV2. 3 A. I was reading the paragraph below that was 4 the 717s. 5 Q. No problem. The 717s are in different AAVs 6 if you look, and I was just starting with the AAV2 7 position. 8 A. Okay. My bad. 9 Q. No problem. Just take a minute, and let me 10 know when you've looked at that paragraph on AAV2. 11 A. Okay. 12 Q. Okay. So the one I'm talking about is the 13 '126 provisional describes an AAV2 mutant containing an 14 amino acid substitution at position 716 in the capsid, 15 correct? 16 A. That's what it says. 17 Q. I want to skip over the paragraph that you 18 were looking at, it says that in 17, and 18 and 19, and 19 go to line 20 that begins in particular aspects of the 20 embodiments described in the previous two paragraphs. 21 Let me know. It's at line 20. Do you see that? 22 A. Yeah. 23 Q. And so we're now talking about -- so the 24 paragraph we looked at before is talking about a single 25 amino acid substitution at position 716 in AAV2, Page 164
1 paragraph on page 18, beginning the invention also 2 provides. Do you see that? 3 A. Um-hum. 4 Q. It says: The invention also provides a 5 chimeric virus vector comprising a chimeric AAV2 6 capsid, comprising an amino acid substitution, and 7 amino acid position 716 in the AAV2 capsid subunits or 8 at the corresponding position in an AAV2 capsid that 9 has been modified to comprise non-AAV2 sequencing. 10 Do you see that? 11 A. Yes. 12 Q. All right. So we talked before about the 13 threonine exception. Now we're talking about a 14 different mutation in AAV2 that's being described, 15 correct? 16 A. Correct. 17 Q. And this mutation is an amino acid 18 substitution at position 716? 19 A. 717. 20 Q. Where do you see 717? 21 A. In the other embodiment, the invention 22 provides a chimeric -- I'm sorry. Are we on the same 23 page? 24 Q. Is it okay if I clarify? 25 A. Sure. Page 163	1 correct? 2 A. Correct. 3 Q. And this paragraph, starting at line 20, is 4 going to talk about specific substitutions at that 5 position, agreed? 6 A. Agreed. 7 Q. And so what it says is in particular 8 aspects of the embodiments described in the previous 9 two paragraphs, meaning one of them is this position 10 716 in AAV2, the substitution is a substitution of an 11 amino acid that cannot be phosphorylated for an amino 12 acid that can be phosphorylated, e.g., threonine. 13 Do you see that? 14 A. Yes. 15 Q. So here they're describing a single amino 16 acid substitution in the AAV capsid that consists of 17 substituting in an amino acid that is not 18 phosphorylatable in place of an amino acid that is 19 phosphorylatable, such as threonine, correct? 20 A. Correct. 21 Q. That's sort of what Dr. Snowdy did. He 22 substituted an amino acid that could not be 23 phosphorylated at position 181 for an amino acid that 24 could be phosphorylated at that position, correct? 25 MS. MORRISON: Objection to the form. Page 165

1 A. That's correct. 2 Q. And if you look at the last sentence in the 3 paragraph we were just looking at, so starting at line 4 25, it says one particular chimeric virus. So now it's 5 getting even more specific, right? We talked about 6 having a substitution at position 716. We talked about 7 the position being one where you substitute in a 8 nonphosphorylatable amino acid in one that can be 9 phosphorylatable. And down at the bottom here it says: 10 A particular chimeric virus comprises an asparagine 11 substitute at this position into the capsid subunit. 12 Do you see that? 13 A. Yes. 14 Q. And so this is describing an AAV2 capsid 15 with a single amino acid substitution, and the 16 substitution is specifically an asparagine being 17 substituted in in place of a phosphorylatable amino 18 acid that was the original, correct? 19 MS. MORRISON: Objection to the form. 20 A. Say it again? 21 Q. Not a problem. 22 So this last sentence is describing an AAV2 23 capsid mutant in which position 716 -- in which at 24 position 716, an asparagine has been substituted in at 25 that position, right? Page 166	1 phosphorylated, such as threonine, correct? 2 MS. MORRISON: Objection to the form 3 of the question. 4 A. So it means there has to be a 5 phosphorylated form there in order to choose to put one 6 in that can't be phosphorylated. 7 Q. Right. Agreed? 8 A. But if there's not a phosphorylated form 9 there, you would not put something in that cannot be 10 phosphorylated. 11 Q. Fair enough. But if there is a 12 phosphorylatable amino acid, such as threonine at 13 position 716 of AAV2, this sentence at the end there is 14 saying substitute it in -- substitute in an asparagine 15 to replace that threonine at position 716, correct? 16 MS. MORRISON: Objection to the form. 17 A. It gives you ten options. 18 Q. And then it chooses one? 19 A. And then it says out of ten, here's one 20 particular one. 21 Q. And the one particular one is putting in an 22 asparagine to replace the threonine, right? 23 A. That's the one that's chosen, yeah. 24 That's the one that was chosen as a 25 particular one, asparagine, out of ten. Page 168
1 A. I'm listening. 2 Q. Do you agree so far? 3 A. Well, I'm confused between the previous two 4 paragraphs. The substitution of an amino acid that 5 cannot be phosphorylated for an amino acid that can be 6 phosphorylated, example, threonine. And then it says 7 alternatively, substitutions can be serine, tyrosine, 8 glycine, alanine, proline, serine(sic), blah, blah, 9 blah, blah, blah into the capsid subunit. One 10 particular chimeric virus comprises an asparagine 11 substituted at the position into the capsid subunit. 12 So basically puts any and every possible 13 scenario at that position. 14 Q. Let's go back, because I don't think so. 15 So the very last sentence says that one 16 particular virus, okay, so let me just back up. 17 We started at paragraph at 17 with an AAV2 18 capsid with a single amino acid substitution at 19 position 716, agreed? 20 A. Correct? 21 Q. Then on line 20, it says that -- well, to 22 get a little bit more specific, that substitution at 23 position 716 is of a particular type. It's of the type 24 where you're substituting in an amino acid that cannot 25 be phosphorylated for an amino acid that can be Page 167	1 Q. Yeah, so it chose that one out of ten, 2 right? 3 A. Yeah. 4 Q. Okay. And then you're aware that 5 Dr. Schaffer is suggesting to substitute in an 6 asparagine for a threonine, correct? 7 A. That is my understanding, yes. 8 Q. Let's look at the next paragraph, the last 9 paragraph on page 18. 10 So now it's discussing a different position 11 or different region of the AAV2 capsid. In this 12 paragraph, starting at position 28, it's discussing 13 positions in the sort of 400 range, around 450. 14 A. Uh-hum. 15 Q. Agreed? 16 A. Correct. 17 Q. And so it says -- and the last sentence of 18 page 18, so that's lines 32 through 34 -- 35, sorry, 19 let me count, 32 through 35, it says: In particular 20 embodiments, the substitution is a substitution of an 21 amino acid that cannot be phosphorylated for an amino 22 acid that can be phosphorylated, e.g. with threonine, 23 right? 24 A. Correct. 25 Q. So it's sort of repeating the disclosures Page 169

1 that we just went through with respect to position 716, 2 but now it's talking about making the same kind of 3 substitution, meaning substituting an amino acid that 4 cannot be phosphorylated for an amino acid that can in 5 this different region of the AAV2 capsid, correct? 6 A. Right. It gets eight examples of where it 7 could be: 447, 448, 449, 450 and 451. So you have a 8 basket of opportunities to insert something, and it's 9 requesting that they go from threonine, which can 10 de-phosphorylate it back to nonphosphorylated amino 11 acids, and then it draws attention to asparagine, 12 that's one of particular. 13 Q. Right. So, again, and it also draws 14 attention to position 450, because it says on page 19 15 at line 3, e.g. substitution of the threonine at 16 position 450 and AAV2, right? 17 A. It says that. 18 Q. So it's drawing attention to position 450 19 in AAV2 where there is a threonine. Then it says in 20 the last sentence: One particular chimeric virus 21 comprises an asparagine substituted at this position, 22 meaning 450, for example, into the capsid subunits, 23 correct? 24 A. Correct. But I wouldn't leave it alone, 25 because in the next paragraph it says: A chimeric Page 170	1 asparagine substituted at this position into the capsid 2 subunits. That's lines 3 to 5 on page 19. 3 A. Yeah, from that position, it draws 4 attention to that. 5 Q. To that, meaning to substituting 6 asparagine -- 7 A. At 450. 8 Q. -- right? And that was the same as we saw 9 at position 716, correct? 10 A. Correct. 11 Q. All right. Now, let's take a look at, 12 please, at page 15 of the '126 provisional. So the 13 same document we've been looking at, Dr. Samulski, but 14 back to page 15. 15 So if you look on page 15 at around line 16 19, there's a sentence that begins: The invention 17 further provides chimeric virus vectors comprising a 18 chimeric AAV capsid, comprising a selective amino acid 19 insertion and/or substitution selected from the group 20 consisting of. Do you see that? 21 A. Yes. 22 Q. And then there's a list. It's a list that 23 makes up this group, and it says that, you know, you 24 can pick any one of these. And so option D at the very 25 bottom it says: A selective amino acid insertion Page 172
1 capsid with a position 454, 455, 56, 57, 58, and then 2 it draws attention to a specific position 457, and 3 basically goes through the same thing again. 4 Q. Except there it's substituting an 5 asparagine for a glutamine, right? 6 A. Glutamine. 7 Q. We're talking -- let's focus on we've got 8 position 450 and position 716, or the '126 provisional 9 says it draws attention, as you say, to substituting 10 the threonines at those positions with asparagine, 11 right? 12 A. With nonphosphorylated amino acids, of 13 which asparagine is chosen as one. 14 Q. Yes, which asparagine is called out as one 15 particular mutant in this case, right? 16 MS. MORRISON: Objection to the form. 17 BY MS. TOKER: 18 Q. Fair enough? 19 MS. MORRISON: Objection to the form. 20 A. I think it ends up saying it represented 21 embodiment, and then asparagine is substituted in the 22 capsid subunit. 23 Q. Well, that's the paragraph we weren't 24 looking at. I was looking at the one above where it 25 says: One particular chimeric virus comprises Page 171	1 and/or substitution that results in an amino acid 2 substitution at amino acid position 716 in the AAV2 3 capsid subunits or corresponding change in other AAV 4 capsids. Do you see that? 5 A. Yes. 6 Q. That's at position 716 that we have just 7 been talking about in the AAV2, correct? 8 A. Correct. 9 Q. And then part E on the next page, page 16, 10 says: A selective amino acid insertion and/or 11 substitution that results in an amino acid substitution 12 at amino acid positions 447 to 453, e.g. position 450, 13 in the AAV2 capsid subunits or corresponding change in 14 AAV capsid, right? 15 A. That's correct. 16 Q. That is calling out that position 450 that 17 we were talking about before, right? 18 MS. MORRISON: Objection to the form. 19 A. That does, in addition to 457. 20 Q. Right. And it mentions that the selective 21 amino acid substitution could be at position 450 in 22 particular, correct? 23 A. It's 457. 24 Q. Right. And at the bottom, at the end of 25 the list, it says: Wherein, the chimeric virus vector Page 173

1 has enhanced transduction and/or an altered 2 immunological profile, as compared with the parent rAAV 3 vector, right? 4 A. That's what it says, correct. 5 Q. Right. So it's saying here that the 6 invention described in this '126 provisional includes 7 making single amino acid substitutions at positions 8 such as 716 and 450 wherein the mutant has enhanced 9 transduction compared with the parent, correct? 10 MS. MORRISON: Objection to the form. 11 BY MS. TOKER: 12 Q. Sorry. I didn't -- I didn't hear an 13 answer. 14 A. Yeah, I was waiting for you to say it 15 again. 16 Q. Oh, sorry. I was just saying that so in 17 this part that we were just looking at on pages 15 to 18 16, the inventors are saying that their invention 19 includes an AAV2 capsid with a single amino acid 20 substitution at position 16(sic) where the mutant has 21 enhanced transduction compared with the parent virus, 22 parent vector, agreed? 23 MS. MORRISON: Objection to the form. 24 I think you said position 16? 25 MS. TOKER: I meant to say position Page 174	1 includes what you call selective amino acid 2 substitutions at position 716, and then another one at 3 position 450, where they -- a fact of the substitution 4 is enhanced transduction as compared to the original 5 AAV2. 6 A. The parent recombinant AAV. So is that 2, 7 the parent? 8 Q. Well, in this case it is, because it 9 classifies position 716 in the AAV2 capsid, and 10 position 450 in the AAV2 capsid. 11 A. The way you read that, that would be 12 accurate. 13 Q. Okay. Now, let's look at page 56 of the 14 '126 provisional. 15 A. 26? 16 Q. 56, I'm sorry, 5-6. Position -- page 56 of 17 the '126 provisional. 18 A. Okay. 19 Q. All right. And these are the claims in the 20 '126 provisional, and I'd like to look at the claim 21 number 57. Do you see that? 22 A. Yes. 23 Q. And so that claim says: A chimeric virus 24 vector comprising -- and then it has an A and a B; the 25 A is a recombinant adeno-associated virus, rAAV genome; Page 176
1 716, I apologize. 2 Q. Do you want me to repeat it once more? 3 A. Please. 4 Q. Third time is a charm. 5 So the inventors of the '126 provisional, 6 of which you are one of these inventors, say that the 7 invention includes an AAV2 capsid mutant with a single 8 amino acid change at position '716 wherein that mutant 9 has enhanced transduction as compared to AAV2. 10 Do you agree? 11 MS. MORRISON: Objection to the form. 12 Apology. 13 A. Compared with the parent, AAV -- 14 recombinant AAV vectors that were -- what am I missing 15 here? 16 Q. Yeah, I'm just -- I'm not trying to make 17 this -- I'm just trying to say, this part is saying 18 that the invention includes single amino acid 19 substitutions at position 716 or position 450, wherein 20 the mutant has enhanced transduction as compared to the 21 original AAV2. 22 A. And these are based on AAV1, 6 and 7 23 serotypes. 24 Q. Well, here it's just saying that the 25 invention includes -- I mean, sure, but the invention Page 175	1 and B, a chimeric AAV capsid comprising a selective 2 amino acid insertion and/or substitution that results 3 in an amino acid substitution at amino acid position 4 450 in the AAV2 capsid subunits or corresponding change 5 in other AAV capsids. Wherein, said chimeric virus 6 vector has enhanced transduction, as compared with the 7 parent rAAV vector. Do you see that? 8 A. Yes. 9 Q. So this claim is describing a mutant AAV2 10 with a single amino acid substitution at position 450, 11 where the mutant has enhanced transduction compared 12 with the original AAV2? 13 MS. MORRISON: Objection to the form. 14 A. I think it's a little bit broader amino 15 acid insertion and/or substitution. So it could be an 16 insertion and a substitution or insertion or a 17 substitution. 18 Q. Sure. My question -- I should have 19 probably clarified. 20 This claim, one of the things that it is 21 describing is mutant AAV2, with a single amino acid 22 substitution at position 450, is where this mutant has 23 enhanced transduction as compared with the original 24 AAV2? 25 A. Correct. Page 177

1 Q. And then we're just going to look down some 2 more. Are you familiar with the idea of a dependent 3 claim versus an independent claim from all of your 4 patents, Dr. Samulski? 5 A. So I would benefit from a refresher, but I 6 know from the word itself it's dependent on the prior 7 claim or something to that effect. 8 Q. Okay. And let me know, I'll try to refresh 9 your recollection, which is that the dependent claim 10 includes everything in the independent claim and then 11 adds something onto it, an additional limitation. 12 Does that sound like what you're familiar 13 with? 14 A. Yeah, that makes sense. 15 Q. Okay. So then looking at these, right, you 16 can see that claims -- we're just going to be looking 17 at 58 through 61, okay. So we're not going to look at 18 62, which starts another claim at the bottom. 19 But you can see that 58 depends on 57; 59 20 depends on 57; then 60 depends from 58; and 59 and 61 21 depends from 60. So this is all sort of a bunch of 22 dependent claims, all depending in different ways from 23 the independent claim, correct? 24 A. Understood. 25 Q. Okay. So we can walk through the whole Page 178	1 went through, right? 2 A. Correct. 3 Q. It's now saying the chimeric virus vector 4 of claim 57, wherein, the chimeric virus vector 5 comprises a chimeric AAV2 capsid comprising an amino 6 acid substitution at amino acid position 450 in the 7 AAV2 capsid. Do you see that on claim 59? 8 A. Yes. 9 Q. All right. So claim 59 is limiting claim 10 57 to having to be a chimeric AAV2 capsid, right? 11 MS. MORRISON: Objection to the form. 12 BY MS. TOKER: 13 Q. Right? 14 A. In the AAV2 capsid, that's what it says. 15 Q. And then it has to also be an amino acid 16 substitution, as opposed to an insertion. 17 MS. MORRISON: Objection to the form. 18 BY MS. TOKER: 19 Q. Correct? 20 A. That's what this says, an amino acid 21 substitution. 22 Q. And then if we look at 60 now, 60, you 23 know, depends from either 58 or 59, but since we're 24 looking at 59, the chimeric virus vector of claim 59, 25 wherein the substitution is a substitution of, and then Page 180
1 thing, but I'll just start -- let's just see how, at 2 least claim 58, narrows it down and sort of how we get 3 down to 61. 4 So 58, sorry, 59 talks about the chimeric 5 virus vector of 57, right? Wherein the vector 6 comprises a chimeric AAV2 capsid comprising an amino 7 acid substitution at amino acid position 450 in the 8 AAV2 capsid. Do you see that? 9 A. Yes. 10 Q. So this dependent claim limits it. You 11 mentioned before that the 57 claim was broader in the 12 sense it could have an insertion and/or substitution, 13 right? 14 MS. MORRISON: Objection to the form. 15 A. Yes, this one has to have a non-AAV2 16 sequence. 17 Q. Well, I'm actually looking at 59, sorry. 18 A. 59. 19 Q. Sorry, let's go back one second. I didn't 20 mean to confuse you. 21 59 and 58 both depend from the independent 22 claim, but I was focusing on 59, okay. So let's go 23 back to that. 24 So 59 says the chimeric virus vector of 25 claim 57, so that's the independent claim that we just Page 179	1 it lists a whole bunch of things: Serine, tyrosine, 2 glycine, alanine, proline, valine, leucine, isoleucine, 3 asparagine or glutamine for threonine, right? 4 A. Right. 5 Q. You have this mutant AAV2 with single amino 6 acid substitution at position 450, and you could be 7 substituting in any of these listed amino acids for the 8 threonine that already exists at that position 450? 9 A. If threonine is at that position, correct. 10 Q. If it is, that's what we're substituting, 11 correct? 12 A. Correct. 13 Q. And then claim 61 now as a furtherance 14 says: The chimeric virus vector of claim 60, which we 15 just read, wherein the substitution is a substitution 16 of asparagine for threonine, correct? 17 A. That's what it says, correct. 18 Q. Okay. So if there is -- and we'll look if 19 there is a threonine at position 450 in AAV2, this 20 claim 61 is describing a single amino acid substitution 21 where that threonine at position 450 is replaced with 22 an asparagine, right? 23 MS. MORRISON: Objection to the form. 24 A. That's what it says. 25 Q. Okay. Now, let's take a look at Figure 1B, Page 181

1 I think we looked at that before in the '126 2 provisional. So we're staying with that, and we're 3 going to turn to Figure 1B towards the end. And what I 4 want to do is look at this figure and take a look at 5 the positions. We have been discussing position 450 in 6 AAV2 and position 716 in AAV2, okay, Dr. Samulski? 7 A. Yes. 8 Q. Are you there? 9 A. Yes. 10 Q. And you see, I think we briefly talked 11 about this before, this is an alignment of the capsids 12 of AAV1 and AAV2, correct? 13 A. That's correct. 14 Q. And just so we don't get too confused, the 15 numbering of the capsid is a little bit different 16 between the two because, you know, they don't always 17 have exactly the same number of amino acids, right? 18 A. Right. The AAV1 has an insertion at 65, 19 and AAV2 doesn't. So you have to adjust it by assuming 20 that that insertion doesn't trick all the other amino 21 acids to be in a different location. 22 Q. Right. And so the numbering of AAV1 and 23 AAV2, while it starts out the same, right, you know, 24 it's beginning the line, you can see that it starts out 25 with both start with one, in the second line both have Page 182	1 Q. And I'll give you a hint. There's a big 2 old arrow right on top of it, but we can count if you 3 want. There's a large arrow that points down near the 4 end of the fourth line. And the number on the top says 5 456, right, but that applies to the AAV1 numbering, not 6 AAV2 numbering, fair? The numbers on the top? 7 A. Correct. 8 Q. Yes, okay. So do you agree, from your 9 counting, that we're looking where the large arrow is, 10 right near the number 456, we're looking at the 11 threonine at positions 450 in AAV2? 12 A. Correct. 13 Q. And you can see that the corresponding 14 amino acid in AAV1 is asparagine, correct? 15 A. Correct. 16 Q. All right. So all of the disclosures we 17 looked at, where it was singling out, substituting in 18 asparagine, asparagine was chosen there and singled out 19 because that's the corresponding amino acid in the 20 AAV1, right? 21 A. Correct. 22 Q. And this '126 provisional is looking to 23 import some of the properties of AAV1 into AAV2? 24 A. Correct. 25 Q. All right. So it's looking into the Page 184
1 115, in the third line both have 229, by the fourth 2 line you can see that AAV1 is starting at amino acid 3 343, and AAV2 is starting at amino acid 342, right? 4 A. Correct. 5 Q. And that's just because, as you said, there 6 was one extra amino acid that that threonine we talked 7 about before, right, in AAV1 and AAV2 doesn't have, 8 correct? 9 A. Correct. 10 Q. All right. So if we look, let's look at 11 position 450. And I just wanted to go through that, 12 because the numbering is slightly different. And when 13 we talk about position 450 and position 716, that's the 14 numbering in AAV2, agreed? 15 MS. MORRISON: Objection to the form. 16 Q. Well, I just mean that in everything we 17 talked about so far, when they say, you know, 18 substitute in position 450 of AAV2 and substitute in 19 position 716 of AAV2, that's the numbering of AAV2, 20 correct? 21 A. Correct. 22 Q. Okay. So we need to find first position 23 450 in AAV2, right? 24 MS. MORRISON: Objection to the form. 25 A. Correct. Page 183	1 sequence of AAV1, and in particular to importing that 2 asparagine in place of the threonine AAV2 to improve 3 the muscle transduction of AAV2? 4 MS. MORRISON: Objection to the form. 5 A. It's pointing to that, and that was an 6 example used, but it actually mentioned multiple amino 7 acids in that area 450, 451. 8 And if you look next to the threonine, it's 9 next to the -- you see there's a phenylalanine there, 10 and there's above it, it's a difference. So there's as 11 much of a change between the next amino acid in 1 and 12 the amino acid in AAV2, that the 450 example would not 13 supersede any of the other options, in my opinion. 14 Q. I was just talking about what was called 15 out in the '126 -- we looked at how it was called out 16 to substitute asparagine, right? 17 A. In that example. In that example. 18 Q. And in fact, there's a big old arrow, right 19 there in that spot, right, in this figure, to call 20 further attention to it? 21 A. In that figure, correct. 22 Q. All right. And now let's look at position 23 716 in AAV2. And that's in the last line. And, again, 24 the numbering is slightly different for the same reason 25 between AAV1 and AAV2. But if you look near the number Page 185

1 720, there's another big arrow that points to threonine 2 716 in AAV2. Do you see that? 3 A. I do. 4 Q. And once again, the corresponding amino 5 acid in AAV1 is asparagine, right? 6 A. Correct. 7 Q. So with the disclosures about substituting 8 in asparagine for threonine at position 716 is also 9 being done because asparagine is the corresponding 10 amino acid in AAV1, correct? 11 A. In this example, correct. 12 Q. Yes. And we read earlier that in the '126 13 provisional, that it discloses that one of the reasons 14 for making this substitution is that threonine is 15 phosphorylatable and asparagine is not, correct? 16 MS. MORRISON: Objection to the form. 17 BY MS. TOKER: 18 Q. Correct? 19 A. That's correct. 20 Q. And so the idea here, which is similar to 21 the results in Snowdy's thesis, is that substituting in 22 a nonphosphorylatable amino acid for a phosphorylatable 23 amino acid in the AAV capsid can enhance transduction 24 of the AAV serotype? 25 MS. MORRISON: Objection to the form. Page 186	1 and found out oh, gee, this was not an optimum change. 2 It should have been aspartic acid, glutamic acid and 3 phenylalanine. 4 So I think trying to draw a strong 5 conclusion that one would do that, one has the option 6 to do that, but I don't think it would infer the 7 positive outcome that we're looking for, because we've 8 seen so many examples where it's gone in the opposite 9 direction. 10 Q. Just to be clear, when you talked about the 11 changes that Snowdy made to the three 12 nonphosphorylatable amino acids, right, there were no 13 negative consequences from changing the 14 phosphorylatable amino acid to a nonphosphorylatable 15 amino acid? 16 A. Correct. 17 Q. There were two that had no effect at all? 18 A. Correct. 19 Q. And a third that enhanced function? 20 A. Correct. 21 Q. Okay. So there was no bad effect in 22 Snowdy -- 23 A. But -- 24 Q. Just let me finish the question. I promise 25 I will let you answer. Page 188
1 A. So I think you're making an assumption that 2 a change is going to be an enhancement and not a 3 detriment. And I think in Snowdy's example, we also 4 saw where he changed the amino acid, it had a negative 5 effect. 6 And here you have two locations you're 7 trying to choose, and you -- the one skilled in the art 8 is making the assumption that if I change one or both 9 of them, they're going to end up being positive effect. 10 But I think we learned our lessons multiple times that 11 it's a dangerous game to play, the assumption that that 12 will end up with a positive or negative effect. 13 If you look at the 720 location that Mavis, 14 who directed us to change that amino acid that was in 15 AAV1 to the amino acid into AAV2, that change, which 16 was obviously very different between the two amino 17 acids, failed to make a difference in the enhancement 18 of muscle transduction. 19 So there is a clear example of the wreck 20 and check analogy, where each and every one of these 21 are as good as it gets as anything else. 22 And I would say if he chooses asparagine 23 over the other amino acids that were chosen, you may 24 fall into the same paradigm, where we chose the 25 threonine but then decided to change all 20 amino acids Page 187	1 There was no bad, no negative effect that 2 was found from substituting in nonphosphorylatable 3 alanine for phosphorylatable serine and threonine, 4 correct? 5 MS. MORRISON: Objection to the form. 6 A. So in that example, the answer was yes. If 7 you take the reference that I think we used in the 8 thesis of John, Jen and Jan, there are two amino acids 9 at position 11, 12 of the teanogen(sic), where when one 10 was changed, it had no effect, and the other one downed 11 the activity 84 percent. 12 So it was, again, unpredictable what would 13 be the outcome. 14 So between proteins and between structures 15 and between serotypes, I don't think there's a guiding 16 principle of one rule follows -- all follow the same 17 rule. I don't know if that makes sense or not. 18 Q. Sure. I was just clarifying that when you 19 had said there was a negative effect from substituting 20 from a nonphosphorylatable, that that wasn't -- 21 A. Technically, I got what you're saying, 22 yeah. 23 Q. So I just want to make sure we agree that 24 when Snowdy substituted in a nonphosphorylatable amino 25 acid as a single amino acid change in AAV2, he either Page 189

1 got no change in transduction or an enhancement, 2 correct? 3 A. Correct. 4 Q. Okay. And so this '126 provisional, I 5 mean, I don't think it's guaranteeing anything, but 6 it's saying, you know, we focus on similarly, in a way, 7 a couple of spots where we think about making a single 8 amino acid change in AAV2, to substitute in a 9 nonphosphorylatable residue for a phosphorylatable one, 10 thinking that it might also enhance transduction. 11 MS. MORRISON: Objection to the form 12 of your question. 13 A. I think you can make those assumption(sic), 14 but I think they would be little -- little confidence 15 that those changes would yield outcomes that you're 16 referring to. 17 I'm going to step back and draw your 18 attention to what we understood during that time, okay. 19 2002, the crystal structure came out. 20 Mavis interpreted it for a handful of us, and we came 21 up with what was called eight variable regions: VR1, 22 VR2, VR3, VR4, and then they were the loop regions, HI 23 loop, loop GH, so forth, and so forth. It ended up 24 determining that only changes in variable region 8 25 would alter tropism. Variable regions in 8 -- loop Page 190	1 MS. MORRISON: Objection to the form. 2 A. So I think Snowdy was looking at 3 phosphorylated and nonphosphorylated by what we assume 4 were basic regions for nuclear localization. 5 So we had a roadmap that was already 6 defining like the variable regions I just described. 7 This was defining another motif that would be 8 important, and therefore he was changing them in the 9 context of this must be important because of these 10 basic regions that are next to it. 11 Q. Right. And he found that that wasn't in 12 fact the case, so he came up with saying I got the 13 opposite result from what I thought, so maybe this is a 14 different mechanism that's leading to this enhanced 15 transduction that I'm seeing, right? 16 MS. MORRISON: Objection to the form. 17 A. That's the way he interpreted the data. 18 Q. And this comes after Snowdy's work, right? 19 This '126 provisional, this is December 2004, and his 20 thesis was already published and deposited in the 21 repository a year before this in December of 2003, 22 correct? 23 A. Correct. 24 Q. So his experiments were already out there 25 at the time of the '126 provisional, correct? Page 192
1 regions in HI were responsible for making the virus' 2 symbol the 665 locations in the HI loop. And so 3 alterations of amino acids there would have been 4 diminished over dose already identified by the variable 5 region to be critical for tropism. 6 So if you're looking for a similarity 7 between AAV8 and rh10 or AAV42 point whatever, and so 8 forth, the variable regions would have been the targets 9 that -- would have been and are the targets by which 10 tropism has been changed. Those are furthest out on 11 the surface. They interact with the receptors. They 12 determine whether you combine, and then they determine 13 whether you can gain entry. 14 The others seem to play a more structural 15 role in whether you assemble the quilt into a 16 meaningful structure, and we're probably less -- were 17 not targets. They were not targets. 18 Q. I was just really focusing on where you 19 point the reader, as it were, in the '126 provisional, 20 and you point the reader to looking at phosphorylatable 21 residues and nonphosphorylatable residues, correct? 22 A. Correct. 23 Q. Just as Snowdy, in fact, was focused on 24 looking at phosphorylatable residues and 25 nonphosphorylatable residues, correct? Page 191	1 MS. MORRISON: Objection to the form 2 of the question. 3 A. I would have to take your word. I don't 4 remember the dates well enough. 5 Q. Well, just -- I mean, we can look at his 6 thesis. His thesis is dated December, you know, 7 whatever it is, 2003? 8 A. 2003. 9 Q. Right. And this is December 2004, agreed? 10 A. Yeah. 11 Q. So his results were, you know, at least a 12 year before this '126 provisional, right? 13 MS. MORRISON: Objection to the form. 14 A. December 15, 2004. 15 Q. Correct. Would you like me -- I can show 16 you -- 17 A. That's okay. I think Snowdy's says 2003. 18 Q. So that would be at least a year before? 19 A. At least A year before publication. 20 Q. Would you agree? 21 A. Yeah. 22 Q. So his results showing that a 23 nonphosphorylatable amino acid -- let me start that 24 over again. 25 His result is showing that substituting in Page 193

1 a nonphosphorylatable amino acid for a phosphorylatable 2 amino acid in AAV2 improved transduction were already 3 known at the time that this was filed? 4 MS. MORRISON: Objection to the form 5 of the question. 6 A. He demonstrated an unexpected result. 7 Q. And the unexpected result was that 8 substituting in a nonphosphorylatable amino acid in 9 place of a phosphorylatable amino acid in the AAV2 10 capsid enhanced transduction? 11 A. Enhanced transduction in HeLa cells. 12 Q. Correct. Agreed? 13 A. Correct. 14 Q. All right. And that was done in 2003, 15 agreed? 16 A. Correct. 17 Q. And the '126 provisional was filed at the 18 end of 2004, agreed? 19 A. Correct. 20 Q. All right. So at the time the '126 21 provisional was filed, Snowdy's result was already 22 known? 23 MS. MORRISON: Objection to the form. 24 BY MS. TOKER: 25 Q. Agreed? Page 194	1 he did this that year, this is why this happened. 2 Because, in other words, he should, you're going to 3 argue that he's an inventor of the two -- of this 4 application just based on time, and I would say I don't 5 think that's how it worked. 6 I think independently we were getting 7 observations, and independently we drew conclusions 8 based on, you know, the chemistry of a charged amino 9 versus an uncharged amino acid, which was more kind of 10 understood in protein chemistry. 11 Q. I wasn't suggesting. I was more suggesting 12 that both of these pieces of work were looking at the 13 effects of substituting in a nonphosphorylatable amino 14 acid for a phosphorylatable amino acid and suggesting 15 that that might enhance transduction. 16 MS. MORRISON: Objection to the form 17 of the question. 18 A. Again, I would be cautious. Those things 19 typically can backfire on you. 20 Q. Perhaps, but we looked at a claim where you 21 specifically called out, you know, a claim in your 22 provision where you specifically called out making that 23 particular substitution, right, at position 450 of 24 substituting in the asparagine for the threonine, and 25 presumably you called it out because you thought it Page 196
1 A. Correct. 2 Q. So the '126 provisional, in a sense, is 3 recommending something similar, based on Snowdy's 4 result, which is you might be able to enhance -- 5 A. So -- 6 Q. Let me just finish. Then you can answer. 7 You might be able to enhance transduction 8 if you make a single amino acid substitution in AAV2 9 replacing a phosphorylatable amino acid with a 10 nonphosphorylatable? 11 MS. MORRISON: Objection to the form 12 of the question. 13 A. So I think you're making an assumption that 14 because one experiment was done, it leads to the 15 understanding of another experiment. And I would say 16 that probably wasn't the case. And in this example if 17 that were -- I want to make sure -- yeah. If that were 18 the case, Snowdy would have been a coinventor. We 19 would never admit someone that contributed something to 20 the understanding of the patent that was being issued, 21 which is why you see Mavis from Florida and Chengwen, 22 had his own position, and students and Dawn had gone to 23 Duke, was at the university. 24 So in that manner, I would say I don't 25 agree with the logic that you're following that because Page 195	1 could be useful in terms of providing a useful enhanced 2 property in the claim? 3 MS. MORRISON: Objection to the form 4 of the question. 5 A. Yeah, I think going on what my 6 understanding of how patents are written, we claimed 7 everything in an effort that we may end up obtaining 8 some of these claims. None of those examples were 9 proven to exist. 10 So as I said, there's just as much 11 probability that they could have a negative effect 12 instead of a positive effect. 13 So to make an assumption that there was 14 direction from one study on basic region alterations to 15 the patent application is more of a reach for me. I 16 think the positions of 450, 458, 716, in my opinion, 17 from what I understand of AAV biology, would not have 18 an impact, but they were included in the patent as the 19 original mutations that may have had an impact. 20 So I guess it's another word for saying 21 covering your bases. 22 Q. Fair enough. And one of the bases you 23 covered was that the threonine to asparagine mutation 24 in position 450 might enhance transduction? 25 MS. MORRISON: Objection to the form Page 197

1 of the question. 2 A. That's one of the covering the bases. 3 Q. Okay. 4 A. I guess I will also clarify, we didn't have 5 any data in those positions to argue for it. It was 6 more assumptions. 7 Q. Well, you had the data that that was the 8 corresponding amino acid in AAV1. You had those data 9 that you did the alignment, and in each case, you 10 looked at the corresponding -- 11 A. Impression. 12 Q. -- in AAV1, correct? 13 A. Correct. 14 Q. So that's some data? 15 A. But as I pointed out, there's other more 16 dramatic changes right next to it that one would say 17 those are data too that should be acknowledged as 18 important. 19 Q. And there are other things that are 20 acknowledged in this '126 provisional also. I'm just 21 simply pointing out that you had data about positions 22 450 and 716. You had other data about other things, 23 but you had data about those two positions having to do 24 with the corresponding amino acid at AAV1, right? 25 A. Correct. Page 198	1 Q. It's infinite, but Snowdy found a finite 2 change of one amino acid that did enhance -- 3 MS. MORRISON: Objection to the form. 4 A. I struggle with Snowdy, because I don't 5 think we're looking at it the same way. 6 Snowdy predicted where to go because of 7 basic regions that were similar to NLS's, otherwise we 8 would have never changed those putative amino acids 9 that could have been phosphorylated. 10 There are too many amino acids that could 11 be phosphorylated. And as an example, the Snowdy 12 results actually surprised us in that it flipped and 13 went the opposite direction. 14 Q. And we talked about that. All right. Fair 15 enough. 16 MS. MORRISON: Are you going to a 17 new -- 18 MS. TOKER: Yes, you want to take a 19 break? 20 MS. MORRISON: Maybe just take a few 21 minutes? 22 THE VIDEOGRAPHER: The time is 3:11. 23 We are off the record. 24 (Recess.) 25 THE VIDEOGRAPHER: The time is 3:26. Page 200
1 Q. Okay. And so therefore it's possible to 2 want to cover that as a base, as you said, you know, 3 based on that information, that there was a 4 nonphosphorylatable amino acid in AAV1 that might 5 enhance transduction of AAV2, if it were substituted in 6 for the phosphorylatable threonine at those positions, 7 correct? 8 A. Might enhance. It might make it unviable. 9 Q. Well, people don't usually claim unviable, 10 right? The idea was that you wanted to claim this in 11 case it turned out to be viable? 12 MS. MORRISON: Objection to the form 13 and argumentative. 14 A. I think I try to bring your attention to 15 why we always look for the positive. The other side is 16 unfortunately a stark part of this. 17 Q. Fair enough. Fair enough. But you were 18 claiming and describing the positive here? 19 A. The positive. 20 Q. And the positive being that that single 21 amino acid substitution could -- the positive side 22 would be that it could enhance transduction? 23 A. It may enhance transduction. It may need 24 to be both. It may need not to be either but something 25 next to it. It's infinite. Page 199	1 We're on the record. 2 BY MS. TOKER: 3 Q. Dr. Samulski, did you talk to your counsel 4 about the substance -- 5 A. No. 6 Q. -- of your deposition during the break? 7 A. Sorry. No. 8 MS. TOKER: I would like to ask the 9 court reporter, please, to mark this 10 document Exhibit 1060. 11 (The document referred to was marked 12 Deposition Exhibit Number 1060 for 13 identification.) 14 Q. Dr. Samulski, the court reporter has just 15 handed you what has been marked Exhibit 1060. 16 Have you seen this document before? 17 A. Yes. 18 Q. So this is a paper published in 2006 on 19 which you are the senior author, correct? 20 A. Correct. 21 Q. The title of this paper is Single Amino 22 Acid Changes can Influence Titer Heparin by name and 23 Tissue Tropism in different Adeno-Associated Virus 24 Serotypes, correct? 25 A. Correct. Page 201

1 Q. Now, this paper says received in June of 2 2006. Do you see that? 3 A. Correct. 4 Q. So presumably the work was carried out 5 sometime before June of 2006? 6 A. Correct. 7 Q. Okay. Was it carried out partly in 2005? 8 Do you know? 9 A. I would assume, but I don't know for sure. 10 Q. Okay. Now, this paper looks at AAV1 and 11 AAV6, correct? 12 A. Correct. 13 Q. We haven't really talked about AAV6 so far 14 today, right? 15 A. Correct. 16 Q. Now AAV1 and AAV6 differ in 6 amino acids, 17 correct? 18 MS. TOKER: Let's go off the record, 19 I'm sorry. 20 THE VIDEOGRAPHER: The time is 3:28. 21 We're off the record. 22 (Recess.) 23 THE VIDEOGRAPHER: The time is 3:29. 24 We're on the record. 25 Page 202	1 amino acid from AAV6 back to AAV1, correct? 2 A. Correct. 3 Q. Now, if you look at the second full 4 paragraph in the left-hand column that begins -- the 5 second paragraph that begins: Despite the high degree 6 of sequence homology. Do you see that paragraph? 7 A. Yeah. 8 Q. All right. It says here that AAV6 was 9 known to be a better liver transducer than AAV1, 10 correct? 11 A. Correct. 12 Q. And that AAV6 was also known to bind 13 heparin, while AAV1 was not known to bind heparin, 14 correct? 15 A. Correct. 16 Q. And that heparin binding could be related 17 to the ability of an AAV serotype to bind to a 18 particular cell receptor, heparin sulfate, correct? 19 A. Correct. 20 Q. And the purpose of making the mutants in 21 this paper, the Wu 2006 paper, was to investigate the 22 molecular determinants of the different liver tropism 23 of the AAV1 and AAV6, correct? 24 A. Partially. 25 Q. Okay. That was one purpose? Page 204
1 BY MS. TOKER: 2 Q. Dr. Samulski, right before this very short 3 break, we were looking at Exhibit 1060, your 2006 4 paper, correct? 5 A. Correct. 6 Q. And the first author is Wu on this paper, 7 right? 8 A. Zhijian Wu, correct. 9 Q. Okay. I might refer to this as the Wu 2006 10 paper, and if I do, you'll know I'm referring to the 11 Exhibit 1060, is that okay? 12 A. Okay. 13 Q. All right. Now, we were just saying that, 14 in, you know, this paper is looking at AAV1 and AAV6, 15 correct? 16 A. Correct. 17 Q. And that it is looking in particular at the 18 6 amino acids that differ between AAV1 and AAV6, right? 19 A. Correct. 20 Q. Now, I'm going to look down at the first -- 21 sorry, let me rephrase that. 22 In general, and this is spelled out in the 23 abstract, but in this paper, you created single amino 24 acid mutants in which you swapped amino acid from AAV1 25 into AAV6 and then vice versa where you swapped an Page 203	1 A. Correct. 2 Q. So at the bottom of this left-hand column, 3 it says, these observations -- meaning what was already 4 known at the time that the work here was done -- it 5 says: These observations suggest that the molecular 6 determinants of heparin binding and possibly liver 7 tropism might lie within the six amino acid residues 8 that differ between AAV1 and AAV6, right? 9 MS. MORRISON: Objection to the form 10 of the question. 11 A. That is correct. 12 Q. And so you were -- one of the things you 13 were doing in this paper is looking at the six amino 14 acids that differ between the two serotypes and making 15 mutations to see if one or more of those amino acids 16 would affect liver tropism of each serotype? 17 A. Wreck and check. 18 Q. Fair enough. But you're making single 19 amino acid mutations, swapping from one serotype into 20 the other, and looking to see the effect on liver 21 tropism? 22 A. So let me clarify. We made -- we published 23 the same amino acid changes. We had to make one, one, 24 one, two, two, two, three, three, three, four, four, 25 four, and after doing all of that, we came back to it. Page 205

1 It appeared that the single amino acids were 2 sufficient. But it wasn't just a single amino acid. 3 It was a comprehensive analysis of combinations, and 4 since there were only six, unlike between other 5 serotypes where there are 48 or 83, this was a doable 6 project. 7 Q. Okay. The ones that are described in this 8 paper say that we generated a series of AAV1 and AAV6 9 mutants by site-directed mutagenesis, right, in the 10 right-hand column? 11 A. Right. I'm clarifying the difference 12 between what was published and what was done, but go 13 ahead. 14 Q. Okay. But part of what was done was 15 swapping these amino acids between AAV6 and AAV1, 16 correct? 17 A. Correct. 18 Q. And part of the reason to do that -- let's 19 just talk about the swapping, since that's what's 20 discussed and what's described here the swapping, part 21 of the reason to do that, or to investigate the six 22 differences between AAV1 and AAV6 was to see whether 23 one or more of these amino acids would determine liver 24 tropism of the two serotypes? 25 A. That was an outcome of the study, yes. Page 206	1 Q. Sure. 2 A. So AAV6 is not a serotype. It was called a 3 serotype, and unfortunately it's identical to AAV1, 4 except for the six amino acids. It was a contaminate 5 that came out of Dusty Miller's lab. He grew it in the 6 presence of an adeno virus that had AAV2 in there, and 7 these six amino acids ended up in AAV1. So 8 Immunologically it's identical to AAV1. So 9 serologically, it's not a serologically distinct 10 capsid. 11 So that misnomer has unfortunately played 12 the field since inception. 13 So what we really had was AAV1, where 14 Mother Nature decided to pop in six amino acids at very 15 distinct locations. And we knew that one behaved 16 different than 1-6, as I would call it. And therefore 17 we were curious what combination of amino acids, or any 18 of the amino acids may -- were responsible for these 19 differences. That's why I said we went through one, 20 one, one, one, one changes, two, two, two, two, two 21 changes, so forth and so on. 22 The receptor for AAV1 is sialic acid, and 23 the same receptor is true for AAV6. 24 This virus is the first example of an AAV 25 having two receptors, and that it appeared to bind the Page 208
1 Q. Okay. Well, it also says at the bottom of 2 the left-hand column, you say that the observations, 3 meaning the information that was available in the prior 4 art, right, suggested that the molecular determinants 5 of heparin binding and possible liver tropism might lie 6 within the six amino acid residues that differ between 7 AAV1 and AAV1, right? 8 A. Right. 9 MS. MORRISON: Objection to the form 10 of the question. 11 BY MS. TOKER: 12 Q. So the paper is saying that based on the 13 information already known, it was suggested -- the 14 information already known suggested that these six 15 differences in amino acids between the two serotypes 16 might determine the differences in liver tropism 17 between the two serotypes? 18 MS. MORRISON: Objection to the form 19 of the question. 20 A. So somehow I have to back up in time so you 21 can understand -- help me better explain the logic 22 behind this. Are you okay with that? If not -- 23 Q. Go for it. 24 A. If not, all of these questions are going to 25 be compounded. Page 207	1 sialic acid, and it appeared to bind the heparin 2 sulfate. We didn't know which one it was using, if 3 either, at that point. 4 So, again, a mutagenesis study, it's like 5 taking a twin of AAV1 and trying to find out why Mother 6 Nature put in these six amino acids. 7 So in that backdrop, we ended up 8 determining that there was an amino acid at position 9 531 that had a charge cluster that allowed it to bind 10 the heparin and appeared to be what was responsible for 11 giving it a different tropid(sic) than its twin, the 12 AAV1. 13 So if you don't have that backdrop, the 14 things you might be extracting from this aren't related 15 to AV serotypes. AV serotypes don't have these type of 16 two receptor, two dominant receptors. This is an 17 artifact of a laboratory accident. 18 Q. Okay, understood. I will not call AAV6 a 19 serotype, I promise, or suggest that it is. 20 So, but what it does show, and what it 21 sounds like you were describing in this left-hand 22 column paragraph that we were looking at, is that the 23 information known at the time your work was done was 24 that these six amino acid changes to AAV1, that happen 25 to be made by Mother Nature, could and do cause a Page 209

1 difference in liver tropism between the two variants? 2 MS. MORRISON: Objection to the form. 3 A. That would be our premise. 4 Q. Is it okay to call them variants? 5 A. Yes. 6 Q. Okay. All right. So then you were looking 7 at these six residues to see if one or more of them was 8 responsible for the differences, for example, in liver 9 tropism between the two variants? 10 A. That's correct. 11 Q. Okay. And you tested heparin binding of 12 the virus mutants that you named in this paper, 13 correct? 14 A. Correct. 15 Q. And, again, you tested, at least as shown 16 here, you may have tested more, but you showed here the 17 testing of the six swapping amino acid mutants that you 18 made between the two variants, correct? 19 A. Correct. 20 Q. And you found that swapping in a particular 21 lysine from an AAV6 into AAV1 at position 531 conferred 22 the heparin-binding ability of AAV6 onto AAV1, correct? 23 A. Correct. 24 Q. And you describe that in the text as an 25 AAV1-E531K substitution, correct? Page 210	1 conferring heparin binding. So conferring an AAV6 2 phenotype onto AAV8? 3 MS. MORRISON: Objection to the form. 4 A. Heparin -- 5 Q. Not a phenotype, I'm sorry. Same property? 6 A. Same property of binding heparin. 7 Q. Fair enough? 8 A. Yeah. 9 Q. Okay. Now, you also tested the ability of 10 the AAV1-E531K mutant to transduce hepatocytes in 11 vitro, correct? 12 A. Point me to that. 13 Q. Yes, I will. I'm sorry, I should have. 14 I'm looking at the bottom right-hand column 15 of 11395, which is the, you know, the page with the 16 figures on it. 17 A. Yeah. 18 Q. So all the way at the bottom, we next 19 investigated whether -- do you see it? 20 A. Yep. Yep. 21 Q. Okay. We next investigated whether lysine 22 531, which appears to be a critical distinguishing 23 feature between AAV6 and AAV1, might possibly explain 24 the enhanced transduction potential of AAV6 compared to 25 AAV1 in hepatocytes in vitro and in vivo, correct? Page 212
1 A. Correct. 2 Q. All right. E, standing for glutamic acid 3 and K standing for lysine, correct? 4 A. Correct. 5 Q. Okay. And, again, just to be clear, that 6 is just a single amino acid change in the AAV1 capsid, 7 correct? 8 A. Correct. 9 Q. You actually also made the same 10 substitution, it says, on the next page, so that's page 11 11395 of this exhibit. I'm looking at the left-hand 12 column, sort of in the middle of the first incomplete 13 paragraph it says: Identical -- I'll wait until you 14 get there. I'm looking at page 11395. So the page 15 with the -- yeah, and the left-hand column under all 16 those figures. 17 A. Identical alterations? 18 Q. Yeah. Identical alteration in serotype 8 19 also confirmed heparin binding supporting the 20 importance of the K531 position in the type 6 VP3 21 capsid subunit. Do you see that? 22 A. Correct. 23 Q. So you made the same substitution of that 24 lysine from AAV6, you made it into AAV8 at that 25 position, and it also showed the same effect of Page 211	1 A. Correct. 2 Q. And hepatocytes are liver cells? 3 A. Correct. Correct. Hep G2 cells. 4 Q. Okay. And so these assays that you 5 describe here, on this page 11396, involved infecting 6 hepatocytes. I'll talk about, sorry, the in vitro ones 7 first. 8 So they involve infecting the hepatocytes 9 in vitro with the AAV1-E531K mutant, and then assaying 10 the amount of a marker transgene that was expressed, 11 correct? 12 A. Correct. 13 Q. And you found that the E531K mutation 14 substantially enhanced the transduction efficiency of 15 AAV1 in vitro in this system, correct? 16 A. Correct. 17 Q. Now, you also did in vivo experiments. So 18 so far you had found that in vitro it looked like this 19 single amino acid change at this position of 531 could 20 confer on AAV1 the hepatocyte transduction ability in 21 vitro of AAV6, correct? 22 MS. MORRISON: Objection to the form 23 of the question. 24 A. Correct. Let me not confuse you. 25 We were using the transthyretin promoter. Page 213

1 So we could only measure liver transduction. So if it 2 went somewhere else, we wouldn't have been able to 3 score that. 4 So tropism was not so much dictated by the 5 single amino acid change, it was the promoter that 6 allowed you to assay that enhancement. 7 Does that make sense? 8 Q. You're saying that you use the same 9 promoter cassette in the in vitro? 10 A. Yeah. So you go to the top of that column 11 it says: The human factor non-expression cassette was 12 driven by a liver-specific transthyretin promoter, 13 which was packaged into AAV1 and AAV1, blah, blah, 14 blah, blah, blah, blah. 15 So once the promoter is tissue specific, 16 whether it's brain, muscle, heart, liver, that's a 17 tissue you can score with -- the virus could be 18 everywhere, but you're scoring the tissue by which the 19 promoter functions in. 20 So we would only have been able to score 21 transduction in liver, not that it changed to tropism, 22 it allowed you to score and enhance infectivity. 23 Q. Sorry, I thought we were just talking in 24 vitro so far? 25 A. Either way. Page 214	1 Q. Right. Understood. But it is still 2 measuring a single amino acid that seems to affect this 3 whole process of the transgene being expressed in these 4 tissue liver tissue or hepatocytes? 5 MS. MORRISON: Objection to the form. 6 A. That's what is measured. 7 Q. Understood. Thank you. Let's see, we just 8 talked about the in vitro. Let's talk about the in 9 vivo that you started in. 10 And so you injected the AAV1-E531K mutant 11 AAV, this time it's carrying a factor 9, carrying 12 the factor 9, H factor 9 transgene. You injected that 13 into the portal veins of mice, correct? 14 A. Correct. 15 Q. And that portal vein carries blood to the 16 liver, right? Yes? 17 A. Correct. 18 Q. It's a way of getting it to the liver, 19 right? 20 A. Right. 21 Q. And then you measure the amount of the 22 transgene expressed in both parenchymal cells and 23 non-parenchymal cells -- how do you pronounce it? 24 A. Parenchymal. 25 Q. Parenchymal. You measured it in both, Page 216
1 Q. But you're only looking at hepatocytes 2 anyway, right? 3 A. Right. So in order to get it to work in 4 hepatocytes, you had to have a liver promoter. And 5 that same cassette when used in animals, actually 6 restricted it that you can only measure it in liver and 7 not in other tissues. 8 So it may have conferred increase eye, 9 muscle, whatever, but we could not have scored it. 10 Does that make sense? 11 Q. Okay. Yes, it does. So you could have 12 gotten enhanced tropism in other tissues, but you just 13 wouldn't have known, right? 14 MS. MORRISON: Objection to the form. 15 A. We would not have been able to deduce that 16 with this cassette and the assays we were doing. 17 Q. Understood. Understood. Right. Because 18 even that way, if the AAV could have successfully 19 delivered its transgene to eye or a muscle or brain or 20 whatever, it wouldn't have been able to be expressed, 21 and you wouldn't have been able to see the marker 22 expression, even if the delivery had happened? 23 A. Correct. So when we loosely use the word 24 tropism in this setting, it's dictated by the promoter, 25 not by the capsid. Page 215	1 right? 2 A. Yeah. 3 Q. So you found from these experiments, and 4 the data are shown in the Figure 3, you looked at -- 5 you looked at a -- you actually looked at a luciferase 6 transgene in vitro, and then you looked at this H 7 factor IX level in vivo, right? 8 A. Correct. 9 Q. So 3A, Figure 3A shows the luciferase 10 transgene in vitro data, correct? 11 A. Correct. 12 Q. And 3B shows the serum h factor IX levels 13 in vivo? 14 A. Correct. 15 Q. And so what you showed was that the 16 AAV1-E531K mutants were substantially better at 17 transducing liver cells and expressing the transgene 18 than the wild-type AAV1 in this assay? 19 MS. MORRISON: Objection to the form. 20 A. Correct. 21 Q. And then you carry that thought bot(sic) 22 assays, and that's part C to determine the vector 23 genome levels in parenchymal and non-parenchymal cells 24 after the portal vein injection, correct? 25 A. Correct. Page 217

1 Q. And so the non-parenchymal cells are just 2 the control here, sort of? 3 A. Hard to argue one way or the other. I 4 don't know if I would call them controls. They could 5 be leaky expression. The AVITR adds promoter activity, 6 which is not restricted to cell type. So there could 7 be leakiness. 8 Q. Okay. But in any event, you found 9 substantially higher vector genome levels produced by 10 injection of AAV1-E531K than were produced by injection 11 of wild-type AAV1, correct? 12 A. That is correct. 13 Q. Okay. Now, you say on page 11396, the same 14 page we've been looking at, down at the bottom of the 15 right-hand column, you say: These data not only 16 speak -- do you see that? I'm like at the second 17 sentence up from the bottom of the right-hand column. 18 The sentence says these data. 19 A. Yes. 20 Q. Found it? 21 A. Yes. 22 Q. Okay. These data not only speak to the 23 importance of the K531 position, but also illustrate 24 the ability of a single amino acid change to confer a 25 serotype-specific trait in another distantly related Page 218	1 You also notice in that figure that the 2 AAV1 531K never reaches the AAV6 level. So there's 3 another amino acid that's also important. It was just 4 too complicated to put this into the story that we 5 needed two amino acids to get this. 6 So it was a, let's take a simple message 7 out and realize it was more complex than even we can 8 explain. 9 Q. Okay, fair enough. So that sentence about, 10 that what we just read: These data, you know, not only 11 speak to the importance of the K531 position, but also 12 illustrate the ability of a single amino acid change to 13 confer a serotype-specific trait in another 14 distantly-related serotype, was referring to the 15 heparin binding? 16 A. Heparin binding. 17 Q. Right. So that you could confer that on 18 AV8 -- sorry, sorry, let me just -- so that you could 19 confer that heparin-binding trait on AAV8 from the 20 single amino acid substitution? 21 A. Right. We could confer an in vitro measure 22 onto a capsid, but not an in vivo measure. 23 Q. Right. Okay. Now, you say also on the top 24 of the left-hand column of the next page, 11397, page 25 11397, you say that -- and I'm looking at the end with Page 220
1 serotype. Do you see that? 2 A. Yes. 3 Q. And that was based on your result between 4 AAV6 and AAV8, correct? 5 A. So that's the story that -- 6 Q. Go ahead. 7 A. -- we didn't publish, because we couldn't 8 see a difference between AAV8 improving its 9 transduction with this change. It was already superior 10 in liver, and we couldn't make it any better. 11 So you can see how we suspiciously said you 12 could put the change in 8 and get it to bind to 13 heparin, but we didn't put 8 in liver and show that we 14 got an increase. 15 And if you take that observation, you're 16 stuck with why did it work for 1 and what I call 1's 17 twin, but didn't work for 8, because 1 and 6 are 18 identical. 19 Q. I thought you were saying you couldn't 20 really measure for 8, because 8 was already very good? 21 A. Well, that's what I'm saying. It did 22 nothing to 8, and you don't know why. Was it because 8 23 was already Mother Nature's best choice where 6 and 1 24 were pretty poor and 6, Mother Nature's variant, got 25 you in the result that worked. Page 219	1 the first incomplete paragraph you say: The results 2 described herein allow, for the first time, a partial 3 mechanistic interpretation of the molecular 4 determinants of liver tropism in AAV6 sectors in 5 comparison with AAV1, right? 6 A. Where are you at? 7 Q. I'm sorry, I'm on the top left-hand 8 column -- no, of the last page, you know, where the 9 references start. Left-hand column page -- 10 A. Nevertheless? 11 Q. Yeah. So I'll go back. 12 Nevertheless, the results described herein 13 allow, for the first time, a partial mechanistic 14 interpretation of the molecular determinants of liver 15 tropism in AAV6 vectors in comparison with AAV1. 16 Do you see that? 17 A. I see that. 18 Q. I'm going to ask my question. 19 A. Ask your question. 20 Q. I will ask my question first. 21 It does say here that the results in this 22 paper involving these single amino acid changes start 23 to help understand the molecular determinants of liver 24 tropism in AAV6 sectors in comparison to AAV1, correct? 25 MS. MORRISON: Objection to the form. Page 221

1 Go ahead. 2 A. That's what it says, but it relates its 3 bias with the liver-specific promoter. 4 So a good reviewer would have said you 5 can't make that statement, because you didn't have a 6 constituted promotor determine if it had anything to do 7 with liver tropism. You already skewed the answer to 8 only be liver, and so you were measuring an increase in 9 infectivity and not an increase in tropism. 10 Does that make sense? 11 Q. You disagree with everybody about the 12 meaning of tropism, Dr. Samulski, including your own 13 coauthors, your students, your lawyers, your patent 14 lawyers, your coinventors from Florida. 15 A. You have that luxury when you've been in it 16 this long to be able to say no guys, you got that one 17 wrong. 18 But in all fairness, when we looked at it 19 in the liver, we could not see it anywhere else. 20 So to attach that to tropism meant we were 21 really speaking to the increase in infectivity. It 22 could be the best muscle vector in the world but we 23 didn't have a muscle promotor. So we couldn't have 24 determined what that outcome was. 25 Q. But isn't -- isn't tropism, even in your Page 222	1 causes damage, and that's where people assign tropism 2 based on the clinical outcome. 3 When we go to vectors, and I'm only going 4 to make you frustrated now, if you think about it. We 5 are not describing AAV tropism. AAV tropism is 6 dependent on the helper virus. That means wherever 7 adenovirus goes, that's where AAV wild-type AAV will 8 replicate. That determines the tropism. 9 Vectors are a prostitute of AAV's tropism, 10 because we determine tropism of where we see a gene 11 being expressed. And that technically is unrelated to 12 the virus, who is restricted by the helper virus where 13 it can grow and where it can't grow. 14 So in these studies, of which I am an 15 abuser of these nomenclature, we say tropism based on 16 expression. We saw gene expression. We didn't measure 17 how many other cells had genes in it, and we could not 18 have measured expression, because they didn't have a 19 promoter that would work in other cells. So we 20 restricted the outcome to be only what we observed by 21 these measurements. 22 Q. I'm going to regret this but I'm going to 23 ask you another question about it. 24 But isn't it still possible, even by your 25 definition here, that you affected tropism, let's say Page 224
1 sort of world view, a preferential -- a preferential 2 ability to transduce tissues? 3 A. No. This is what I have been trying to get 4 across. 5 Q. Okay. 6 A. A virus can bind to a receptor and it can 7 start the process of entry. That's tropism. Anything 8 after that are a series of steps that determine 9 infectivity. 10 So, for example, when you do get the Polio 11 virus vaccine, and it replicates in your gut, it is 12 restricted from replicating in the neurons, not because 13 the virus can't bind to the neurons, it has had a 14 mutation at 543 that won't let it get translated. It's 15 a plus-strand RNA. And the fact that it can't get 16 translated and make copies, it can't replicate and kill 17 your motor neurons and paralyze you. But the virus 18 that's made will go to those cells and bind and do the 19 entry step. It's the neurons that can't take the 20 genome after that and have it continue its life cycle. 21 So tropism, when we say tropism, we say oh, 22 Polio virus is tropic for neuroma cells. It binds to 23 epithelial cells in the gut and replicates that allows 24 it to get into Peyer's patches that allows it to get to 25 neuronal cells. It's the neuronal cells is where it Page 223	1 what you did -- I mean, you said yourself the promoter 2 was the same, right, in both constructs, the AAV1 and 3 the AAV6, right? 4 A. It's the same promoter. 5 Q. So something then had to be improved before 6 you got to gene expression in this whole pathway in 7 liver cells? 8 A. Now I'm going to drive you really crazy. 9 Q. Okay. 10 A. Okay. Because there is ample data, in 11 fact, we're plagued with data that AAV will get in the 12 cell but it won't express. It's sitting there waiting 13 for something to happen. And there's now data seven 14 years later in a monkey we can find the AAV virus in 15 the eye, but it's never turned on -- it's never uncoded 16 and turned on its genome. These motif may simply be 17 allowing it to do a last step in transduction; entry 18 step, an un-coding step, whatever. 19 The most recent observation that we've 20 uncovered, which is going to frustrate everybody, is we 21 found out that the capsid can decide after it's in the 22 nucleus whether to let the genome express or not 23 express. 24 And that paper you brought up by Rebecca 25 Haberman was the first example, which we were not able Page 225

1 to explain how AAV can go into neurons, and it was on 2 with one promoter, but not with the other, and then 3 when you switched promoters, it was on and not on. 4 So it was neurons -- excitatory neurons and 5 inhibitory neurons. We were stopping excitatory 6 neurons from making seizures, and we were successful. 7 So we put a regulator, promotor in that 8 virus, in that cassette, and put it back in the same 9 place, and all of a sudden inhibitory neurons were 10 being turned off. We were like, wait a minute? How 11 the hell did that happen? The virus was in both cells, 12 but it was controlling which one was on and which one 13 was off. 14 Any and all of these modifications could be 15 determining tropism at steps that we have yet to 16 unravel. 17 So when I struggle, and I apologize, 18 because you're asking very different type of 19 questions -- isn't it possible, could someone at this 20 skill do this or that -- I'm basically saying no, no, 21 no, no, because we don't know how it works. We never 22 understood and we still don't know how it works, and 23 each time we do this, we peel back another layer of the 24 onion. 25 So I'm hesitant to go on record saying Page 226	1 nearly at the end of this paragraph, so don't get your 2 hopes up. We anticipate that further studies mapping 3 the potential role of single amino acid changes in 4 closely-related AAV serotypes and variants, as 5 described above for AAV1 and AAV6, are likely to shed 6 light on molecular determinants of capsid receptor 7 interactions, tissue tropism, immune profile, and other 8 aspects of the AAV life cycle in general, right? 9 A. The entire kitchen sink was thrown in 10 there. 11 Q. Well, and one element in the kitchen sink 12 is tropism, and you're saying here that these kind of 13 studies of single amino acid changes can shed light on 14 molecular determinants of tropism, right? 15 A. That's what we said. 16 Q. That is what you said. Okay. 17 Now, I would like to look at the crystal 18 structure data in this paper, which is on page 11395. 19 Now, you used -- okay. So let's just talk 20 about this figure for a second so that we're all on the 21 same page. 22 So the left -- so there's three boxes on 23 the top and three boxes on the bottom, correct? 24 A. Yes. 25 Q. All right. And each column of two boxes Page 228
1 things that I know are(sic) not been proven yet. I 2 hope that apology of why the field is so -- still stuck 3 in the mud. 4 Q. Appreciate it. But you got to yell at your 5 coauthors, because in the very next paragraph here it 6 says: It should be noted that similar studies with 7 autonomous parvoviruses suggest that the molecular 8 determinants controlling post range and tropism in 9 autonomous parvoviruses can often be narrowed down to a 10 few critical amino acid residues in the capsid, right? 11 A. Correct. 12 Q. So you agree at least -- 13 A. Correct. 14 Q. -- that tropism can be determined by a few 15 critical amino acid residues from the capsid? 16 A. Okay. So with autonomous parvoviruses, 17 you're measuring disease now. 18 Q. Okay. 19 A. Not expression. A disease. So if you 20 change an amino acid, and you don't get a disease, you 21 can safely say we affected the tropism. 22 Q. Well, keep yelling at your coauthors, 23 because further down on the page it says: We 24 anticipate -- and I'm nearly at the end of this 25 paragraph. I'm not done with this reference, but I'm Page 227	1 relates to a different AAV variant, correct? 2 A. Correct. 3 Q. So the first two are AAV2, right? 4 A. Correct. 5 Q. And those pictures are based on the crystal 6 structure of Wu, et al.? 7 A. Correct. 8 Q. And the other two column, there's a middle 9 one that's AAV6 and a right-hand one that's AAV1, 10 correct? 11 A. Correct. 12 Q. Those are based on modeling that your group 13 did using the coordinates of the Wu published crystal 14 258 structure for AAV2, correct? 15 A. Yeah, that was done Mavis's student. 16 Q. Okay. All right. And Mavis was in Florida 17 at this time? 18 A. That's correct. 19 Q. And she's a -- she was faculty? 20 A. She was the faculty. 21 Q. I see. All right. And so from the 22 coordinates of the AAV2 actual structure, you, meaning, 23 everybody collectively, not -- I understand she did it, 24 but, you know, this paper modeled the capsid surface of 25 AAV6 and AAV1, correct? All right. Yes? Page 229

1 A. Correct. 2 Q. All right. Now, you looked -- in 3 particular, you were interested to see where this 4 important 531 residue was in AAV6. It's a license. So 5 you were looking to see in the AAV6 model where does 6 K531 fall on the capsid surface, correct? 7 A. Correct. 8 Q. And now you say, if you look at the 9 right-hand column of 11395, so underneath the modeling 10 pictures you say -- make sure I find the exact right 11 thing. You say: When the capsid is viewed 12 perpendicular -- I'm reading like almost at the top of 13 the right-hand column, but not quite. Do you see the 14 sentence beginning when the capsid is viewed on the 15 right-hand column? 16 A. When the capsid is viewed perpendicular to 17 the threefold axis? 18 Q. Yeah, so when the capsid is viewed 19 perpendicular to the threefold axis, the site chain for 20 K531 is surface exposed and forms a continuation for 21 the basic patch between K533 and R488 as well as K528 22 and R567 at the base of each threefold spike of the 23 AAV6 capsid surface model, right? 24 A. Correct. 25 Q. So here you're talking about a basic patch Page 230	1 MS. MORRISON: Objection to the form 2 of the question. 3 BY MS. TOKER: 4 Q. I'm going to say it. 5 A. You're welcome to say it. 6 So let me help you a little bit here. 7 Q. Yep. 8 A. When a -- surface receptors on the outside 9 of the virus and it binds, it goes through a 10 confirmation of change, and I'll use Polio virus for 11 example. When it binds to its receptor, it actually 12 opens up allowing other surface molecules to interact 13 with one another. 14 So having the basic patch at the bottom of 15 the receptor couldn't do nothing more than after it 16 binds to the receptor, it gives it more affinity to 17 have a stronger interaction and hold onto that, and 18 thereby remember step one is binding, step two is 19 entry. 20 And so we would view that, based on Mavis', 21 later Crystal's, where she actually show the receptor 22 interacting with those spikes, that it opens up like a 23 flower and exposes things that aren't on the surface to 24 be more surface exposed. 25 Q. So just so I understand what you just said, Page 232
1 that obviously involved a bunch of basic residues, 2 which are lysine and arginine, correct? 3 A. Correct. 4 Q. And they listed them all here, all the 5 amino acids that make up this basic patch, correct? 6 A. Correct. 7 Q. And you've located K531 in this basic 8 patch? 9 A. Correct. 10 Q. All right. And this basic patch is at the 11 base of each three-fold spike, right? 12 A. Correct. 13 Q. And a spike is a peak, fair? 14 A. Correct. 15 Q. All right. So this basic patch, where this 16 important, or this paper residue 531 is located, is at 17 the base of a peak, not on the peak or at the top of 18 the peak, correct? 19 MS. MORRISON: Objection to the form. 20 A. Correct. 21 Q. And, you know, despite the fact that it's 22 at the base of the peak, as opposed to on the peak, or 23 at the top of the peak, you found in your experiment 24 that it could be a determinant of, I'm going to say 25 tropism. Page 231	1 things that are not on the peak or, you know, at the 2 top of the peak, can still -- can influence the binding 3 to the receptor based on conformational changes that 4 occur upon binding? 5 A. Yep, correct. 6 Q. So that you can't, again, necessarily look 7 at the surface and say well, you know, K531 is at the 8 base of the peak so it won't affect binding? 9 A. Correct. 10 Q. Okay. All right. 11 MS. TOKER: Can we go off the record 12 for a moment? 13 THE VIDEOGRAPHER: The time is 4:13. 14 We're off the record. 15 (Recess.) 16 THE VIDEOGRAPHER: The time is 4:13. 17 We're on the record. 18 BY MS. TOKER: 19 Q. All right. Dr. Samulski. I would like to 20 look at a different exhibit. I will ask the court 21 reporter to mark this as 1061. 22 (The document referred to was marked 23 Deposition Exhibit Number 1061 for 24 identification.) 25 Q. All right, Dr. Samulski. The court Page 233

1 reporter has just handed you what has been marked as 2 Exhibit 1061. Do you recognize this document? 3 A. Is this a different one? Dawn Bowles, it's 4 like the same group. I guess it -- 5 Q. My first question is does this look 6 familiar? 7 A. Yeah, I recognize it a little bit. 8 Q. Okay. So just for the record, this is US 9 Patent 7,892,809, and Dr. Samulski, you're a named 10 inventor on this patent, correct? 11 A. Yes. 12 Q. This patent issued February 22 of 2011, 13 right? 14 A. Correct. 15 Q. I don't know if you know, but do you happen 16 to know that this patent does not expire until 2026? 17 A. Is that true? I didn't know that. 18 Q. I'm just asking if you happen to know that? 19 A. I don't know that. 20 Q. All right. That's fair. 21 All right. This patent claims priority to 22 the '126 provisional that we spent a lot of quality 23 time on today, and I'll point you to where that is on 24 the patent you're holding. 25 So if you look at the '809 patent, the Page 234	1 A. Okay. 2 Q. The provisional was filed on December 15 of 3 2004, and this patent says that the provisional is 4 related to it. 5 A. Okay. So they are one in the same, I 6 guess, is what you're saying? 7 Q. Well, they are not exactly the same, and 8 we're going to look at one of the differences between 9 them. But they are related, and we can look at some of 10 that. 11 You will see, if you want to look a little 12 bit, there are a lot of the disclosures that we talked 13 about in the '126 are part of the '809, but there are 14 additional. 15 A. Oh, in addition to the other stuff, okay. 16 Q. Yes. And if you want to just take a moment 17 and just kind of look at it, that's fine, and we're 18 going to walk through some. 19 A. The same set of inventors, right? 20 Q. You tell me, but it looks like same 21 inventors, correct? 22 A. Yeah, Josh, Chengwen, Dawn, yeah, okay. 23 Q. Okay. And this -- I'll just say one more 24 thing about the cover before you open it. This was 25 originally filed on December 15 of 2005. If you look Page 236
1 Exhibit 1061, and if you look down on the left column, 2 it says related US application data. Do you see that? 3 Just keep going down the left-hand column. And there's 4 a little bold headline that says related -- 5 A. Yes, I see that. 6 Q. All right. And you can check the number, 7 it says provisional application number 60/636126 filed 8 on December 15 of 2004. 9 And Dr. Samulski, feel free to check that 10 number against the '126 provisional that we were 11 looking at before. 12 A. This is the provisional? 13 Q. Yes. 14 A. Okay. What does that mean? So is this the 15 real patent and this is not the patent? 16 Q. Okay. So, I mean, if you don't know, based 17 on your experience, I can see if I can refresh your 18 recollection that you file a provisional first and it 19 may ultimately -- 20 A. Issue. 21 Q. -- turn into, right, an issued patent. 22 A. Okay. So this is the published provisional 23 and this is the patent? 24 Q. It's not actually published, but it was 25 filed. Page 235	1 down, it says PCT filed December 15 of 2005. 2 Do you see that? 3 A. 2005? 4 Q. Yeah. So your provisional was filed on 5 December 15, 2004. This was filed a year later on 6 December 15 of 2005. 7 A. Okay. And it was issued in 2011? 8 Q. Correct. 9 A. And it's almost expired because of the 10 20-year thing? 11 Q. I'm not going to represent stuff to you. 12 You're the witness. But if you didn't have to know it, 13 that was fine. I was just asking if you happened to 14 know about that. But that's okay. Sorry. 15 A. This takes forever. 16 Q. That's fair. I'm ready to go. I just want 17 to make sure you weren't -- 18 A. Yeah, I'm looking at just trying to 19 familiarize myself. 20 Q. Take a moment. That's fine. 21 (Pause.) 22 A. 263, 265, no, it doesn't have the 716 and 23 the 450 we were talking about. There's animal data. 24 Interesting. 25 Okay. I think we can jump in the deep end. Page 237

1 Q. I do want to take you up on what you just 2 said. It does have the 716 and the 450. 3 A. No, I meant no more data to validate those. 4 Q. Oh, sorry, but it does discuss -- 5 A. It's the same discussion, yeah. 6 Q. Okay. So I just wanted to look at a couple 7 of additional things here. I wanted to look at the 8 summary of the invention under column one, which is 9 just at the beginning after all the figures. 10 So in this one, the figures are at the 11 beginning instead of at the end, and then the patent 12 begins, yeah, exactly. 13 So if you could look at the summary of the 14 invention? 15 A. Yes. Where it says the present invention 16 provides? 17 Q. Exactly. The present invention provides 18 chimeric virus vectors that have been designed to 19 exhibit one or more properties of interest, e.g. 20 enhanced tissue tropism, right? 21 A. Correct. 22 Q. All right. So here again we're looking at 23 AAV mutants that enhance tissue tropics, right? 24 MS. MORRISON: Objection to the form 25 of the question. Page 238	1 Q. So they're talking about a single amino 2 acid from AAV1 that they put into AAV2, right? 3 A. Correct. 4 Q. Now, I'd like to take a look in this patent 5 at claim 22, which is in column 75, so towards the end 6 column 75. It's near at the end. 7 A. The column? 8 Q. Sorry, column. Now we have the issue 9 patents, so we're going to be looking at the numbers at 10 the top. 11 A. 75, yeah. 12 Q. So I was looking at column 75 and claim 22, 13 which is around line 13. 14 A. Yeah. 15 Q. Let me know when you have got that. 16 A. Okay. 17 Q. Okay. So then claim 22 describes a 18 chimeric adeno-associated virus AAV capsid comprising a 19 selective amino acid substitution at amino acid 20 position 450 in an AAV2 capsid subunit having the amino 21 acid sequence, with sequence ID number 3, or 22 corresponding change in another, and there's a typo, it 23 says AVV, but it should say AAV, I think, AAV capsid 24 subunit? 25 A. Correct. Page 240
1 A. Related to the assay that you were scoring 2 the promoters or tissue -- 3 MS. TOKER: Sorry, we have to go off 4 the record. 5 THE VIDEOGRAPHER: The time is 4:23. 6 We're off the record. 7 (Recess.) 8 THE VIDEOGRAPHER: The time is 4:23. 9 We're on the record. 10 BY MS. TOKER: 11 Q. All right. We were just looking, right 12 before the interruption, under the summary of the 13 invention, where it says that: This patent discloses 14 mutants, mutant AAV vectors that have been designed to 15 exhibit one or more properties of interest, such as 16 enhanced tissue tropism, right? 17 A. Correct. 18 Q. All right. And -- okay. 19 Now, and, again, we're talking about as we 20 were before with the '126 provisional -- well, I'll 21 just read it. It says: The inventors have identified 22 the key amino acid of AAV1 responsible for enhanced in 23 vivo transduction and selectively engineered this amino 24 acid into the backbone of AAV2 and AAV3B, right? 25 A. Correct. Page 239	1 Q. And that's part A. 2 And then part B says: A nucleic acid 3 comprising an AAV terminal repeat sequence and a 4 heterologous nucleic acid sequence, wherein, the 5 nucleic is packaged within the chimeric AAV capsid. 6 So that's the, what we were looking at 7 before, that's an independent claim, correct? 8 A. Correct. 9 Q. And then claim 23 is a dependent claim, 10 right? Because it says: The chimeric virus vector of 11 claim 22. Does that make sense to you, Dr. Samulski? 12 A. Yes. 13 Q. So claim 23 depends from claim 22, correct? 14 A. Correct. 15 Q. All right. And it now adds on a limitation 16 to what we just read from claim 22, and it says: The 17 chimeric virus vector of claim 22, wherein the 18 substitution, is a substitution of asparagine for 19 threonine, correct? 20 A. Correct. 21 Q. So in your issue patent, your '809 patent, 22 you also claimed a single amino acid substitution at 23 position 450 of AAV2, where the threonine that's 24 normally there was replaced by asparagine, correct? 25 MS. MORRISON: Objection to the form Page 241

1 of the question. 2 A. Correct. 3 Q. I would like to look at in this same 4 document, the '809 patent, speaking of frustrating, I'm 5 going to have us look at the definition of tropism 6 again, because no document would be complete without a 7 discussion of tropism. So that's column 8, around line 8 60. 9 A. Yeah. 10 Q. It's at the bottom of this table one. 11 There's a paragraph of text on column 8. 12 Do you see that? 13 A. Yes. 14 Q. It says: The term tropism, as used herein, 15 refers to preferential entry virus into certain cell or 16 tissue types or preferential interaction of the cell 17 surface that facilitates entry into certain cell and 18 tissue types optionally and preferably followed by 19 expression, e.g. transcription and optional translation 20 of sequences carried by the viral genome in the cell, 21 e.g. for a recombinant virus expression of the 22 heterologous nucleotide. You see that, right? 23 A. Yep. 24 Q. So once again it defines the tropism as the 25 way you defined it originally, but with this optional Page 242	1 tropism, Dr. Samulski. So I'm going to say tropism. 2 This AAV2.5 has different tropism from both 3 parent vectors, from -- it was different from both AAV1 4 and AAV2, correct? 5 MS. MORRISON: Objection to the form 6 of the question. 7 A. It was different from both of the parents. 8 Q. Different in terms of tropism, is my 9 question, correct? 10 A. Expression profile. 11 Q. You call it tropism here? 12 A. Yeah, but we're back to expressing vector 13 9. You see what I'm saying? 14 Q. Okay. And so that's -- I understand you 15 don't agree with it, but it's being referred to here as 16 tropism, correct? 17 A. Okay. It may be referred there as tropism. 18 Q. Okay. And so what you found was that 19 the -- I mean, it's called tropism. So you say the 20 experiment demonstrated that the AAV2.5 it gained 21 muscle tropism, it lost liver tropism, and it gained a 22 new kind of tropism for a totally different kind of 23 cell astrocytes, correct? 24 A. Correct. 25 Q. All right. And so despite the fact that Page 244
1 addition that it can include expression even of a 2 heterologous nucleotide sequence from a recombinant 3 virus, correct? 4 A. Correct. 5 Q. Now, I would like to look at column 38, 6 please, which is right before the sequence listed, 7 there's a column 38 that says example 4. 8 A. 38. 9 Q. And do you see a heading that says example 10 4? 11 A. Yes. 12 Q. Okay. And so this example 4 describes some 13 experiments having to do with transduction of different 14 tissues by the 2.5 variance that we talked about 15 before, correct? 16 A. Correct. 17 Q. And that was the one that you or that your 18 company, AskBio, took to clinical trials in children 19 with DME, correct? 20 A. Correct. 21 Q. Now, this particular -- is it okay if I 22 call it AAV2.5? 23 A. Yeah. 24 Q. So AAV2.5 had different tropism or, yeah, 25 well, I'm going to call it tropism, because you call it Page 243	1 this vector gained a non-muscle tropism, astrocytes are 2 not muscle, correct? 3 A. Correct. 4 Q. All right. You still believe that it could 5 be a useful therapy to use for -- to shed muscular 6 dystrophy, which you had originally been creating this 7 vector to enhance -- let me start that over, okay. 8 That was a mess. I'm going to try again. 9 You were originally looking to enhance -- 10 you need a drink? 11 A. I'm okay. 12 Q. You were originally in the '809 patent and 13 in the '126 provisional that we were looking at, 14 looking to enhance the muscle tropism of AA2, correct? 15 A. Correct. 16 Q. With the idea of using it for, you know, 17 potentially for a disease as a gene therapy, such as 18 Duchenne muscular dystrophy, correct? 19 A. Correct. 20 Q. Here you found that this AAV2.5 gained a 21 tropism in a different kind of cell astrocytes, right? 22 A. Correct. 23 Q. But you still used it for its improved 24 muscle tropism in DMD patients, correct? 25 A. So I'm trying to -- I want to make sure Page 245

1 that was the experiment in astrocyte cells or in 2 animals that showed up in astrocytes? 3 Q. Okay. So -- 4 A. It was -- 5 Q. So let's be clear on that. I'm sorry, I 6 did skip over the experimental details. But it says, 7 right, that it was injected into mouse, right? 8 A. Yeah. 9 Q. Okay. So these are in animals, correct? 10 A. Correct, okay. 11 Q. So this was in vivo, agreed? 12 A. But this is a different gene. It's GFP 13 where the original was vector 9. 14 Q. Yeah, this example we didn't see that in 15 the '126, that's new for the '809, and you're welcome 16 to compare, but that's here. 17 So yes, this is a different set from 18 experiments that we had looked at in the '126. 19 So I guess sort of the question to start 20 off is you agree that this was looked at in vivo in the 21 animals, correct? 22 A. Correct. 23 Q. Okay. And you found that there was a 24 gained -- a new tropism for astrocytes? 25 A. Correct. Page 246	1 didn't inject it intravenously anyway. You were using 2 it intramuscularly. 3 MS. MORRISON: Objection to the form 4 of the question. 5 A. For those studies, it was requested by the 6 FDA data be done IM, intramuscularly. 7 Q. Okay. 8 A. Which reduced even more likelihood that it 9 would ever get access to the brain. 10 Q. Right. So you can have a vector that can 11 have multiple different cellular tropism or tissue -- 12 let me rephrase. 13 You can have a vector that can have 14 multiple different tissue tropisms, but the way you use 15 it, can still -- it can still be useful to target one 16 of the multiple tropisms of the vector. 17 MS. MORRISON: Objection to the form 18 of the question. 19 A. So I think what you're proposing is if you 20 had multiple tropisms attached to a capsid, the root of 21 administration may restrict access to those tropisms. 22 I would put forth that if it's intravenous delivery, it 23 has access to essentially everything. If it's 24 intramuscular, it's restricted. If it's intravitreal, 25 it's restricted, intracranial, so forth and so on. Page 248
1 Q. All right. And that new tropism for 2 astrocytes did not stop you from using AAV2.5 to target 3 muscle? 4 MS. MORRISON: Objection to the form 5 of the question. 6 Q. In Duchenne muscular dystrophy? 7 A. Yeah, I think one thing you might want to 8 take into account is the AAV2 won't cross the blood 9 brain barrier. So the only way to determine that it 10 went into astrocytes was to physically put it in the 11 brain, that means injecting into the brain. 12 And so using it for muscular dystrophy 13 where you add it systemically would never put a risk of 14 going to not astrocytes. 15 So that was basically a physical blocker to 16 a putative tropism. So there was probably no risk 17 involved. 18 Q. Right. But even in your 2.5 clinical 19 trial, you didn't inject it intravenously. You 20 injected it -- 21 A. Yeah. I'm sorry. 22 Q. You injected it intramuscularly, correct? 23 A. That's correct. 24 Q. So in that sense also, the fact that the 25 vector had an additional tropism didn't stop you. You Page 247	1 So that the virus is not restricted. 2 You're not using the virus based on its promiscuity. 3 You're restricting it based on the root of 4 administration. 5 Q. Right. So you can have a promiscuous 6 vector, but based on the root of administration, you 7 can target specific tissues within the promiscuous 8 tropism of the vector? 9 A. Correct. And IV would probably not be a 10 root of administration that you can control that 11 promiscuity. 12 Q. Okay, right. But -- fair enough. Okay. 13 Do you know whether you ever licensed this 14 patent, the '809? 15 A. I have no idea. 16 Q. Okay. I'm going to mark another exhibit. 17 A. While you're drawing attention, can I also 18 draw attention to the fact that the claim set that you 19 were describing, the claims for 3B required two changes 20 instead of one? 21 Q. Let's stick to my questions for now, and 22 I'm about to grab the next exhibit, so just give me one 23 second. 24 Let's go ahead and mark this as 25 Exhibit 1062. Page 249

1 Dr. Samulski, the court reporter has just 2 handed you what has been marked Exhibit 1062. 3 (The document referred to was marked 4 Deposition Exhibit Number 1062 for 5 identification.) 6 Q. Have you seen this before? 7 A. Yes, I'm the senior author. 8 Q. You're the senior author. 9 This is a paper by Dawn Bowles and a number 10 of authors, including yourself, correct? 11 A. Correct. 12 Q. And this was, I think it says it was 13 published online in 2011. That's under the sort of the 14 abstract right before the introduction. 15 A. Yes. Correct. 16 Q. Who is Jade Samulski? 17 A. My nephew. 18 Q. Oh, and this has some of the people that we 19 looked at as inventors, you know, the named inventors 20 of the '126 provisional and the '809 patent, correct? 21 A. Correct. 22 Q. Including, for example, Joshua Grieger and 23 Mavis McKenna and others, correct? 24 A. And Dawn Bowles, correct. 25 Q. And Dawn Bowles, correct. Page 250	1 So this is basically the science behind the 2 vector, and he was trying to establish a clinical 3 outcome that we thought was not justified by the 4 science. 5 Q. Just -- I'm curious, what was the clinical 6 outcome that he was trying to sell? 7 A. There was no way you could provide benefit 8 to a child with DMD if you did an IM injection. So we 9 were opposed to the clinical trial from the beginning. 10 And he went ahead and did it and published it, and then 11 we came back speaking to the science of it. 12 Q. Of how you had developed the AAV2.5 vector? 13 A. Yeah, and this is more basic science than 14 this. But it was a difference in opinions of do you 15 use a kid to find out an experiment; and we were no. 16 That's why I said 2.5 was the wrong virus, we were 17 quite aware of that, but he took it and went and did 18 something we didn't support. 19 Q. Got it. But this, nonetheless, does 20 describe, in your view, accurately the science of how 21 you developed, for example, the AAV2.5 vector, is that 22 fair? 23 A. Correct, that's fair. 24 Q. So let's see, you discuss here, you say in 25 the abstract that you -- this is the first clinical Page 252
1 Now, a couple of the authors here are 2 listed as having their affiliation as Asklepios 3 BioPharmaceutical, Inc., which is AskBio, correct? 4 A. Correct. 5 Q. And those authors are Scott McPhee and Jade 6 Samulski, correct? 7 A. Correct. 8 Q. And what position was Scott McPhee at 9 AskBio? 10 A. Technically, I don't remember. But there 11 was only two members of the company; Jade and Scott, so 12 I think they shared whatever roles. 13 Q. Okay. And so this paper describes the 14 clinical trial using AAV2.5, correct? 15 A. It's one of two papers. The first paper 16 was done by Jerry Mendell, published in the New England 17 Journal of Medicine. Jerry Mendell and it was 18 published in the New England Journal of Medicine, and 19 this paper Jerry and others refused to put their names 20 on it. 21 Q. Why did they refuse to put their names on 22 it? 23 A. So we had a difference of opinion on what 24 he was trying to do clinically and what we thought we 25 were doing scientifically. Page 251	1 trial using AAV2.5 derived from a rational design 2 strategy, correct? 3 A. Correct. 4 Q. And we talked about that. That's the 5 strategy that we discussed, right, in the '126 6 provisional? 7 A. Correct. 8 Q. You say under the introduction -- you say 9 in the introduction, there's a section labeled 10 introduction at the bottom of the left-hand column that 11 talks in general about DMD as a disease, right? And 12 then it says at the bottom: DMD is X-linked and is 13 genetically inherited, caused by mutations in 14 dystrophin, a large 427-kilodalton cytoskeletal protein 15 that is normally expressed. And then you listed some 16 different tissues; skeletal and cardiac muscle as well 17 as smooth muscle, brain and retina in various isoforms, 18 correct? 19 A. Correct. 20 Q. And so isoforms are different forms of this 21 very large dystrophin protein, correct? 22 A. Correct. 23 Q. And each isoform has different forms of 24 this very large dystrophin protein, is that fair? 25 A. Fair. Page 253

1 Q. And depending on the isoform, depending on 2 which parts of the dystrophin protein the isoform has, 3 it will be expressed potentially in a different place 4 in the body? 5 MS. MORRISON: Objection to the form 6 of the question. 7 A. That's my assumption. 8 Q. Okay. And so right here you're saying that 9 it's expressed in a variety of tissues both, what, 10 muscle, brain and retina, correct? 11 A. Correct. 12 Q. All right. 13 A. There's a reference in the article that 14 describes that. Just to be clear, we didn't document 15 that. We're just referencing the literature. 16 Q. That it was known at this time that there 17 were studies that showed that isoforms of dystrophin 18 were spread throughout this variety of tissues, 19 correct? 20 A. Correct. 21 Q. Now, you say on page -- the next page, page 22 444, so that's the second page of Exhibit 1062, and 23 you're talking in -- on this page about -- again, the 24 strategy that you used to come up with AAV2.5, correct? 25 A. Correct. Page 254	1 the AAV2 capsid, which offers the benefits of a 2 well-defined safety profile, coupled with purification 3 ease. Do you see that? 4 A. Yes. 5 Q. And that's pretty much what we looked at in 6 the '126 provisional is this strategy, correct? 7 MS. MORRISON: Objection to the form 8 of the question. 9 A. That's similar to what we were just looking 10 at, correct. 11 Q. In the '126? 12 A. In the '126. 13 Q. Right. All right. So just turning to the 14 next page, where you say on the left-hand column -- I'm 15 looking at the first full paragraph. You say: The 16 engineering and testing of this chimeric capsid 17 meaning -- 18 MS. MORRISON: I'm sorry, counsel, 19 where are you? 20 MS. TOKER: I'm sorry, left-hand 21 column of page 445, first full paragraph 22 that begins the novel AAV2.5. 23 Q. Do you have that? 24 A. Yes. 25 Q. I'm just looking at the end. You say: The Page 256
1 Q. And you say at the bottom of the left-hand 2 column: It is clear that no single natural AAV 3 serotype will be useful for every clinical application. 4 Do you see that? 5 A. Yes. 6 Q. You go on to say that essentially, and we 7 will see what it says exactly. But you basically say 8 we now -- we are able to, and we now understand that we 9 should design our AAV vectors based on our needs, fair? 10 MS. MORRISON: Objection to the form 11 of the question. 12 A. Yes, I think that's what it's implying. 13 Q. And it says just a little bit further down 14 on the right-hand column, you say: Instead of 15 attempting to fit a known AAV, I'm sorry, I think -- 16 tell me when you're -- 17 A. I'm good. 18 Q. You're good okay, sorry. 19 So I'm on the right-hand column of page 20 444, and it says: Instead of attempting to fit a known 21 AAV serotype to a disease process or coevolve multiple 22 traces of a single capsid, we chose to use a rational 23 approach to identify capsid regions on alternative AAV 24 serotypes responsible for enhanced skeletal muscle 25 transduction and to combine these modifications into Page 255	1 engineering and testing of this chimeric capsid in 2 clinical setting is providing a paradigm, whereas the 3 investigator is no longer obligated to natural viral 4 isolates for gene delivery and can therefore address 5 additional clinical concerns, e.g. capsid immune 6 response, that lie outside of primary objective of 7 measuring therapeutic transgene expression. 8 Do you see that? 9 A. Correct. 10 Q. All right. So you're, again, talking about 11 rational design engineering of AAV to serve particular 12 purposes? 13 A. Yeah, I think I was emphasizing more that 14 because of the way this trial was done, we were trying 15 to extract safety data related to empty capsids in one 16 arm and vector with transgene in the other arm so that 17 we are no longer obligated to using the vectors or the 18 natural viral isolates, but you might be able to 19 discern novel safety data with these chimeric capsids, 20 which you couldn't do before. 21 Q. Okay. I mean, you also address safety in 22 the '126 provisional, where you said you were 23 interested in rational design that would preserve some 24 safety aspects of AAV2 and also enhance transduction, 25 right? Page 257

1 MS. TOKER: What were you going to 2 say? 3 MS. MORRISON: I was going to object, 4 but I didn't want to interrupt. 5 MS. TOKER: I'm done with the 6 question. 7 MS. MORRISON: Objection to the form 8 of the question. 9 A. So when we say safety, I think I'm going 10 back to historical perspective. AAV2 was the only one 11 in humans. It was the only one that the FDA had to 12 rely on. So there was what we called a safety profile 13 attached to that. Nobody died. And nobody -- so that 14 was like if you're going to choose something to go in, 15 try to choose 2. And if your 2 won't do what you want 16 it to do, then you're stuck trying to use rational 17 design to make 2 potentially successful. 18 After that, there was an onslaught of 19 forget rational design, just take AAV1, that was 20 Glybera the first drug approved, SMA, which was AAV9, 21 which was another. So people stopped worrying about 22 safety and just starting use you different serotypes. 23 But we were hamstrung by what the FDA would 24 say they would let go into a human. And I think in 25 order to be fair, the amount of resources it took to do Page 258	1 A. If I'm not mistaken, Laura Coruzzi. 2 Q. Okay. What were you asked to do, you know, 3 when you were contacted about this? 4 A. Provide some expert opinions about AAV 5 capsids. 6 Q. Anything in addition -- anything more 7 specific providing opinions? 8 A. No. Mostly navigating the minefield of AAV 9 capsids and tropisms and so forth and so on. 10 Q. Have you ever consulted for REGENXBIO 11 before? 12 A. No. 13 Q. Have you ever had any dealings of any kind 14 with REGENXBIO? 15 A. Yes, many different interactions. The CSO, 16 Olivier Danos, and I were both affiliated with the 17 first wave of gene therapy companies. It was called 18 Somatics. he was from Richard Mulligan's group, and I 19 was a consultant at that point in time. And over the 20 years we've interacted scientifically. 21 Then I think he went to France, and he was 22 in charge of the AFM, the French version of the DMD, 23 MDA, and we were collaborators, scientific 24 collaborators. And then I think he went to Viagen, and 25 he was there for a short stint where we interacted. Page 260
1 a clinical trial was too expensive to take each one in. 2 So we were trying to attach it to AAV2. 3 Does that make sense? 4 Q. Yes, understood. 5 MS. TOKER: Okay. I would like to 6 take a break and sort of see what my last 7 bit will be. 8 THE VIDEOGRAPHER: The time is 4:56. 9 We are off the record. 10 (Recess.) 11 THE VIDEOGRAPHER: The time is 5:09. 12 We are on the record. 13 BY MS. TOKER: 14 Q. Dr. Samulski, you didn't talk about the 15 substance of your deposition? 16 A. No. Just about pasta and carbohydrates. 17 Q. Well, I'm sorry I missed that one. 18 But, okay. When were you first contacted 19 about serving as an expert in this proceeding? 20 A. Oh, gosh. I'm not going to be able to 21 remember a date. It was maybe a year ago or so. I 22 can't remember. 23 Q. About a year ago? 24 A. Maybe, yeah. 25 Q. And do you know who contacted you? Page 259	1 Q. Sorry, who was this? 2 A. Olivier Danos, the CSO for REGENX, chief 3 scientific officer. 4 Q. Current CSO? 5 A. I think he's current. 6 Q. Okay. And so you've had dealings you 7 say -- 8 A. Twenty-five years, thirty years of just -- 9 he went there. We met in different places. 10 So when he was at REGENX, we would invite 11 him to meetings to represent the science and stuff like 12 that. 13 Q. At UNC you mean? 14 A. No. No. No. Conferences. You pick 15 people that are in an industry and people that are in 16 academics, and you try to have a mixture of 17 representation science wise. 18 Q. When you were organizing conferences in the 19 field of gene therapy? 20 A. Correct. 21 Q. Okay. And -- 22 A. The same would hold true for Jim Wilson, 23 and there was multiple interactions with Ken Miller, 24 who was the CEO, just by default, at the same meetings 25 and things like that. Employees that worked in our Page 261

1 space were limited. 2 So they would either come on his lab, my 3 lab, that lab, and he would be working for REGENX for a 4 number of years. 5 Q. Had your company had dealings with 6 REGENXBIO so, you know, AskBio or Bamboo or any of 7 the -- 8 A. Yeah, AskBio -- I'm going to say -- I'm 9 going to get it wrong, but there was like either a 10 horse trade of a license, they wanted cell 11 complimentary, and this was when all of the technology 12 was at GSK. So I don't think it was REGENX as much as 13 it was the University of Pennsylvania. 14 Q. Okay. So why don't we just clarify that a 15 little bit. 16 So your company, AskBio, had license 17 exchange with -- 18 A. GSK. 19 Q. GSK, but not with REGENX? 20 A. No. GSK, I think, held all of the U Penn 21 IP before it went into REGENX. I think they gave it 22 back to the university, or I don't know if they had any 23 ownership or not when they gave it back. But there was 24 an effort by GSK wanted our self-complimentary 25 technology. So I think we did a horse trade where Page 262	1 relationships and things. I don't think we 2 crossed-licensed or collaborated at any significant 3 level. 4 Q. Just to be clear, is the, whatever IP that 5 you licensed originally from GSK, is that now held by 6 REGENX? 7 A. No. 8 Q. Sorry? 9 A. No. 10 Q. No. Oh, okay. And your companies, AskBio 11 or Bamboo, have not otherwise been involved in 12 licensing U Penn technology or REGENX Biotechnology? 13 A. No. No. 14 Q. Okay. Now, are you receiving compensation 15 for your expert services here? 16 A. For here? 17 Q. For this proceeding. Sorry. 18 A. I hope so. 19 Q. Are you being paid? 20 A. I'm being told I am. I hope so. 21 Q. And how much have they told you they are 22 going to pay you for these services? 23 A. They didn't give me a number, and I don't 24 know what we ended up settling on. I don't spend 25 time -- Page 264
1 nobody spent any money, and we said we would like to 2 explore your AAV8 capsid. 3 Q. So this was a license for AAV8? 4 A. I think it was. 5 Q. Okay. So you and GSK -- 6 A. GSK. 7 Q. Do you remember what time frame? 8 A. I'm not -- I couldn't give you anything 9 accurate. I'm going to say 2000, maybe. 10 Q. So you had, as you say, a horse trade where 11 you licensed some of your self-complimentary technology 12 to GSK, and they licensed some University of 13 Pennsylvania technology to AskBio? 14 A. AskBio, yeah. 15 Q. And you think that that license involved 16 AAV8? 17 A. I think so. That was probably the only 18 capsid we were looking at at that time. 19 Q. Okay. And subsequent to that, or in 20 addition to that, have you had any similar or any 21 dealings with licenses having to do with REGENX or the 22 University of Pennsylvania? 23 A. No. I think after that, GSK gave the 24 package back to U Penn, and then I think REGENX spun 25 out of that. And then that was more of the scientific Page 263	1 Q. You did this without knowing how much 2 they're paying you, Dr. Samulski. 3 A. You won't understand it, but those are not 4 priorities in my life. 5 Q. Any ballpark of how much you are being 6 paid? Any idea? If you don't know, if you really 7 don't know, that's fine? 8 A. I don't. 9 Q. You don't? 10 A. I don't. 11 Q. Out of curiosity then, you know, why did 12 you decide to, you know, take part in this, and, you 13 know, subject yourself to questioning on this and all 14 that kind of stuff? 15 A. I think that's an easy answer. There's few 16 people that have been in the field long enough to know 17 it from its inception to where we're at. And I think 18 today you probably, unfortunately, got drenched with 19 the confusion that the field has brought upon itself by 20 promiscuously using terms and technologies that aren't 21 correct. And as a result of that, there always seems 22 to be what I call pseudo-dogma that gets out there 23 compared to reality. And so if I can bring any clarity 24 with that before it gets any worse, I almost feel 25 obligated. Otherwise, it's lost forever. Page 265

1 Q. Have you done other consulting then? 2 A. Yeah. 3 Q. What? 4 A. I did for Novartis and Genzyme were in a 5 similar, what I call pissing match, and who was 6 responsible for generating self-complimentary vectors, 7 and Genzyme had Barry Carter's patent, which was not 8 functional, couldn't practice it. And Novartis was 9 using the self-complimentary AAV for all of the SMA 10 kits. And so that was a very similar one where they 11 were trying to say what is a self-complimentary 12 molecule, why is it different than a double-stranded 13 genome and stuff like that. 14 So most of mine was trying to put the 15 guardrails on what can you call it, what will it do, 16 and how does it work. 17 Q. Anything -- any other consulting besides 18 that? 19 A. Consulting? 20 Q. Well, yeah. 21 A. I get asked all the time to consult. Too 22 many -- I can't even go through the list. But I don't 23 charge. I just do whatever they need, kind of thing, 24 and then they donate to the Columbus Children 25 Foundation. Page 266	1 Q. Other than listening, and I will have to 2 say that's a first for my -- an answer to how did you 3 prepare for your deposition question. Other than 4 listening to the audio -- so you recorded your own 5 declaration? 6 A. It's an AI recording. 7 Q. Okay. 8 A. The voice is actually pretty pleasant to 9 listen to, especially if you're trying to get to sleep. 10 Q. I will have to suggest that for my own 11 experts. 12 So other than listening to the AI-generated 13 audio recording of your declaration, did you meet with 14 anyone to prepare? 15 A. No. Physically, no. 16 Q. Or -- 17 A. Zoom, we had some Zooms, where we talked 18 through things. 19 Q. And I'm not asking you about the substance, 20 the subject matter. I'm just asking you now about sort 21 of the facts of who you met with and when and so forth 22 and so on. 23 So who did you meet with to prepare? 24 A. Mostly only Zooms. Nothing in person. 25 Q. And who was on the Zoom? Again, I'm not Page 268
1 Q. I meant more in sort of any other pissing 2 matches, is the technical term that -- 3 A. I haven't been asked in any formal way. 4 MS. MORRISON: I almost did a spit 5 take. 6 MS. TOKER: Apologies to the room. 7 Q. About how many hours did you spend 8 preparing your declaration? 9 A. You don't want to know. 10 Q. Sure I do. 11 A. For this one, like I said, I had to read so 12 many of my own papers out of 20 years ago. I would say 13 over two weeks of effort put into this. 14 Q. And about how many hours over the two 15 weeks? 16 A. Oh, I'm talking two weeks of hours. 17 Q. Okay. 18 A. Not two weeks of doing it, but of hours. I 19 would accumulate up to two weeks. 20 Q. And about how long did you spend preparing 21 for your deposition today? 22 A. Can I share it with you? I have an audio 23 of my declaration, and I listen to it everywhere I 24 drive so I can remember it, because I'm at an age now 25 where I'm losing my memory. Page 267	1 asking you to tell me what was discussed. 2 A. Our colleagues here, and Laura Coruzzi, who 3 is probably on the computer. 4 Q. Okay. And how many meetings did you have? 5 They can't help you. You have to just tell me whatever 6 you remember. 7 A. They're a blur; five, six, seven. 8 Q. Okay. And about how many hours? 9 A. They lasted about an hour each. I fit into 10 the Zoom schedule. 11 Q. Did you talk to anyone at REGENXBIO to 12 prepare? 13 A. No. I don't have any contacts with those 14 guys these days. 15 Q. Or anyone at the University of 16 Pennsylvania? 17 A. No. Unfortunately, no. 18 MS. TOKER: I think I do not have any 19 further questions, barring if you have some, 20 I may have some follow up to that. 21 MS. MORRISON: Sure. I do have a few 22 questions. 23 Can we go off the record for just like 24 two minutes? 25 THE VIDEOGRAPHER: The time is 5:22. Page 269

1 We are off the record. 2 (Recess.) 3 THE VIDEOGRAPHER: The time is 5:24 4 we're on the record. 5 EXAMINATION BY COUNSEL FOR REGENXBIO: 6 BY MS. MORRISON: 7 Q. Dr. Samulski, I just have a few questions 8 for you. 9 Where is the dystrophin protein primarily 10 expressed in the body? 11 A. Muscle. 12 Q. And if you were going to make a gene 13 therapy that included a dystrophin gene, what tissues 14 would you target with that gene therapy? 15 A. Muscle. 16 Q. Okay. I want to look at what was marked as 17 Exhibit 1059, it's this provisional, it's right here. 18 I want to start on the very first, front page. And I 19 just want to ask you about each of these inventors. 20 So who is Dawn Bowles? 21 A. Dawn Bowles was a post doc in the lab. 22 Q. How long had she been a post doc in your 23 lab at the end of 2004? 24 A. I'm going to say three to four years, 25 maybe. You're pull my memory now. Page 270	1 masked that site. So she did it at the structural 2 level, structural crystal. 3 Mavis, for us, would guide us with GPS 4 coordinates of if you want to do this, you should knock 5 out these amino acids or change those amino acids. 6 And we had a very productive relationship 7 where she gave us guidance and we made the wreck and 8 check and came back with biology. 9 Q. When you say Mavis, Dr. McKenna gave you 10 GPS coordinates, you mean to amino acids or something 11 else? 12 A. Yes, amino acids. 13 Q. So would you call Dr. McKenna. Was she a 14 person of ordinary skill in the art? More than that? 15 How would you characterize that? 16 A. There's been no other crystal structure 17 since she's passed away. So she is and has been the 18 whole definition of the AAV structural biology. 19 Q. So more than ordinary skill in the art -- 20 A. More than ordinary. 21 Q. Who is Joseph Rabinowitz? 22 A. He was a post doc of mine, probably when 23 this was happening, he was finishing leaving the lab 24 and he went to Pennsylvania, got a faculty position at 25 Temple and started his own lab. Page 272
1 Q. Josh Grieger we already talked about. 2 A. Is a graduate student. 3 Q. I'll skip him. 4 Who is Chengwen Li? 5 A. Chengwen Li was another post doc in the 6 lab. 7 Q. Okay. And who is Mavis McKenna? You 8 mentioned her several times today. 9 A. Mavis McKenna is the, unfortunately passed 10 away, but the goddess of the AAV crystal structure. 11 She solved all the crystal structures for all the 12 serotypes. So she was the mecca who understood AAV 13 structure. 14 Q. And what do you mean she was the -- can you 15 give a little more description when you say she was the 16 goddess of AAV crystal structure? 17 A. Mavis was on the paper when they started 18 the AAV2 crystal structure. And then she independently 19 solved AAV1, 2, 3, 4, 5, 6, 7, 8, 9, and then she 20 solved the structure of the heparan binding to the AAV. 21 She met the first antibodies crystal structures 22 interacting with the motifs that sat on the surface 23 demonstrating that the three -- the prongs that set out 24 at the three protocol where basically where viruses 25 would get blocked from tropism because the antibody Page 271	1 And Joe was one who was responsible for 2 developing how you connect AAV2 rep and swap in 3 different serotypes and produce production packaging 4 systems. 5 So Joe was an innovator that opened the 6 field up to trying different serotypes, otherwise we 7 were stuck with AAV2. 8 Q. So would you call Dr. Rabinowitz a person 9 of ordinary skill in the art? 10 A. No. 11 Q. What would you call him? 12 A. He was extraordinary in that he developed 13 technology that enabled the whole field to go forward 14 and that was probably not typical for a graduate 15 student, so most post DOCS. 16 Q. And is he still in the field? 17 A. I think so. 18 Q. Okay. And then the last person listed on 19 here is Richard Samulski. That's you, right, 20 Dr. Samulski? 21 A. I think that's correct. 22 Q. And at the time of this in late 2004, how 23 long had you been in the field? 24 A. Are you serious? Since 1978. 25 Q. So more than 25 years at this time? Page 273

1 A. Correct. 2 Q. All right. So let's look, if you go to the 3 back of this, it's at Figure 1A, which I think we 4 looked at before. 5 A. Yes. 6 Q. So you talked -- do you remember when you 7 looked at this with Ms. Toker? 8 A. Yes. Yes. 9 Q. And so the top of this Figure 1A says 36 10 candidates based on triage versus 83 from AAV1/2 11 alignment. Do you see that? 12 A. Yes. 13 Q. Can you explain what that means? 14 A. Well, what you typically do is you line 15 them up in the linear form that's on this page, and you 16 look for differences. And when you see differences, 17 you then look to see if they are similar amino acids or 18 completely different amino acids. If they're similar, 19 you would not rank those as high as a priority as if 20 they were different. And when I say different, we're 21 talking about is it hydrophilic versus hydrophobic? Is 22 it charged versus uncharged? Is it bulky group versus 23 a non-bulky group? And that was kind of a standard art 24 in the field. You look for amino acids that were 25 different, and if you saw a cluster of amino acids, you Page 274	1 for changes, but looking for hemology that said this 2 might be like that, because they have a similar repeat 3 clustering of charge. 4 Q. So I want to ask you a couple of follow-up 5 questions on that. 6 You mentioned you looked for to narrow -- I 7 think you said -- please correct me if I'm wrong -- to 8 narrow from the 83 to 36, you looked for amino acids 9 that were similar versus ones that were different in 10 terms of hydrophilicity, the size of the amino acid or 11 the charge. Do I have that right? 12 A. Correct. 13 Q. And so when you were choosing the 36 from 14 83, did you want to -- what were you -- were you 15 looking for amino acids that were similar or ones that 16 were different? 17 MS. TOKER: Objection to the form. 18 A. Different. We were looking for those that 19 were different. 20 Q. Okay. And you also talked about variable 21 region 8, and can you just -- what is variable -- where 22 is variable region 8 in the capsid? 23 A. If you look at the 580 region. 24 Q. Okay. 25 A. That cluster down there is where the virus Page 276
1 paid a little bit more attention, because it said it's 2 not just one, there's a group of them. And this is 3 where the variable regions that Mavis identified, so 4 it's eight variable regions that exist on this capsid, 5 and this is where most likely tropism is going to be 6 dictated. 7 So we focused on variable regions of which 8 variable region 8 exists in the 550, 560 in that 9 region, if you see that, the 585, there's lots of 10 differences there. That's where my lab mapped the 11 first AAV receptor identified, which was heparin 12 sulfate. 13 And, again, AAV -- all of the AAVs now when 14 they do libraries or any type of tropism studies, they 15 put their inserts into this variable region 8 because 16 on that footprint, this is the one that's the surface 17 sticking out most likely to read the Braille of what's 18 on the cell and then attach the receptor by initiating 19 a cascade of the next events. 20 So in that type of theme, that's where you 21 would have focused changes and things like that. 22 Again, when we looked at the nuclear 23 localization signals, those were clusters that were 24 understood from previous publications. So those were 25 like hemology searches. They weren't so much looking Page 275	1 goes from the linear map to the three-dimensional map. 2 It's the protrusion that sticks out from the virus, so 3 it's the furthest away from the core of the virus, and 4 it has a series of charged amino acids that are 5 involved in binding to receptors. 6 Q. And when you were talking about amino acids 7 that are similar versus different, I want to focus on 8 serine and asparagine. Are those similar, or are they 9 different? 10 MS. TOKER: Objection, form. 11 A. So, again, we would look for those that are 12 different, whether they're charged and be 13 phosphorylated and things of that nature over ones that 14 are not likely to be what's called post-translated 15 modified, PTMs. 16 Q. And I want to ask about the crystal 17 structure you talked about -- sorry. If you could go 18 to Figure 1B, the next figure, and this Figure 1B says: 19 12 candidates that tested based on amino acid and 20 crystal structure analyses. Do you see that? 21 A. Yes. 22 Q. And how did you select these 12 candidates 23 from the 36 in Figure 1A? 24 A. Mostly from Mavis, who ended up giving us 25 whether they were loop structures, or whether they were Page 277

1 concerned(sic), what's called beta barrel sheets, it 2 means they're very concerned(sic), and she was able to 3 identify from her crystal structure where she said -- 4 can I try to use this, which isn't going to show up on 5 the type? 6 Q. Sure. 7 A. VP3 is like a glove, okay. The thumb is 8 the HI loop, and the macavirus you had five of these 9 that snap together like a Lego: Click, click, click, 10 click, click. And what Mavis was able to show us was 11 that two amino acids in this HI loop are kissing three 12 amino acids from its neighbor; and therefore if you 13 wanted to make a change that may be involved in better 14 infection in muscle, make those changes so it opens up 15 and releases the DNA. 16 So that's where we went and made 2.5 based 17 on her taking the linear molecule and folded it into 18 this structure. And after she folded it into the 19 three-dimension, it was, guess what, this neighbor is 20 talking to this neighbor. 21 And so we had a different perspective of 22 how the virus worked. It was not just linear 23 differences. It was three-dimensional. 24 Q. And when you say you had a different 25 perspective, do you mean based on the crystal structure Page 278	1 Q. Is there an arrow anywhere within 50 amino 2 acids of position 665? 3 A. I'd have to count those, but I don't think 4 there's anything within proximity. 5 Q. Okay. All right. If you could go back in 6 this exhibit, Exhibit 1059, to page 9, and let me know 7 when you're there, Dr. Samulski. 8 A. Yeah. 9 Q. Opposing counsel looked at the definition 10 of the term tropism with you. I want to look at the 11 next paragraph. And do you see that there is a 12 definition of the term infection in that paragraph? 13 A. Yes. 14 Q. And also a definition of the term 15 transduction in that paragraph? 16 A. Yes. 17 Q. Okay. How does this application define 18 infection? 19 A. You want me to read it or just tell you? 20 Q. Just tell me in your own words. 21 A. Infection is the steps by which the virus 22 enters the cell and proceeds through a productive 23 infection. 24 Q. And is that consistent with how you talked 25 about infection in your declaration and your opinions? Page 280
1 or something else? 2 MS. TOKER: Objection to the form. 3 A. We had a different perspective based on the 4 crystal structure and how she allowed us to see who was 5 talking to who. 6 Q. Were there other people in the field at 7 that time, other than Dr. McKenna, who could have done 8 that kind of analysis? 9 A. The autonomous parvovirus people could have 10 stepped over in our field and helped. 11 Q. Could a person of ordinary skill in the art 12 at that time have done that analysis? 13 MS. TOKER: Objection, form. 14 A. In answering that question, I still can't 15 do it after 50 years of being in this space. It's a 16 different -- it's a different training. 17 Q. And in Figure 1B, I see there are -- it 18 mentions 12 candidates. What are the arrows in this 19 figure describing. There's arrows, do you see those? 20 A. Yes. 21 Q. Are those the 12 candidates? 22 A. Those are 12 candidates. 23 Q. Okay. And is there an arrow at position 24 665? 25 A. No. Page 279	1 A. I think so. 2 Q. Okay. And then how does this application 3 define transduction, again, in your own words? 4 A. Transduction would be expression of a gene 5 product that's carried by the viral genome, the vectors 6 in the transgene cassettes. 7 Q. You talked during your examination earlier 8 about the threonine insertion from AAV1 to AAV2. 9 Do you remember that conversation? 10 A. Yes. 11 Q. You were starting to say something during 12 your examination about removal of the threonines. If 13 you could explain that, please? 14 A. When we were doing the wreck and check 15 approach towards this AAV1, as we discussed earlier, 16 was an insertion, and so the alignment was off by one 17 amino acid. 18 So when Mavis identified that abnormality, 19 she said transfer the threonine over so you can mimic 20 more of what AAV1's loop looks like, and we did that. 21 And that's where 2.5 came from, and eventually 2.1. 22 It's that sole amino acid, of which when we changed all 23 20 amino acids, it was not the preferred amino acid. 24 So as a good scientist, we went to AAV1 and 25 say let's take Mother Nature's three best amino acids Page 281

1 and replace threonine, and we put them in one by one, 2 and lo and behold they were superior than threonine. 3 And I barked at my students saying you didn't do the 4 control. Take out the threonine so it looks like AAV2 5 and no longer goes into muscle. 6 So she took out the threonine, and to our 7 shock, it was the best muscle transduction we've ever 8 seen. 9 So it was complete spinning of the magnetic 10 poles from what we were doing as a systematic dogmatic, 11 if you do this, then this should happen and so forth 12 and so on. And we still don't understand that puzzle 13 to this day. 14 Q. You looked at a bunch of experiments that 15 were done in this provisional. How much time and work 16 went into these experiments that went into this 17 provisional? How many years or hours, however you want 18 to characterize it? 19 MS. TOKER: Objection to the form. 20 A. I would say every time we published a 21 paper, that was at least a four or five-year effort. 22 And depending on the number of authors that are on it, 23 they are all contributing with typically a lead author 24 that's totally dedicated for that four and a half 25 years, four to five years. That would be an average. Page 282	1 page 12. 2 A. Am I reading this one? 3 Q. Yes, that's the right one. And at the very 4 bottom, starting around line 29, the paragraph that 5 starts in representative embodiments? 6 A. Um-hum. 7 Q. This paragraph describes making -- 8 potentially making changes at amino acid positions. 9 I'm not going to read them all, but it's 260 through 10 268, correct? 11 A. Correct. 12 Q. So that's nine different potential changes? 13 A. Correct. 14 Q. And then if you go to the next page, page 15 13, and look at line 18, do you see where I am in the 16 paragraph that starts in particular embodiments? 17 A. Yes. 18 Q. And this paragraph is continuing to talk 19 about making changes, those same sorts of changes, 20 right? 21 A. Yes. 22 Q. And do you see the sentence that starts 23 based on crystal structure analysis? 24 A. Yes. 25 Q. What does it mean when it says: It will be Page 284
1 Some of them were longer. The 2.5 was much longer, 2 because it was the first time we've ever decided to try 3 to do rational design. Didn't know what we were doing, 4 so we had to wreck and check, wreck and check, wreck 5 and check. 6 Q. And was anyone else in the field, to your 7 knowledge, doing this sort of rational design that's 8 described in this provisional? 9 MS. TOKER: Objection. 10 BY MS. MORRISON: 11 Q. At this time? 12 MS. TOKER: Objection, form. 13 A. Ask that question again? 14 Q. Sure. Was any -- to your knowledge, was 15 anyone else doing this sort of rational design that's 16 described in this application at this time in 2005? 17 MS. TOKER: Objection, form. 18 A. I think we were the lead in this and 19 essentially all the other efforts that were going on, 20 which meant we had like a three or four-year head start 21 over everybody else. Once these were published, I 22 think it was fairly straightforward that people can say 23 oh, I see what he's doing. We should start doing that. 24 Q. Okay. I want to look at just a couple of 25 the changes. If you can go back to this provisional to Page 283	1 clear that in some instances, the 2 insertion/substitution can be moved one, two, three, 3 four or even five amino acids in either direction, and 4 it goes on. What is that referring to? 5 A. So this is going to the fact that the 6 crystal structure is actually working in three 7 dimension, and if you're in a loop, the loop could be 8 changed essentially anywhere from the base through that 9 fluidity back to a structured base. 10 So picking a specific location may not be 11 the optimum one. You may need to shift it in the left 12 direction or the right direction. And, again, the 13 crystal structure allowed you to see what that was 14 because if it was the beta barrel sheets, they had 15 rigidity, but if it was a loop, it had flexibility in 16 it, and we were referring to the loop, the HI loop, the 17 265 loop. 18 Q. Okay. I want to look now at the paragraph 19 that goes from the bottom of page 14 to the top of page 20 16 of this Exhibit 1059. Do you see where I am? It 21 says representative embodiment. 22 A. Yeah. 23 Q. This paragraph refers again to positions 24 260 through 268. Do you see that? 25 A. Yes. Page 285

1 Q. And then the sentence that is at the very 2 bottom of the page, suitable amino acids, do you see 3 where I am? 4 A. Yes. 5 Q. It says: Suitable amino acids include, and 6 it lists a number of amino acids. Do you see that? 7 A. Yes. 8 Q. And then the last sentence says: In 9 particular, embodiments of threonine is inserted or 10 substituted into the capsid subunit. Do you see that? 11 A. Yes. 12 Q. And what is that referring to? 13 A. Of all the amino acids that were being 14 suggested, threonine would be the preferred. 15 Q. So this particular paragraph is talking 16 about substituting the threonine into the subunit? 17 MS. TOKER: Objection, form. 18 A. Correct. Correct. 19 Q. All right. And then if you look at the 20 bottom of this page, paragraph that starts the 21 invention further provides? 22 A. Yes. 23 Q. What's being described, just in your own 24 words, in subparagraphs A through F that go from pages 25 15 to 16? Page 286	1 in this group of 260 to 268, you can do that in 2 combination with any of the others. 3 Q. Okay. All right. If you could turn to 4 page 18 at line 20. 5 A. In particular? 6 Q. Yes. And if you could just -- well, I'll 7 read that sentence for the record, and then I'll ask 8 you a question about it. 9 On page 18, line 20 of Exhibit 1059, it 10 says: In particular, aspects of the embodiments 11 described in the previous two paragraphs, the 12 substitution is a substitution of an amino acid that 13 cannot be phosphorylated for an amino acid that can be 14 phosphorylated. Do you see that? 15 A. Yes. 16 MS. TOKER: Objection. You left out a 17 word. 18 MS. MORRISON: Oh, I apologize. What 19 word did I leave out? 20 MS. TOKER: Substitution is a 21 substitution of an. 22 MS. MORRISON: An amino acid. 23 MS. TOKER: Amino acid, right. 24 MS. MORRISON: Okay. 25 Q. The substitution is a substitution of an Page 288
1 A. All the possibilities that one could start 2 to try substitutions or insertions. 3 Q. And so paragraphs A through F give about 4 ten different additional -- no, I'm sorry, more than 5 that, almost 20 different positions that could be 6 substituted? 7 MS. TOKER: Objection, form. 8 A. Some number that it could count up. 9 Q. Well, it's four individual ones in 10 paragraphs A through D, and then it lists in paragraphs 11 E and F positions 447 through 460? 12 MS. TOKER: Objection, form. 13 BY MS. MORRISON: 14 Q. Is that right? 15 MS. TOKER: Objection, form. 16 A. There's six in that one, and there's 17 another six in the other. So 12. There are about 19, 18 20, almost 20. 19 Q. Okay. And then on the same page 16, 20 there's a paragraph starting at line 11 that says the 21 modifications. Do you see that? 22 A. Yes. 23 Q. Just read that to yourself, and then in 24 your own words, what's this paragraph talking about? 25 A. Well, it's saying that if you make a change Page 287	1 amino acid that cannot be phosphorylated for an amino 2 acid that can be phosphorylated. 3 Did I read that correctly, doctor? 4 A. Yes. 5 Q. Now, what is that referring to? What does 6 that mean? 7 A. The substitution is a substitution of an 8 amino acid that cannot be phosphorylated for an amino 9 acid that can be phosphorylated, for example, 10 threonine. I don't know what you're asking me. 11 Q. Sure. So is this talking about removing a 12 threonine or substituting one in? 13 MS. TOKER: Objection, form. 14 A. It's putting threonine in. 15 MS. MORRISON: All right. I don't 16 have any further questions. Thank you, 17 Dr. Samulski. 18 FURTHER EXAMINATION BY COUNSEL FOR PETITIONER 19 BY MS. TOKER: 20 Q. I do. Let's just look at what you just 21 said, Dr. Samulski, on that paragraph. 22 So we're going to go back to page 18, line 23 20. And it says: In particular aspects of the 24 embodiments described in the previous two paragraphs, 25 the substitution is a substitution of an amino acid Page 289

1 that cannot be phosphorylated for an amino acid that 2 can be phosphorylated, e.g. threonine, right? Correct? 3 A. Correct. 4 Q. This is talking about substituting in to 5 replace a threonine with an amino acid that cannot be 6 phosphorylated, correct? 7 A. That's the way it reads. 8 Q. Right. And that's how you understand that, 9 correct? 10 A. Correct. 11 Q. Okay. So and we've talked about this 12 before that at position 716, for example, in AAV2, 13 there is a threonine that here they're saying you would 14 substitute in, for example, an asparagine to replace 15 the threonine in AAV2, correct? 16 MS. MORRISON: Objection to the form. 17 A. That's what it's implying. 18 Q. Okay. Now, to determine whether or not an 19 amino acid is phosphorylatable, you don't need a 20 crystal structure, do you? 21 A. It helps. If they're on the surface, 22 they're more likely to be phosphorylated -- 23 Q. I'm sorry, I didn't ask if it was 24 phosphorylated. I asked if it was phosphorylatable? 25 A. Oh, phosphorylatable? Page 290	1 crystal structure, correct? 2 A. Would have the potential. 3 Q. Would have the potential to be 4 phosphorylated, correct? 5 A. Correct. 6 Q. And similarly, a person of ordinary skill, 7 without a crystal structure, could look at a threonine 8 in a linear sequence and know that it has the potential 9 to be phosphorylated, correct? 10 A. Correct. 11 Q. Okay. I would like to look back at the 12 definition that you looked at with counsel just now of 13 infectivity on page 9 of the '126 provisional. 14 A. Which one are we in? 15 Q. I'm sorry, the '126, yeah, you got that, 16 and page 9. And you looked at the definition of 17 infection, right, as defined in the second paragraph on 18 page 9? 19 A. Um-hum. 20 Q. Correct? 21 A. Correct. 22 Q. So first of all, in your declaration, you 23 didn't use the term infection. You used the term 24 infectivity, correct? 25 A. Correct. Page 292
1 Q. Yeah. 2 A. No, the crystal structure wouldn't be as 3 essential. 4 Q. Right. You could look at a sequence and 5 know whether or not a particular amino acid is capable 6 of being phosphorylated, correct? 7 A. Right. But if you wanted more accurate, 8 you would look at a crystal structure. 9 Q. Okay. A crystal structure could give you a 10 lot of information, no question. But my question is 11 just simply, you and a person of ordinary skill in the 12 art could look at a linear sequence and know which 13 amino acids are phosphorylatable and which are not, 14 correct? 15 A. Potentially phosphorylatable. 16 Q. Potentially phosphorylatable, correct? 17 A. Correct. 18 Q. So a person of ordinary skill in the art 19 looking at a linear protein sequence could look at an 20 asparagine and know without a crystal structure that 21 asparagine cannot be phosphorylated, correct? 22 A. That would be correct. 23 Q. And that same person of ordinary skill in 24 the art could look at a linear protein sequence and 25 know that a serine could be phosphorylated without a Page 291	1 Q. Okay. So the word infection here you're 2 defining a different term than you've defined in your 3 declaration, correct? 4 A. I'm not sure if that's -- I'm not sure what 5 the two -- 6 Q. They are different words, right? 7 A. You mean the ending of the word infection 8 versus infectivity? 9 Q. Sure. Sure. 10 A. I'm only going by what you're saying. 11 Q. Okay. Fair enough. So let's look at what 12 you said in your declaration versus what you said in 13 the '126 provisional. 14 If you could look back, sorry to make you 15 do it, look back at your declaration, please, 16 Dr. Samulski, paragraph 38. Page 22, paragraph 38. 17 A. Right. 18 Q. You said here: Infectivity, on the other 19 hand, generally refers to the steps that occur after 20 the AAV vector reaches the cell of tissue of interest, 21 i.e., intake of the AAV vector into the cell 22 translocation to the nucleus and expression of the gene 23 of interest, right? Correct? 24 A. Correct. 25 Q. And in the '126 provisional, you say: As Page 293

1 used herein, infection of a cell by a parvovirus AAV or 2 recombinant virus vector of the invention means that 3 the parvovirus AAV enters the cell to establish an 4 active, i.e., lytic infection. Do you see that? 5 A. Yes. 6 Q. That's not the same definition, is it? 7 A. They're not the same. 8 Q. Right. 9 A. AAV is different than a recombinant virus. 10 Q. So -- but the two definitions are not the 11 same, the definition in the '126 and the definition in 12 your declaration, right? 13 A. I think what you're -- what I might be 14 confusing reading or -- a lytic infection is 15 infectivity, i.e., lytic infection, that is -- that can 16 only happen when(sic) a parvovirus, not a vector. 17 Q. But you say infectivity in your declaration 18 relates to a vector? 19 A. Right. Infectivity, on the other hand, 20 refers to steps that occur after the AAV vector reaches 21 the cell tissue of interest uptake, blah, blah, blah, 22 blah, blah, blah, blah, blah. 23 Q. So you're using the term infectivity to 24 refer to something to do with a vector in your 25 declaration, right? Page 294	1 an AAV vector, right? That's what you say. You use 2 the term AAV vector, right? 3 A. Infection of cells by a parvovirus AAV or a 4 recombinant viral vector means that parvovirus/AV 5 enters the cell to establish an active means expressing 6 lytic infection. So it has to be able to express. 7 Q. Fair enough. But you mention gene 8 expression from the viral genome in your definition of 9 tropism, right? 10 A. Whatever the cargo is, if it's a viral 11 genome or if it's the transgene of interest. 12 MS. TOKER: Okay. Fair enough. Let's 13 see, I do not have any questions, pending 14 any more questions from counsel, and then I 15 may have some additional ones. 16 MS. MORRISON: I don't have any more 17 questions. 18 MS. TOKER: Okay. Then we're done for 19 the day. 20 THE VIDEOGRAPHER: We are off the 21 record at 6:00 p.m., and this concludes 22 today's testimony. 23 The master video files will be 24 retained by Veritext Legal Solutions. 25 (Whereupon the deposition was concluded at 6:00 p.m.) (Signature reserved.) Page 296
1 A. In my declaration, I'm referring to the 2 steps that AAV is required to do to go through an 3 infection. 4 Q. Okay. And one of those steps is expression 5 of the gene of interest, right? 6 A. Yes. 7 Q. You don't mention gene expression in 8 relation to infection in your '126? 9 A. No, I didn't. 10 Q. You mention it in relation to tropism, 11 right? 12 A. No. Lytic means it has to express in order 13 to be lytic. 14 Q. But you just told me that an AAV vector 15 can't have a lytic infection? 16 A. So let's make sure we're on the same page. 17 AAV is going through the same steps of 18 binding entry transfer, and then it releases the 19 genome, and that genome gets expressed from the 20 promoter that one chooses. 21 In the AAV infection, it does the exact 22 same thing, and the genome is expressed from the AAV 23 promoters: P5, P19 and the P40, and that's what ensues 24 as a lytic infection. 25 Q. In your declaration, you're talking about Page 295	1 SIGNATURE PAGE 2 3 4 5 6 7 _____ 8 RICHARD JUDE SAMULSKI, Ph.D. 9 10 11 SUBSCRIBED AND SWORN to before me this _____ 12 day of _____, 2025. 13 14 15 _____ 16 NOTARY PUBLIC 17 18 My Commission expires: _____ 19 20 21 22 23 24 25 Page 297

1 C E R T I F I C A T E 2 I, Marisa Munoz-Vourakis, Stenographic Reporter, RMR, 3 CRR and Notary Public, the officer before whom the 4 foregoing proceeding was conducted, do hereby certify that 5 the witness whose testimony appears in the foregoing 6 proceeding, were duly sworn by me; that the testimony of 7 said witness were taken by me to the best of my ability 8 and thereafter transcribed under my supervision; and that 9 the foregoing pages, inclusive, constitute a true and 10 accurate transcription of the testimony of the witness. 11 I do further certify that I am neither counsel for, 12 related to, nor employed by any of the parties to this 13 action in which this proceeding was conducted, and 14 further, that I am not a relative or employee of any 15 attorney or counsel employed by the parties thereof, nor 16 financially or otherwise interested in the outcome of the 17 action. 18 19 IN WITNESS WHEREOF, I have hereunto subscribed my name 20 this 10th of MARCH, 2025. 21 22 23 24 <i>Marisa Munoz-Vourakis</i> MARISA MUNOZ-VOURAKIS Notary #20032900127 25 Page 298	1 _X_ 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 __ Federal R&S Not Requested - Reading & Signature was not 10 requested before the completion of the deposition. 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 Page 300
1 Susan Morrison 2 morrison@fr.com 3 MARCH 10, 2025 4 RE: Sarepta Therapeutics, Inc. v. The Trustees Of The University Of Pennsylvania 5 3/6/2025, Dr. Richard Jude Samulski, (#7189290). 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 Contact Veritext when the sealed original is required. 21 __ Waiving the CA Code of Civil Procedure per Stipulation of 22 Counsel - Original transcript to be released for signature 23 as determined at the deposition. 24 __ Signature Waived – Reading & Signature was waived at the 25 time of the deposition. Page 299	1 Sarepta Therapeutics, Inc. v. The Trustees Of The University Of Pennsylvania 2 Dr. Richard Jude Samulski (#7189290) 3 E R R A T A S H E E T 4 PAGE____ LINE____ CHANGE_____ 5 _____ 6 REASON_____ 7 PAGE____ LINE____ CHANGE_____ 8 _____ 9 REASON_____ 10 PAGE____ LINE____ CHANGE_____ 11 _____ 12 REASON_____ 13 PAGE____ LINE____ CHANGE_____ 14 _____ 15 REASON_____ 16 PAGE____ LINE____ CHANGE_____ 17 _____ 18 REASON_____ 19 PAGE____ LINE____ CHANGE_____ 20 _____ 21 REASON_____ 22 _____ 23 _____ 24 (Dr. Richard Jude Samulski) Date 25 _____ Page 301

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

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

(A) to review the transcript or recording; and

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

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

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

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

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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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