


1 IN THE UNITED STATES DISTRICT COURT
2 FOR THE MIDDLE DISTRICT OF PENNSYLVANIA
3 HARRISBURG DIVISION

3 TAMMY KITZMILLER, et al., : CASE NO.
4 Plaintiffs : 4:04-CV-02688
5 vs. :
6 DOVER SCHOOL DISTRICT, : Harrisburg, PA
7 Defendant : 3 November 2005
8: 1:00 p.m.

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11  TRANSCRIPT OF CIVIL BENCH TRIAL PROCEEDINGS
12 TRIAL DAY 21, AFTERNOON SESSION
13 BEFORE THE HONORABLE JOHN E. JONES, III
14 UNITED STATES DISTRICT JUDGE
15

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I N D E X
Kitzmiller vs. Dover Schools
4:04-CV-2688
Trial Day 21, Afternoon Session
4 November 2005

PROCEEDINGS

DEFENSE WITNESSES

Page

Dr. Scott Minnich:	
Direct examination by Mr. Muise	5
Cross examination by Mr. Harvey	145

1 PROCEEDINGS

2 THE COURT: Be seated, please. All right,
3 good afternoon to everyone. We have the first
4 witness then of the afternoon.

5 MR. MUISE: Your Honor, I know there was a
6 discussion during the lunch break over the
7 exhibits, and if we perhaps maybe could move
8 for those admissions, I believe there's no
9 objections on any of the exhibits.

10 THE COURT: Do you want to do them now?
11 All right, sure.

12 MR. MUISE: So it might be worthwhile to get
13 that housekeeping measure taken care of.

14 THE COURT: All right, I'll just read the
15 numbers and not describe them if you think
16 there's no objection, and you can for the sake
17 of speed, D-4, D-5, D-7, D-9, D-10, D-19,
18 actually these are all defendant's, 20, 21, 24,
19 25, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41,
20 42, 43, 54, 164, 284, 286, 287, 85, 86, 100,
21 116. What did I miss on the defendant's
22 exhibits?

23 MR. MUISE: I believe that's the complete
24 list. I don't think Mr. Gillen reviewed --

25 THE COURT: Say again? I'm sorry.

1 MR. MUISE: Yes, I believe that was the
2 complete list, Your Honor. That's all the
3 exhibits.

4 MR. ROTHSCHILD: Your Honor, you said 285,
5 and I didn't have that on my list. So --

6 THE COURT: No, if I said it I misspoke.
7 284 and 286. If I said that I misspoke.

8 MR. ROTHSCHILD: And then I thought there
9 was an Exhibit 50, and I don't remember what
10 it is, but I have that on my list.

11 THE COURT: What is D-50? Why don't we
12 check?

13 MR. ROTHSCHILD: D-50 is --

14 COURTROOM DEPUTY: It's already in.
15 It's already in.

16 MR. ROTHSCHILD: My mistake. Thank you.

17 THE COURT: You got to get up pretty early
18 to keep up with Liz, Mr. Rothschild.

19 MR. ROTHSCHILD: 5:20 this morning, Your
20 Honor.

21 THE COURT: Anything else? Any objections?

22 MR. MUISE: That's it, Your Honor.

23 MR. ROTHSCHILD: No objection.

24 THE COURT: All right, they're all admitted
25 the. Cross, P-817, P-91, and P-179. Any

1 additional exhibits that I've missed? And
2 are you moving for those, or are you moving
3 those in I should say.

4 MR. ROTHSCCHILD: Those we are moving in,
5 and if you could just give me just one moment,
6 I believe that's everything.

7 (Brief pause.)

8 MR. ROTHSCCHILD: That's it, Your Honor.

9 THE COURT: All right. No objection?
10 All right, then they're admitted as well.
11 All right. Having covered that, we're ready.

12 MR. MUISE: Thank you, Your Honor.
13 Defendants call Dr. Scott Minnich.

14 (Dr. Scott Minnich was called to testify
15 and was sworn by the courtroom deputy.)

16 COURTROOM DEPUTY: State your name, and
17 spell it for the record, please.

18 THE WITNESS: My name is Scott A. Minnich.
19 S-C-O-T-T, middle initial A, M-I-N-N-I-C-H.

20 DIRECT EXAMINATION BY MR. MUISE:

1 21 Q. Good afternoon, Dr. Minnich.

22 A. Good afternoon.

2 23 Q. Your Honor, may I approach?

24 THE COURT: You may.

25 (Brief pause.)

3 1 Q. Dr. Minnich, I've just provided you with
2 two binders. One of them is a black binder
3 marked as exhibits, which have some of the
4 exhibits that we'll be using for the course
5 of your testimony to assist you in your
6 reference. In the blue binder is a copy of
7 the demonstrative exhibits that we'll be using
8 through the course of your testimony again to
9 assist you from the witness stand. Sir, where
10 do you reside?

11 A. In Moscow, Idaho.

4 12 Q. And, sir, I'd ask you if you could please
13 open up that exhibit binder, the black binder
14 if you could, to Exhibit 201-A, as in Alpha.
15 It should be under Tab 1?

16 A. Got it.

5 17 Q. Is that a copy of your curriculum vitae,
18 sir?

19 A. It is. It's an abbreviated form for a
20 grant that was submitted.

6 21 Q. I want to, I want you to refer to it as
22 we go through some of your background and
23 qualifications to give expert opinions in this
24 case. Sir, what is your profession?

25 A. I'm an associate professor at the

1 University of Idaho in microbiology.

7 2 Q. Are you a tenured professor?

3 A. I am.

8 4 Q. And you said you teach at the University
5 of Idaho?

6 A. Correct.

9 7 Q. How long have you taught there?

8 A. Since 1989.

10 9 Q. Where else have you taught?

10 A. I was at Tulane for a year previous to
11 that.

11 12 Q. And what subjects have you taught at the
13 University of Idaho?

14 A. General microbiology for undergraduate
15 majors. Food microbiology, molecular genetic
16 techniques. I currently teach a 600 level
17 course, six credit course in infectious disease
18 for first year medical students.

12 19 Q. And what other subjects do you presently
20 teach?

21 A. Infectious disease and general
22 microbiology.

13 23 Q. You've been teaching science at the college
24 and graduate level for approximately eighteen
25 years, is that correct?

1 A. Correct.

14 2 Q. You said you're a microbiologist. Could
3 you explain for us what it is that you do as a
4 microbiologist?

5 A. Well, the primary focus is microorganisms,
6 in my particular case pathogenic organisms or
7 infectious disease agents. All the biological
8 sciences, you know, the disciplines have kind
9 of bled together. So we do molecular biology,
10 biochemistry, and are even doing a little bit of
11 cell biology, but primarily molecular genetics
12 is my bread and butter.

15 13 Q. And how would that different at all with
14 say a biochemist?

15 A. Again, you know, those are somewhat
16 artificial distinctions. I mean, we're more
17 focused at genetic programming of organisms
18 and how they respond to their environment,
19 biochemists may be looking at specific, you
20 know, organelles or suborganelles and how
21 they're assembled, and we do a little bit of
22 that as well.

16 23 Q. How would a microbiologist then differ
24 from a cell biologist?

25 A. A cell biologist is looking at more global

1 effects, you know, cell responses, involves
2 generally a lot of microscopy, and we don't do
3 a lot of that.

17 4 Q. And I know during the course of your
5 testimony we're going to be using some difficult
6 scientific terms and so forth, so I would ask if
7 you could, we need to speak slowly and loud and
8 clearly so our court reporter here can do his
9 best job taking all this down, okay?

10 A. I'll do my best.

18 11 Q. What is the name of the department that you
12 teach in at the University of Idaho?

13 A. My department is microbiology, molecular
14 biology, and biochemistry.

19 15 Q. Does that department then include all three
16 of those disciplines that we discussed, cell
17 biologists, biochemists, and microbiologists?

18 A. Correct.

20 19 Q. Now, sir, in your work and in your
20 profession do you conduct experiments?

21 A. I do.

21 22 Q. What is the focus of your experimental
23 work?

24 A. Right now we're focused on I'd say the
25 discipline of host parasite interactions.

1 So we work on bacterial infectious agents and
2 how they adapt during the infectious process.

22 3 Q. Does that focus on the bacterial flagellum
4 and the type three secretory systems?

5 A. It is. We've worked on that for the last
6 ten years in terms of these are two systems that
7 in our organism the genus Yersinia have opposing
8 regulations. So outside the host the cells
9 build a flagellum. Once they inspect a
10 mammalian host, flagellum biosynthesis is turned
11 off and you turn on the weapons systems that
12 these organisms have. So we've used those two
13 aspects kind of as opposing markers to follow
14 regulatory events.

23 15 Q. So the focus of your experimental work, I
16 assume also the focus of your research, and that
17 would include the bacterial flagellum and the
18 type three secretory systems?

19 A. Correct.

24 20 Q. Sir, do you incorporate intelligent design
21 into your experimental and research work?

22 A. I think the principles of intelligent
23 design are what we would call reverse
24 engineering would be, you know, a very
25 prominent part of what we do.

25 1 Q. And we're going to get into a little bit
2 more detail about that later in your testimony.
3 Sir, I want to talk about your education. What
4 degrees do you hold and where did you get them
5 from?

6 A. I have an undergraduate degree, a BS in
7 bacteriology and public health from Washington
8 State University.

26 9 Q. What year was that, sir?

10 A. Good question. 1975.

27 11 Q. If you want to look at your CV to help
12 refresh --

13 A. Okay.

28 14 Q. Okay, go ahead.

15 A. I obtained a masters degree in microbiology
16 from the University of Idaho, and a Ph.D. from
17 Iowa State University in 1981 in microbiology.

29 18 Q. Now, when you got your Ph.D. in
19 microbiology, what was the dissertation
20 that you wrote?

21 A. My research dissertation was on the
22 development of a rapid immunoassay for the
23 detection of salmonella. So it was really the
24 first application of enzyme immunoassays, which
25 are kind of a standard diagnostic procedure now,

1 to detecting salmonella.

30 2 Q. Would you give us a thumbnail sketch of
3 what this was about?

4 A. Yeah, it's an antibody based assay, and
5 our goal was to make something that was very
6 rapid. So the problem that we had, you know,
7 particularly in the food industry that it
8 could take up to a week using conventional
9 microbiological techniques to verify, detect
10 and verify that salmonella was present. This
11 was a rapid screening procedure that reduced
12 that time period to about 24 to 36 hours. So
13 for the food industry there was, you know,
14 incredible savings in terms of warehousing costs
15 before food is released. The FDA has zero
16 tolerance with respect to salmonella in foods.
17 So the test was developed as a prototype as a
18 graduate student, and then through the next four
19 years it was commercialized and applied to the
20 food industry. Variants of that procedure are
21 still used today.

31 22 Q. You got to see your work go from the
23 inception of an idea through the experimental
24 all the way to the commercialization of the
25 idea?

1 A. Correct.

32 2 Q. Did this work also include work on the
3 bacterial flagellum?

4 A. It did, because the antibodies we were
5 using were directed against the flagellar
6 filament, which is distinctive for the
7 salmonella. We had to have an assay that
8 incorporated the detection of over 2,400
9 different what we call serotypes, or variants,
10 of salmonella.

33 11 Q. Sir, do you belong to any professional
12 memberships?

13 A. I do. I'm a member of the American
14 Association for the Advancement of Science
15 and the American Society for Microbiology.

34 16 Q. I want to talk about some of you, we have
17 listed here positions and honors. That's how
18 you have it listed in your CV. You were on a
19 sabbatical from October of 2003 to May of 2004,
20 is that correct?

21 A. That's correct.

35 22 Q. And for what purpose?

23 A. I was a subject matter expert for the
24 Defense Intelligence Agency in Iraq. So I
25 served with the Iraq Survey Group looking for

1 weapons of mass destruction.

36 2 Q. What was the purpose of the need for a
3 microbiologist to be part of this survey group?

4 A. Well, that was the focus of the Iraq Survey
5 Group based on the intelligence that Iraq had
6 reestablished both their chemical and biological
7 weapons, or their nuclear, but we weren't part
8 of that aspect, but their programs. So our job
9 was to travel around the country and look for
10 these materials.

37 11 Q. How were you selected for that position?

12 A. I had a phone call in September of 2003,
13 actually August of 2003, asking if I had any
14 students in my laboratory that had military
15 experience. In part because we're registered
16 with the Center of Disease Control to work with
17 select agents, and that requires now with the
18 new regulations after 9/11 that everybody in my
19 laboratory has FBI clearance, and so I think we
20 were on a checklist of people that worked with
21 organisms that were of concern and, you know,
22 my remark was no, I didn't have any students
23 that fit that category, but in subsequent
24 conversations, you know, I was intrigued by
25 the idea, and volunteered.

38 1 Q. And why did you volunteer?

2 A. I volunteered because I grew up in a
3 military family. Both my father and
4 father-in-law are West Pointers, and it's
5 an area that I'm very interested in. Obviously,
6 I mean, it's work that we do, and it was an
7 opportunity to do field work and serve my
8 country at the same time.

39 9 Q. Sir, you said you've been teaching at the
10 University of Idaho since 1989 in microbiology,
11 correct?

12 A. Right.

40 13 Q. Is that correct?

14 A. That's correct.

41 15 Q. You also were a post-doctoral fellow at
16 Princeton University from 1984 to 1987, is that
17 correct?

18 A. That's correct.

42 19 Q. Could you tell us what that was?

20 A. This was after my doctorate, working in a
21 laboratory, the primary focus was developmental
22 regulation of flagellum biosynthesis, and one of
23 the model organisms for this system, caulobacter
24 crescentus.

43 25 Q. So during this period of research you

1 worked on flagellar biosynthesis, is that
2 correct?

3 A. That's correct.

44 4 Q. And you also were a post-doctoral fellow
5 at Purdue University from 1981 to 1983, is that
6 correct?

7 A. That's correct.

45 8 Q. And what did you do there?

9 A. There I was working in a molecular genetics
10 laboratory. The project focused on cloning and
11 studying the regulation of a toxin made by
12 bacillus thuringiensis. So that sounds kind of
13 esoteric, but this is the BT toxin that was put
14 into plants by Monsanto. So really the first
15 application of genetic engineering in
16 agricultural crops. So we cloned the gene,
17 studied its regulation, we handed it over to
18 Monsanto, it was modified, put into maize,
19 soybeans, you name it, cotton.

46 20 Q. Now, when you were at Purdue University
21 doing this work did you also engage in any
22 collaborative efforts with other faculty at
23 Purdue University?

24 A. Yes. There was an individual in the food
25 science department, Dr. Swaminathan, that had

1 worked on for years on salmonella detection.
2 We knew each other's work, so we started
3 collaborating. And I actually took my graduate
4 work ideas that he had as well and took our
5 assay to the next level. So it was a very
6 profitable interaction. Dr. Swaminathan I think
7 is just retiring this year as branch chief for
8 enteric disease at the Center for Disease
9 Control.

47 10 Q. During that collaborative effort did you
11 work on the bacterial flagellum?

12 A. We did. Again this was the focus of what
13 we called the antigen that we were trying to
14 detect.

48 15 Q. Now, you've published articles in peer
16 reviewed science journals, is that correct?

17 A. I have.

49 18 Q. Approximately how many?

19 A. 25 to 30. I'm missing a few on here,
20 but --

50 21 Q. And what are some of the journals that
22 you've published in?

23 A. Proceedings of the National Academy of
24 Science, Journal of Molecular Biology, and
25 Molecular and Microbiology, and Journal of

1 Bacteriology, which are really the primary
2 journals for what I work on. Applied
3 Environmental, there are a few others.

51 4 Q. Has there been a focus of your peer
5 reviewed science journal articles?

6 A. Over the last ten years we've focused on
7 flagellum biosynthesis and type three secretory
8 system regulation and pathogenic organisms.

52 9 Q. And again this is the focus of your
10 experimental work?

11 A. Correct.

53 12 Q. Through your experiments, your research,
13 and your writings have you become familiar
14 with the scientific evidence as it relates
15 to Darwin's theory of evolution?

16 A. I have.

54 17 Q. Would it be fair to say that your focus
18 is principally on the molecular level?

19 A. Correct.

55 20 Q. So you're a fellow with The Discovery
21 Institute, is that correct?

22 A. I am.

56 23 Q. And what does that mean?

24 A. My name is on one of their web pages listed
25 as a fellow. So it's more of a networking

1 opportunity, you know, for people that are
2 interested in this area of intelligent design.

57 3 Q. Are you an employee of The Discovery
4 Institute?

5 A. No. No, I'm not.

58 6 Q. Do they have any control over the work that
7 you do?

8 A. None whatsoever.

59 9 Q. Do they direct your work?

10 A. No.

60 11 Q. So is it fair to say that you're not on The
12 Discover Institute payroll?

13 A. I'm not.

61 14 Q. Has anyone ever accused you of that?

15 A. Yeah, there was an incident in 2003 in May
16 when Robert Pennock was invited to give a
17 seminar --

18 MR. HARVEY: Objection. Relevance, hearsay.

19 MR. MUISE: Your Honor, we'll all say we've
20 been hearing a lot of testimony today, or
21 throughout the course of this trial, vilifying
22 Discovery Institute, you know, talking about
23 this grand agenda. Some of it's been expressed
24 by their experts. I'm going through his
25 qualifications and I'm just demonstrating that

1 a lot these accusations aren't true, that these
2 are independent scientists who are working on
3 this for scientific reasons.

4 THE COURT: But he's not being offered to
5 defend The Discovery Institute.

6 MR. MUISE: That's correct, Your Honor,
7 but the fact is in terms of his, in terms of
8 his background and qualifications, I mean this
9 is how they've been really vilifying these
10 individuals --

11 THE COURT: I say again, I understand that,
12 and in another time and in another place he
13 might be competent to talk about how as a
14 fellow The Discovery Institute ran into some
15 difficulties, but for today I think it's
16 stipulated, his credentials are stipulated to,
17 and now we're going to get sidetracked on why
18 his bona fides as a fellow at The Discovery
19 Institute were called into question, and I just
20 don't think that's relevant. I understand, it
21 is not central or necessarily important to me
22 that we engage in an independent debate on The
23 Discovery Institute. It's just not helpful to
24 me, and I'll tell you that. So why don't we
25 proceed. I'll sustain the objection.

1 BY MR. MUISE:

62 2 Q. Sir, you're an advocate for intelligent
3 design?

4 A. I am.

63 5 Q. Is Darwin's theory of evolution
6 inconsistent with your private religious
7 beliefs?

8 A. No.

64 9 Q. Do you have a religious equipment to
10 intelligent design?

11 A. I don't.

65 12 Q. Why did you get involved with intelligent
13 design?

14 A. I read Mike Behe's book soon after it was
15 published, and of course he uses the bacterial
16 flagellum as a paradigm for, you know, his term
17 irreducible complexity, and I had arrived at
18 some of these same conclusions. So it intrigued
19 me, there was a friend I had in the physics
20 department that was interested in these
21 questions as well. So I think together we
22 started looking into these questions and what
23 intelligent design was and what it claimed, and
24 so it kind of blossomed from there.

66 25 Q. So how long have you been involved with

1 or associated with intelligent design?

2 A. Probably since about 1997, '98, or so.

67 3 Q. Have you ever been involved with
4 creationism or creation science?

5 A. No.

68 6 Q. Why not?

7 A. You know, I'm old enough that I was around
8 during those debates, and I never participated
9 because I don't agree with the approach. I
10 don't think you mix religion with your science.
11 I don't think you use Genesis as a filter of how
12 you interpret your scientific data, you know,
13 empirical evidence.

69 14 Q. So what is your commitment then to
15 intelligent design?

16 A. I think it fits. I think it's a good
17 paradigm. We can discuss that as we go through
18 some of the slides, but it's consistent with the
19 empirical evidence and standard scientific
20 reasoning that we employ.

70 21 Q. Do you perceive efforts on the part of
22 opponents of intelligent design to equate
23 intelligent design with creationism?

24 A. I think there is. You know, often times
25 when it's mentioned in the press it's referred

1 to as intelligent design creationism,
2 anti-evolutionism, you know, these types of
3 terms are often equated, and I think that's
4 a misrepresentation.

71 5 Q. Sir, is there unanimity amongst biologists
6 regarding all aspects of Darwin's theory of
7 evolution?

8 A. No, there isn't.

72 9 Q. Is intelligent design different in that
10 respect?

11 A. No. There's a broad spectrum of people in
12 terms of, you know, how they interpret the data.

73 13 Q. Does intelligent design continue to
14 develop?

15 A. Yes. I mean, it's I think developed quite
16 a bit since my involvement, and maybe if you
17 trace it back to the early 90's.

74 18 Q. Now, sir, you testified that you authored
19 numerous peer reviewed articles, many in
20 scientific journals, and I believe you testified
21 the one area in which you published the most was
22 on the topics of molecular biology and in the
23 past ten years specifically the bacterial
24 flagellum and the type three secretory system.
25 Is that fair?

1 A. Correct.

75 2 Q. Have you authored any articles appearing
3 in peer reviewed science journals that make
4 intelligent design arguments?

5 A. Not directly.

76 6 Q. You say not directly. Are there articles
7 that provide support for intelligent design
8 arguments that you've published?

9 A. I think so. I think all of them do.
10 I think they're, you know, dissecting intricate
11 components of subcellular organelles that
12 support the general contention of irreducible
13 complexity and design.

77 14 Q. I want to ask you if you agree with this
15 testimony that was provided by Dr. Miller. He
16 testified that, "It is a standard scientific
17 practice for scientists to point to the
18 scientific literature, to point to observations
19 and experiments that have been done by other
20 people in other laboratories, have been peer
21 reviewed, have been published, and to cite to
22 that evidence, cite to those data, and to cite
23 to those experiments in their arguments." Do
24 you agree with that?

25 A. I agree with that. That's standard

1 practice in scientific, you know, endeavors.

78 2 Q. And is that what intelligent design is
3 doing?

4 A. Yes.

79 5 Q. This is something that scientists do
6 routinely?

7 A. Oh, yes. It's critical.

80 8 Q. I want to ask you if you also agree with
9 Dr. Miller that the question is not whether you
10 or any other scientists have done experiments
11 in your own laboratories that have produced
12 evidence for a particular claim, the question
13 is whether or not the inference that you or
14 other scientists drawing your analysis from
15 that data are supported. Do you agree with
16 that?

17 A. I do. I think, you know, that's part of
18 the scientific endeavor. I mean, either you're
19 doing your own experiments and the data that
20 you generate you try to fit into the general
21 knowledge that's available, whether it's
22 consistent or inconsistent, and you can look
23 at other people's data through this published
24 and view it perhaps from a different perspective
25 and come up with a new interpretation. And

1 that's standard. I think Watson and Crick are
2 examples of that in terms of doing minimal
3 experiments, but at the same time taking
4 information from various sources and melding
5 it into an explanatory model, and so that can
6 be profitable.

81 7 Q. Explain for us what you -- you mentioned
8 Crick and Watson. What are you referring to?

9 A. Well, the fact that, you know, they really
10 didn't do any wet lab experiments. They took
11 Shordhop's work from Columbia University,
12 Rosalyn Franklin's x-ray crystallography data
13 coordinate in terms of the structure of
14 nucleotides and built models and came up with
15 a double helical structure, so --

82 16 Q. And those are the two that received the
17 Nobel prize for --

18 A. Right.

83 19 Q. -- developing the architecture I guess of
20 the double helix, DNA?

21 A. Right, solving instruction.

84 22 Q. Now, is this method, this process, is this
23 what intelligent design advocates are engaged
24 in?

25 A. Well, I don't want to equate it with, you

1 know, in terms of something that is critical as
2 a double helix, but at the same time we're
3 looking at across the landscape of empirical
4 data and asking the question does it fit with
5 the Darwinian mechanism of mutation and natural
6 selection to generate, you know, the deep
7 diversity of life.

85 8 Q. Now, you testified previously that you
9 though do experiments that you believe
10 supports intelligent design?

11 A. I do. I do.

86 12 Q. Are there peer reviewed articles that make
13 arguments for aspects of intelligent design that
14 you're aware of?

15 A. I think there are around ten of them now
16 that are in the literature that address this,
17 I'm not sure of an exact number, but within the
18 last couple of years.

87 19 Q. Do you perceive a bias against publishing
20 intelligent design articles in science journals?

21 A. I think there's --

22 MR. HARVEY: Objection, Your Honor.

23 Speculation.

24 MR. MUISE: I'm asking for his perception,
25 Your Honor.

1 THE COURT: I think it's a fair question.

2 I'll overrule the objection. You can answer.

3 THE WITNESS: I think that's on public
4 record, there's a paper published by a journal
5 from the Smithsonian Institute last summer by
6 Stephen Meyer. Brixter and Berg was the editor,
7 and I think it was a --

8 MR. HARVEY: Your Honor, objection.
9 Hearsay. He has no firsthand knowledge
10 of it.

11 THE COURT: Well, the question was a yes
12 or no question. The answer was yes. That was
13 accepted. The objection was overruled on that
14 basis. If he gets into the particulars he may
15 be getting into hearsay.

16 MR. MUISE: But he testified as to
17 perception. If he has an understanding,
18 he said it's a public record. I mean, you're
19 saying that --

20 THE COURT: A newspaper article is not a
21 public record, and you've certainly argued
22 vigorously in this case that it's not, and
23 we've spent a lot of time on that. Mr. Muise.
24 You want to tell me now it's a public record?
25 We can spare a lot of argument tomorrow if it

1 is.

2 MR. MUISE: Your Honor, I mean, a public
3 record not in the sense of I think the term
4 that you're using with the hearsay.

5 THE COURT: No, it's not in the way that I'm
6 using it. It's the way that we've argued it.
7 Don't insult my intelligence. It's not. The
8 objection is sustained.

9 MR. MUISE: I understand, Your Honor. And
10 I certainly did not intend to convey any message
11 that I was --

12 THE COURT: I understand that. Let's keep
13 going. Proceed.

14 BY MR. MUISE:

88 15 Q. Sir, you authored an article entitled
16 Genetic Analysis of Coordinate Flagella in
17 Type Three Regulatory Circuits and pathogenic
18 Bacteria, correct?

19 A. I did.

89 20 Q. And was this article published?

21 A. It was published in the proceedings of a
22 meeting in 2004.

90 23 Q. And who was it published by?

24 A. The Wessex Institute. It's an institute
25 of higher education in the U.K.

91 1 Q. It's not a religious organization?

2 A. No.

92 3 Q. This article was part of a conference, is
4 that correct?

5 A. That's right. It was a conference titled
6 "Design In Nature II" that was held in Rhodes,
7 Greece in July of that year.

93 8 Q. And what was this conference about?

9 A. The conference I think would fit under the
10 broad category of a new area in science called
11 biomimetics where engineers, architects are
12 brought together with biologists to, as a
13 mechanism of cross fertilization. Engineers
14 are recognizing that biological systems have
15 solved some pretty difficult problems, and
16 so there's a lot in terms of nanotechnology
17 structural analysis that can be gleaned from
18 biological systems.

94 19 Q. Do you consider this article to be an
20 intelligent design article?

21 A. Primarily it's a review of our work looking
22 at coordinate regulation in type three systems,
23 but there's a section where I address
24 intelligence aspects of it.

95 25 Q. Who attended this conference? I believe

1 you said there were engineers and scientists?

2 A. Biologists, engineers, design engineers,
3 aircraft engineers, architects.

96 4 Q. Was this a creationists conference?

5 A. No.

97 6 Q. Now, this article that was published by
7 the Wessex Institute, was it peer reviewed?

8 A. There was, you had to submit the paper
9 before it would be accepted or before you
10 could provide or present it at the conference.
11 So I actually wrote that when I was in Baghdad,
12 communicated it by e-mail, and it was peer
13 reviewed, I'm not sure what the peer review is,
14 it's not as rigorous as, you know, a primary
15 journal article, but there is that process.

98 16 Q. Could you just briefly explain for us what
17 this article is about? We're going to be
18 talking about it in more detail later in your
19 testimony, but if you could just give us sort
20 of a thumbnail sketch?

21 A. Well, it looks at the work that we've been
22 involved with why bacteria repress motility in
23 a mammalian host environment and how they
24 activate type three secretion systems and why
25 these systems are segregated. It also addressed

1 the question that had come up in these debates
2 on intelligent design that the type three
3 secretory system represented a structural
4 intermediate for the flagellum, and Ken Miller
5 has published on this. And so there were
6 arguments against that position in particular.

99 7 Q. Did this conference demonstrate the utility
8 of intelligence design as a scientific theory?

9 A. I think so, in terms of our approach and
10 what we found out.

100 11 Q. How so?

12 A. Well, again the types of the questions we
13 asked looking for reasons why these two systems
14 would be regulated in an opposing manner, the
15 reverse engineering techniques that proved
16 profitable. We also, although I don't want to
17 bore everybody with the details, but in part to
18 me the most interesting aspect is that one of
19 the organism we work with, yersinia pestis,
20 which causes the bubonic plague, so this is an
21 organism that's estimated to have killed two
22 hundred million people in recorded history,
23 activates its virulence genes by temperature.

24 So we were interested in terms of what's
25 the thermostat, how does the cell sense

1 temperature and how does it shut genes off and
2 turn others on, and it turned out through a
3 genetic approach mutational analysis that the
4 trigger, from one sense you can look at this
5 almost as kind of dissecting the trigger of a
6 nuclear weapon in terms of its potential effect,
7 turned out to be DNA itself, which was a
8 surprise to us.

9 It told us that the DNA molecule is just
10 not a reservoir for digital information, but
11 the three-dimensional structure that it can
12 conform to under different environments imparts
13 information as well, and that was a surprising
14 observation and I think we did that by reverse
15 engineering and looking at temperature
16 parameters of DNA molecules.

101 17 Q. Sir, are you familiar with the book Of
18 Pandas and People?

19 A. I am.

102 20 Q. Did you contribute to any portions of
21 this book?

22 A. I did not.

103 23 Q. Are you aware of any prior drafts of
24 this book?

25 A. No.

104 1 Q. I take it then you didn't contribute
2 to any prior drafts of the Pandas book?

3 A. I didn't.

105 4 Q. Sir, is it your understanding that this
5 book Pandas is part of the controversy in
6 this lawsuit?

7 A. I'm aware of that.

106 8 Q. What is your understanding of how this book
9 will be used at the Dover High School?

10 A. It's mentioned in a short statement read to
11 students before the, to biology students, 9th
12 grade biology students, and it's also on deposit
13 or reserve or in the library as, you know, a
14 reference in the library.

107 15 Q. Now, this book was published in 1993,
16 correct?

17 A. That's correct.

108 18 Q. Would you recommend that it be used as the
19 primary text for a biology class?

20 A. No, I would not.

109 21 Q. Why not?

22 A. Well, it's not a primary biology text, and
23 I think that's stated in the introduction.

110 24 Q. And the other reason?

25 A. Well, it's outdated as well. It's an old

1 book. I mean, in the course of biology ten
2 years is light years now in terms of our
3 progression.

111 4 Q. Would you recommend that it be used in the
5 manner that Dover High School is using it?

6 A. I do.

112 7 Q. Do you have experience with this book being
8 used in a biology course at the high school
9 level?

10 A. I do. I had children that attended private
11 school in Moscow, Idaho. Being a scientist they
12 asked me to review their biology curriculum.
13 They had, you know, a curriculum that I thought
14 was inadequate. I recommended that they use
15 Miller and Levine, which I think is the same
16 book that's being used in Dover, and supplement
17 it with Pendas and People.

113 18 Q. What year was this?

19 A. I'm not sure exactly. I'd say '95 or '96.

114 20 Q. Are they still using the Pendas book?

21 A. They still have it. In fact, I got a copy
22 from them.

115 23 Q. Why did you recommend Pendas as a
24 supplement?

25 A. It addresses some of the aspects of

1 Darwinian evolution from a different perspective
2 in terms of the fossil record, in term of other
3 interpretations of homology, molecular aspects.
4 There was I think in this book a brief
5 introduction to, although not stated, but
6 irreducible complexity, the blood clotting
7 system, that Mike Behe contributed.

116 8 Q. Did you think it was beneficial for the
9 students to have exposure to this book?

10 A. Yes. I think any time you expose students
11 to, you know, different interpretations it's
12 good. It promotes critical thinking.

117 13 Q. Have you subsequently had any experience
14 with these students from this school since
15 recommending this curriculum change?

16 A. Two of the students came through our
17 department and have since graduated, and
18 they were excellent students. Both of them
19 I think had published peer reviewed papers by
20 the time they had finished their undergraduate
21 degrees, which is an outstanding achievement
22 for undergraduates.

118 23 Q. Do you have any way of assessing their
24 critical thinking skills compared with other
25 students?

1 MR. HARVEY: Objection, Your Honor. Beyond
2 the scope of the expert report. I have not
3 objected for a few questions here, figuring a
4 little latitude is appropriate, but it's clearly
5 not the area with which he's been proffered and
6 the content of his expert report.

7 THE COURT: Mr. Muise?

8 MR. MUISE: I'm going to move on, Your
9 Honor. I think what it's establishing is
10 obviously with regard to his expertise from
11 the perspective of science education. I
12 haven't proffered him obviously yet as an
13 expert.

14 THE COURT: Well, just the critical skills
15 of the students who would have, along with his
16 own child --

17 MR. MUISE: I'm sorry, Your Honor?

18 THE COURT: Whose critical skills are we
19 talk about?

20 MR. MUISE: The students'.

21 THE COURT: The students in his own child's
22 class?

23 MR. MUISE: No, these are students who have
24 gone through this biology course where the
25 curriculum included Pandas as part of the

1 supplemental books, and --

2 THE COURT: That would appear to be beyond
3 the scope of this report. I think you can
4 probably concede that point.

5 MR. MUISE: Well, in the report he
6 specifically talks about Pandas being a
7 good book and it promotes good science
8 education.

9 THE COURT: If I recall the testimony
10 correctly, correct me if I'm wrong, sir, this
11 is a school that your child attends and they
12 use Pandas as an ancillary resource?

13 THE WITNESS: Right. I mean, my children
14 have since graduated, but --

15 THE COURT: But when they were there they
16 used it?

17 THE WITNESS: They did, right.

18 THE COURT: I don't know what basis he could
19 judge -- well, I do know the basis he could
20 judge, but it does appear to go outside the
21 report, Mr. Muise. Unless you can, if you can
22 point me to something in the report, and it's a
23 long report, if there's something in there that
24 you want to hang your hat on, I'll listen.

25 MR. MUISE: Well, it's not just the report.

1 He was asked about these same questions during
2 his previous deposition, and on his report he
3 said, "I read and am familiar with the text of
4 Pandas, it's a good text, it critically analyzes
5 various aspects of Darwin's theory, it asks
6 critical questions in terms of the evidence
7 and mechanism required to drive evolution. Such
8 questions are essential for the advancement of
9 science, makings students aware of the
10 controversy in the science community, it's good
11 to students and it's good to science."

12 COURT REPORTER: Mr. Muise? Mr. Muise?

13 THE COURT: Yes, we have lots of time.

14 Slow your cadence down if you could.

15 MR. MUISE: Your Honor, I mean I can, I
16 think I've got through the testimony of the
17 part that I wanted to and I can move on to
18 the next --

19 THE COURT: Well, that may be a fair
20 question once we get out of the -- we're
21 still on qualifications, are we not?

22 MR. MUISE: We are.

23 THE COURT: All right. Why don't you --
24 I'll reserve judgment. If you want to come back
25 around and lay a foundation for that question

1 on your examination, I'll hear any objection
2 Mr. Harvey has at that time. So why don't
3 we move on. I'll sustain it, but with needs
4 to reassert it, I think there's maybe a
5 foundational problem with the question, too,
6 but that wouldn't stop you necessarily from
7 asking it under different circumstances.

8 BY MR. MUISE:

119 9 Q. Dr. Minnich, do you think that schools
10 should teach students the theory of evolution?

11 A. Absolutely.

120 12 Q. Why?

13 A. It's critical. I mean, it's critical to
14 biology to have a firm foundation in evolution.

121 15 Q. By advocating intelligent design is it your
16 goal to not have the theory of evolution taught
17 in a biology class?

18 A. Not at all.

122 19 Q. Has that ever been your goal?

20 A. No.

123 21 Q. At this point do you believe that
22 intelligent design should be fully integrated
23 into a science curriculum?

24 A. I don't.

124 25 Q. Why not?

1 A. Well, you've got an old textbook and you
2 lack the standards for teachers and assessment
3 for students.

125 4 Q. You think it's appropriate to supplement
5 the science curriculum by making the students
6 aware of intelligent design as Dover has done
7 in this case?

8 A. Yes, I think it's advantageous.

126 9 Q. There's one last area on your CV I want
10 to address, and that's the research support.

11 A. Correct.

127 12 Q. What is significant about research support
13 for a scientist?

14 A. Well, to be successful and to do
15 experiments you've got to have extramural
16 support and, you know, it's to be likened
17 to running a small business within a research
18 community. You know, I have to pay my graduate
19 students, technicians, pay for supplies, animal
20 care, and there's overhead associated with it as
21 well. So funding is very important.

128 22 Q. Have you been awarded any significant
23 grants?

24 A. Well, right now we have an NIH grant
25 for five years for, with myself and two

1 collaborators, for 1.8 million dollars.

129 2 Q. And what is significant about NIH grants?

3 A. Well, I mean for infectious disease
4 that's the primary source for funding. It's
5 competitive.

130 6 Q. Now, the research that you're being funded
7 by NIH, does that include research on the
8 flagellum and the type three secretory system?

9 A. It does.

10 MR. MUISE: Your Honor, may it please the
11 court, I tender Dr. Scott Minnich as an expert
12 in microbiology, evolution, intelligent design,
13 and science education.

14 MR. HARVEY: Your Honor, I don't believe
15 this expert was proffered previously in science
16 education, and I'm not aware of that. His
17 reference in the expert report to Pandas and
18 People being good science, and his general
19 statement about it being good to make students
20 aware of the controversy, but there's no
21 reference to an expert in science education.

22 MR. MUISE: Your Honor, I mean we stipulated
23 to the qualifications of the matters that were
24 covered in the expert report. He testified
25 that using Pandas, making students aware of

1 intelligent design, was good for science
2 education. He's been teaching science for
3 eighteen years at the college level.

4 THE COURT: Did you have a, and I may have
5 known this and forgotten it, but was there a
6 written stipulation as to the expert or just
7 simply an understanding?

8 MR. MUISE: There's a written stipulation
9 I believe, I don't have a copy in front me, but
10 I believe it says effective of the matters that
11 were covered in the expert reports, that their
12 experts would testify as to the matters
13 addressed in the expert reports.

14 MR. HARVEY: Your Honor, addressing the
15 defendant's pretrial memorandum, it says will
16 testify, it says questions, in other words
17 critical questions in terms of the evidence
18 and mechanism required to drive evolution are
19 essential to the advancement of science and
20 that making students aware of the controversy
21 in the science community is good for students
22 and is good for science.

23 THE COURT: Well, we're having a bench
24 trial, and your objection is that he's being
25 offered on science education. But it seems to

1 me that the real objection gets to potential
2 testimony that would be outside of his report,
3 isn't it?

4 MR. HARVEY: That's correct, Your Honor.
5 And I don't believe he has been qualified in
6 the area of teaching at the high school level
7 for example.

8 THE COURT: Well, I understand that, and
9 that may go to a specific objection, but so we
10 don't waste time on this, which becomes at some
11 point a semantical argument, I'll take a precise
12 objection as it goes to his testimony on that
13 point, but I'm going to overrule your objection
14 at this point and allow him to testify on that
15 basis. I think that's the better course rather
16 than to try to split hairs at this point as to
17 what he's qualified to testify, what area he's
18 qualify to testify. And you have his report.
19 If you have an objection as to an individual
20 question or an area that Mr. Muise gets into,
21 I'll hear your objection on that, all right?
22 So we accept him for the purposes and
23 qualifications as set forth by Mr. Muise,
24 and Mr. Muise, you may proceed with your
25 examination.

1 BY MR. MUISE:

131 2 Q. Thank you, Your Honor. Dr. Minnich, I want
3 to first review with you the opinions you intend
4 to offer in this case before we get to the basis
5 for these opinions. Sir, do you have an opinion
6 as to whether intelligent design is science?

7 A. I do.

132 8 Q. What is that opinion?

9 A. It is.

133 10 Q. Do you have an opinion as to whether
11 intelligent design makes testable scientific
12 claims?

13 A. I do.

134 14 Q. And what is that opinion?

15 A. It does.

135 16 Q. Do you have an opinion as to whether
17 intelligent design causes a causative
18 argument for design?

19 A. I do.

136 20 Q. What is that opinion?

21 A. It does.

137 22 Q. Do you have an opinion as to whether
23 intelligent design requires the action of
24 a supernatural creator?

25 A. I do.

138 1 Q. What is that opinion?

2 A. It does not.

139 3 Q. Do you have an opinion as to whether
4 intelligent design is creationism?

5 A. I do.

140 6 Q. What is that opinion?

7 A. It is not.

141 8 Q. Do you have an opinion as to whether
9 intelligent design is a religious belief?

10 A. I do.

142 11 Q. And what is that opinion?

12 A. It is not.

143 13 Q. Do you have an opinion as to whether
14 Darwin's theory of evolution is a fact?

15 A. I do.

144 16 Q. And what is that opinion?

17 A. It is not.

145 18 Q. Do you have an opinion as to whether there
19 are gaps and problems with Darwin's theory of
20 evolution?

21 A. I do.

146 22 Q. Sir, what is that opinion?

23 A. There are such gaps.

147 24 Q. Do you have an opinion as to whether making
25 students aware that Darwin's theory is not a

1 fact promotes good science education?

2 A. I do.

148 3 Q. And what is that opinion?

4 A. I think it does. It does.

149 5 Q. Do you have an opinion as to whether making

6 students aware of the existence of gaps and

7 problems with Darwin's theory of evolution

8 promotes good science education?

9 A. I do.

150 10 Q. And what is that opinion?

11 A. It does, definitely.

151 12 Q. Do you have an opinion as to whether making

13 students aware of intelligent design promotes

14 good science education?

15 A. I do.

152 16 Q. And what is that opinion?

17 A. It does.

153 18 Q. Sir, do you have an opinion as to whether

19 providing students with the opportunity to

20 review the book Of Pandas and People promotes

21 good science education?

22 A. It does.

154 23 Q. Do you have an opinion on that?

24 A. I do, and it does.

155 25 Q. Thank you. Sir, I want to talk now about

1 the, turn now to the nature of the intelligent
2 design argument, and I believe you have provide
3 some demonstratives to assist in your testimony
4 here, is that correct?

5 A. That's correct.

156 6 Q. Sir, what is intelligent design?

7 A. We have summarized here in the first slide.
8 I'll just read it, "Intelligent design is a
9 scientific theory, and it holds that the deep
10 complexity and clearly evident design in
11 organisms is the result of an intelligent agent
12 or cause. Given that even the simplest cells
13 are comprised of nanomachines that currently
14 defy our own intelligent capability to produce,
15 yet have the general features of many machines
16 we have made on a larger scale, intelligent
17 design theory is simply an inference to the best
18 explanation as to the origin of the design."
19 If I could just summarize this perhaps in a more
20 simpler form?

157 21 Q. Yes.

22 A. All biologists recognize design in nature.
23 So I think the question boils down to whether or
24 not it's real design or apparent design, as some
25 people hold. Thirty years ago we didn't know

1 about molecular machines and this concept of
2 irreducible complexity, which we'll talk more
3 about. We didn't know the sophistication of the
4 information storage system in nucleic acids of
5 RNA and DNA that have been likened to digital
6 code that surpasses anything that a software
7 engineer at Microsoft at this point can produce.
8 Certainly Darwin didn't know about this.

9 So we don't have a Darwinian mechanism
10 to explain these things in terms of natural
11 selection and mutation or variation. On the
12 positive side, because these are similar to
13 machines that we have made in a macro scale,
14 we know what it takes to make them. We know
15 digital information storage systems that we
16 can infer design, looking at the empirical
17 evidence, and maybe a uniformitarian aspect of
18 cause and effect in the world that we live in,
19 when we find these things they're the product of
20 intelligence.

21 So we're looking at the empirical evidence.
22 We find irreducible complex systems. When we
23 find these in any other context they're the
24 product of intelligence, we infer by standard
25 scientific inference or reasoning that these

1 systems are also the product of intelligence,
2 and we leave it at that.

158 3 Q. Is intelligent design based on any
4 religious beliefs or convictions?

5 A. No. Again, it's looking at the public
6 evidence or the empirical evidence.

159 7 Q. And if you could just summarize the
8 intelligent design argument, I know you
9 have an exhibit to assist you.

10 A. Yes, we'll just go this, we infer design
11 when we see parts that appear to be arranged for
12 a purpose. The strength of the inference is
13 quantitative. The more parts that are arranged,
14 the more intricately they interact, the stronger
15 our confidence is for design. The appearance of
16 design in aspects of biology is overwhelming by
17 the community's own admission. Since nothing
18 other than intelligence cause has been
19 demonstrated to be able to yield such a
20 strong appearance of design, Darwinian claims
21 notwithstanding, the conclusion that design seen
22 in life is real design is rationally justified.

160 23 Q. Does intelligent design make a causative
24 argument for design?

25 A. Again it does. I mean, there's a negative

1 aspect in the sense that for any of these
2 systems that we'll talk about we don't have a
3 Darwinian mechanism to explain them. The
4 positive side is we do know where such systems
5 originate from our own experience of cause and
6 effect.

161 7 Q. The purposeful arrangement of parts?

8 A. The purposeful arrangement of parts in
9 molecular machines that have the appearance of
10 machines that we make that are the product of
11 intelligent design engineers.

162 12 Q. Now, does the book Pandas make this point?

13 A. It talks about, and there's a quote here,
14 the ordering of independent pieces into a
15 coherent whole to accomplish a purpose which
16 is beyond any single component of the system
17 is characteristic of intelligence. So this
18 is kind of a prestatement I think before the
19 coining of the term irreducible complexity.

163 20 Q. And the quote you read was from page 144,
21 is that correct?

22 A. Correct.

164 23 Q. And that's Defendant's Exhibit 220. Sir,
24 is intelligent design science?

25 A. It is. Again just to restate, it's looking

1 at the empirical evidence, the public evidence.

165 2 Q. And from this empirical evidence it makes
3 inferences, is that correct?

4 A. Right, using standard scientific reasoning
5 of cause and effect we see machines that in
6 every aspect look like machines that engineers
7 produce. We don't have a Darwinian mechanism
8 to explain these things in terms of the
9 intermediates. So we can infer that these
10 are the product of intelligence.

166 11 Q. Sir, can you give us an example of design
12 at the molecular level?

13 A. Yeah, I've got a couple of slides, you
14 know, this is I'm sure has been hammered to
15 some degree already, but this is a bacterial
16 flagellum. This is a system that I work on.

17 THE COURT: We've seen that.

18 A. I know.

167 19 Q. You're going to see a little bit more of
20 it, Your Honor.

21 A. I kind of feel like Zsa Zsa's fifth
22 husband, you know? As the old adage goes,
23 you know, I know what to do but I just can't
24 make it exciting. I'll try.

25 THE COURT: Any further questions,

1 Mr. Muise?

2 MR. MUISE: He's doing fine right now,
3 Your Honor.

4 THE COURT: For our last witness we get
5 stand-up. You may proceed.

6 A. All right, this is out of a standard
7 biochemistry textbook that's used for the
8 advanced graduate, or undergraduate and graduate
9 students, Voet and Voet, but it's a cartoon
10 of bacterial flagellum from a grand negative
11 organism, and this is what we refer to as the
12 parts. I mean, we've got a drive shaft here,
13 this is the hook protein, or the U joint, it
14 spins. This is the propeller, or the filament.
15 We've got bushings, we've got a stator and a
16 rotor. This thing self assembles from the
17 inside out in a programmed manner. Most of my
18 research has focused on the genetic programming
19 of when to make these things, and also on the
20 assembly of the filaments. But it's a true
21 rotary engine. The size of is about 45
22 nanometers. So forty-five billionths of a
23 meter in size.

168 24 Q. You specialize your focus and research
25 on the flagellum, is that correct?

1 A. That's correct.

169 2 Q. And you've done experiments on flagellum?

3 A. I have.

170 4 Q. And have written peer reviewed articles
5 about it?

6 A. Yes.

171 7 Q. Now, as your prior testimony intimated
8 there's been a good deal of focus on the
9 bacterial flagellum. I guess we could probably
10 call this the bacterial flagellum trial. Why
11 the focus on this particular organelle?

12 A. Well, I think it's, I mean it's just a
13 logical thing, because of all the molecular
14 machines that we know about in biological
15 systems, we know more about the bacterial
16 flagellum than any. I mean, this was first
17 discovered in E. coli and salmonella, which are
18 really the gold standard for doing molecular
19 genetics, and teasing apart these types of
20 machines.

21 This in terms of organelle development
22 synthesis, we know an incredible amount about
23 it. It's also been a primary model system
24 starting in the early days for signal
25 transduction, a field of biology in terms of

1 how an organism reads its environment and makes
2 appropriate decisions in terms of, you know, in
3 this case directional flow. So it has served us
4 very well in terms of working out simple signal
5 transduction systems which have paid off an
6 astonishing coin as we've applied the same
7 principles of their study to higher organisms.
8 So in essence this is a system that will maker
9 or break, you know, intelligent design, because
10 it's the one we know the most about.

172 11 Q. So it's a system that we have a lot of
12 data available, correct?

13 A. Correct.

173 14 Q. And it's a well defined system?

15 A. It's well defined. I mean, we know all
16 the genes involved, we know a lot about its
17 assembly, but there's still questions about
18 how the motor actually works, some of the
19 biophysics, but other than that I think of
20 any molecular machine this one is the most
21 well understood and most defined.

174 22 Q. Sir, would it be fair to say that this is
23 not just an organelle that intelligent design
24 proponents have randomly selected to use for
25 their arguments?

1 A. No, no, not at all.

175 2 Q. Is it fair to say that if you were going
3 to find support for your arguments or support
4 against your arguments, this would probably be
5 the organelle that you would have to address in
6 the literature?

7 A. Sure.

176 8 Q. Now, Dr. Behe and you just covered some of
9 the components of the bacterial flagellum, and
10 they appeared to be identified or named in using
11 names that we sort of recognize as part of
12 engines and as part of machines. Are those
13 labels that scientists actually apply to these
14 components?

15 A. Right. I mean, again this is out of a
16 textbook, and you know, some may say that well,
17 if you draw something to look like a machine it
18 becomes a machine, but this is a true rotary
19 engine, and by definition it's got to have a
20 rotor and stator and drive shaft and U joint
21 for propulsion. It's an amazing engine I don't
22 think just to me, but, you know, the people,
23 those of us that work on it are fascinated by
24 it.

25 In E. coli these things will rotate at

1 about 17,000 RPM's on average, although there's
2 some marine vibrios where these engines have
3 been blocked at 100,000 RPM's. It's essentially
4 a massless engine, so it can reverse direction
5 in less than a quarter turn of the rotor. So,
6 you know, it's got two gears, forward and
7 reverse, water cooled, battery powered. It's
8 a fascinating system.

177 9 Q. Now, the conclusion that something was
10 designed, does that require knowledge of the
11 designer?

12 A. No. Absolutely not.

178 13 Q. Why not?

14 A. Well, I mean, we can infer design, but the
15 science isn't going to tell us anything about
16 the designer unless it's, you know, signed on
17 one of these components, and we haven't found
18 that yet.

179 19 Q. So is it accurate for people to claim or to
20 represent that intelligent design holds that the
21 designer is God?

22 A. No, absolutely not.

180 23 Q. Has science answered this question, the
24 source of design --

25 A. No.

181 1 Q. -- in your view?

2 A. No.

182 3 Q. Now, we're going to, we'll be returning
4 to the bacterial flagellum a little bit later.
5 I put up here a quote that I believe we heard
6 already once in this trial from Theodosius
7 Dobzhansky, did I pronounce that right?

8 A. Correct, Russian evolutionist.

183 9 Q. It says, "Nothing in biology makes sense
10 outside the light of Evolution." Do you agree
11 with this quote?

12 A. I don't. Not to belittle the importance of
13 evolution, but this hasn't been my experience.

184 14 Q. Why?

15 A. Well, let's go to the next slide, and I've
16 got a couple of quotes that I picked from my
17 expert report. This is from a review by Carl
18 Woese, it was published last year. He talks
19 about this aspect, if could read it, "Molecular
20 biology's success over the last century has come
21 solely from looking at certain ones of the
22 problems biology poses (the gene and the nature
23 of the cell) and looking at them from a purely
24 reductionist point of view," and this is part of
25 Carl's point, you know, he disagrees with

1 reductionism.

2 "It's produced an astounding harvest."

3 So a reductionist approach to biology has
4 been astounding. "The other problems, evolution
5 and the nature of biological form, molecular
6 biology chose to ignore, either failing outright
7 to recognize them or dismissing them as
8 inconsequential as historical accidents,
9 fundamentally inexplicable, and irrelevant to
10 our understanding of biology. Now, this should
11 be cause for pause."

12 So here you have, you know, Carl Woese
13 really saying that there's this period in the
14 last fifty years when molecular biology has kind
15 of reigned that we've ignored the question of
16 evolution, and this is a period I think where
17 we've had the greatest increase in our
18 understanding of biological systems I'd say
19 probably over the whole millennium beforehand.

185 20 Q. And who is Carl Woese?

21 A. He's a professor at the University of
22 Illinois, a prominent evolutionary biologist.
23 I have utmost respect for him.

186 24 Q. He's not an intelligent design advocate?

25 A. No, no.

187 1 Q. And if you'd just note, this is, it's
2 listed here as Defendant's Exhibit 251, if
3 you can just confirm that that's the exhibit
4 that you're referring to, and it should be in
5 your exhibit binder under Tab 5.

6 A. Yes, that's correct.

188 7 Q. And that's the article A New Biology For
8 A New Century?

9 A. Correct.

189 10 Q. I believe you have some additional
11 demonstratives to make this point?

12 A. Yes. The next slide, this is a paper
13 published in Cell in 2000. So Cell I think
14 is most prestigious journal for biologists to
15 publish in. Primary research articles of some
16 length. It won't go into the nature of science.
17 Simon Conway Morris is a paleontologist at
18 Cambridge University. This is the introduction
19 to his paper which is a review titled Evolution:
20 Bringing Molecules Into the Fold. "When
21 discussing organic evolution the only point
22 of agreement seems to be: 'It happened.' Given,
23 therefore, this history and the most recent and
24 spectacular advances in microbiology, it may
25 seem curmudgeonly, if not perverse, to even hint

1 that our understanding of evolutionary processes
2 and mechanisms is incomplete. Yet, this review
3 has exactly that intention."

4 So again this is one of the most prominent
5 paleontologists, worked on the Burgess shale,
6 Cambrian explosion, remarking that molecular
7 biology had spectacular advances and, you know,
8 I think with this knowledge, and going back and
9 addressing fundamental questions in terms of
10 evolution is justified. When you consider that
11 statement, you know, the only consensus seems to
12 be that it happened. Beyond that, you know,
13 mechanisms, our understanding of mechanisms,
14 processes, are incomplete.

190 15 Q. In this article, I believe it's marked as
16 Defendant's Exhibit 255, and it's Tab 9 in your
17 exhibit binder, can you verify that for us, sir?

18 A. That's correct.

191 19 Q. I'll move to the next exhibit, which is a
20 paper by Lenski, et al., and I believe it's
21 marked as Defendant's Exhibit 252, which will
22 be under Tab 6 in the exhibit binder that you
23 have. Are you familiar with this paper and its
24 findings?

25 A. I am.

192 1 Q. What does this paper purport to conclude?

2 A. Well, if you go to -- well, this is a paper
3 addressing evolutionary origin of complex
4 features, really looking at the infusion of new
5 genetic information in organisms and trying to
6 look at, you know, the mechanism of that.

193 7 Q. Now, Professor Pennock is one of the
8 co-authors of this paper, is that correct?

9 A. That's correct.

194 10 Q. And he's an expert who testified for
11 plaintiffs, and he appeared rather giddy about
12 the results that they achieved in this paper.
13 Do you share his enthusiasm?

14 A. I like the paper, and I like the quotes.
15 The thing that I hesitate when I bring this up
16 first you all is, and I'll show you in the next
17 slide, but this is out of Richard Lenski's lab,
18 and they've been doing experiments over the last
19 twenty years, long-term evolutionary of E. coli
20 and hemostats or fermenters, looking at changes
21 over, up to 40,000 generations, and --

195 22 Q. These are on living --

23 A. Living, on escherichia coli, again our
24 standard model for these type of studies, and
25 this in less than 20,000 generations they see

1 the infusion of new information, but this is
2 a mathematical model. These are virtual
3 organisms. So I think there's a limitation,
4 which I mentioned in my expert report.

196 5 Q. How do the results of these digital
6 organisms compare with Lenski's results
7 with living organisms?

8 A. Well, again you see change at a faster
9 pace than the real experiment, so I think it's
10 somewhat backward, I'm not a computer scientist,
11 I don't understand the software, so there's
12 limitation there as well and I'm the first to
13 admit it, but as I read this paper it seems like
14 there's a targeted logical program that these
15 organisms can adapt to by mutation, much like
16 viruses in your computer systems. So that's
17 what they're measuring this change to.

197 18 Q. You picked a particular quote from this
19 paper I guess to emphasize your points regarding
20 that quote from Dobzhansky, is that correct, on
21 this next line?

22 A. Right. That, and also the fact that
23 students are often confronted with the absolute
24 statement that Darwinism is fact, or if not
25 evolution is fact and, you know, this is from

1 the introduction of this paper that was, you
2 know, in Nature. From the outset Darwin
3 realized that organs of extreme perfection
4 and complication, such as the eye, posed a
5 difficulty to his theory." I mean, this is the
6 argument of design.

7 "Such features are much too complex to
8 appear de novo, and he reasoned that they must
9 evolve by incremental transitions through many
10 intermediate states, sometimes undergoing
11 changes in function." This is variation in
12 natural selection. "Now, there exists
13 substantial evidence concerning the evolution
14 of complex features that supports Darwin's
15 general model. Nonetheless, it's difficult to
16 provide a complete account of the origin of any
17 complex feature, owing to the extinction of
18 intermediate forms, imperfection of the fossil
19 record, and incomplete knowledge of the genetic
20 and developmental mechanisms that produced such
21 features."

22 So in summary, if you go to the next slide,
23 there's this admission in this paper, in Simon
24 Conway Morris's paper, Woese addresses these
25 facts as well, that we lack intermediate

1 structures, we lack fossils, we don't have an
2 adequate knowledge of how natural selection can
3 introduce novel genetic information. That's
4 the point of this paper with virtual organisms
5 and mathematical and computer simulation, and
6 then from my own experience going back to
7 Dobzhansky's quote, "Nothing in biology makes
8 sense outside the light of information," I have
9 my own experience as well that I would like
10 to --

198 11 Q. Please tell us your experience with regard
12 to that quote that nothing makes sense in
13 biology in light of evolution.

14 A. In my entire academic training as an
15 undergraduate or graduate student or as a
16 post-doc at Purdue and Princeton University,
17 I never once took a formal course in evolution.
18 In fact, when I requested it as a graduate
19 student, you know, to include it on my graduate
20 student study plan, it was refused by my
21 committee with a, you know, you don't have time
22 to do it, it's not necessary.

23 So that has been my experience as a
24 biologist and a practicing, you know,
25 experimental biologist, I've never been

1 required to take a single course in evolution.
2 My exposure formally was in my undergraduate 100
3 and 200 level introductory biology classes were
4 we got basic evolution, you know, Haeckel's
5 embryos, peppered moths, founder effect. So
6 the basis tenets were there, but in terms of
7 really looking at this in detail, I haven't.

8 Now, this isn't unique to me. When
9 I, in my department of molecular biology,
10 microbiology, and biochemistry there's only one
11 other faculty member, although we've had three
12 or four that have joined the department in the
13 last year, so I can't say that absolutely, but
14 since my tenure there in 1989 one person has
15 took an actual course in evolution as a graduate
16 student. So I find this amazing that, you know,
17 we're doing hard-core molecular biology, and
18 this was never part of our training.

19 I'm the only person and one other faculty
20 member that have read Darwin, which again, you
21 know, I think is a problem. I would like to
22 correct that. I think it should be required
23 that all students in biology read Darwin's
24 Origin of the Species and be required to take
25 a rigorous course at some level, preferably

1 early on in their undergraduate degree careers,
2 in evolution, because, you know, I find this
3 ironic situation that although I've never been
4 required to take this material, you know, in my
5 training, the point now where I'm questioning
6 its importance in my discipline, you know, has
7 been quite an amazing experience.

199 8 Q. How so has it been quite an amazing
9 experience?

10 A. Well, it's difficult to say. I mean,
11 it's almost like you're a heretic in the camp.
12 I mean, I'll put it like that.

200 13 Q. So to sort of summarize through some of
14 these quotes from prominent evolutionary
15 biologists and from your own experience, we
16 had the greatest advances in biology perhaps
17 in this last half century, and it's been
18 primarily at the molecular level, is that fair
19 to say?

20 A. Correct. I mean, molecular biology is
21 focusing primarily on E. coli first and then
22 extrapolating what we learn there to more
23 difficult systems, eukaryotic systems, yeah,
24 it's been an incredible period.

201 25 Q. Yet evolution has been practically

1 inconsequential in the development of this
2 information that we've gathered?

3 A. Carl Woese states that in his paper. I
4 mean, some people considered it inconsequential.
5 It was ignored, a historical accident.

6 MR. MUISE: Your Honor, I'm going to start
7 moving into another area. I don't know if this
8 may be a time to break.

9 THE COURT: Yeah, why don't we, I think that
10 makes good sense. Why don't we break here for
11 about twenty minutes, and we'll resume with the
12 witness's testimony after that intermission,
13 and we will return after the break. Thank you.

14 (Recess taken at 2:14 p.m. Proceedings
15 resumed at 2:36 p.m.)

16 THE COURT: Be seated, please. You may
17 resume.

18 BY MR. MUISE:

202 19 Q. Thank you, Your Honor. Dr. Minnich, when
20 you were defining intelligent design earlier in
21 your testimony you noted the "deep complexity
22 and clearly evident design in organisms." Do
23 other scientists recognize this complexity in
24 evidence of design?

25 A. Yes. All biologists see design in nature,

1 and this is really part of this central
2 question, is it real design or apparent design,
3 and how do we differentiate between the two.
4 This is a cover of Cell again, this is our
5 premier journal. From a review issue, once a
6 year they run a review issue, this is from 1999
7 I believe.

203 8 Q. I believe it's 1998.

9 A. '98, okay, I can't remember, but
10 macromolecular machines, this dealt with the
11 machines of life, and I think the cover really
12 sums it up. Across the landscape of biological
13 systems we find these incredible macromolecular
14 machines.

204 15 Q. And they dedicated an entire issue?

16 A. Exactly. The entire issue is looking at
17 specific machines in the cell that we knew a
18 lot about.

205 19 Q. And just I guess for purposes of the record
20 this cover can also be found as Exhibit 203-C,
21 Charlie. I believe another slide from an
22 article that appeared in there in this
23 particular journal, this issue, from Bruce
24 Alberts, is that correct?

25 A. Correct. Bruce Alberts at the time was

1 National Academy of Science president. He's
2 an evolutionist, so you know, I don't want to
3 misinterpret his position on any of this, but
4 it's an interesting article titled The Cell as a
5 Collection of Protein Machines: Preparing the
6 Next Generation of Molecular Biologists. Some
7 of the things that he notes, the complexity of
8 the cell's macromolecular machines was not
9 anticipated."

10 In the introduction of this article he
11 states as a graduate student in the 1960's they
12 looked at the, you know, cells that they were
13 working on, E. coli at the time, as really a bag
14 of enzymes operating on the second order of
15 kinetics, or diffusion kinetics, "Our current
16 view of the cell is vastly different." In fact,
17 he says, "We've always underestimated the cell
18 in this review." More complex than the view of
19 the cell when Dr. Alberts was a graduate
20 student, okay, so I covered that.

21 Dr. Alberts advocates in this article
22 incorporating the principles of design
23 engineering into biology curricula for this
24 next generation of molecular biologists
25 as a means to dissect the interactions of

1 macromolecular machines now identified in
2 even the simplest cells. The point being that
3 for us to get to the next level of understanding
4 at the cellular and subcellular level, how all
5 these molecular machines not only function
6 independently in and of themselves, but how
7 they're coordinately regulated as a consortium
8 machines to carry out the cell's duty will be
9 the job more of the design engineer or a systems
10 analyst. These are true factories.

11 So I find it incredible. In fact, in
12 the acknowledgments he acknowledges Jonathan
13 Albert, I don't know the relationship, for the
14 information in terms of how design engineers
15 approach these types of problems. We're going
16 to need this, you know, the age of cloning and
17 sequencing is over, to get to the next step.
18 We're going to incorporate design engineering.

206 19 Q. And again this article is marked as
20 Defendant's Exhibit 253, and I just want to
21 verify if you look under Tab, I believe it's Tab
22 7 in your exhibit binder if you would, in the
23 black binder, if you'd verify this as the
24 article you're referring to?

25 A. Correct.

207 1 Q. I believe you have another section from
2 this issue of the journal that you want to use
3 to emphasize your points?

4 A. Right. Can I just read one quote out
5 of this article, because again it's important
6 to understand that Bruce Alberts is an
7 evolutionist. In fact, he's co-author of the
8 book on how to teach evolution at the secondary
9 level, published by the National Academy. But
10 on the first page of this article at the bottom,
11 why do we call --

208 12 Q. I'm sorry, you're referring to Exhibit 253?

13 A. Correct, 253, on the first page. "Why do
14 we call the large protein assemblies that
15 underlie cell function protein machines?
16 Precisely because like the machines invented by
17 humans to deal efficiently with the macroscopic
18 world, these protein assemblies contain highly
19 coordinated moving parts. Within each protein
20 assembly intermolecular collisions are not only
21 restricted to a small set of possibilities, but
22 retain, reaction C depends on reaction B, which
23 in turn depends on reaction A, just as it would
24 in the machine of our common experience." So
25 emphasizing that this is almost a definition of

1 purposely ordered parts that you find in Pandas
2 and People or it might be a used definition of
3 irreducible complexity, highly ordered parts
4 that perform a function.

209 5 Q. And you have another demonstrative aid?

6 A. Right.

210 7 Q. I guess another excerpt from this journal
8 itself, right?

9 A. Correct. I think this is what I just read,
10 isn't it? Oh, no, this is actually from the
11 table of contents for this issue. "Again, like
12 machines invented by humans to deal efficiently
13 with the macroscopic world, protein assemblies
14 contain highly coordinated moving parts.
15 Reviewed in this issue of cell are the protein
16 machines that control replication,
17 transcription, splicing, nucleocytoplasmic
18 transport, protein synthesis, protein assembly,
19 protein degradation, and protein translocation,
20 the machines that underlie the workings of all
21 living things."

22 Across the landscape again these are the
23 machines that are performing every function in
24 the cell. Highly sophisticated machines, many
25 of which when we dissect them have all the

1 hallmarks of machines that design engineers have
2 made in our macro world. So again the
3 inference, you know, we have the question the
4 appearance of design, is it real or just
5 apparent? We don't have a Darwinian mechanism
6 to explain the appearance of these in a
7 step-wise manner. At the same time we do know
8 from our common experience, you know, cause and
9 effect in the world, that when we find these
10 types of machines, they're the product of
11 intelligence, and these surpass anything that
12 yet, you know, that we can make ourselves.
13 It's an inference, it's a logical inference.

211 14 Q. I believe we have another slide with our
15 friend, the bacterial flagellum.

16 A. Right. Again this is my machine, and David
17 DeRosier at Brandeis University has done an
18 incredible amount of work on this. In a review
19 article in Cell in 1998 he wrote, "More so than
20 other motors, the flagellum resembles a machine
21 designed by a human," all right? So there's
22 question of design. As biologists we all
23 recognize it. It's a true rotary engine.

212 24 Q. Is that an understatement by Dr. DeRosier?

25 A. Yeah, I guess you would have to say,

1 because we have yet engineered a machine that
2 can self assemble and function, you know,
3 actually have its own software written that
4 can call up and decide when and how many of
5 these to make, where to put them, etc. So
6 it's incredible, I mean, when you look at the
7 parameters of this machine.

213 8 Q. And this, and again for reference purposes
9 this is from Defendant's Exhibit 274, and if you
10 can just look in your exhibit binder, I believe
11 it's Tab 11, is this the article from which
12 you're quoting from?

13 A. Correct. That's correct.

214 14 Q. Now, you indicated these living organelles
15 are described as machines by you and by these
16 scientists. Are they in fact machines?

17 A. They are. I mean, again they have all the
18 components of a rotary engine. Rotor, stator,
19 U joints, bushings, drive shaft, that's how
20 they're described, and by definition a rotary
21 engine has to have these components, regardless
22 of the scale. I want to point out, too, you
23 know, just for the record that we didn't know
24 these things existed twenty or thirty years ago
25 this was the surprise.

1 Again emphasizing what Bruce Alberts says,
2 our conception of the cell has changed radically
3 in the last twenty to thirty years. In terms
4 of how we view the cell he says that we've
5 always underestimated it, I have another quote
6 here by some colleagues, but I think it's
7 perfectly legitimate to go back and ask is
8 natural selection mutation sufficient to prove
9 or to build this type of sophisticated
10 machinery.

215 11 Q. But the bacterial flagellum isn't the only
12 machine in a cell, correct?

13 A. No, no.

216 14 Q. And I believe you have some additional
15 exhibits to point out some other machines?

16 A. Yeah, I've included another rotary engine,
17 the ATPase we find in prokaryotic and eukaryotic
18 cells. This is a description of the torque
19 generated in the transfer of this energy to ATP
20 synthesis. ATP is the energy currency of a
21 cell, is generated by oxidation reduction
22 reactions in the cell, and essentially what you
23 do is you push protons across a membrane, much
24 like you would collect water behind a dam, and
25 then you bleed through ATPase, which acts as a

1 turbine. For every third of a turn, or 120
2 degree turn of this rotor, you generate
3 essentially one adenine triphosphate molecule.

4 The point being here I think is this group
5 conceded all, makes this point in their article
6 in Cell that if one ATP consumed for 120 degrees
7 is one of, one may anticipate from the make of
8 this motor the efficiency of our ATPase is
9 nearly 100 percent, far superior to a Honda V-6.
10 This is a direct quote out of this article. So
11 it's approaching 100 percent efficiency in these
12 machines that are being produced by the random
13 events and selection of Darwinian mechanism.

217 14 Q. I believe you have a schematic here of ATP?

15 A. Yes, this is a cartoon, again it's a rotary
16 engine like the flagellar, it's a much smaller
17 scale, but you can see that you've got a stator
18 here and a rotor with a rem ATP is generated as
19 this turbine turns around up here.

218 20 Q. Are engineers studying these machines?

21 A. Right, I think that's -- the fascinating
22 thing to me, and this is in part why I
23 participated in this conference in Rhodes in
24 biomimetics is that engineers and architects
25 have recognized that biology, systems in biology

1 have solved some pretty complex problems, and
2 when you consider nanotechnology, the
3 application of this, computer applications,
4 pharmaceutical applications, engineers are
5 coming to biologists to learn about these
6 systems and how they may, you know, practically
7 apply them. So when you consider the bacterial
8 flagellum, the speed at which it rotates, the
9 fact that it can, you know, reverse direction in
10 less than a turn, I mean that's like any time
11 you have a machine that can stop and start, it's
12 the equivalent in machine language of a one and
13 zero. I mean, you can have that application in
14 terms of designing computers that are
15 biologically based.

219 16 Q. Have you been asked to give presentations
17 to engineers about these molecular machines?

18 A. I have in my university, the University
19 of Idaho, I've given one talk to the physics
20 department just based on the bacterial flagellum
21 as a nanomachine. They're interested in the
22 fluid dynamics of the system and how it operates
23 at this scale, and also to, I believe it was a
24 mechanical engineering department.

220 25 Q. And I believe you have a few other examples

1 of design in nature?

2 A. Yeah. So the other thing that I think
3 caught us by surprise is the sophistication
4 of the information storage system of the cell.
5 DNA and RNA are really information systems
6 that store digital information just like our
7 computers do. This is out of a textbook, this
8 is a genetic code that was solved in the 1960's
9 by Caron at Harvard and Nirenberg at the NIH,
10 and essentially you have as we all know from
11 basic biology there are four nucleotides that
12 make up genetic information, and there are
13 twenty amino acids. It's combination of three
14 of these letters that determine each amino acid
15 if this translation is occurring between
16 nucleotide language to protein language.

17 So for instance U in the first position,
18 we call this the five prime positions, the
19 center position U, and U in the third position
20 codes for phenylalamine. UUC also codes for
21 phenylalamine. With four digits there are 64
22 combination. So we have 64 three letter codons.
23 Now, when this was determined in the 60's, so
24 this is really the Rosetta Stone of genetics,
25 when this was determined in the 60's there was

1 an intuitive recognition that there seemed to
2 be a bias in the code for amino acids that if
3 you had a point mutation, for instance if you
4 have UUU and you changed this last U to a C,
5 you get the same amino acids.

6 So there's redundancy. UCU or UCC, UCA,
7 UCG all code for a series. You either get the
8 same amino acid or a similar amino acid in
9 terms of its chemical properties. So that was
10 intuitively obvious. Now, if this is a product
11 of arbitrary chance and necessity, to quote
12 Minot, then there's no reason that this code
13 is chosen over any other. Francis Crick
14 referred to this as a frozen accident. Carl
15 Woese in his paper "Owed to the Code" states
16 that the genetic code has not evolved.

17 Now, with computer analysis we can actually
18 look at all of the random codes that can be
19 generated. There are millions of codes that
20 can be generated with the parameters of twenty
21 amino acids and four nucleotide bases, and ask
22 is there a bias, is there a better code to
23 minimize the effect of point mutations, because
24 that's really what we're seeing in this code,
25 and it turns that the natural code according to

1 this author Hays when this has been analyzed
2 against millions of other arbitrary codes is
3 optimized to minimize the effects of point
4 mutations, okay, the very thing required to
5 drive evolution.

6 We have a code that from the get go is
7 optimized to minimize the effects of point
8 mutation. Now, that to me, and my colleagues,
9 too, when we've discussed this causes them to
10 pause. I mean, people just stop and get
11 reflective. That to me has a signature of
12 design on it, okay, that you have a, this is
13 a sophisticated, this is the most sophisticated
14 information storage system that we know of.
15 It's true digital code we've got, it codes for
16 algorithms.

17 Now we're talking about the cell working on
18 fuzzy logic, which is non-linear, which is much
19 more complicated than we considered in the past,
20 and if this is a product of undirected chance
21 and necessity, I find that difficult, you know,
22 that nothing that Microsoft and Bill Gates's
23 engineers yet have come close to producing an
24 information storage system like this. That's
25 what we're talking about in terms of design and

1 looking back. We didn't know about this system
2 fifty years ago I mean, when the code was broken
3 in the 60's. Certainly Darwin didn't know about
4 it.

5 So you have this most sophisticated
6 information storage system coupled with
7 macromolecular machines that are also highly
8 sophisticated, with ordered parts that we by
9 definition call are irreducibly complex, it's
10 appropriate to go back and ask is a Darwinian
11 mechanism sufficient to account for the
12 appearance of these.

221 13 Q. You said that the DNA has been shown to
14 resist point mutations, is that correct?

15 A. It's not that it resists it, but if
16 you have a point mutation, which is common
17 either in replication or just exposure to the
18 environment, perhaps mutagens or UV, light that
19 you can get a mutation in one of these codons,
20 you know, to convert a U to a C, or what we call
21 a transition or a transversion mutation, and
22 often you'll get either the same amino acid or
23 an amino acid that's related in terms of its
24 chemical properties so that you don't disruption
25 of that protein that's produced with that

1 mutational event. Now, it doesn't eliminate
2 it completely, but there is, we recognize that
3 there is this bias. This is optimized to negate
4 the effect of point mutation.

222 5 Q. So it's optimized to negate point mutations
6 which are necessary for that selection to
7 function?

8 A. Right. That's one of the driving forces
9 obviously of evolution.

223 10 Q. Dr. Minnich, why isn't this just the
11 argument from incredulity?

12 A. I mean, that's -- Dawkins makes that
13 argument that because I can't imagine a
14 mechanism that would produce this that I
15 suffer from incredulity, and I'm, darn it,
16 you know, we are trained to be skeptics. We
17 are trained to look at things through, you know,
18 a very narrow lens. We're to be our own worst
19 critics, and it seems like in any other practice
20 of science that's how we operate, except when it
21 comes to an explanation of the origin of these
22 systems, and then we're accused of being, you
23 know, suffering from incredulity because we
24 can't imagine how these came about.

25 We don't have the intermediates. Again

1 for any biochemical pathway we don't have the
2 phylogenetic history for any biochemical pathway
3 or subcellular organelle. Yet as a scientist I
4 am supposed to accept this without blinking that
5 this is a product of a Darwinian mechanism, and
6 I'm sorry, these are highly sophisticated
7 systems, and I know from experience that when
8 you see a machine, a rotary engine, in any other
9 contest, you would assume that there's an
10 engineer around, and those are the arguments
11 that we're making.

224 12 Q. I believe you have another example, you
13 described the sliding clamp. Could you describe
14 this?

15 A. This is DNA polymerase on the right,
16 so this is the copying mechanism for DNA
17 replication. What I find interesting, actually
18 this was a paper that was given to me by a
19 colleague who we disagree with in terms, but
20 he thought I'd be interested in it. The clamp
21 protein here, which forms this donut around this
22 double helix of DNA, in eukaryotic organisms or
23 higher organisms there's a dimer. We call it in
24 yeast PCNA protein.

25 In *E. coli* we also have a clamp protein,

1 this is a prokaryotic, a more primitive
2 organism, it's a trimer. It's a beta subunit
3 of E. coli polymerase. Now, if we compare the
4 protein sequences that form this structure
5 between E. coli and yeast, we wouldn't pick
6 them up as being similar in a computer search.
7 Now, this is, all organisms are required to
8 replicate their DNA. You would think that
9 this would be a highly conserved process by
10 definition if prokaryotics eventually evolved
11 eukaryotes from some common ancestor, but what
12 we find is a protein that has almost an exact
13 superimposable structure, one on the other,
14 forming the same function, but completely
15 different amino acid sequences.

16 This is a remarkable example of
17 convergence, and there are many examples of this
18 coming out now at the molecular, and as we'll
19 talk about Simon Conway Morris says even at the
20 organismal level. We can't, at present we don't
21 understand the properties of protein folding,
22 so we couldn't make a protein to form this
23 structure as a base for the assembly of the
24 other components of DNA polymerase. Yet we find
25 in nature that this has happened twice for the

1 same function, the same structure, but a
2 different amino acid sequence. I mean, that's
3 an incredible finding.

225 4 Q. Is that what you mean by convergence?

5 A. Convergent, right.

226 6 Q. I believe you have another example, a gated
7 portal. Could you explain what this is?

8 A. The gated portal, so this is looking from
9 the nucleus of a eukaryotic organism, and I
10 don't think it shows up with that well on this
11 slide, but this is a portal, or actually a gate,
12 so you have to have traffic material from the
13 nucleus to the outside, from the outside back
14 into the nucleus.

15 These are proteins of nucleic acids, and we
16 have these gate systems or turnstiles, and we
17 find that there's a very sophisticated postal
18 system in the cell that components of the cell
19 will have, you know, a molecular zip coding that
20 will direct them, first of all allow them to go
21 through this portal, and then afterwards direct
22 them to their location wherever they're required
23 in the cell. That whole postal system of zip
24 coding, how, you know, a protein made of a
25 cytoplasm is directed to the membrane or to

1 endoplasmic verticulum is an incredible area
2 of research and interest as well, and --

227 3 Q. So this is an informational transport
4 system, is that --

5 A. Correct, correct. So there's, you know,
6 this is a cross section of that. So here would
7 be the nuclear membrane and the components that
8 have been defined by mutational analysis that
9 dictate what can come through or what can go
10 back through the nucleus. So proteins
11 synthesized in the cytoplasm and in the ruthear
12 have to come back through if they're regulatory
13 proteins and interact with DNA. So there's a
14 very important regulatory system in terms of
15 recognizing these proteins and directing them
16 to their locales.

228 17 Q. Dr. Minnich, it appears from your testimony
18 and sometimes from the prior quotes you have
19 from other scientists that our understanding
20 of the complexity of life has, especially at
21 the molecular level, has probably advanced
22 exponentially in the last half century. Is
23 that fair to say?

24 A. Oh, for sure. For sure.

229 25 Q. Dr. Alberts acknowledged that in the

1 article that you cited to, is that correct?

2 A. Right.

230 3 Q. Are there other scientists as well that
4 make that observation?

5 A. Right, I have a quote from the journal
6 *Bacteriology*, you know, from Richard Losick
7 at Harvard and Lucy Shapiro who works on an
8 organism that I used to work with. I know Lucy,
9 but --

231 10 Q. Where is she now?

11 A. She's at Stanford. She's department chair
12 in developmental biology at Stanford, *Changing*
13 *Views on the Nature of the Bacterial Cell* from
14 *Biochemistry to Cytology*. She would be a
15 contemporary of Bruce Alberts having gone
16 through I think graduate training in the 60's.
17 So these people that are kind of reaching
18 retirement age are starting to reflect back on
19 their careers I think during the most fruitful
20 research period in the history of biology, and
21 these are not uncommon statements.

22 So let me read what these two individuals
23 say, "How profoundly our view of the bacterial
24 cell has changed since we first started our
25 lifelong fascination with life's smallest

1 creatures." They're both microbiologists.

2 "Who would have imagined that bacteria have
3 proteins that assemble into rings, that cluster
4 at the poles of cells, that localize delocalize
5 as a function of the cell cycle, or that bounce
6 off the ends of the cell with a periodicity of
7 tens of seconds.

8 "Who would have suspected that the origins
9 replication move to the poles of cells, that the
10 machinery for replicating DNA is stationary, and
11 that it is the chromosome that moves through the
12 chromosome duplicating factory, or that plasmids
13 would jump from the cell center or the cell
14 quarter points following their replication."
15 The point I just want to make is that our view
16 of the cell, even the simplest cell, has changed
17 profoundly, and we are, there are scientists
18 that have come through are, you know, awe struck
19 in terms of the beauty and complexity of the
20 systems that we're studying.

232 21 Q. How is this relevant or implicate
22 intelligent design?

23 A. Again the molecular machines that we find
24 that I work on were not anticipated, they
25 weren't predicted. They have the appearance

1 of machines that engineers make. I'm going to
2 hammer this point home, but I think it's
3 critical to understand that we don't have a
4 Darwinian mechanism for the step-by-step
5 intermediates to get there or build these
6 machines, and we know from definitional work
7 on these machines that they're irreducibly
8 complex, and we'll go over that in the next
9 section. But again you take away one component,
10 you trash the machine. That's how you study
11 them. That's how we figure out what the parts
12 are in each individual system that, you know, is
13 our pleasure to work on.

233 14 Q. I believe we have one last quote which I
15 believe we've seen already in this trial.

16 A. Right, from Mr. Dawkins and The Blind
17 Watchmaker. "Biology is the study of
18 complicated things that give the appearance
19 of having been designed for a purpose." As
20 biologists we all see the design, and you can
21 be like Richard Dawkins and argue that it's
22 only apparent design. If there is a natural
23 mechanism, a Darwinian mechanism, a variation
24 on the mutation that can produce it, I'm more
25 reserved, I guess more conservative and say,

1 you know, to me it's real design, and it's a
2 scientific argument.

234 3 Q. And I believe you've prepared a summary?

4 A. Okay. Our view of the cell is vastly
5 different from when Darwin's theory was first
6 proposed, let alone our view over forty years
7 ago. The cell is now recognized as being orders
8 of magnitude more complex and sophisticated than
9 Darwin envisaged. While our understanding of
10 the complexity of the cell has increased by
11 orders of magnitude, the mechanism to generate
12 the complexity, mutation and natural selection,
13 has remained constant, although there's some new
14 avenues of research that I find very exciting in
15 this last part. It's reasonable to revisit the
16 question, again it's reasonable to revisit the
17 question as to whether natural selection is
18 sufficiently up to the task of design
19 engineering this recognized sophistication we
20 find in even the simplest of cells.

235 21 Q. Do other scientists who are not intelligent
22 design advocates recognize the lack of an
23 adequate Darwinian explanation for this
24 complexity in evident design?

25 A. I have a quote from Carl Woese in that

1 paper that was cited earlier alluding to this
2 fact, and I don't think I'm taking this out of
3 context. "The creation of the enormous amount
4 of and degree of novelty needed to bring forth
5 modern cells is by no means a matter of waving
6 the usual wand of variation and selection. What
7 was there, what proteins were there to vary in
8 the beginning? Did all proteins evolve from one
9 aboriginal protein to begin with? Hardly
10 likely.

11 "Evolution's rule, to which there are
12 fortunately a few exceptions," which he doesn't
13 give, "is that you can't get there from here.
14 Our experience with variation and selection in
15 the modern context does not begin to prepare us
16 for understanding what happened when cellular
17 evolution was in its very early rough and tumble
18 phases of spewing forth novelty." All right, so
19 Carl Woese is saying essentially in these early
20 stages of evolution, whatever parameters were at
21 work are not present today, which again, I mean,
22 bears on the question of doing the science.

23 I mean, there were conditions by admission
24 perhaps that we can't reproduce. You know,
25 we've got to recognize that, and I think it's

1 important for students to recognize that, but
2 maybe the important thing here, evolution's rule
3 to which there are fortunately a few exceptions
4 is you can't get there from here. It means we
5 can't, we don't have the intermediates to
6 account from how we got from the simple to the
7 complex.

236 8 Q. And this article you're quoting from, if
9 you can again refer to your exhibit binder,
10 Defendant's Exhibit 251, and it should be I
11 believe at Tab 5, is that the article you're
12 referring to?

13 A. I'll check. That's correct.

237 14 Q. I just need to backtrack because I don't
15 believe we identified the exhibit number for
16 the article from Losick and Shapiro that you
17 referred to previously, and I believe it's at
18 Defendant's Exhibit 257, which would be at Tab
19 10. Is that the article you're referring to by
20 Losick and Shapiro?

21 A. Correct.

238 22 Q. Now, Carl Woese is not an intelligent
23 design advocate, is that correct?

24 A. Absolutely not. I mean, he's a well known
25 and like I said respected evolutionary biologist

1 at the University of Illinois.

239 2 Q. Now, we've been talking about Darwin's
3 theory of evolution. What's the common
4 understanding of Darwin's theory? I should
5 say his principal contribution.

6 A. His principal contribution was the
7 mechanism to account for the variation that
8 we see. So natural selection coupled with
9 variation, which from a neo-Darwinian
10 perspective once we understood genetic
11 information was that mutation, natural selection
12 over time.

240 13 Q. We're talking about the mechanism of
14 evolution?

15 A. Yes.

241 16 Q. Is Darwin's theory of evolution a fact?

17 A. In terms can we demonstrate mutation and
18 selection? Yes. In terms of extrapolating that
19 to larger systems or going from, you know, the
20 evolution of some of these machines that we're
21 talking about, we don't have the evidence.

242 22 Q. Are there gaps and problems with the
23 Darwinian theory of evolution?

24 A. There are.

243 25 Q. Is there a principal contention that you

1 have for the ability of this mechanism of
2 natural selection to explain the origin of
3 life that concerns intelligent design?

4 A. Right, when you look at the origin of life
5 problem, yeah, I mean, you know, we don't, we
6 can't reproduce it. It's a lot of speculation.

244 7 Q. Let me perhaps rephrase that question
8 because it wasn't as clear as I wanted it to
9 be. Is there a principal contention you have
10 with the explanatory power of the theory of
11 evolution that is particularly relevant for
12 intelligent design?

13 A. I'm not quite sure what you're getting at,
14 and other than the fact that we've got to
15 explain, you know, these machines which I
16 say by definition are irreducibly complex.

245 17 Q. Can natural selection account for the
18 origin of these complex molecular machines?

19 A. Not at present. Again, we don't have the
20 mechanism. I think that natural selection can
21 preserve them, and this is in part I think where
22 we may, you know, if I could look at in a
23 crystal ball and see a melding of these two
24 ideas. Natural selection is definitely a
25 preservative. The question is whether or not

1 it's generative and if it can produce these
2 novel structures de novo, but certainly once
3 these structures are around it has a
4 preservative effect, which is very, very,
5 very important in our study of biology.

246 6 Q. Well, can natural selection account for the
7 information storage systems required for the
8 production of these molecular machines?

9 A. No. No. We have no understanding in terms
10 of how nucleic acid information systems evolved,
11 and in fact in our chemical experiments, looking
12 at primordial conditions we can't get cytosine
13 in all of the methods that have been tested to
14 date.

247 15 Q. How about do we have a phylogenetic history
16 of the single biochemical pathway for things
17 such as the flagella?

18 A. No. Again I think I stated this that, you
19 know, Jim Shapiro at the University of Chicago,
20 Harold, a retired microbiologist at Colorado
21 State, says we don't have a single phylogenetic
22 history of a biochemical pathway or a
23 subcellular organelle. A lot of conjecture,
24 wishful thinking I think to paraphrase their
25 view.

248 1 Q. And who was that view that you were just
2 paraphrasing?

3 A. Harold is a microbiologist, although
4 Shapiro has made similar statements. Jim
5 Shapiro in an article that I just read last
6 week, a fascinating article, said there's no
7 contrivance of man that comes close to the
8 simplest cell or one of the subcellular
9 organelles.

249 10 Q. Now, the theory of evolution, particularly
11 natural selection we've been talking about here,
12 has it been able to explain the existence of a
13 genetic code?

14 A. No.

250 15 Q. Has it been able to explain the
16 transcription of DNA?

17 A. No.

251 18 Q. Has it been able to explain the translation
19 of M-RNA?

20 A. No.

252 21 Q. Has been it been able to explain the
22 structure and function of the ribosome?

23 A. No.

253 24 Q. Can it explain the existence of motility
25 organelles such as the bacterial flagellum?

1 A. No.

254 2 Q. Can it explain the development of the
3 pathways for the construction of organelles
4 such as the flagellum?

5 A. No. Like I said, we have to phylogenetic
6 history. I've worked on the bacterial flagellum
7 for years and there's to my knowledge not a
8 paper that can tell me, you know, the
9 evolutionary assembly of this by a step-wise
10 mutation selection program, and we may never
11 know it. That's the problem.

255 12 Q. Is it fair to say that under this
13 relatively broad category of difficulties
14 that we just went through lies much of the
15 structure and the development of life?

16 A. Oh, for sure.

256 17 Q. And does this then cause you to question
18 whether a Darwinian framework is the proper way
19 to approach such questions?

20 A. That's why I'm testifying here. I mean
21 it's because of the scientific constraints I
22 see in Darwinian explanation.

257 23 Q. Some of the plaintiffs' experts have
24 described intelligent design as a science
25 stopper. Would you agree with that?

1 A. Absolutely not. I mean, turn it around.
2 If you just say, you know, like Woese, wave a
3 magic wand of variation and selection, where
4 does that get you? You know, I think from my
5 own personal perspective, having something
6 designed implies that there's purpose and, you
7 know, I can start teasing apart that purpose
8 and apply that in different ways, like a design
9 engineer or a systems analyst would approaching
10 the machine where you don't have the blueprints,
11 you don't have the owner's manual, and that's
12 the beauty of it.

258 13 Q. So you're a working scientist, I mean you
14 kind of roll up your sleeves and go into
15 laboratories and conduct experiments quite
16 regularly?

17 A. Yeah. That's my passion.

259 18 Q. Do you know employ principles and concepts
19 from intelligent design in your work?

20 A. I do.

260 21 Q. And I'd like for you to explain that
22 further. I know you're prepared several
23 slides to do that.

24 A. Okay, this is just a reiteration in terms
25 of how we function in the laboratory during the

1 last half century, we've gained a greater
2 understand of biology at the molecular level
3 than the entire history of efforts in the
4 proceeding millennia, and I don't think that's
5 an overstatement. The vast inroads we have made
6 in our understanding of the cell came by
7 techniques essential to a design engineer.

261 8 Q. If you can read on from "our understanding
9 of the cell"?

10 A. All right. I lost my place, let's see.
11 Came by techniques essential to a design
12 engineer, not elements derived from the theory
13 of evolution. The mainstay technique of modern
14 biology has made use of the concept of
15 irreducible complexity of the cell's subsystems.
16 And if I can have the next slide I'll iterate on
17 what I mean by that.

262 18 Q. This concept of irreducible complexity,
19 that was coined by Dr. Behe, is that correct?

20 A. Right, right, but I think any working
21 molecular geneticist recognizes that this really
22 explains the approach that we take. This is
23 from Mike's, one of his publication, but I
24 co-opted it here, "By irreducibly complex I mean
25 a single system which is necessarily composed

1 of several well-matched interacting parts that
2 contribute to the basic function and where the
3 removal of any one of the parts causes a system
4 to effectively cease functioning."

263 5 Q. Is this your understanding of the concept
6 of irreducible complexity?

7 A. Correct.

264 8 Q. And I just want to know that this was from
9 an article written by Dr. Behe which has I
10 believe already been admitted as Defendant's
11 Exhibit 203-H, for hotel. Is irreducible
12 complexity one of the, I guess one of the
13 arguments or components of the intelligent
14 design argument, is that correct?

15 A. Right. And I find it difficult when, you
16 know, even this definition is challenged,
17 whether or not it's real or not, because to me
18 as a geneticist this is really restatement of
19 Beadle and Tatum's principle back in the 30's,
20 the two individuals that got molecular genetics
21 going in the last century, you know. One gene,
22 one enzyme, the idea you can use mutational
23 analysis to knock out as individual gene and
24 produce a phenotype, all right -- so if we can
25 go to the next slide.

265 1 Q. Let me just ask you one question before you
2 move on. You have here in this definition, this
3 system, underlined, bold, and in capitals, what
4 purpose was --

5 A. I think because often this is the part
6 that's misunderstood in terms of some of the
7 people that debate these issues, you know.
8 It's not, we're not saying that you can't find
9 components of a given molecular machine
10 associated with another machine and another
11 function. I mean, I have no problem with
12 microevolution co-opts and the certain parts,
13 there are plenty of examples like this.

14 The point being the system that's being
15 studied, the bacterial flagellum, if you take
16 out one of the components of the type three
17 secretion system of the flagellum, we know that
18 we can build it, the cells don't move. That's
19 not to say that you can't have a type three
20 system involved in another function in the cell.
21 But for the system that's being addressed it's
22 irreducible and complex when the fact that we've
23 identified all the components based on
24 mutational analysis.

266 25 Q. Do you find that those who argue against

1 this concept of irreducible complexity change
2 the definition to create a straw man to knock
3 it down?

4 A. You know, I don't know if I'd say straw man
5 or it's intentional. I mean, it's one way you
6 can construe it, but I think it's a subtle but
7 important definition that we're talking just
8 about one system of the cell that we're
9 addressing through mutational analysis, and
10 again you can have components that may be
11 similar in other systems that could be addressed
12 separately, but it's a key point.

267 13 Q. If you could, I know we have another slide
14 for this, break down for us this concept of
15 irreducible complexity and how you employ it
16 in your work in the lab.

17 A. Okay. Molecular machines are comprised of
18 a core set of components that are arranged for a
19 purpose essential for function of that machine.
20 If one of these components is removed from the
21 machine, there's a resulting overall loss of
22 function. If there's no function, then there's
23 nothing to select, you know, from a Darwinian
24 perspective, or you have to assume that there
25 would be some selective advantage for an

1 intermediate, but this implies that mutations
2 in genes encoding pieces of a molecular
3 machinery will yield selectable phenotypes
4 based on this loss of function.

268 5 Q. Could you explain that?

6 A. Selectable phenotypes for a geneticist
7 means that you mutagenize these cells. The
8 hard part for us is coming with a screen or a
9 selection to separate all the mutations that
10 have occurred from the ones that you want to
11 study in the system that you're interested in.
12 I'll show you a picture of how this works in the
13 lab really simply to get this point across, but
14 this process of using mutagenesis and devising
15 genetic screens and selections to identify loss
16 of function has yielded astonishing findings
17 over the last sixty years.

18 This is the bread and butter of molecular
19 genetics. If these systems we worked on weren't
20 irreducibly complex, we would know very little
21 about them. This is a mechanism how the fact
22 that we want to identify all the components of a
23 given molecular machine, we make mutants that
24 trash the system, sort out, map the mutations,
25 how many genes are involved, and then start

1 piecing it back together. It's a very reverse
2 engineering procedure more attuned to, you know,
3 this concept of intelligent design or reverse
4 the design process to understand how these
5 systems work.

269 6 Q. Break down for us further this concept of
7 mutagenesis, and I believe you have a slide --

8 A. Sure. All right. I work on the bacterial
9 flagellum, understanding the function of the
10 bacterial flagellum for example by exposing
11 cells to mutagenic compounds or agents, and then
12 scoring for cells that have attenuated or lost
13 motility. This is our phenotype. The cells can
14 swim or they can't. We mutagenize the cells, if
15 we hit a gene that's involved in function of the
16 flagellum, they can't swim, which is a scorable
17 phenotype that we use. Reverse engineering is
18 then employed to identify all these genes. We
19 couple this with biochemistry to essentially
20 rebuild the structure and understand what the
21 function of each individual part is. Summary,
22 it is the process more akin to design that
23 propelled biology from a mere descriptive
24 science to an experimental science
25 in terms of employing these techniques.

270 1 Q. Do you have some examples employing this
2 particular concept of the flagella?

3 A. I do, in the next slide. Hopefully this
4 will cut to the chase and show you what we're
5 talking about. This is an organism that my
6 students and I work on. This is a petri dish
7 about 15 millimeters size, filled with this soft
8 auger food source for the organism. It's soft
9 in the sense the organisms can swim in it, but
10 it has some rigidity that they just don't slosh
11 around. Now, each one of these areas showing
12 growth were inoculated with a toothpick of
13 cells, the wild type parent here. So this is
14 yersinia enterocolitica, a good pathogen, double
15 bucket disease if you ingest it.

271 16 Q. That's the center?

17 A. Yeah, that's the center, okay? So it can
18 swim. So it was inoculated right here, and over
19 about twelve hours it's radiated out from that
20 point of inoculant. Here is this same derived
21 from that same parental clone, but we have a
22 transposon, a jumping gene inserted into a rod
23 protein, part of the drive shaft for the
24 flagellum. It can't swim. It's stuck, all
25 right? This one is a mutation in the U joint.

1 Same phenotype. So we collect cells that have
2 been mutagenized, we stick them in soft agar,
3 we can screen a couple of thousand very easily
4 with a few undergraduates, you know, in a day
5 and look for whether or not they can swim.

272 6 Q. I'm sorry, just so we're clear on the
7 record, the two you're talking about on the
8 bottom left, the first one was the bottom left
9 and the second one was the bottom right?

10 A. Right.

273 11 Q. Where you took away a portion of the
12 flagella?

13 A. We have a mutation in a drive shaft protein
14 or the U joint, and they can't swim. Now, to
15 confirm that that's the only part that we've
16 affected, you know, is that we can identify
17 this mutation, clone the gene from the wild
18 type and reintroduce it by mechanism of genetic
19 complementation. So this is, these cells up
20 here are derived from this mutant where we have
21 complemented with a good copy of the gene.

22 One mutation, one part knock out, it can't
23 swim. Put that single gene back in we restore
24 motility. Same thing over here. We put, knock
25 out one part, put a good copy of the gene back

1 in, and they can swim. By definition the system
2 is irreducibly complex. We've done that with
3 all 35 components of the flagellum, and we get
4 the same effect.

274 5 Q. And those top left and the top right were
6 restored bacterial flagellum --

7 A. Right.

275 8 Q. -- with the one missing part?

9 A. This is an essential aspect of doing these
10 types of study to show that it's a single
11 component you're dealing with. You complement
12 with only that gene and show that you restore
13 function.

276 14 Q. I believe you have another diagram?

15 A. In this manner we've, in other labs, so
16 this would be a compilation of work done in a
17 number of laboratories around the world. We've
18 contributed to part of this right here and the
19 front end up here, but this is a blueprint for
20 building a flagellum. You know, you have a
21 master control switch that's turned on when it's
22 appropriate. To make a flagellum, turn on the
23 first set of genes, you lay down, you know, a
24 base plate on the inner membrane, and you start
25 assembling from inside of the cell out.

1 So we're putting in, you know, a drive
2 shaft, another ring, our U joint. There are
3 checkpoint controls like just in the assembly
4 of any machine. If there's a defective part
5 there's a feedback loop that will shut down
6 expression of all the succeeding genes to
7 conserve energy in the cell. Eventually you
8 have this rotary engine with a propeller that
9 can extend about five to ten lengths of the
10 cell.

277 11 Q. So this is a blueprint of the flagellum
12 that was developed through using this
13 mutagenesis technique that you're referring to?

14 A. Right. That and biochemistry and cell
15 biology, I think David DeRosier's done a lot
16 of work with the mutants, you know, showing
17 their assembly. You get these, we call them
18 rivet-like structures. So different mutants
19 you can actually isolate these structures at
20 various stages.

278 21 Q. Would it be accurate to say then the design
22 principle which I believe you referred to them
23 as work because these systems are irreducibly
24 complex, is that correct?

25 A. By definition. Again, you know, this is

1 how we do this type of work.

279 2 Q. Now, there are some scientists, and
3 Dr. Miller is one of them, that claim that
4 the bacterial flagellum is not irreducibly
5 complex, and he'll point to the type three
6 secretory systems to make his argument. Are
7 those arguments correct?

8 A. I think they were a valid argument when
9 they first came out. In fact, we worked on
10 type three secretion systems. So when we're
11 talking about that, this structure over here
12 on the right side of this slide, this is an
13 electron micrograph, this is essentially a micro
14 or a nano syringe for the plague organism, like
15 I said, this has killed two hundred million
16 people alone, and most Gram-negative pathogens
17 have them.

18 We were working on the regulation between
19 motility in *Yersinia enterocolitica* and
20 expression of virulence genes which involved a
21 subset of these proteins back in the early 90's,
22 and in fact we made the hypothesis that the
23 toxins made in this system, we didn't know about
24 type three secretory systems at the time,
25 actually using Occam's Razor would be the

1 flagellum. I mean, we had good genetic evidence
2 that the flagellum could be used for other than
3 secretion of flagellar proteins, but there's a
4 subset of proteins involved in both of these at
5 the base that dictate what proteins are secreted
6 through these structures.

7 You build a flagellum from the inside out,
8 all the components are transported through this
9 hollow core and assembled at the distal tip, and
10 with this nano syringe you make toxins and
11 they're actually injected into your white blood
12 cells when you make contact. They're a subset
13 of common proteins between those, and so after
14 reading Mike's book I actually corresponded
15 with him and said, you know, we may have an
16 intermediate for the flagellum.

17 That's a possibility based on our early
18 studies of this. These structures were
19 identified in 1998 by electron microscopy
20 finally, and Dr. Miller, Ken Miller has said
21 that these are the intermediate structure for
22 flagellum biosynthesis, and I was willing to
23 entertain that view. But since then our own
24 work and work in other laboratories I think is
25 showing that it's actually the other way around,

1 that the type three system if anything has been
2 derived from the flagellum. In one of my papers
3 I make that argument. So really to explain this
4 structure you have to presuppose the very thing
5 you're trying to explain. In fact it's being
6 derived from a more complex system.

280 7 Q. Are both of these systems irreducibly
8 complex?

9 A. By definition I mean all the components
10 for the type three system were identified by
11 mutational analysis, and in this case
12 attenuation of virulence.

281 13 Q. Would it be fair to say that if the type
14 three secretory system was found to have
15 preceded the bacterial flagellum, we'd still
16 have difficulty with trying to determine how
17 that one system that functions as a secretory
18 system could then become a separate system that
19 functions as a motor, flagellar motor?

20 A. Right. I mean, that would be a positive
21 argument, I mean, in the sense that it could be
22 an intermediate. But again I think the evidence
23 is falling heavily against it. But sure, but
24 having a nano syringe and developing that into
25 a rotary engine, you know, is a big leap.

282 1 Q. You wrote a paper, and we showed it up here
2 on this next slide, they referred to previously,
3 "The Genetic Analysis of Coordinate Flagella in
4 Type Three Regulatory Circuits and Pathogenic
5 Bacteria," and I believe it's listed as
6 Defendant's Exhibit 254, which should be under
7 Tab 8 in the exhibit binder. If you can confirm
8 that that's the article?

9 A. That's correct.

283 10 Q. Could you explain a little further this
11 article, its findings and its implications for
12 intelligent design?

13 A. Again it's a review of the reason, you
14 know, that we've teased out why pathogenic
15 organisms regulate production of a flagellum
16 in a host environment, and they switch between
17 these type three systems. We show in this paper
18 that there is a logical reason for this, because
19 if you operate these systems simultaneously, in
20 other words if we artificially express flagellum
21 protein, which makes up the filament of the
22 flagellum in the host environment, it will be
23 recognized and secreted by that nano syringe.

24 In fact, will be injected into a white
25 blood cell. Since over the last three to four

1 years we've come to recognize that the sentinel
2 cells of our innate immune system, white blood
3 cells, neutrophils, dendritic cells, have on
4 their surface a receptor looking for bacterial
5 flagellum as a pattern recognition molecule of
6 an invader, and if that receptor gets tickled
7 with flagellum it will induce the innate immune
8 response and an inflammatory response.

9 So the whole point I think it comes into
10 play is why a lot of organisms shut off motility
11 in the host environment is to hide this protein
12 from invading cells, or from the sentinel cells,
13 the white blood cells, that they're going to
14 encounter. That has lots of ramifications. It
15 explains *yersinia pestis*, the bubonic plague
16 organism, is nonmotile even though it has
17 residual flagellar genes in its chromosomes.

18 Flagellar dysentery, the organism that
19 causes bacterial dysentery, has flagellar genes
20 in its genome, but it's nonmotile. *Bordetella*
21 *pertussis*, which we were all immunized for as
22 kids, whooping cough, has flagellar genes in its
23 chromosome, but it doesn't express them because
24 they all operate type three systems. The point
25 being if the type three system is going to be

1 an intermediate, there would be to have sometime
2 in their history where they would both be
3 operational, and that would really work against
4 the organism.

5 I'm going into detail and I don't want to
6 bore people with it, but I find it, you know,
7 fascinating that these important pathogens have
8 lost flagellar synthesis over time, and there's
9 a reason for it in terms of this. We're
10 actually taking purified flagellum, knowing
11 this interaction and why it's dangerous to
12 expose white blood cells to flagellum. We can
13 take purified flagellum, expose a mouse by
14 aerosol or intranasal, and the next day
15 challenge it with ten lethal doses of yersinia
16 pestis or francisella tularensis, which causes
17 tularemia, and it shows significant delay time
18 to death or even protection. I mean, this has
19 been, this is really going to change things in
20 terms of how we look at the initial stages of
21 disease --

22 THE COURT: Did you get that, Wes?

23 THE WITNESS: Am I boring you, judge?

24 THE COURT: Oh, you're not boring me, but
25 I'm concerned about his ability to get -- Wes

1 of course drew the short straw in the court
2 reporter pool for the afternoon, and I'm just
3 concerned that Wes got that. You're going to
4 have to, when you get to a term, what my concern
5 is when you get to a term like several of the
6 terms to try to spell that. Not to protract
7 things, but --

8 THE WITNESS: I apologize.

9 BY MR. MUISE:

284 10 Q. If you could go back, you mentioned several
11 diseases and bacteria. If you could restate
12 those perhaps spell to help us out. The disease
13 for the whooping cough and some of the others
14 that you've mentioned.

15 A. Okay, in terms of you yersinia,
16 Y-E-R-S-I-N-I-A, pestis. That's the bubonic
17 plague organism. Shigella, S-H-I-G-E-L-L-A,
18 bordetella, B-O-R-D-E-T-E-L-L-A, so these are
19 all organisms that operate type three systems
20 that have lost the ability to make a flagellum
21 over time. But the point I'm trying to make is
22 that by approaching this kind of in a systems
23 analysis way it suddenly make sense why
24 organisms regulate these systems, why they're
25 not displaying those proteins, and then we

1 can take advantage of this in terms of our
2 understanding of the innate or nonspecific
3 immune response and manufacture really novel
4 vaccines. New adjuvants, we can use flagellum,
5 you know, packed with epitopes for plague or
6 tularemia or other organisms, and --

285 7 Q. Can you spell those, too? Tularemia was
8 one.

9 A. Right, T U-L-A-R-E-M-I-A I think. I almost
10 have to see it to write it. From Tulare County.
11 Okay, so the point being that this has all kinds
12 of applications in our own work.

286 13 Q. And so you, by looking at this from our
14 perspective of real design you're finding a
15 great deal of utility in applying that approach
16 to it in terms of actually perhaps providing
17 some antibodies or some way to resist these
18 things that will be beneficial to, beneficial
19 results for the community?

20 MR. HARVEY: Objection. Leading. I think
21 he's summarizing a lot of testimony. He's not
22 developing the testimony or moving it along
23 there, which I wouldn't object to, because it
24 does tend to move things along. I think he's
25 testifying, and that's not proper when you've

1 got your own witness, particularly an expert
2 witness, who should be able to explain.

3 MR. MUISE: Your Honor, it was an attempt to
4 summarize, we had some fits and starts with the
5 spelling of these bacteria, and it was just an
6 attempt to summarize --

7 THE COURT: I think -- it's a close call,
8 but I think it's a fair summary at this point.
9 I understand the point. So I'm going to
10 overrule the objection. You can proceed.

11 MR. MUISE: Do you recall the question?

12 THE WITNESS: Repeat the question.

13 THE COURT: Wes, why don't you read the
14 question back for us.

15 (The record was read by the reporter.)

16 THE WITNESS: Close enough.

17 BY MR. MUISE:

287 18 Q. Do you have an answer to that question?

19 A. Yes, I agree. I think, you know, going
20 back to Bruce Alberts that we're looking at
21 this thing kind of from the systems perspective
22 and --

288 23 Q. Dr. Minnich, another complaint that's often
24 brought up, and plaintiffs' experts brought it
25 up in this case, is that intelligent design is

1 not testable. It's not falsifiable. Would you
2 agree with that claim?

3 A. No, I don't. I have a quote from Mike
4 Behe. "In fact, intelligent design is open to
5 direct experimental rebuttal. To falsify such
6 a claim a scientist could go into the
7 laboratory, place a bacterial species lacking
8 a flagellum under some selective pressure,
9 for motility say, grow it for ten thousand
10 generations and see if a flagellum or any
11 equally complex system was produced. If that
12 happened my claims would be neatly disproven."

289 13 Q. Is this an experiment that could be done
14 in a lab?

15 A. It could be, and I, you know, would say
16 that, you know, up the ante. I'll give somebody
17 a time three secretory system intact and the
18 missing proteins required to convert it into a
19 flagellum and let them go, see if you can get a
20 flagellum from a type three system. That's a
21 falsifiable doable experiment. That's just the
22 type of experiment that could be subjected to
23 this type of analysis.

290 24 Q. Would this be an experiment that you would
25 do?

1 A. You know, I think about it, I would be
2 intrigued to do it. Knowing the tolerance
3 limits for these proteins and how they would
4 assemble I wouldn't expect it to work. But
5 that's my bias.

291 6 Q. You think natural selection could account
7 for that, take the type three secretory system,
8 the additional proteins, and see if natural
9 selection can build a bacterial flagellum from
10 that?

11 A. I'm not convinced that it could, but again
12 it's a plausible experiment. They should write
13 a grant and see if we can do it.

292 14 Q. One of the examples that had come up in the
15 course of this trial and I know you're somewhat
16 familiar with, you addressed it in your expert
17 report, it's listed "Icon of Evolution:
18 Antibiotic Resistance." Is this a good example
19 of evolution in practice?

20 A. I don't think so.

293 21 Q. Why not?

22 A. Because it really, it's an extrapolation
23 from the data. It's a good example of
24 adaptation, you know, and here I'm talking
25 about point mutations conferring resistance

1 to specific antibiotics like streptomycin,
2 which is commonly used as a demonstration.
3 You can show a population of cells are sensitive
4 to this drug, put them under selective pressure,
5 isolate mutants that are resistant. It comes
6 with an extreme fitness cost.

7 You know, from my own experience in this
8 you can almost, almost a doubling of the
9 generation time required. These organisms have
10 a difficult time competing. Once the selective
11 pressure is removed you can get compensatory
12 mutations, and this has been shown in the
13 literature, that restore the growth rate, but
14 only for the conditions in which you're doing
15 the experiments.

16 In actuality in biology we have a term for
17 this referred to as Mueller's Ratchet, and that
18 essentially says that when you have a mutation
19 that you turn the ratchet once you're limiting
20 the organism's ability to respond to the next
21 environmental condition required for an
22 adaptational response. And so the more
23 environmental insults or mutations that occur,
24 you're turning this ratchet down tighter and
25 tighter to the point where you're going to limit

1 the organism's ability to eventually survive.

2 So you can show this in this laboratory,
3 it's a beautiful demonstration of adaptation in
4 mutation, but to extrapolate this to the general
5 principles of going from the simple to the
6 complex I think it's out of bounds. If anything
7 it's showing limits or the shortcomings of
8 mutation. I don't think it has anything to do
9 with the complexifying mutations required to
10 drive evolution.

294 11 Q. I guess quoting from Carl Woese, you can't
12 get there from here?

13 A. Yeah, that's exactly it.

295 14 Q. Now, based on your testimony thus far
15 it would seem that the new information about
16 molecular biology calls into question some of
17 the previous assumptions about evolution, is
18 that fair?

19 A. I think that's definitely fair.

296 20 Q. And do scientists other than intelligent
21 design advocates recognize this?

22 A. Yes. This was in the literature. I can
23 go back and look at this paper by Simon Conway
24 Morris, again this is a paleontologist at
25 Cambridge University, well known, this article

1 titled Evolution: Bringing Molecules into the
2 Fold, you know, this is the one where he says
3 that he's going to do this perverse thing about
4 addressing the problems in evolution in the
5 abstract, and he goes through the problems that
6 we have. We cannot still differentiate
7 phenotype from genotype.

8 In other words, the outward expression,
9 the morphology of an organism from its genome,
10 we have a problem in terms of phylogenetic
11 assignments and looking at phylogenetic
12 histories, related histories of derived from
13 molecular clocks versus the fossil record.
14 They're out of sync. Molecular clocks tend
15 to indicate the organisms are much more older
16 than fossil record. The paleontologists argue
17 their interpretation is correct. Molecular
18 biologists will argue that their interpretation
19 is correct.

20 This has to be resolved. When we look at
21 molecular data we get conflicting phylogenies.
22 If you compare a cytochrome amino acid
23 sequences, which was done back in the 60's
24 and the 70's, compared the ribosomal RNA
25 sequences, compared the superoxide dismutates,

1 other essential conserve genes or proteins in
2 the cell, you'll generate a different phylogeny
3 depending upon whether you're looking at one
4 individually or in combination, and this is now
5 being superseded by comparing entire genomes.

6 So bioinformatics is going to be critical
7 in this next stage. You have this question of
8 convergence that we mentioned before again with
9 a beta protein, beta subunit of DNA polymerase,
10 Morris remarks in a couple of examples in this
11 paper and even says if evolution is channelled,
12 in the sense that it's always coming up with the
13 same solution being different routes, pretty
14 complex problems, in his mind teleology is back
15 on the table for discussion.

16 Now, this is a paper in Cell, and he says
17 it's interesting that physicists are reaching
18 the same conclusion in terms of the anthropic
19 principle or the fine tuning principles of the
20 universe. He cites Barrow and Tipler, one of
21 which is a design proponent. As physicists he
22 also cites a reference in terms of biology of
23 Michael Denton, who has been involved in
24 intelligent design and wrote a book previously
25 to the one cited in this article, Evolution: A

1 Theory in Crisis. So here you have a well
2 known paleontologist looking at the problems of
3 evolution, recognizing that they're real,
4 and considering maybe this word teleology,
5 purpose, should be back on the table for
6 discussion.

297 7 Q. Does he use that term in the paper?

8 A. He does. In the discussion at the end.

298 9 Q. Dr. Minnich, I'd like you just to sort of
10 summarize some of these points that you've been
11 discussing here.

12 A. I think if you look at the Carl Woese's
13 paper and read it carefully, he says that
14 nothing in evolution should be not subject to
15 intense review. He even says common descent
16 was a conjecture, an idea of 19th century
17 biologists, that somehow got set in stone. We
18 shouldn't be stuck to it. But I think in terms
19 of my experience, we're dealing with dogmatism
20 versus science and where the data is leading us.

21 Again to emphasize, we can't differentiate
22 genotype from pheno. I read a paper last week,
23 you know, one of the best phylogenetic histories
24 we have is fossil horses in North America.

25 These have been, you know, from the Pleistocene

1 and Miocene time period, and I'm not a
2 paleontologist, but I'm interested in the
3 molecular analysis. These have been well
4 characterized in terms of their phylogenetic
5 history and taxonomy, molecular techniques,
6 isolation of fossil DNA comparing to
7 mitochondrial sequences shows that this
8 phylogeny is artificial, that they're all in
9 the same taxa, perhaps even in the same species.

10 It can't explain the origin of information.
11 This is still a major question in biology, and
12 we're dealing with the most sophisticated
13 information storage system that we know about.
14 We can't explain how life initiated. Origins.
15 We can't explain the existence of the genetic
16 code, this frozen accident I referred to.
17 Convergent examples in evolution are causing
18 people to question, and this is at the molecular
19 level, the organismal level.

20 So I would say that quoting Tulkingshorn,
21 we're in a situation much like the physicists
22 were at the end of the last century, and we
23 suffer from this triumphal arrogance where we
24 think everything can be explained by our
25 Darwinian methodology, just like physicists,

1 everything can be explained in Newtonian
2 mechanics. I think we're at a turning point,
3 and that's not to say that all the work before
4 is not valuable. I think it's critical. I
5 think -- I love reading evolution, and these
6 are important contributions to understanding of
7 life, but I'm convinced there's something more
8 there, and that's why I'm here.

299 9 Q. Dr. Minnich, I want to sort of shift our
10 focus a little bit and talk a little bit about
11 creationism. Is there a popular understanding
12 of this term?

13 A. Creationism has to deal with viewing
14 scientific or the empirical evidence through
15 a literal interpretation of Genesis, six-day
16 creation event.

300 17 Q. What is creation science?

18 A. Again these are scientists that are
19 limiting how they interpret the data through
20 a scriptural context of Genesis, a literal
21 interpretation of Genesis.

301 22 Q. Plaintiffs countering that intelligent
23 design is not science but rather creationism,
24 are they correct?

25 A. No. We have don't have any precommitment

1 to any scripture, revelation, religion. Just
2 looking at the empirical data and using
3 scientific, standard scientific reasoning of
4 cause and effect and asking is it real design or
5 only apparent design.

302 6 Q. Dr. Miller made a claim that if the
7 bacterial flagellum was designed, then it
8 had to be created and therefore it was special
9 creationism. Is that accurate?

10 A. I don't agree with that. I mean, it
11 doesn't say anything about how it was designed,
12 over what time period it was designed, how it's
13 been modified, you know, over time in terms of
14 evolutionary events. So I would disagree.

303 15 Q. Could the bacterial flagellum be designed
16 over time under intelligent design theory?

17 A. Yes. I don't think we're limited by that.

304 18 Q. May I approach the witness, Your Honor?

19 THE COURT: You may.

305 20 Q. Dr. Minnich, I've handed you what's been
21 marked as Defendant's Exhibit 220, a copy of Of
22 Pandas and People, and I believe you testified
23 previously you're familiar with this book,
24 correct?

25 A. I am.

306 1 Q. If I could direct your attention to page
2 99?

3 A. Okay.

307 4 Q. Towards the bottom and then continuing on
5 to the next pages it says, "Intelligent design
6 means that various forms of life began abruptly
7 through an intelligent agency with their
8 distinctive features already intact. Fish with
9 fins and scales, birds with feathers, beaks, and
10 wings, etc., " and it goes on to say, this is
11 the next page, "Some scientists have..." --

12 A. Can I interrupt? You're on 99? I don't
13 see that on page 99.

308 14 Q. Page 99 at the bottom if you look, I'm
15 sorry.

16 A. Okay.

309 17 Q. Look at the last paragraph.

18 A. Mine says, "Darwin has subjected a view of
19 intelligent..." --

310 20 Q. Correct.

21 A. Okay.

311 22 Q. Keep going down five lines.

23 A. Okay.

312 24 Q. So we're at, "Intelligent design means"?

25 A. Right, intelligent design means.

313 1 Q. Let me read this again for you again.
2 "Intelligent design means that various forms
3 of life began abruptly through an intelligent
4 agency with their distinctive features already
5 intact. Fish with fins and scales, birds with
6 feathers, beaks, and wings, etc." And it goes
7 on to say, Some scientists have arrived at this
8 view since fossil forms first appeared in the
9 rock record with their distinctive features
10 intact and apparently fully functional rather
11 than gradually developing." Do you see that?

12 A. I see that.

314 13 Q. Sir, is it your understanding that
14 creationism requires an abrupt appearance
15 of life on earth?

16 A. Creationism, you know, scientific
17 creationism, yeah, ex nihilo appearance of
18 life forms.

315 19 Q. Is this ex nihilo appearance of life forms,
20 is that a theological concept?

21 A. Yes, yes. Out of nothing.

316 22 Q. Does this statement in Pandas that I just
23 reviewed with you, does this make intelligent
24 design creationism?

25 A. No, I don't think so. I mean, this is a

1 literal interpretation of the fossil record
2 where you see the sudden appearance of these
3 forms, you know, fish with fins, etc. in a
4 geologic record. From my interpretation this
5 isn't ex nihilo, you know, creation from
6 nothing.

317 7 Q. Are you familiar with other scientists who
8 are not intelligent design advocates making
9 statements regarding the fossil record using
10 the term abrupt appearance?

11 A. Right. I mean, this is common in
12 paleontology literature. From my understanding
13 Woese even talks about it in the one paper
14 saltational events.

318 15 Q. What's a saltational event?

16 MR. HARVEY: Your Honor, I'm going to
17 object. A question or two on paleontology
18 might have been not something to object to,
19 but this man isn't a paleontologist. He has
20 no expertise in paleontology whatsoever.

21 MR. MUISE: He's testifying here also about
22 this particular book and that intelligent design
23 science is not creationism. He mentioned in
24 Carl Woese's article which he's been testifying
25 to --

1 THE COURT: Heard that. Heard the last
2 thing. Isn't he getting into paleontology?

3 MR. MUISE: All I'm asking him, Your Honor,
4 he used the term saltational event. I asked him
5 what does he mean by that, and that's the end of
6 the question.

7 THE COURT: Well, whether it's the end or
8 not, isn't that paleontology?

9 MR. MUISE: Well, he used the term, and I'm
10 asking him what he means.

11 THE COURT: Well, the objection is that he's
12 not qualified. Tell me why he is. Tell me
13 where it's in his report. Tell me -- it's a
14 technical objection, but it's an objection
15 that's founded in the lack of qualifications.

16 MR. MUISE: He's testifying about the book,
17 Your Honor. That's what he's, about it being
18 good for science, and he said so in his report.
19 He used the term, all I asked him was the term
20 about saltational events and what did he mean by
21 saltational events. He's familiar with the
22 literature. He cited from Carl Woese's article.
23 Carl Woese is a person he's been relying on in
24 most of his testimony.

25 THE COURT: All right. That's your

1 argument. I'll sustain the objection.

2 You'll have to ask a different question.

3 BY MR. MUISE:

319 4 Q. Dr. Minnich, is intelligent design a
5 religious belief?

6 A. No.

320 7 Q. Why not?

8 A. Because again there's no precommitment to
9 any religious tenet or system.

321 10 Q. Is intelligent design inherently religious
11 or advance a religious belief?

12 A. No. Again, I think we're looking at the
13 empirical evidence and asking, you know,
14 specific questions in terms of the Darwinian
15 mechanism and alternative interpretations.

322 16 Q. Do creationists in the sense that
17 plaintiffs and their experts have used in
18 this case require physical evidence to draw
19 their conclusions?

20 A. No, I mean I think by definition if you're
21 a creationist, you're going to rely on the
22 authority of scripture regardless of any
23 evidence that's presented.

323 24 Q. Is that different from a proponent of
25 intelligent design?

- 1 A. Yes.
- 324 2 Q. How so?
- 3 A. Again we're looking at the evidence first
- 4 and not making any precommitment or filtering it
- 5 through any revelation or religious position.
- 325 6 Q. Are intelligent design's conclusions or
- 7 explanations based on any religious,
- 8 theological, or philosophical commitments?
- 9 A. No.
- 326 10 Q. Sir, do you adhere to the literal reading
- 11 of the Book of Genesis?
- 12 A. I don't.
- 327 13 Q. Does intelligent design require adherence
- 14 to the literal reading of the Book of Genesis?
- 15 A. It does not.
- 328 16 Q. Do you believe that the earth is no more
- 17 than six to ten thousand years old?
- 18 A. I believe the earth is according to the
- 19 estimates 4.5 billion years old.
- 329 20 Q. Is that the estimate that's accepted by
- 21 the scientific community?
- 22 A. Yes.
- 330 23 Q. Does intelligent design require adherence
- 24 to the belief that the earth is no more than six
- 25 to ten thousand years old?

1 A. It does not.

331 2 Q. Sir, do you adhere to the flood geology
3 point of view which is advanced by creationists?

4 A. I don't.

332 5 Q. Does intelligent design require adherence
6 to the flood geology point of view advanced by
7 creationists?

8 A. No.

333 9 Q. I have to -- let me strike that and go back
10 because I misstated my question. Do you adhere
11 to the flood geology point of view advanced by
12 creationists?

13 A. No.

334 14 Q. And let me again ask does intelligent
15 design require adherence to the flood geology
16 point of view advanced by creationists?

17 A. No.

335 18 Q. Does intelligent design require the action
19 of a supernatural creator acting outside the
20 laws of nature?

21 A. No.

336 22 Q. Now, in your deposition you claim that the
23 NASA SETI project, which stands for the "Search
24 for Extraterrestrial Intelligence," that that
25 program was seeking a supernatural explanation

1 by searching for intelligence from space. Do
2 you recall that?

3 A. I do.

337 4 Q. And you also indicated that Nobel laureate
5 Francis Crick's claim of directed panspermia was
6 a supernatural explanation for the origin of
7 life, do you recall that?

8 A. I do.

338 9 Q. In what sense were you using supernatural
10 to describe these explanations?

11 A. I think in my deposition I made it clear
12 that these were above our normal experience, or
13 natural experience. So I categorized them as if
14 they're are not natural to our experience they
15 would be supernatural in that limited sense of
16 the word.

339 17 Q. Is it not true that from a scientific
18 perspective these explanation are actual natural
19 explanations?

20 A. They would be, right.

340 21 Q. Does intelligent design rule out these sort
22 of explanations for the source of design?

23 A. Not at all.

341 24 Q. Can science identify the source of design
25 at this point?

1 A. No.

342 2 Q. Does intelligent design rule out a natural
3 explanation for design foundation?

4 A. It doesn't.

343 5 Q. We heard quite a bit of testimony during
6 the course of this trial about methodological
7 naturalism, and I believe you indicated in your
8 deposition you see that as placing limits on
9 intelligent design, is that correct?

10 A. It does. It can. In the sense that it
11 limits explanations it can be advanced, but it
12 has the same kind of stricture on other avenues
13 of scientific research as well.

344 14 Q. Does methodological naturalism necessarily
15 exclude intelligent design from the realm of
16 science?

17 A. No, it doesn't.

345 18 Q. Why not?

19 A. Again, I mean, there could be a natural
20 cause for the systems we're trying to explain.

346 21 Q. Sir, are you aware that there's a statement
22 that is being read to the students which is part
23 of the controversy in this case?

24 A. I am aware.

347 25 Q. I'd like to read that to you here in a

1 moment. This is a statement read to the
2 students from the January 2005. "The
3 Pennsylvania academic standards require
4 students to learn about Darwin's theory of
5 evolution and eventually take a standardized
6 test of which evolution is a part. Because
7 Darwin's theory is a theory it continues to be
8 tested as new evidence is discovered.

9 "The theory is not a fact. Gaps in the
10 theory exist for which there is no evidence.
11 A theory is defined as a well tested explanation
12 that unifies a broad range of observations.
13 Intelligent design is an explanation of the
14 origins of life that differs from Darwin's view.
15 The reference book *Of Pandas and People* is
16 available for students who might be interested
17 in gaining an understanding of what intelligent
18 design actually involves.

19 "With respect to any theory, students are
20 encourage to keep an open mind. The school
21 leaves the discussion of the origins of life to
22 individual students and their families. As a
23 standards driven district, class instruction
24 focuses upon preparing students to achieve
25 proficiency on standards based assessments."

1 Sir, did I read anything to you in that short
2 statement that in your expert opinion will cause
3 any harm to a student's science education?

4 A. Not in my opinion.

348 5 Q. Sir, let me ask you, I want to go through a
6 couple of these sentences. "Because Darwin's
7 theory is a theory, it continues to be tested as
8 new evidence is discovered." Is that true?

9 A. That's true.

349 10 Q. A theory is not a fact, is that true?

11 A. I think we talked about that today, yes.
12 That's true.

350 13 Q. Gaps in the theory exist for which there's
14 no evidence. Is that true?

15 A. That's true.

351 16 Q. And a theory is defined as a well tested
17 explanation that unifies a broad range of
18 observations. Is that a good definition of
19 a theory?

20 A. Yes, it is.

352 21 Q. It says, "Intelligent design is an
22 explanation of the origin of life that
23 differs from Darwin's view." Is that true?

24 A. That's true.

353 25 Q. Sir, in your expert opinion should students

1 be made aware of this information?

2 A. Yes.

354 3 Q. Do you believe it will promote science
4 education?

5 A. I do.

355 6 Q. Dr. Alters, who testified on behalf of the
7 plaintiffs, made the following comments about
8 in his opinion the effect or impact of this
9 statement. I want to read you from his
10 testimony, and he's referring to this, the
11 statement I just read to you. "Now, what this
12 policy is doing is saying that there's this
13 other scientific view that belongs, it belongs
14 in the game of science, and it's the one that
15 most students will perceive as God friendly.
16 It has an intelligent designer. Evolution
17 doesn't.

18 "Now students are going to be in there
19 discussing out on the playground, discussing in
20 their class, among themselves or whatever, that
21 the unit that they're now about to hear about,
22 the evolution unit, that's now coming up is the
23 one that's not God friendly, the one scientific
24 theory that doesn't mention God. But this other
25 so-called scientific theory, intelligent design,

1 is God friendly because there's a possibility
2 that God has this other theory.

3 "What a terrible thing to do to kids. I
4 mean, to make them have to think about defending
5 their religion before learning a scientific
6 concept, how ridiculous. This is probably the
7 worst thing I've ever heard of in science
8 education." What's your reaction to that those
9 comments?

10 MR. HARVEY: Objection, Your Honor. Outside
11 the scope of his expert report. He didn't
12 submit an expert report in rebuttal to
13 Dr. Alters' report. No mention of the statement
14 in the expert report. I don't think it's
15 proper.

16 MR. MUISE: Your Honor, it's all in line
17 with why he believes this is good science
18 education. We've had one expert making these
19 claims, and I'm asking him to comment on those
20 claims as part of his opinion to demonstrate why
21 this should be a part of science education.
22 This was testimony from trial. To say he didn't
23 have it in his expert report is --

24 THE COURT: What was testimony from trial?

25 MR. MUISE: What I just read, Your Honor.

1 THE COURT: Well, I understand that. That
2 begs the question, the question has been raised
3 by Mr. Harvey's objection is, is it in his
4 export report. I do not believe it is. I think
5 you can probably concede that point. Obviously
6 it can't be because the report was prepared
7 prior to Dr. Alters' testimony. Now, the
8 objection then states that there's no rebuttal
9 report that contains this. So in effect he's
10 claiming I think that he's not qualified, and
11 surprised. What do you say about that?

12 MR. MUISE: Your Honor, he's testifying
13 about the --

14 THE COURT: I know what --

15 MR. MUISE: I understand that.

16 THE COURT: I know exactly what he's
17 testifying about. Don't reiterate what he's
18 testifying about. Tell me why I should allow
19 the testimony based on the fact that it's not
20 in the report and that it's, well, fundamentally
21 not in the report, and I think there's a
22 qualification objection inherent in this that I
23 allowed Mr. Harvey to reserve. Dr. Alters in
24 his testimony could take this one step further,
25 he's qualified in that area to render that

1 opinion. Was he not?

2 MR. MUISE: Dr. Minnich is also rendering
3 an opinion that he's qualified regarding this
4 particular policy at issue and whether
5 intelligent design is science and whether
6 it's beneficial for the students.

7 THE COURT: No, that makes no sense what you
8 just said. Dr. Alters was qualified prior to
9 his testimony on the subject of, in the realm of
10 whether he could testify as to whether or not
11 this was good practice to read this statement
12 to 9th grade students. Now, I understand the
13 purposes of this witness generally, but you
14 haven't qualified him on that point. It's on
15 education, and --

16 MR. MUISE: I'm saying you accepted him for
17 science education. Is that --

18 THE COURT: I accepted him subject to, don't
19 misunderstand what I said, subject to objections
20 by Mr. Harvey. Now, the objection goes
21 generally to qualifications and -- it goes
22 broadly to qualifications, but it goes precisely
23 now to a statement outside the report. Now,
24 you had the ability, and in fact you have the
25 obligation if he's going to render an opinion

1 in this area to supplement the report and you
2 didn't do that. So strictly speaking it appears
3 to me to fall considerably outside the report.
4 He may have an opinion on this, I understand
5 that, but it's both outside the report and it's
6 both that and not within the qualifications as I
7 perceive them to be. I also said if you lay a
8 foundation I might consider it. There is no
9 foundation for the opinion, and therefore the
10 objection is at this point sustained.

11 BY MR. MUISE:

356 12 Q. Dr. Minnich, should schools such as Dover
13 make students aware of intelligent design as a
14 scientific theory during their class instruction
15 on Darwin's theory of evolution?

16 A. Through the reading of this one-minute
17 thing, yeah, sure.

357 18 Q. Why?

19 A. I think it promotes critical thinking.
20 It indicates to students that there's important
21 problems that are being discussed in this
22 important area of biology, and it will serve
23 their education well.

358 24 Q. Should schools such as Dover make Pandas
25 available to students as a reference book?

1 A. Yes.

359 2 Q. And why?

3 A. I think it's a valuable resource. It's
4 another way of looking at empirical evidence
5 and how it can interpreted, whether it's a
6 fossil record or molecular data.

360 7 Q. In your expert opinion does the Dover
8 policy at issue in this case promote good
9 science?

10 A. Overall I think it does.

11 MR. MUISE: No further questions, Your
12 Honor.

13 THE COURT: Thank you, Mr. Muise. All
14 right, it's about eleven after 4:00. Do you
15 want to get into cross today, or do you want
16 to --

17 MR. HARVEY: I'm happy to give it a start.

18 THE COURT: We might as well use the time
19 we have and go until 4:30. So you can proceed,
20 Mr. Harvey.

21 MR. HARVEY: Your Honor, may I approach the
22 witness?

23 THE COURT: You may.

24 CROSS EXAMINATION BY MR. HARVEY:

361 25 Q. Dr. Behe -- excuse me, that was a Freudian

1 slip.

2 A. We're clones.

362 3 Q. I didn't, that was not on purpose, I assure
4 you.

5 THE COURT: Obviously the flagellum has you
6 mixed up.

363 7 Q. Dr. Minnich, did anyone help you prepare
8 your expert report in this case?

9 A. No, actually I wrote this over a fairly
10 short period of time, so it reflects I think
11 some of that speed.

364 12 Q. Now, you and Dr. Behe both, or together,
13 you make the same claim, the claim of
14 irreducible complexity?

15 A. Correct.

365 16 Q. And essentially if I understand your
17 contention, it is that an irreducibly complex
18 system is one in which it cannot function unless
19 all the parts are there, and you take away one
20 part and the system ceases to function, correct?

21 A. Correct.

366 22 Q. And the point that you're trying make for
23 purposes of evolution is that irreducibly
24 complex systems in your view cannot evolve?

25 A. I think it's a problem for evolution. In

1 other words, for each intermediate part you have
2 to have some selective advantage to that
3 intermediate structure, and that hasn't been
4 demonstrated. We know that if you remove one
5 part you have no function, and then if you have
6 no function you've got nothing to select.

367 7 Q. You didn't originate this idea of
8 irreducible complexity as a problem for
9 evolution, did you?

10 A. No. I think Mike Behe coined the term, but
11 underlying is the basic argument of design is to
12 account for these complex structures that we
13 find in nature to have the appearance of design,
14 is it real design or apparent.

368 15 Q. Well, and in support of your argument today
16 you spent a certain amount of time with pictures
17 of what you called motors. Did I understand
18 that correctly?

19 A. Correct.

369 20 Q. And you told us that the bacterial
21 flagellum was a true rotary engine, right?

22 A. By definition in the literature that's what
23 we find.

370 24 Q. And I wrote in my notes that you said it
25 was incredible, is that correct?

1 A. Right.

371 2 Q. Do you remember that?

3 A. I used that.

372 4 Q. And you said it has all the components of
5 a rotary engine?

6 A. Correct.

373 7 Q. I guess what I'm trying to say is you're
8 really convinced that this looks a lot like a
9 machine that a human would make?

10 A. Right, and I think the literature supports
11 that.

374 12 Q. Now, Dr. Behe did not originate the concept
13 of irreducible complexity, putting aside the
14 word irreducible complexity, but the concept
15 of irreducible complexity as a problem for
16 evolution, did he?

17 A. I don't know, you know, the entomology of
18 the phrase, so --

375 19 Q. Are you aware that that specific problem
20 was posed in the creationist literature, the
21 creation science literature, as a problem for
22 evolution?

23 A. No, I'm not. I'm not aware of.

376 24 Q. Take a look at what's been marked as P-853.
25 A. 853.

377 1 Q. Please, and Matt, if you can bring it up.

2 A. Are these in order?

378 3 Q. It's towards the back. I can help you if
4 you like.

5 THE COURT: You can approach.

6 A. I got it.

379 7 Q. Dr. Minnich, I'm showing you a publication
8 of the Creation research Society Quarterly from
9 June of 1994. Do you see that?

10 A. I do.

380 11 Q. That's two years before Dr. Behe published
12 Darwin's Black Box, isn't it?

13 A. I'll take your word for it.

381 14 Q. You don't know what year Dr. Behe published
15 Darwin's Black Box?

16 A. '96, '97, I'm not --

382 17 Q. I'd like to -- have you ever seen this
18 publication before?

19 A. No, I haven't.

383 20 Q. Well, I'd like you to go to pages, there's
21 page numbers in the upper, in the corners, in
22 the upper corners, and I'd like you to look at
23 pages 16 to 21. I'm not going to ask you to
24 read it, but I'd just like you to look at it and
25 see -- Matt, if you could page through beginning

1 with page 16 to 21, we'll go through it, I'll
2 invite you to read it if you'd like to, but if
3 you see on page 16 there's a section that begins
4 "bacterial motility"?

5 A. I see it.

384 6 Q. And then on the next page if you turn the
7 page you'll see, Matt, if you can just highlight
8 the language in the lower right-hand column?

9 Yeah, right there, the words "bacterial
10 flagellum," and it's a description of the
11 bacterial flagellum in this piece of literature
12 from this creation science organization, and
13 then if you turn the page again to page 18,
14 there's a description there of the bacterial
15 flagella rotor. Can you highlight that lower
16 paragraph there, Matt? And you'll see it says,
17 "As resolved by electron microscopy, it consists
18 of a series of flanges, grooves, and wheels,
19 yes, wheels, mounted on an axil and turning on
20 bearing surfaces with an efficiency that would
21 be the pride of any industrial research and
22 development operation." Do you see that?

23 A. I see it.

385 24 Q. And then if you'd just please turn the
25 page one more time, there's a diagram, and it's

1 actually Figure 9 in this, and Matt, if you
2 could blow up Figure 9? You have to go to the
3 next page. I'd like the language at the bottom,
4 please. And then if you could, would it be
5 possible to put up Dr. Minnich's slide 18?

6 (Brief pause.)

386 7 Q. And I'd like to ask you just to look at
8 that. Do you see on the Figure 9 from this
9 creation research society publication that
10 there's a picture of the motor rotor complex
11 of the bacterial flagellum?

12 A. Yes, I see.

387 13 Q. And that's very similar to the picture you
14 put up of the bacterial flagellum, isn't that
15 correct?

16 A. Well, I don't know in terms of the labeling
17 of the parts. I haven't read the --

388 18 Q. Well, actually that's what I'd like you to
19 look at for just a second. You'll see that you
20 have labeled something called the universal
21 joint on your, that's D-274, right?

22 A. Right, and again this is, this picture is
23 out of a biochemistry textbook, Voet and Voet.

389 24 Q. I understand.

25 A. Okay.

390 1 Q. I understand. But I just want to -- you
2 have a picture of the universal joint?

3 A. Right.

391 4 Q. And then if you look to the picture that's
5 in the creation research society publication,
6 you'll see that there's, that that diagram has
7 a universal joint as well. Do you see --
8 actually if you look at the bottom and the
9 language at the bottom.

10 A. What's the letter designation?

392 11 Q. It's actually "H," letter designation "H".

12 A. Okay.

393 13 Q. It's called the connective hook universal
14 joint.

15 A. Right.

394 16 Q. And that's the same as in your diagram?

17 A. Correct.

395 18 Q. And then if you look, there's in this
19 Figure 9 from P-853 there's something that's
20 designated "MR," and that's the motor ring?

21 A. Okay.

396 22 Q. And you have motor rings in yours as well,
23 is that right?

24 A. Okay.

397 25 Q. Do you agree?

1 A. I agree.

398 2 Q. And then there's something called, in this
3 Plaintiff's Exhibit 853 there's something called
4 a stationary ring, and in yours you have, also
5 have something in that same place, except it's
6 called an "S" ring, is that right?

7 A. Now we know that that's a single structure
8 in the "S" ring.

399 9 Q. In this Plaintiff's Exhibit 853 there is
10 something that's designated with "AX," and it's
11 called the axil. Do you see that?

12 A. Correct.

400 13 Q. And in yours you have the same thing except
14 it's called the drive shaft, right?

15 A. Right.

401 16 Q. You see that's the same function, right?

17 A. Right.

402 18 Q. Do I have that right? And of course they
19 both have what's been marked as "F," which is
20 the filament. Do you see that?

21 A. I see it.

403 22 Q. Now, and if you turn to page to the next
23 page of this publication, on page 20 -- Matt,
24 can you bring this up? On the left-hand side
25 of the page, about one-third of the way down

1 there's a reference there to bacterial
2 nanomachines. Do you see that?

3 A. I see it.

404 4 Q. And that's the same way you referred to the
5 bacterial flagellum, isn't it?

6 A. I referred to it as a nanomachine or a
7 macromolecular machine.

405 8 Q. A bacterial nanomachine?

9 A. Right. That's explained in the literature,
10 right.

406 11 Q. And then here's where the claim of
12 essentially what I believe is irreducible
13 complexity comes in, if you look on the
14 right-hand side of the page it says -- it's
15 actually the first full sentence on the
16 right-hand side underneath the diagram, it says,
17 "However, it is clear from the details of their
18 operation that nothing about them works unless
19 every one of their complexly fashioned and
20 integrated components are in place." Do you
21 see where it says that?

22 A. I see it.

407 23 Q. And then finally, and I'll bring this to a
24 close, if you go to the abstract on the page,
25 page 13? Matt, if you could just highlight the

1 second half of that, beginning with the word
2 "in terms of biophysical complexity"? I'll
3 read it to you, it says, "In terms of
4 biophysical complexity, the bacterial rotor
5 flagellum is without precedent in the living
6 world. To the micromechanician of industrial
7 research and development operations it has
8 become an inspirational, albeit formidable
9 challenge to best efforts of current technology,
10 but one ripe with potential for profitable
11 applications. To evolutionists the system
12 presents an enigma. To creationists it offers
13 clear and compelling evidence of purposeful
14 intelligent design." Do you see that?

15 A. I see it.

408 16 Q. And I'd like you to agree with me,
17 Dr. Behe, that that is essentially the
18 same argument --

19 A. Minnich.

409 20 Q. I did it again, I'm sorry. I'll just ask
21 the court reporter just when he hears that to
22 just put in Minnich. I'd like you to agree with
23 me, to know whether you agree with me that that
24 is the same argument that you have advanced here
25 today in your direct testimony.

1 A. Right, I mean in terms of -- I don't have
2 any problem with that statement. And I would
3 add that Howard Berg at Harvard University
4 refers to the bacterial flagellum as the most
5 efficient machine known in the universe. So
6 across the board whether, I don't -- what are
7 we arguing here?

410 8 Q. I'm just, you're just confirming for me,
9 and I think you just did, that what we have
10 just reviewed in this Plaintiff's 853 is the,
11 precisely the same argument that you advanced
12 today in support of your, in your direct
13 testimony, isn't that correct?

14 A. Yeah, in essence I mean I don't disagree
15 with you. If you're trying to make a connection
16 with creationism though I would disagree.

17 MR. HARVEY: Well, let's take a look at
18 another exhibit. Could you please go in your
19 binder to what's been marked as -- Your Honor,
20 am I going to be able to run over for a few
21 minutes? Because if not I might as well stop.

22 THE COURT: Why don't we -- Wes has been out
23 here a while, because we've had an extended
24 second session this afternoon because we started
25 early, so I think this would probably be a good

1 time to break. We'll invoke the mercy rule for
2 Wes's benefit because of a lot of complicated
3 testimony this afternoon. All right, you're
4 going to be able to wrap up obviously it would
5 appear to me your cross and any redirect
6 comfortably within the morning tomorrow?

7 MR. HARVEY: It's very much my intention
8 to do so.

9 THE COURT: All right. Let's try to shoot
10 for that. We'll reconvene for what appears to
11 be our final day at 9:00 a.m. tomorrow. We will
12 have all morning to complete this witness's
13 testimony. My best guess is that we would
14 reconvene after lunch and we'll have the
15 evidentiary arguments as we spoke about
16 yesterday, and then we will follow with the
17 closing arguments by counsel in the afternoon.

18 MR. ROTHSCHILD: Your Honor, one question.
19 What is your plan or ascertainment for the order
20 of closing arguments?

21 THE COURT: Well, it's your burden.

22 MR. ROTHSCHILD: Right.

23 THE COURT: So --

24 MR. ROTHSCHILD: My view is that we would
25 then go second if that's acceptable.

1 MR. THOMPSON: Your Honor, I believe the
2 plaintiffs have always gone first.

3 THE COURT: Yeah, why would you go second
4 if it's your burden?

5 MR. ROTHSCHILD: I think my understanding
6 it was my burden, and I was not planning on
7 rebuttal, but that I would go second.

8 THE COURT: No, I would allow you to reserve
9 for rebuttal if you want, but the way I see it
10 you'd go first and I'll allow you to reserve
11 time for rebuttal. I think that's appropriate
12 under the circumstances for the plaintiff to do
13 that, but I think you ought to go first, I agree
14 with Mr. Thompson in that regard, and then we'll
15 hear from the defendant, defendants, and then if
16 you want to carve out part of your time for
17 suitable rebuttal, and you're aware of, if
18 you're not Liz will tell you how much time you
19 have left out of the hour that each side
20 appropriated for your openings, closings, and
21 in the case of the plaintiff the rebuttal, there
22 will be one rebuttal as to the plaintiff. If we
23 didn't make that clear before, that's the way we
24 should do it. All right? Anything further?

25 MR. HARVEY: No, Your Honor.

1 THE COURT: All right, we'll see you all at
2 9:00 a.m. tomorrow. We'll be in recess until
3 then.

4 (Court was adjourned at 4:27 p.m.)

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1 Tammy Kitzmiller, et al. vs. Dover Schools

2 4:04-CV-02688

3 Trial Day 21, Afternoon Session

4 4 November 2005

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I hereby certify that the proceedings
and evidence are contained fully and accurately
in the notes taken by me on the trial of the
above cause, and that this copy is a correct
transcript of the same.

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s/ Wesley J. Armstrong

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Wesley J. Armstrong

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Registered Merit Reporter

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