

PATRICK T. GILLEN, ESQUIRE
RICHARD THOMPSON, ESQUIRE
ROBERT J. MUISE, ESQUIRE
For the Defendants

I N D E X T O W I T N E S S E S

<u>FOR THE DEFENDANTS</u>	<u>DIRECT</u>	<u>CROSS</u>	<u>REDIRECT</u>	<u>RECROSS</u>
---------------------------	---------------	--------------	-----------------	----------------

Michael Behe				
--------------	--	--	--	--

By Mr. Muise				
--------------	--	--	--	--

	3			
--	---	--	--	--

1 THE COURT: Good morning to all. Mr. Muise,
2 if it's Tuesday, we must be on the blood clotting.

3 MR. MUISE: We will be getting to blood
4 clotting, immunity systems, and many more complex
5 systems, Your Honor.

6 THE COURT: All right. You may proceed.

7 MR. MUISE: Thank you.

8 (Whereupon, Michael Behe, Ph.D., resumed the
9 stand and testimony continued.)

10 **DIRECT EXAMINATION (CONTINUED)**

11 BY MR. MUISE:

12 Q. Good morning, Dr. Behe.

13 A. Good morning.

14 Q. Before we do get to the blood clotting, I need to
15 circle back to sort of cover one housekeeping matter.

16 MR. MUISE: If I may approach the witness,
17 Your Honor?

18 THE COURT: Yes.

19 BY MR. MUISE:

20 Q. Sir, I've handed you what has been marked as
21 Defendants' Exhibit No. 237, which is an article from
22 Saier, correct?

23 A. That's right.

24 Q. Is that one of the articles that you referenced
25 during your testimony and appeared on one of the slides

1 regarding the type III secretory system?

2 A. Yes, it is.

3 Q. Okay. Thank you, sir. Sir, yesterday, just to
4 sort of recap and bring us to where we need to begin
5 this morning, I had asked you if some scientists had
6 argued that there is experimental evidence that complex
7 biochemical systems can arise by Darwinian processes,
8 and I believe you indicated there were two that are
9 offered, correct?

10 A. That's right.

11 Q. And the first one was the lac operon?

12 A. Yes.

13 Q. And we discussed that yesterday?

14 A. Yes.

15 Q. And what is the second one?

16 A. The second one concerns what's called the blood
17 clotting cascade, the system for clotting blood in
18 animals. And I should say that, emphasize again that
19 this is the second example of an experimentally -- an
20 experimental result that was offered as evidence against
21 some of the arguments that I made in Darwin's Black Box.

22 In this one, this is directed more to the
23 question of irreducible complexity than to the question
24 of whether Darwinian processes can put together a
25 complex system.

1 Q. Now, sir, we've put up on the slide a figure,
2 6-5, that appears on page 142 in the Pandas text. Can
3 you explain what we see here?

4 A. That's right. This is an electron micrograph of
5 some red blood cells caught in a meshwork of a protein
6 called fibrin, which forms a blood clot. And most
7 people, when they think about blood clotting, if they
8 think about it at all, it appears to be a simple
9 process.

10 When somebody cuts themselves, a minor cut slows
11 down, stops, and heals over, and it doesn't seem like --
12 it doesn't seem like much at all. But thorough
13 investigation over the past 40 to 50 years has shown
14 that the blood clotting system is a very intricate
15 biochemical system. And I believe there's an
16 illustration of it on the next slide.

17 Q. Now you referred to, I believe, a blood clotting
18 cascade, is that correct?

19 A. That's right.

20 Q. Can you explain a little bit to us as you're
21 explaining what we see here on this particular diagram?

22 A. Okay, sure. Yeah, this is a figure of the blood
23 clotting cascade taken from the biochemistry textbook by
24 Voet and Voet, which is widely used in colleges and
25 universities around the country. You see all these

1 names of things and arrows. The names of things are
2 very complex proteins of the complexity or sometimes
3 more complex than the hemoglobin that I showed
4 yesterday.

5 In blood clotting, the material that forms the
6 clot cannot, of course, be in its solid clotted form
7 during the normal -- during the normal life of an animal
8 or all of the blood would be clotted, and that would be
9 inconsistent with its life. So the material of the clot
10 that actual eventually forms the clot exists as
11 something called fibrinogen, which is actually a soluble
12 pre-cursor to the clot material.

13 It floats around in your bloodstream during
14 normal times. But when a cut occurs, fibrinogen is
15 transformed into something called fibrin, and that
16 happens when another protein comes along and cuts off a
17 small piece of fibrinogen, a specific piece which
18 exposes a sticky site on it, sticky in the sense of
19 those two proteins yesterday that I saw that -- that I
20 showed you that had complimentary surfaces.

21 It exposes a sticky site on the surface of the
22 fibrinogen, which allows the many copies of fibrinogen,
23 now turned into fibrin, to aggregate and stick to each
24 other, forming the blood clot.

25 But what is the component that cuts fibrinogen

1 and activates it? Well, the component is another
2 protein called thrombin. But now we've got the same
3 problem again. If thrombin were going around cutting
4 fibrinogen and turning it into fibrin, all the blood
5 would clot, and that would congeal the blood and kill
6 the animal.

7 So thrombin itself is an inactive form called
8 prothrombin, so it has to be activated when a cut
9 occurs. And that's the responsibility of another
10 protein. And that protein exists in an inactive form,
11 and it's -- the activation of that is the responsibility
12 of another protein.

13 So in the blood -- it's called a blood clotting
14 cascade because one component acts on the next which
15 acts on the next which acts on the next and so on. Now
16 notice that the blood clotting cascade actually has what
17 are called two branches. There is one in this box up
18 here is labeled the intrinsic pathway. And this is
19 labeled the extrinsic pathway. So there are actually
20 two branches to this blood clotting cascade.

21 Q. I believe this section is addressed in the
22 textbook Pandas, correct?

23 A. Yeah, that's correct. On the left is a figure
24 from Of Pandas and People illustrating the blood
25 clotting cascade. And that was drawn after the

1 illustration from the textbook by Voet and Voet. On the
2 right-hand side is the illustration for the blood
3 clotting cascade that appears in Darwin's Black Box.

4 I discussed the blood clotting cascade in one
5 chapter of that -- of my book, and the illustration is
6 very similar to the one in Pandas.

7 Q. I believe the diagram in Pandas is found on page
8 143?

9 A. Yes, that's right.

10 Q. Now these two diagrams, the one that appears in
11 Darwin's Black Box and one of the blood clotting cascade
12 appear, to my eye, to be virtually similar or almost
13 exactly similar?

14 A. Yeah, they are very similar, except for the color
15 in Pandas and so on. And that's because I wrote the
16 discussion in Pandas and, of course, also in my own
17 book. So the figures are very similar between the two.

18 Q. Now you testified yesterday that you coined the
19 term irreducible complexity in Darwin's Black Box, which
20 was published in 1996, is that correct?

21 A. Yes.

22 Q. So that book was published actually three years
23 after Pandas was written, is that accurate?

24 A. Yes, that's correct.

25 Q. Is it accurate to say then that the concept of

1 irreducible complexity was not fully developed when you
2 had written that section in Pandas on blood clotting in
3 1993?

4 A. Yes, that's right. I was still contemplating the
5 idea.

6 Q. Does Pandas, however, discuss the complexity of
7 this system, the blood clotting system?

8 A. Yes, it does. It elucidates all the parts of the
9 system.

10 Q. Is that discussion consistent with your
11 discussion in Darwin's Black Box?

12 A. Yes, it introduces the concept of the purposeful
13 arrangement of parts and says that's how we perceive
14 design.

15 Q. That's introduced in the Pandas book?

16 A. Yes, uh-huh.

17 Q. When you talk about the purposeful arrangement of
18 parts, that's similar to what you were discussing
19 yesterday in your testimony, is that correct?

20 A. Yes.

21 Q. So is the scientific explanation of the blood
22 clotting system similar to the -- the discussion in
23 Pandas similar to the blood clotting cascade scientific
24 explanation in Darwin's Black Box?

25 A. That's right, they're essentially the same. I

1 think it's more detailed in Darwin's Black Box.

2 Q. In fact, you did use the similar diagrams?

3 A. Yes, that's correct.

4 Q. To explain the two?

5 A. Yes, uh-huh.

6 Q. I believe the next slide we have is, this is from
7 your -- you discussed this and treated this as well in
8 your book Debating Design, is that correct?

9 A. That's right. When I wrote Darwin's Black Box,
10 and when Darwin's Black Box was subsequently reviewed by
11 people, some of them looked at the argument about the
12 blood clotting cascade and argued against what I had
13 written in Darwin's Black Box.

14 And I thought that the counterarguments were
15 themselves flawed, and so I answered some of those
16 arguments in a variety of cites, but most recently in
17 the chapter in that book, Debating Design, published by
18 Cambridge University Press from the year 2004.

19 I wrote The Blood Clotting Cascade. Having dealt
20 with some common misconceptions about intelligent
21 design, I will examine two systems that were proposed as
22 serious counterexamples of my claim of irreducible
23 complexity. One of them discussed in that article is
24 the blood clotting cascade.

25 Q. If you could then, explain to us how you refute

1 the claims that are made that the blood clotting cascade
2 is experimental evidence to refute irreducible
3 complexity?

4 A. Okay. In the next slide, I believe that shows an
5 excerpt from an article written by a man named Russell
6 Doolittle entitled A Delicate Balance, which appeared in
7 a publication called the Boston Review in 1997. Now
8 Russell Doolittle is a very eminent scientist, a
9 professor of biochemistry at the University of
10 California, San Diego.

11 He's a member of the National Academy of
12 Sciences, and has worked on the blood clotting system
13 for the past 45 years or so. And this article was a
14 part of the symposium organized by Boston Review, which
15 again is published by MIT, and contained contributions
16 from a number of academics, scientists discussing my
17 book and discussing a book that had been recently
18 published by Richard Dawkins of Oxford University.

19 Participants included myself, Russell Doolittle,
20 James Shapiro, who is a professor of microbiology at the
21 University of Chicago, Alan Orr, who is a professor of
22 evolutionary biology at the University of Rochester,
23 Robert DiSilvestro, who is a professor of biochemistry
24 at Ohio State, and a number of other people as well.

25 And in his essay, Professor Doolittle argued

1 that, in fact, there was experimental evidence showing
2 that the blood clotting system was not irreducibly
3 complex. And he said the following. Let me read the
4 quote. Quote, Recently the gene for plaminogen (sic) --
5 and that's actually a typo. There should be an S there.
6 The gene for plaminogen (sic) was knocked out of mice --
7 which means that it was destroyed by molecular
8 biological methods -- and predictable, those mice had
9 thrombotic complications because fibrin clots could not
10 be cleared away.

11 Let me stop a second and explain that plasminogen
12 is a protein that acts as a chemical scissors which cuts
13 up and removes blood clots once the clot has finished
14 its job. Let me resume the quote from Russell
15 Doolittle. Not long after that, the same workers
16 knocked out the gene for fibrinogen in another line of
17 mice. Again, predictably, these mice were ailing,
18 although in this case, hemorrhage was the problem.

19 Let me stop again and explain that fibrinogen,
20 remind you, is the pre-cursor of the clot material
21 itself, the pre-cursor of those fibers. And what do you
22 think happened when these two lines of mice were
23 crossed? For all practical purposes, the mice lacking
24 both genes were normal.

25 Contrary to claims about irreducible complexity,

1 the entire ensemble of proteins is not needed. Music
2 and harmony can arise from a smaller orchestra. So
3 Professor Doolittle's point, if I just might briefly
4 say, was that, if you knock out one component of the
5 blood clotting cascade, yes, those mice have problems.

6 If you knock out a different component in a
7 different line of mice, yes, those mice have problems,
8 too. But if you make a string of mice in which both of
9 those components were missing, then the mice are normal
10 and the blood clotting cascade is okay. And so
11 presumably then, that shows that the blood clotting
12 cascade is not irreducibly complex.

13 Q. Was there a particular study that Professor
14 Doolittle is referring to?

15 A. Yes, it's shown on the next slide. This is the
16 article that he was referencing in his own essay. It's
17 entitled Loss of Fibrinogen Rescues Mice from the
18 Pleiotropic Effects of Plasminogen Deficiency. Now if
19 we could go to the next slide.

20 Now because of the phrase, rescues mice, in the
21 title, Professor Doolittle thought that the mice missing
22 both components were normal. But it turns out, that was
23 a misreading of the article.

24 In the abstract of the article itself, the
25 authors write, quote, Mice deficient in plasminogen and

1 fibrinogen are phenotypically indistinguishable from
2 fibrinogen deficient mice. Now translated that into
3 English on the next slide.

4 That means that mice missing both components have
5 all the problems that mice missing fibrinogen only have.
6 Their blood does not clot. They hemorrhage. Female
7 mice die during pregnancy. They are not normal. They
8 are not promising evolutionary intermediates. So if we
9 look at this table of the symptoms of the various
10 strings of mice, we can see what the authors meant by
11 that phrase, rescues mice.

12 Lacking plasminogen, mice can't remove blood
13 clots once their job is done and their blood circulation
14 gets interfered with and they develop problems such as
15 thrombosis, ulcers, and so on. Lacking fibrinogen, they
16 can't clot blood in the first place, and they have a
17 different suite of symptoms.

18 When they lack both, they have been rescued from
19 the symptoms of plasminogen deficiency, but only to
20 suffer the symptoms of fibrinogen deficiency. And if
21 you think about it for just a minute, it's easy to
22 understand what is going on. When an animal lacks
23 plasminogen, it can't remove blood clots and its
24 circulation becomes impeded and it suffers problems.

25 Lacking fibrinogen, it can't make clots in the

1 first place, and so hemorrhage is a problem. Lacking
2 both, it doesn't matter that it's lacking plasminogen,
3 because the plasminogen's job is to remove blood clots
4 after the job is finished. But the mouse missing both
5 components can't form clots in the first place. So
6 there are no clots to remove.

7 Q. Has subsequent work verified those results?

8 A. Yes, here's a table of not only the work that was
9 cited in this discussion here on plasminogen fibrinogen,
10 but also subsequent work by the same group of scientists
11 who knocked out other components of the blood clotting
12 cascade, including something called prothrombin and
13 something else called tissue factor.

14 And if you look at the -- under the column
15 labeled effect, in each case the blood clotting cascade
16 is broken. They suffer hemorrhage. They cannot clot
17 their blood. And that is exactly the result you would
18 expect if, in fact, the blood clotting cascade were
19 irreducibly complex, as I had written.

20 Q. So Professor Doolittle's refutation of your
21 claims was based on a misreading of the study, is that
22 correct?

23 A. That's right. He misread the original paper that
24 he pointed to. And if I could make a couple of points
25 based on this. As I said, this study, or this essay by

1 Professor Doolittle and the one I discussed yesterday by
2 Professor Miller were the two examples which offered
3 experimental evidence that either irreducible complexity
4 was not correct or that random mutation and natural
5 selection could explain complex biochemical systems.

6 But if you look at the exact studies that were
7 offered as support for Darwinian evolution, and you look
8 at them closely, in reality, they highlight the
9 difficulties for Darwinian evolution. So I think this
10 is an illustration of how a scientist's preconceptions
11 about the truth of a theory or the validity of a theory
12 can affect his reading of the evidence.

13 And one more point is that, Professor Doolittle,
14 of course, is a very eminent scientist. Professor
15 Miller is, too. And they're quite capable of surveying
16 the entire scientific literature for studies that they
17 think are problems for my argument for intelligent
18 design.

19 And nonetheless, when they surveyed the whole
20 literature, and they seemed to be motivated to look for
21 counterexamples to intelligent design, when they do so,
22 they offer studies such as this, which are, at best,
23 very problematic and none of which, I would say, are
24 arguments against intelligent design.

25 So in my mind, I conclude that since highly

1 motivated capable scientists who could advance arguments
2 or who could point to studies that have created problems
3 for intelligent design, that they have failed to do so,
4 makes me confident that intelligent design is a good
5 explanation.

6 Q. Now these article findings, the actual findings
7 in these articles, is that what you would expect to find
8 for an irreducibly complex system?

9 A. Yes, that's right. This is completely consistent
10 with my expectations.

11 Q. As far as you know, has Professor Doolittle ever
12 acknowledged that he misread that paper?

13 A. Yes, he has.

14 Q. And if I could --

15 MR. ROTHSCCHILD: Objection. Hearsay, Your
16 Honor. I would move to strike.

17 MR. MUISE: Your Honor, he just -- he has an
18 understanding that Professor Doolittle has indicated he
19 has misread this paper.

20 MR. ROTHSCCHILD: If he has a basis, I'd like
21 to see it.

22 THE COURT: Well, it's his understanding,
23 and I'm take it for that. I won't take it as a matter
24 of fact. His understanding is, he didn't quote
25 something that Professor Doolittle said. It's simply,

1 I'll take it as his understanding, and you're free to
2 cross-examine him and present rebuttal evidence, if you
3 see fit. So it's overruled.

4 BY MR. MUISE:

5 Q. Dr. Behe, I'd ask you to look at the exhibit
6 binder that I had provided you yesterday. It's at your
7 table in front of you. If you go to tab 17, please.

8 A. Yes.

9 Q. You'll see an exhibit marked Defendants' Exhibit
10 272. Is that the article by Russell Doolittle that
11 you've been referring to here in your testimony?

12 A. Yes, that's correct. This is a web version.

13 MR. ROTHSCHILD: Objection, Your Honor. I
14 want to make clear, I think that's not the
15 acknowledgment of the mistake, it's just the article
16 that's being referred to. I just want to clarify that.

17 MR. MUISE: I think the question was pretty
18 clear.

19 BY MR. MUISE:

20 Q. That's the article in the Boston Review that
21 you're referring to?

22 A. Yes, this is Russell Doolittle's article in the
23 Boston Review.

24 THE COURT: Does that resolve the objection?

25 MR. ROTHSCHILD: Yes. I just want to

1 clarify, this was not Dr. Doolittle's acknowledgment of
2 a mistake.

3 THE WITNESS: Yes.

4 THE COURT: All right.

5 BY MR. MUISE:

6 Q. Dr. Behe, does anyone else know how the blood
7 clotting cascade can be explained in Darwinian fashion
8 and other proposed examples or explanations?

9 A. No, that's one of the very nice things about
10 science is that, if there is no explanation in the
11 science library in scientific literature, and if leaders
12 in the field do not know how something could have come
13 about, and presumably they know the literature very,
14 very well, then one can be confident that not only do
15 they not know how something could have been done, but
16 nobody else in the world knows how that could have been
17 done as well. And that's important to keep in mind
18 because some people claim that nonetheless.

19 Q. And that's my next question. There have been
20 individuals that nonetheless have made such claims, and
21 do you have some slides to bring that up?

22 A. Yes, that's correct. On the next slide is an
23 excerpt from an article by a man named Michael Ruse.
24 Michael Ruse is a professor of philosophy of science
25 currently at Florida State University. And in

1 particular, he's a philosopher interested in Darwinian
2 thought.

3 And he's written many books on Darwin, his ideas,
4 the history around them, and so on. And several years
5 after my book came out in 1998, Professor Ruse wrote an
6 article entitled Answering the Creationists, Where They
7 Go Wrong and What They're Afraid Of, and had it
8 published in a magazine called Free Inquiry. And he
9 said the following in the article.

10 Quote, For example, Behe is a real scientist, but
11 this case for the impossibility of a small-step natural
12 origin of biological complexity has been trampled upon
13 contemptuously by the scientists working in the field.
14 They think his grasp of the pertinent science is weak
15 and his knowledge of the literature curiously, although
16 ventsly, outdated.

17 For example, far from the evolution of clotting
18 being a mystery, the past three decades of work by
19 Russell Doolittle and others has thrown significant
20 light on the ways in which clotting came into being.
21 More than this, it can be shown that the clotting
22 mechanism does not have to be a one-step phenomenon with
23 everything already in place and functioning. One step
24 in the cascade involves fibrinogen, required for
25 clotting, and another, plaminogen -- there's that typo,

1 missing the S -- required for clearing clots away.

2 And he goes on in his article to quote that
3 passage from Russell Doolittle's Boston Review essay
4 that I showed on the slide a couple slides ago. So this
5 excerpt, in my view, shows that Professor Ruse relies
6 completely on Professor Doolittle's explanation for the
7 blood clotting cascade and has no independent knowledge
8 of his own.

9 As a matter of fact, the fact that the same typo,
10 the same misspelling of plasminogen occurs in Professor
11 Ruse's essay makes me think that he relied on Professor
12 Doolittle even for the spelling of the components of the
13 cascade. So the point is that, even though Professor
14 Ruse is a prominent academic concerned with Darwin and
15 Darwinian thought, he has no knowledge that Professor
16 Doolittle does not have concerning the blood clotting
17 cascade.

18 Q. Do you have another example, sir?

19 A. Yes, another person has written on this, a man
20 named Neil Greenspan, who is a professor of pathology at
21 Case Western Reserve University, and he wrote an article
22 in a magazine called The Scientist in the year 2002
23 entitled Not-so-intelligent Design. In the article, he
24 writes the following. Quote, The Design advocates also
25 ignore the accumulating examples of the reducibility of

1 biological systems. As Russell Doolittle has noted in
2 commenting on the writings of one ID advocate -- and
3 perhaps I can be forgiven if I think he means me -- mice
4 genetically altered so they lack either thrombin or
5 fibrinogen have the expected abnormal hemostatic
6 phenotypes. However, when the separate knockout mice
7 are bred, the double knockouts apparently have normal
8 hemostasis, reducible complexity after all, at least in
9 the laboratory.

10 So the reasoning here exactly mimics the
11 reasoning of Russell Doolittle in his Boston Review
12 article. And let me just point out here that he talks
13 about thrombin or fibrinogen, but the study was actually
14 on plasminogen and fibrinogen. So again, I think this
15 illustrates that even a scientist has -- even a
16 scientist writing publicly on this topic, even a
17 scientist writing publicly on this topic in order to
18 argue against intelligent design has no more knowledge
19 of this than Professor Doolittle has.

20 And once more, I think this speaks to the point
21 of how firmly a theory can guide persons' thinking. I
22 think the fact that Professor Ruse relied so heavily on
23 Professor Doolittle, and Professor Greenspan did, too,
24 and apparently they did not even go back and read the
25 article on blood clotting that was being disputed, shows

1 that they are so confident in Darwinian evolution that
2 they don't think they have to, you know, check the
3 facts.

4 They can rely on the authority of a person like
5 Professor Doolittle. So I think that shows the grip of
6 a theory on many people's thinking.

7 Q. Do you have an additional example?

8 A. Yes, one other excerpt here. In 1999, the
9 National Academy of Sciences issued a booklet called
10 Science and Creationism. And in it, they write the
11 following, quote, The evolution of complex molecular
12 systems can occur in several ways. Natural selection
13 can bring together parts of a system for one function at
14 one time, and then at a later time, recombine those
15 parts with other systems of components to produce a
16 system that has a different function.

17 Genes can be duplicated, altered, and then
18 amplified through natural selection. The complex
19 biochemical cascade resulting in blood clotting has been
20 explained in this fashion.

21 Let me make a comment on this. Professor
22 Doolittle is a member of the National Academy of
23 Sciences. There is no other member of the National
24 Academy who knows anything more about blood clotting
25 than Professor Doolittle. But if Professor Doolittle

1 does not know how Darwinian processes could have
2 produced the blood clotting cascade, as I think is
3 evident from his pointing to an inappropriate paper in
4 his attempt to refute a challenge to Darwinian
5 evolution, then nobody in the National Academy knows
6 either. I should also -- well, I'll --

7 Q. Do they cite any papers or experiments to support
8 this claim, the National Academy of Sciences, in this
9 particular booklet?

10 A. No. That's a very interesting point. They
11 simply assert this. They do not cite any paper in any
12 journal to support this. And it's an interesting point,
13 if I may say so. I've heard said earlier in this trial
14 that not every utterance by a scientist is a scientific
15 statement.

16 And that's something that I entirely agree with.
17 And it's also true that not every utterance by a
18 scientist even on science is a scientific statement.
19 And it's also true that not even, not every
20 proclamation, or not every declaration by a group of
21 scientists about science is a scientific statement.

22 Scientific statements have to rely on physical
23 evidence. They have to be backed up by studies. And
24 simply saying that something is so does not make it so.
25 In fact, this statement of the National Academy is

1 simply an assertion. It is not a scientific statement.

2 Q. Does the National Academy of Sciences, in this
3 document that you referenced, give any other examples of
4 complex biochemical systems that have been explained?

5 A. This is the only example that they point to.

6 Q. In his testimony, Dr. Miller has pointed to the
7 work of, I believe, you pronounce is Jiang, J-i-a-n-g --

8 A. Yes.

9 Q. -- and Doolittle and Davidson, et al, to argue
10 against the irreducible complexity of the blood clotting
11 system. Do you agree with his assessment of those
12 studies?

13 A. No, I do not.

14 Q. And you have some diagrams to explain this
15 further, sir?

16 A. Yes, I do. This is a slide from Professor
17 Miller's presentation showing work from Jiang and
18 Doolittle. And he also shows a diagram of the blood
19 clotting cascade. And notice again, it's a branched
20 pathway with the intrinsic pathway and the extrinsic
21 pathway.

22 And Professor Miller makes the point that in DNA
23 sequencing studies of something called a puffer fish,
24 where the entire DNA of its genome was sequenced, and
25 scientists looked for genes that might code for the

1 first couple components of the intrinsic pathway, they
2 were not found.

3 And so Professor Miller demonstrated that by --
4 if you could push to start the animation -- Professor
5 Miller demonstrated that by having those three
6 components blanked out in white. Nonetheless, puffer
7 fish have a functioning clotting system. And so
8 Professor Miller argued that this is evidence against
9 irreducible complexity.

10 But I disagree. And the reason I disagree is
11 that I made some careful distinctions in Darwin's Black
12 Box. I was very careful to specify exactly what I was
13 talking about, and Professor Miller was not as careful
14 in interpreting it.

15 In Darwin's Black Box, in the chapter on blood
16 clotting cascade, I write that, a different difference
17 is that the control pathway for blood clotting splits in
18 two. Potentially then, there are two possible ways to
19 trigger clotting. The relative importance of the two
20 pathways in living organisms is still rather murky.
21 Many experiments on blood clotting are hard to do. And
22 I go on to explain why they must be murky.

23 And then I continue on the next slide. Because
24 of that uncertainty, I said, let's, leaving aside the
25 system before the fork in the pathway, where some

1 details are less well-known, the blood clotting system
2 fits the definition of irreducible complexity.

3 And I noted that the components of the system
4 beyond the fork in the pathway are fibrinogen,
5 prothrombin, Stuart factor, and proaccelerin. So I was
6 focusing on a particular part of the pathway, as I tried
7 to make clear in Darwin's Black Box.

8 If we could go to the next slide. Those
9 components that I was focusing on are down here at the
10 lower parts of the pathway. And I also circled here,
11 for illustration, the extrinsic pathway. It turns out
12 that the pathway can be activated by either one of two
13 directions. And so I concentrated on the parts that
14 were close to the common point after the fork.

15 So if you could, I think, advance one slide. If
16 you concentrate on those components, a number of those
17 components are ones which have been experimentally
18 knocked out such as fibrinogen, prothrombin, and tissue
19 factor.

20 And if we go to the next slide, I have red arrows
21 pointing to those components. And you see that they all
22 fall in the area of the blood clotting cascade that I
23 was specifically restricting my arguments to. And if
24 you knock out those components, in fact, the blood
25 clotting cascade is broken. So my discussion of

1 irreducible complexity was, I tried to be precise, and
2 my argument, my argument is experimentally supported.

3 Q. Now just by way of analogy to maybe help explain
4 further. Would this be similar to, for example, a light
5 having two switches, and the blood clotting system that
6 you focus on would be the light, and these extrinsic and
7 intrinsic pathways would be two separate switches to
8 turn on the system?

9 A. That's right. You might have two switches. If
10 one switch was broke, you could still use the other one.
11 So, yes, that's a good analogy.

12 Q. So Dr. Miller is focusing on the light switch,
13 and you were focusing on the light?

14 A. Pretty much, yes.

15 Q. I believe we have another slide that Dr. Miller
16 used, I guess, to support his claim, which you have some
17 difficulties with, is that correct?

18 A. Yes, that's right. Professor Miller showed these
19 two figures from Davidson, et al, and from Jiang, et al,
20 Jiang and Doolittle, and said that the suggestions can
21 be tested by detailed analysis of the clotting pathway
22 components.

23 But what I want to point out is that whenever you
24 see branching diagrams like this, especially that have
25 little names that you can't recognize on them, one is

1 talking about sequence comparisons, protein sequence
2 comparisons, or DNA nucleotide sequence comparisons. As
3 I indicated in my testimony yesterday, such sequence
4 comparisons simply don't speak to the question of
5 whether random mutation and natural selection can build
6 a system.

7 For example, as I said yesterday, the sequences
8 of the proteins in the type III secretory system and the
9 bacterial flagellum are all well-known, but people still
10 can't figure out how such a thing could have been put
11 together. The sequences of many components of the blood
12 clotting cascade have been available for a while and
13 were available to Russell Doolittle when he wrote his
14 essay in the Boston Review.

15 And they were still unhelpful in trying to figure
16 out how Darwinian pathways could put together a complex
17 system. And as we cited yesterday, in Professor
18 Padian's expert statement, he indicates that molecular
19 sequence data simply can't tell what an ancestral state
20 was. He thinks fossil evidence is required.

21 So my general point is that, while such data is
22 interesting, and while such data to a non-expert in the
23 field might look like it may explain something, if it's
24 asserted to explain something, nonetheless, such data is
25 irrelevant to the question of whether the Darwinian

1 mechanism of random mutation and natural selection can
2 explain complex systems.

3 Q. So is it your opinion then, the blood clotting
4 cascade is irreducibly complex?

5 A. Yes, it is.

6 Q. Now Professor Pennock had testified that he was
7 co-author on a study pertaining to the evolution of
8 complex features. Does this study refute the claim of
9 irreducible complexity?

10 A. No, it does not.

11 Q. And I believe we put up a slide indicating the
12 paper that was apparently by Lenski and Pennock,
13 correct?

14 A. That's right. Richard Lenski, and Professor
15 Pennock was co-author, and several other co-authors as
16 well. This is the first page of that article. Let me
17 reemphasize that the last two systems that I talked
18 about, the lac operon and the blood clotting cascade
19 were ones in which experiments were done on real
20 biological organisms to try to argue against intelligent
21 design and irreducible complexity.

22 This study of Lenski is a computer study, a
23 theoretical study not using live organisms, one which is
24 conducted by writing a computer program and looking at
25 the results of the computer program.

1 If I could have the next slide. This is an
2 excerpt from the abstract of that paper. Let me read
3 parts of it. It says, quote, A long-standing challenge
4 to evolutionary theory has been whether it can explain
5 the origin of complex organismal features, close quote.
6 Let me just stop there to emphasize that these workers
7 admit that this has been a long-standing problem of
8 evolutionary theory.

9 MR. ROTHSCILD: Objection. This
10 mischaracterizes the document.

11 THE COURT: Elaborate on that objection.

12 MR. ROTHSCILD: I'm sorry?

13 THE COURT: Elaborate on the objection. You
14 say he's mischaracterizing --

15 MR. ROTHSCILD: This is a long-standing
16 challenge not a long-standing problem.

17 THE COURT: Well, I think he's
18 characterizing something and not necessarily reading
19 from it. What are you objecting to?

20 MR. ROTHSCILD: I think he's
21 mischaracterizing it. That's my objection.

22 THE COURT: Again, you'll have him on cross.
23 This is direct examination. I'll overrule the
24 objection. You may proceed.

25 BY MR. MUISE:

1 Q. Dr. Behe, just for reference, the article you are
2 referring to is published in 2003, is that correct?

3 A. That's correct, yes.

4 Q. Continue, please.

5 A. So apparently, this had not been explained up
6 until at least the publication of this paper. The
7 authors continue, quote, We examined this issue using
8 digital organisms, computer programs that
9 self-replicate, mutate, compete and evolve. Let me
10 close quotes there.

11 You have to remember that the labeling of these
12 things as organisms is just a word. These things are
13 not flesh and blood. These things are little computer
14 programs. There are strings of instructions. And a
15 comparison of these to real organisms is kind of like
16 comparing an animated character in some movie to a real
17 organism.

18 So the authors go on. And the next slide,
19 please. And this is the first figure on the first page
20 of their article. And I just want to emphasize, this is
21 just an illustration emphasizing that these -- there are
22 computer instructions. Each one of these are little
23 computer instructions; swap, nand, nand, shift R. They
24 have no similarity to biological features, biological
25 processes. You see over here little strings of ones and

1 zeroes.

2 These are characters in a computer memory. These
3 are not anything biological. Let me say that,
4 theoretical studies of biology can oftentimes be very
5 useful. And I'm certainly not denigrating the use of
6 computer in studying biology. But one has to be
7 careful, very careful that one's model, computer model
8 mimics as closely as possible a real biological
9 situation. Otherwise, the results one obtains really
10 don't tell you anything about real biology.

11 And I think that the Lenski paper, it does not
12 mimic biology in the necessary way. And that's shown on
13 the next slide.

14 Q. Let me just, to clarify. So a crucial question
15 is whether or not it's a good model for biological
16 process, is that correct?

17 A. Yes, that's right.

18 Q. And you don't believe this is one?

19 A. No, I think it misses the point and it assumes
20 what should be proven instead. And let me try to
21 explain that with an excerpt from the article itself.
22 The authors write in their discussion, quote, Some
23 readers might suggest that we stacked the deck by
24 studying the evolution of a complex feature that could
25 be built on simpler functions that were also useful,

1 close quote.

2 Let me stop there to comment that, yes, that is
3 exactly what I would suggest, that they stacked the
4 deck. They built a model in which there was a
5 continuous pathway of functional Features very close
6 together in probability, which is exactly the question
7 that's under dispute in real biological organisms. Is
8 there such a pathway in real biological organisms?

9 So to assume that in your computer model is
10 stacking the deck. Let me go back to the abstract.
11 They continue, quote, However, that is precisely what
12 evolutionary theory requires. Now I'll close quote
13 there, and let me comment on that.

14 Just because your theory requires something does
15 not mean it exists in nature. James Clerk Maxwell's
16 theory required ether. Ether does not exist. So just
17 because a theory requires it is no justification for
18 saying that building a model shows something about
19 biology.

20 Q. Dr. Behe, if you could, just so we're clear on
21 the record, because I'm not sure if we have it that
22 clear, can you identify the title and the specifics of
23 this article, so we're clear on what specific article
24 you're referring to?

25 A. Yes, this is an article by Lenski, Ofria,

1 Pennock, and Adami published in the year 2003. The
2 title is The Evolutionary Origin of Complex Features
3 published in the journal Nature, volume 423, pages 139
4 to 144.

5 Q. Thank you. And the authors go on to say in their
6 discussion, indeed, our experiments showed that the
7 complex feature never evolved when simpler functions
8 were not rewarded. This is not surprising to me. This
9 shows the difficulty of irreducible complexity. If you
10 do not have those closely stacked functional states, if
11 you have to change a couple things at once before you
12 get a selectable property, then I have been at pains to
13 explain, that's when Darwinian theory starts to fail,
14 not when you have things close together.

15 And to build them into your model is, again,
16 begging the question. The fact that when they do not
17 build that into their model, they run into problems that
18 complex features then don't evolve. That is exactly
19 what I would expect. I would cite this as evidence
20 supporting my own views.

21 Q. Have other scientists made similar criticisms?

22 A. Yes. A couple years ago, there was an article
23 published by two scientists named Barton and Zuidema
24 published in a journal called Current Biology. The
25 title of the article is Evolution, The Erratic Path

1 Towards Complexity.

2 And much of the article is a commentary on the
3 work by Lenski and co-workers. And if I could just read
4 a couple excerpts from that article. They make a couple
5 interesting points. The authors say, complex systems,
6 systems whose function requires many interdependent
7 parts, that is irreducible complexity systems in my
8 view, are vanishingly unlikely to arise purely by
9 chance.

10 Darwin's explanation of their origin is that
11 natural selection establishes a series of variants, each
12 of which increases fitness. This is an efficient way of
13 sifting through an enormous number of possibilities,
14 provided there is a sequence of ever-increasing fitness
15 that leads to the desired feature, close quote.

16 So that's the exact -- that's the big question.
17 Is there such a pathway, or is it, as it certainly
18 appears, that one has to make large numbers of changes
19 before one goes from a functional selectable state to a
20 second functional selectable state? And Barton and
21 Zuidema continue in their discussion.

22 They say, in Lenski's artificial organisms, the
23 mutation rate per site is quite high. So, in other
24 words, if I might make my own comment, they are using --
25 they are using factors which are not common for

1 biological organisms.

2 Now picking up with the paper again. So that
3 favorable pairs can be picked up by selection at an
4 appreciable rate. This would be unlikely in most real
5 organisms because, in these, mutation rates at each
6 locus are low. In other words, again, they are building
7 into the model exactly the features they need to get the
8 result they want.

9 But building it into your model does not show
10 that that's what exists in nature. And Barton and
11 Zuidema comment further, quote, Artificial life models
12 such as Lenski, et al's, are perhaps interesting in
13 themselves, but as biologists, we are concerned here
14 with the question of what artificial life can tell us
15 about real organisms.

16 It's -- it can be productive and it can be
17 interesting to do such studies as Lenski, et al, did.
18 But the big question is, do they tell us anything about
19 real organisms? And I am very skeptical that this study
20 does so.

21 Q. Now have you done some work yourself that's
22 somewhat similar?

23 A. Yes, indeed. A year ago, as I mentioned earlier
24 in my testimony, David Snoke and myself published a
25 paper in the journal Protein Science entitled Simulating

1 Evolution by Gene Duplication of Protein Features that
2 Require Multiple Amino Acid Residues.

3 In this, we also -- it was essentially a
4 theoretical study using computer programs to try to
5 mimic what we thought would occur in biology. But we
6 tried, as closely as possible, to mimic features of real
7 proteins and real mutation rates that the professional
8 literature led us to believe were the proper reasonable
9 values.

10 And when we used those values, the short, the
11 gist of the matter is that, once -- if there is not a
12 continuous pathway, if one has to make two or three or
13 four amino acid changes, those little changes from that
14 figure of two interacting proteins that I talked about
15 yesterday, if one has to make several changes at once,
16 then the likelihood of that occurring goes -- drops
17 sharply in the length of time, and the number of
18 organisms in a population that one would need to have
19 that goes up sharply.

20 Q. Would it be fair to say that your model is closer
21 to biological reality?

22 A. Well, I certainly think so.

23 Q. Now Dr. Miller testified that the immune system
24 is being explained by Darwinian theory. Do you agree
25 with that?

1 A. No, I do not.

2 Q. And so I'd ask you if you could explain why not?

3 A. Yes. On the next slide is a -- is the first
4 slide of Professor Miller's discussion of this topic and
5 his presentation simply showing a model of an
6 immunoglobulin protein. And here is kind of a little
7 cartoon version of the same thing, the immunoglobulin
8 protein.

9 He goes on the next slide to take an excerpt from
10 my book where in a chapter where I discussed the immune
11 system and argue that, in fact, it is not well-explained
12 by Darwinian processes but, in fact, is better explained
13 by design.

14 Q. Can you explain that Sisyphus reference?

15 A. Yeah, okay. Sisyphus. I said, Sisyphus himself
16 would pity us. That was just a literary flourish there.
17 Sisyphus is a figure from mythology who was doomed for
18 eternity to have to roll a bolder up a hill, and
19 whenever he got to the top of the hill, the bolder would
20 roll back, and he would have to start all over again.

21 This was meant to indicate frustration. And I
22 argued that Darwinian attempts at explanations would be
23 similarly frustrating.

24 Q. I just want to make a point clear. You said
25 there were two examples where those who claim that

1 irreducible complexity does not work or is not a valid
2 explanation, they use experimental evidence, and that
3 was the blood clotting system and the lac operon. How
4 does the immunity system, is that experimental evidence
5 or is that a theoretical claim?

6 A. No, this is mostly a theoretical claim. There is
7 no experimental evidence to show that natural selection
8 could have produced the immune system. And I think
9 that's a good example of the different views that people
10 with different theoretical frameworks bring to the
11 table.

12 If we could show the next slide. Professor
13 Miller shows this slide from a reference that he cited
14 by Kapitonov and Jurka, and he has titled Summary,
15 Between 1996 and 2005, each element of the transposon
16 hypothesis has been confirmed. He has this over this
17 diagram.

18 But again, as I mentioned previously, whenever
19 you see diagrams like this, we're talking about sequence
20 data, comparison of protein, sequences, or gene
21 sequences between organisms. And such data simply can't
22 speak to the question of whether random mutation and
23 natural selection produced the complex systems that
24 we're talking about.

25 So Professor Miller -- so, in my view, this data

1 does not even touch on the question. And yet Professor
2 Miller offers as compelling evidence. And one more
3 time, I view this as the difference between two people
4 with two different expectations, two different
5 theoretical frameworks, how they view the same data.

6 And I'd like to take a little bit of time to
7 explain why such studies do not impress me. And I'll do
8 so by looking at one of the papers that Professor
9 Doolittle -- I'm sorry, Professor Miller, that's his
10 name, cited in his presentation, Kapitonov and Jurka,
11 that was published this year.

12 I just want to go through, and just kind of as a
13 quick way to show why I am not persuaded by these types
14 of studies. I want to excerpt some sentences from this
15 study to show what I consider to be the speculative
16 nature of such studies.

17 For example, in this excerpt, the authors say,
18 something indicates that they may be important. This
19 may indicate. It may be encoded. It might have been
20 added. If so, it might have been derived.
21 Alternatively, it might have been derived from a
22 separate unknown transposon. It was probably lost. And
23 we have a lot more of those, one more slide at least.

24 It says, we cannot exclude the possibility. In
25 any case, the origin appears to be a culmination of

1 earlier evolutionary processes. If so, this might have
2 been altered. Again, without going into the detail of
3 the article, I just wanted to emphasize those phrases to
4 point out what I consider to be the very speculative
5 nature of such papers.

6 Here's what I view to be the problem. The
7 sequence of the proteins are there. The sequence of the
8 genes are experimentally determined. And the question
9 is, what do we make of that information? People like
10 Professor Miller and the authors of this paper working
11 from a Darwinian framework simply fit that data into
12 their framework.

13 But to me, that data does not support their
14 framework. It does not offer experimental evidence for
15 that framework. They're simply assuming a background of
16 Darwinian random mutation and natural selection and
17 explaining it -- or fitting it into that framework, but
18 they're not offering support for it.

19 Q. Dr. Behe, is there another paper that scientists
20 point to for the support that the immune system can be
21 explained by this Darwinian process?

22 A. Yes, there is. There is one more that I have to
23 discuss. Here is a recent paper, again the year 2005,
24 by Klein and Nikolaidis entitled The Descent of the
25 Antibody-Based Immune System by Gradual Evolution. And

1 on the next slide is an excerpt from the initial part of
2 their discussion where they say, quote, According to a
3 currently popular view, the Big Bang hypothesis, the
4 adaptive immune system arose suddenly, within a
5 relatively short time interval, in association with the
6 postulated two rounds of genome-wide duplications.

7 So these people, Klein and Nikolaidis, are going
8 to argue against what is the currently popular view
9 among immunologists and people who study the immune
10 system on how that system arose.

11 Q. And what is the Big Bang hypothesis that's
12 referred to here?

13 A. Well, that's kind of a label that they put on to
14 kind of indicate the fact that the immune system appears
15 in one branch of animals, the vertebrates, and any
16 obvious pre-cursors or functional parts of such a system
17 do not appear to be obvious in other branches of
18 animals.

19 So it seems like the immune system arose almost
20 complete in conjunction with the branching of
21 vertebrates from invertebrate.

22 Q. Do scientists acknowledge that or treat that as a
23 problem for Darwin's theory?

24 A. Well, in my experience, no, nobody treats such a
25 thing as a problem for Darwin's theory.

1 Q. Do you consider it a problem?

2 A. I certainly consider it a problem. But other
3 scientists who think that Darwinian evolution simply is
4 true don't consider much of anything to be a problem for
5 their theory.

6 Q. Why do you consider it a problem?

7 A. Because the -- as Darwin insisted, he insisted
8 that adaptations had to arise by numerous successive
9 slight modifications in a very gradual fashion. And
10 this seems to go against the very gradual nature of his
11 view.

12 Q. Now has this paper been held up by scientists as
13 refuting claims against intelligent design?

14 A. Yes, it has. As a matter of fact, Professor
15 Miller cited it in his expert report, although he didn't
16 refer to it in his testimony. Additionally, I attended
17 a meeting on evolution at Penn State in the summer of
18 2004 where one of the authors, Juan Kline, spoke on his
19 work, and he interpreted it in those terms.

20 Q. Now we have some quotes, I believe, from this
21 paper that you want to highlight?

22 A. Yes. Again, I want to pull out some excerpts
23 from that paper just to show you why I regard this as
24 speculative and unpersuasive. For example, they start
25 with, by saying, quote, Here, we sketch out some of the

1 changes and speculate how they may have come about. We
2 argue that the origin only appears to be sudden. They
3 talk about something as probably genuine.

4 It probably evolved. Probably would require a
5 few substitutions. It might have the potential of
6 signaling. It seems to possess. The motifs presumably
7 needed. One can imagine that a limited number. It
8 might have been relatively minor. Quote, The kind of
9 experimental molecular evolution should nevertheless
10 shed light on events that would otherwise remain
11 hopelessly in the realm of mere speculation. They're
12 talking about experiments that have yet to be done.

13 Next slide, I have even more such quotations.
14 These factors are probably genuine. Nonetheless. They
15 might have postdated. Nevertheless. Albeit. It seems.
16 This might have been. These might represent. They
17 might have been needed. This might have functioned.
18 This might have. And this might have contributed.

19 So again, this is just a shorthand way of trying
20 to convey that, when I read papers like this, I do not
21 see any support for Darwin's theory. I read them as
22 speculative and -- but nonetheless, people who already
23 do believe in Darwin's theory fit them into their own
24 framework.

25 Q. Now Dr. Miller cited numerous papers in his

1 testimony to support his claims on irreducible
2 complexity, the type III secretory system, and so forth.
3 Have you done a review of those papers and have some
4 comments on them that you prepared slides for?

5 A. Yes, I did. I went through many of the papers
6 that Professor Miller cited, as many as I could, and
7 simply, as a shorthand way of trying to indicate or
8 trying to convey why I don't regard any of them as
9 persuasive, I simply did a search for the phrases,
10 random mutation, which is abbreviated here in this
11 column, RM, and the phrase, natural selection.

12 Random mutation, of course, and natural selection
13 are the two elements of the Darwinian mechanism. That
14 is what is at issue here. And so this is, you know,
15 this is, of course, a crude and perhaps shorthand way,
16 but nonetheless, I think this illustrates why I do not
17 find any of these papers persuasive.

18 When I go through the papers that Professor
19 Miller cited on the blood clotting cascade, Semba, et
20 al, Robinson, et al, Jiang and Doolittle, there are no
21 references to those phrases, random mutation and natural
22 selection.

23 Q. Some of your indications on this slide, you have
24 0 with asterisks and some without. Is there a reason
25 for that?

1 A. Yes. The papers that have asterisks, I scanned
2 by eye. I read through them visually. Ones that do not
3 have an asterisk, I was able to do a computer search for
4 those phrases because they are on the web or in computer
5 readable form. I have a number of other such tables.

6 On the next one are references that Professor
7 Miller cited on the immune system. And again, none of
8 these references contain either those phrases, random
9 mutation and natural selection. There were a couple
10 more references on the immune system that Professor
11 Miller cited, and they didn't contain those phrases
12 either.

13 In references for the bacterial flagellum and the
14 type III secretory system, there was one paper by Hauch,
15 a review in 1998 that did use the phrase natural
16 selection. However, that phrase did not occur in the
17 body of the paper. It was in the title of one of the
18 references that Hauck listed.

19 And on the next slide, I think there are papers
20 cited by Professor Miller on common descent of
21 hemoglobin. And again, those phrases are not there. I
22 think there's another slide or two, if I'm not mistaken.
23 This is the one on what he described as molecular trees,
24 Fitch and Margoliash, from 1967. And I didn't find the
25 phrase there either. So again, this is a shorthand way

1 of showing why I actually considered these off-the-point
2 and unpersuasive.

3 Q. So all these papers that are being used to
4 provide evidence for Darwin's theory of evolution, in
5 particular, the mechanism evolution of natural
6 selection, yet they don't mention random mutation or
7 natural selection in the body of the works?

8 A. That's correct.

9 Q. Could you summarize the point then, Dr. Behe,
10 that you are making with, referring to these studies and
11 the comments you made about the speculative nature of
12 some of these studies?

13 A. Yes. Again, much of these studies, in my view,
14 are speculative. They assume a Darwinian framework.
15 They do not demonstrate it. And certainly, you know,
16 certainly scientists should be free to speculate
17 whatever they want. You know, science usually starts
18 with speculation, but it can't end with speculation.

19 And a person or, and especially a student, should
20 be able to recognize and differentiate between
21 speculation and actual data that actually supports a
22 theory.

23 Q. So it would be beneficial to point this sort of
24 feature that you just described, point that out to
25 students?

1 A. I very much think so.

2 MR. MUISE: Your Honor, we're going to be
3 moving again into another subject, and it appears to be
4 close to the time for a break.

5 THE COURT: Yeah, why don't we take a break
6 at this point. I think that makes good sense. We'll
7 break for 20 minutes at this juncture, and we'll return
8 and pick up direct examination at that point.

9 (Whereupon, a recess was taken at 10:11 a.m.
10 and proceedings reconvened at 10:36 a.m.)

11 THE COURT: All right. Mr. Muise, you may
12 continue.

13 MR. MUISE: Thank you, Your Honor.

14 BY MR. MUISE:

15 Q. Dr. Behe, Dr. Miller severely criticized Pandas
16 for its treatment of the topic of protein sequence
17 similarity. Do you agree with his assessment?

18 A. No, I don't.

19 Q. And I would ask you to explain why not?

20 A. On the next slide, we see one of Professor
21 Miller's slides, the first, I think, in his sequence
22 where he very severely criticized the book Of Pandas and
23 People for its treatment of the question of why similar
24 proteins in separate organisms have the differences in
25 their sequence that they do.

1 And on the next slide, this is again a slide from
2 Professor Miller. He reproduces a figure from Pandas
3 which shows -- it's hard to read on here -- that the
4 difference in the number of amino acids of a protein
5 called cytochrome c, which is a small protein which is
6 involved in energy metabolism and which has about 100
7 amino acids in it, the difference between that protein
8 which occurs in fish is about 13 percent.

9 About 13 amino acids differ between the fish
10 cytochrome C and frog cytochrome C; and about 13 or so
11 between bird and fish cytochrome C; and about 13 between
12 mammalian cytochrome C and fish cytochrome C. So that
13 remarkably, the proteins in these different organisms
14 all seem to have roughly the same number of differences,
15 although the differences are not the same differences,
16 but they have the same number of differences from fish
17 cytochrome C.

18 And Pandas discusses this in their text. And
19 Professor Miller -- Professor Miller takes Pandas to
20 task because he says that, in fact, this is a
21 well-studied and a problem that has been solved by
22 evolutionary theory. For example, he says, in fact,
23 these sequence differences confirm that each of these
24 organisms is equi-distant from a common ancestor, which
25 is the actual prediction of evolutionary theory.

1 He has a little tree diagram there, too. But one
2 has to realize that, in fact, Professor Miller is
3 mistaken. Evolutionary theory does not predict that.
4 Or one could say, evolutionary theory predicts that in
5 the same sense that evolutionary theory predicted that
6 the vertebrate embryos, as drawn by Haeckle, should be
7 very, very similar to it; or the prediction of
8 evolutionary theory after newer results came out, that
9 vertebrate embryos could vary by quite a bit; or the
10 prediction of evolutionary theory that the type III
11 secretory system would be a good pre-cursor for the
12 flagellum; or the prediction of evolutionary theory that
13 the flagellum -- or that the type III secretory system
14 might be derived easily from a flagellum.

15 So, in fact, what we have, I will try to make
16 clear, is an instance where experimental science comes
17 up with data, and the data is attempted to be fit into a
18 framework. But this data was not predicted by any
19 evolutionary theory.

20 Q. How was Pandas' treatment of this compared with
21 what Dr. Miller found?

22 A. In my view, Pandas' treatment of this topic is
23 actually much more accurate than Professor Miller's
24 discussion of the same topic in his testimony here.
25 Professor Miller, in his discussion, where he says that,

1 evolutionary theory predicts this remarkable amount of
2 difference, is referring to something, although he does
3 not call it such, something called the molecular clock
4 hypothesis.

5 And notice that, in fact, in Pandas, on the page
6 opposite to the figure that Professor Miller used in his
7 presentation, there is a section entitled A Molecular
8 Clock where they go through and discuss some issues with
9 it, which I will talk about later on.

10 Q. Just to be clear for the record, the diagram,
11 figure 9 that you've been referring to that Dr. Miller
12 cited in his testimony, appears on page 38 of Pandas, is
13 that correct?

14 A. Yes.

15 Q. And the discussion of the molecular clock
16 appearing on the subsequent page appears on page 39 of
17 Pandas, as indicated in this slide, is that correct?

18 A. That's correct.

19 Q. Do you have some slides and discussion as to how
20 this molecular clock problem is treated in the science
21 community?

22 A. Yes, I do, and it will probably take about 10
23 minutes or so to go through it. So please be patient.
24 But here is a cover of the Biochemistry textbook that I
25 referred to frequently here by Voet and Voet, which is

1 used in many universities and colleges across the
2 country.

3 And they have a section on the molecular clock
4 hypothesis and on cytochrome C in which they discuss
5 these issues. Let's imagine -- I'm going to try to
6 explain a molecular clock. Let's imagine that these
7 lengths of time -- these lines represent time. And down
8 at the bottom of the screen is a time -- a distant time
9 ago, and up at the top is modern time.

10 And the branches here represent events in the
11 course of life where a population of organisms split
12 into two -- split into two, and one branch went off to
13 form one group of organisms and another group went off
14 to form a different type of organisms.

15 Q. If I might just interrupt briefly. You're
16 referring to a phylogenetic tree that has vertical lines
17 that branch off to each other, and that's what you're
18 referring to the vertical lines running, two at the top
19 of the diagram, and then they branch off into different
20 sections?

21 A. That's correct. That's exactly right.

22 Q. Could you continue, please?

23 A. Yes. So, for example, at this branch, a
24 population of organisms split off that went on to become
25 plants, and at this branch, a population split off which

1 went on to become animals.

2 Now I suppose that before any split in the
3 population, the pre-cursor population organisms had a
4 cytochrome c with a certain sequence. We'll say there
5 was a hundred letters. Just think of a string of a
6 hundred letters; Z, Q, A, L, W.

7 Now, however, when we get to this branch point,
8 we have a group of organisms going off to form the
9 animals, another going off to form the plants. They no
10 longer interbreed, and so that string of a hundred
11 letters representing cytochrome c can't accumulate
12 mutations in it separately.

13 So, for example, suppose once every year or so,
14 the cytochrome c in the branch that is forming the
15 plants suffered a mutation, so that one of those letters
16 changed from what it had been. And similarly, in the
17 branch going off to form the animals, once every hundred
18 years or so, one of those letters changed into
19 something.

20 Not necessarily the same. Maybe a different one.
21 So that after a while, those two sequences would be
22 different. And suppose every hundred years, that
23 happened, one change, one change, one change, and so on.
24 After a while, you'd start to accumulate a number of
25 changes.

1 Now further suppose that along the line to
2 animals, the population of animals split into two, one
3 line leading to, say, insects, and another line leading
4 to mammals. Now you could have the same thing with the
5 cytochrome c sequence that had been mutating all along,
6 but now they split into two populations, and now these
7 two populations also begin to accumulate mutations
8 independently.

9 But notice here, they start right at the branch
10 point with the same sequence. But after, say, a hundred
11 years, this will have one difference with what it had at
12 the beginning. This one will have one difference, too.
13 And they don't necessarily have to be the same
14 difference.

15 So they'll start to accumulate differences with
16 each other between, say, the branch leading to the
17 insects and the branch leading to the mammals. Now
18 here's the point. Any sequence along this branch should
19 have accumulated the same number of sequences between
20 any sequence on this branch.

21 So that the number of differences between insects
22 and plants should be roughly the same between, as that
23 between mammals and plants. Any animal and any plants
24 should have roughly the same number of differences.
25 Whereas between subgroups of animals that have split off

1 from each other earlier than animals did from plants,
2 they will have had less time to accumulate differences
3 in their amino acid sequences. And so they will have --
4 so they will have fewer differences.

5 Q. You mean, if they split off later. You said,
6 earlier. They were split off later, correct?

7 A. Thank you. Yes, later. So Professor Miller has,
8 I believe, this sort of model in mind, which is commonly
9 -- which is a common way of thinking of these things in
10 science.

11 So the idea is that, since fish branched off from
12 those other groups of vertebrates, mammals, birds, and
13 so on, the fish, under this model, would be expected to
14 have the same number of differences in their amino acid
15 sequences between themselves and all those other
16 vertebrate groups.

17 Q. So here you have plants splitting off at the same
18 time as the insects or you have the same -- you have the
19 same connection between insects and plants as plants and
20 mammals?

21 A. That's right. So the critical point is that, the
22 difference between animals, any animal group like
23 mammals and plants and insects and plants, they should
24 have the same difference between animals and plants, no
25 matter what the subgroup of animals.

1 But between animals which branch off -- groups of
2 animals which branched off at an earlier -- or from each
3 other earlier to the current time, they would have less
4 time to accumulate differences. And I believe this is
5 what Professor Miller had in mind.

6 However, this model has some difficulties with it
7 which are well recognized and have been discussed in the
8 literature for over 40 years. For example, I said,
9 suppose every hundred years or so, a mutation occurred.
10 Okay. Well, suppose that in this branch, every hundred
11 years or so, a mutation occurred. But in this branch,
12 suppose a mutation occurred every 50 years.

13 And suppose when these split, the mutation rate
14 again changed somewhat. Now you would not expect this
15 nice, neat pattern to occur. Now you would expect a
16 jumble. It's not quite clear what one might expect.
17 And it turns out, that's a real problem because it's
18 thought that most mutations accumulate in a lineage when
19 an organism reproduces.

20 When an organism reproduces, the DNA in it has to
21 be replicated, and that gives a chance for mutations to
22 come into the DNA. But different organisms can
23 reproduce at greatly differing rates. For example, a
24 fruit fly might have a generation time of two weeks, and
25 an elephant might have a generation time of 20 years.

1 So if the number of mutations that a protein or
2 gene underwent was proportional to the number of
3 generations, you might expect a lineage with quickly
4 reproducing organisms to accumulate mutations much more
5 quickly, and the one with slowly reproducing organisms
6 to accumulate more slowly.

7 And I believe this is -- on the next slide, there
8 shows discussion from the Biochemistry textbook
9 explaining exactly that point. Let me quote from it.
10 Quote, Amino acid substitutions in a protein mostly
11 result from single base changes in the gene specifying
12 the protein. If such point mutations mainly occur as a
13 consequence of errors in the DNA duplication process,
14 then the rate at which a given protein accumulates
15 mutations would be constant with respect to numbers of
16 cell generations.

17 Not with time. With numbers of cell generations.
18 If, however, the mutations process results from a random
19 chemical degradation of DNA, then the mutation rate
20 would be constant with absolute time. So here's this
21 complication. If most mutations occur during
22 replication, you wouldn't expect this difference that we
23 see in cytochrome c.

24 If, for some reason, mutations occurred constant
25 with time, well, then you might expect that. But the

1 problem is, we know of no reason why that necessarily --
2 that has to be so, why a mutations have to -- would have
3 to occur constant in time.

4 Q. Is there a problem in addition to this
5 generational rate change?

6 A. Yes, that's one complication, but there's another
7 one as well. And that's that, this so-called molecular
8 clock seems to tick at different rates in different
9 proteins. And this is an illustration again from the
10 Biochemistry textbook that applies to this point.

11 On the bottom, the X axis, this is time. This is
12 200 million, 400 million, a billion years, and so on.
13 This is number of -- or percent amino acid sequence
14 difference. And the idea is that, here's the line for
15 cytochrome c.

16 Organisms which diverge about 200 million years
17 ago have these many sequence differences; about 400
18 million years ago, have these many, and so on. Look at
19 how nice and neat that is. However, for another
20 protein, hemoglobin, the molecular clock seems to tick
21 faster. For the same amount of time, hemoglobin has
22 maybe twice as many mutations.

23 Another region of a protein called a
24 fibrinopeptide seems to accumulate mutations extremely
25 rapidly. And a fourth protein, if you can look at the

1 bottom of the figure, it's hard to see, for something
2 called histone H4, barely accumulates any mutations at
3 all. Organisms in very widely separated categories have
4 virtually identical histone H4's.

5 Now to resolve this problem, it was postulated
6 that perhaps this has to do with the number of amino
7 acid residues in a protein that are critical for its
8 function. Perhaps in some proteins, you know, most of
9 the amino acid residues cannot be changed or it destroys
10 the function and would destroy the organism.

11 And in others, maybe some can be changed, but not
12 others. And so you can change those. And perhaps in
13 another group, almost all of them can be changed without
14 really affecting the function. And so that's an
15 interesting idea. But there are also difficulties with
16 that because, under that model, you would predict that
17 if you changed the amino acid sequence of histone H4,
18 then that should cause problems for an organism, because
19 all of its, or most of its, or practically all of its
20 amino acids are critical for function. But
21 experimentally, that is not supported, as shown on the
22 next slide.

23 Q. Is this -- so you've done work in this area with
24 the histone H4 and the molecular clock?

25 A. Yes, uh-huh. I've written this commentary in

1 1990 in a journal called Trends in Biochemical Sciences,
2 commenting on the work of somebody else who
3 experimentally took an organism called yeast into the
4 lab and altered its histone H4 and actually chopped off
5 a couple amino acids at the beginning portion of that
6 protein.

7 And when he looked, it seems that it didn't make
8 any difference to the organism. The organism grew just
9 as well without those mutations, which is surprising,
10 which is not what you would expect if all of those
11 residues were critical for the function of that protein,
12 histone H4.

13 Later on, in the year 1996, I and a student of
14 mine, Sema Agarwal, we were interested in this problem
15 of histone H4 and molecular clock, and so we
16 experimentally altered some amino acid residues into
17 protein and changed them into different amino acids,
18 with the expectation that these might destroy the
19 function of the protein. But it turned out not to.

20 These positions, these amino acids could be
21 substituted just fine, which is unexpected, and which
22 kind of complicates our interpretation of the molecular
23 clock hypothesis. So there are two complications;
24 complications upon complications.

25 One, we would expect the number of mutations to

1 accumulate with generation time, but it seems to
2 accumulate, for some unknown reason, with absolute time.
3 And the second is that, proteins accumulate mutations at
4 different rates. We would expect that it would have to
5 do with how vulnerable they are to mutations, and
6 mutations might destroy the function of one protein that
7 evolved slowly, but that is not experimentally
8 supported.

9 Q. Now has this problem been discussed in the
10 scientific literature?

11 A. Yes, this has been continuously discussed ever
12 since the idea of the molecular clock hypothesis was
13 first proposed in the early 1960's by two men named
14 Emile Zuckerkandl and Linus Pauling. And here are a
15 couple of papers which deal with the difficulties of the
16 molecular clock hypothesis.

17 Here's a recent one, Gillooly, et al, published
18 in the Proceedings in the National Academy of Sciences,
19 entitled The Rate of DNA Evolution, Effects of Body Size
20 and Temperature on the Molecular Clock. In this
21 publication, they say that, in fact, the size of an
22 organism and temperature can affect how fast or how slow
23 this clock might tick.

24 Francisco Ayala has written on this frequently.
25 Here's one from 1997. And I should say, Francisco Ayala

1 is a very prominent evolutionary biologist. He wrote an
2 article in 1997 entitled Vagaries of the Molecular
3 Clock. And I think the title gets across the idea that
4 there are questions with this hypothesis.

5 And in 1993, a researcher named Tomoka Ohta
6 published an article in the Proceedings of the National
7 Academy of Sciences entitled An Examination of the
8 Generation-time Effect on Molecular Evolution in which
9 she considers exactly that complication that the
10 textbook Voet and Voet pointed out, this generation-time
11 effect.

12 You know, why shouldn't organisms that reproduce
13 more quickly accumulate more mutations. I have another
14 slide just from one more recent paper. This paper by
15 Drummond, et al, is entitled Why Highly Expressed
16 Proteins Evolve Slowly. And it's referring to the
17 sequence evolution that I've been discussing.

18 It was published in the Proceedings of the
19 National Academy of Sciences, and this was from an
20 online version. This is so recent that I don't think it
21 has yet appeared in print. The point I want to make
22 with this is that, these people treat this question as a
23 currently live question.

24 They start off by saying, a central problem in
25 molecular evolution is why proteins evolve at different

1 rates. So that question I was trying to illustrate with
2 histone H4, why does one protein tick faster and another
3 one tick more slowly, that's still -- that is still
4 unknown.

5 And I think I will skip the rest of this slide
6 and go to the next slide and just point out a couple
7 words here. Drummond, et al, say, Surprisingly, the
8 best indicator of a protein's relative evolutionary rate
9 is the expression level of the encoding gene.

10 The only point I want to make with this is that,
11 they are reporting what is a surprise, what was not
12 expected, which was not known, you know, 40 years ago,
13 which has only been seen relatively recently. And they
14 say, quote, We introduce a previously unexplored
15 hypothesis, close quote.

16 And the point I want to emphasize is that, here
17 in this paper published, you know, weeks ago, that they
18 are exploring new hypotheses to try to understand why
19 proteins have the sequences that they do.

20 Q. So in summary, this protein sequence, the fact
21 that the equi-distant from a common ancestor is not what
22 evolutionary theory would actually predict?

23 A. That's right. Evolutionary theory makes no firm
24 prediction about this anymore than it makes a firm
25 prediction about the structure of vertebrate embryos.

1 Q. It's a common understood problem that biologists
2 are trying to resolve at this point?

3 A. Yes, within the community of scientists who work
4 on this. People have been working on it for decades.

5 Q. Is this a problem that an American Biology
6 teacher should be aware of?

7 A. Yes, an American Biology teacher should be aware
8 of it, because an article on this very topic was
9 published in the magazine, American Biology Teacher, a
10 couple years ago, which is put out by the National
11 Association of Biology Teachers.

12 And the article is entitled Current Status of the
13 Molecular Clock Hypothesis. And one of the first --
14 this is a red arrow that I added to the figure. One of
15 the first subsections of the article is entitled How
16 Valid is the Molecular Clock Hypothesis? And if you'll
17 advance to the next slide, let me just read the last
18 line from the paper.

19 The author says, The validity of a molecular
20 clock, except in closely related species, still remains
21 controversial. So the point is that, extrapolating
22 across wide biological distances, such as from fish to
23 other vertebrates, that is controversial.

24 Maybe similar species, species of mice or some
25 such thing, okay. But when you try to extrapolate

1 further, the model is quite controversial.

2 Q. How does Pandas then address this issue?

3 A. Well, I have here the section from Pandas
4 entitled The Molecular Clock where they discuss exactly
5 all these things. They discuss the molecular clock, the
6 standard molecular clock model, the naive molecular
7 clock model, and then they discuss complications with
8 it.

9 Let me just read this section from Pandas on the
10 molecular clock. They write, quote, Some scientists
11 have suggested that the idea of a molecular clock solves
12 the mystery. The explanation they advance is that there
13 is a uniform rate of mutation over time, so quite
14 naturally, species that branched off from a common
15 ancestor at the same time in the past will now have the
16 same degree of divergence in their molecular sequences.

17 There are some serious shortcomings, however,
18 with this explanation. First, mutation rates are
19 thought to relate to generation times, with the mutation
20 rates for various molecules being the same for each
21 generation.

22 The problem comes when one compares two species
23 of the same taxon, say two mammals, with very different
24 generation times. Mice, for instance, go through four
25 to five reproductive cycles a year. The number of

1 mutations, therefore, would be dramatically higher than,
2 say, those of an elephant.

3 Thus, they should not reflect similar percent
4 sequence divergences for comparable proteins. Besides
5 that, the rates of mutations are different for different
6 proteins even of the same species. That means that, for
7 the molecular clock idea to be correct, there must be
8 not one molecular clock, but thousands.

9 So let me point out here that, in this section,
10 Pandas describes the simple molecular clock idea that
11 was proposed 40 years ago by Zuckerkandl and Pauling,
12 and then talks about the two complications for the
13 model, which are common knowledge and are taught in
14 basic science texts that deal with this issue, the
15 generation time problem and the fact that different
16 proteins accumulate mutations at different rates.

17 And as I have shown from the literature I just
18 cited, that continue to be live issues in the scientific
19 community.

20 Q. In that section you read from on the molecular
21 clock from Pandas are found on page 39, is that correct?

22 A. Yes, that's correct.

23 Q. Again, returning to that slide that Dr. Miller
24 presented in his testimony?

25 A. Yes. I just wanted to go back to that slide

1 where Dr. Miller says -- again, I should say that, in
2 his testimony, which I attended, he, you know,
3 excoriated Pandas on this point. And he says -- on his
4 slide, he says, in fact, the information we have
5 confirms that each of these organisms is equidistant
6 from a common ancestor, which is the actual prediction
7 of evolutionary theory.

8 And that's simply is incorrect. And in my view,
9 Pandas is treating problems that Professor Miller,
10 treating real live problems that Professor Miller shows
11 no signs of being aware of. So I think a student
12 reading this section would actually get a better
13 appreciation for this subject than otherwise.

14 Q. Dr. Behe, in Dr. Miller's testimony, he also
15 criticized another example found in Pandas that had a
16 message such as, quote, John loves Mary, written on the
17 beach, would be a sure sign of intelligence.

18 He claimed that any philosopher, any logician
19 would spot the mistake in logic, because we know a human
20 made that message, and probably made it with a stick,
21 because we have seen such things happen in our own
22 experience. Do you agree with this reasoning?

23 A. No, I disagree with Professor Miller's reasoning.

24 Q. And if I can just say, the example that John
25 loves Mary, and we have a slide up, that's on page 7 of

1 Pandas, correct?

2 A. Yes, that's right.

3 Q. Again, could you explain why you disagree with
4 this reasoning?

5 A. Yes. The inference from the -- the inference
6 from the existence of designed objects in the -- in our
7 world of experience to the conclusion of design in life
8 is an example of an inductive inference. And I think I
9 explained earlier that, in an inductive inference, one
10 always infers from examples of what we know to examples
11 of what we don't know.

12 And the strength of the inference depends on
13 similarities between the, between the inference in
14 relevant properties. For example, in the Big Bang
15 hypothesis, scientists extrapolated, or used inductive
16 reasoning of their knowledge of explosions from our
17 everyday world from things like fireworks and canon
18 balls and so on.

19 They extrapolated from their experience that the
20 motion of objects away from each other bespeaks an
21 explosion. They extrapolated from our common everyday
22 experience to something that nobody had ever seen
23 before, an entirely new idea, that the universe itself
24 began in something like a giant explosion.

25 Nonetheless, they were confident that this was a

1 good idea because they thought the relevant property,
2 the parts moving rapidly away from each other, was what
3 we understand from an explosion. And that's how science
4 often reasons.

5 In the same way, the purposeful arrangement of
6 parts in our everyday experience bespeaks design.
7 Pandas is exactly right, that if we saw such a message
8 on the beach, we could conclude that it had been
9 designed. And William Paley is exactly right, that if
10 we stumbled across a watch in a field, that we would
11 conclude that it was designed, because in each case
12 there is this strong appearance of design from the
13 purposeful arrangement of parts.

14 Now we have found purposeful arrangement of parts
15 in an area where we didn't expect to, in the very
16 cellular and molecular foundation of life, in the cell.
17 The cell again was not understood in Darwin's day. And
18 it is much better understood now. And from the new
19 information we have, again, we see this purposeful
20 arrangement of parts, and it's -- by inductive
21 reasoning, we can apply our knowledge of what we see in
22 our everyday world to a different, completely different
23 realm.

24 And so that sort of inference has been done in
25 science throughout the history of science, and it's a

1 completely valid inference for Pandas to make.

2 Q. Now we've heard some testimony throughout the
3 course of this trial of a program called SETI, S-E-T-I,
4 a project, I believe, that stands for the search for
5 extraterrestrial intelligence?

6 A. Yes.

7 Q. Are you familiar with that project?

8 A. Yes, I am.

9 Q. Whose project is that?

10 A. The search for extraterrestrial intelligence is a
11 project that was, for a while, was sponsored by the
12 federal government. It involved scientists scanning the
13 skies with detectors to see if they could detect some
14 electromagnetic signal that might point to intelligence.

15 Q. Is there a comparison with that project to the
16 discussion you had in here with the John loves Mary on
17 the beach?

18 A. Yes. Again, if they detected something that
19 seemed to have a purposeful arrangement of parts, if
20 they saw something that bespoke a message, then even
21 though we have had no experience with other entities
22 from off the Earth trying to send us a message,
23 nonetheless, we could still be confident that an
24 intelligent agent had designed such a message.

25 And again, whenever we see John -- things like

1 John loves Mary, we can be confident of that. And when
2 we see the purposeful arrangement of parts in the cell,
3 the argument is that, we can be confident of that, that
4 that bespeaks design as well.

5 Q. I want to bring this discussion somewhat down to
6 the molecular level, and ask you whether or not new
7 genetic information can be generated by Darwinian
8 processes. And I want to be more specific and ask
9 whether new genetic information can be generated by
10 known processes such as gene duplication and exon
11 shuffling?

12 A. Well, that's a topic about which you have to be
13 very careful and make distinctions.

14 Q. Okay. Let's start with the gene duplication. If
15 you could explain what that is in the context of
16 generating new genetic information?

17 A. Well, gene duplication is a process whereby a
18 segment of DNA gets copied twice or gets duplicated and
19 replicated so that where one gene was present before, a
20 second copy of the exact same gene is now present in the
21 genome of an organism. Or sometimes larger segments can
22 be duplicated, so you can have multiple copies of
23 multiple genes.

24 Q. Are you saying, duplication, like photocopying,
25 is just making another copy of the gene that was

1 originally existing?

2 A. Yeah, that's a good point. It's important to be
3 aware that gene duplication means that you simply have a
4 copy of the old gene. You have not done anything new.
5 You've just taken the same gene and copied it twice. So
6 it would be like, like photocopying a page. And now you
7 have two pages, but it's just a copy of the first one,
8 it's not something fundamentally new.

9 It would be like saying, the example of Pandas
10 here with John loves Mary. If you walked down the sand
11 another five yards or something, and you came across
12 another message that says, John loves Mary, well, that's
13 interesting, but you don't have anything fundamentally
14 new.

15 Q. Can there be variations though in the duplication
16 of those genes?

17 A. Well, once a gene has duplicated, then the idea
18 goes that, perhaps one of those two copies can continue
19 to perform the function that the single copy gene
20 performed before the duplication, and the other one is
21 sort of a spare copy.

22 Now it's available to perhaps undergo mutation,
23 and mutation accumulate changes, and perhaps Darwinian
24 theory postulates. Perhaps it can go on to develop
25 brand new properties.

1 Q. Does this generate new information? And if you
2 use that John loves Mary example to help explain
3 perhaps?

4 A. Well, again, you have to be careful. Nobody
5 disputes that random mutation and natural selection can
6 do some things, can make some small changes in
7 pre-existing systems. The dispute is over whether that
8 explains large complex functional systems.

9 And to leave the world of proteins for a second,
10 to look at John loves Mary, suppose we're looking at the
11 spare copy, and the first copy was continuing to fulfill
12 the function of conveying that information. Well, you
13 know, suppose you changed a letter. Suppose you changed
14 the final n in the word John to some other, some other
15 letter, like r. That would not spell a name in the
16 English language.

17 So that's kind of an analogy to saying that, you
18 might lose the function of the message in the terms. In
19 the terms of protein, the protein might no longer be
20 functional. But you might get to closeby. You might
21 get to closeby messages. For example, if you deleted
22 the r and the y from the end of Mary, you might get to
23 John loves Ma, or some such thing. But you're not going
24 to get anything radically different from that.

25 Q. So you are operating with the copy. The copy is

1 operating with those same letters, the John loves Mary,
2 or some variation or deletions of that subset?

3 A. That's right. A copy is a copy. It's
4 essentially the same thing. And now the big problem
5 that Darwinian processes face is, now what do you do?
6 How do you generate a new complex function?

7 Q. And that's with gene duplication that we just
8 talked about. Could you explain a little bit about exon
9 shuffling in the context of generating new complex
10 information?

11 A. Yes, exon shuffling is a little bit more
12 involved. It turns out that the gene for a protein can
13 contain regions of DNA that actually code for regions of
14 a protein interrupted by regions of DNA that don't code
15 for regions of a protein. And the regions that code for
16 the part of the protein are called exons.

17 Now it turns out that, in cellular processes,
18 similar to gene duplication and other processes, too,
19 one can duplicate separate exons and sometimes transfer
20 them to different places in the genome and other such
21 processes. But to make it more understandable, we can
22 go back to the analogy of John loves Mary.

23 And in this sense, exon shuffling might be
24 expected to generate something like, instead of John
25 loves Mary, perhaps Mary loves John, or John Mary loves,

1 or something like that. But again, it's kind of a
2 mixture of pre-existing properties, and we're not
3 generating something fundamentally new.

4 Q. So, for example, you couldn't generate Brad loves
5 Jen from exon shuffling using your beach example?

6 A. No, I hope not.

7 Q. Do these concepts, particularly gene duplication,
8 exon shuffling, do they have any impact on the concept
9 of irreducible complexity that you've been discussing
10 quite a bit throughout your testimony?

11 A. Yes. In fact, there is an important point to
12 recognize here. Russell Doolittle knew all about the
13 processes of gene duplication and exon shuffling. And
14 as a matter of fact, in the blood clotting cascade, many
15 proteins look similar to each other, and they're often
16 times pointed to as examples of exon shuffling.

17 But nonetheless, that knowledge did not allow him
18 to explain how the blood clotting system might have
19 arisen. Again, these are sequence comparisons. And
20 such information simply does not speak to the question
21 of random mutation and natural selection being able to
22 build complex new biochemical structures.

23 In the same way, the people who are investigating
24 the type III secretory system and the bacterial
25 flagellum know all about gene duplication and exon

1 shuffling. And nonetheless, that information has not
2 allowed them to explain the origin of either of those
3 structures.

4 So those are interesting processes. And people
5 who are convinced of Darwinian theory include those
6 processes in their theory, but they do not explain --
7 they do not explain where new complex systems come from.
8 And it's an example of somebody accommodating this
9 information to an existing theory rather than getting
10 information that actually experimentally supports the
11 theory.

12 Q. So can random mutation and natural selection
13 generate new information?

14 A. Well, again, that's -- you have to be careful.
15 You can make small changes in pre-existing systems. And
16 that's clearly the case. One can clearly do that. But
17 there has been no demonstration to show that such
18 processes can give rise to new complex systems such as
19 we've been suggesting. And there are many reasons to
20 think that it would be extremely difficult to do so.

21 Q. Have you prepared some slides with a couple --
22 several quotes that make this point?

23 A. Yes, I do. This first one is an excerpt from a
24 paper from John Maynard Smith, which I spoke about
25 earlier, from 1970 entitled Natural Selection and the

1 Concept of a Protein Space. Let me read the first
2 excerpt.

3 Quote, It follows that if evolution by natural
4 selection is to occur, functional proteins must form a
5 continuous network which can be traversed by unit
6 mutational steps without passing through nonfunctional
7 intermediates, close quote. Again, let me explain.

8 If you can remember the figure of two proteins
9 binding to each other that I showed in -- I showed
10 yesterday, he is speaking of unit mutational steps in
11 terms of one of those interactions, maybe a plus charge
12 and a minus charge or a hydrophobic group and another
13 hydrophobic group.

14 And so to get two proteins to -- or proteins to
15 start change into something new and different with
16 different properties, each one of those changes would
17 have to be a beneficial one, or at least not cause any
18 difficulties for the problem. And actually, seeing how
19 that could happen is extremely difficult.

20 And continuing on this slide. I'm sorry. Could
21 you back up one slide? Thank you. The bottom part of
22 the quotation, he says, quote, An increase in the number
23 of different genes in a single organism presumably
24 occurs by the duplication of an already existing gene
25 followed by divergency. So here, he's kind of

1 describing the standard scenario which -- scenario,
2 which is standard in Darwinian thinking, that one has
3 gene duplication and then divergence of the sequence of
4 a gene, and that gives a brand new interesting and
5 complex protein.

6 But notice that I, of course, underlined and
7 bolded the word presumably. Well, presumably, you know,
8 is a presumption. And it may be true, and it may not.
9 But presumptions are not evidence. And so in order to
10 support this idea, one needs more than the presumption
11 that it occurs.

12 Q. Do you have another citation to a science text?

13 A. Yes, I do. Here's an excerpt from an article by
14 a man named Alan Orr, who is an evolutionary biologist
15 at the University of Rochester. And again, this speaks
16 to the same consideration, that you have to be able to
17 have a pathway that step by tiny step could lead from
18 one functional protein to another.

19 He says, quote, Given realistically low mutation
20 rates, double mutants will be so rare that adaptation is
21 essentially constrained to surveying, and substituting,
22 one mutational step neighbors. Thus, if a double mutant
23 sequence is favorable, but all single amino acid mutants
24 are deleterious, adaptation will generally not proceed.

25 Again, this makes the point that, if you only

1 need to change one little step, Darwinian evolution
2 works fine. But if you need to change two things before
3 you get to an improved function, the probability of
4 Darwinian processes drops off dramatically.

5 If you need three things, it drops off, you know,
6 even more dramatically. And nonetheless, as I showed in
7 that figure of interacting proteins, even to get two
8 proteins to stick together, multiple groups are
9 involved.

10 Q. Did you write about something similar in a paper?

11 A. Yes. The paper that I published with David Snoke
12 last year speaks exactly to this topic. It's entitled
13 Simulating Evidence by Gene Duplication of Protein
14 Features that Require Multiple Amino Acid Residues.

15 And in this theoretical study, we showed that,
16 again, if you need one change, that's certainly doable.
17 If you need two amino acid changes before you get a
18 selectable function, the likelihood of that drops
19 considerably. Three or more, now you're really in the
20 very, very improbably range. So again, gene duplication
21 is not the answer that it's often touted to be.

22 Q. Can you make an analogy here at all to -- you
23 talked about Maxwell and the ether theory?

24 A. Yes. When Darwinian -- adherence to Darwinian
25 theory, when they view that there are similar genes in

1 different -- in the same organism, and they infer a
2 process of gene duplication, it is simply their
3 theoretical framework, which is saying, such a process
4 must be important in generating new and complex
5 structures.

6 That has not been demonstrated. Just like James
7 Clerk Maxwell knew that light was a wave and inferred
8 from his theory that there must be an ether, modern
9 Darwinists infer from something we know, the existence
10 of gene copies to an unproved role of such a process in
11 generating complex biochemical systems.

12 Q. Now Dr. Miller says that Pandas necessarily
13 rejects common descent, and points to a figure -- I
14 believe it was 4.4 on page 99 -- showing separate lines
15 representing categories of animals rather than a
16 branching tree. Do you regard that as ruling out common
17 descent?

18 A. No, I don't. And here's a figure that I made up
19 in the upper right-hand corner. It's figure 4.4 from
20 Pandas, which is the figure that Professor Miller
21 showed, which shows straight lines instead of a
22 branching tree, which is the traditional representation
23 of how -- of the fossil record.

24 Nonetheless, here I regard this as simply trying
25 to describe the data without a theoretical framework,

1 without the branched lines in between. One has to
2 realize that these lines do not occur in the fossil
3 record. These are theoretical constructs.

4 And how one groups things together is theory
5 building rather than data itself. I viewed this as
6 Pandas trying to describe the data without the framework
7 of the existing theory. And I might add that, this was
8 figure 4.4. And earlier, a couple pages earlier, Pandas
9 describes the traditional interpretation of the fossil
10 record in terms of a branching tree.

11 And in this section, section 96 through 100, the
12 meaning of gaps in the fossil record, Pandas describes
13 the traditional tree diagram for the fossil record, and
14 then points to statements by biologists, saying that
15 there seem to be difficulties in this sort of
16 representation, and then goes on to discuss what
17 interpretations, what ideas have been offered to try to
18 account for the form of the fossil record.

19 Pandas writes, Several interpretations have been
20 offered to resolve this problem. That is, that the tree
21 of life doesn't seem to be as continuous as one might
22 expect. Number 1, they say, imperfect record. That is,
23 maybe not all organisms left representative of
24 fossilized specimens. Number 2, incomplete search. And
25 that is, maybe we simply haven't looked in the right

1 places or looked in all the places on the Earth, and
2 maybe when we do, then we will find what we expect to be
3 there.

4 Number 3, what they call jerky process, or which
5 has been called punctuated equilibrium, which was an
6 idea advanced by Steven J. Gould and Niles Eldredge in
7 the 1970's, whereby it said that the mode or the tempo
8 of evolution is one in which a species or a branch of
9 life stays pretty much constant for a long period of
10 time, and then within a relatively short period of time,
11 large changes occur.

12 And then fourth, they say, well, perhaps -- they
13 suggest something called the sudden appearance or face
14 value interpretation, saying that, well, maybe if we see
15 the sudden appearance of some feature or organism in the
16 fossil record, then that, in fact, might be what
17 happened.

18 Nonetheless, as I say, they discuss all of these
19 possibilities, including the standard interpretation.
20 And at the end of the section, they write that,
21 scientists should not accept the face value
22 interpretation of the fossil record without also
23 exploring the other possibilities, and even then, only
24 if the evidence continues to support it.

25 So as I read this, Pandas is telling students

1 that they should follow the data where the data lead.
2 And if the data lead from this model to another model,
3 or from that model to a second model, then a scientific
4 attitude toward the problem is to follow the data, where
5 the data go.

6 Q. Dr. Behe, does intelligent design necessarily
7 rule out common descent?

8 A. No, it certainly does not.

9 Q. Now we've heard testimony from several witnesses
10 claiming that the theory of evolution is no different
11 than, say, the germ theory of disease, so there's no
12 reason to pay any special attention to it. Do you agree
13 with that?

14 A. No, I disagree.

15 Q. And why?

16 A. Well, in a number of ways, evolutionary theory is
17 unique. It's been my experience that students have a
18 number of misconceptions about the theory. They confuse
19 facts with theoretical interpretations. They do not
20 make distinctions between the components of evolutionary
21 theory.

22 And perhaps, most strikingly, a number of people
23 have made very strong extra-scientific claims for the
24 implications of evolutionary theory.

25 Q. Now I just want to return to something you had

1 said about your experience with students. You testified
2 that you teach a course called popular arguments on
3 evolution, is that correct?

4 A. Yes, that's right.

5 Q. And you've been teaching that for 12 years?

6 A. Roughly, yes.

7 Q. Now are there some standard misconceptions that
8 you can point to about the theory of evolution that you
9 find your students bringing to the class?

10 A. Yes. In my experience, a number of students come
11 in thinking that, in fact, evolution is completely true;
12 that is, they don't make a distinction between fact and
13 theory, they don't think it will be falsified, or they
14 don't think there's a possibility of it being falsified.

15 They also confuse various components of
16 evolutionary theory. For example, you can ask a
17 student, you know, why they think Darwinian evolution is
18 correct? And they'll say, you know, because, you know,
19 because of the dinosaurs. And they're mistaking change
20 over time with the question of natural selection. And
21 they will assume that the existence of animals in the
22 past necessarily means that animals in the present were
23 derived from them by random mutation and natural
24 selection.

25 Oftentimes also, students think that utterly

1 unsolved problems, such as the origin of life, have, in
2 fact, been solved by science. I had students tell me
3 that, gee, it's true, right, that science has shown
4 genes being produced in origin of life experiments. So
5 in my experience, students bring a number of
6 misconceptions to this issue.

7 Q. One of the first ones you indicated is that they
8 believe that Darwin's theory of evolution is a fact as
9 opposed to a scientific theory?

10 A. That's right.

11 Q. Does intelligent design seek to address some of
12 these misconceptions?

13 A. Yes. Yes, it does. One way is -- one way to
14 address the problem of students not understanding that
15 the distinction between fact and theory is to at least
16 have at least one more theoretical framework in which to
17 treat facts.

18 If a student has only one theory and a group of
19 facts to think of, it's extremely difficult to
20 distinguish what is theory and what is fact. The little
21 lines connecting various points on, say, a protein
22 sequence comparison are theory, but students can often
23 confuse them, confuse them to be facts.

24 Q. Do you believe these students will be better
25 prepared if they had learned that Darwin's theory of

1 evolution was not a fact and that gaps and problems
2 existed within this theory?

3 A. Yes, I certainly do. They would see that, in
4 fact, if you can look at the data in a couple ways, then
5 they'll more easily distinguish data from interpretation
6 or from theory. And if they are aware that there are
7 problems in a theory, then perhaps they won't expect --
8 they won't, again, confuse it with a fact, they'll
9 understand that there are some problems that are
10 unresolved.

11 Q. Now you made some indication previously in your
12 answer to my question that there are claims made about
13 the theory that go beyond biology, is that true?

14 A. Yes, that's certainly true.

15 Q. And do you have some slides to demonstrate some
16 of those examples?

17 A. Yes, I have a couple of slides, four slides over
18 -- that point to this. For example, in the high school
19 textbook Biology, which was written by Professor Kenneth
20 Miller and his co-author, Joseph Levine, this is the
21 1995 version, I think, the third edition, in a section
22 entitled The Significance of Evolutionary Theory, the
23 authors write, quote, The influence of evolutionary
24 thought extends far beyond biology. Philosopher J.
25 Collins has written that, quote, there are no living

1 sciences, human attitudes, or institutional powers that
2 remain unaffected by the ideas released by Darwin's
3 work, close quote.

4 In another example of the implications, the
5 profound implications beyond biology that some people
6 see for Darwin's theory, there's a section in his book,
7 Finding Darwin's God, A Scientist's Search for Common
8 Ground Between God and Evolution, where Dr. Miller
9 writes that, quote, God made the world today contingent
10 upon the events of the past. He made our choices
11 matter, our actions genuine, our lives important. In
12 the final analysis, He used evolution as the tool to set
13 us free.

14 So here is a scientific theory which is being
15 used to support the idea that we are free, we are free,
16 in apparently some metaphysical sense, because of the
17 work of Darwin. In another example -- it's just that --
18 for example, the expert, Professor John Hauck, the
19 theologian from Georgetown University, has written a
20 number of books, including God After Darwin, a Theology
21 of Evolution.

22 Further example, in -- the evolutionary
23 biologist, Richard Dawkins, in his book, The Blind
24 Watchmaker, writes, Darwin made it possible to be an
25 intellectually-fulfilled atheist.

1 If I could have the next slide. Thank you. The
2 Darwinian philosopher, Daniel Dennett, who's at Tufts
3 University, has described Darwinism as a universal acid
4 that destroys our most cherished beliefs. And he says,
5 quote, Darwin's idea had been born as an answer to
6 questions in biology, but it threatened to leak out,
7 offering answers, welcome or not, to questions in
8 cosmology, going in one direction, and psychology, going
9 in the other direction.

10 If the cause of design in biology could be a
11 mindless, algorithmic process of evolution, why couldn't
12 that whole process itself be the whole product of
13 evolution, and so forth, all the way down? And if
14 mindless evolution could account for the breathtakingly
15 clever artifacts of the biosphere, how could the
16 products of our own real, quote, unquote, minds be
17 exempt from an evolutionary explanation? Darwin's idea
18 thus also threatened to spread all the way up,
19 dissolving the illusion of our own authorship, our own
20 divine spark of creativity and understanding.

21 So again, Professor Dennett sees implications for
22 Darwin's theory that are profound and that extend well
23 beyond biology. Another philosopher by the name of Alex
24 Rosenberg, who's at Duke University, published an
25 article a few years ago in the journal Biology and

1 Philosophy that, quote, No one has expressed the
2 destructive power of Darwinian theory more effectively
3 than Daniel Dennett. Others have recognized that the
4 theory of evolution offers us a universal acid, but
5 Dennett, bless his heart, coined the term.

6 In short, it, that is Darwin's idea, has made
7 Darwinians into metaphysical Nihilists denying that
8 there is any meaning or purpose to the universe, close
9 quote. So again, a number of philosophers, a number of
10 scientists, and so on, see very, very profound
11 implications in Darwin's theory.

12 Two more quotations on this last slide on this
13 topic. Larry Arnhart is a professor of political
14 science at Northern Illinois University. He wrote a
15 book entitled Darwinian Natural Right, The Biological
16 Ethics of Human Nature. And in it, he writes -- and in
17 it, he writes the following, that, quote, Darwinian
18 biology sustains conservative social thought by showing
19 how the human capacity for spontaneous order arises from
20 social instincts and a moral sense shaped by natural
21 selection in human evolutionary history.

22 So let me emphasize that he sees implications for
23 politics from Darwin's theory. And the same -- and a
24 Princeton University philosopher by the name of Peter
25 Singer has written a book entitled A Darwinian Left,

1 Politics, Evolution, and Cooperation. And in it, he
2 writes that we should try to incorporate a Darwinian
3 ethic of cooperation into our political thought.

4 So the gist of Professor Singer's book is that,
5 Darwinian ideas support a liberal political outlook.
6 And he argues for that. So, again, these -- all of
7 these people see profound implications for Darwin's
8 theory well far beyond biology.

9 Q. These are non-scientific claims, correct?

10 A. Yes, that's correct.

11 Q. Have you come across any similar claims made
12 about, say, the germ theory of disease?

13 A. I have never seen the germ theory of disease
14 argued to say how we should conduct our political life.

15 Q. How about atomic theory?

16 A. I have never seen atomic theory used in such
17 profound senses either. So my point then is that, it is
18 perfectly rationale to treat a scientific theory, which
19 so many people have claimed such profound implications
20 for, to treat it differently from other scientific
21 theories for which such far-reaching implications have
22 not been claimed.

23 It might be very important, and I think a school
24 district would be very justified to say that, since this
25 particular theory seems to reach far beyond its

1 providence, then we should take particular care in
2 explaining to our students exactly what the data is for
3 this theory, exactly what is the difference between
4 theory and fact, exactly what is the difference between
5 theory and interpretation. And so I think such an
6 action would be justified.

7 Q. Sir, I want to ask you some questions about
8 creationism as it relates to intelligent design. First
9 of all, let me ask you, does creationism have a popular
10 meaning or is there a popular understanding of that
11 term?

12 A. Well, again, you have to be careful, because many
13 words in these discussions can have multiple meanings.
14 And if you're not very careful about your definitions,
15 you'll easily become confused.

16 Creationism -- creationist has sometimes been
17 used, as John Maddox, the editor of Nature, used it,
18 simply to mean somebody who thinks that nature was begun
19 by a supernatural act, by God, and the laws of nature
20 perhaps were made of God, and unfolded from there
21 nonetheless.

22 Q. That would be similar to Dr. Miller's view
23 towards evolution that he had written in his book
24 Finding Darwin's God?

25 A. Yes, that seems to be consistent with what he

1 wrote. But nonetheless, in the popular useage,
2 creationism means -- creationist means somebody who
3 adheres to the literal interpretation of the first
4 several books -- or first several chapters of the Book
5 of Genesis in the Bible, somebody who thinks that the
6 Earth is relatively young, on the order of, say, 10,000
7 years, that the major groups of plants and animals and
8 organisms were created ex-nihilo in a supernatural acts
9 by a supernatural being, God, that there was a large
10 worldwide flood which is responsible for major features
11 of geology, and so on.

12 Q. Now we've heard different terms; young-earth
13 creationism, old-earth creationism, and special
14 creationism. And you have familiarity with those terms,
15 is that correct?

16 A. Yes, that's right.

17 Q. Is intelligent design creationism, whether you
18 call it young-earth creationism, old-earth creationism,
19 or special creationism?

20 A. No, it is not.

21 Q. And why not?

22 A. Creation -- creationism is a theological concept,
23 but intelligent design is a scientific theory which
24 relies exclusively on the observable, physical,
25 empirical evidence of nature plus logical inferences.

1 It is a scientific idea.

2 Q. Is it special creationism?

3 A. No, it is not special creationism.

4 Q. Again, why not?

5 A. Again, for the same reason. Creation is a
6 theological religious concept. And intelligent design
7 is a scientific idea, which is based exclusively on the
8 physical, observable evidence plus logical processes.

9 Q. Dr. Miller has made a claim that if the bacterial
10 flagellum, for example, was designed, then it had to be
11 created, and is, therefore, special creationism. Is
12 that accurate?

13 A. No, that is inaccurate. The reason it's --
14 again, creation is a theological concept. It is a
15 religious concept. But intelligent design is a
16 completely scientific concept which supports itself by
17 pointing to observable, physical, empirical facts about
18 the world, about life, and makes logical inferences from
19 them.

20 Q. Does intelligent design require that the
21 bacterial flagellum, for example, instantaneously appear
22 from nothing?

23 A. No, it does not.

24 Q. Why not?

25 A. Because intelligent design focuses exclusively on

1 the deduction of design from the purposeful arrangement
2 of parts. And it says nothing directly about how the
3 design was effected, whether it was done quickly, or
4 slowly, or whatever. So it has nothing to say about
5 that.

6 Q. Could the bacterial flagellum have been designed
7 over time?

8 A. Yes, it could.

9 Q. Does intelligent design require ex-nihilo
10 creation?

11 A. No, it does not.

12 Q. Why not?

13 A. Because again, the term ex-nihilo creation is a
14 theological concept, a religious concept. And
15 intelligent design is a scientific idea that relies on
16 observable facts about nature plus logical inferences.

17 Q. Is there, again, an analogy you can make here to
18 the Big Bang theory?

19 A. Yes. Yes, there is. Again, many people,
20 including many scientists, saw in the Big Bang theory
21 something that had theological implications, maybe this,
22 this Big Bang was ex-nihilo creation by a supernatural
23 being. And many people who saw that didn't like that.
24 Nonetheless, the Big Bang theory itself is an utterly
25 scientific theory because it relies on observations,

1 physical observations, empirical observations about
2 nature, and reasons from those observations using
3 logical processes.

4 Q. Is intelligent design a religious belief?

5 A. No, it isn't.

6 Q. Why not?

7 A. Intelligent design requires no tenet of any
8 particular religion, no tenet of any general religion.
9 It does not rely on religious texts. It does not rely
10 on messages from religious leaders or any such thing.
11 The exclusive concern of intelligent design is to
12 examine the empirical and observable data of nature and
13 reason from that using logical processes.

14 Q. Now some claim that intelligent design advances a
15 religious belief, that it is inherently religious and
16 not science. Do you agree?

17 A. No. Again, no more than the Big Bang theory is
18 inherently religious. Although the Big Bang theory and
19 intelligent design might be taken by some people to have
20 theological or philosophical implications, both of them
21 rely on observed evidence, empirical evidence, and
22 logical reasoning.

23 Neither the Big Bang nor intelligent design
24 relies on any religious tenet, points to any religious
25 books, or any such thing.

1 Q. Do creationists in the sense that Plaintiffs and,
2 I believe, their experts use in this case require
3 physical evidence to draw their conclusions?

4 A. No. Actually, it's interesting that one could be
5 a creationist without any physical evidence. One could
6 rely -- a creationist could rely for his belief in
7 creation on, say, some religious text or in some private
8 religious revelation or some other non-scientific
9 source.

10 So a creationist does not need any physical
11 evidence of the kind that, for example, Richard Dawkins
12 sees in life that leads him to think that life has the
13 strong appearance of design or the kind that David
14 DeRosier sees in the bacterial flagellum. A creationist
15 can believe in creation without any such physical
16 evidence.

17 Q. Is that different than from a proponent of
18 intelligent design?

19 A. Yes, that's vastly 180 degrees different from
20 intelligent design. Intelligent design focuses
21 exclusively on the physical evidence. It relies totally
22 on empirical observations about nature. It does not
23 rely on any religious text. It does not rely on any
24 other such religious information. It relies exclusively
25 on physical evidence about nature and logical

1 inferences.

2 Q. Are intelligent design's conclusions or
3 explanations based on any religious, theological, or
4 philosophical commitment?

5 A. No, they are not.

6 Q. Again, can you draw any comparisons between
7 intelligent design and the Big Bang theory in this
8 regard?

9 A. Yes. Again, the -- both the Big Bang theory and
10 intelligent design may have philosophical or theological
11 implications in the view of some people, but again, both
12 are scientific theories. Both rely on observations
13 about nature. Both make reasoned conclusions from those
14 observations about nature.

15 Q. Does intelligent design require adherence to the
16 literal reading of the Book of Genesis?

17 A. No, it does not.

18 Q. Does intelligent design require adherence to the
19 belief that the Earth is no more than 6 to 10,000 years
20 old?

21 A. No, it doesn't.

22 Q. Does intelligent design require adherence to the
23 flood geology point of view which is advanced by
24 creationists?

25 A. No, it doesn't.

1 Q. Does intelligent design require the action of a
2 supernatural creator acting outside of the laws of
3 nature?

4 A. No, it doesn't.

5 Q. Could you explain?

6 A. Yes. Making an analogy again to the Big Bang
7 theory, the Big Bang theory is a theory which is
8 advanced simply to explain the observations that we have
9 of nature, and it does so by making observations and
10 making inferences. It does not posit any supernatural
11 act to explain the Big Bang. It leaves that event
12 unexplained.

13 Perhaps in the future, science will find an
14 explanation for that event. Perhaps it won't. But
15 nonetheless, the Big Bang is a completely scientific
16 theory. Again, intelligent design is a scientific
17 theory that starts from the data -- the physical,
18 observable data of nature, and makes reasoned
19 conclusions from that and concludes intelligent design.

20 Scientific information does not say what is the
21 cause of design. It may never say what is the cause of
22 design. But nonetheless, it remains the best scientific
23 explanation for the data that we have.

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

1 A. No, not at this point.

2 Q. Does intelligent design rule out a natural
3 explanation for the design found in nature?

4 A. No, it does not rule it out.

5 Q. Could you explain?

6 A. Yes. Again, harkening back to the Big Bang
7 theory, the Big Bang theory was proposed, and the cause
8 of the Big Bang was utterly unknown. It's still utterly
9 unknown. But nonetheless, the Big Bang theory is a
10 scientific theory.

11 The Big Bang theory does not postulate that the
12 Big Bang was a supernatural act. Although, you know, it
13 simply posits no explanation whatsoever. In the same
14 sense, intelligent design is a scientific theory
15 advanced to offer -- advanced to explain the physical,
16 observable facts about nature.

17 It cannot explain the source of the design and
18 just leaves it as an open question.

19 Q. We've heard testimony about methodological
20 naturalism. Are you familiar with that term?

21 A. Yes, I am.

22 Q. I believe you indicated in your deposition that
23 you thought it hobbles or even constrains intelligent
24 design, is that correct?

25 A. Yes, that's right.

1 Q. How does it do so?

2 A. Well, any constraint on what conclusion science
3 can come to hobbles all of science. Science should be
4 an open, no-holds-barred struggle to obtain the truth
5 about nature. When you start putting constraints on
6 science, science suffers.

7 Yesterday, I discussed a man named Walter Nernst
8 who said that the timelessness of nature, the infinity
9 of time was a necessary constraint on a scientific
10 theory. Science had to operate within that framework.
11 If he had prevailed, progress, real progress in science
12 would have been severely constrained.

13 Another reason why methodological naturalism can
14 be a constraint on science is because oftentimes people
15 don't think -- don't separate neatly categories in their
16 own minds. For example, I showed the -- I showed the
17 quotation from John Maddox, the editor of Nature, who
18 found the Big Bang theory philosophically unacceptable
19 and was reluctant to embrace it because of that.

20 There are other scientists in the past, one named
21 Fred Hoyle, who rejected the Big Bang theory because he
22 did not like its non-scientific, extra-scientific
23 implications. So to the extent that people confuse a
24 scientific theory with extra-scientific implications
25 that some people might draw from it, then that might --

1 that might be a constraint upon the theory.

2 Q. Despite these constraints, does intelligent
3 design still fit within the framework of methodological
4 naturalism?

5 A. Yes. Despite the constraints, it certainly does,
6 just as the Big Bang theory does.

7 Q. Now we've heard some testimony about space aliens
8 and time traveling biologists. And I believe you made
9 some similar reference to that in your book, Darwin's
10 Black Box, is that correct?

11 A. Yes.

12 Q. And why was that?

13 A. Well, this was, you know, a tongue-in-cheek
14 effort to show people that, you know, intelligent design
15 does not exclude natural explanations, although some,
16 you know, explanations we might wave our hands to think
17 up right now might strike many people as implausible,
18 they are not, you know, utterly illogical.

19 And it was kind of a placemaker to say that maybe
20 some explanation will occur to us or be found in the
21 future which will, in fact, be a completely natural one.

22 Q. Now the space alien claim in particular seems to
23 fall hard on the ear of a lay person. But has that been
24 a claim that has been advanced by a notable scientist to
25 explain the natural phenomena?

1 A. Yes, that's right. Surprisingly, in the year
2 1973, a man named Francis Crick, the eminent Nobel
3 laureate who discovered the double helicle shape of DNA
4 with James Watson, he published, with a co-author named
5 Leslie Orgle, he published a paper entitled Directed
6 Panspermia, which appeared in the science journal
7 Icarus.

8 And the gist of the paper was that the problems
9 trying to think of an unintelligent origin of life on
10 Earth were so severe that perhaps we should consider the
11 possibility that space aliens in the distant past sent a
12 rocket ship to the Earth filled with spores to seed life
13 on the early Earth.

14 Q. This was a claim advanced by a Nobel laureate?

15 A. Yes, Francis Crick.

16 Q. And the article in which his arguments appear,
17 was this a peer reviewed science journal?

18 A. Yes, the journal Icarus.

19 Q. Was this just a tongue-in-cheek, so to speak,
20 explanation on behalf of Francis Crick?

21 A. No, it wasn't. He mentioned it first in that
22 1973 article, and he repeated the same claim in a book
23 he published in '88 and interviews he gave later on.
24 And from what I understand, he still thought it was a
25 reasonable idea up until his death recently.

1 Q. Sir, I'd ask you to direct your attention to the
2 exhibit binder that I have provided for you, and if you
3 could go to tab 14. There is an exhibit marked as
4 Defendants' Exhibit 203-E as echo. Is that the article
5 from Francis Crick that you've been testifying about?

6 A. Yes, this is Francis Crick's article on Directed
7 Panspermia.

8 Q. Is the search for intelligence causes a
9 scientific exploration?

10 A. Yes, it is.

11 Q. Again, do you have any examples that we could
12 point to?

13 A. Well, one good example is one that I mentioned
14 earlier, which is this project called the SETI project,
15 S-E-T-I, which stands for search for extraterrestrial
16 intelligence, where scientists use instruments to scan
17 space in the hope of finding transmissions or some
18 signals that may have been sent by extraterrestrial
19 sources.

20 And they are confident that they could be able to
21 distinguish those signals from the background noise,
22 background radiation, electromagnetic phenomena of
23 space.

24 Q. Again, that's a scientific exploration?

25 A. Yes, a number of scientists are involved in that.

1 MR. MUISE: Your Honor, I'm just -- do you
2 intend to go to 12:30?

3 THE COURT: I was thinking more 12:15,
4 unless you think that this is an appropriate break
5 point. Your call.

6 MR. MUISE: I certainly have more than 15
7 minutes. This next section might be divided in that 15,
8 so my preference would be to take the lunch break and
9 come back and then complete the direct during the first
10 session after lunch.

11 THE COURT: All right. We'll return then
12 at, let's say, 1:25, this afternoon, after a suitable
13 lunch break, and we'll pick up with your next topic on
14 direct at that time. We'll be in recess.

15 (Whereupon, a lunch recess was taken at
16 12:04 p.m.)

17

18

19

20

21

22

23

24

25

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25

CERTIFICATION

I hereby certify that the proceedings and evidence are contained fully and accurately in the notes taken by me on the within proceedings, and that this copy is a correct transcript of the same.

/s/ Wendy C. Yinger

Wendy C. Yinger, RPR
U.S. Official Court Reporter
(717) 440-1535

The foregoing certification of this transcript does not apply to any reproduction by any means unless under the direct control and/or supervision of the certifying reporter.