

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

3 TAMMY KITZMILLER, et al., : CASE NO.
4 Plaintiffs : 4:04-CV-02688
5 vs. :
6 DOVER SCHOOL DISTRICT, : Harrisburg, PA
7 Defendant : 17 October 2005
8 : 1:20 p.m.

7 TRANSCRIPT OF CIVIL BENCH TRIAL PROCEEDINGS
8 TRIAL DAY 10, AFTERNOON SESSION
9 BEFORE THE HONORABLE JOHN E. JONES, III
10 UNITED STATES DISTRICT JUDGE

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I N D E X
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DEFENSE WITNESSES

Dr. Michael Behe:
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P R O C E E D I N G S

1
2 THE COURT: Be seated, please. All right.
3 We return, and Mr. Muise, you may continue.

4 DIRECT EXAMINATION CONTINUED

5 BY MR. MUISE:

6 Q. Thank you, Your Honor. Dr. Behe, I want to
7 ask you some questions about the term theory and
8 its understanding in the science community. As
9 the record has shown so far that the statement
10 that is read to the students in this case uses
11 this definition, "A theory is defined as a well
12 tested explanation that unifies a broad range of
13 observations." Is that a good definition of a
14 theory?

15 A. Yes, it seems to be.

16 Q. Are you aware of the National Academy of
17 Sciences' definition of the word theory?

18 A. Yes, I've heard it.

19 Q. Let me see if this is what your
20 understanding of that definition is. In
21 science "a well substantiated explanation
22 of some aspect of the natural world that can
23 incorporate facts, laws, inferences, and tested
24 hypotheses." Do you agree with that definition?

25 A. Well, that's certainly one definition of

1 the word theory, but you have to be sensitive
2 to the fact that the word theory can be used in
3 other senses as well.

4 Q. It can be used in other senses in the
5 scientific community?

6 A. Yes, in the scientific community itself.

7 Q. Now, using the National Academy of
8 Sciences' definition of theory, does that
9 mean a theory is almost certainly right?

10 A. No, it's not. And that might surprise some
11 people unless you, until you start to think of
12 a couple of examples, and perhaps I'd like to
13 discuss two examples of a well substantiated
14 theory that was widely held, but nonetheless
15 which turned out to be incorrect. The first --

16 Q. I'm sorry, and you prepared a slide to make
17 this point?

18 A. I did, but first let me mention something
19 else. Before -- let me ask, let me mention an
20 older example that most people are familiar
21 with, and that's the example of geocentrism, the
22 idea that the earth is the center of the solar
23 system, the center of the universe, and that the
24 stars and sun circle around the earth. Now, it
25 turns out that was very well substantiated

1 because people could look up and watch the stars
2 and the sun circle around the earth.

3 So they had very good evidence to support
4 their view. Furthermore, that theory was used
5 for ages to help sailors and so on navigate the
6 seas. So it was pretty well substantiated.

7 Nonetheless, of course as everybody knows it
8 turned out to be incorrect, and Copernicus

9 proposed that in fact the sun is the center of
10 the solar system and that the earth, while
11 revolving on its axis, travels around the sun.

12 So again that's an old example, but nonetheless
13 it shows that a well accepted theory nonetheless
14 is not necessarily correct.

15 **Q.** And you have an example of that in more
16 modern times?

17 **A.** Yes, a more modern example from the late
18 19th century is something called the ether
19 theory of the proposition of light, and that's
20 shown on this slide here. I pulled off an
21 article from the web describing ether theory
22 from the Encyclopedia Britannica, and they say
23 that, "The ether theory in physics, ether is a
24 theoretical universal substance believed during
25 the 19th century to act as the medium for

1 transmission of electromagnetic waves, much as
2 sound waves are traveled elastically such as
3 air. "The ether was assumed to be weightless,
4 transparent, frictionless, undetectable
5 chemically or physically, and literally
6 permeating all matter and space."

7 Now, this theory arose from the fact that
8 it was known that light was a wave, and like
9 waves in the ocean and waves in air that we
10 perceive as sound, waves need a medium to travel
11 in. But if light is a wave, what does it travel
12 in in space? Ether. Ether was the medium
13 through which light traveled.

14 Q. Who was it that was the proponent of this
15 theory?

16 A. Well, it's a good thing we use this article
17 from the Encyclopedia Britannica, because on the
18 next slide we see that a man named James Clerk
19 Maxwell, who was arguably the greatest physicist
20 of the 19th century, wrote an article for the
21 Ninth Edition of Encyclopedia Britannica in the
22 1870's, the title of which was *Ether*. And you
23 should keep in mind when he wrote this for this
24 publication, this was not going to be read not
25 only by the general public at large, but by all

1 physicists as well.

2 So he was writing of the idea as it was
3 commonly held at that time in the highest levels
4 of physics, and he wrote the following:

5 "Whatever difficulties we may have in forming
6 a consistent idea of the constitution of the
7 ether, there can be no doubt that the
8 interplanetary and interstellar spaces are not
9 empty, but are occupied by a material substance
10 or body which is certainly the largest and
11 probably the most uniform body of which we have
12 any knowledge."

13 Now, later on Einstein's work caused
14 physics to abandon the ether theory. Physicists
15 no longer believed that the ether does in fact
16 fill space, but let's look further on the next
17 slide. This is a copy of James Clerk Maxwell's
18 article taken from a collection of his papers,
19 his article on the ether, and I want to
20 concentrate on the lower portion down here and
21 I think on the next slide that's blown up a
22 little bit.

23 I'm not going to read this, I'm just going
24 to point out that you can observe that he's
25 using a lot of precise numbers about the energy

1 of light by the sun, and it turns out he's using
2 that to do calculations, and in the calculations
3 he is deducing the properties of the ether. For
4 example, these large red arrows are pointing to
5 the coefficient of rigidity of ether, which is
6 given by the formula $R_0 V^2$, which is
7 842.8.

8 The next red arrow points to a line labeled
9 density of ether, which is equal to R_0 , which is
10 equal to 9.36 times 10 to the minus 19th power.
11 Now, the point I want to make using this slide
12 is that James Clerk Maxwell, the greatest
13 physicist of his time, whose equations for
14 electricity and magnetism are still ought to
15 physics students today, was using his well
16 accepted theory to do precise calculations
17 and deduce precise physical properties of a
18 substance that did not exist. And so the point
19 is that even a well accepted theory, even a
20 feature which seems to be required by something
21 else such as the wave nature of light, can
22 nonetheless be inaccurate and turned out to be
23 not only wrong, but utterly imaginary.

24 Q. Again I guess that would demonstrate the
25 nature that scientific theories are tentative,

1 is that correct?

2 A. Yes, I think that it helps to make that
3 claim that scientific theories are tentative
4 more than just a hypothetical claim. The
5 history of science is replete with examples of
6 what seemed to be correct explanations which
7 turned out to be incorrect.

8 Q. Now, is Darwin's theory of evolution a
9 theory in the sense of the National Academy
10 of Sciences' definition?

11 A. Well, it partly is and partly isn't.

12 Q. Did you prepare a slide to demonstrate that
13 point?

14 A. Yes. A slide here is an excerpt from a
15 book written by a man named Ernst Mayr, who,
16 Ernst Mayr was a very prominent evolutionary
17 biologist, who died just I think last year at
18 the age of 100, and was privy to a lot of the
19 development of what's called neo-Darwinian
20 theory in the middle of the 20th century, and he
21 wrote a book entitled *One Long Argument*, and in
22 it he makes the case that Darwin's theory is not
23 some single entity, and let me just quote from
24 that.

25 He says, "In both scholarly and popular

1 literature one frequently finds references to
2 Darwin's theory of evolution as though it were
3 a unitary entity. In reality, Darwin's theory
4 of evolution was a whole bundle of theories,
5 and it is impossible to discuss Darwin's
6 evolutionary thought constructively if one does
7 not distinguish its various components. The
8 current literature can easily lead one perplexed
9 over the disagreements and outright
10 contradictions among Darwin specialists, until
11 one realizes that to a large extent these
12 differs of opinion are due to a failure of some
13 of these students of Darwin to appreciate the
14 complexity of his paradigm." So you have to
15 realize that Darwin's theory is not a single
16 claim. There are multiple claims within what's
17 called Darwin's theory, and they can be, they
18 can have different levels of evidence behind
19 them.

20 Q. Did he break out these five claims in this
21 *One Long Argument* that you're referring to?

22 A. Yes, he did. He went on to say, well what
23 are those ideas that are grouped together under
24 Darwin's theory? He called them, he identified
25 five different components, the first of which is

1 "evolution as such." He says this is the theory
2 that the world is not constant or recently
3 create nor perpetually cycling, but rather is
4 steadily changing. So what we might call change
5 over time.

6 Q. Is that a theory or is it an empirical
7 observation of facts? How would you describe
8 that?

9 A. Well, yeah, I myself would call that more
10 an observation rather than a theory. We see
11 that the earth seems to have changed over time.
12 The second --

13 Q. Go ahead.

14 A. The second aspect of Darwin's theory that
15 Mayr discerned was common descent. This is the
16 theory that, "Every group of organisms descended
17 from a common ancestor and that all groups of
18 organisms, including animals, plants, and
19 microorganisms, go back to a single origin of
20 life on earth." The third point is something
21 called multiplication of species. This theory
22 explains the origin of enormous organic
23 diversity.

24 I won't read the rest of the quote there,
25 but it's just a question why are there so many

1 species, the multiplication of species. The
2 fourth component of Darwin's theory according to
3 Mayr is something called gradualism. According
4 to this theory, "Evolutionary change takes place
5 through the gradual change of populations and
6 not by the sudden saltational production of
7 new individuals that represent a new type." So
8 gradualism, things thing gradually over time.

9 And the last component according to Mayr is
10 natural selection. According to this theory,
11 "Evolutionary change comes through the abundant
12 production of genetic variation, the relatively
13 few individuals who survive, owing to
14 particularly well adapted combinations of
15 inheritable characters, give rise to the next
16 generation." So this is what's commonly called
17 survival of the fittest.

18 Q. Is this strength of the scientific evidence
19 equal for each of these five separate claims?

20 A. No, they vary greatly in the strength of
21 evidence that's behind each of those.

22 Q. Has it been your experience that supporters
23 of Darwin's theory of evolution and opponents of
24 intelligent design have conflated the evidence
25 for the occurrence of evolution, the change over

1 time, with the evidence for the mechanism of
2 evolution, natural selection?

3 A. Yes. In my experience many people confuse
4 the various parts of Darwin's theory. They
5 don't make the distinction that Ernst Mayr
6 makes, and people see that there has been change
7 in the world and a lot of people then assume
8 that because there has been change in the world,
9 then it must have been change driven by natural
10 selection. And that's a mistaken conclusion.

11 Q. Are there other senses in which the word
12 theory is used by scientists?

13 A. Yes. You have to realize that scientists
14 themselves use the word theory in a very broad,
15 with a very broad range of senses. Not only in
16 the sense that the National Academy gave to it,
17 but scientists themselves use it to indicate
18 many other things.

19 Q. Now, you did a search of Pub Med searching
20 for the term theory, is that correct?

21 A. Yes, that's right. In order to illustrate
22 how scientists themselves use the word theory,
23 I did a search in a database called Pub Med,
24 which is maintained by the National Library of
25 Medicine, which is a division of the National

1 Institutes of Health of the federal government,
2 and this is a database of abstracts and titles
3 of almost all biological articles that are
4 published. It contains millions and millions of
5 articles.

6 Q. And have you prepared several slides to
7 demonstrate this point?

8 A. Yes, I have. In this first one, which
9 might be a little bit hard for me to read, but
10 nonetheless the red arrow down here, I certainly
11 won't read the whole abstract, but if you can
12 see the little red arrow down here, let me just
13 read a phrase from this. This says that, "This
14 study does not support the previous theory."
15 And so they are using the word theory here
16 to mean a previous idea that has now been shown
17 to be wrong or have evidence against it.

18 Q. If I may, Dr. Behe, just interrupt you here
19 briefly that might help you in your testimony as
20 well, if you go to the exhibit book that you've
21 been provided, and if you look under Tab 8 I
22 believe, there's an exhibit marked Defendant's
23 Exhibit 203-A, as in Alpha.

24 A. Oh, okay. Yes.

25 Q. Is that the search that you conducted on

1 Pub Med in which the slides are derived from?

2 A. Yes, that's correct. Yes, uh-huh.

3 Q. And if it will help you to perhaps look at
4 those as opposed to trying to review it on the
5 screen, work between the two.

6 A. Okay. Thank you. And the next slide up on
7 the screen here is if you follow the red arrows,
8 and those points to other occasions of the word
9 theory, it says in this article, "The membrane
10 pacemaker theory of aging is an extension of the
11 oxidative stress theory of aging." So in here
12 the scientists are using the word theory to
13 explain, or to refer to ideas that are very
14 limited in scope, which may or may not have much
15 evidence to support them.

16 So in a much different sense than the
17 National Academy used in its booklet. You
18 could go to -- oh, thank you for the next slide.
19 Let me just see if I can find that one article.
20 Here it is. Okay. If you look at this other
21 article from Pub Med, it's pointing to a
22 sentence that begins, "In theory, change in
23 climate would be expected to cause changes
24 elsewhere."

25 So again a scientist here is using the

1 world theory to refer to, you know, we would
2 expect this to happen, a kind of expectation.
3 Now, I put up here a publication of my own that
4 I published with my dissertation advisor Walter
5 Englander, and if you could read the top it
6 reads, "mixed gelation theory," and it refers to
7 mixtures of sickle cell hemoglobin with other
8 types of hemoglobin. So again we were using the
9 word theory to describe ideas and results that
10 have a very limited providence.

11 And finally on the next slide this is an
12 article taken from an issue of *Science Magazine*
13 seven years ago, a special issue which focused
14 on the question of why is there sexual
15 reproduction. And the article was entitled "Why
16 Sex? Putting Theory to the Test," and the
17 author said the following. "Biologists have
18 come up with a profusion of theories since first
19 posing these questions a century ago." These
20 questions meaning why is there sexual
21 reproduction, and again the author here is
22 using the word theory in terms of competing
23 hypotheses, competing ideas, none of which have
24 much evidence behind it, none of which have wide
25 acceptance in the scientific community.

1 Q. I want to return to Ernst Mayr and ask you
2 are the parts of Darwin's theory as he's listed
3 here well tested?

4 A. No, they are not. If you look at the
5 top ones, evolution as such, common descent,
6 multiplication of species, those are all well
7 tested. The claim of gradualism is in my
8 opinion rather mixed. There's evidence for,
9 and some people argue against it. But the
10 component of Darwin's theory natural selection
11 which is sometimes viewed as the mechanism that
12 Darwin proposed for evolution is very poorly
13 tested and has very little evidence to back
14 it up.

15 Q. I want to go through in a little bit more
16 detail on some of these claims. Going back to
17 that first claim, and I believe you testified
18 probably akin to an empirical observation, is
19 that correct?

20 A. Yes, evolution as such that the world
21 is changed over time, and life as well.

22 Q. Does intelligent design refute the
23 occurrence of evolution?

24 A. No, it certainly has no argument with this
25 component of Darwin's theory. As a matter of

1 fact I think there is a, on the next slide
2 there's an excerpt from *Of Pandas and People*
3 where the authors write, "When the word is used
4 in this sense, that is the sense of change over
5 time, it is hard to disagree that evolution is a
6 fact. The authors of this volume certainly have
7 no dispute with that notion. *Pandas* clearly
8 teaches that life has a history, and that the
9 kinds of organisms present on earth have changed
10 over time." And let me make the point that
11 Ernst Mayr calls this component evolution as
12 such. That is the basic idea of evolution.

13 Q. So when you hear a claim that intelligent
14 design is anti-evolution, are those accurate?

15 A. No, they are completely inaccurate.

16 Q. Returning back to the slide with Ernst
17 Mayr, the second claim, does intelligent design
18 speak to that second claim of common descent?

19 A. No. Intelligent design looks to see if
20 aspects of life exhibit a purposeful arrangement
21 of parts as evidenced by their physical
22 structure. It does not say how such a thing
23 might have happened.

24 Q. Is common descent nevertheless addressed in
25 *Pandas*?

1 A. Yes. I've read sections that do address
2 common descent.

3 Q. How does it fit then within intelligent
4 design?

5 A. Well, some people point to empirical
6 difficulties that they see for common descent,
7 but common descent itself is not a claim, either
8 for or against is not a claim of intelligent
9 design theory.

10 Q. Would it be accurate then to say it's
11 viewed more as a difficulty with Darwinism
12 rather than a claim for intelligent design?

13 A. Yes, that's correct. Common descent
14 applies more to Darwinian claims, which claim
15 descent with modification, than it does to
16 intelligent design, because intelligent design
17 is focused exclusively on the question of
18 whether we can discern the effects of
19 intelligence in life.

20 Q. In which of these claims is intelligent
21 design focused principally upon?

22 A. Intelligent design focuses exclusively on
23 the fifth claim of Ernst Mayr, or the fifth
24 component that Ernst Mayr identified in Darwin's
25 theory, that of natural selection, or in other

1 words what is the mechanism of evolution, how
2 could such things happen.

3 Q. Is it your view that that is where the
4 scientific evidence for these five claims is
5 perhaps the weakest?

6 A. Yes, that is in fact the most poorly
7 supported aspect of Darwin's theory. As a
8 matter of fact, that's where the evidence in
9 my view points away from Darwin's theory.

10 Q. Again so does intelligent design question
11 all parts of Darwin's theory of evolution?

12 A. No. It focuses exclusively on the question
13 of the mechanism of evolution, and I tried to
14 make that clear as this picture shows. This is
15 an issue of something called the reports of the
16 National Center for Science Education, which
17 is a group which strongly advocates for the
18 teaching of Darwinian evolution in school, and
19 I wrote a letter to the editor of *The Reports*,
20 which was published in an issue approximately
21 four years ago.

22 And here's an excerpt from that letter
23 where I explain, "The core claim of intelligent
24 design theory is quite limited. It says nothing
25 directly about how biological design was

1 produced, who the designer was, whether there
2 has been common descent, or other such
3 questions. Those can be addressed separately."
4 It says, "Only that design can be empirically
5 detected in observable features of physical
6 systems."

7 And I go on to say, "As an important
8 corollary it also predicts that mindless
9 processes such as natural selection or the
10 self-organization scenarios favored by Shanks
11 and Joplin will not be demonstrated to be able
12 to produce irreducible systems of the complexity
13 found in cells." So I tried to clearly explain
14 that the only focus of intelligent design is on
15 the mechanism of evolution, or the question of
16 whether or not aspects of life show the marks
17 of intelligent design.

18 Q. And you said this was published in *The*
19 *Reports* by the National Center for Science
20 Education?

21 A. Yes, that's correct.

22 Q. And that's an organization where Dr. Kevin
23 Padian is the president?

24 A. Yes, I understand he's the president of
25 that.

1 Q. And Dr. Alters and Forrest are also
2 associated with this organization?

3 A. I think Dr. Forrest is and Dr. Miller
4 is. I'm not sure about Dr. Alters, and also
5 Professor Pennock has a reply in that same
6 issue of *The Reports*.

7 Q. Now, Dr. Miller in his expert report that
8 he's provided in this case said that Darwin's
9 theory actually has many mechanisms. Do you
10 agree with that?

11 A. No, I disagree, and here is a little copy
12 of Professor Miller's expert report, and he
13 lists a number of things, including genetic
14 recombination, transposition, horizontal gene
15 transfer, gene duplication, sexual selection,
16 developmental mutation and so on, and he says
17 that, "The relative importance of these and
18 other mechanisms of evolution, these conflicts
19 continue to motivate."

20 So he seems to be calling these mechanisms.
21 He's making a mistake here. Except for sexual
22 selection, all the other components listed in
23 his report, gene transfer, transposition,
24 recombination, are simply ways that diversity
25 is generated in nature. But diversity has to be

1 acted upon in Darwin's understanding by natural
2 selection. So natural selection is the only
3 mechanism of Darwinian evolution. The sexual
4 selection that he lists, that is a mechanism,
5 but it's a subset of natural selection where
6 features have selected value due to the
7 consideration of their ability to allow an
8 organism to attract mates or otherwise
9 reproduce.

10 Q. Do other scientists agree with your
11 position on this?

12 A. Yes, they do. Here's an excerpt from
13 an article by a man named Jerry Coyne, who
14 was writing in a magazine called *The New*
15 *Republic*. Now, Jerry Coyne is a professor of
16 evolutionary biology at the University of
17 Chicago and a vocal opponent of intelligent
18 design, as the title of the article shows.
19 He writes an article entitled *The Case Against*
20 *Intelligent Design*.

21 Nonetheless, he disputes what Professor
22 Miller has said, the idea that he had talked
23 about, Jerry Coyne says the following, "Since
24 1859 Darwin's theories have been expanded, and
25 we now know that some evolutionary change can be

1 caused by forces other than natural selection.
2 For example, random and nonadaptive changes in
3 the frequencies of different genetic variance,
4 the genetic equivalent of coin tossing, have
5 produced evolutionary changes in DNA sequences,"
6 and here is an important point.

7 "Yet, selection is still the only known
8 evolutionary force that can produce the fit
9 between organism and environment, or between
10 organism and organism, that makes nature seem
11 designed." So Professor Coyne was saying that
12 well, there can be random genetic changes in
13 organisms, but the only mechanism pertinent to
14 the discussion of whether there is design in
15 nature or not is Darwin's idea of natural
16 selection.

17 **Q.** Do any other scientist besides intelligent
18 design proponents question the ability of
19 natural selection to explain various aspects
20 of life?

21 **A.** Yes, a number of scientists who are not
22 design proponents also question the ability of
23 natural selection to account for features of
24 life, and one example is shown on this slide,
25 a man named Stewart Kauffman, who is a professor

1 of biology at the University of Toronto now, in
2 1993 wrote a book called *The Origins of Order:
3 Self organization and Selection in Evolution*,
4 and that was published by Oxford University
5 Press, and in the introduction to his book he
6 wrote the following, "Darwin's answer to the
7 sources of the order we see all around us is
8 overwhelmingly an appeal to a single singular
9 force: natural selection. It is this single
10 force view which I believe to be inadequate, for
11 it fails to notice, fails to stress, fails to
12 incorporate the possibility that simple and
13 complex systems exhibit order spontaneously."
14 So in this quotation Professor Kauffman
15 is summarizing his view that the Darwinian
16 mechanism of natural selection is inadequate
17 to explain some features of biology.

18 Q. Does Dr. Kauffman still maintain that view?

19 A. Yes, he does. He also contributed an
20 article to the book *Debating Design*, to which
21 I and others also contributed, which was
22 published by Cambridge University Press last
23 year in which he reiterates his views about
24 self-organization and complexity. He wrote in
25 the underlying bold portion, "Much of the order

1 in organisms I believe is self organized and
2 spontaneous. Self-organization mingles with
3 natural selection in barely understood ways to
4 yield the magnificence of our teeming biosphere.
5 We must therefore expand evolutionary theory."
6 In other words natural selection is not
7 sufficient. We have to expand evolutionary
8 theory to include something else other than
9 natural selection if we want to explain what
10 we see in biology.

11 Q. Sir, you've already shown that the theory
12 of evolution does not consist of a single claim,
13 and you testified that proponents of the theory
14 of evolution tend to conflate evidence for one
15 claim to support another claim, and also you
16 testified that opponents of ID, intelligent
17 design, claim that it's anti-evolution, and you
18 showed a slide of *Pandas* which refutes that
19 particular claim. Now, when we say, when we use
20 the term Darwin's theory of evolution, what is
21 the common understanding for that?

22 A. Well, the common understanding is that
23 natural selection has driven all of the change
24 in the world, we see in the biological world.

25 Q. Now, the evolution as such, understanding

1 that life is changed over time, that was
2 understood before Darwin's time, is that
3 correct?

4 A. Yes. People have been proposing such
5 things for I think a couple of hundred years
6 before Darwin's day. Darwin's distinctive
7 contribution to this discussion was the proposal
8 of natural selection. It was he who had
9 proposed what people considered to be a
10 completely unintelligent mechanism for the
11 production of the complexity of life.

12 Q. With that understanding, sir, is Darwin's
13 theory of evolution a fact?

14 A. No. No theory is a fact.

15 Q. Are there gaps and problems with Darwin's
16 theory of evolution?

17 A. Yes, there are.

18 Q. Is there one principal contention you have
19 with the explanatory power of the theory of
20 evolution that's is particularly relevant for
21 intelligent design?

22 A. Yes, I think the major overwhelming problem
23 with Darwin's theory is what I summarized in my
24 expert report. I stated the following, "It is
25 my scientific opinion that the primary problem

1 with Darwin's theory of evolution is the lack of
2 detailed, testable, rigorous explanations for
3 the origin of new complex biological features."

4 MR. ROTHSCCHILD: Your Honor, objection, just
5 to the extent I just want to make sure that the
6 expert report is not coming into evidence. I
7 don't object to the slide as long as that's
8 clear.

9 MR. MUISE: The report is not coming, Your
10 Honor. It's just for demonstrative purposes to
11 demonstrate his opinion.

12 THE COURT: I'll consider that just to be a
13 clarification objection.

14 MR. ROTHSCCHILD: Thank you, judge.

15 THE COURT: There's no need for a ruling.
16 You can proceed.

17 BY MR. MUISE:

18 Q. Dr. Behe, do scientists who do not adhere
19 to intelligent design share your opinion of
20 this?

21 A. Yes, they do. A couple of examples are
22 shown next. Here is an excerpt from a book by a
23 man named Franklin Harold, who's an emeritus
24 professor of chemistry at Colorado State
25 University, and four years ago he published a

1 book entitled *The Way of the Cell* with Oxford
2 University Press, and he quote, "We must concede
3 that there are presently no detailed Darwinian
4 accounts of the evolution of any biochemical
5 system, only a variety of wishful speculations."
6 So he also seems to share that view.

7 Q. Has Dr. Miller acknowledged such problems?

8 A. Yes. Dr. Miller himself wrote in his
9 expert statement, "Living cells are filled of
10 course with complex structures," and let's skip
11 down to the underlying bold statement, he
12 continues, "One might pick nearly any cellular
13 structure, the ribosome for example, and claim
14 correctly that its origin has not been explained
15 in detail by evolution." So again everybody
16 agrees that Darwinian theory has not given an
17 explanation of many, many features of life.

18 Q. With that in mind, sir, I have some
19 specifics I want to ask you. Has the theory
20 of evolution, in particular natural selection,
21 explained the existence of the genetic code?

22 A. No.

23 Q. Has the theory of evolution, in particular
24 natural selection, explained the transcription
25 of DNA?

1 A. No.

2 Q. Has the theory of evolution, in particular
3 natural selection, explained translation of "M"
4 RNA?

5 A. No.

6 Q. Has the theory of evolution, in particular
7 natural selection, explained the structure and
8 function of the ribosome?

9 A. No.

10 Q. Has the theory of evolution, in particular
11 natural selection, explained the structure of
12 the cytoskeleton?

13 A. No.

14 Q. Has the theory of evolution, in particular
15 natural selection, explained nucleosome
16 structure?

17 A. No.

18 Q. Has the theory of evolution, in particular
19 natural selection, explained the development of
20 new protein interactions?

21 A. No.

22 Q. Has the theory of evolution, in particular
23 natural selection, explained the existence of
24 the proteosoma?

25 A. No.

1 Q. Has the theory of evolution, in particular
2 natural selection, explained the existence of
3 the endoplasmic reticulum?

4 A. No.

5 Q. Has the theory of evolution, in particular
6 natural selection, explained the existence of
7 motility organelle such as the bacterial
8 flagellum in the eucaryotic syllium?

9 A. No.

10 Q. Has the theory of evolution, in particular
11 natural selection, explained the development of
12 the pathways for the construction of the syllium
13 and flagella?

14 A. No.

15 Q. Has the theory of evolution, in particular
16 natural selection, explained the existence of
17 defensive apparatus such as the immune system
18 and blood clotting system?

19 A. No.

20 Q. Sir, is it fair to say that under this
21 broad category of difficulties that we just
22 reviewed lies much of the structure and
23 development of life?

24 A. Yes, that's correct.

25 Q. Does this cause you to question whether a

1 Darwinian framework is the right way to approach
2 such questions?

3 A. Yes, it does, because if Darwinian theory
4 is so fruitless at explaining the very
5 foundation of life, the cell, then that makes
6 a person reasonably doubt whether it's, whether
7 some other explanation might be more fruitful.

8 Q. Sir, in your expert opinion is there a
9 problem with falsification of Darwin's theory?

10 A. Yes, there's a big problem with that.
11 Falsification is roughly the idea that there
12 is some evidence which would make somebody
13 change his mind that a theory was right or not
14 right. In many instances Darwinian theory is
15 extremely difficult to falsify, and let me give
16 one example. On the next slide is shown a
17 figure of vertebrate embryos taken from a
18 biochemistry textbook by Voet and Voet, and this
19 is the biochemistry textbook that is used widely
20 in colleges and universities across the United
21 States.

22 The figure here is drawn after a figure
23 that was first drawn in the 19th century by a
24 man named Ernst Haeckel, who was an embryologist
25 and supporter of Darwin's theory. As you see in

1 the figure, the vertebrate embryos all begin by
2 looking virtually identical, very extremely
3 similar, and yet in the course of their
4 development they develop into completely
5 different organisms. A fish, reptile, bird,
6 amphibian, human, and so on. And Ernst Haeckel
7 thought it was exactly in accord with what
8 Darwin expected.

9 And the reasoning is illustrated by a
10 quotation on the next slide from a book entitled
11 *Molecular Biology of the Cell*, which was written
12 by Bruce Alberts, who I mentioned earlier was
13 president of the National Academy of Sciences.
14 One of his co-authors is James Watson, the Nobel
15 laureate who with Francis Crick won the prize
16 for discovering the double helical shape of DNA,
17 and other illustrious authors. And in the
18 textbook they explain those embryological facts
19 by saying the following, "Early developmental
20 stages of animals whose adult forms appear
21 radically different are often surprisingly
22 similar.

23 "Such observations are not difficult to
24 understand. The early cells of an embryo are
25 like cards at the bottom of a house of cards.

1 A great deal depends on them, and even small
2 changes in their properties are likely to result
3 in disaster." So if I can summarize their
4 reasoning here, the authors were saying these
5 extremely similar embryos are exactly what we
6 expect, because in vertebrates the basic body
7 plan is being laid down in the early
8 generations. And if you upset the foundation
9 of a structure, that's likely to essentially
10 destroy it.

11 So what we expect is for later stages of
12 development to be dissimilar, but the earlier
13 stages to be very, very similar. Nonetheless,
14 it turns out that those drawings were incorrect,
15 and a number of years ago in the late 1990's the
16 journal *Science* ran a story about a study that
17 had been done to try to reproduce Haeckel's,
18 results, and it turns out they could not be
19 reproduced. And the story was entitled
20 *Haeckel's Embryos: Fraud Rediscovered*, and if
21 you look at the illustration in the news story,
22 on the bottom row one sees the drawings of
23 embryos as Haeckel produced them, and on the top
24 row you see photographs of embryos which were
25 taken by a modern team of embryologists, looking

1 very, very much different.

2 And on the next slide are excerpts from
3 the news story. It was written, it says,
4 "Generations of biology students may have been
5 misled by a famous set of drawings of embryos
6 published 123 years ago by Ernst Haeckel.

7 'The impression they give that the embryos are
8 exactly alike is wrong,' says Michael
9 Richardson, an embryologist at St. George's
10 Hospital Medical School in London," and he was
11 the lead author of the study which showed the
12 incorrectness of Haeckel's results.

13 "Not only did Haeckel add or omit features,
14 but he also fudges the scale to exaggerate
15 similarities." Now, here is the point with
16 respect to the topic of falsification. Since
17 these studies have appeared, no Darwinian
18 biologist that I'm aware of has decided that
19 Darwinian biology is incorrect. But if a
20 theory, Darwin's theory, can live with one
21 result, and its utter opposite with virtually
22 identical embryos and with significant variation
23 in the embryos, then it says nothing about that
24 topic.

25 It doesn't predict anything. It will live

1 with whatever result experimental science comes
2 up with, which means that Darwin's theory has
3 nothing significant to say about a major feature
4 of life, embryology, because if you think about
5 it, if one kind of organism is to give rise to
6 another kind of organism over time, then the
7 embryological plan for building that first
8 organism has to change into the embryological
9 plan to build the second kind of organism, and
10 yet how that could happen is a topic that
11 Darwin's theory of evolution does not address in
12 the least.

13 Q. Sir, if I could direct your attention to
14 the exhibit book, under Tab 16, Defendant's
15 Exhibit 271?

16 A. Number 16 did you say?

17 Q. Tab 16, that's right. Is that a copy of
18 that article, it's an on-line version of
19 *Haeckel's Embryos: Fraud Rediscovered?*

20 A. Yes, it's a copy of the article that does
21 not have the illustrations in it.

22 Q. Was the article written by Elizabeth --

23 A. Pennisi.

24 Q. Pennisi, the one you've been referring to?

25 A. Yes.

1 Q. Does the bacterial flagellum in the Type 3
2 secretory system, and we're going to be talking
3 about these in a little bit greater detail
4 later, but is there an analogy also with regard
5 to the falsifiability that you could --

6 A. Yes. As I'll discuss later, again
7 Darwinian theory can't decide whether the
8 Type 3 secretory system might have arisen from
9 the flagellum, the flagellum from the secretory
10 system, whether both developed independently,
11 or other pertinent questions. So again the
12 question of falsifiability, if it doesn't, can't
13 predict any of those, then it has nothing to say
14 about those features.

15 Q. Now, does Darwin's theory have difficulty
16 explaining what we see in nature regarding
17 sexual reproduction?

18 A. Yes, turns out that it does. It was
19 realized not long after Darwin published his
20 theory, it was realized by a man named August
21 Weisman that Darwinian theory actually predicts
22 that most organisms should reproduce asexually
23 because, one reason is because Darwinian theory,
24 one goal of an organism, goal in the terms of a
25 better evolutionary result, is to get more of

1 the organism's genes into the next generation.
2 If an organism reproduced asexually by clonal
3 reproduction, the offspring would contain all of
4 the genes of the organism. But during sexual
5 reproduction, for each offspring reproduced the
6 parent gets only half of its genes into the next
7 generation.

8 And this has been a conundrum that has been
9 unsolved in Darwinian theory for over a century,
10 and during that time scientists have not just
11 been sitting around. They've been trying very
12 hard to come up with explanations for that, and
13 as a matter of fact they've come up with so many
14 suggestions, so many theories, that in 1999 a
15 man named Kondrashov published an article in the
16 journal *Heredity* entitled *Classification of*
17 *Hypotheses on the Advantage of Amphimixis*, and
18 for amphimixis read sexual reproduction. There
19 were so many competing ideas that he had to
20 classify them into groups to try to keep better
21 track of them, and he --

22 Q. This was written in 1993?

23 A. Yes, in 1993, about ten years ago. Let me
24 just read the first sentence here, "After more
25 than a century of debate, the major factors of

1 the evolution of reproduction are still
2 obscure."

3 Q. If I could direct your attention again to
4 your exhibit book, Tab Number 9, and it's listed
5 as Defendant's 270, is that the article you're
6 referring to?

7 A. Yes, that's the one. And if I could
8 continue the quote after the bolded text, he
9 continues, "During the past 25 years, hypotheses
10 have become so numerous and diverse that their
11 classification is a necessity. The time is
12 probably right for this. No fundamentally new
13 hypothesis has appeared in the last five years,
14 and I would be surprised and delighted if some
15 important idea remain unpublished." So he was
16 expressing his view that an exhaustive look had
17 been done and that we have not yet come up with
18 an answer.

19 Q. Do you have additional slides and articles
20 to demonstrate this point?

21 A. Yes, that's right. This was in 1993. In
22 the year 1998 *Science*, the journal *Science*
23 issued a special issue which focused on the
24 evolution of sex, and in that the leadoff
25 article of a number of articles in that issue

1 was the one entitled *Why Sex? Putting Theory to*
2 *the Test*. Now, notice the word theory is not
3 being used in the sense that the National
4 Academy gives to it.

5 And if you look at this little abstract
6 which is, or this little blurb up on the
7 left-hand corner I think on the next slide
8 that's enlarged, it stated that, "After decades
9 of theorizing about the evolutionary advantages
10 of sex, biologists are at last beginning to test
11 their ideas in the real world." So let notice a
12 couple of things about that.

13 Again they're using theory, theorizing, in
14 a sense like brainstorming. Furthermore, they
15 say that this brainstorming, this theorizing
16 goes on ahead of the activity of testing it.
17 And furthermore that the testing can be put off
18 decades from when the theorizing takes place.

19 Q. If I could direct your attention again to
20 the exhibit book under Tab 10 and there's an
21 exhibit listed, Defendant's Exhibit Number 269,
22 is that a copy, it looks like an on-line version
23 copy of the article that you're referring to?

24 A. Yes, that's right.

25 Q. I believe you have another slide you'd like

1 to cite?

2 A. Yes. There's an excerpt from this article
3 which is on the next I think -- oh, yes, I'm
4 sorry. Yes, this is kind of a repeat of one
5 that I've done already, "Biologists have come up
6 with a profusion of theories since first posing
7 these questions a century ago." So clearly this
8 is an idea that has stumped science for a very
9 long time. Another excerpt from the article is
10 shown on the next slide. The author writes,
11 "How sex began and why it thrived remains a
12 mystery. Why did sex overtake asexual
13 reproduction?" I'm going to skip down here,
14 and the author continues, "Sex is a paradox in
15 part because if nature puts a premium on genetic
16 fidelity, asexual reproduction should come out
17 ahead. All this shuffling is more likely to
18 break up combinations of good genes than to
19 create them. Yet nature keeps reshuffling the
20 deck."

21 Q. And if I could just so the record is clear,
22 those last two quotes that you read from were
23 from which articles?

24 A. They were from the article *Why Sex? Putting*
25 *Theory to the Test* by Bernice Wuethrich.

1 Q. Again do you have another slide to make
2 this point?

3 A. Yes, I do. This is a quotation of a man
4 named George Williams. George Williams is a
5 prominent evolutionary biology at the State
6 university of New York at Stonybrook, and he
7 wrote a book in the mid 1970's entitled *Sex and*
8 *Evolution*, and a part of that book was quoted in
9 a book recently by Richard Dawkins of Oxford
10 University, and the quotation is this. "This
11 book," that is George Williams' book, "this book
12 is written from a conviction that the prevalence
13 of sexual reproduction in higher plants and
14 animals is inconsistent with current
15 evolutionary theory. There is a kind of crisis
16 at hand in evolutionary biology," and Dawkins
17 comments on this quotation on the next slide.

18 Richard Dawkins, an evolutionary biologist
19 at Oxford University, Dawkins says, this is
20 Dawkins speaking, "Maynard Smith and Hamilton,"
21 which refers to two prominent evolutionary
22 biologists, "said similar things. It is to
23 resolve this crisis that all three Darwinian
24 heroes along with others of the rising
25 generation, labored. I shall not attempt an

1 account of their efforts, and certainly I have
2 no rival solution to offer myself."

3 So the point is that this problem is still
4 unresolved, and yet this goes to the very heart
5 of evolutionary theory, or a theory of evolution
6 that expects that most species would reproduce
7 asexually can be likened to a theory of gravity
8 that expects that most objects will fall up.
9 And in either case a reasonable person might
10 wonder if the theory is missing some large piece
11 of the puzzle, and certainly I think as an
12 educator students should be apprised of facts
13 like these.

14 Q. Sir, does Darwin's theory account for the
15 origins of life?

16 A. No, Darwin's theory does not even address
17 the origin of life.

18 Q. Is this an unsolved scientific problem?

19 A. Yes, it certainly is. And it also poses,
20 it poses a large problem for Darwin's theory
21 as well, and --

22 Q. What is that problem?

23 A. I think I have a little excerpt from my
24 expert report in which I dealt with that
25 question, and I said the following, "The problem

1 that the *Origin of Life* poses for Darwin's
2 theory is the following. If the beginning of
3 life required something extra, something in
4 addition to the unintelligent operation of
5 natural processes that Darwin's theory invokes,
6 then it would be fair for a curious inquirer to
7 wonder if those other processes ended with the
8 beginning of life, or if they continued to
9 operate throughout the history of life," and
10 I'll stop there, close quote. So the point is
11 this. If we cannot explain the origin of life
12 by unintelligent processes, and if intelligent
13 processes were in fact involved with that, then
14 we might wonder did they continue throughout the
15 history of life, or did they stop at that point.

16 Q. Sir, do you have an additional slide to
17 make this point regarding the questions of the
18 origins of life is left unresolved?

19 A. Yes, I do. Just a couple. It's easy to
20 find scientists involved in a study of the
21 origin of life who are very willing to say that
22 we have not a clue as to how life started, and
23 here's a convenient source, this was an
24 interview by PBS with a man named Andrew Knoll,
25 who is an eminent professor of biology at

1 Harvard who studies the early development of
2 life, and one of the topics they wanted to speak
3 with him over was, "Why it's so devilishly
4 difficult to figure out how life got started."

5 And on the next slide they put the question
6 to Andrew Knoll, they say, "How does life form?"
7 And Professor Knoll says, "The short answer is
8 we don't really know how life originated on
9 this planet." And skip a bit, "We remain in
10 substantial ignorance." Next slide, they asked
11 another question, the interviewer asked, "Will
12 we ever solve the problem of the origin of
13 life?"

14 And Knoll says, "I don't know. I imagine
15 my grandchildren will still be sitting around
16 saying that it's a great mystery." So that
17 here's a person involved in studying the origin
18 of life who says quite frankly that we don't
19 know what's going on and he doesn't have any
20 particular expectation that our grandchildren
21 will understand the origin of life.

22 Q. Sir, if I could direct your attention to
23 the exhibit book under Tab 12, Defendant's
24 Exhibit Number 267, is that the interview that
25 you've just been testifying to?

1 A. Yes, it is.

2 Q. I'd like to direct your attention to what
3 I have put up on the screen here is an excerpt
4 from a booklet entitled *Science and Creationism*
5 which was put out by the National Academy of
6 Sciences in 1999, and if you could please read
7 that quote?

8 A. Yes. The National Academy wrote, "For
9 those who are studying the origin of life, the
10 question is no longer whether life could have
11 originated by chemical processes involving
12 nonbiological components. The question instead
13 has become which of many pathways might have
14 been followed to produce the first cell," and
15 I'll stop there, close quote.

16 Q. Do you have any problems with this
17 statement?

18 A. Yes. I find it very disturbing, because
19 in that statement you don't see any reference
20 to the results of workers in the field. You
21 don't see any reference to the data of what
22 people have come up with. Instead, in this
23 publication they focus on the attitudes of the
24 scientists involved, and while the attitudes
25 might be an interesting sociological phenomenon,

1 they do not go to the question of whether we
2 can explain the origin of life.

3 And furthermore, this booklet is written
4 for teachers and indirectly then for their
5 students, and by advising teachers or letting
6 teachers or by saying this to teachers, it seems
7 to me the National Academy is encouraging them
8 to have their students think of this problem in
9 the same way that workers have been doing for
10 the past fifty years in the same way that has
11 proved fruitless for over half a century.

12 Q. Sir, is there a scientific controversy
13 regarding intelligent design in evolution?

14 A. Yes, there is.

15 Q. And what leads you to that conclusion?

16 A. Well, in addition to, you know, the
17 articles and counterarticles and things that
18 have been mentioned earlier in the day, and
19 besides the conferences and symposia that I have
20 attended, there have also been a number of
21 published books and articles debating design,
22 and a good example of that is shown on the
23 screen here, this is the cover of the book
24 entitled, excuse me, *Debating Design: From*
25 *Darwin to DNA*, and it was edited by two people,

1 William Dembski, who's a philosopher and
2 mathematician and intelligent design proponent,
3 and Michael Ruse, who's a professor of the
4 philosophy of science and a student of Darwinian
5 thought, and in this number of academics
6 contributed chapters arguing not only about
7 intelligent design and Darwinism, but also
8 complexity theory, self-organization, and other
9 views as well.

10 Q. And I believe you testified previously
11 that some of the experts that are testifying
12 on behalf of plaintiffs in this case have also
13 contributed chapters to this particular book?

14 A. That's correct. Kenneth Miller has a
15 chapter in there. I think Robert Pennock has
16 a chapter in there as well.

17 Q. And I believe you also testified during
18 the qualifications portions that you contributed
19 a chapter to a book that was written by Robert
20 Pennock, scientists debating the question of
21 intelligent design?

22 A. That's correct, published by MIT Press.

23 Q. And there was also a similar book --

24 MR. ROTHSCILD: Objection, Your Honor.
25 I think it's mischaracterizing the title.

1 MR. MUISE: Your Honor, I didn't say what
2 the title was. It's what the --

3 MR. ROTHSCCHILD: I think he did say it,
4 Your Honor.

5 MR. MUISE: The nature of the book. I don't
6 believe I stated the title. If I stated the
7 title --

8 THE COURT: How did he mischaracterize it?

9 MR. ROTHSCCHILD: He called it scientists
10 debating intelligent design, or something to
11 that effect. He used the word scientists. It's
12 actually *Intelligent Design and Its Critics*, if
13 it's the Pennock edited book.

14 MR. MUISE: Okay. I don't see much a
15 distinction with that, Your Honor, but --

16 MR. ROTHSCCHILD: It think it's a loaded
17 question.

18 THE COURT: Well, for the record you don't
19 doubt, Mr. Muise, that's the correct title, or
20 do you? Let's just be clear.

21 MR. ROTHSCCHILD: Sorry, *Intelligent Design*,
22 *Creationism, and Its Critics*, I am corrected.

23 MR. MUISE: I believe that's the correct
24 title, Your Honor. I'm just verifying.

25 (Brief pause.)

1 MR. MUISE: Let's go back to your --

2 THE COURT: Just so we're --

3 MR. MUISE: I do have it here, Your Honor,
4 and I just want to make it clear what the title
5 is, and I believe Mr. Rothschild is accurate.

6 THE COURT: All right. Then there's no need
7 for a ruling on it. You can just clarify it for
8 the record.

9 BY MR. MUISE:

10 Q. The book by Robert T. Pennock was entitled
11 *Intelligent Design, Creationism and Its Critics:*
12 *Philosophical, Theological and Scientific*
13 *Perspectives*, is that correct?

14 A. That's correct.

15 Q. And that book was published by the MIT
16 Press?

17 A. That's correct, yes.

18 Q. You contributed an article making
19 scientific arguments for intelligent design
20 in that book?

21 A. That's correct, I did.

22 Q. I should clarify, you submitted a chapter,
23 is that correct?

24 A. Yes that's, right.

25 Q. Were there other scientists who submitted

1 chapters in that particular book?

2 A. Yes. There were several arguing against
3 my ideas and several others arguing on other
4 points.

5 Q. Were these scientists making scientific
6 arguments in that book?

7 A. Yes.

8 Q. Again similarly I believe there was a book
9 that was edited by John Campbell and Steve Meyer
10 entitle *Darwinism: Design in Public Education*,
11 is that correct?

12 A. Yes, that's right.

13 Q. Published by Michigan State University
14 Press?

15 A. Yes, that's correct.

16 Q. And several scientists and others
17 contributed articles for that particular
18 book, is that correct?

19 A. Yes, that's right.

20 Q. If I could direct your attention to the
21 exhibit, Tab 13, marked as Defendant's Exhibit
22 266.

23 A. Yes.

24 Q. Do you know what that, what is Defendant's
25 Exhibit 266?

1 A. It is a publication in the journal
2 *Theoretical Biology* by two authors, Richard
3 Thornhill and David Ussery entitled *A*
4 *Classification of Possible Roots of Darwinian*
5 *Evolution*.

6 Q. And who are Thornhill and Ussery?

7 A. They are two scientists, David Ussery is
8 at the Institute of Biotechnology and Technical
9 University of Denmark and, Technical University
10 of Denmark, and Thornhill I'm not quite sure of.

11 Q. Is that an article that was published in
12 a scientific journal?

13 A. Yes, the *Journal of Theoretical Biology* is
14 indeed a scientific journal.

15 Q. What was that article about?

16 A. As its title implies, it was trying to
17 group, put into groups possible pathways that
18 a Darwinian evolutionary pathway might take,
19 and it was particularly concerned with the
20 problem of irreducible complexity.

21 Q. Did it particularly refer to irreducible
22 complexity?

23 A. Yes, it did. It refers to irreducible
24 complexity by name I'm certain, virtually
25 certain, and it makes reference to my book

1 as well to illustrate the problem.

2 Q. So would it be fair to say based on these
3 articles and books and symposia that you've been
4 attending that scientists are debating this
5 issue in scientific and academic circles?

6 A. Yes, that's what I would say.

7 MR. MUISE: Your Honor, I'm about to start
8 into another area. I know we've only been going
9 for an hour, but I'm not sure how that'll work
10 out.

11 THE COURT: No, keep going.

12 MR. MUISE: Okay.

13 THE COURT: Because we've not been at it
14 long enough to take a break.

15 BY MR. MUISE:

16 Q. Dr. Behe, I'd like to return to the concept
17 irreducible complexity, which you testified was
18 a term that you coined in *Darwin's Black Box*, is
19 that correct?

20 A. Yes, that's right.

21 Q. Now, you testified that the design
22 arguments speaks of the purposeful arrangement
23 of parts. Are there any other aspects of the
24 design argument?

25 A. Yes, and that's correct. There are other

1 aspects, and they're shown on the next slide.
2 Just like Ernst Mayr showed that there were
3 several aspects to Darwinian theory, there are
4 aspects to the intelligent design argument. The
5 intelligent design argument itself, the positive
6 argument for it is the purposeful arrangement of
7 parts, as I have described.

8 However, in an inductive argument, if
9 somebody else offers a counterexample to the
10 induction, then one has to address that to make
11 the inductive argument stand. So there's also
12 a negative argument which says that despite
13 Darwinian claims that the inductive positive
14 argument is unrefuted, that is that Darwinism
15 cannot account for the purposeful arrangement
16 of parts.

17 Q. So that's your argument against the
18 plausibility of a Darwinian explanation for
19 design, is that correct?

20 A. Yes, that's right.

21 Q. Do you have several slides that further
22 make this point?

23 A. Yes. Now, what would make Darwinian
24 explanations seem implausible? Well, Charles
25 Darwin himself wrote how his argument could be

1 refuted. In his writings in his book *On the*
2 *Origin of Species* he wrote that, "If it could be
3 demonstrated that any complex organ existed
4 which could not possibly have been formed by
5 numerous successive slight modifications, my
6 theory would absolutely break down," adding,
7 "but I can find out no such case."

8 In this passage Darwin was emphasizing that
9 his was a gradual theory. Natural selection had
10 to improve things slowly, in tiny steps over
11 long periods of time. If it seemed that things
12 were improving rapidly, in big leaps, then it
13 would start to look suspiciously as if random
14 mutation and natural selection were not the
15 cause.

16 **Q.** Have other scientists acknowledged that
17 this is an argument against Darwin's theory of
18 evolution?

19 **A.** Yes. In his book *Finding Darwin's God*
20 Kenneth Miller has written that, "If Darwinism
21 cannot explain the interlocking complexity of
22 biochemistry, then it is doomed."

23 **Q.** I believe we have a quote from another
24 prominent scientist?

25 **A.** Yes. Richard Dawkins in his recent book

1 *The Ancestor's Tail*, from which I quoted
2 recently, wrote "That it is perfectly legitimate
3 to propose the argument from irreducible
4 complexity, which is a phrase I use, as a
5 possible explanation for the lack of something
6 that doesn't exist, as I did, for the absence
7 of wheeled mammals." Let me take a second to
8 explain Dawkins' reference.

9 He's saying that this problem is a problem
10 for biology, but nonetheless he thinks that
11 everything in biology has a Darwinian
12 explanation. So that whatever we do see in
13 biology necessarily is not irreducibly complex,
14 and I think in my opinion that's an example of
15 begging the question. But he does recognize the
16 concept of irreducible complexity.

17 **Q.** Sir, I'd like at this point for you to
18 define irreducible complexity, and we have a
19 slide here.

20 **A.** Yes, in my article from the journal *Biology*
21 *and Philosophy*, I defined it this way. "By
22 irreducibly complex, I mean a single system
23 which is necessarily composed of several well
24 matched interacting parts that contribute to the
25 basic function, and where the removal of any one

1 of the parts causes the system to effectively
2 cease functioning."

3 Q. Now, you have up there "necessarily"
4 in italics. Is there a reason for that?

5 A. Yes, the definition that I gave in *Darwin's*
6 *Black Box* did not have those italicized words
7 necessarily, but after the books came out and an
8 evolutionary biologist at the University of
9 Rochester named Allen Orr pointed out that it
10 may be the case that if you had a system that
11 was already functioning, already doing some
12 function, it's possible for a part to come
13 along and just assist the system in performing
14 its function, but after several changes perhaps
15 it might change in such a way that the extra
16 part has now become necessary to the function of
17 the system but that could have been approached
18 gradually.

19 And I, in thinking about it I saw that he
20 was thinking of examples that I did not have
21 in mind when I wrote the book. So I kind of
22 tweaked the definition here in this article to
23 try to make it clear and try to exclude those
24 examples that I didn't have in mind.

25 Q. Is it a common practice within the science

1 community for a scientist to adjust, modify, or
2 tweak their theories based on criticisms that
3 they get from other scientists?

4 A. Oh, sure. That's done all the time.
5 Nobody is perfect, nobody can think of
6 everything at once, and a person is always
7 grateful for criticism and feedback that helps
8 to improve an idea.

9 Q. Does criticism undermine the idea that
10 you were trying to convey by irreducible
11 complexity?

12 A. No, it didn't. It clarified it, and after
13 his, after reading his SI I saw that he was
14 thinking of things that I did not have in mind.
15 So I tried to clarify that.

16 Q. You have this system in underlying
17 capitalized and in red. What's the purpose
18 for that?

19 A. Well, that to me has turned into a point
20 of confusion because some people, including
21 Professor Miller, have been focusing the
22 discussion on the parts of the system and saying
23 if one removes a part and then can use the part
24 for some other purpose, then they say that means
25 that it's not irreducibly complex, but that is

1 not the definition I gave to irreducible
2 complexity, that is not the concept of
3 irreducible complexity that I described in
4 *Darwin's Black Box*. I said that if you take
5 away one of the parts from the system, the
6 system, the function of the system itself ceases
7 to work, and whether one can use the part for
8 anything else is beside the point.

9 Q. So then it is fair to say Dr. Miller's uses
10 the wrong definition of your concept and then
11 argues against that different definition to
12 claim that your concept is incorrect?

13 A. Yes. It's a mischaracterization, yes.

14 Q. Now, Dr. Padian testified on Friday that
15 the concept of irreducible complexity applies
16 above the molecular level, is that correct?

17 A. No, that is incorrect. In *Darwin's Black*
18 *Box* I was at pains to say that the concept of
19 irreducible complexity applies only to systems
20 where we can enumerate the parts, where we can
21 see all the parts and how they work, and I said
22 that in biology therefore that necessarily means
23 systems smaller than a cell, systems whose
24 active molecular components we can elucidate.

25 When you go beyond a cell, then you're

1 necessarily talking about a system, an organ
2 or animal or any such thing, that is so complex
3 we don't really know what we're dealing with,
4 and so it remains a black box, and so the term
5 irreducible complexity is confined to molecular
6 examples.

7 Q. Well, I want to read to you several
8 sections, passages from *Pandas* that Dr. Padian
9 referred to as claiming that this is the concept
10 of irreducible complexity, and I'd like your
11 comment on each one of those as I go through.
12 The first one, "Multifunctional adaptations
13 where a single structure or trait achieves two
14 or more functions at once is taken as evidence
15 by the proponents of intelligent design of their
16 theory," and the reference is page 72 of *Pandas*.

17 A. Well, if -- I'm sorry, what is the question
18 then?

19 Q. The question is, is that a definition or
20 is that within your concept of irreducible
21 complexity?

22 A. No, that's not the way I define the term,
23 and I'm not quite sure what he has in mind.

24 Q. And the second example is, "Proponents
25 of intelligent design maintain that only a

1 consummate engineer could anticipate so
2 effectively the total engineering requirements
3 of an organism like the giraffe." That's a
4 citation from page 71. Is that a reference
5 to the concept of irreducible complexity?

6 A. No, it isn't. Again, irreducible
7 complexity focuses on the cell and systems
8 smaller, because we have to elucidate all the
9 parts, and you have to keep in mind that the
10 parts of a biological system are molecular
11 parts, even though most people commonly think
12 of large organisms. Let me just say that, you
13 know, that you should keep in mind that
14 Darwinism has other problems beyond irreducible
15 complexity. So *Pandas* might have been pointing
16 to those.

17 Q. Two more such examples. The third one, two
18 more of out of four, this is the third out of
19 four, "But it has not been demonstrated that
20 mutations are able to produce the highly
21 coordinated parts of novel structures needed
22 again and again by macroevolution." And again,
23 is that referring to the concept of irreducible
24 complexity?

25 A. Well, again unless he's referring to the

1 molecular level, then no, that is not correct.
2 It turned out that molecular changes, small
3 changes in DNA can actually cause large changes
4 in an organ. You might lose the finger or get a
5 duplicate of a finger or some such thing, so you
6 have to apply the concept of irreducible
7 complexity to the molecular level.

8 Q. And the last example, "Design theory
9 suggest that various forms of life began
10 with their distinctive features already intact,
11 fish with fins and scales, birds with feathers,
12 beaks, and wings," that's a reference to page 25
13 of *Pandas*. Is that a reference to the concept
14 of irreducible complexity?

15 A. No, it is not. Again one more time, the
16 concept of irreducible complexity applies to
17 the molecular level simply because in biology
18 the molecular level is where changes are taking
19 place. There are active components. That's
20 where the rubber meets the road in biology.
21 So one has to restrict one's self to that level.

22 Q. Is that the level where we can identify the
23 components of the systems?

24 A. Yes, that's the critical thing. We have
25 to see how things are working so we can realize

1 what's going on and decide whether or not an
2 explanation is plausible.

3 Q. So it would be fair to say those four
4 examples I read to you may illustrate or
5 highlight other difficulties with Darwin's
6 theory, but they're not specifically addressed
7 in the concept of irreducible complexity?

8 A. Yes, that's right. Just because
9 irreducible complexity is a problem, that
10 doesn't mean that it's the only problem.

11 Q. Now, again can you give us an example of an
12 irreducibly complex biochemical system?

13 A. Yes, an excellent example is again the
14 bacterial flagellum, which uses a large number
15 of parts in order to function, and again if you
16 remove the components, if you remove the
17 propeller, if you remove the hook region, if
18 you remove the drive shaft or any multiple parts
19 of the flagellum, it does not work. It's ceases
20 to function as a propulsive device.

21 Q. Now, Professor Miller has testified that
22 the flagellum is not irreducibly complex. Do
23 you agree with him?

24 A. No, I don't.

25 Q. I'd like for you to go through and explain

1 your objections to his claim.

2 A. Okay. This is a slide from Professor
3 Miller's presentation on the flagellum.
4 Let me just first read through the slide
5 completely and then I want to point to several
6 mischaracterizations that are contained on the
7 slide. He writes, "The observation that there
8 are as yet no detailed evolutionary explanations
9 for certain structures in the cell, while
10 correct, is not a strong argument for special
11 creation, 'design.' As Michael Behe has made
12 clear, the biochemical argument from design
13 depends upon a much bolder claim, namely that
14 the evolution of complex biochemical structures
15 cannot be explained even in principle."

16 This has three mischaracterizations I'd
17 like to point out in turn. The first one is
18 what many people considered to be an informal
19 logical fallacy, and that is called poisoning
20 the well. It is given the reader a, leading the
21 reader to suspect the other person's argument.
22 It's kind of a version of an *ad hominem*
23 argument. When he uses the term special
24 creation and quotation in design, that looks to
25 me like he's indicating to the reader that the

1 people who make these arguments are trying to
2 mislead you into thinking that this is design,
3 but it's really special creation.

4 What's more, again the word creation has
5 very negative overtones and is used as a
6 pejorative in many academic and scientific
7 circles. Furthermore, the phrase special
8 creation occurs nowhere in *Darwin's Black Box*.
9 I never used the phrase special creation in
10 any of my writings except perhaps to say that
11 intelligent design does not require this. And
12 so again I think it is a mischaracterization
13 and it appears to me an attempt to kind of
14 prejudice the reader against this, against my
15 argument.

16 The second point is this. The second
17 mischaracterization is this. He says, "The
18 observation that there are as yet no detailed
19 evolutionary explanations for certain structures
20 in the cell, while correct, is not a strong
21 argument for special creation that is 'design.'"
22 Here Professor Miller is doing something more
23 understandable. He's essentially is viewing my
24 theory through the lens of his own theory. So
25 all he sees is essentially how it conflicts with

1 his own theory and thinks that that's all there
2 is to it.

3 But as I have explained throughout the day
4 today, if we could go to the next slide, that
5 an inability to explain something is not the
6 argument for design. The argument for design is
7 when we perceive the purposeful arrangement of
8 parts, the purposeful arrangement of parts such
9 as we see in the flagellum, such as we see the
10 molecular machinery such as described in that
11 special issue of *Ce77* and so on.

12 We can go to the next slide, this is a copy
13 of the first slide of Professor Miller's, the
14 third mischaracterization is this. He says, "As
15 Michael Behe has made clear, the biochemical
16 argument from design depends upon a much bolder
17 claim, namely that the evolution of complex
18 biochemical structures cannot be explained even
19 in principle." This is a mischaracterization.
20 It's essentially absolutizing my argument.
21 It's making overstating my argument in order to
22 make it seem brittle, to make it more easily
23 argued against.

24 Q. Have you addressed such a claim in *Darwin's*
25 *Black Box*?

1 A. Yes, if you read *Darwin's Black Box* you
2 see that I say the following, "Even if a system
3 is irreducibly complex and could not have been
4 produced directly, however one cannot definitely
5 rule out the possibility of an indirect
6 circuitous route. As the complexity of an
7 interacting system increases though, the
8 likelihood of such an indirect route drops
9 precipitously."

10 So here I was arguing well, there's a big
11 problem for Darwinian theory. These things
12 can't be produced directly, but nonetheless
13 you can't rule out an indirect route, but
14 nonetheless building a structure by changing
15 its mechanism and changing its components
16 multiple times is very implausible and the
17 likelihood of such a thing, the more complex
18 it gets, the less likely it appears. So the
19 point is that I was careful in my book to
20 qualify my argument at numerous points, and
21 Professor Miller ignores those qualifications.

22 Q. Do these qualification also demonstrate
23 the tentative nature in which you hold your
24 theories?

25 A. Yes, that's right. I always -- well, I try

1 to state it in what I thought was a reasonable
2 way and in a tentative way as well.

3 Q. I believe we have a couple of more slides
4 from Dr. Miller that you --

5 A. Yes, this is essentially a continuation.
6 These will be slides number 2 and 3 from his
7 slides on the flagellum. This is just a
8 continuation of his overstated arguments.
9 He says, "The reason that Darwinian evolution
10 can't do this is because the flagellum is
11 irreducibly complex," and he quotes my
12 definition of irreducible complexity from
13 *Darwin's Black Box*, and continue on the next
14 slide.

15 And he states that, "That claim is the
16 basis of the biochemical argument for design."
17 But again that is not the basis for the
18 biochemical argument for design. The basis
19 for the biochemical argument for design is the
20 purposeful arrangement of parts. Irreducible
21 complexity shows the difficulties for Darwinian
22 processes in trying to explain these things.

23 Q. Now, Dr. Miller claims that natural
24 selection can explain the flagellum. Do
25 you agree with that claim?

1 A. I'm sorry, can you restate that?

2 Q. Dr. Miller claims that natural selection
3 can explain the bacterial flagellum. Do you
4 agree with that claim?

5 A. No, I disagree, and we go on to the next
6 slide, which is another one of Professor
7 Miller's slides from his presentation on the
8 bacterial flagellum, and he tried to explain
9 molecular machines using kind of simple concepts
10 to try and make it more understandable to a
11 broad audience. So for example on the
12 right-hand side which he labels "Evolution,"
13 he has little colored hexagons, which are exist,
14 which are separated, and then he has the
15 hexagons forming little groups and arrows
16 pointing between the hexagons and the groups of
17 hexagons, and finally there is kind of a large
18 aggregation of hexagons.

19 On this, which he labels "Design," he
20 has the colored hexagons separate and arrows
21 pointing to a larger aggregation of hexagons.
22 Now, I'm sure Professor Miller was trying to
23 get across a concept which is difficult, but in
24 my viewing and my understanding and presenting
25 it this way, this overlooks enormous problems

1 that actual molecules would encounter in the
2 cell.

3 Q. Have you addressed these claims in other
4 writings that you have done?

5 A. Yes. Professor Miller has presented
6 exactly the same argument in several other
7 settings, and I have addressed it several
8 times, most recently in my chapter in *Debating*
9 *Design*, and if you go to the next slide --

10 Q. Is this a figure from that book, *Debating*
11 *Design*?

12 A. Yes, this is Figure 2 from that chapter.
13 And the slide is entitled "An irreducibly
14 complex molecular machine, can it arise from
15 individual functional precursors." I used little
16 colored squares instead of hexagons, but
17 nonetheless the concept is kind of the same.
18 The colored squares are supposed to represent
19 individual proteins which perhaps existed in
20 the cell already, there is six different ones,
21 and the complex molecular machine now is
22 supposed to be an aggregate of all six proteins
23 with a new function that the system has that the
24 individual parts did not have. Unfortunately
25 while this illustrates, you know, something, it

1 leaves out many concepts which are critical to
2 evaluating the likelihood of such a thing. May
3 I continue?

4 Q. Yes, go ahead.

5 A. For example, proteins, the components of
6 molecular machines are not little colored
7 squares. They are not little colored hexagons.
8 They are very complex entities which we will see
9 in a second. Additionally, notice this red
10 square. The red square with the little arrow
11 places it against the green square and the
12 yellow and the blue. Why is it there? Why
13 didn't it go down there? Why is it sticking to
14 B and C and D? Why doesn't it float away?

15 None of those questions are answered, this
16 is an oversimplified way to look at a very
17 complex problem. For example, let me just make
18 one more comment. Notice that in machines in
19 our common experience, if you put a part in a
20 place different from where it usually is, that
21 often times breaks the machine. If in an
22 outboard motor you took the propeller and you
23 put it on top instead of down by the rotor, then
24 the machine would not function. And it's the
25 exact same way for molecular machines.

1 Q. Have you prepared some slides to
2 demonstrate some of the more complexity
3 of these parts?

4 A. Yes, I'm afraid we're going to have to
5 go a little bit into the complexity of these
6 molecular systems.

7 THE COURT: Do you want to break here,
8 Mr. Muise?

9 MR. MUISE: That would be wonderful, Your
10 Honor.

11 THE COURT: Why don't we do that, let's take
12 a 20-minute break here, and we'll return and
13 we'll pick up with those slides at the end of
14 the recess. We'll be in recess.

15 (Recess taken at 2:48 p.m. Proceedings
16 resumed at 3:13 p.m.)

17 THE COURT: Be seated, please. You can pick
18 it up where you left off, Mr. Muise.

19 CONTINUED DIRECT BY MR. MUISE:

20 Q. Thank you, Your Honor. Dr. Behe, before we
21 broke we were talking about how proteins aren't
22 simply colored squares or hexagons, that they
23 are far more complex than that, including what
24 makes them stick together in any particular
25 order, and I want to return back to that. We

1 put up a slide which has some indication I
2 believe of proteins, and I'd like you to explain
3 what you meant, that they're more complex than
4 just these colored hexagons.

5 A. Yes, sure. Let me preface my explanation
6 by saying this, that in talking about these
7 matters there's kind of, an intelligent design
8 proponent and a Darwinian theorist who have
9 different goals. A Darwinian wants to persuade
10 his audience that evolution isn't all that
11 difficult, it's doable, and so will not always
12 attend to all the complexity of a system,
13 whereas in order to show the difficulties
14 for undirected unintelligent processes, an
15 intelligent design proponent has to show all
16 of the very severe complexity of systems, and
17 that's often times hard to do because people
18 often times don't have the patience to attend
19 to it, but I apologize in advance but I have to
20 attend to some of the complexities here.

21 So on this slide there are three figures
22 taken from a biochemistry textbook by Voet and
23 Voet of the protein, of the same protein, a
24 protein named hemoglobin. Hemoglobin is the
25 protein that binds oxygen and carries it from

1 your lungs and dumps it off in peripheral
2 tissues such as your fingers and so on. Now,
3 this is a rendering of the structure of
4 hemoglobin, and actually this rendering itself
5 does not show the full complexity of hemoglobin.
6 Let's focus --

7 Q. You're referring to Figure 8-63 on this
8 slide?

9 A. Yes, that's correct. Let's focus on this
10 yellow glob here. You'll notice a number of
11 circles. They represent atoms in one of what
12 are called the protein chains of hemoglobin,
13 but the amino acids in that protein chain are
14 actually different. So if it was actually
15 rendered in more detail you would see a lot of
16 different colors of atoms, indicating different
17 groups and so on, and the identity of all these
18 amino acids is also frequently very critical to
19 the function of a protein.

20 Hemoglobin itself consists an aggregate of
21 four proteins designated here by the blue and
22 the green and the light blue colors, and it is
23 the aggregate of the four protein chains, that
24 is the active molecular machine in this cell
25 that carries oxygen from your lungs to your

1 tissues. Nonetheless, a drawing like this of
2 such a complex system is often times bewildering
3 to students, and so artists with the proper
4 purpose of getting across some conceptual points
5 to students will draw simplified renditions of
6 the same figure.

7 For example, in the lower left here this
8 is also supposed to be a rendition of the same
9 protein hemoglobin. But in here the only atoms
10 that are represented are things called the alpha
11 carbons of each amino acid, and the artist has
12 kind of shaded it to show the different
13 directions in which the protein chain is
14 heading. One can also to make a legitimate
15 point to students simplify the drawing even
16 further, and here's another rendering of
17 hemoglobin in Voet and Voet.

18 Here each very, very complex protein chain
19 is rendered as a simple square, and the O_2
20 represents the oxygen that each protein is
21 supposed to be carrying. Now, all of these
22 are legitimate renderings of the protein
23 hemoglobin, but when we discuss these matters
24 and we discuss difficulties with evolution and
25 we discuss arguments for intelligent design, we

1 have to keep in mind that this is the actual
2 protein, this is the actual machine in the cell,
3 and so these are the things that we have to deal
4 with.

5 Q. Again that last figure you're referring to
6 is 8-63?

7 A. That's right, uh-huh.

8 Q. And the two previous, the one just previous
9 to that was Figure 10-37 and the one prior to
10 that 10-13?

11 A. That's correct. Now, let's consider
12 a further point. We have this yellow
13 conglomeration of circles representing the
14 atoms of the protein chain, with this blue one
15 and this green one and this light blue one. Why
16 do they stick together? Why don't they just
17 float away? How come they are in the
18 arrangement they are? Why don't we have the
19 yellow one over here? The green one down here?

20 Well, it turns out that proteins arrange
21 themselves. Molecular machines are actually
22 much more sophisticated than the machines of
23 our common experience, because in our common
24 experience with things like say outboard motors,
25 an intelligent agent assembles the parts of

1 those machines. But in the cell the molecular
2 machines have to assemble themselves. How do
3 they do that? They do it by having surfaces
4 which are both geometrically and chemically
5 complementary to the proteins to which they're
6 supposed to bind, and I think --

7 Q. Do you have a slide to demonstrate that
8 for us?

9 A. Yes, I do. I think it's the next one.
10 Okay, remember here's another little cartoon
11 version which gets rid of some complexity of
12 the system in order to make an important point
13 to students. This is also a figure taken from
14 the biochemistry textbook Voet and Voet. This
15 is meant to convey why two molecules, why two
16 proteins bind to each other specifically in the
17 cell. This one up here is supposed to represent
18 one protein. The second one is supposed to be
19 this greenish area, and it's supposed to have a
20 depression in it in which the yellowish protein
21 binds to and sticks.

22 Now, let me point out a couple of things.
23 You'll notice that the shapes of the proteins
24 are matched to each other. They're
25 geometrically complementary, kind of like a

1 hand in a glove. But not only are they
2 geometrically complementary, they're also
3 chemically complementary. You see these little
4 circles and NH and this thing here? Well, these
5 are chemical groups on the surface of the two
6 binding proteins, and they attract each other.
7 Certain groups attach other groups.

8 I think the easiest to understand is the
9 one right here, there's a red circle marked with
10 a minus sign in it. That indicates an amino
11 side chain of a protein that has a negative
12 charge. When it binds to the larger one, notice
13 that on the surface of the larger protein
14 there's this blue circle with a plus sign in it.
15 That is taken, that is meant to indicate an
16 amino acid side chain with a positive charge.
17 Negative and positive charges attract. So
18 therefore these guys stick together.

19 If this were a negative charge these two
20 proteins would not stick together. They would
21 float away from each other. It's not sufficient
22 to have just one group in the protein be
23 complementary to another group in a protein.
24 Usually proteins have multiple amino acids that
25 stick together and cause them to bind to each

1 other. For example, look up here, this little
2 circle labeled H. H is supposed to stand for
3 something called hydrophobic, which essentially
4 means oily. It doesn't like to be in contact
5 with water.

6 It lines up with another H on the green
7 protein so that the two oily groups can stick
8 together and avoid water. So it's kind of like
9 oil, you know, oil and water, they don't mix.
10 If they're in this configuration the two oily
11 groups can stick together and be away from
12 water, and there are other groups, too, which
13 I won't go into which exhibit things call
14 hydrogen bonding which also help the proteins
15 stick together.

16 So in molecular machines, in aggregates
17 of proteins, all of the proteins which are
18 sticking together have to have all these
19 complementary surfaces in order for them to
20 bind their correct partners. If they do not
21 have the complementary surface, they don't bind
22 and the molecular machine does not form. Now,
23 interestingly, remember Darwin's theory says
24 that evolution has to proceed in small steps,
25 tiny steps.

1 Well, one way something like this might
2 form is by, you have to have mutations that
3 might produce each of these interactions at a
4 time. For example, I think there's a quotation
5 from an article in *Nature* which kind of make
6 this point, and I'll explain it after I quote
7 it, it's from an article by a man named John
8 Maynard Smith, who is a very prominent
9 evolutionary biologist who died about a year
10 ago I believe, and he wrote in a paper called
11 *Natural Selection and the Concept of a Protein*
12 *Space*, which was published in *Nature* in 1970,
13 "It follows that if evolution by natural
14 selection is to occur, functional proteins must
15 form a continuous network which can be traversed
16 by unit mutational steps without passing through
17 nonfunctional intermediates," and by unit
18 mutational steps, we mean each of those pluses,
19 each of those H's, each of those OH's and so on
20 that I showed you in that little cartoon drawing
21 on the previous slide.

22 If for example a mutation came along that
23 changed a positive into a negative charge and
24 disallowed an interaction that needed to occur,
25 that would be a detrimental one. John Maynard

1 Smith is saying that we need to proceed, you
2 know, one step at a time. So the point is that
3 those little colored squares are enormously
4 complex in themselves, and further the ability
5 to get them to bind specifically to their
6 correct partners also requires much more
7 additional information. It is not a single step
8 phenomenon. You have to have the surfaces of
9 two proteins to match.

10 Q. A difficulty of getting two changes
11 at once?

12 A. Yes, that's exactly right. If you can
13 do this one tiny, tiny step at a time, then
14 Darwinian evolution can work. If you need to
15 make several changes at once, two, three, four,
16 there were multiple interactions that were
17 required for those two proteins to bind. If
18 you need multiple interactions, the plausibility
19 of Darwinian evolution rapidly, rapidly
20 diminishes.

21 Q. And have other scientists made similar
22 observations?

23 A. Yes. On the next slide an evolutionary
24 biologist by the name of Allen Orr, who's at the
25 University of Rochester, published an article in

1 a journal called Biology entitled *A Minimum on*
2 *the Number of Steps Taken in Adaptive Walks* in
3 which he makes this similar point. He says,
4 "Given realistically low mutation rates, double
5 mutants will be so rare that adaptation is
6 essentially constrained to surveying and
7 substituting one mutational step neighbors.
8 Thus, if a double mutant sequence is favorable,
9 but all single amino acid mutants are
10 deleterious, adaptation will generally not
11 proceed," and translating that into more
12 colloquial English it means that you have to
13 change again those groups one at a time, and
14 if you need to change two at a time in order to
15 get a favorable interaction, then you are
16 running into a big roadblock for Darwinian
17 processes.

18 Q. Now, have you done any writing or research
19 that emphasizes this particular point?

20 A. Yes. On the next slide I believe is a copy
21 of an article that I published with David Smoke
22 which was published last year in the journal
23 *Protein Science*, which is entitled *Simulating*
24 *Evolution by Gene Duplication of Protein*
25 *Features that Require Multiple Amino Acid*

1 *Residues*, and in this paper we were addressing
2 exactly that problem. What happens if you need
3 to change a couple of amino acids before you get
4 a selective effect?

5 And the gist of the conclusion is if you
6 need to change two at once or three at once,
7 then again the expectation that that will happen
8 at a probability becomes much smaller, the
9 length of time one would have to wait for such
10 a mutation to show up is much longer, the
11 population size of a species would have to be
12 much, much longer to have an expectation of such
13 a mutation occurring.

14 Q. And this particular article, the one
15 you wrote with David Smoke, you testified to
16 previously?

17 A. Yes, that's the same one.

18 Q. I believe we have a diagram to further make
19 this point?

20 A. Yes. Here again is a little simplified
21 cartoon version of how proteins might interact,
22 simply to point out the problem that is not
23 apparent in the earlier drawings. Now I've made
24 the shapes of those colored proteins, I've
25 altered the shapes. Now the A is a circle and

1 what's that, a C, the C is a rectangle, and the
2 other proteins have other shapes. How do we get
3 those to bind into a conglomerate molecular
4 machine?

5 In order to get them to bind to each other
6 we have to alter their surfaces to be
7 geometrically and chemically complementary, and
8 that is a large and long, tall evolutionary
9 order. As a matter of fact, it's so tall that
10 one can reasonably conclude that something like
11 this would not be expected to occur. So the
12 point I want to make here is that even if one
13 was to have parts in the cell which if they
14 could develop binding sites to bind to each
15 other, and if that binding together would
16 produce a new selectable property, that still
17 does not help in Darwinian processes, because
18 you still have the problem of adjusting many,
19 many different things before you get the final
20 result.

21 Q. And this diagram is a figure from the
22 chapter that you wrote in *Debating Design*,
23 is that correct?

24 A. Yes. That's Figure 2.

25 Q. And that's the chapter that you've already

1 testified to previously?

2 A. Yes, that's correct.

3 Q. And I believe we have a slide with the
4 figure legend?

5 A. Yes, that's right. I make this point
6 exactly in my article in that book *Debating*
7 *Design*. Let's just look at the bold and
8 underlined text. It's says, "Thus, the problem
9 of irreducibility remains even if the separate
10 parts originally had individual functions."
11 So even if the parts can do something on their
12 own, that does not explain how one can get a
13 multipart molecular machine in a cell.

14 Q. I just want to point out that that figure
15 legend in the figure is from pages 352 to 370
16 in your chapter?

17 A. No, that's the whole chapter. The figure
18 legend is on one of those pages.

19 Q. As well as that previous diagram?

20 A. Yes, that's correct.

21 Q. Dr. Behe, if I understand you correctly, so
22 even if there are similar separate parts are in
23 the cell, that doesn't explain irreducible
24 complexity?

25 A. That's correct.

1 Q. Dr. Miller testified about something
2 called the Type 3 secretory system, the TTSS,
3 and he said that that showed that the flagellum
4 was not irreducibly complex, do you agree
5 with that assessment?

6 A. No, I disagree. That's a
7 mischaracterization.

8 Q. Why do you disagree?

9 A. Well, I think we have some slides from
10 Professor Miller's presentation, and he said
11 that, let us start with the bacteria flagellum,
12 and he has a drawing of the flagellum from a
13 recent paper. Let me just make another similar
14 point. You see these little three, four-letter
15 abbreviations all over here? Each one of those
16 is of the complexity of a hemoglobin molecule
17 that I showed on an earlier slide. Each one of
18 those has all the sophistication, all the needs
19 to have very complex features to bind together
20 that hemoglobin had.

21 Can you press the slide again to advance
22 the figure on this same thing of Professor
23 Miller's? Professor Miller says that well,
24 okay, you start with the bacterial flagellum,
25 and if you remove the pieces, then he says,

1 press again, please, he says, "That leaves just
2 ten," and he says, his characterization, his
3 mischaracterization of my argument is that
4 what's left behind should be non-functional.

5 And if we go to the next slide of Professor
6 Miller's, he says, "But it's not. Those ten
7 parts are fully functional as a protein
8 secretion system," but again I tried to be very
9 careful in my book to say that we are focusing
10 on the function of the system, of the bacterial
11 flagellum, and while a subset of the flagellum
12 might be able to be used as something else, if
13 you take away those parts it does not act as a
14 rotary motor. So it is irreducibly complex as I
15 tried to carefully explain. I'm sorry.

16 Q. So is it fair to say that Dr. Miller makes
17 a misrepresentation of what your claim is by his
18 representation?

19 A. This is a mischaracterization, yes, that's
20 correct, and I think I pointed that out on the
21 next slide. I pointed this out, as I said
22 earlier we've debated this back and forth for
23 a while. I pointed it out recently in my book
24 chapter. I write, "Miller asserted that the
25 flagellum is not irreducibly complex because

1 some proteins of the flagellum could be missing,
2 and the remainder could still transport proteins
3 perhaps independently.

4 "Again he was equivocating, switching the
5 focus from the function of the system to act as
6 a rotary propulsion machine to the ability of a
7 subset of the system to transport proteins
8 across a membrane. However, taking away the
9 parts of flagellum certainly destroys the
10 ability of the system to act as a rotary
11 propulsion machine as I have argued. "Thus,
12 contra Miller, the flagellum is indeed
13 irreducibly complex."

14 Q. Dr. Behe, even if that is true, doesn't the
15 Type 3 secretory system help us to explain the
16 flagellum, the development of the flagellum?

17 A. No, it does not help in the least. And
18 that may be surprising to some people, so let
19 me take a second to explain. Most people when
20 they see an argument such as Professor Miller
21 presents will naturally assume that well,
22 perhaps this part, this system that had fewer
23 parts, the Type 3 secretory system, maybe that
24 was a stepping stone, maybe that was an
25 intermediate on the way to the more complex

1 bacterial flagellum.

2 But in fact a number of scientists have
3 said that's not true, and perhaps we could see
4 the next slide. Yes, thank you. For example,
5 in a paper published by Nguyen, et al. five
6 years ago they investigated the Type 3 protein
7 secretion system, and they said the following,
8 "We suggest that the flagellar apparatus was the
9 evolutionary precursor of Type 3 protein
10 secretion systems."

11 In other words, they're saying that from
12 their investigation it looked like the more
13 complex type or more complex flagellum came
14 first, and then the system with fewer parts,
15 the Type 3 secretory system came second and
16 perhaps was derived from that. Exactly what
17 the opposite of what one might first expect.

18 Q. Have scientists reached different
19 conclusions?

20 A. Yes, and it turns out that other groups
21 have reached different conclusions from those
22 of Nguyen at all. For example, in a paper
23 published by Gophna, et al. recently in 2003 in
24 the journal *Gene* they write, "The fact that
25 several of the Type 3 secretory system proteins

1 are closely related to flagellar export protein
2 has led to the suggestion that the TTSS has
3 evolved from flagella. Here we reconstruct the
4 evolutionary history of four conserved Type 3
5 secretion proteins and their phylogenetic
6 relationships with flagellar paralog." And
7 then they say, "The suggestion that Type 3
8 secretory system genes have evolved from genes
9 and coding flagellar proteins is effectively
10 refuted." In other words. They say that
11 the conclusion of the first group was incorrect.
12 Instead they suggest that the Type 3 secretory
13 system and the flagellum developed independently
14 of each other, perhaps from the same precursor
15 gene. And I think on the --

16 Q. We have another study on this issue,
17 correct?

18 A. Yes. I think that's right. In the year
19 2004 a man named Milton Sayer, who was the one
20 of the authors, the senior author actually on
21 the study by Nguyen, et al. that I referred to a
22 couple of slides ago, wrote an article in a
23 journal called *Transient Microbiology* called
24 *Evolution of Bacterial Type 3 Protein Secretion*
25 *Systems*, he says the following, "It is often

1 not possible to prove directionality of an
2 evolutionary process. At present, too little
3 information is available to distinguish between
4 these possibilities with certainty. As is often
5 true in evaluating evolutionary arguments, the
6 investigator must rely on logical deduction and
7 intuition.

8 "According to my own intuition and the
9 arguments discussed above, I prefer pathway
10 2 for the Type 3 system deriving from the
11 flagellum. What's your opinion?" So I think
12 you can see from this the very tentative nature
13 of the results regarding the Type 3 secretory
14 system and the flagellum that in fact what is
15 going on is very much up in the air.

16 Q. And again I believe we have another result
17 from --

18 A. Yes. Let me apologize that again this is a
19 complex subject, and so you really have to delve
20 into it to come to a firm conclusion. This is a
21 quotation from a review article by a man named
22 Robert Macnab who was a professor of biology at
23 Yale University who died in the year 2003, and
24 this article was actually published
25 posthumously. It's entitled *Type 3 Flagellar*

1 *Protein Export and Flagellar Assembly*. It was
2 published in journal *Biochemica Biophysica Acta*,
3 and I underlined words that emphasized the
4 tentativeness and the speculative nature of
5 discussions on this topic.

6 Robert Macnab wrote, "It has been suggested
7 that the Type 3 virulence factor secretion
8 system evolved from the Type 3 flagellar protein
9 export system since flagella are far more
10 ancient, existing in very diverse genre than
11 the organisms which are targets for Type 3
12 virulence systems. However, it is possible that
13 the original targets were other bacteria. Also,
14 the possibility of lateral gene transfer cannot
15 be ruled out.

16 "Finally, one could argue that evolution
17 from a less complex structure, the needle
18 complex, to a more complex one, the flagellum,
19 is more probable than the other way around,"
20 and he continues I think on the next slide, and
21 I think I'll pass over much of this quotation
22 and just go to the last line of his article, and
23 he says, "As the above discussion indicates,
24 there is much about the evolution of Type 3
25 systems that remains mysterious."

1 So let me point out that in the past couple
2 of years we've had investigators suggest that in
3 fact the flagellum came first and the Type 3
4 secretory system came after it. We've had other
5 investigators suggest that the Type 3 secretory
6 system came first and the flagellum came after
7 it. We've had other investigators suggest that
8 the Type 3 secretory system and the flagellum
9 arose independently, perhaps from similar genes,
10 so --

11 **Q.** Dr. Behe, so what do these widely different
12 opinions mean?

13 **A.** Well, maybe we could go to the next slide.
14 To me it means this. We see the little cartoon
15 drawing of the flagellum here, and this is a
16 cartoon drawing of the Type 3 secretory system.

17 **Q.** I'm sorry, this is one of Dr. Miller's
18 slides?

19 **A.** I'm sorry, yes. This is Dr. Miller's
20 slide. Science knows a lot of information
21 about the structure of the Type 3 secretory
22 system, a lot of information about the structure
23 and function of the flagellum. It knows the
24 sequences of proteins of the flagellum. It
25 knows the sequences of the proteins of the Type

1 3 secretory system. It sees many similarities
2 between them, both in the amino acid sequence
3 and function, and it still can't tell how one
4 arose or whether one arose first, the other
5 second, or whether they arose independently.

6 So this to me drives home the point that
7 such information simply does not come out of
8 Darwinian theory. Much like our discussion of
9 Haeckel's embryos earlier in the day, Darwinian
10 theory can live with any result that
11 experimental science comes up with on this
12 question and then goes back and tries to
13 rationalize the results afterwards *post hoc*,
14 and so to a person like myself this exemplifies
15 the fact in fact these results have nothing to
16 do with Darwinian theory. They are no support
17 at all for the claim that natural selection
18 could have produced them. Quite the contrary.

19 Q. I just need to backtrack for one moment.
20 If I may approach the witness, Your Honor?

21 THE COURT: You may.

22 Q. Dr. Behe, I handed you what's been marked
23 as Defendant's Exhibit, 238 correct?

24 A. Yes.

25 Q. Is that the study from Nguyen that you

1 referenced in your testimony on the section
2 of the Type 3 secretory systems?

3 A. Yes, that's correct.

4 Q. It was inadvertently left out of your book,
5 but I just wanted to make sure you identified it
6 as an exhibit. You can just keep that with you
7 and I'll retrieve it later.

8 A. Thank you.

9 Q. I want to see if I can get you correct,
10 Dr. Behe. It's your opinion that this also
11 shows that even knowledge of the structure and
12 sequences of two systems doesn't necessarily
13 give a clue as to how these systems might have
14 arisen, is that true?

15 A. That's exactly right.

16 Q. And could you explain that further? And
17 I believe we have some additional slides for
18 that.

19 A. Yes, I think some text with actually
20 Professor Padian wrote as part of his expert
21 report illustrates this problem, and I'd like
22 to quote you several sections from that report.
23 On the next slide Professor Padian said the
24 following. He said that, "Darwin's main
25 concern, however, was with the mechanism of

1 natural selection, which cannot be observed
2 directly in the fossil record."

3 So to me this means you cannot see natural
4 selection. You see fossils, and how you
5 classify those fossils and what explanations
6 you come up with them is not based directly on
7 the evidence. Rather, it's provided by your
8 theory. And I think we have a further quote
9 from Professor Padian. He said the following,
10 and this is a long quote, so --

11 Q. If you could read it a little bit slower
12 for our court reporter when you are reading
13 these quotes, please? Thank you.

14 A. Okay. "Molecular biology has produced
15 tremendously powerful tools to compare the DNA
16 sequence of all manner of living organisms, and
17 a few extinct ones, and so help to derive their
18 evolutionary relationships. However, molecular
19 systematics can say nothing about the
20 relationship or role of fossil organisms to
21 each other or to living lineages," and he gives
22 an example.

23 "For example, several recent molecular
24 analyses agree that whales and hippos are each
25 other's closest relatives. From this conclusion

1 some authors have suggested that because both
2 kinds of animals spend time in the water, their
3 common ancestors would have been aquatic. Only
4 the fossil record could show that this inference
5 is incorrect. Therefore, hippos and whales,
6 even if they are each other's closest relatives
7 among living animals, did not have a common
8 ancestor that lived in the water, but that was
9 terrestrial. Only paleontological research and
10 materials could demonstrate this."

11 And let me make a point about this.

12 Professor Padian is saying that molecular
13 studies of DNA sequence of whales and hippos
14 suggested or led to the suggestion that both
15 animals had aquatic ancestors. But they didn't.
16 They had terrestrial ancestors. That means that
17 the molecular information is compatible with
18 either result, with the ancestors being aquatic
19 or the ancestors being terrestrial.

20 That means that the molecular information
21 can't decide what the ancestors were and
22 therefore it can't tell what the selective
23 pressure was or other factors of what might
24 have caused an ancestor of those organisms to
25 produce what we see in the modern world. So

1 that means that does not speak to Darwin's claim
2 that natural selection drove evolution, okay?

3 Well, molecular data can't decide the question.

4 But nonetheless, Professor Padian told us
5 that paleontology did. Paleontology discovered
6 what seemed to be ancestor of both hippos and
7 whales, and saw that they are terrestrial
8 organisms. So can paleontology tell us whether
9 it was natural selection that drove the
10 evolution of these organisms? Well, no. On
11 the previous slide he said explicitly natural
12 selection is not shown directly in the fossil
13 record.

14 That means that there is nothing that can
15 show from the fossil record or from molecular
16 data that current organisms derive by a process
17 of natural selection from organisms in the past
18 or how such a thing might have happened. That
19 means that in fact the inference that such a
20 thing did is simply a theoretical construct in
21 which we try to fit that data into our current
22 theory. The current theory either predicts it,
23 does not predict it, and may be consistent with
24 such evidence, but a lot of theories might be
25 consistent with the same evidence.

1 And I think that, bring it back to the
2 flagellum, I think that's illustrated in the
3 flagellum and Type 3 secretory system 2. We
4 know all the molecular data, we know lots of
5 structural and functional studies, and yet we
6 still can't tell how natural selection could
7 have produced them.

8 **Q.** So are you saying then at best the
9 evidence, and you were talking about sequence
10 comparisons and in particular the fossil record,
11 at best they may be consistent with natural
12 selection but they also may be consistent with
13 any number of mechanisms that might be derived?

14 **A.** That's exactly right. Perhaps intelligent
15 design, perhaps complexity theory, perhaps
16 something else. But consistent does not, is
17 not the same thing as evidence for a theory.

18 **Q.** And the next slide we have is another quote
19 from Dr. Padian that I'd like you to comment
20 about.

21 **A.** I think this also throws light on this
22 topic. Professor Padian said in his expert
23 statement, he said, "Darwin was not talking
24 about how major new adaptive change took place.
25 He was talking about how minor variations could

1 be selected. He was really talking about the
2 baby steps of evolution. He made only the most
3 passing references to how new major adaptive
4 types might emerge," and I could comment that
5 no one disputes or certainly no one I'm aware of
6 disputes that Darwinian processes, Darwinian
7 mechanism, can explain some things in life. And
8 certainly nobody disputes that baby steps could
9 be explained by random mutation and natural
10 selection. It is exactly the new major adaptive
11 types and new molecular systems for myself as a
12 biochemist that is the focus of dispute.

13 Q. So again though when you say nobody
14 refutes, is that saying that intelligent design
15 does not refute this notion of baby steps that
16 Dr. Padian is referring to?

17 A. That's right. It is very happy to say that
18 Darwinian processes are consistent with those.

19 Q. Here I believe is a continuation of that
20 particular statement from his report.

21 A. Yes, this is Professor Padian continued,
22 referring to Darwin, he said, "Though he was
23 convinced that would happen in the course of
24 time," and let me just comment on that. Well,
25 that's interesting that he was convinced that

1 would happen, but another way of saying that is
2 that Darwin assumed that these small changes
3 would add up to larger changes, or to major new
4 adaptive features, but that is exactly the point
5 of contention. And for a point of contention an
6 assumption is not evidence, let alone proof. So
7 I see this as very pertinent to the question of
8 things like the flagellum Type 3 secretory
9 system and other things as well.

10 Q. So is it clear, I guess in summarizing you
11 think that the flagellum is in fact irreducibly
12 complex, correct?

13 A. Yes, that's right.

14 Q. Does that affect necessarily the positive
15 argument for intelligent design?

16 A. Well, yes. Let's perhaps we can look at
17 another slide here that I just wrote out some
18 text to make this point clear. It's this. For
19 the past number of, past hour or so we've been
20 talking about the argument against Darwinian
21 processes, but I want to re-emphasize to say
22 that it is important to keep in mind that the
23 positive inductive argument for design is in
24 the purposeful arrangement of parts.

25 Irreducible complexity, on the other hand,

1 is an argument to show that Darwinism, the
2 presumptive alternative to design, is an
3 unlikely explanation. However, one also has
4 to be careful to remember that Darwinism isn't
5 positively demonstrated by attacks on the
6 concept of irreducible complexity. Darwinism
7 can only be positively supported by convincing
8 demonstrations that it is capable of building
9 the machinery of the degree of complexity found
10 in life. In the absence of such convincing
11 demonstration it is rationally justified
12 to think that design is correct.

13 Q. So an argument against irreducible
14 complexity is not necessarily an argument
15 against design?

16 A. An argument against irreducibly complexity
17 is not an argument against design, and more
18 importantly it's not an argument in favor of
19 Darwinian evolution.

20 Q. Have other scientists agreed that Darwinian
21 theory has not yet explained complex biochemical
22 systems?

23 A. Yes. I recall there on that slide that I
24 say Darwinism can only be positively supported
25 by convincing demonstrations, and almost

1 everybody agrees that such demonstrations have
2 not yet been forthcoming. For example, on the
3 next slide these are quotations taken from a
4 number of reviews of my book *Darwin's Black Box*,
5 most of these are by scientists. The first one
6 James Shreeve, a science writer, but all of them
7 making the point that we do not yet have
8 Darwinian explanations for such complex
9 structures.

10 For example, James Shreeve, the science
11 writer, writing the New York Times said,
12 "Mr. Behe may be right that given our current
13 state of knowledge, good old Darwinian evolution
14 cannot explain the origin of blood clotting or
15 cellular transport," and James Shapiro, who is a
16 professor of microbiology at the University of
17 Chicago, wrote in a review that, "There are no
18 detailed Darwinian accounts for the evolution of
19 any fundamental biochemical or cellular system,
20 only a variety of wishful speculations."

21 Jerry Coyne, who's a professor of
22 evolutionary biology at the University of
23 Chicago wrote in a review of the book in the
24 journal *Nature*, "There is no doubt that the
25 pathways described by Behe are dauntingly

1 complex, and their evolution will be hard to
2 unravel. We may forever be unable to envisage
3 the first protopathways."

4 And Andrew Pomiankowski, who is an
5 evolutionary biologist I believe at the
6 University College London, wrote in a review
7 in *New Scientist*, "Pick up any biochemistry
8 textbook and you will find perhaps two or three
9 references to evolution. Turn to one of these
10 and you will be lucky to find anything better
11 than 'evolution selects the fittest molecules
12 for their biological function.'"

13 So this is a sampling of writings by
14 scientists agreeing with the point that no,
15 we do not have these demonstrations yet that
16 Darwinian processes can produce complex
17 biological systems.

18 Q. And these were scientists, and in one case
19 a science writer, who are commenting on your
20 particular book, correct?

21 A. Yes.

22 Q. And have scientists in other contexts made
23 similar claims?

24 A. Yes, another good comment on this was by
25 Franklin Harold, who I mentioned before, he's

1 an emeritus professor of biochemistry at
2 Colorado State University, and in his book *The*
3 *Way of the Cell* published by Oxford University
4 Press in 2001 he kind of echos James Shapiro.
5 He says, "We must concede that there are
6 presently no detailed Darwinian accounts of the
7 evolution of any biochemical system, only a
8 variety of wishful speculations," and perhaps
9 I might add that besides these people one can
10 add also complexity theorists, who also like
11 Stuart Kauffman who also deny that such things
12 have been explained in Darwinian theory.

13 Q. Sir, have some scientists argued that
14 there is experimental evidence that complex
15 biochemical systems can arise by Darwinian
16 processes?

17 A. Yes, there have been a total of two such
18 arguments which I regard to be very important,
19 because these were claims that there had been
20 experimental demonstrations, not just
21 speculations, not just stories, but experimental
22 demonstrations that either irreducible
23 complexity was incorrect or that complex
24 systems could be built by Darwinian processes.

25 Q. And one of those claims was raised by

1 Dr. Miller, is that correct?

2 A. That's correct. I think on the next slide
3 we see that he wrote in his book *Finding*
4 *Darwin's God*, which was published in 1998, he
5 said, "A true acid test used the tools of
6 molecular genetics to wipe out an existing
7 multipart system and then see if evolution can
8 come to the rescue with a system to replace it."

9 So here he was making the point well, here
10 one test of this claim of irreducible complexity
11 and the ability of Darwinian processes to make
12 complex systems, well, is to find a complex
13 system in a cell, destroy it, and then see if
14 random mutation and natural selection can come
15 back and replace it. And I have to say I agree
16 that's an excellent test of that claim. However,
17 I disagree with Professor Miller's further
18 comments and conclusions.

19 Q. What was the particular system that he was
20 looking at?

21 A. Well, he was referring to what is shown in
22 a little cartoon version on the next slide.
23 This is a figure again taken from that
24 biochemistry textbook by Voet and Voet
25 discussing a system called the lac operon.

1 Now, an operon is a little segment of DNA in
2 a bacteria which codes for a couple of genes,
3 and genes code for proteins, and the proteins
4 usually have related functions or function as a
5 group, and one of them is called the lac operon
6 which is used to, the proteins of which are
7 necessary for the bacterium Escherichia coli to
8 metabolize a sugar called lactose, which is a
9 milk sugar.

10 And it consists of a number of parts. No,
11 let's go back one slide, please, I'm sorry. All
12 these little squares here, this little green
13 thing represents a very complex protein called
14 a repressor, which will bind to the DNA, and
15 when it binds there it stops another protein
16 called an RNA preliemerase from binding to the
17 same spot, and therefore the information carried
18 by these genes is not expressed, and that's
19 important because the sugar lactose is usually
20 not present in the bacteria's environment, and
21 making proteins that metabolize lactose in the
22 absence of that sugar would be wasting energy.

23 So the bacterium wants to keep that turned
24 off until lactose is around. So the repressor
25 turns off the operon, and that means that the

1 genes for these three proteins here are not
2 turned on, not expressed. This first one, which
3 is labeled Z, codes, is the gene for a protein
4 called a beta galactosidase, okay? That's
5 actually the enzyme which chops up lactose.
6 We don't have to go into the detail of how
7 that happens.

8 This little thing marked Y codes for
9 something called a permease. Now, a permease
10 it turns out is a protein whose job it is to
11 allow the lactose to enter the bacterial cell.
12 The bacterial cell is surrounded by a membrane
13 which generally acts as a barrier to large
14 molecules, and there's this specialized protein,
15 this specialized machine called a permease
16 which, when lactose is around, grabs the lactose
17 from outside the cell, turns it around, and
18 allows it to enter to the inside of the cell.

19 In the absence of that permease the lactose
20 might be present in abundance in the bacteria's
21 environment, but it can't get inside the cell.
22 And so the bacterium can't use it. One other
23 detail of this before I go on is that this
24 repressor kind of sticks to the beginning of
25 the gene and turns it off, but when lactose is

1 present in the environment a small molecule
2 which is a derivative of lactose can bind to the
3 repressor, and that, and again start thinking in
4 terms of the complex shape and structure of
5 hemoglobin, when that happens it interacts in
6 specific ways in order and causes the shape of
7 the repressor to change, and that changed shape
8 makes it now no longer geometrically and
9 chemically complementary to the site that it
10 bound on the lac operon, and it falls off.

11 So in the presence of the inducer the repressor
12 falls off, this prelimerase can come along and
13 those proteins get made in the cell.

14 Q. Would you like the next slide?

15 A. Yes, thank you. Now I'm going to simplify,
16 after that discussion I'm going to try to
17 simplify nonetheless. So let me just list
18 some parts of the lac operon. There's the
19 galactosidase, the repressor, the permease, all
20 three of which are proteins, and something that
21 I've written IPTG/allolactose. That is the
22 small molecule which can bind to the repressor
23 and cause to it fall off of the operon,
24 allolactose is something, is a metabolite
25 of lactose itself, and that's the substance

1 which usually binds to the repressor in the
2 cell, but there's also an artificial chemical
3 called IPTG, which stands for isopropyl
4 thiogalactoside, which is sold by chemical
5 supply companies, which mimics the action of the
6 allolactose, and when a scientist comes and
7 dumps some IPTG into the beaker, that binds to
8 the repressor and causes those genes to be
9 expressed, to be turned on.

10 Okay, those are the parts of the lac
11 operon. Now, for purposes of further
12 illustration let me just mention that in
13 E. coli there are thousands of genes, and many
14 of them are grouped into operons. Unbeknownst
15 to the experimenter, whose name is Barry Hall,
16 there also existed in the E. coli another operon
17 called the EBG operon, which he called it that
18 because it stands for evolved beta
19 galactosidase. He thought this protein evolved
20 in response to the selective pressure that he
21 put on it, and it turns out that that operon
22 also codes for a galactosidase, another
23 galactosidase and another repressor as well.

24 Q. So this was the system that Dr. Miller was
25 talking about in --

1 A. Yes, I'm afraid this is the background for
2 the system that he started to discuss in his
3 book.

4 Q. Which he sees it as experimental evidence
5 to refute the irreducible complexity claim?

6 A. Yes, that's right, and if you look on the
7 next slide you'll see the part of his book where
8 he discusses that. He says of the system, he
9 says, "Think for a moment. If we were to happen
10 upon the interlocking biochemical complexity of
11 the re-evolved lactose system, wouldn't we be
12 impressed by the intelligence of its design.
13 Lactose triggers a regulatory sequence that
14 switches on the synthesis of an enzyme that then
15 metabolizes lactose itself.

16 "The products of that successful lactose
17 metabolism then activate the gene for the lac
18 permease, which ensures a steady supply of
19 lactose entering the cell. Irreducible
20 complexity, what good would the permease be
21 without the galactosidase? No good of course."
22 And he continues that same discussion on the
23 next slide, he continues, "By the very same
24 logic applied by Michael Behe to other systems,
25 therefore, we can conclude that this system had

1 been designed, except we know that it was not
2 designed. "We know it evolved, because we
3 watched it happen right in the laboratory. No
4 doubt about it, the evolution of biochemical
5 systems, even complex multipart ones, is
6 explicable in terms of evolution. Behe is
7 wrong."

8 Q. Is Dr. Miller right?

9 A. No. Dr. Miller is wrong. Now, Professor
10 Miller is always enthusiastic and he always
11 writes and speaks with great excitement, but I
12 say that when you examine his arguments closely,
13 under close inspection they simply don't hold up
14 and this is enormously exaggerated, and the
15 results of researcher Barry Hall that he is
16 describing here I would happily have included
17 as an example of irreducible complexity in
18 *Darwin's Black Box*.

19 So let me please try to explain why I say
20 that. Reading Professor Miller's prose one
21 would get, and I certainly did get when I first
22 read it, the impression that this system was
23 completely knocked out in that it completely
24 came back under the experiments that Barry Hall
25 conducted. But it turns out of this multipart

1 system, only one part, the protein beta
2 galactosidase, was knocked out by experimental
3 method.

4 Everything else, the repressor, the
5 permease, and we'll see later IPTG, and
6 importantly as well other proteins which did
7 very, very similar jobs in the cell, were left
8 behind. And the worker Barry Hall himself was
9 always very careful to say that he was only
10 knocking out that one protein.

11 Q. The galactosidase?

12 A. Yes, that's correct. I think on the next
13 slide he makes that point. This is a quotation
14 from a paper by Professor Hall recalling his
15 experiments that he did earlier on the lac
16 operon. He says the following, "All of the
17 other functions for lactose metabolism,
18 including lactose permease and the pathways
19 for metabolism of glucose and lactose, the
20 products of lactose hydrolysis, remain intact.
21 Thus, reacquisition of lactose utilization
22 requires only the evolution of a new," and this
23 should be a beta, "beta galactosidase function."

24 So let me point out that what he did in his
25 laboratory was to take an E. coli bacterium and

1 using molecular biological methods to knock out
2 or destroy the gene for that one part of the lac
3 operon, the beta galactosidase. He left the
4 permease intact, he left the repressor intact,
5 everything else was intact. He just had to get
6 one more component of the system.

7 And what he saw was that he did get
8 bacteria that were again able to use lactose.
9 And when he did the experiments in the 1970's,
10 that's all he saw. He saw he had bacteria that
11 could grow when they were fed lactose. But
12 years later after methods had developed and
13 after he had the ability to do so, he asked
14 himself what protein was it that took over the
15 role of the beta galactosidase, and he named it
16 EBG, evolved beta galactosidase.

17 But when he looked at it further he found
18 it to be a very similar protein to the one that
19 he had knocked out. Essentially it was almost
20 a spare copy of the protein that had been
21 destroyed. So this slide makes a couple of
22 points. Let me just point to a couple. The
23 EBG protein that took the place of the beta
24 galactosidase is homologous to lac proteins.
25 That's a technical term, that means they're

1 very similar. Their protein structures, their
2 sequences are pretty similar, and odds are
3 good that they have the same sort of activity.

4 What's more, after further investigation
5 Professor Hall showed that even the unmutated,
6 even the EBG galactosidase before he did his
7 experiment, the unmutated galactosidase could
8 already hydrolyze, although it was inefficient.
9 So again this was almost a spare copy of the
10 protein, and I think on the next slide, I'll
11 skip that last point on the next slide to drive
12 home the point I want to show you what are the
13 amino acid sequences of the area around what's
14 called the active site of the protein, which is
15 kind of the business end where the lactose binds
16 and where the chemical groups reside which will
17 cause it to be hydrolyzed into two component
18 parts.

19 Notice this. Look at these sequence of
20 letters. Now, I know that they don't mean much
21 to most people in here, but notice the sequence
22 of letters, these are the amino acid sequences,
23 abbreviations for the amino acid sequence of
24 various beta galactosidase enzymes found in
25 E. coli and a related species. Notice here,

1 let's start in here, there's an R here,
2 HEHEMYEHW. Look up top, there's RHEHEMYEHW, the
3 same thing on the lower one, too. They're
4 active sites, their business ends are almost
5 identical. Like I said, these are essentially
6 spare copies of each other.

7 Q. So in fact it wasn't a new evolved element
8 to this system. It was a spare part that was
9 already existing?

10 A. Well, it was there and it did undergo small
11 changes. But nobody, nobody denies that
12 Darwinian evolution can make small changes in
13 preexisting systems. Professor Miller was
14 claiming that a whole new lactose utilizing
15 system had been evolved in Barry Hall's
16 laboratory, and that's, you know, that's very,
17 very greatly exaggerated.

18 Q. Again do you have additional slides to
19 emphasize the point?

20 A. Yes. This might be hard to explain, but
21 Professor Hall says in one of his papers that,
22 "The evidence indicates that either AS-92 and
23 sys trip 977," these are the same of some amino
24 acids, "are the only acceptable amino acids at
25 those positions, or that all of the single based

1 substitutions that might be on the pathway to
2 other amino acid replacements at those sites,
3 are so deleterious that they constitute a deep
4 selective valley that have not been transversed
5 in the two billion years since those proteins
6 emerged from a common ancestor." Now, translated
7 into --

8 Q. Yes, please into English.

9 A. -- more common language, that means that
10 that very similar protein could only work if
11 it became even more similar to the beta
12 galactosidase that it replaced, and if you
13 then also knock out that EBG galactosidase, no
14 other protein in Professor hall's experience was
15 able to substitute for the beta galactosidase.
16 So the bottom line, the bottom line is that the
17 only thing demonstrated was that you can get
18 tiny changes in preexisting systems, tiny
19 changes in preexisting systems, which of course
20 everybody already had admitted.

21 Another interesting point, another
22 interesting point is shown on that figure
23 from Voet and Voet, the inducer, this little
24 red dot, this little red dot actually stands
25 for this chemical that binds to the repressor

1 which changes its shape which causes it to fall
2 off of the operon and allow the premerase to
3 come in and transcribe that information. Well,
4 it turns out that the EBG operon, this place in
5 the DNA and E. coli that had that spare beta
6 galactosidase, did not have a spare permease.

7 So the system was stuck, because it didn't
8 have its own permease. When the repressor binds
9 to this operon, the normal lac operon, if there
10 weren't any lactose around then the repressor
11 would be essentially stuck there indefinitely.
12 And even if lactose were present outside the
13 cell, it had no way to get inside the cell. So
14 what Barry Hall did to allow his experiment to
15 continue was that he added the inducer. He
16 added that artificial chemical IPTG that he can
17 buy from a chemical supply house, and he took
18 some and sprinkled it in the beaker for the
19 specific purpose of allowing the bacteria to
20 survive so that it could take these small little
21 steps to produce a new beta galactosidase.

22 **Q.** You have a slide to demonstrate that?

23 **A.** Yes. And Barry Hall was always very
24 careful to explain exactly how these experiments
25 were performed, and he brought it directly to

1 the attention of readers when he described his
2 system. For example he writes, "At this point
3 it is important to discuss the use of IPTG in
4 these studies. Unless otherwise indicated, IPTG
5 is always included in media containing lactose,"
6 and that italics is Barry Hall's emphasis. He
7 wanted to make sure his reader understood
8 exactly what he was doing.

9 "The sole function of the IPTG is to induce
10 synthesis of the lactose permease and thus to
11 deliver lactose to the inside of the cell.

12 Neither constitutive nor the inducible of all
13 strains grew on lactose in the absence of IPTG."

14 In other words, if this intelligent agent, Barry
15 Hall, had not gone to the store and gotten some
16 IPTG to help the bacteria survive, they would
17 not have lived. This would not have occurred in
18 the wild. This tells us virtually nothing about
19 how Darwinian evolution could produce complex
20 molecular systems.

21 Q. So again this system would not have worked
22 in nature but for Barry Hall interjecting the
23 IPTG to make this system work?

24 A. Yes. I should point out that Professor
25 Miller does not mention this aspect of Barry

1 Hall's experiments in his discussion, in his
2 book *Finding Darwin's God*.

3 Q. Is that a significant oversight?

4 A. Well, I certainly would have included it.

5 MR. MUISE: Your Honor, we're about to move
6 into the blood clotting system, which is really
7 complex.

8 THE COURT: Really? We've certainly
9 absorbed a lot, haven't we?

10 MR. MUISE: We certainly have, Your Honor.
11 This is Biology 2. It's a quarter past, and if
12 we're going to go until 4:30, it's probably not
13 worthwhile to start up on the blood clotting
14 because it's fairly complex and heavy and a lot
15 of it is going to be --

16 THE COURT: Well, we don't have an issue as
17 to his availability through the day tomorrow I
18 assume?

19 MR. MUISE: He's available, Your Honor, for
20 as long as we need him.

21 THE COURT: Any objection if we --

22 MR. ROTHSCHILD: No. He started it.

23 THE COURT: I was just waiting to see what
24 you'd say.

25 MR. MUISE: We've gone from Biology 101 to

1 advanced biology. So this is where we get.

2 THE COURT: We will recess then for today,
3 and we'll reconvene at 9:00 tomorrow and
4 we will pick up with Mr. Muise's direct
5 examination at that time. So have a pleasant
6 good evening, and we'll see you tomorrow.

7 (Court was adjourned at 4:15 p.m.)

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1 Tammy Kitzmiller, et al. vs. Dover Schools

2 4:04-CV-02688

3 Trial Day 10, Afternoon Session

4 17 October 2005

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I hereby certify that the proceedings
9 and evidence are contained fully and accurately
10 in the notes taken by me on the trial of the
11 above cause, and that this copy is a correct
12 transcript of the same.

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s/ Wesley J. Armstrong

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Wesley J. Armstrong

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