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

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

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

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I N D E X
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PROCEEDINGS

P R O C E E D I N G S

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

DIRECT EXAMINATION CONTINUED

BY MR. MUISE:

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

A. Yes, it seems to be.

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

A. Yes, I've heard it.

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

A. Well, that's certainly one definition of

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

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

A. Yes, in the scientific community itself.

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

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

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

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

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

So they had very good evidence to support their view. Furthermore, that theory was used for ages to help sailors and so on navigate the seas. So it was pretty well substantiated. Nonetheless, of course as everybody knows it turned out to be incorrect, and Copernicus proposed that in fact the sun is the center of the solar system and that the earth, while revolving on its axis, travels around the sun. So again that's an old example, but nonetheless it shows that a well accepted theory nonetheless is not necessarily correct.

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

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

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

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

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

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

physicists as well.

So he was writing of the idea as it was commonly held at that time in the highest levels of physics, and he wrote the following: "Whatever difficulties we may have in forming a consistent idea of the constitution of the ether, there can be no doubt that the interplanetary and interstellar spaces are not empty, but are occupied by a material substance or body which is certainly the largest and probably the most uniform body of which we have any knowledge."

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

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

of light by the sun, and it turns out he's using that to do calculations, and in the calculations he is deducing the properties of the ether. For example, these large red arrows are pointing to the coefficient of rigidity of ether, which is given by the formula Ro V squared, which is 842.8.

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

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

is that correct?

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

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

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

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

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

He says, "In both scholarly and popular

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

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

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

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

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

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

Q. Go ahead.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

A. Oh, okay. Yes.

Q. Is that the search that you conducted on

Pub Med in which the slides are derived from?

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

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

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

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

So again a scientist here is using the

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

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

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

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

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

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

Q. Does intelligent design refute the occurrence of evolution?

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

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

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

A. No, they are completely inaccurate.

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

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

Q. Is common descent nevertheless addressed in Pandas?

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

29 Q. How does it fit then within intelligent design?

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

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

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

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

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

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

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

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

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

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

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

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

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

34 Q. And you said this was published in The Reports by the National Center for Science Education?

A. Yes, that's correct.

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

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

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

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

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

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

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

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

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

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

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

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

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

39 Q. Do any other scientist besides intelligent design proponents question the ability of natural selection to explain various aspects of life?

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

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

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

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

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

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

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

42 Q. Now, the evolution as such, understanding

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

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

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

A. No. No theory is a fact.

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

A. Yes, there are.

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

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

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

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

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

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

MR. ROTHSCHILD: Thank you, judge.

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

BY MR. MUISE:

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

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

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

47 Q. Has Dr. Miller acknowledged such problems?

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

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. No.

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

A. Yes, that's correct.

Q. Does this cause you to question whether a

Darwinian framework is the right way to approach such questions?

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

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

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

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

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

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

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

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

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

very, very much different.

And on the next slide are excerpts from the news story. It was written, it says, "Generations of biology students may have been misled by a famous set of drawings of embryos published 123 years ago by Ernst Haeckel. 'The impression they give that the embryos are exactly alike is wrong,' says Michael Richardson, an embryologist at St. George's Hospital Medical School in London," and he was the lead author of the study which showed the incorrectness of Haeckel's results.

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

It doesn't predict anything. It will live

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

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

A. Number 16 did you say?

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

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

65 Q. Was the article written by Elizabeth --

A. Pennisi.

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

A. Yes.

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

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

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

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

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

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

69 Q. This was written in 1993?

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

the evolution of reproduction are still obscure."

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

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

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

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

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

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

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

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

A. Yes, that's right.

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

to cite?

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

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

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

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

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

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

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

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

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

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

77 Q. Is this an unsolved scientific problem?

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

78 Q. What is that problem?

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

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

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

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

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

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

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

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

A. Yes, it is.

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

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

82 Q. Do you have any problems with this statement?

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

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

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

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

A. Yes, there is.

84 Q. And what leads you to that conclusion?

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

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

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

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

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

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

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

MR. ROTHSCHILD: Objection, Your Honor. I think it's mischaracterizing the title.

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

MR. ROTHSCHILD: I think he did say it, Your Honor.

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

THE COURT: How did he mischaracterize it?

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

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

MR. ROTHSCHILD: It think it's a loaded question.

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

MR. ROTHSCHILD: Sorry, Intelligent Design, Creationism, and Its Critics, I am corrected.

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

(Brief pause.)

MR. MUISE: Let's go back to your --
THE COURT: Just so we're --
MR. MUISE: I do have it here, Your Honor, and I just want to make it clear what the title is, and I believe Mr. Rothschild is accurate.
THE COURT: All right. Then there's no need for a ruling on it. You can just clarify it for the record.
BY MR. MUISE:
88 Q. The book by Robert T. Pennock was entitled Intelligent Design, Creationism and Its Critics: Philosophical, Theological and Scientific Perspectives, is that correct?
A. That's correct.
89 Q. And that book was published by the MIT Press?
A. That's correct, yes.
90 Q. You contributed an article making scientific arguments for intelligent design in that book?
A. That's correct, I did.
91 Q. I should clarify, you submitted a chapter, is that correct?
A. Yes that's, right.
92 Q. Were there other scientists who submitted

chapters in that particular book?

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

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

A. Yes.

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

A. Yes, that's right.

95 Q. Published by Michigan State University Press?

A. Yes, that's correct.

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

A. Yes, that's right.

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

A. Yes.

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

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

99 Q. And who are Thornhill and Ussery?

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

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

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

101 Q. What was that article about?

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

102 Q. Did it particularly refer to irreducible complexity?

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

as well to illustrate the problem.

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

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

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

THE COURT: No, keep going.

MR. MUISE: Okay.

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

BY MR. MUISE:

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

A. Yes, that's right.

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

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

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

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

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

A. Yes, that's right.

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

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

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

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

108 Q. Have other scientists acknowledged that this is an argument against Darwin's theory of evolution?

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

109 Q. I believe we have a quote from another prominent scientist?

A. Yes. Richard Dawkins in his recent book

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

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

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

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

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

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

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

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

112 Q. Is it a common practice within the science

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

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

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

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

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

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

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

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

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

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

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

When you go beyond a cell, then you're

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

A. No, I don't.

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

your objections to his claim.

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

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

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

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

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

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

But as I have explained throughout the day today, if we could go to the next slide, that an inability to explain something is not the argument for design. The argument for design is when we perceive the purposeful arrangement of parts, the purposeful arrangement of parts such as we see in the flagellum, such as we see the molecular machinery such as described in that special issue of Cell and so on.

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

127 Q. Have you addressed such a claim in Darwin'S Black Box?

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

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

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

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

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

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

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

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

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

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

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

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

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

that actual molecules would encounter in the cell.

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

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

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

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

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

134 Q. Yes, go ahead.

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

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

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

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

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

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

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

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

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

CONTINUED DIRECT BY MR. MUISE:

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

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

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

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

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

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

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

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

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

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

Here each very, very complex protein chain is rendered as a simple square, and the O sub 2 represents the oxygen that each protein is supposed to be carrying. Now, all of these are legitimate renderings of the protein hemoglobin, but when we discuss these matters and we discuss difficulties with evolution and we discuss arguments for intelligent design, we

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

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

A. That's right, uh-huh.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

141 Q. A difficulty of getting two changes at once?

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

142 Q. And have other scientists made similar observations?

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

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

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

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

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

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

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

A. Yes, that's the same one.

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

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

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

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

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

A. Yes. That's Figure 2.

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

testified to previously?

A. Yes, that's correct.

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

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

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

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

150 Q. As well as that previous diagram?

A. Yes, that's correct.

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

A. That's correct.

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

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

153 Q. Why do you disagree?

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

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

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

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

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

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

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

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

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

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

bacterial flagellum.

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

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

156 Q. Have scientists reached different conclusions?

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

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

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

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

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

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

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

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

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

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

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

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

159 Q. Dr. Behe, so what do these widely different opinions mean?

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

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

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

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

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

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

THE COURT: You may.

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

A. Yes.

163 Q. Is that the study from Nguyen that you

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

A. Yes, that's correct.

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

A. Thank you.

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

A. That's exactly right.

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

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

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

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

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

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

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

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

And let me make a point about this. Professor Padian is saying that molecular studies of DNA sequence of whales and hippos suggested or led to the suggestion that both animals had aquatic ancestors. But they didn't. They had terrestrial ancestors. That means that the molecular information is compatible with either result, with the ancestors being aquatic or the ancestors being terrestrial.

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

that means that does not speak to Darwin's claim that natural selection drove evolution, okay? Well, molecular data can't decide the question.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

A. Yes, that's right.

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

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

Irreducible complexity, on the other hand,

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

174 Q. So an argument against irreducible complexity is not necessarily an argument against design?

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

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

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

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

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

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

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

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

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

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

A. Yes.

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

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

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

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

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

179 Q. And one of those claims was raised by

Dr. Miller, is that correct?

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

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

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

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

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

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

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

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

This little thing marked Y codes for something called a permease. Now, a permease it turns out is a protein who is job it is to allow the lactose to enter the bacterial cell. The bacterial cell is surrounded by a membrane which generally acts as a barrier to largish molecules, and there's this specialized protein, this specialized machine called a permease which, when lactose is around, grabs the lactose from outside the cell, turns it around, and allows it to enter to the inside of the cell.

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

present in the environment a small molecule which is a derivative of lactose can bind to the repressor, and that, and again start thinking in terms of the complex shape and structure of hemoglobin, when that happens it interacts in specific ways in order and causes the shape of the repressor to change, and that changed shape makes it now no longer geometrically and chemically complementary to the site that it bound on the lac operon, and it falls off. So in the presence of the inducer the repressor falls off, this prelimerase can come along and those proteins get made in the cell.

181 Q. Would you like the next slide?

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

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

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

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

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

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

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

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

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

184 Q. Is Dr. Miller right?

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

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

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

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

185 Q. The galactosidase?

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

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

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

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

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

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

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

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

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

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

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

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

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

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

188 Q. Yes, please into English.

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

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

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

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

189 Q. You have a slide to demonstrate that?

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

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

"The sole function of the IPTG is to induce synthesis of the lactose permease and thus to deliver lactose to the inside of the cell. Neither constitutive nor the inducible of all strains grew on lactose in the absence of IPTG." In other words, if this intelligent agent, Barry Hall, had not gone to the store and gotten some IPTG to help the bacteria survive, they would not have lived. This would not have occurred in the wild. This tells us virtually nothing about how Darwinian evolution could produce complex molecular systems.

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

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

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

191 Q. Is that a significant oversight?

A. Well, I certainly would have included it.

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

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

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

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

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

THE COURT: Any objection if we --

MR. ROTHSCHILD: No. He started it.

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

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

advanced biology. So this is where we get.

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

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

Tammy Kitzmiller, et al. vs. Dover Schools

4:04-CV-02688

Trial Day 10, Afternoon Session

17 October 2005

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

s/ Wesley J. Armstrong

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Wesley J. Armstrong

Registered Merit Reporter

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