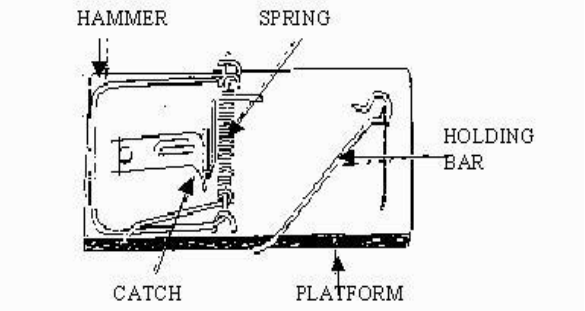
Intelligent Design Postulate: Life's Irreducible Complexity

Irreducible Complexity: The Challenge to Darwinian Evolutionary Explanations of Biochemical Structures

> ***"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."***
>
> --Charles Darwin, Origin of Species

With this statement, Charles Darwin provided a criterion by which his theory of evolution could be falsified. The logic was simple: since evolution is a gradual process in which slight modifications produce advantages for survival, it cannot produce complex structures in a short amount of time. It's a step-by-step process which may gradually build up and modify complex structures, but it cannot produce them suddenly.

Darwin, meet Michael Behe, biochemical researcher and professor at Lehigh University in Pennsylvania. Michale Behe claims to have shown exactly what Darwin claimed would destroy the theory of evolution, through a concept he calls "irreducible complexity." In simple terms, this idea applies to any system of interacting parts in which the removal of any one part destroys the function of the entire system. An irreducibly complex system, then, requires each and every component to be in place before it will function. As a simple example of irreducible complexity, Behe presents the humble mousetrap.



It contains five interdependent parts which allow it to catch mice: the wooden platform, the spring, the hammer (the bar which crushes the mouse against the wooden base), the holding bar, and a catch. Each of these components is absolutely essential for the function of the mousetrap. For instance, if you remove the catch, you cannot set the trap and it will never catch mice, no matter how long they may dance over the contraption. Remove the spring, and the hammer will flop uselessly back and forth-certainly not much of a threat to the little rodents. Of course, removal of the holding bar will ensure that the trap never catches anything because there will again be no way to arm the system.

Now, note what this implies: an irreducibly complex system cannot come about in a gradual manner. One cannot begin with a wooden platform and catch a few mice, then add a spring, catching a few more mice than before, etc. No, all the components must be in place before it functions at all. A step-by-step approach to constructing such a system will result in a useless system until all the components have been added. The system requires all the components to be added at the same time, in the right configuration, before it works at all.

How does irreducible complexity apply to biology? Behe notes that early this century, before biologists really understood the cell, they had a very simplistic model of its inner workings. Without the electron microscopes and other advanced techniques that now allow scientists to peer into the inner workings of the cell, it was assumed that the cells was a fairly simple blob of protoplasm. The living cell was a "black box"-something that could be observed to perform various functions while its inner workings were unknown and mysterious. Therefore, it was easy, and justifiable, to assume that the cell was a simple collection of molecules. But not anymore. Technological advances have provided detailed information about the inner workings of the cell. Michael Denton, in his book Evolution: A Theory in Crisis, states "Although the tiniest bacterial cells are incredibly small, weighing less than 10^-12 grams, each is in effect a veritable microminiaturized factory containing thousands of exquisitely designed pieces of intricate molecular machinery, made up altogether of one hundred thousand million atoms, far more complicated than any machine built by man and absolutely without parallel in the non-living world." In a word, the cell is complicated. Very complicated.

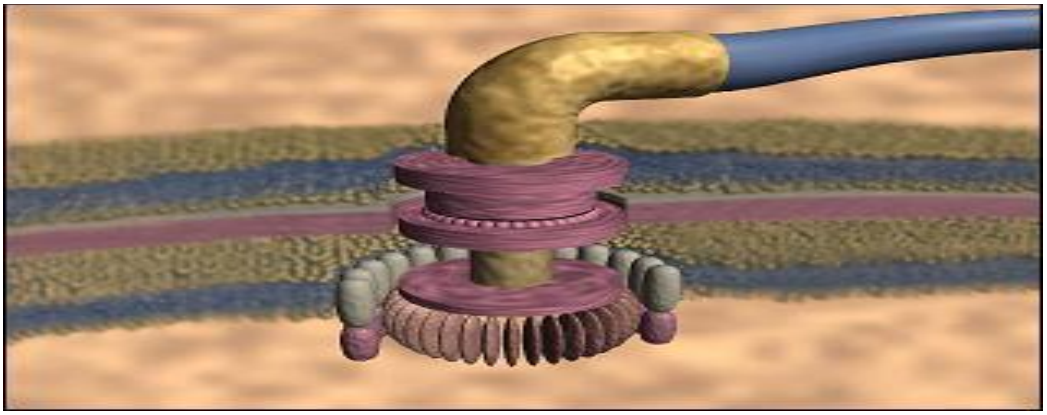
In fact, Michael Behe asserts that the complicated biological structures in a cell exhibit the exact same irreducible complexity that we saw in the mousetrap example. In other words, they are all-or-nothing: either everything is there and it works, or something is missing and it doesn't work. As we saw before, such a system cannot be constructed in a gradual manner-it simply won't work until all the components are present, and Darwinism has no mechanism for adding all the components at once.

Remember, Darwin's mechanism is one of gradual mutations leading to improved fitness and survival. A less-than-complete system of this nature simply will not function, and it certainly won't help the organism to survive. Indeed, having a half-formed and hence non-functional system would actually hinder survival and would be selected against. But Behe isn't the only scientist recognizing irreducible complexity in nature. In 1986, Michael J. Katz, in his *Templets and the explanation of complex patterns* (Cambridge: Cambridge University Press, 1986) writes:

> ***"In the natural world, there are many pattern-assembly systems for which there is no simple explanation. There are useful scientific explanations for these complex systems, but the final patterns that they produce are so heterogeneous that they cannot effectively be reduced to smaller or less intricate predecessor components. As I will argue ... these patterns are, in a fundamental sense, irreducibly complex..."***

Katz continues that this sort of complexity is found in biology:

> ***"Cells and organisms are quite complex by all pattern criteria. They are built of heterogeneous elements arranged in heterogeneous configurations, and they do not self-assemble. One cannot stir together the parts of a cell or of an organism and spontaneously assemble a neuron or a walrus: to create a cell or an organisms one needs a preexisting cell or a preexisting organism, with its attendant complex templets. A fundamental characteristic of the biological realm is that organisms are complex patterns, and, for its creation, life requires extensive, and essentially maximal, templets."***



The bacterial flagellum is a cellular outboard motor that bears the marks of intelligent design.

Behe presents several examples of irreducibly complex systems to prove his point, but I'll just focus on one: the cilium. Cilia are hair-like structures, which are used by animals and plants to move fluid over various surfaces (example, cilia in your respiratory tree sweep mucous towards the throat and promote elimination of contaminants) & by single-celled organisms to move through water. Cilia are like "oars" to contain their own mechanism for bending. That mechanism involves tiny rod-like basic structures called microtubules arranged in a ring. Adjacent microtubules are connected to each other by two types of "bridges" - a flexible linker bridge and an arm that can "walk" up neighboring microtubule. The cilia bends by activating the "walker" arms, and the sliding motion that this tends to generate is converted to a bending motion by the flexible linker bridges.

Thus, the cilium has several essential components: stiff microtubules, linker bridges, and the "motors" in the form of walker arms. While my description is greatly simplified (Behe notes that over 200 separate proteins have been identified in this particular system), these 3 components form the basic system, and show what is required for functionality. For without one of these components, the system simply will not function. We can't evolve a cilium by starting with microtubules alone, because the microtubules will be fixed and rigid-not much good for moving around. Adding the flexible linker bridges to the system will not do any good either-there is still no motor and the cilia still will not bend. If we have microtubules and the walker arms (the motors) but no flexible linker arms, the microtubules will keep on sliding past each other till they float away from each other and are lost.

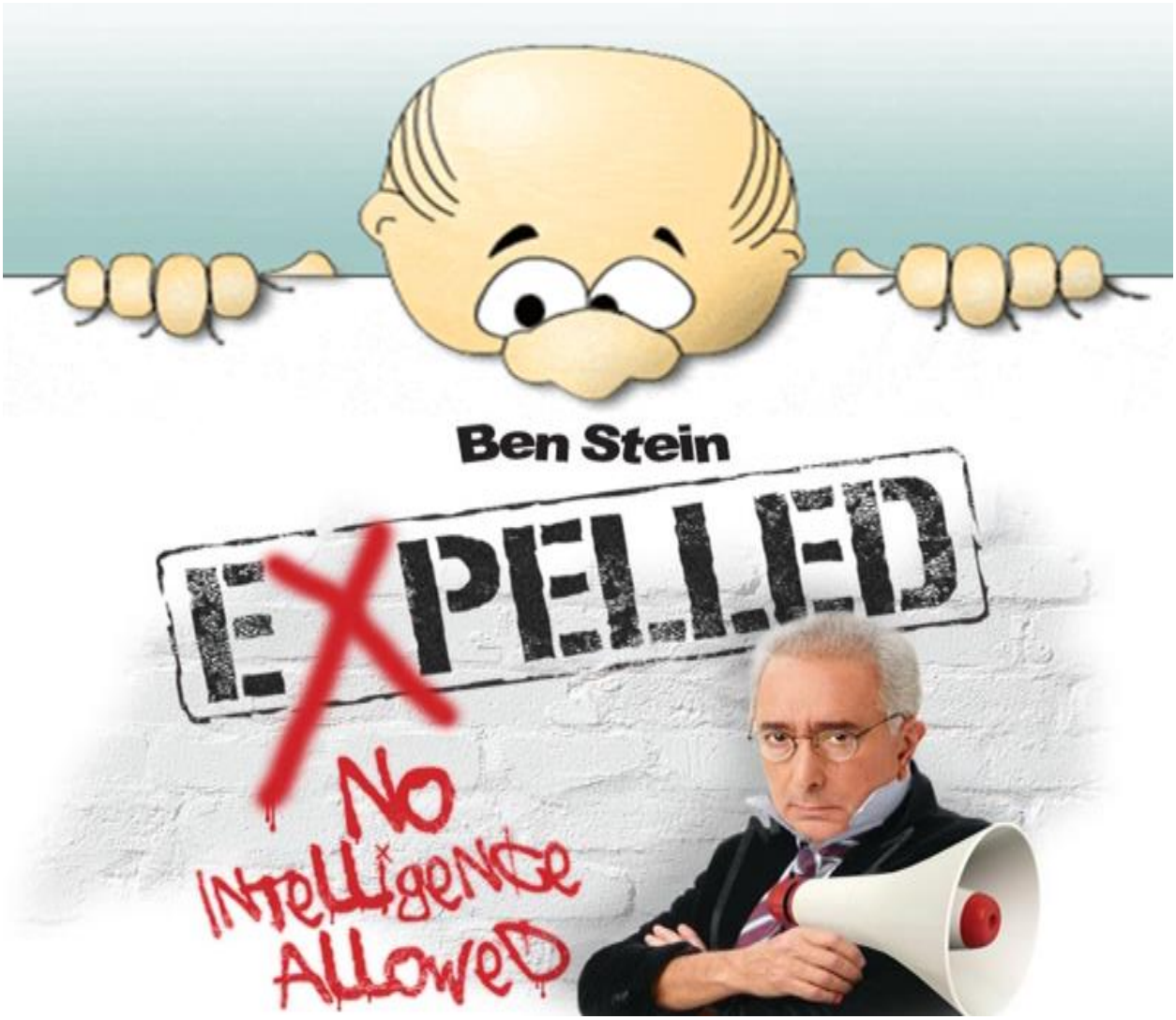
This is only one of many biochemical systems that Behe discusses in his book, Darwin's Black Box. Other examples of irreducible complexity include the light-sensing system in animal eyes, the transport system within the cell, the bacterial flagellum, and the blood clotting system. All consist of a very complex system of interacting parts which cannot be simplified while maintaining functionality.

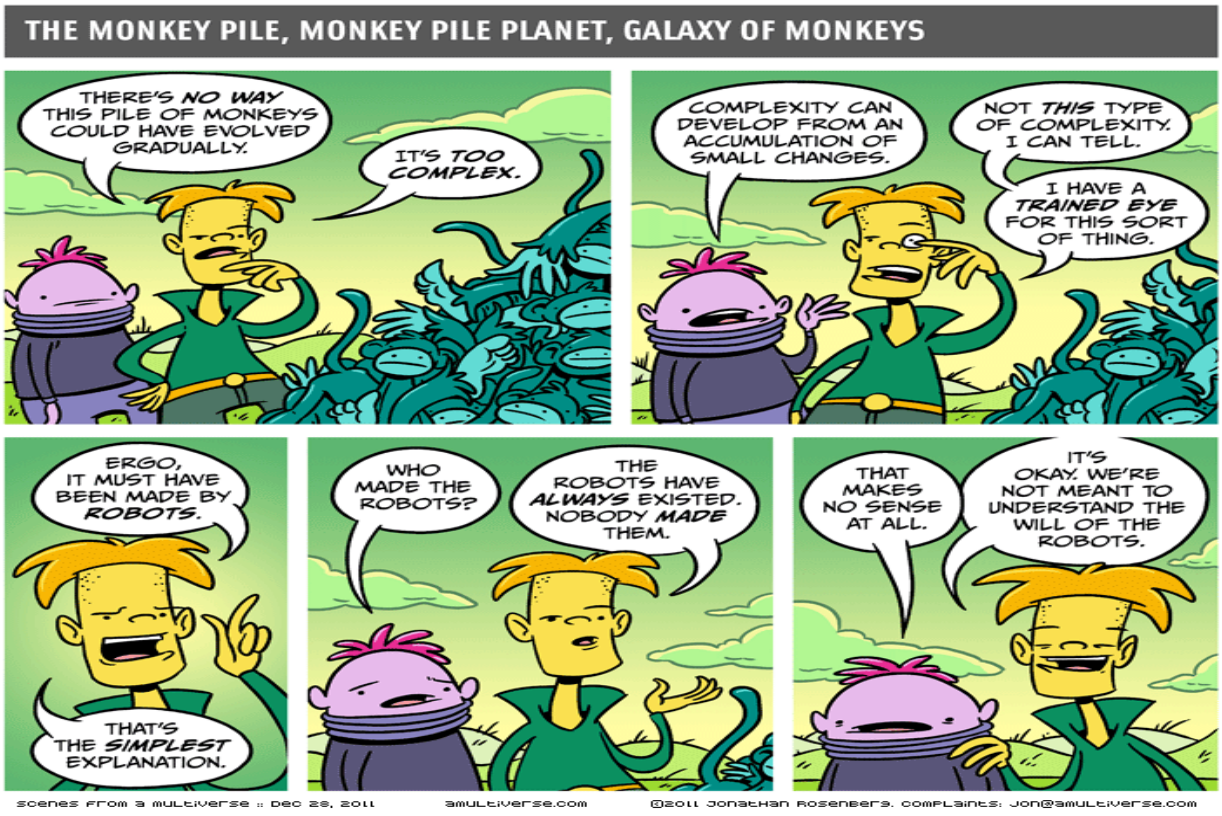
Since the publication of Darwin's Black Box, Behe has refined the definition of irreducible complexity. In 1996 he wrote that "any precursor to an irreducibly complex system that is missing a part is by definition nonfunctional."(Behe, M)

Evidence for Intelligent Design from Biochemistry, a speech given at the Discovery Institute's God & Culture Conference, August 10, 1996 Seattle, WA. http://www.arn.org/docs/behe/mb_idfrombiochemistry.htm). By defining irreducible complexity in terms of "non-functionality," Behe casts light on the fundamental problem with evolutionary theory: evolution can't produce something where there would be a non-functional intermediate. Natural selection only preserves or "selects" those structures which are functional. If it is not functional, it cannot be naturally selected. Thus, Behe's latest definition of irreducible complexity is as follows:

> ***"An irreducibly complex evolutionary pathway is one that contains one or more unselected steps (that is, one or more necessary-but-unselected mutations). The degree of irreducible complexity is the number of unselected steps in the pathway." (A Response to Critics of Darwin's Black Box, by Michael Behe, PCID, Volume 1.1, January February March, 2002; iscid.org/)***

Evolution simply cannot produce complex structures in a single generation as would be required for the formation of irreducibly complex systems. To imagine that a chance set of mutations would produce all two hundred proteins required for cilia function in a single generation stretches the imagination beyond the breaking point. And yet, producing one or a few of these proteins at a time, in standard Darwinian fashion, would convey no survival advantage because those few proteins would have no function-indeed, they would constitute a waste of energy for the cell to even produce. Darwin recognized this as a potent threat to his theory of evolution-the issue that could completely disprove his idea. So, the question must be raised: Has Darwin's theory of evolution "absolutely broken down?" According to Michael Behe, the answer is a resounding "yes."


Ben Stein
EXPELLED
No
Intelligence
Allowed


THE MONKEY PILE, MONKEY PILE PLANET, GALAXY OF MONKEYS
THERE'S NO WAY THIS PILE OF MONKEYS COULD HAVE EVOLVED GRADUALLY.
IT'S TOO COMPLEX.
COMPLEXITY CAN DEVELOP FROM AN ACCUMULATION OF SMALL CHANGES.
NOT THIS TYPE OF COMPLEXITY. I CAN TELL.
I HAVE A TRAINED EYE FOR THIS SORT OF THING.
ERGO, IT MUST HAVE BEEN MADE BY ROBOTS.
THAT'S THE SIMPLEST EXPLANATION.
WHO MADE THE ROBOTS?
THE ROBOTS HAVE ALWAYS EXISTED. NOBODY MADE THEM.
THAT MAKES NO SENSE AT ALL.
IT'S OKAY. WE'RE NOT MEANT TO UNDERSTAND THE WILL OF THE ROBOTS.
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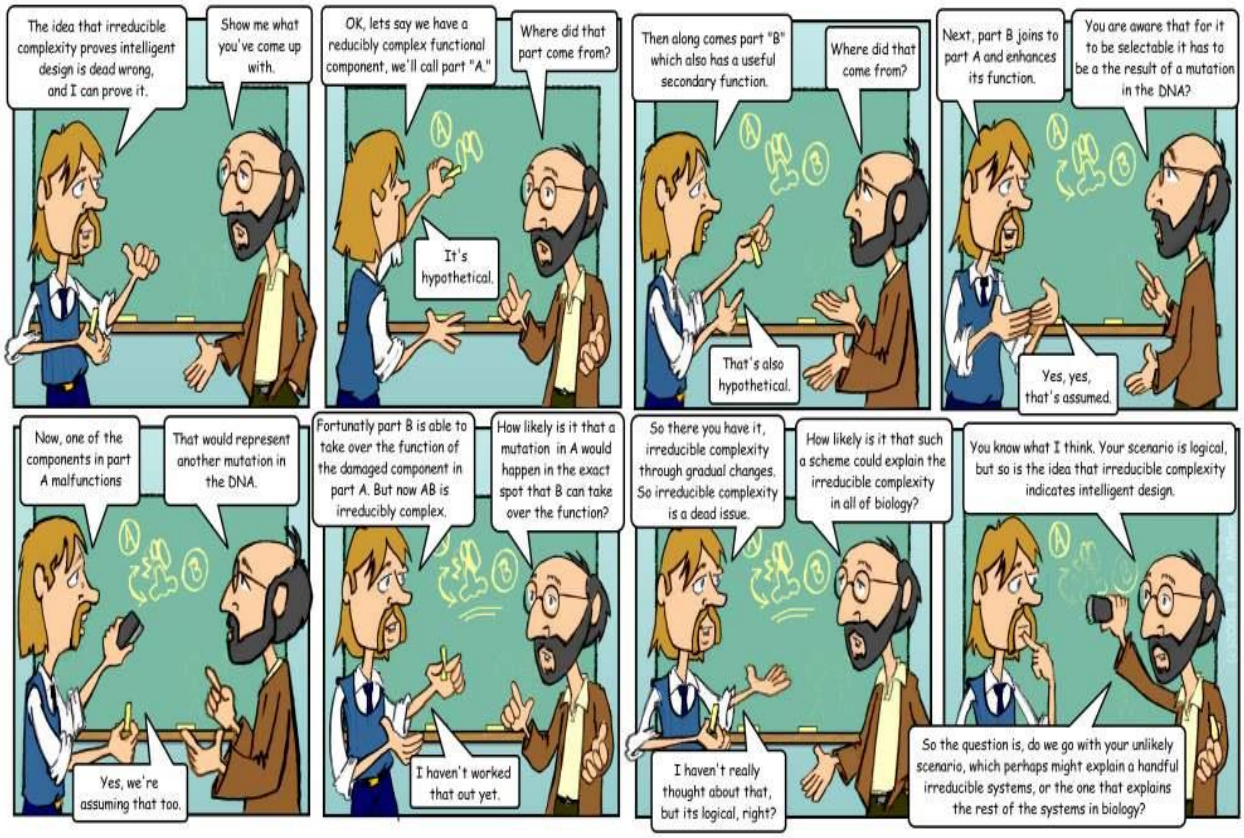

The idea that irreducible complexity proves intelligent design is dead wrong, and I can prove it.
Show me what you've come up with.
OK, lets say we have a reducibly complex functional component, we'll call part "A."
Where did that part come from?
It's hypothetical.
Then along comes part "B" which also has a useful secondary function.
Where did that come from?
That's also hypothetical.
Next, part B joins to part A and enhances its function.
You are aware that for it to be selectable it has to be a the result of a mutation in the DNA?
Yes, yes, that's assumed.
Now, one of the components in part A malfunctions
That would represent another mutation in the DNA.
Yes, we're assuming that too.
Fortunatly part B is able to take over the function of the damaged component in part A. But now AB is irreducibly complex.
How likely is it that a mutation in A would happen in the exact spot that B can take over the function?
I haven't worked that out yet.
So there you have it, irreducible complexity through gradual changes. So irreducible complexity is a dead issue.
How likely is it that such a scheme could explain the irreducible complexity in all of biology?
I haven't really thought about that, but its logical, right?
You know what I think. Your scenario is logical, but so is the idea that irreducible complexity indicates intelligent design.
So the question is, do we go with your unlikely scenario, which perhaps might explain a handful irreducible systems, or the one that explains the rest of the systems in biology?

Argument: ‘Irreducible complexity’

. *Scientific American* states the problem this way:

14. Living things have fantastically intricate features—at the anatomical, cellular and molecular level— that could not function if they were any less complex or sophisticated. The only prudent conclusion is that they are the products of intelligent design, not evolution.

This ‘argument from design’ is the backbone of most recent attacks on evolution, but it is also one of the oldest. In 1802, theologian William Paley wrote that if one finds a pocket watch in a field, the most reasonable conclusion is that someone dropped it, not that natural forces created it there. By analogy, Paley argued, the complex structures of living things must be the handiwork of direct, divine invention. Darwin wrote *On the Origin of Species* as an answer to Paley: he explained how natural forces of selection, acting on inherited features, could gradually shape the evolution of ornate organic structures. [*SA* 83]

Indeed, Gould, who was an expert on the history of evolution, agreed that Darwin was writing to counter Paley. This is another way of saying that he had an anti-theistic agenda,[1] as discussed in chapter 2. This doesn’t stop many churchian academics kowtowing to every pronouncement made by Darwin and his God-hating successors, who in return regard them as contemptuously as Lenin regarded his ‘useful idiot’ allies in the West.[2]

Could the eye have evolved?

It’s interesting to note that the eye, which evolutionists claim is an example of ‘bad design’ leftover from evolution (previous chapter), presents their greatest challenge as an example of superb ‘irreducible complexity’ in God’s creation.

Scientific American says:

Generations of creationists have tried to counter Darwin by citing the example of the eye as a structure that could not have evolved. The eye's ability to provide vision depends on the perfect arrangement of its parts, these critics say. Natural selection could thus never favor the transitional forms needed during the eye's evolution—what good is half an eye? Anticipating this criticism, Darwin suggested that even 'incomplete' eyes might confer benefits (such as helping creatures orient toward light) and thereby survive for further evolutionary refinement. [*SA* 83]

First, this overlooks the incredible complexity of even the simplest light-sensitive spot. Second, it's fallacious to argue that 51 percent vision would necessarily have a strong enough selective advantage over 50 percent to overcome the effects of genetic drift's tendency to eliminate even beneficial mutations.[3]

Biology has vindicated Darwin: researchers have identified primitive eyes and light-sensing organs throughout the animal kingdom and have even tracked the evolutionary history of eyes through comparative genetics. (It now appears that in various families of organisms, eyes have evolved independently.) [*SA* 83]

Scientific American **contradicts itself here.** If the evolutionary history of eyes has been tracked through comparative genetics, how is it that eyes have supposedly evolved independently? Actually, evolutionists recognize that eyes must have arisen independently at least 30 times because there is no evolutionary pattern to explain the origin of eyes from a common ancestor. What this really means is that since eyes cannot be related by common ancestor, and since they are here, and only materialistic explanations are allowed, hey presto, there's proof that they evolved independently!

Simulation of eye evolution

PBS 1 goes to great lengths to convince us that the eye could easily have evolved. Dan Nilsson explained a simplistic computer simulation he published in a widely publicized paper.[4] Taking his cue from Darwin, who started with a light-sensitive spot when 'explaining' the origin of the eye, Nilsson's simulation starts with a light-sensitive layer, with a transparent coating in front and a light-absorbing layer behind.

Here is how the simulation proceeds. Firstly, the light-sensitive layer bends gradually into a cup, so it can tell the direction of light rays increasingly well. This continues until it is curved into a hemisphere filled with the transparent substance.

Secondly, bringing the ends together, closing the aperture, gradually increases the sharpness of the image, as a pinhole camera does, because a smaller hole cuts out light. But because of the diffraction of light if the hole is too small, there is a limit to this process. So thirdly, the shape & refractive index gradient of the transparent cover change gradually to a finely focusing lens. Even if we were generous and presumed that such computer simulations really have anything to do with the real world of biochemistry, there are more serious problems.

However, the biochemist Michael Behe has shown that even a 'simple' light-sensitive spot requires a dazzling array of biochemicals in the right place and time to function. He states that each of its 'cells makes the complexity of a motorcycle or television set look paltry in comparison' and describes a small part of what's involved:[5]

When light first strikes the retina a photon interacts with a molecule called 11-*cis*-retinal, which rearranges within picoseconds to *trans*-retinal. (A picosecond [10^{-12} sec] is about the time it takes light to travel the breadth of a single human hair.) The change in the shape of the retinal molecule forces a change in the shape of the protein, rhodopsin, to which the retinal is tightly bound. The protein's metamorphosis alters its behavior. Now called metarhodopsin II, the protein sticks to another protein, called transducin. Before bumping into metarhodopsin II, transducin had tightly bound a small molecule called GDP. But when transducin interacts with metarhodopsin II, the GDP falls off, and a molecule called GTP binds to transducin. (GTP is related to, but different from, GDP.)

GTP-transducin-metarhodopsin II now binds to a protein called phosphodiesterase, located in the inner membrane of the cell. When attached to metarhodopsin II and its entourage, the phosphodiesterase acquires the chemical ability to 'cut' a molecule called cGMP (chemical relative of both GDP & GTP). Initially there are a lot of cGMP molecules in the cell, but the phosphodiesterase lowers its concentration, just as a pulled plug lowers the water level in a bathtub.

A transparent layer is also far more difficult to obtain than the researchers think. The best explanation for the cornea's transparency is diffraction theory, which shows that light is not scattered if the refractive index doesn't vary over distances more than half the wavelength of light. This in turn requires a certain very finely organized structure of the corneal fibers, which in turn requires complicated chemical pumps to make sure there is exactly the right water content.[6]

Therefore, these simulations do not start from simple beginnings but presuppose vast complexity even to begin with. Also, in their original paper, researchers admitted ‘an eye makes little sense on its own,’ because the ability to perceive light is meaningless unless the organism has sophisticated computational machinery to make use of this information. For example, it must have the ability to translate ‘attenuation of photon intensity’ to ‘a shadow of a predator is responsible’ to ‘I must take evasive measures,’ and be able to act on this information for it to have any selective value. Similarly, the first curving, with its slight ability to detect the direction of light, would only work if the creature had the appropriate ‘software’ to interpret this. Perceiving actual images is more complicated still. And having the right hardware & software may not be enough—people who have their sight restored after years of blindness take some time to learn to see properly. It should be noted that much information processing occurs in the retina before the signal reaches the brain.

It is also fallacious to point to a series of more complex eyes in nature, and then argue that this presents an evolutionary sequence. This is like arranging a number of different types of aircraft in order of complexity, then claiming that the simple aircraft evolved into complex ones, as opposed to being designed. For one thing, eyes can’t descend from other eyes *per se*; rather, organisms pass on genes for eyes to their descendants. This is important when considering the nautilus eye, a pinhole camera. This cannot possibly be an ancestor of the vertebrate lens/camera eye, because the nautilus as a whole is not an ancestor of the vertebrates, even according to the evolutionists!

Rotary motors in the bacterial flagellum

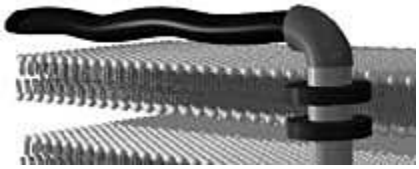
Scientific American cites another difficult example of irreducible complexity—the rotary motors on bacterial flagellum, but it really has no answers.

15. Recent discoveries prove that even at the microscopic level life has a quality of complexity that could not have come about through evolution.

‘Irreducible complexity’ is the battle cry of Michael J. Behe of Lehigh University, author of *Darwin’s Black Box: The Biochemical Challenge to Evolution*. As household example of irreducible complexity, Behe chooses the mousetrap—a machine that could not function if any of its pieces were missing and whose pieces have no value except as parts of the whole.

What is true of the mousetrap, he says, is even truer of the bacterial flagellum, a whiplike cellular organelle used for propulsion that operates like an outboard motor. The proteins that make up a flagellum are uncannily arranged into motor components, a universal joint, and other structures like those that a human engineer might specify. The possibility that this intricate array could have arisen thru evolutionary modification is virtually nil, Behe argues, and that bespeaks intelligent design. [*SA* 84]

Indeed, it does (see diagram below).



Bacterial flagellum with rotary motor, with the following features:

- **Self-assembly and repair**
- **Water-cooled rotary engine**
- **Proton motive force drive system**
- **Forward and reverse gears**
- **Operating speeds of up to 100,000 rpm**
- **Direction reversing capability within 1/4 of a turn**
- **Hard-wired signal transduction system with short-term memory**

[from Bacterial Flagella: *Paradigm for Design*, video, <www.arn.org/arnproducts/videos/v021.htm>]

He makes similar points about the blood's clotting mechanism and other molecular systems. Yet evolutionary biologists have answers to these objections. First, there exist flagellae with forms simpler than the one that Behe cites, so it is not necessary for all of those components to be present for a flagellum to work. The sophisticated components of this flagellum all have precedents elsewhere in nature, as described by Kenneth R. Miller of Brown University and others. [*SA* 84]

Miller is hardly the epitome of reliability. Behe has also responded to critics such as Miller.[7] In fact, the entire flagellum assembly is extremely similar to an organelle that *Yersinia pestis*, the bubonic plague bacterium, uses to inject toxins into cells. [*SA* 84]

This actually comes from the National Center for Science Education's misuses of the research of Dr Scott Minnich, a geneticist and associate professor of microbiology at the University of Idaho. He is a world-class expert on the flagellum who says that belief in design has given him many research insights. His research shows that the flagellum won't form above 37°C, and instead some secretory organelles form from the same set of genes. But this secretory apparatus, as well as the plague bacterium's drill apparatus, are a *degeneration* from the flagellum, which Minnich says came first although it is more complex.[8]

The key is the flagellum's component structures, which Behe suggests have no value apart from their role in propulsion, can serve multiple functions that would have helped favor their evolution. [*SA* 84]

Actually, what Behe says he means by irreducible complexity is that the flagellum could not work without about 40 protein components all organized in the right way. *Scientific American*'s argument is like claiming that if the components of an electric motor already exist in an electrical shop, they could assemble by themselves into a working motor. However, the right organization is just as important as the right components.

The final evolution of the flagellum might then have involved only novel recombination of sophisticated parts initially evolved for other purposes. [*SA* 84]

Minnich points out that only about 10 of the 40 components can be explained by co-option, but the other 30 are brand new. Also, the very process of assembly *in the right sequence* requires other regulatory machines, so is in itself irreducibly complex.[9]

Blood clotting

Scientific American cites another serious problem for evolution—blood clotting.

Similarly, the blood-clotting system seems to involve the modification and elaboration of proteins that were originally used in digestion, according to studies by Russell F. Doolittle of the University of California at San Diego. So some of the complexity that Behe calls proof of intelligent design is not irreducible at all. [*SA* 84]

This is once more bluff by the atheist Doolittle, or at least poor reading comprehension. He cited recent experiments showing that mice could survive with only two of the components of the blood clotting cascade (plasminogen and fibrinogen) eliminated. This supposedly showed that the current cascade was not irreducibly complex but clearly *reducibly* complex. But the experiment *really* showed that the mice lacking both components were better off than one lacking only plasminogen, because the latter suffer from uncleared clots. But the former are hardly as healthy as Doolittle implied, because the only reason they don't suffer from uncleared clots is that they have no functional clotting system at all! A non-functioning clotting system (despite possessing all the many remaining components) is hardly an evolutionary intermediate that natural selection could refine to produce a proper clotting system. Rather, this experiment is evidence against this, because the next step (i.e., from lacking both plasminogen and fibrinogen to fibrinogen only) would be selected *against* because of the uncleared clots.[10]

Complexity of a different kind—**'specified complexity'**—is the cornerstone of the intelligent-design arguments of William Dembski of Baylor University in his books *The Design Inference* and *No Free Lunch*. Essentially, his argument is that living things are complex in a way that undirected, random processes could never produce. The only logical conclusion, Dembski asserts, in an echo of Paley 200 years ago, is that some superhuman intelligence created and shaped life.

Dembski's argument contains several holes. It is wrong to insinuate that the field of explanations consists only of random processes or designing intelligences. Researchers into nonlinear systems and cellular automata at the Santa Fe Institute and elsewhere have demonstrated that simple, undirected processes can yield extraordinarily complex patterns. Some of the complexity seen in organisms may therefore emerge through natural phenomena that we as yet barely understand. But that is far different from saying that the complexity could not have arisen naturally. [*SA* 84]

Talk about blind faith! But in practice, as Dembski points out, specified complexity in all cases but biology is used as evidence of design, including the search for extraterrestrial intelligence. Since biological complexity is the only exception proposed by evolutionists, it smacks of special pleading.[11] In addition to the human eye, the flagellum, and blood clotting, there's a host of other examples of irreducible complexity in nature. Earlier I alluded to the dynamic sticking mechanism in the legs of insects. The sticky feet of geckos is another example of God's ingenuity.[12] Its structure is described by its evolutionary discoverers as 'beyond the limits of human technology.' [13] Still other examples of design include the lobster eyes with their unique square reflecting geometry that inspired advanced x-ray telescopes and beam producers,[14] the ATP synthase motor.

1. Carl Wieland, Darwin's real message: have you missed it? *Creation* **14**(4):16–19 (September–November 1992); J. Sarfati, review of K. Birkett, *The Essence of Darwinism*; see Evangelical compromise misses the essentials.

2. J. Sarfati, The Skeptics and their 'Churchian' Allies.

3. See my discussion about the evolution of the eye in Stumbling Over the Impossible: Refutation of Climbing Mt Improbable, *Journal of Creation* **12**(1):29–34, 1998; see Eye evolution, a case study.

4. D.E. Nilsson and S. Pelger, A Pessimistic Estimate of the Time Required for an Eye to Evolve, *Proc. R. Soc. Lond.* B **256**:53–58, 1994.

5. M.J. Behe, *Darwin's Black Box: The Biochemical Challenge to Evolution* (New York, NY: The Free Press, 1996), p. 46.

6. P.W.V. Gurney, Dawkins's Eye Revisited, *Journal of Creation* **15**(3):92–99, 2001.

7. Behe responds to various critics <www.trueorigin.org/behe08.asp>

8. See Scott Minnich, Bacterial Flagella: Spinning Tails of Complexity and Co-Option <www.idurc.org/yale-minnich.html>

9. *Unlocking the Mysteries of Life*, video, Illustra Media, 2002.

10. For more information, see Behe's In Defense of the Irreducibility of the Blood Clotting Cascade <www.trueorigin.org/behe03.asp>

11. Russell Grigg, A brief history of design, *Creation* **22**(2):50–53 (March–May 2000).

12. J. Sarfati, Great gecko glue? *Creation* **23**(1):54–55 (December 2000–February 2001).

13. K. Autumn *et al.*, Adhesive Force of a Single Gecko Foot Hair, *Nature* **405**(6787): 681–685 (8 June 2000); perspective by H. Gee, Gripping Feat, same issue, p. 631.

14. J. Sarfati, Lobster eyes—brilliant geometric design, *Creation* **23**(3)12–13 (June–July)

Molecular Evidence of Human Origins

by Bert Thompson, Ph.D.
Brad Harrub, Ph.D.

The molecular evidence clearly demonstrates that mitochondrial Eve is **not** the "most-recent common ancestor of all humans on Earth today." The reality is that one of the most critical assumptions behind such a concept has now been disproved. Mitochondrial DNA is not exclusively received from the maternal side—researchers now know that a father's mtDNA can cross into the egg. But what about the second assumption—that mutations occur at constant rates?

BROKEN MOLECULAR CLOCKS

Researchers who made the initial announcement about Eve not only gave a location for this amazing female, but also proposed the time period during which she was supposed to have lived. However, in order for the mtDNA theory to be of any practical use, those scientists had to assume that random mutations in the DNA occurred at documented, steady rates. For example, if they speculated that there was one mutation every 1,000 years, and if they found a difference of 10 mutations between us and our ancient hypothetical ancestor, they then could infer that that ancestor lived ten thousand years ago. Scientists—who use this concept to determine the age of mitochondrial Eve—refer to this proposed mutation rate as a "molecular clock." One group of researchers described the process as follows:

> The hypothesis of the molecular clock of evolution emerged from early observations that the number of amino acid replacements in a given protein appeared to change linearly with time. Indeed, if proteins (and genes) evolve at constant rates, they could serve as molecular clocks for timing evolutionary events and reconstructing the evolutionary history of extant species (Rodriguez-Trelles, et al., 2001, 98:11405, parenthetical item in orig.).

It sounds good in theory, but the actual facts tell an entirely different story. As these same researchers went on to admit:

> The neutrality theory predicts that the rate of neutral molecular evolution is constant over time, and thus that there is a molecular clock for timing evolutionary events. It has been observed that **the variance of the rate of evolution is generally larger than expected** according to the neutrality theory, **which has raised the question of how reliable the molecular clock is or, indeed, whether there is a molecular clock at all**.... The observations are inconsistent with the predictions made by various subsidiary hypotheses proposed to account for the overdispersion of the molecular clock (98:11405, emp. added).

Another study that was published in 2002 pointed out a built-in, natural bias for older ages that result from use of the molecular clock. The researchers who carried out the study noted:

> There is presently a conflict between fossil- and molecular-based evolutionary time scales. Molecular approaches for dating the branches of the tree of life frequently lead to substantially deeper times of divergence than those inferred by paleontologists. Here we show that molecular time estimates suffer from a methodological handicap, namely that they are asymmetrically bounded random variables, constrained by a nonelastic boundary at the lower end, but not at the higher end of the distribution. **This introduces a bias toward an overestimation of time** since divergence, which becomes greater as the length of the molecular sequence and the rate of evolution decrease....

Despite the booming amount of sequence information, molecular timing of evolutionary events has continued to yield conspicuously deeper dates than indicated by the stratigraphic data. Increasingly, the discrepancies between molecular and paleontological estimates are ascribed to deficiencies of the fossil record, while sequence-based time tables gain credit. **Yet, we have identified a fundamental flaw of molecular dating methods, which leads to dates that are systematically biased towards substantial overestimation of evolutionary times** (Rodriguez-Trelles, et al., 2002, 98:8112,8114, emp. added).

But the problems do not stop with systematic biases towards older ages. Ann Gibbons authored an article for the January 2, 1998 issue of *Science* titled "Calibrating the Mitochondrial Clock," the subheading of which read as follows: "Mitochondrial DNA appears to mutate much faster than expected, prompting new DNA forensics procedures & raising troubling questions about the dating of evolutionary events." In that article, she discussed new data which showed that the mutation rates used to obtain mitochondrial Eve's age no longer could be considered valid.

Evolutionists have assumed the clock is constant, ticking off mutations every 6,000 to 12,000 years or so. But if the clock ticks faster or at different rates at different times, some of the spectacular results—such as dating our ancestors' first journeys into Europe at about 40,000 years ago—may be in question (279:28).

Gibbons then quoted Neil Howell, a geneticist at the University of Texas Medical Branch in Galveston, who stated: "We've been treating this like a stopwatch, and I'm concerned that it's as precise as a sun dial. I don't mean to be inflammatory, but I'm concerned that we're pushing this system more than we should" (279:28). Gibbons concluded:

Regardless of the cause, evolutionists are most concerned about the effect of a faster mutation rate. For example, researchers have calculated that "mitochondrial Eve"—the woman whose mtDNA was ancestral to that in all living people—lived 10,000 to 200,000 years ago in Africa. **Using the new clock, she would be a mere 6,000 years old** (1998, 279:29, emp. added).

"Mitochondrial Eve" a mere 6,000 years old—instead of 200,000?! Gibbons quickly went on to note, of course, that "no one thinks that's the case" (279:29). She ended her article by discussing the fact that many test results are (to use her exact word) "inconclusive," and went on to lament the fact that "for now, so are some of the evolutionary results gained by using the mtDNA clock" (279:29).

But it gets worse. The "evolutionary results gained by using the mtDNA clock" are not just "inconclusive." They're **wrong**! In the January 2003 edition of the *Annals of Human Genetics*, geneticist Peter Forster of Cambridge authored an article ("To Err is Human") in which he documented that, to use his words, **"more than half of the mtDNA sequencing studies ever published contain obvious errors**." He then asked: "Does it matter? Unfortunately, in many cases it does." Then came the crushing blow for "Mitochondrial Eve": "**fundamental research papers, such as those claiming a recent African origin for mankind** (Cann,1987; Vigilant,1991) ...**have been criticized, and rejected due to the extent of primary data errors**". Then, as if to add salt to an already open and bleeding wound, Dr. Forster acknowledged that the errors discovered thus far are "only the tip of the iceberg...," and that "there is no reason to suppose that DNA sequencing errors are restricted to mtDNA" (67[1]:2,3).

Just one month later, *Nature* weighed in with an exposé of its own. In the February 20, 2003 issue, Carina Dennis authored a commentary on Forster's work titled "Error Reports Threaten to Unravel Databases of Mitochondrial DNA." Dennis reiterated the fact that "more than half of all published studies of human mitochondrial DNA (mtDNA) sequences contain mistakes." Then, after admitting that the "published mtDNA sequences are popular tools for investigating the evolution and demography of human populations," she commented:

[T]he problem is far bigger than researchers had imagined. The mistakes may be so extensive that geneticists could be drawing incorrect conclusions to studies of human populations and evolution (421:773). In her report, Dennis quoted Eric Shoubridge, a geneticist at McGill University's Montreal Neurological Institute in Canada, who investigates human diseases resulting from problems with mtDNA. His response was: "I was surprised by the number of errors. What concerns me most is that these errors could be compounded in the databases" (421:773). In 1981, the complete sequence of human mtDNA — known as the "Cambridge Reference Sequence"—was published in a database format for scientists to use in their research (see Anderson, et al., 1981). It is from that initial database that many of the mtDNA sequences have been taken and used to predict, among other things, the Neolithic origin of Europeans (Simoni, et al., 2000) and the "factuality" of the creature known as "Mitochondrial Eve." Yet Dr. Forster has been busily engaged in making corrections to that 1981 database almost since its inception, and has compiled his own database of corrected mitochondrial sequences.

Eric Shoubridge (quoted above) is not the only one who is "concerned" about Peter Forster's findings. Neil Howell, vice president for research at MitoKor, a San Diego-based biotech company whose speciality is mitochondrial diseases, suggested that Forster's error-detection method "may even **underestimate** the extent of the errors" (as quoted in Dennis, 421:773-774, emp. added).

Until approximately 1997, we did not have good empirical measures of mutation rates in humans. However, that situation greatly improved when geneticists were able to analyze individual DNA from well-established family trees going back several generations. One study revealed mutation rates in mitochondrial DNA were **eighteen times higher than previous estimates** (Parsons, et al., 1997).

What has been the response of the scientific community? Let Forster answer: "Antagonism would be an understatement in some cases" (as quoted in Dennis, 421:773). He did note, however, that, at times, some of the scientists whose published papers have been found to contain the errors were "forthcoming in resolving discrepancies in sequences." That's nice—since "truth" and "knowledge" are what science is supposedly all about (our English word "science" derives from the Latin *scientia*, meaning knowledge).

We now know that the two key assumptions behind the data used to establish the existence of "mitochondrial Eve" are **not just flawed, but wrong**. The assumption that mitochondrial DNA is passed down only by the mother is completely incorrect (it also can be passed on by the father). And, the mutation rates used to calibrate the so-called "molecular clock" are now known to have been in error. (To use the words of Rodriguez-Trelles and his coworkers, the method contains a "fundamental flaw.") In the end, where does all of this leave "Mitochondrial Eve"? We could not put it any plainer than Dr. Forster did when he said that "fundamental research papers, such as those claiming a recent African origin for mankind have been criticized and rejected due to the extent of primary data errors." Criticized—**and rejected**?!

Philip Awadalla and his coworkers noted in *Science:* "Many inferences about the pattern and tempo of human evolution and mtDNA evolution have been based on the assumption of clonal inheritance. Their inferences will now have to be reconsidered" (1999, 286:2525). Yes, they will. The same year that Awadalla, et al., published their paper on recombination in mitochondrial DNA, Evelyn Strauss published a paper in *Science* ("Can Mitochondrial Clocks Keep Time?") in which she noted:

The DNA sequences pouring in from sequencing projects have fueled the effort and extended the clock approach to many genes in the cell nucleus. But the wash of data has uncovered some troubling facts. **It's now clear that in many cases, the main assumption underlying molecular clocks doesn't hold up**: Clocks tick at different rates in different lineages and at different times....

For the clock to work with either sort of DNA [nuclear or mitochondrial—BT/BH], nucleotide changes must tick away steadily so scientists can convert the number of nucleotide differences seen between two organisms into the number of years since they diverged. Different genes evolve at different rates, depending on the selective forces upon them, but the model requires only that each gene's clock maintains its own rate. **Early work hinted that this might not always be true & now a plethora of data shows that many genes don't conform to this model** (1999, 283:1435,1436, emp. added).

John Avise, an evolutionary geneticist at the University of Georgia in Athens, went so far as to remark: "There's an emerging consensus that there are significant rate heterogeneities across different lineages. How big they are and how to deal with them is very much a matter of concern" (as quoted in Strauss, 283:1435). Avise observed that the problems with the molecular clock are a "matter of concern." Phillip Awadalla suggested that the inferences that have been drawn from those clocks "will now have to be reconsidered." Ann Gibbons reported "the evolutionary results gained by using the mtDNA clock" are "inconclusive." When each of these writers made those statements, they had no idea about the "bomb" that was about to be dropped on the evolutionary community regarding the inaccuracy of huge sections of reported mitochondrial DNA data. Just as evolutionists thought it could not possibly get any worse—it did!

Poor Eve. How many times, we wonder, will she have to die before she finally can be buried—permanently—and left to "rest in peace"? We suggest that, instead of merely "reconsidering" their theory and attempting to revamp it accordingly, evolutionists need to admit, honestly and forthrightly, the clock is "broken," and that mitochondrial Eve, as it turns out, has existed only in their minds, not in the facts of the real world. Science works by analyzing the data and forming hypotheses based on those data. Science is not supposed to "massage" the data until they fit a certain preconceived hypothesis. All of the conclusions that have been drawn from research on mitochondrial Eve via the molecular clock must now be discarded as unreliable. But this is just the "tip of the iceberg." The molecular evidence against evolutionary theory does not stop there. Consider the complexity involved in packing all of that genetic information into a cell, and then passing it on. The mechanics underlying genetics is mind-boggling and yet, it's very real. Read on.

THE SECOND CODE AND "JUNK DNA"

During the 1950s, while James Watson, Francis Crick, Maurice Wilkins, and Rosalind Franklin were racing to see who could be the first in print with the molecular structure of DNA, no one could have imagined the immense molecular complexity that humans had discovered. The race to unravel the genetic code of life was on. Almost exactly fifty years later, on February 16, 2001, a special issue of *Science* was devoted almost entirely to the human genome. In that report, scientists revealed that the genome consisted of 2.91 billion nucleotide base pairs. However, this rough draft had been accomplished using a "shotgun" approach to the entire genome, and as such, there were numerous gaps left to fill. On April 14, 2003, the International Human Genome Consortium announced the successful completion of the Human Genome Project—more than two years ahead of schedule. The press report read: "**The human genome is complete & the Human Genome Project is over**" (see "Human Genome Report...," 2003, emp. added). But the puzzle is nowhere close to being solved. **Having now completed the human genome, it appears there may be a second—more complex—code left to unravel**. As Elizabeth Pennisi observed:

All this work is making clear that buried in DNA sequence is a regulatory code akin to the genetic code "but infinitely more complicated," says Michael Eisen, computational biologist at Lawrence Berkeley National Laboratory in California.... Manolis Dermitzakis of the Wellcome Trust Sanger Institute in Cambridge, U.K., agrees: "**The complexity of the genome is much higher than we have defined for the past 20 years. We have to change our way of thinking**" (2004, 304:632, emp. added).

So now we discover that there is a code buried within the code. In fact, as Michael Eisen admitted, this second code is "infinitely more complicated." And yet, we are expected to believe that this massive network of complexity simply arose as the result of some cosmological/biological accident? Pennisi lamented:

> Molecular biologists may have sequenced the human genome, but it's going to take molecular cryptographers to crack its complex code. Genes, keystones to the development and functioning of all organisms, can't by themselves explain what makes cows cows and corn corn. The same genes have turned up in organisms as different as, say, mice and jellyfish. Instead, new findings from a variety of researchers have made clear that it's the genome's exquisite control of each gene's activity—and not the genes per se—that matters most (p. 632).

The genetics sequence is vital. But what is becoming more evident all the time is that the way in which genes are regulated is even a more critical factor. For instance, Savante Pääbo and his colleagues noted in the April 12, 2002 issue of *Science* that certain genes are far more active in the human brain than in the chimp brain (see Enard, et al., 2002). And as if that were not complicated enough, researchers now have discovered regulatory DNA also is playing a key role in transcription.

Add to this the fact that we know today that there are sections of DNA within a gene that don't code for any part of the protein, but rather are purposefully "spliced out," and one begins to realize the sophistication involved in this second code. **Introns** are sections of DNA evolutionists frequently refer to as "junk DNA" because those sections do not appear to serve any known role in creating proteins. When mRNA copies DNA, these introns are cut out before a new synthesized RNA strand leaves the nucleus (what remains is referred to as **exons**). The question should be asked: How did this specific mechanism to splice out very specific portions occur, and why did it "evolve" in the first place? Why would nature select to have "junk DNA" present in the genome? The reality is that this complex information system was designed by an omnipotent Designer—and it is obvious from the fact that it is referred to as "junk" DNA that some scientists have yet to grasp the full import of God's handiwork.

In order to better understand how this second code affects an individual, we need to examine what is taking place inside the cell. Consider the following description of the mechanics involved in creating a particular protein that's needed within the cell…

A double-helix molecule of DNA is composed of two polynucleotide chains wound around each other. Three-dimensionally, the helix twists in the right-handed direction (think of two strands of rope twisted around each other in the clockwise direction). This tightly bound structure is located within the nucleus of a cell where the genetic information needed for the protein is housed.

The first "step" is commonly called **transcription**—where the genetic material from DNA is synthesized into RNA. When our bodies want to make new proteins, the location of DNA that contains that information must be unwound and "read" by a molecular enzyme known as RNA polymerase. We know today that dozens of molecules (mostly proteins) are required to carry out this carefully choreographed event. RNA polymerase is an enzyme that "reads" DNA & synthesizes a complementary strand of RNA using nucleotides that must match up with the base pairs on the DNA. Keep in mind that all of this is occurring within the nucleus of a cell, and the RNA polymerase must "travel" down the DNA strand in the correct direction to make the needed protein.

Remember, too, that RNA polymerase is a three-dimensional molecular machine composed of a dozen different small proteins. So before a protein can be built, RNA polymerase must be present in the correct 3D configuration. A microscopic investigation into the structure of RNA polymerase reveals a pair of jaws that appears to grip the DNA, a clamp that holds the molecular strand in place, a three-dimensional pore through which RNA nucleotides probably enter, and tiny grooves through which the newly synthesized RNA strand may thread out of the enzyme.

You may recall being told in various biology classes about the different "types" of RNA, each of which has a different job. For instance:

- MRNA —Messenger RNA: Encodes the amino acid sequence of a polypeptide.
- TRNA—Transfer RNA: Brings the amino acids to ribosomes during translation.
- RRNA—Ribosomal RNA: With ribosomal proteins, makes up the ribosomes (organelles that translate mRNA).
- SnRNA—Small nuclear RNA: With proteins, forms complexes that are used in RNA processing in eukaryotes (not found in prokaryotes).

The next step cannot occur until the introns (a.k.a. "junk DNA") have been spliced out, so that step must take place within the nucleus. Transcription occurs in the nucleus to produce a "pre-mRNA" molecule. The pre-mRNA is typically processed to produce the mature mRNA. Part of the job of the pre-mRNA is to remove the introns from the nucleotide sequence and splice the exons into a translatable mRNA, which then can exit the nucleus.

The second major step in protein synthesis is one in which the information encoded in mRNA is deciphered (or **translated**) into sequences of amino acids. This process occurs in a cellular organelle known as a ribosome. In cells without a nucleus, transcription and translation occur simultaneously; that is, translation begins while the mRNA is still being synthesized. In cells that possess a nucleus (like the majority with which we are familiar), transcription occurs in the nucleus, and translation takes place in the cytoplasm. Thus, this complex system had to "devise" a method to get the newly synthesized RNA strand through the bilipid membrane of the nucleus, out into the cytoplasm, and onto a ribosome. [Believe it or not, this is a "condensed summary" of the transcription phase.]

Recall that the building blocks of DNA are bases (designated as A, C, G, T) that are "read" in groups of three. Each "three-letter" group codes for a specific amino acid (e.g., ACG codes for threonine, while TAC codes for tyrosine). The newly synthesized piece of genetic material makes its way to a ribosome where it then is "read," and amino acids are joined together to form the protein. Once the DNA code has been read, the appropriate amino acids then are brought in one at a time and joined together by peptide bonds to make a protein. Raven and Johnson summed up the translation phase in the following manner:

> Protein synthesis is carried out on the ribosomes, which bind to sites at one end of the mRNA and then move down the mRNA in increments of three nucleotides. With each step of the ribosome's progress, it exposes a three-base sequence to binding by a tRNA molecule at the complimentary nucleotide sequence. Ultimately, the amino acid carried by that specific tRNA molecule is added to the end of the growing polypeptide chain (1989, p. 307).

[Again, that was another "condensed summary." We do not have the space here to discuss the fact that once the protein has been formed, it then must fold itself into the correct three-dimensional shape. Consider for just a moment that in the time it took you to read the condensed version of this complex process, numerous proteins were being formed in many of the cells throughout your body.]

IRREDUCIBLE COMPLEXITY

Charles Darwin understood that evolutionary theory rested on one key point—that all parts of a system must be the products of slight, successive changes that work together. He wrote, in fact: "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" (1859, p. 219). More than a century later, Richard Dawkins would contend:

> **One hundred and twenty five years on, we know a lot more about animals and plants than Darwin did, and still not a single case is known to me of a complex organ that could not have been formed by numerous successive slight modifications. I do not believe that such a case will ever be found. If it is...I shall cease to believe in evolution (1986, p. 91).**

Ten years after Dawkins penned those words, a powerful challenge arose for Darwinian evolution—one that demonstrates examples of the criterion that Darwin suggested would "absolutely break down" evolutionary theory. The answer lies in "irreducible complexity." In his book, *Darwin's Black Box*, Lehigh University biochemist Michael Behe pointed out:

> **What type of biological system could not be formed by "numerous, successive, slight modifications"? Well, for starters, a system that is irreducibly complex. By irreducibly complex, I mean a single system composed of several well-matched, interacting parts that contribute to the basic function, wherein the removal of any one of the parts causes the system to effectively cease functioning. An irreducibly complex system cannot be produced directly (that is, by continuously improving the initial function, which continues to work by the same mechanism) by slight, successive modifications of a precursor system, because any precursor to an irreducibly complex system that is missing a part is by definition nonfunctional (1996, p. 39).**

Within the pages of his book, Dr. Behe pointed out several prominent examples of systems that cannot be explained by successive incremental changes. He examined in detail the intricate complexity of a cell's cilium, and that of the bacterial flagellum. In detailing the sophistication of these molecular motors, he noted:

> **The rotary nature of the bacterial flagellar motor was a startling, unexpected discovery. Unlike other systems that generate mechanical motion (muscles, for example) the bacterial motor does not directly use energy that is stored in a "carrier" molecule such as ATP. Rather, to move the flagellum it uses the energy generated by a flow of acid through the bacterial membrane.... The bacterial flagellum, in addition to proteins already discussed, requires about forty other proteins for function (1996, pp. 70, 71, parenthetical item in orig.).**

He then went on to observe:

> **In summary, as biochemists have begun to examine apparently simple structures like cilia and flagella, they have discovered staggering complexity, with dozens or even hundreds of precisely tailored parts.... As the number of required parts increases, the difficulty of gradually putting the system together skyrockets, and the likelihood of indirect scenarios plummets. Darwin looks more and more forlorn (p. 73).**

Naturalistic evolution cannot offer an adequate explanation for the origin of all of the microscopic parts to these complex systems. As William Dembski remarked in his classic book, *Intelligent Design*:

The irreducible complexity of such biochemical systems counts powerfully against the Darwinian mechanism, and indeed against any naturalistic evolutionary mechanism proposed to date. Moreover, because irreducible complexity occurs at the biochemical level, there is no more fundamental level of biological analysis to which the irreducible complexity of biochemical systems can be referred, and at which a Darwinian analysis in terms of selection and mutation can still hope for success (1999, p. 149).

An unbiased observation demonstrates that the molecular components of the dynein ATPase motors in cilia and flagella can be "reduced" to the simplest level, and yet without each one of the functional parts, the "organ" will not work.

Italo Calvino's book, *Invisible Cities*, presents a dialogue between Marco Polo and Kublai Khan.

Marco Polo describes a bridge stone by stone.

"But which is the stone that supports the arch?" Kublai Khan asks.

"This bridge is not supported by one stone or another," Marco Polo answers, "but by the line of the arch that they form."

Kublai Khan remains silent, reflecting. Then he adds, "Why do you speak to me of the stones? It is only the arch that matters to me."

Polo answers, "Without stones there is no arch" (1974).

And that is exactly the point. These complex systems require many simple pieces, but none of them is beneficial on its own; making the flagellum work requires all of the pieces. As evolutionist Michael Denton remarked:

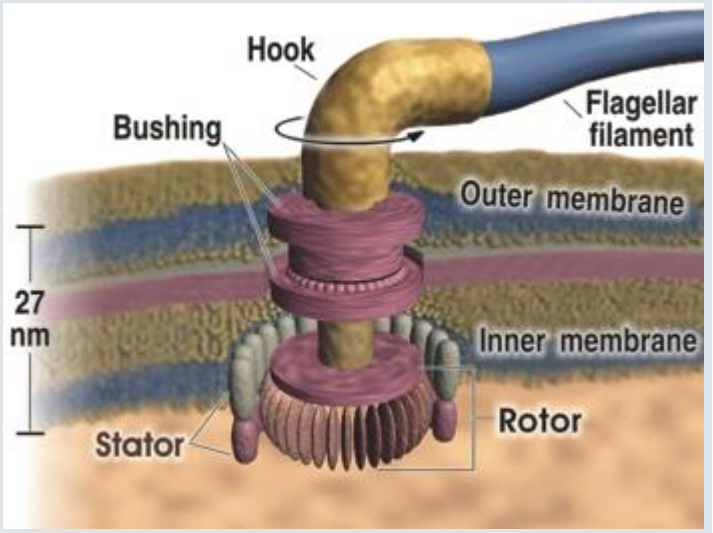
The bacterial flagellum and the rotary motor which drives it are not led up to gradually through a series of intermediate structures and, as is so often the case, it is hard to envisage a hypothetical evolutionary sequence of similar rotors through which it might have evolved gradually (1985, p. 225).

Darwin's criterion for failure has been met in molecular machines and irreducible complexity. The question, then, that must be asked is this: will Richard Dawkins "cease to believe in evolution?"

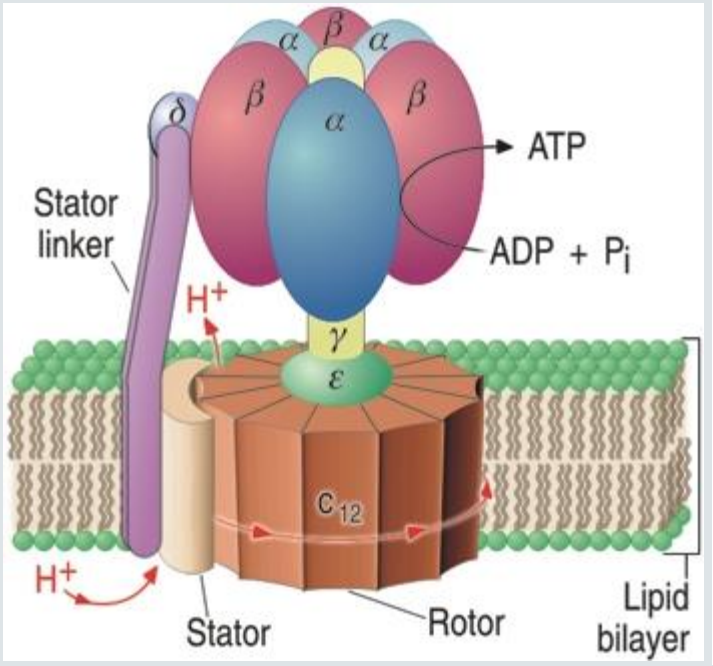
MOLECULAR MOTORS

Evolutionists routinely contend that early life was simple, and subsequently has evolved into more complex forms. German evolutionist Ernst Haeckel, who faked embryological drawings in support of Darwinian theory, purported that a cell was a "simple little lump of albuminous combination of carbon" (as quoted in Farley, 1979, p. 73.). As Michael Behe put it, Haeckel believed that the interior of the cell was "not much different from a piece of microscopic Jell-O" (1996, p. 24). But today we know differently. We no longer think "Jell-O"; rather, we think of the famous (or infamous!) Interstate highway 405 around Los Angeles as a more accurate description. As Behe commented:

Shortly after 1950, science advanced to the point where it could determine the shapes and properties of a few of the molecules that make up living organisms. Slowly, painstakingly, the structures of more and more biological molecules were elucidated, and the way they work inferred from countless experiments. The cumulative results show with piercing clarity that life is based on **machines**—machines made of molecules! Molecular machines haul cargo from one place in the cell to another along "highways" made of other molecules, while still others act as cables, ropes, and pulleys to hold the cell in shape (1996, p. 4, emp. in orig.).



Top: Bacterial flagellum with rotary motor, courtesy of Access Research Network (Art Battson)
Bottom: ATP synthase motor; image by Charles McCown



Consider the validity of evolutionary theory **now**, since **five families** of these structurally complex motors have been identified! The February 21, 2003 issue of *Cell* included a review by Ronald Vale titled "The Molecular Motor Toolbox" (112:467-480). In the abstract that accompanied his article, Dr. Vale noted: "Recent genomic and functional studies suggest that five cargo-carrying motors emerged in primitive eukaryotes and have been widely used throughout evolution" (p.467). He then described these "evolved" motors as follows:

A cell, like a metropolitan city, must organize its bustling community of macromolecules. Setting meeting points and establishing the timing of transactions are of fundamental importance for cell behavior. The high degree of spatial/temporal organization of molecules and organelles within cells is made possible by protein machines that transport components to various destinations within the cytoplasm (p. 467).

Vale then went into extreme detail, reviewing everything we know about these five major motor-engine families that ferry cargo around the cell: actin, dynein, conventional homodimeric kinesin, heterotrimeric kinesin II, and Unc 104/KIF1. But throughout his review, one point became painfully clear: there still is a great deal of information that we do not yet understand about these amazingly complex motors. As Vale himself admitted:

Fifteen years ago, only a few molecular motors were known. In contrast, complete inventories of molecular motors are now available in a number of diverse organisms. While these remarkable accomplishments have answered many questions, the genomic inventories also have exposed many areas of ignorance (p. 477).

Dr. Behe's book brilliantly exposed the complexity of these structures, and as a result, numerous scientists are echoing his initial observations. A United Kingdom research team headed by Stan Burgess imaged thousands of the tiny molecules that work something like railroad handcars (Burgess, et al., 2003, 421:715). These dynein motors have a ring-shaped, hexagonal head of six AAA proteins, to which is added a C-terminal domain of the protein. Emerging out of one side, and in the same plane as the ring, is what researchers refer to as a "stalk," which has a structure on the end that attaches to microtubules in the cell. These microtubules are like train tracks running throughout the cell. Emerging out of the other end is a stem that attaches to whatever cargo needs to be transported. The stem is fastened to the ring by a linker, which seems to act like a ratchet on a gear during the cycle. In the same issue of *Nature* in which the Burgess study was published, Richard Vallee & Peter Hook provided a review of the study titled "A Magnificent Machine." They noted: "The protein displays a degree of gymnastic ability that is rarely seen" (2003, 421:701).

Words like "remarkable," "magnificent," and "intricately complex" fill the literature as scientists struggle to figure out exactly how these miniature motors can run so efficiently and effectively. In an interview, Joshua Shaevitz, co-author of a study published in the *Proceedings of the National Academy of Sciences,* commented: "This is one of the most efficient engines anyone has ever seen. Some estimates put it at near 100 percent efficiency. It's an amazing little thing" (as quoted in Swartz, 2003). In an article titled "Acid Stops Bacteria Swimming," Kendall Powell noted:

> "This is a motor with quite remarkable properties," says Robert Macnab of Yale University in New Haven, Connecticut, who studies the assembly of bacterial motors. "It runs like a battery, moves like a ship's propeller, has a gear switch so it can rotate in either direction, and it's under the control of information from environment. These are biological functions at their most simplified form, and yet there are 60 different types of components in this little engine" (2003).

This is hardly the description of a "simple biological function"! While evolutionists may continue to fondly embrace blind chance, a number of serious questions still remain. What, exactly, keeps all of these engines from colliding on the tracks? What (Who?) is responsible for the switching of the tracks? How do these motors "know" specifically what cargo to carry? And perhaps most important of all, how did they get here in the first place? Add to this the fact that most "primitive" life forms such as Archaea and eubacteria possess these same molecular machines, and the pressure **really** begins to mount rapidly for evolutionists.

Evolutionist Richard Dawkins stated in the preface to his book, *The Blind Watchmaker*: "The complexity of living organisms is matched by the elegant efficiency of their apparent design. If anyone doesn't agree that this amount of complex design cries out for an explanation, I give up!" (1986, p. ix). We agree. And this is the same Richard Dawkins who admitted:

> The more statistically improbable a thing is, the less we can believe that it just happened by blind chance. **Superficially the obvious alternative to chance is an intelligent Designer** (1982, 94:130, emp. added).

We, on the other hand, suggest that it is not "superficial" to acknowledge that where there is obvious design, there is, just as obviously, a designer. In fact, for once, we actually find ourselves in agreement with our unbelieving colleagues in science. As atheistic physicist Paul Ricci wrote in *Fundamentals of Critical Thinking*: "'Everything designed has a designer' is an analytically true statement" (1986, p. 190). Indeed, it is. Where there is design, there must, by definition, be a designer. The time has come for evolutionists to stop "marveling" at these "intricately complex" finely tuned motors, and, instead, to acknowledge the "remarkable," "magnificent," and "intricately complex" design behind them.

CONCLUSION

One of the best arguments against evolution is the complexity, intricacy, ingenuity, beauty, and design of the molecules in living systems.

Michael Denton affirmed: "Molecular biology has shown even the simplest of all living systems on earth today, bacterial cells, are exceedingly complex objects. Although the tiniest bacterial cells are incredibly small, weighing less than 10^{-12}gms, each in effect a veritable microminiaturized factory containing thousands of exquisitely designed intricate pieces of molecular machinery, made up altogether of one hundred thousand million atoms, far more complicated than any machine built in the non-living world (1985, p. 250).

How can blind chance account for the information stored in the molecular structure of DNA? And how can "slight modifications" account for the complex highway of molecular motors? The reality is, they cannot. Centuries ago, Greek philosopher Democritus stated that everything that exists in the Universe is the end result of chance and necessity. Today, even with all of our advanced knowledge of the molecular world around us, many people remain dedicated to such an idea. As G.K. Chesterton once remarked: "When men stop believing in God, they do not believe in **nothing**; they believe in **anything**.

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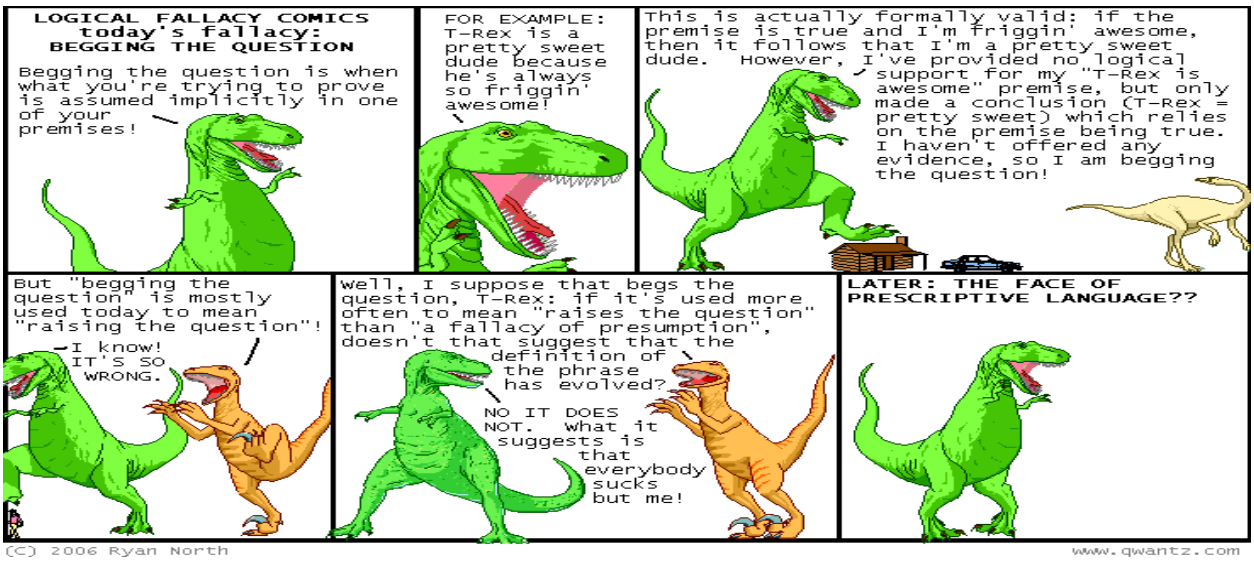


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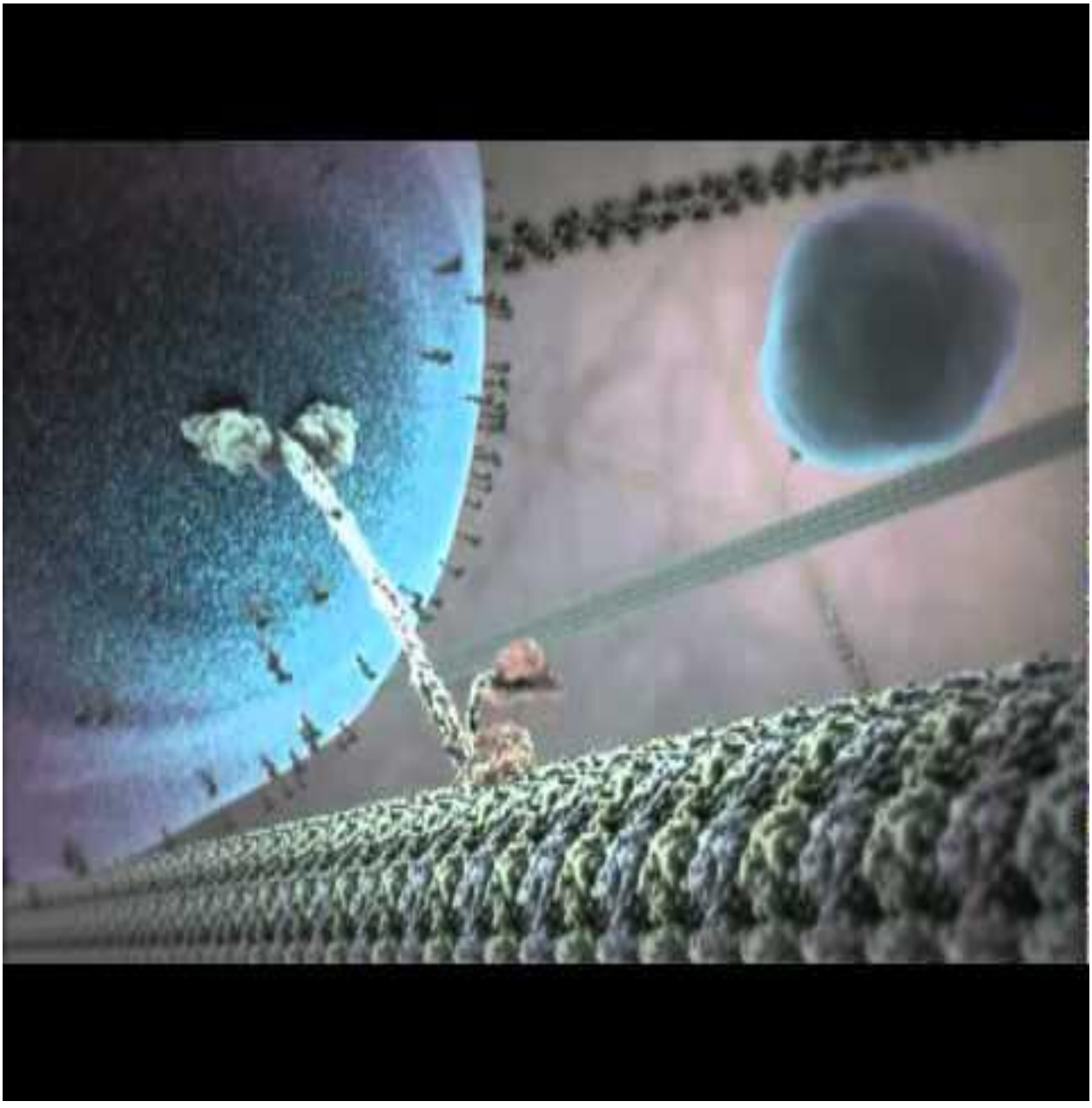

Edited by David L. Burris