

MOLECULAR EVIDENCE OF HUMAN ORIGINS—[PART II]

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

[EDITOR'S NOTE: Part I of this two-part series appeared in the April issue. Part II follows below and continues, without introductory comments, where the first article ended.]

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 10,000 years ago. Scientists—who use this concept to determine the age of mitochondrial Eve—refer to this proposed mu-

tation 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 overestima-**

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tion 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 and 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 that the clock is constant, ticking off mutations every 6,000 to 12,000 years or so. But if the clock ticks faster or at dif-

ferent 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, et al., 1987; Vigilant, et al., 1991) **...have been criticized, and rejected due to the extent of primary data errors”** (67 [1]:2, emp. added). 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 (2003, 421:773, emp. added).

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

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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 DNA from individuals with well-established family trees going back several generations. One study revealed that mutation rates in mitochondrial DNA were **eighteen times higher than previous estimates** (see 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, and 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.” Philip Awadalla suggested that the inferences that have been drawn from those clocks “will now have to be reconsidered.” Ann Gibbons reported that “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 the 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, that the clock is “broken,” and that mito-

chondrial 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 is 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 and 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, a 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 that 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 do not 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 that 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 newly 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 just a few of the mechanics involved in creating a particular protein that is needed within the cell. [We realize that this material may be a bit complicated—but that is exactly the point. How could such a complex information system arrive by random chance? Also, bear in mind that this discussion will not address how an organism allegedly evolved the ability to detect a need for a particular protein, how DNA or RNA evolved, how DNA and RNA “know” one protein from another, or how different types of cells could have evolved. We simply want to point out the intricacy involved in creating just a single protein.]

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 and 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 three-dimensional 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 pro-

tein. 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. At each step of the ribosome's progress, it exposes a three-base sequence to binding by a tRNA molecule with the complimentary nucleotide sequence. Ultimately, the amino acid carried by that particular 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 sys-

tems can be referred, and at which a Darwinian analysis in terms of selection and mutation can still hope for success (1999, p. 149).

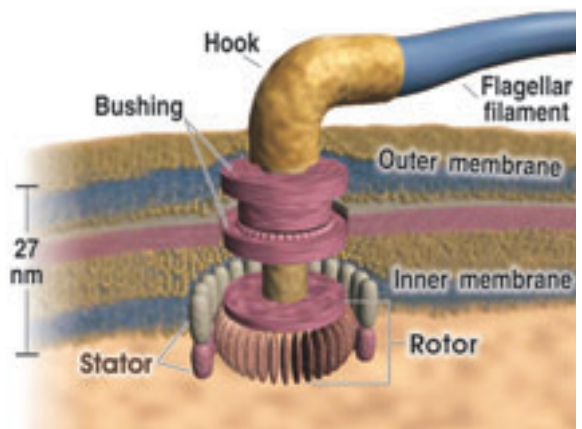
An unbiased observation demonstrates that the molecular components of the dynein ATPase motors in cilia and flagella can be "reduced" to the simplest level, and yet without each one of the functional parts, the "organ" will not work.

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

Marco Polo describes a bridge stone by stone.

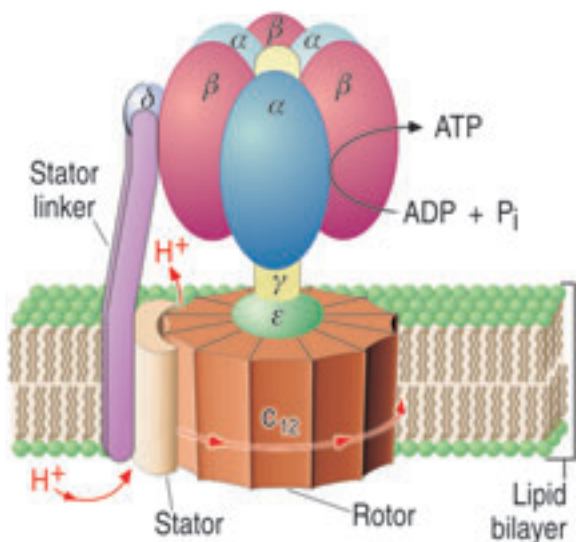
"But which is the stone that supports the arch?" Kublai Khan asks.

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



Top: Bacterial flagellum with rotary motor, courtesy of Access Research Network (Art Battson)

Bottom: ATP synthase motor; image by Charles McCown



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 mi-

croscopic 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.).

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 im-

portance 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 and 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

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June 10-12	Wendover, KY	(606) 526-8151

Kyle Butt

June 1	Gulf Shores, AL	(251) 968-7769
June 24-26	Nashville, TN	(615) 833-8480

Eric Lyons

June 8	Gulf Shores, AL	(251) 968-7769
June 15	Florence, AL	(256) 766-3617

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 "remarkable," "magnificent," and "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 that 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 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 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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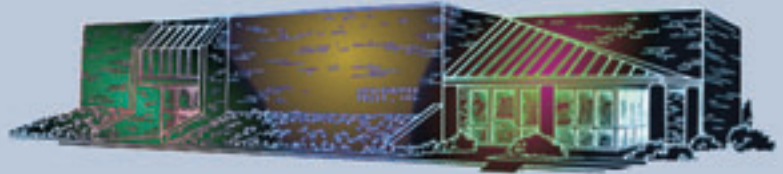
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NOTE FROM THE EDITOR



EXCITING TIMES, THESE!

I could not begin to count the number of times that I've said to one or more members of my staff, "I wish our friends, financial supporters, and customers could spend a day or two with us here in our offices—just to see how exciting it is to work here, and witness what actually takes place behind the scenes!" But because those friends, financial supporters, and customers are spread quite literally around the world, I know that I probably am not going to get my wish anytime soon. So, I have decided to use my "Note from the Editor" this month to provide a brief update on some of the things that are going on "behind the scenes" currently at A.P. These truly are exciting times for our work!

First, I might mention that our extremely popular Web site (www.ApologeticsPress.org) continues to draw hundreds of thousands of visitors each year (it receives approximately 250,000 page-hits per month!). If you have not visited it recently, please do. The material on the home page changes every Monday around noon—and everything that has appeared there in the past is archived so it's easy to retrieve. [The site's search engine is a dream to use; try it, and you'll see what I mean.] Anytime you need cutting-edge material on "hot-topic" current issues (euthanasia, stem-cell research, the creation/evolution controversy, etc.), make it a point to visit the Apologetics Press Web site first. Chances are, you'll find exactly what you need—and then some!

Second, we have just sent two new books to the printer. *The Anvil Rings* (volume 2) by Eric Lyons examines (as did the first volume) alleged biblical contradictions and discrepancies. Eric has done his usual thorough job in answering the skeptics' and infidels' charges. The book is a veritable treasure trove of invaluable material, much of which is unavailable anywhere else. Our second new book, *The Quran Unveiled* by Dr. Dave Miller, presents a calm-yet-candid comparison between Islam's book of scripture and the Bible. I do not overstate the case when I say that every Muslim, and every Christian, should read this volume in a spirit of open-minded investigation. There is no doubt in my mind that those who do so will be surprised by what they find. Both of these books are due back to us from the printer around June 30. I will announce their availability as soon as we receive them.

[We are extremely grateful for several specific donations from churches and/or individuals that made the printing of these two books possible. Other new books will be announced shortly.]

Third, we are getting ready for the arrival of our summer interns. In fact, by the time you read this, several of them will already be here, and will be busily at work on their assignments. Regular readers of *Reason & Revelation* already know the quality of young men these interns represent. Each of them (they range in age from 13 to 23) has gone through a lengthy, in-depth, and extremely laborious selection process (which includes, among other things, their submission of written essays, our checking of their personal and professional references, an "entrance interview" at our offices, etc.). Each young man was hand picked for his deep spirituality, keen intellect, and multiple talents. In addition, each of them has been selected for his particular area of expertise. Some interns are interested in the sciences (specifically, astrophysics, cytology, biochemistry, genetics, marine biology, psychology, etc.). Some are concentrating on biblical issues (Greek, Hebrew, Aramaic, Old Testament, New Testament, etc.). And some are working in multi-disciplinary areas (such as biomedical ethics, philosophy of science, and so on).

Each of the young men is a straight-A student who is incredibly self-disciplined and self-motivated. In addition, each is an outstanding writer or public speaker (truth be told, most of them are **both!**). Later this year, and well into the next, you will see their articles appear on our Web site, in our journals, and elsewhere. [All of the interns write for *Discovery*, our magazine on Scripture and science for children; the older ones also write on occasion for *Reason & Revelation*—as Nathaniel Nelson did for this month's issue (in the *Resources* section).] I will have much more to say about our interns in the months ahead—but for now, I simply wanted to mention them. They contribute a **lot** of zest and freshness to our efforts, as well as a **lot** of valuable in-depth research and excellent writing. Expect great things from them—and from all of us. Exciting times, these! **Truly exciting!**

Bert Thompson