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THE MOLECULAR EVIDENCE OF HUMAN ORIGINS [PART I]

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

While many evolutionists proclaim that human DNA is 98% identical to chimpanzee DNA, few would allow themselves to receive a transplant using chimpanzee organs. As a matter of fact, in the 1960s, American doctors tried transplanting chimp organs into humans, but in all cases the organs were totally unsuitable. **The claim of 98% similarity between chimpanzees and humans is not only deceptive and misleading, but also is scientifically incorrect.** Today, scientists are finding increasing numbers of differences in chimpanzee and human DNA. For instance, a 2002 research study proved that human DNA was **at least 5% different** from that of chimpanzees—and that number probably will continue to grow as we compare the actual base sequences that comprise human DNA (see Britten, 2002).

In 1962, James Watson and Francis Crick received the Nobel Prize in Physiology or Medicine for their discovery concerning the molecular structure of DNA. Thirteen years later, the declaration was made that “the average human polypeptide is more than 99 percent identical to its chimpanzee counterpart” (King and Wilson, 1975, pp. 114-115). This genetic similarity in the proteins and nucleic acids, however, left a great paradox—why do we not look or act like chimpanzees if our genetic material is so similar? King and Wilson recognized this quandary, and wrote: “The molecular similarity between chimpanzees and humans is extraordinary because they differ far more than many other sibling species in both anatomy and life” (p. 113). Nevertheless, the results matched what evolutionists had

hoped to find, and as such, the claim has reverberated through the halls of science for decades as compelling evidence that humans evolved from an ape-like ancestor.

One year following the Nobel ceremony for Watson and Crick, chemist Emile Zuckerkandl noted that the protein sequence of hemoglobin in humans and the gorilla differed by only 1 out of 287 amino acids. Zuckerkandl remarked: “From the point of view of hemoglobin structure, it appears that the gorilla is just an abnormal human, or man an abnormal gorilla, and the two species form actually one continuous population” (1963, p. 247). The molecular and genetic evidence only strengthened the evolutionary foundation for those who alleged that humans had emerged from primate ancestors. Professor of physiology Jared Diamond even titled one of his books *The Third Chimpanzee*, thereby viewing the human species as basically just another big mammal. From all appearances, it seemed that evolutionists had indeed won a battle—humans were 98% identical to chimpanzees. However, after spending his professional career looking for evolutionary evidence in molecular structures, biochemist Christian Schwabe admitted:

Molecular evolution is about to be accepted as a method superior to paleontology for the discovery of evolutionary relationships. As a molecular evolutionist I should be elated. **Instead it seems disconcerting that many exceptions exist to the orderly progression of species as determined by molecular homologies;** so many in fact that I think the exception, the quirks, may carry the more important message (1986, p. 280, emp. added).

On April 14, 2003, the International Human Genome Sequencing Consortium (led in the United States by the National Human Genome Research Institute and the Department of Energy) announced the successful completion of the Human Genome Project. The Consortium had completed its task a full two years ahead of schedule, and sequenced the entire human genome of 3.1 billion base pairs (see “Human Genome Report...,” 2003). Before this massive project was created, scientists estimated that humans possessed 80,000 to 100,000 genes (a gene is a section of DNA

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that is a basic unit of heredity; the genome constitutes the total genetic composition of an organism). As preliminary data from the genome project began to arrive, a special issue of *Science*, published on February 16, 2001, set the number of genes in the human genome at between 35,000 and 40,000 (see Pennisi, 2001, 291:1178). One year later almost to the day, *Science* reported a revised number—70,000 (Shouse, 2002, 295:1457; Haney, 2002). As of the writing of this article, the number stands at 20,000-25,000 (see “How Many Genes...,” 2005). It appears that approximately 1.5% of the human genome consists of genes that code for proteins. These genes are clustered in small regions, with large amounts of “non-coding” DNA (referred to as “junk DNA”) between the clusters. The specific function of these non-coding regions is only now being determined. These findings indicate that even if **all** the human genes were different from the genes of a chimpanzee, the DNA still could be 98.5 percent similar if the non-coding DNA of humans and chimpanzees was identical.

Because DNA is a linear array of those four bases—A, G, C, and T—only four possibilities exist at any specific point in a DNA sequence. The laws of chance tell us that two random sequences from species that have no ancestry in common will match at about one in every four sites. Thus even two unrelated DNA sequences will be 25 percent identical, not 0 percent identical (Marks, 2000, p. B-7).

Therefore a human and **any** earthly DNA-based life form must be at least 25% identical. Would it be correct, then, to suggest that daffodils are one-quarter human? The idea that daffodils are one-quarter human is neither profound nor enlightening, but rather ridiculous! There is hardly any biological comparison—except perhaps the DNA—that would make daffodils appear similar to humans. As Marks conceded:

[M]oreover, the genetic comparison is misleading because it ignores qualitative differences among genomes.... Thus, even among such close relatives as human and chimpanzee, we find that the chimp’s genome is estimated to be about 10 percent larger than the human’s; that one human chromosome contains a fusion of two small chimpanzee chromosomes; and that the tips of each chimpanzee chromosome contain a DNA sequence that is not present in humans (p. B-7).

The truth is, if we took all of the DNA from every cell, and then compared the DNA in monkeys and humans, the 4-5% difference in DNA would represent approximately **200 million differences in a human body, compared to that of an ape!** To help make this number understandable, consider the fact that if evolutionists were forced to pay you one penny for every one of those differences, you would walk away with \$2,000,000. Given those proportions, a 5% difference does not sound so small.

Furthermore, in 2004, researchers studying chromosome 22 of the chimpanzee reported: “83% of the 231 coding sequences,

including functionally important genes, show differences at the amino acid sequence level” (see International Chimpanzee Chromosome 22 Consortium, 2004). The scientist who headed the consortium, Yoshitaki Sakaki, told *The Scientist* that the difference is “much more complicated than we initially imagined or speculated” (as quoted in Holding, 2004). In fact, Dr. Sakaki went on to note: “83% of the genes have changed between the human and the chimpanzee—only 17% are identical—so that means that the impression that comes from the 1.2% [sequence] difference is [misleading]. In the case of protein structures, it has a big effect.” A “big effect” indeed!

CHROMOSOMAL COUNTS

It would seem to make sense that if humans and chimpanzees were genetically identical, then the manner by which they store DNA also would be similar. Yet it is not. DNA, the fundamental blueprint of life, is tightly packed into chromosomes. All cells that possess a nucleus contain a specific number of chromosomes. Common sense would necessitate that organisms that share a common ancestry would possess the same number of chromosomes. However, chromosome numbers in living organisms vary considerably. For example, certain animals, such as the mosquito (*Culex pipiens*) and nematode worm (*Caenorhabditis elegans*) have only 6, while a black mulberry (*Morus nigra*) plant has 308 (see Sinnott, et al., 1958). In addition, complexity does not appear to affect the chromosomal number. The radiolaria, a simple protozoan, has over 800, while humans possess 46. Chimpanzees, on the other hand, possess 48 chromosomes. A strict comparison of chromosome number would indicate that we are more closely related to the Chinese muntjac (a small deer found in Taiwan’s mountainous regions), which also possesses 46 chromosomes.

This hurdle of differing numbers of chromosomes may appear trivial, but we would do well to remember that chromosomes contain genes, which themselves are composed of DNA spirals. If the blueprint of DNA locked inside those chromosomes codes for only 46 chromosomes, then how can evolution account for the **loss** of two entire chromosomes? The job of DNA is to continually reproduce itself, and if we infer that this change in chromosome number occurred via evolution, then we are asserting that the DNA locked in the original number of chromosomes did not do its job correctly or efficiently. Considering that each chromosome carries many genes, los-

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	PREDICTION	FACTS
	Simple to Complex	Chromosome Count
↑	Man	Fern—512
	Dog	Crayfish—200
	Bat	Dog—78
	Herring Gull	Herring Gull—68
	Reptiles	Reptiles—48
	Fern	Man—46
	Crayfish	Bat—32

Comparison of chromosome numbers in various organisms

ing chromosomes does not make physiological sense, and very likely would prove deadly for new species. Are evolutionists ready to argue that **losing** chromosomes fits in with their “survival of the fittest” protocol? No respectable biologist would suggest that by **removing** one chromosome (or several), a new species likely would be produced. Science never has recorded an animal species created through the addition or removal of an entire chromosome. To remove even one chromosome could potentially remove the DNA codes for millions of vital body factors. Eldon Gardner summed it up by saying: “Chromosome number is probably more constant, however, than any other single morphological characteristic that is available for species identification” (1968, p. 211). Humans always have had 46 chromosomes, whereas chimps always have had 48.

To make matters worse for evolutionists, they must contend with the problem of explaining what happens when one loses (or gains) the genes found on a chromosome. We know today that a single gene controls many different traits. Scientifically, the process is known as pleiotropy (from the Greek *pleion*, meaning more, and *tropism*, meaning responses). Simply put, it means that genes act on a whole host of traits or actions. As Strauss remarked: “The emerging richness of pleiotropy means that any simple Darwinian notion of what is going on during natural selection has to be abandoned” (2005). Sally Otto, a University of British Columbia professor, commented: “When you knock out a single gene in an organism, you can today see that it affects the expression of something in the order of hundreds of other genes” (as quoted in Strauss, 2005). She went on to com-

ment: “You can’t change selection on one thing without changing everything.” Yet, many high school textbooks still promote the incorrect idea that each gene somehow is responsible for a single action or trait. Given the vast effects of losing even a single gene, one can only wonder what would happen at the loss of an entire chromosome, which consists of **many genes!**

REAL GENOMIC DIFFERENCES

One of the downfalls of previous molecular genetic studies has been the limit at which chimpanzees and humans could be compared accurately. Scientists often would use only 30 or 40 known proteins or nucleic acid sequences, and then from those they would extrapolate their results for the entire genome. Today, however, we have the majority of the human genome sequences, almost all of which have been released and made public. **This allows scientists to compare individual nucleotide base pair sequences between humans and primates—something that was not possible prior to the Human Genome Project.** In January 2002, a study was published in which scientists had constructed and analyzed a first-generation human/chimpanzee comparative genomic map. This study compared the alignments of 77,461 chimpanzee bacterial artificial chromosome [BAC] end sequences to human genomic sequences. Fujiyama and his colleagues “detected candidate positions, including two clusters on human chromosome 21 that suggest large, nonrandom regions of differences between the two genomes” (2002, 295:131). In other words, the comparison revealed some “large” differences between the genomes of chimps and humans.

Amazingly, the authors found that only 48.6% matched chimpanzee nucleotide sequences. And the human Y chromosome was only 4.8% identical to the chimpanzee sequences! This study analyzed the alignments of 77,461 chimpanzee sequences to human genomic sequences obtained from public databases. Of these, 36,940 end sequences were unable to be mapped to the human genome (p. 131). Almost 15,000 of those sequences that did not match with human sequences were speculated to “correspond to unsequenced human regions or are from chimpanzee regions that have diverged substantially from humans or did not match for other unknown reasons” (p. 132). While the authors noted that the quality and usefulness of the map should “increasingly improve as the finishing of the human genome sequence proceeds” (p. 134), the data already support what creationists have suggested for years—the 98% equivalency figure between chimpanzees and humans is grossly misleading, as Britten’s study revealed (Britten, 2002).

Exactly **how** misleading came to light in a news article—“Jumbled DNA Separates Chimps and Humans”—published in the October 25, 2002 issue of *Science*. The first three sentences of the article, written by Elizabeth Pennisi (a staff writer for *Science*), represented a “that was then, this is now” type of admission of defeat. She wrote:

For almost 30 years, researchers have asserted that the DNA of humans and chimps is at least 98.5% identical. Now research reported here last week at the American Society for Human Genetics meeting suggests that **the two primate genomes might not be quite as similar after all.** A closer look has uncovered nips and tucks of homologous sections of DNA that weren’t noticed in previous studies (298:719, emp. added).

Genomicists Kelly Frazer and David Cox of Perlegen Sciences in Mountain View, California, along with geneticists Evan Eichler and Devin Locke of Case Western University in Cleveland, Ohio, compared human and chimp DNA, and discovered a wide range of insertions and deletions (anywhere from between 200 bases to 10,000 bases). Cox commented: “The implications could be profound, because such genetic hiccups could disable entire genes, possibly explaining why our closest cousin seems so distant” (as quoted in Pennisi, 298:721).

Roy Britten, of the California Institute of Technology in Pasadena, analyzed both chimp and human genomes with a customized computer program. To quote Pennisi:

He compared 779,000 bases of chimp DNA with the sequences of the human genome, both found in the public repository GenBank. Single-base changes accounted for 1.4% of the differences between the human and chimp genomes, and insertions and deletions accounted for an additional 3.4%, he reported in the 15 October [2002] *Proceedings of the National Academy of Sciences*. Locke's and Frazer's groups didn't commit to any new estimates of the similarity between the species, but **both agree that the previously accepted 98.5% mark is too high** (298:721, emp. added).

While the Locke and Frazer team was unwilling to commit to any new estimate of the similarity between chimps and humans, Britten was not. In fact, he titled his article in the October 15, 2002 *Proceedings of the National Academy of Sciences*, "Divergence between Samples of Chimpanzee and Human DNA Sequences is 5%" (Britten, 99:13633-13635). In the abstract accompanying the article, he wrote:

The conclusion is that the old saw that we share 98.5% of our DNA sequence with chimpanzee is probably in error. For this sample, a better estimate would be that 95% of the base pairs are exactly shared between chimpanzee and human DNA (99:13633, emp. added).

The news service at NewScientist.com reported the event as follows:

It has long been held that we share 98.5 per cent of our genetic material with our closest relatives. That now appears to be wrong. In fact, we share less than 95 per cent of our genetic material, a **three-fold increase in the variation between us and chimps** (see Coghlan, 2002, emp. added).

It seems that, as time passes and scientific studies increase, humans appear to be less like chimps after all. In a separate study, Barbulescu and colleagues also uncovered another major difference in the genomes of primates and humans. In their article "A HERV-K Provirus in Chimpanzees, Bonobos, and Gorillas, but not Humans," the authors wrote: "**These observations provide very strong evidence that, for some fraction of the genome, chimpanzees, bonobos, and gorillas are more closely related to each other than they are to humans**" (2001, 11:779, emp. added). Such data go squarely against what evolutionists have contended for decades—that chimpanzees are closer genetically to humans than they are to gorillas. Another study using interspecies representational difference analysis (RDA) between

humans and gorillas revealed **gorilla-specific DNA sequences** (Toder, et al., 2001)—that is to say, gorillas possess sequences of DNA that are not found in humans. The authors of this study suggested that the sequences found in gorillas, but not humans, "could represent either ancient sequences that got lost in other species, such as human and orang-utan, or, more likely, recent sequences which evolved or originated specifically in the gorilla genome" (9:431).

The differences between chimpanzees and humans are not limited to genomic variances. In 1998, a structural difference between the cell surfaces of humans and apes was detected. After studying tissues and blood samples from great apes, and sixty humans from various ethnic groups, Elaine Muchmore and colleagues discovered that human cells are missing a particular form of sialic acid (a type of sugar) found in all other mammals (1998, 107 [2]:187). This sialic acid molecule is found on the surface of every cell in the body, and is thought to carry out multiple cellular tasks. This seemingly "minuscule" difference can, in fact, have far-reaching effects, and might explain why surgeons were unable to transplant chimp organs into humans in the 1960s. Keeping this in mind, a person never should declare, with a simple wave of the hand, "chimps are almost identical to us," simply because of a large genetic overlap.

Homology (i.e., similarity) does not prove common ancestry. The entire genome of the tiny nematode (*Caenorhabditis elegans*) also has been sequenced as a tangential study from the Human Genome Project. Of the 5,000 best-known human genes, 75% have matches in the worm (see "A Tiny Worm Challenges Evolution"). Does this mean that we are 75% identical to a nematode? Just because living creatures share some genes with humans does not mean there is a linear ancestry. Biologist John Randall admitted this when he wrote:

The older textbooks on evolution make much of the idea of homology, pointing out the obvious resemblances between the skeletons of the limbs of different animals. Thus the "pentadactyl" [five bone—BT/BH] limb pattern is found in the arm of a man, the wing of a bird, and flipper of a whale, and this is held to indicate their common origin. Now if these various structures were transmitted by the same gene couples, varied from time to time by mutations and acted upon by environmental selection, the theory would make good sense. Unfortunately this

is not the case. Homologous organs are now known to be produced by totally different gene complexes in the different species. The concept of homology in terms of similar genes handed on from a common ancestor has broken down... (as quoted in Fix, 1984, p. 189).

Yet textbooks and teachers still proclaim that humans and chimps are 98% genetically identical. The evidence clearly demonstrates vast molecular differences—differences that can be attributed to the fact that humans, unlike animals, were created in the image and likeness of God (Genesis 1:26-27).

"MITOCHONDRIAL EVE"

On the first day of 1987, a scientific "discovery" seized the attention of the popular press. The original scientific article that caused all the commotion—"Mitochondrial DNA and Human Evolution"—appeared in the January 1, 1987 issue of *Nature*, and was authored by Rebecca Cann, Mark Stoneking, and Allan C. Wilson (see Cann, et al., 1987). These three scientists announced that they had "proven" that all modern human beings can trace their ancestry back to a single woman who lived 200,000 years ago in Africa. This one woman was nicknamed "Eve" (a.k.a., "Mitochondrial Eve")—much to the media's delight. An article in the January 26, 1987 issue of *Time* magazine bore the headline, "Everyone's Genealogical Mother: Biologists Speculate that 'Eve' Lived in Sub-Saharan Africa" (Lemonick, 1987). A year later, that "speculation" became a major *Newsweek* production titled "The Search for Adam and Eve" (Tierney, et al., 1988). The provocative front cover presented a snake, tree, and a nude African couple in a "Garden of Eden" type setting. The biblical-story imagery was reinforced as the woman offered a piece of fruit to the man. Sadly, *Newsweek* and other media outlets have not been quite so quick to give readers an update on Mitochondrial Eve.

In 2002, we authored an article ("The Demise of 'Mitochondrial Eve'") on this topic for the Apologetics Press Web site. In that article, we noted how rapidly things can, and often do, change in science. As an example of that fact, we discussed the well-known evolutionary icon, "Mitochondrial Eve," a female who was alleged to have lived in Africa at the beginning of the Upper Pleistocene period (between 100,000 and 200,000 years ago). Eve had been described as the most-recent common ancestor of all humans on Earth today. In fact,

as late as mid-2002, some evolutionists still were touting her as exactly that—in spite of overwhelming scientific evidence to the contrary. Geneticist Spencer Wells, in his book, *The Journey of Man: A Genetic Odyssey*, referred to Eve as “a real person who lived at that time—the common ancestor of everyone alive today” (2002, p. 54). Spencer went on to inform his readers:

Crucially, though, the fact that a single ancestor gave rise to all of the diversity present today does not mean that this was the only person alive at the time—only that the descendant lineages of the other people alive at the same time died out (p. 32).

This makes a great “just-so” story. But is any of it true? As we pointed out in our 2002 article on “The Demise of ‘Mitochondrial Eve,’” no, it is not. The scientists who performed the original work that led to the creation of Eve (see Cann, et al., 1987) used estimates of the frequency of mutations that occur in the DNA within a cell’s mitochondria, in an attempt to determine how far back in time our alleged “most-recent common ancestor” could be traced (an explanation of how this works follows below). In performing this work, the researchers **assumed** that **all** the DNA in the cell’s mitochondria had been passed down generation by generation **only** by the female. Other evolutionists who performed similar studies continued to make that same assumption—until reports began appearing in 1999, documenting that mitochondrial DNA also can be (and often is) passed down generation to generation by the **male**. This information destroyed the basic assumption upon which “Mitochondrial Eve” had been built—and ultimately led to her “demise.”

A word of explanation is in order at this point. For decades, evolutionists had been trying to determine the specific geographical origin of humans—whether we all came from one specific locale, or whether there were numerous smaller pockets of people placed around the globe. When they set out to determine the specific geographical origin of humans, a curious piece of data came to light. As they considered various human populations, Africans seemed to show much more genetic variation than non-Africans (i.e., Asians, Europeans, Native Americans, Pacific Islanders, et al.). According to molecular biologists, this increased variability is the result of African populations being older, thus, having had more time to accumulate mutations and diverge from one another. This assumption led some researchers to postulate that

Africa was the ancient, much-touted “cradle of civilization” from which all of humanity had emerged.

The genetic material (DNA) in a cell’s nucleus controls the functions of the cell, bringing in nutrients from the body and making hormones, proteins, and other chemicals. Outside the nucleus is an area known as the cytoplasmic matrix (generally referred to as the cytoplasm), which contains, among other things, tiny bean-shaped organelles known as mitochondria. These often are described as the “powerhouses” or “energy factories” of the cell.

Mitochondria contain their own DNA, which they use to make certain proteins; the DNA in the nucleus oversees production of the rest of the proteins necessary for life and its functions. However, mitochondrial DNA (mtDNA) was thought to be special for two reasons. First, it is short and relatively simple (in comparison to the DNA found within the nucleus), containing only thirty-seven genes (instead of the 20,000+ genes located in the nuclear DNA). This makes it relatively easy to analyze. Second, unlike nuclear DNA, which each person inherits in equal portions from both parents, mitochondrial DNA was thought to be passed on only through the mother’s line (more about this later). Working from the assumption that mtDNA is passed down to the progeny **only by the mother**, Dr. Cann and her coworkers believed that each new cell should contain copies of only the egg’s mitochondria. In trying to draw the human family tree, therefore, researchers took a special interest in these minute strands of the genetic code. What they **really** were interested in locating, of course, was the variations in mitochondrial DNA from one group of people to another.

Although our mtDNA should be, in theory at least, the same as that found in our mother’s mtDNA, small changes (known as mutations) in the genetic code can, and do, arise. On rare occasions, mutations are serious enough to do harm. More frequently, however, the mutations have no effect on the proper functioning of either the DNA or the mitochondria. In such cases, the mutational changes will be preserved and carried on to succeeding generations.

Theoretically, if scientists could look farther and farther into the past, they would find that the number of women who contributed the modern varieties of mitochondrial DNA gets less and less until, finally, we arrive at one “original” mother. She, then, would be the only woman out of all the women living in her day to have a daughter

in every generation till the present. Coming forward in time, we would see that the mtDNA varieties found within her female contemporaries were gradually eliminated as their daughters did not have children, had only sons, or had daughters who did not have daughters. This does not mean, of course, that we would **look like** this alleged ancestral mother; rather, it means only that we would have gotten our mitochondrial DNA from her.

To find this woman, researchers compared the different varieties of mtDNA in the human family. Since mtDNA occurs in fairly small quantities, and since the researchers wanted as large a sample as possible from each person, they decided to use human placentas as their source of the mtDNA. So, Rebecca Cann and her colleagues selected 145 pregnant women and two cell lines representing the five major geographic regions: 20 Africans, 34 Asians, 46 Caucasians, 21 aboriginal Australians, and 26 aboriginal New Guineans (Cann, et al., 1987, 325:32). All placentas from the first three groups came from babies born in American hospitals. Only two of the 20 Africans actually were born in Africa.

After analyzing a portion of the mtDNA in the cells of each placenta, they found that the differences “grouped” the samples by region. In other words, Asians were more like each other than they were like Europeans, people from New Guinea were more like each other than they were like people from Australia, and so on.

Next, they saw two major branches form in their computer-generated tree of recent human evolution. Seven African individuals formed one distinct branch, which started lower on the trunk than the other four groups. This was because the differences among these individuals were much greater than the differences between other individuals and other groups. More differences mean more mutations, and hence more time to accumulate those changes. If the Africans have more differences, then their lineage must be older than all the others. The second major branch bore the non-African groups and, significantly, a scattering of the remaining thirteen Africans in the sample. To the researchers, the presence of Africans among non-Africans revealed an African common ancestor for the non-African branches, which, likewise, meant an African common ancestor for both branches. The nickname “Eve” stuck to this “hypothetical common ancestral mother,” and fired the imagination of the media.

Having concluded, then, that the African group was the oldest, Dr. Cann and her colleagues wanted to find out just **how** old the group might be. To do this, they used what is known as a “molecular clock” that, in this case, was based on mutations in the mtDNA. The rate at which the clock ticked was determined from the accumulation of changes over a given period of time. For example, if the assumption was made that there was one mutation every 1,000 years, and if scientists 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.

The researchers looked in two places for their figures. First, they compared the mtDNA from humans with that from chimpanzees, and then used paleontology and additional molecular data to determine the age of the supposed common ancestor. This (and similar calculations on other species) revealed a mutation rate in the range of 2% to 4% per million years. Second, they compared the groups in their study that were close geographically, and took the age of the common ancestor from estimated times of settlement as indicated by anthropology and archaeology. Again, 2% to 4% every million years seemed reasonable to them.

Cann, and her coworkers suggested that the common mitochondrial ancestor diverged from all others by an average of 0.57% (325:34), which meant that she must have lived sometime between approximately 140,000 ($0.57 \div 4 \times 1,000,000$) and 290,000 ($0.57 \div 2 \times 1,000,000$) years ago. The figure of 200,000 was chosen as a suitable round number.

The results obtained from analysis of mitochondrial DNA eventually led to what is known in evolutionary circles as the “Out of Africa” theory. This is the idea that the descendants of Mitochondrial Eve were the only ones to colonize Africa and the rest of the world, supplanting all other hominid populations in the process. Many (although not all) evolutionists claim that such an interpretation is in accord with archaeological, paleontological, and other genetic data (see, for example, Stringer and Andrews, 1988; for an opposing viewpoint, see the written debate in the April 1992 issue of *Scientific American*).

While most evolutionists have accepted the mitochondrial DNA tree, they differ widely in their views regarding both the source of the nuclear DNA and the “humanity” of Eve. Some believe that Eve contributed **all** the nuclear DNA, in addition to the mitochondrial DNA. Some believe she was an “archaic” *Homo sapiens*, while others believe she was fully human. The exact interpretation is hotly debated because mitochondrial DNA is “something of a passenger in the genetic processes that led to the formation of new species: it therefore neither contributes to the formation of a new species nor reveals anything about what actually happened” (Lewin, 1987, 238:24). As Wells went on to observe:

As we have seen, people inherit their genes from their parents, so the study of genetic history is also a study of the history of the people carrying these genes. Ultimately, though, we hit a barrier when we trace back into the past beyond a few thousand generations—

there is simply no more variation to tell us about these questions of very deep history. Once we reach this point, there is nothing more that human genetic variation can tell us about our ancestors. We all coalesce into a single genetic entity—“Adam” in the case of the Y-chromosome, “Eve” in the case of mtDNA—that existed for an unknowable period of time in the past. While this entity was a real person who lived at that time—the common ancestor of everyone alive today—we can’t use genetic methods to say very much about **their** ancestors. We can ask questions about how Adam and Eve relate to other species (are humans more closely related, as a species, to chimpanzees or sturgeons?), but we cannot say anything about what happened to the human lineage itself prior to the coalescence point (2002, p. 54, emp. in orig.).

The “reality” of Eve as the “most-recent common ancestor of all humans on Earth today,” however, depended upon two important “ifs.” **If** humans received mtDNA only from their mothers, then researchers could “map” a family tree using that information. And, **if** the mutations affecting mtDNA had indeed occurred at constant rates, then the mtDNA could serve as a molecular clock for timing evolutionary events and reconstructing the evolutionary history of extant species. But, as we pointed out in our 2002 Web article, “The Demise of ‘Mitochondrial Eve,’” it is the “ifs” in these two sentences where the problem lies. The fact is, **we now know that both assumptions are wrong!**

First, let us examine the assumption that mtDNA is derived exclusively from the mother. In response to a paper in *Science* in 1999, anthropologist Henry Harpending of the University of Utah lamented: “There is a cottage industry of making gene trees in anthropology and then interpreting them. This paper will invalidate most of that” (as quoted in Strauss, 1999, 286:2436). Just when women thought they were getting their fair shake in science, the tables turned. As one study noted:

Women have struggled to gain equality in society, but biologists have long thought that females wield absolute power in a sphere far from the public eye: in the mitochondria, cellular organelles whose DNA is thought to pass intact from mother to child with no paternal influence. On page 2524 however, a study...finds signs of mixing between maternal and paternal mitochondrial DNA (mtDNA) in humans and chimpanzees. **Because biologists have used mtDNA as a tool to trace human ancestry and relationships,**

SPEAKING SCHEDULES

Dr. Bert Thompson

May 6-8	Haleyville, AL	(205) 486-6963
May 13-15	Mechanicsville, VA	(804) 746-8224

Dr. Brad Harrub

April 29-May 1	Hillsboro, TN	(931) 596-2541
June 12-15	Tyler, TX	(903) 592-0809

Dr. Dave Miller

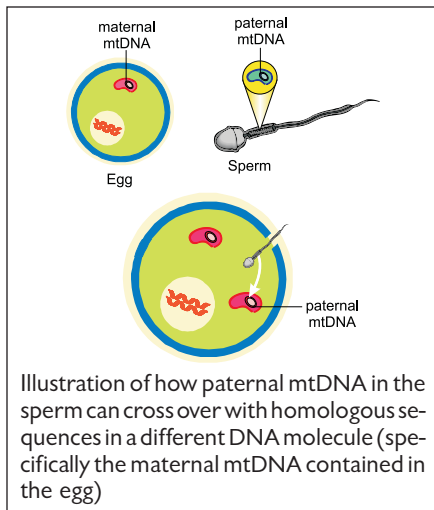
May 6-8	Jacksonville, FL	(904) 693-2274
May 13-14	Pulaski, TN	(931) 363-2777

Kyle Butt

May 20-21	Lake City, FL	(386) 752-6010
June 1	Gulf Shores, AL	(251) 968-7769

Eric Lyons

May 4, 11, 18, 25	Birmingham, AL	(205) 988-5808
June 8	Gulf Shores, AL	(251) 968 - 7769



the finding has implications for everything from the identification of bodies to the existence of a “mitochondrial Eve” 200,000 years ago (Strauss, 286:2436, emp. added).

One year later, researchers made the following startling admission:

Mitochondrial DNA (mtDNA) is generally assumed to be inherited exclusively from the mother.... Several recent papers, however, have suggested that elements of mtDNA may sometimes be inherited from the father. This hypothesis is based on evidence that mtDNA may undergo recombination. If this does occur, maternal mtDNA in the egg must cross over with homologous sequences in a different DNA molecule; paternal mtDNA seems the most likely candidate.... **If mtDNA can recombine, irrespective of the mechanism, there are important implications for mtDNA evolution and for phylogenetic studies that use mtDNA (Morris and Lightowlers, 2000, 355:1290, emp. added).**

And now we know that these are more than small “fractional” amounts of mtDNA coming from fathers. The August 2002 issue of the *New England Journal of Medicine* reported:

Mammalian mitochondrial DNA (mtDNA) is thought to be strictly maternally inherited.... Very small amounts of paternally inherited mtDNA have been detected by the polymerase chain reaction (PCR) in mice after several generations of interspecific backcrosses.... We report the case of a 28-year-old man with mitochondrial myopathy due to a novel 2-bp mtDNA deletion.... We determined that the mtDNA harboring the mutation was paternal in origin and accounted for **90 percent** of the patient’s muscle mtDNA (Schwartz and Vissing, 2002, 347:576, emp. added).

Ninety percent! A 2002 study concluded:

Nevertheless, even a single validated example of paternal mtDNA transmission suggests that the interpretation of inheritance patterns in other kindreds thought to have mitochondrial disease should not be based on the dogmatic assumption of absolute maternal inheritance of mtDNA.... The unusual case described by Schwartz and Vissing **is more than a mere curiosity (Williams, 2002, 347:611, emp. added).**

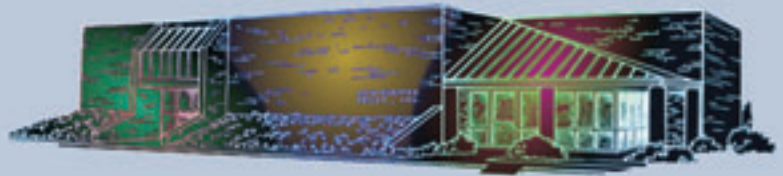
And all this time, evolutionists have been selectively shaping our family tree by using what was alleged to be only **maternal** mtDNA!

[to be continued]

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NOTE FROM THE EDITOR



ANNOUNCING: REVISED VERSIONS OF TWO OF OUR MOST POPULAR KIDS' BOOKS

What child—growing up in a home where God is revered and His Word is respected—has not asked the question: “Mom (or dad), how do you know the Bible is from God?” It’s a valid question. And it deserves an equally valid answer.

What, then, should be a parent’s (or teacher’s) response? It will not do to simply say, “Well, honey, we just ‘know in our heart’ that the Bible is God’s Word,” or “Well, we’ve always been taught, and we’ve always believed, that the Bible is the Word of God.” Those are not appropriate (or adequate) answers for a young, inquiring mind. We must do better. And each child who bothers to ask the question deserves better! In 2002, we announced the availability of Kyle Butt’s new book for kids, *How do You Know the Bible is from God?*, which was written to help both parents and teachers “do better.” The book was an instant best seller.

Now I am pleased to announce the availability of the revised edition of this extremely popular book. The original edition was the very first book we had published in a hardback version, and also was the first book we had published in full color throughout! The original was indeed a thing of beauty to behold. The revised version is even more so!

The original was an 8.5 x 11-inch book; the revised edition is a more “kid friendly” 8 x 8-inch volume. And while the new book contains the same trustworthy, reliable text as the first edition (which parents and teachers alike will appreciate), it also is filled with additional illustrations and crisp, fresh artwork.

The new 62-page book has a full-color, firm, laminated cover (designed to last a long time in the hands of a child), and sells for only \$7.95. Truth be told, it would be a steal at twice the price.

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In addition, several months ago, we also revised our children’s coloring book on dinosaurs (which always is popular with “the younger set”). Kids, as we all recognize, love dinosaurs. The coloring book not only gives them lots of dinosaurs to color, but also offers a variety of activities such as mazes, puzzles, connect the dots, and cartoons (featuring our famous mole-sleuth, Digger Doug, and his friend, Iguana Don).

The book, which is printed on crisp, white, crayon-friendly paper, will keep budding artists busy for hours. One of the greatest things about this particular coloring book, however, is that it doesn’t just “keep kids busy.” It also teaches them biblical and/or scientific truths about these “terribly great lizards.” [If you watch a child interact with the book, you’ll be surprised at how frequently he or she stops to **read**—not **color**.] At \$2.00, this is a great gift for a child. Call us toll free at 800/234-8558 to order these two volumes. Order both for only \$7.95 (plus shipping) until May 30 and save \$2.

Bert Thompson

