IS EVOLUTION A FACT?
— A REBUTTAL TO AN EVOLUTIONIST'S CLAIMS —

During the summer of 2009 I was invited to participate in a written, online debate on the topic of organic evolution. One disputant, an evolutionist, had agreed to affirm the following proposition:

> **RESOLVED**: Macroevolution (as suggested by the General Theory of Evolution—as opposed to microevolution, as suggested by the Special Theory of Evolution) **is** a fact, and as such, represents a correct scientific explanation of the origin of the Universe and life on Earth.

I agreed to respond in the negative to the evolutionist's written arguments. Once that had been accomplished, a reverse process would ensue. As a non-evolutionist, I would affirm the following proposition:

> **RESOLVED**: Macroevolution (as suggested by the General Theory of Evolution—as opposed to microevolution, as suggested by the Special Theory of Evolution) is **not** a fact, and as such, does not represent a correct scientific explanation of the origin of the Universe and life on Earth.

[**NOTE TO THE READER**: In my affirmative argument, I will deal with the so-called "factuality" of organic evolution. Here, however, I simply plan to offer rebuttals to the alleged proofs of evolution offered by my evolutionist opponent.]

EVOLUTIONIST'S FIRST ARGUMENT: BACTERIAL ANTIBIOTIC RESISTANCE

Thanks to various drug treatments available during the 1960s and 1970s, humanity appeared to have won the battle against many dreaded diseases. Today, however, microbial antibiotic resistance makes it seem as if initial appearances may have been deceptive, and subsequent jubilation premature. According to some evolutionists, "the culprit is evolution" (Miller, 1999, p. 50). In fact, Harvard evolutionary biologist Stephen Palumbi even went so far as to suggest that "bacterial evolution outwits one antibiotic after another" (as quoted in Hayden, 2002, 133[4]:48).

Most well-read people are familiar with the concept—microorganisms that change over time, seem at first to perceive, and then thwart, our most-impressive medical efforts to kill them. But do they do it "on purpose"? And is evolution "the culprit"? The answer to both questions is "No."

Evolutionists frequently use this idea of "the rapid evolution of microorganisms" as "observed proof" for evolution, and are fond of making statements such as these: "Bacteria experience far more mutations because there are so many more individuals and generations. This and the short reproductive cycle allow beneficial mutations to be exploited by natural selection rapidly" (Berra, 1990, p. 54). The claim is that drug-resistant strains of many types of such organisms have evolved from strains that, at one time, were susceptible to these same drug treatments. Evolutionists would have us believe that microorganisms are "selectively adapting" to our drug treatments through a mechanism that involves genetic mutations—and that this portends great strides for organic evolution. Scientific studies, however, indicate an alternative explanation for this acquired immunity—one that argues **against** organic evolution.

Researchers Monica Sala and Simon Wain-Hobson (of the world-famous Pasteur Institute in France) published a paper several years ago titled, "Are RNA Viruses Adapting or Merely Changing" (2000). In this particular study, the scientists examined 85 sets of proteins from viruses that were known to infect bacteria, plants, and mammals. According to the evolutionary hypothesis, once drug therapy alleviates the majority of susceptible microorganisms, only those that remain have mutated during replication and thus are resistant. Evolutionists believe that this represents a type of natural selection taking place, in which mutations "purposefully" confer drug resistance. Speaking about bacterial replication, Miller stated, "The result is unavoidable, given the millions of genetic duplications that occur in a bacterial population in just a few days. Sooner or later, the 'right' mutation shows up, and it causes the individual bacteria that possess it to prosper at our expense" (1999, p. 50).

However, the data from Sala and Wain-Hobson indicated that the changes we were seeing were due to simple genetic drift (i.e., **random** genetic variations) rather than a response to drugs. Furthermore,

these studies demonstrated that this genetic drift occurred at a constant rate, even when microorganisms were subjected to drug treatments (in other words, organisms changed whether or not they had been exposed to drugs). Plus, the appearance of "drug resistance" may not be as "new" as evolutionists have led us to believe. Modeling studies examining HIV-resistant mutants have demonstrated that drug resistant strains were present **before** drug therapy began (see Ribeiro and Sebastian, 2000), which indicates that the changes in these viruses are occurring randomly, rather than in response to a particular drug. There is additional evidence to substantiate such a claim. In an article titled "Superbugs not Super After All," scientist Carl Wieland wrote,

> "That some germs were already resistant to man-made antibiotics before these were invented is common knowledge to microbiologists. Soil samples from villages where modern antibiotics had never been used show that some of the germs are already resistant to drugs like methicillin which have never existed in nature" (1997/1998, 20[1]:11).

Additionally, in 1988 researchers did autopsies on three of the Northwest Passage explorers who froze to death in the Arctic in 1845. Bacteria from their colons were cultured (with great care, in order to avoid any possible contamination), and found that many of the bacteria already were resistant to the most-powerful modern-day antibiotics (see Wieland, 1994; McGuire, 1998).

Furthermore, microorganisms like bacteria do not become resistant to antibiotics merely by experiencing genetic mutations. In fact, there are at least three known genetic mechanisms by which such resistance can be conferred. First, there are instances where **mutations** produce antibiotic-resistant strains of microorganisms. Second, there is the process of **conjugation**, which among bacteria is analogous to copulation in humans. During conjugation, two bacterial cells join, and an exchange of genetic material occurs. Inside many bacteria there is a somewhat circular piece of self-replicating, extra-nuclear DNA known as a plasmid, which codes for enzymes necessary for the bacteria's viability. Certain of these enzymes, coincidentally, are able to catalyze the breakdown of antibiotics, thus conferring upon the bacteria resistance to antibiotics. During conjugation, plasmids in one organism that are responsible for resistance to antibiotics can be transferred to an organism that previously did not have such resistance. Third, bacteria can incorporate into their own genetic machinery short, foreign pieces of DNA through either of two types of DNA transposition—**transformation** or **transduction**. In transformation, a piece of DNA from the surrounding environment (perhaps left there when another bacterium died) is absorbed into the bacterial cell. In transduction, a foreign piece of DNA is transported physically into the cell via a virus. As a result of incorporating new genetic material, an organism can become resistant to antibiotics when heretofore it was not.

Do microorganisms change over time? Yes. Are they "purposefully evolving"? No. First, the genetic mutations responsible for antibiotic resistance in bacteria do not arise as a result of the "need" of the organisms to develop such resistance. As evolutionist Douglas Futumya noted,

> "...the adaptive 'needs' of the species do not increase the likelihood that an adaptive mutation will occur; mutations are not directed toward the adaptive needs of the moment.... Mutations have causes, but the species' need to adapt isn't one of them" (1983, pp. 137,138).

What does this mean? Simply put, bacteria did not "mutate" as a result of being exposed to antibiotics; the mutations responsible for the resistance were present in the bacterial population even prior to the discovery or use of the antibiotics. Joshua Lederberg's experiments on streptomycin-resistant bacteria in 1952 showed that bacteria that never had been exposed to the antibiotic already possessed the mutations that conferred the resistance (see Lederberg and Lederberg, 1952).

Second, while certain pre-existing mutations may confer to bacteria antibiotic resistance, such mutations also may decrease the organism's viability in other ways. For example, "the surviving strains are usually less virulent, and have a reduced metabolism and so grow more slowly. This is hardly a recommendation for 'improving the species by competition' (i.e., survival of the fittest)" [Bowden, 1991, p. 56, parenthetical item in orig.]. Just because a mutation provides an organism with a certain trait does not mean necessarily that the organism **as a whole** has been helped. For example, people afflicted with the mutant gene for sickle-cell anemia are "carriers" of the disease, but do not die from it. Such people are inex-

plicably resistant to malaria, which at first would seem to be an excellent example of a "good mutation." However, that is not the entire story. While it is true that such people are resistant to malaria, it also is true that they are not as healthy, do not possess much stamina, and do not live as long as their unafflicted counterparts. Bacteria may be resistant to a certain antibiotic, but that resistance frequently comes at a price (e.g., reduced metabolism, slower growth, etc.). From an evolutionary point of view, in the grand scheme of things this is harmful, not beneficial.

Third, regardless of how bacteria acquired their antibiotic resistance (i.e., by mutation, conjugation, or by transposition), **the fact remains that they still are exactly the same bacteria *after* receiving that trait as they were *before* receiving it**. This so-called "proof" of evolution turns out to be not vertical **macro**evolution but horizontal **micro**evolution (i.e., adaptation). In other words, these bacteria "...are still the same bacteria and of the same type, being only a variety that differs from the normal in its resistance to the antibiotic. No new 'species' have been produced" (Bowden, p. 56). Thus, no real "organic evolution" has occurred.

In commenting on the changing, or sharing, of genetic material, Walter ReMine suggested that "it has not allowed bacteria to arbitrarily swap major innovations such as the use of chlorophyll or flagella. The major features of microorganisms fall into well-defined groups that seem to have a nested pattern like the rest of life" (1993, p. 404). What does Dr. ReMine mean by his use of the term "nested pattern"? Microbiologists have studied quite extensively two specific genera of bacteria in their attempts to understand antibiotic resistance: *Escherichia* and *Salmonella*. Of these, the genus *Escherichia* perhaps has been used more than any other in the studies of genetic mutations. In speaking about *Escherichia* in an evolutionary context, the renowned zoologist of France, Pierre-Paul Grassé, observed,

> "Bacteria, the study of which has formed a great part of the foundation of genetics and molecular biology, are the organisms which, because of their huge numbers, produce the most mutants…. [B]acteria, despite their great production of intraspecific varieties, exhibit a great fidelity to their species. The bacillus *Escherichia coli*, whose mutants have been studied very carefully, is the best example. The reader will agree that it is surprising, to say the least, to want to prove evolution and to discover its mechanisms and then to choose as a material for this study a being which practically stabilized a billion years ago" (1977, p. 87).

While no one doubts that, because of bacteria's sheer numbers and brief reproductive cycles, they potentially would be exposed to more mutations than other organisms, the fact remains that such mutations have not produced "new" organisms. *E. coli* (to choose just one example of many) has undergone billions of years' worth of mutations (according to the evolutionists' timetable), yet still remains "stabilized" in its "nested pattern." Species remain intact in spite of the sharing, or changing, of genetic material. So what, exactly, is it that natural selection has allegedly accomplished in favor of organic evolution? An antibiotic-resistant *Escherichia coli* is **still** *Escherichia coli*. Neither mutations nor DNA transposition has altered the fact that bacteria remain exactly what they have always been—**down to their very genus and species**. The suggestion that the development of antibiotic resistance in bacteria somehow "proves" organic evolution is horribly flawed. Evolution requires change across phylogenetic boundaries, and in the case of antibiotic-resistant bacteria, that has not occurred—which means that no true (organic) macroevolution has taken place.

EVOLUTIONIST'S SECOND ARGUMENT: DNA AND HEREDITY

In the 1940s, scientist O.T. Avery showed that traits could be passed from one bacterium to another via a chemical known as deoxyribonucleic acid [DNA] (see Avery, et al., 1944, 79:137-158). The late, eminent taxonomist of Harvard, Ernst Mayr, wrote concerning this event, "A new era in developmental genetics was opened when Avery demonstrated that DNA was the carrier of the genetic information" (1997, p. 166). The still-new science of genetics was advanced greatly in 1953 by the discovery of the chemical code within cells that provides the genetic instructions. It was in that year that James D. Watson of the United States, and Francis H.C. Crick of Great Britain, published their landmark paper in *Nature* about the composition and helical structure of DNA (1953, 171:737-738). Nine years later, in 1962, they were

awarded the Nobel Prize in Medicine or Physiology for their stellar achievement in elucidating the structure of DNA.

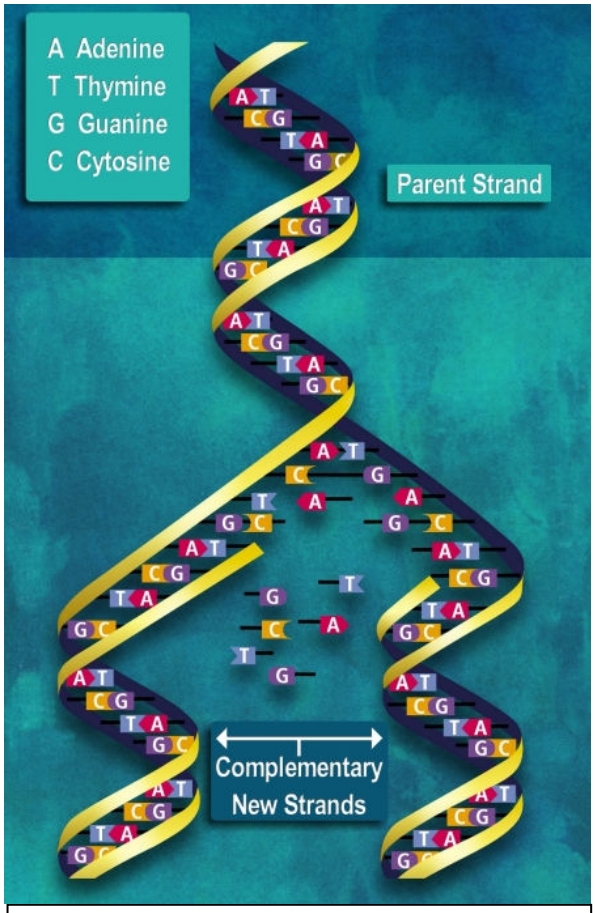

Figure 1 — DNA shown in double-helix, parent-strand form (top), and during replication of two new complementary strands (bottom). Source: U.S. Department of Energy Human Genome Program [on-line], URL: http://www.ornl.gov/hgmis

DNA, Genes, and Chromosomes

In most organisms, the primary genetic material is DNA [although some viruses, primarily retroviruses, contain only RNA (see Nicholl, 1994, pp. 9-10; Ridley, 1999, p. 9).] What is DNA, and how does it work? [It is not my intention here to present an in-depth examination of the inner workings of the DNA molecule since excellent discussions are available elsewhere (see Kautz, 1988, pp. 43-47; Davis and Kenyon, 1989, pp. 62-64; Suzuki and Knudtson, 1989, pp. 41-45).] In his book, *The Case Against Accident and Self-Organization,* Dean Overman provided the following valuable summary:

> "A DNA molecule is comprised of thousands of long chains of nucleotides (polynucleotides) each consisting of three parts. One part is the pentose or five carbon sugar known as deoxyribose. A second part is a phosphate group, and the third part is a nitrogen base of either adenine (A), guanine (G), cytosine (C) or thymine (T). Alternating sugar and phosphate molecules connect each nucleotide chain in a ladder type configuration coiled around a central axis in a twisted double spiral or helix. The two chains run in opposite directions with 10 nucleotides per turn of the helix. The rungs of the bases are pairs of either adenine and thymine (A-T) or cytosine with guanine (C-G). A relatively weak hydrogen bond connects these bases..." (1997, p. 34). [See Figure 1.]

Genes, then, are specific segments of DNA (but not all DNA assumes the form of genes; some resides in extranuclear organelles such as plasmids, and some is non-coding). Chromosomes—which consist of DNA and other material—are macromolecules composed of repeating nucleotides that serve as carriers for genes, with thousands of genes being aligned along each chromosome. [Not all human genes, however, are found on chromosomes; a few reside within mitochondria located in the cytoplasm; see Ridley, 1999, p. 9.] Each chromosome consists of a pair of long (roughly three feet), tightly coiled, double-stranded DNA molecules, with each chromosome possessing one long arm and one short arm separated by a middle "pinch point" known as a centromere.

Every living organism has a specified number of chromosomes in each of its somatic cells. For example, a corn cell has 20, a mouse has 40, a gibbon has 44, and a human has 46. Germ cells in humans, however, have only 23 chromosomes each so that during the union of the male and female gametes, the total will be the standard human number of 46 (23 + 23). As a result, genes are inherited in pairs consisting of one portion from the father and one from the mother, thereby ensuring genetic diversity.

An average gene consists of about 1,000 nucleotides [Figure 2] that normally appear in triplets such as AGC or ATG (see Perloff, 1999, p. 72). While most triplets specify amino acid production, some function as a "stop" command, just as a telegram might contain "stop" to end a sentence. All living organisms—humans, animals, and plants—depend on this code for their existence. Furthermore, each gene is the blueprint the cell uses to assemble a protein that is composed of a long necklace of amino acids (with

each protein consisting of a distinct sequence of those amino acids). [A typical protein contains approximately 300 amino acids (see Macer, 1990, p. 2).]

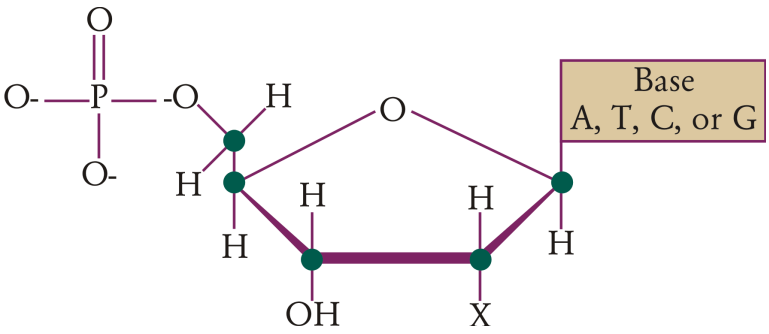

Figure 2 — The structure of a nucleotide. Circles represent carbon atoms. In DNA the sugar is deoxyribose, with a hydrogen atom at position X. In DNA, the base can be A,G,C, or T; in RNA, the base can be A,G.C, or U.

Thanks to the progress that has been made in both genetics and molecular biology, we now possess techniques by which it is possible to determine the exact chemical sequence of any gene from any organism. The **genotype** is the complete set of genes that the organism possesses—something determined at the time of conception for multicellular organisms. It is the same in all cells of an individual organism. The genotype of all cells derived from a particular cell will be the same, unless a mutation occurs. [It is estimated that 90% of all known gene mutations occur in autosomal chromosomes (as opposed to sex chromosomes—see Macer, 1990, p. 4).] For organisms that reproduce sexually, the **genotype** of each new individual will be different since the genes from the two parents are combined. The **phenotype** of an individual is determined by the constant interaction of their genotype and the environment.

The DNA molecule truly is amazing, but it still has certain built-in limits. As evolutionary geneticist Richard Lewontin remarked, "DNA is a dead molecule, among the most nonreactive, chemically inert molecules in the living world" (2000, p. 141). Evolutionist Matt Ridley referred to DNA as "a helpless, passive piece of mathematics, which catalyses no chemical reactions" (1999, p. 17). What is the point of such statements? Evolutionist Jonathan Wells explained:

> "Although molecular biology has demonstrated conclusively that DNA carries the genetic code for the amino acid sequences of proteins, this is not sufficient to specify a whole organism. Combining DNA with all the ingredients necessary for protein synthesis does not make a cell.... Molecular biology has shown that an organism's DNA specifies the building materials. It turns out, however, that **the assembly instructions are largely in other components of the cell**, and that the floor plan has not yet been discovered. So there are clearly other factors involved in heredity and development besides DNA" (1998, pp. 62,64).

Strictly speaking, of course, DNA is not actually a **self**-replicating molecule. As Lewontin explained:

> "DNA has no power to reproduce itself. Rather it is produced out of elementary materials by a complex cellular machinery of proteins.... The newly manufactured DNA is certainly a **copy** of the old, and the dual structure of the DNA molecule provides a complementary template on which the copying process works...[but] no living molecule is self-reproducing" (2000, p. 142, emp. in orig.).

DNA **does** replicate, however. And the process by which it does so is an enormously complex one with many different components that interact to ensure the faithful transfer of genetic information to the next generation. Biochemist Michael Behe noted, "A large number of parts have to work together to that end. In the absence of one or more of a number of the components, DNA replication is either halted completely or significantly compromised, and the cell either dies or becomes quite sick" (1998, p. 185). What, then, is involved in reproducing the DNA molecule so that it can be passed from cell to cell and generation to generation?

Once the structure of DNA finally was elucidated, scientists discovered how, during cell division, the DNA is replicated to produce a genome [the organism's total genetic content] for each new daughter cell. The secret lies in the pairing of the bases—A to T, and G to C. During the replication process, the two

complementary strands of DNA "unzip" down the middle. A new strand then begins to form alongside each of the originals, laying in an A wherever there is an opposing T, a T where there is an A, a G to a C, and a C to a G. The end result is two new double-stranded portions of DNA that, in most instances, are identical to the originals in their base sequences. Ridley described the process by comparing the genetic material to a book.

> "The genome is a very clever book, because in the right conditions it can both photocopy itself and read itself. The photocopying is known as **replication**, and the reading as **translation**. Replication works because of an ingenious property of the four bases: A likes to pair with T, and G with C. So a single strand of DNA can copy itself by assembling a complementary strand with Ts opposite all the As, As opposite all the Ts, Cs opposite all the Gs and Gs opposite all the Cs. In fact, the usual state of DNA is the famous **double helix** of the original strand and its complementary pair intertwined.
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> "To make a copy of the complementary strand therefore brings back the original text. So the sequence ACGT becomes TGCA in the copy, which transcribes back to ACGT in the copy of the copy. This enables DNA to replicate indefinitely, yet still contain the same information.
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> "Translation is a little more complicated. First the text of a gene is **transcribed** into a copy by the same base-pairing process, but this time the copy is made not of DNA but of RNA, a very slightly different chemical.... This RNA copy, called the **messenger RNA**, is then edited....
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> "The messenger is then befriended by a microscopic machine called a **ribosome**, itself made partly of RNA. The ribosome moves along the messenger, translating each three-letter codon in turn into one letter of a different alphabet, an alphabet of twenty different **amino** acids, each brought by a different version of a molecule called **transfer RNA**. Each amino acid is attached to the last to form a chain in the same order as the codons. When the whole message has been translated, the chain of amino acids folds itself up into a distinctive shape that depends on its sequence. It is now known as a **protein**.
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> "Almost everything in the body, from hair to hormones, is either made of proteins or made by them. Every protein is a translated gene" (1999, pp. 6,7,8, emp. in orig.).

Yes, the process described above is utterly amazing. But no less amazing is the fact that it takes place in a DNA fiber that is only two millionths of a millimeter thick (barely visible under an electron microscope). Yet the amount of information contained within it "is so immense in the case of human DNA that it would stretch from the North Pole to the equator if it was typed on paper, using standard letter sizes" (Gitt, 1997, p. 90). Bruce Anderson observed, "If the tightly coiled DNA strands inside a single human adult were unwound and stretched out straight, they would cover the distance to the moon half a million times. Yet when coiled, all the strands could fit inside a teaspoon" (1980, p. 50).

The DNA molecule must be incredibly stable, since the genetic information stored within it may need to function in a living organism for up to a century or more. It also must be completely reproducible so that its complex informational content can be passed successfully from generation to generation. As it turns out, DNA does, in fact, possess each of these traits, and thereby fulfills the necessary and essential criteria of stability and replicability. Are we to be convinced, however, that all of this astonishing complexity, orderliness, design, and function **occurred merely by chance**?

Since the elucidation of the genetic code in the mid-1950s, materialists have suggested that those mythical parents, "Father Time" and "Mother Nature," gave birth to the genetic code via purely chance processes. As Nobel laureate Jacques Monod put it, "Chance alone is the source of every innovation, of all creation in the biosphere.… All forms of life are the product of chance..." (1972, pp. 110,167). Such a view, however, ascribes to "chance" properties that it does not, and cannot, possess. Sproul, Gerstner, and Lindsley addressed this logical fallacy and concluded:

> "Chance is incapable of creating a single molecule, let alone an entire universe. Why not? Chance is no thing. It is not an entity. It has no being, no power, no force. It can effect nothing for it has no causal power within it" (1984, p. 118).

Chance cannot create. And it certainly cannot create something as complex as the genetic code. Furthermore, as evolutionary science writer Matt Ridley observed, "DNA is information, a message written in a code of chemicals" (1999, p. 13). And, as information scientist Werner Gitt correctly noted, "Cod-

ing systems are not created arbitrarily, but they are optimized according to criteria.... Devising a code is a **creative mental** process. Matter can be a **carrier** of codes, but it cannot **generate** codes" (1997, pp. 59,67, emp. added). Whence, then, has come the genetic code? What "creative mental process" imposed the information on it that it contains? In their textbook, *The New Biology,* evolutionists Robert Augros and George Stanciu wrote:

> "What cause is responsible for the origin of the genetic code and directs it to produce animal and plant species? It cannot be matter because of itself matter has no inclination to these forms.... **There must be a cause** apart from matter that is able to shape and direct matter. Is there anything in our experience like this? Yes, there is: our own minds. The statue's form originates in the mind of the artist, who then subsequently shapes matter, in the appropriate way.... **For the same reasons there must be a mind that directs and shapes matter in organic forms**" (1987, p. 191, emp. added).

In speaking of the origin of the genetic code, and the simultaneous appearance of the decoding mechanism that accompanies it, evolutionist Caryl Haskins lamented, "By a pre-Darwinian (or a skeptic of evolution after Darwin) **this puzzle would surely have been interpreted as the most powerful** sort of evidence for special creation" (1971, 59:305, parenthetical comment in orig., emp. added). The late evolutionist Carl Sagan of Cornell University admitted,

> "The number of possible ways of putting nucleotides together in a chromosome is enormous. Thus **a human being is an extraordinarily improbable object**. Most of the $10^{2.4 \times 10^9}$ possible sequences of nucleotides would lead to complete biological malfunction" (1997, 22:967, emp. added).

Sir Francis Crick therefore observed:

> "An honest man, armed with all the knowledge available to us now, could only state that in some sense, **the origin of life appears at the moment to be almost a miracle**, so many are the conditions which would have had to have been satisfied to get it going" (1981, p. 88, emp. added).

United Nations scientist A.E. Wilder-Smith offered the following observation about the origin of the genetic code:

> "The almost unimaginable complexity of the information on the genetic code along with the simplicity of its concept (four letters made of simple chemical molecules), together with its extreme compactness, **imply an inconceivably high intelligence behind** it. Present-day information theory permits no other interpretation of the facts of the genetic code" (1976, pp. 258-259, emp. added).

This is the very point that Gitt made in his 1997 book on information theory when he wrote, "The coding system used for living beings is optimal from an engineering standpoint. This fact strengthens the argument that it was a case of **purposeful design** rather than fortuitous chance" (p. 95, emp. added). British evolutionist Richard Dawkins once observed, "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). I suggest, however, that since the genetic code "appears to be almost a miracle" which "implies an inconceivably high intelligence behind it," then it hardly is "superficial" to believe that it did not happen merely by chance.

Function and Design of the Genetic Code

Faithful, accurate cellular division is critically important, of course, because without it life could not continue. In his presidential address to the British Association for the Advancement of Science, William Bateson made this startling admission: "Descent used to be described in terms of blood. Truer notions of genetic physiology are given by the Hebrew expression 'seed.' If we say he is 'of the seed of Abraham,' we feel something of the **permanence and indestructibility** of that germ which can be divided and scattered among nations, but remains recognizable in type and characteristic after 4,000 years" (1914, emp. in orig.). Seventy-five years later, not much had changed. Suzuki and Knudtson commented, for example:

> "Yet long before the concept of the "gene" crystallized in human consciousness early in this century, human beings felt compelled to search for ways to make sense of at least the most visible evidence of biological inheritance that surrounded them. For they could not help noticing the recurring pattern of reproduction in the natural world by which every form of life seemed to generate new life—'according to its own kind.'

> The keen-eyed agriculturalists among them could not have missed the similarity between successive generations of livestock and crops. Nor was it possible to ignore the sometimes uncanny resemblances between members of one's own immediate family or ancestral lineage" (1989, p. 32).

Suzuki and Knudtson went on to suggest, however, that these poor humans lived in a state of "scientific innocence," and thus could be excused for not knowing any better. But is it necessarily a state of "scientific innocence" to rely on empirical observations and common sense? John Gribbin, himself an evolutionist, has admitted that "...once a fertilized, single human cell begins to develop, the original plans are **faithfully copied** each time the cell divides (a process called mitosis) so that every one of the thousand million million cells in my body, and in yours, contains a **perfect replica** of the original plans for the whole body" (1981, p. 193, parenthetical comment in orig., emp. added). Wilder-Smith noted:

> "Nobel laureate F.H. Crick has said that if one were to translate the coded information on one human cell into book form, one would require one thousand volumes each of five hundred pages to do so. And yet the mechanism of a cell can copy faithfully at cell division all this information of one thousand volumes each of five hundred pages in just twenty minutes" (1976, p. 258, emp. added).

Sparrows produce nothing but sparrows and human beings produce nothing but human beings because all organisms faithfully reproduce copies of their own genetic code. Dr. Bateson spoke of the **permanence and indestructibility** of the "seed." Dr. Gribbin said the code is copied **faithfully**. Suzuki and Knudtson commented on the **recurring pattern of reproduction**. It matters little what terms these evolutionists use; their point is still clear—all living things reproduce "after their kind."

However, while it is important to recognize that although "faithful reproduction" at the cellular level is essential, life could not sustain itself without the existence and continuation of the extremely intricate genetic code contained within each cell. Scientific studies have shown that the hereditary information contained in the code found within the nucleus of the living cell is universal in nature. Regardless of their respective views on origins, all scientists acknowledge this. Evolutionist Richard Dawkins observed, "The genetic code is universal.... The complete word-for-word universality of the genetic dictionary is, for the taxonomist, too much of a good thing" (1986, p. 270). Darrel Kautz agreed when he wrote, "It is recognized by molecular biologists that the genetic code is universal, irrespective of how different living things are in their external appearances" (1988, p. 44). Or, as Matt Ridley put it in his 1999 book, *Genome*,

> "Wherever you go in the world, whatever animal, plant, bug or blob you look at, if it is alive, it will use the same dictionary and know the same code. **All life is one**. The genetic code, barring a few tiny local aberrations, mostly for unexplained reasons in the ciliate protozoa, is the same in every creature. We all use exactly the same language.
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> **"This means—and religious people might find this a useful argument—that there was only one creation, one single event when life was born**.... The unity of life is an empirical fact" (pp. 21-22, emp. added).

It is the genetic code which ensures that living things reproduce faithfully "after their kind," exactly as the principles of genetics state that they should. Such faithful reproduction, of course, is due both to the immense complexity and the intricate design of that code. It is doubtful that anyone cognizant of the facts would speak of the "simple" genetic code. A.G. Cairns-Smith has explained why.

> "Every organism has in it a store of what is called **genetic information**.... I will refer to an organism's genetic information store as its **Library**.... Where is the Library in such a multicellular organism? The answer is everywhere. With a few exceptions, every cell in a multicellular organism has a complete set of all the books in the Library. As such an organism grows, its cells multiply and in the process the complete central Library gets copied again and again.... The human Library has 46 of these cord-like books in it. They are called chromosomes. They are not all of the same size, but an average one has the equivalent of about 20,000 pages.... Man's Library, for example, consists of a set of construction and service manuals that run to the equivalent of about a million book-pages together" (1985, pp. 9,10, emp. in orig.).

Wilder-Smith concurred with such an assessment when he wrote,

> "Now, when we are confronted with the genetic code, we are astounded at once at its simplicity, complexity and the mass of information contained in it. One cannot avoid being awed at the sheer density of in-

> formation contained in such a miniaturized space. When one considers that the entire chemical information required to construct a man, elephant, frog, or an orchid was compressed into two minuscule reproductive cells, one can only be astounded. **Only a sub-human could not be astounded**. The almost inconceivably complex information needed to synthesize a man, plant, or a crocodile from air, sunlight, organic substances, carbon dioxide and minerals is contained in these two tiny cells. If one were to request an engineer to accomplish this feat of information miniaturization, one would be considered fit for the psychiatric line" (1976, pp. 257-259, emp. in orig.).

It is no less amazing to learn that even what some would call "simple" cells (e.g., bacteria) have extremely large and complex "libraries" of genetic information stored within them. For example, the bacterium *Escherichia coli*, which is by no means the "simplest" cell known, is a tiny rod only a thousandth of a millimeter across and about twice as long, yet "it is an indication of the sheer complexity of *E. coli* that its Library runs to a thousand page-equivalent" (Cairns-Smith, p. 11).

In the section he authored on the topic of "life" for the *Encyclopaedia Britannica*, Carl Sagan observed that a single human being is composed of what he referred to as an "ambulatory collection of 10^{14} cells" (1997, 22:965). He then noted, "The information content of a simple cell has been established as around 10^{12} bits, comparable to about a hundred million pages of the *Encyclopaedia* Britannica" (22:966). Dr. Sagan estimated that if a person were to count every letter in every word in every book of the world's largest library (approximately 10 million volumes), the total number of letters would be 10^{12}, which suggests that **the "simple cell" contains the information equivalent of the world's largest library** (1974, 10:894)! Evolutionist Paul Ferrigno admitted, "The complexity of Millennium domes, Eiffel towers and 'Ferris wheels' are likely just pale reflections of life at the heart of the cell" (2000, p. 366). Evolutionist Richard Dawkins acknowledged that the cell's nucleus "contains a digitally coded database larger, in information content, than all 30 volumes of the *Encyclopaedia Britannica* put together. And this figure is for **each** cell, not all the cells of a body put together" (1986, pp. 17-18, emp. in orig.).

A human body is composed of over 250 different kinds of cells (red blood cells, white blood cells, muscle cells, fat cells, nerve cells, etc.—Baldi, 2001, p. 147), totaling approximately 100 trillion cells in an average adult (Fukuyama, 2002, p. 58). These cells come in a variety of sizes and shapes, with different functions and life expectancies. For example, some cells (e.g., male spermatozoa) are so small that 20,000 would fit inside a capital "O" from a standard typewriter, each being only 0.05 mm long. Some cells, placed end-to-end, would make only one inch if 6,000 were assembled together. Yet all the cells of the human body, if set end-to-end, would encircle the Earth over 200 times. Even the largest cell of the human body, the female ovum, is unbelievably small, being only 0.01 of an inch in diameter.

Cells have three major components. First, each cell is composed of a cell membrane that encloses the organism. Second, inside the cell is a three-dimensional cytoplasm—a watery matrix containing specialized organelles. Third, within the cytoplasm is the nucleus, which contains most of the genetic material, and which serves as the control center of the cell.

The lipoprotein cell membrane (lipids/proteins/lipids—known as a bilipid membrane) is approximately 0.06-0.08 of a micrometer thick, yet allows selective transport into, and out of, the cell. Evolutionist Ernest Borek has observed, "The membrane recognizes with its uncanny molecular memory the hundreds of compounds swimming around it and permits or denies passage according to the cell's requirements" (1973, p. 5). Inside the cytoplasm, there are over 20 different chemical reactions occurring at any one time, with each cell containing five major components for: (1) communication; (2) waste disposal; (3) nutrition; (4) repair; and (5) reproduction. Within this watery matrix there are such organelles as the mitochondria (over 1,000 per cell in many instances) that provide the cell with its energy. The endoplasmic reticulum is a transport system that carries materials from one part of the cell to the other. Ribosomes are miniature protein-producing factories. Golgi bodies store the proteins manufactured by the ribosomes. Lysozomes within the cytoplasm function as garbage disposal units. Vacuoles aid in intracellular cleaning processes.

While all of these microscopic organelles point to a non-chance origin, the truly amazing intricate complexity of a cell is observed within the nucleus, for it is within the nucleus that the genetic code is to be found. The nucleus is the control center of the cell, and is separated from the cytoplasm by a nuclear

membrane. Within the nucleus is the genetic machinery of the cell (chromosomes and genes containing deoxyribonucleic acid—DNA). The DNA is a supermolecule that carries the coded information for the replication of the cell. It has been estimated that if the DNA from a single human cell were removed from the nucleus and unraveled from its spiral configuration, it would be approximately six feet long and would contain over 3 billion base pairs. It also has been suggested that if all the DNA in an adult human were placed end-to-end, it would reach to the Sun and back (186 million miles) 400 times. If transcribed into English, the chemical code (deoxyribonucleic acid—DNA) in the human genome (i.e, in a spermatozoon or ovum) would fill a 300-volume set of encyclopedias of approximately 2,000 pages each (Baldi, 2001, p. 21).

Yet just as amazing is the fact that all the genetic information needed to reproduce the entire human population (around six billion people) could be placed into a space of about one-eighth of a square inch. In comparing the amount of information contained in the DNA molecule with a much larger computer microchip, evolutionist Irvin Block remarked, "We marvel at the feats of memory and transcription accomplished by computer microchips, but these are gargantuan compared to the protein granules of deoxyribonucleic acid, DNA" (1980, p. 52).

It also should be noted that the DNA molecule does something that we as humans have yet to accomplish: it stores coded information in a chemical format and then uses a biologic agent (RNA) to decode and activate it. As Darrel Kautz stated, "Human technology has not yet advanced to the point of storing information chemically as it is in the DNA molecule" (1988, p. 45, emp. in orig.). The intricate and complex nature of the DNA molecule—combined with the staggering amount of chemically coded information that it contains—speaks unerringly to the fact that this "supermolecule" simply could not have come into existence due to blind chance and random natural forces operating through eons of time, as evolutionists have claimed. This is not an adequate explanation for the inherent complexity of the DNA molecule. Does coded information happen by chance? And could the decoding system (RNA and ribosomes) just happen by chance as well? Hardly!

It does not take much convincing, beyond facts such as these, to see that the genetic code is characterized by orderliness, complexity, and adeptness in function. The order and complexity themselves are nothing short of phenomenal. But the **function** of this code is perhaps its most impressive feature, as Wilder-Smith explained when he suggested that the coded information

> "...may be compared to a book or to a video or audiotape, with an extra factor coded into it enabling the genetic information, under certain environmental conditions, to read itself and then to execute the information it reads. It resembles, that is, a hypothetical architect's plan of a house, which plan not only contains the information on how to build the house, but which can, when thrown into the garden, build entirely of its own initiative the house all on its own without the need for contractors or any other outside building agents.... Thus, it is fair to say that the **technology** exhibited by the genetic code is orders of magnitude higher than any technology man has, until now, developed. What is its secret? The secret lies in its ability to store and to execute incredible magnitudes of conceptual information in the ultimate molecular miniaturization of the information storage and retrieval system of the nucleotides and their sequences" (1987, p. 73, emp. in orig.).

This "ability to store and to execute incredible magnitudes of conceptual information" is where DNA comes into play. Wilder-Smith concluded, "The information stored on the DNA molecule is that which controls totally, as far as we at present know, by its interaction with its environment, the development of all biological organisms" (p. 73). E.H. Andrews summarized how this can be true:

> "The way the DNA code works is this. The DNA molecule is like a template or pattern for the making of other molecules called 'proteins....' These proteins then control the growth and activity of the cell which, in turn, controls the growth and activity of the whole organism" (1978, p. 28).

Thus, the DNA contains the information that allows proteins to be manufactured, and the proteins control cell growth and function, which ultimately are responsible for each organism. The genetic code, as found within the DNA molecule, is vital to life as we know it. And it is likely that many people have not stopped to consider the exact terminology with which the genetic code is described in the scientific literature. Lester and Bohlin observed:

> "The DNA in living cells contains coded information. It is not surprising that so many of the terms used in describing DNA and its functions are language terms. We speak of the genetic **code**. DNA is **transcribed** into RNA. RNA is **translated** into protein.... Such designations are not simply convenient or just anthropomorphisms. They accurately describe the situation" (1984, pp. 85-86, emp. in orig.).

What may we conclude regarding the infinitely complex genetic code found within the DNA in each cell? Sir Fred Hoyle concluded that the notion that the code's complexity could be arrived at by chance is "nonsense of a high order" (1981a, p. 527). Thaxton, Bradley, and Olsen, in their classic text on the origin of life, addressed the implications of the genetic code found within the DNA molecule.

> "We know that in numerous cases certain effects always have intelligent causes, such as dictionaries, sculptures, machines and paintings. We reason by analogy that similar effects have intelligent causes. For example, after looking up to see 'BUY FORD' spelled out in smoke across the sky we infer the presence of a skywriter even if we heard or saw no airplane. We would similarly conclude the presence of intelligent activity were we to come upon an elephant-shaped topiary in a cedar forest.
>
> "In like manner an intelligible communication via radio signal from some distant galaxy would be widely hailed as evidence of an intelligent source. Why then doesn't the message sequence on the DNA molecule also constitute prima facie evidence for an intelligent source? After all, DNA information is not just analogous to a message sequence such as Morse code, it is such a message sequence....
>
> "We believe that if this question is considered, it will be seen that most often it is answered in the negative simply because it is thought to be inappropriate to bring a Creator into science" (1984, pp. 211-212, emp. in orig.).

The complexity and intricacy of the DNA molecule—combined with the staggering amount of chemically coded information it contains—speak unerringly to the fact that this "supermolecule" simply could not have happened by blind chance. As Andrews observed,

> "It is not possible for a code, of any kind, to arise by chance or accident.... A code is the work of an intelligent mind. Even the cleverest dog or chimpanzee could not work out a code of any kind. It is obvious then that chance cannot do it.... This could no more have been the work of chance or accident than could the 'Moonlight Sonata' be played by mice running up and down the keyboard of my piano! Codes do not arise from chaos" (pp. 28-29).

Indeed, codes do not arise from chaos. As Richard Dawkins (quoted earlier) correctly remarked, "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). But it hardly is "superficial" to suggest that the obvious alternative to chance is an intelligent designer.

My opponent in this debate chooses to believe that "DNA and heredity" (as he put it) provide proof of organic evolution. But that is merely the inference he, personally, has chosen to draw. John Rennie, writing as the editor of *Scientific American*, once admitted that "the historical nature of macroevolutionary study involves **inference** from fossils and DNA **rather than direct observation**" (2002, 287[1]:80, emp. added). The **evidence** is the same for both evolutionists and non-evolutionists. The **inferences** drawn from that evidence, however, are not. Can an unbiased person—after having read the above material on the design and function of the DNA molecule—reasonably infer that the mind-boggling complexity, orderliness, and purpose of this intriguingly intricate and amazingly accurate biochemical code **just happened merely by chance**? I will leave you, the reader, to answer that question for yourself.

EVOLUTIONIST'S THIRD ARGUMENT: THE FOSSIL RECORD

I must candidly admit that I was as surprised as I was shocked when I saw "the fossil record" included in my opponent's list of alleged evolutionary proofs. Why so? Whereas it **used** to be the case that evolutionists went to the fossil record in an attempt to substantiate their theory, that rarely is the case today. As long ago as 1981 British evolutionist Mark Ridley authored an article defending the concept of evolution as a "scientific fact," yet quickly admitted what, even at that time, had come to be common knowledge among those involved in the evolution controversy. He wrote, "No real evolutionist, whether gradualist or punctuationist, uses the fossil record as evidence in favour of the theory of evolution as opposed to

special creation" (1981, 90:831). [On a lighter note, I hope you will not think ill of me for asking, in view of Dr. Ridley's assessment, if my opponent in this debate would consider himself to be a "real evolutionist"?]

However, since my opponent chose to include "the fossil record" among his alleged proofs for the theory of macroevolution, I will be happy to discuss what has been called "the record of the rocks." The fact that fossils occur, and that they represent the environments in which they once lived, is not under dispute. It is the **interpretation** placed on those fossils by evolutionists that non-evolutionists call into question. And for good reason! In his book, *Bones of Contention*, evolutionist Roger Lewin asked in regard to the famous Piltdown fraud:

> "How is it that trained men, the greatest experts of their day, could look at a set of modern human bones —the cranial fragments—and 'see' a clear simian signature in them; and 'see' in an ape's jaw the unmistakable signs of humanity? The answers, inevitably, have to do with the scientists' expectations and their effects on the interpretation of data.... Data are just as often molded to fit preferred conclusions. And the interesting question then becomes 'What shapes the preference of an individual or group of researchers?' not 'What is the truth?'" (1987, pp. 61,68).

Philip Johnson commented in a similar vein in his book, *Darwin on Trial*, when he wrote, "The Darwinist approach has consistently been to find some supporting fossil evidence, claim it as 'proof' for evolution, and then ignore all the difficulties" (1991, p. 84). As you will see in the discussion that follows, that assessment is, unfortunately, all too accurate.

For example, the methodology of the evolutionist in interpreting both the location and the importance of various fossils within the geological record is widely recognized as relying upon circular reasoning. The process begins with the **assumption** that life has progressed from the simple to the complex (i.e., evolution is true). On that basis, then, the fossils are arranged in order from the simple to the complex. "Voilà!," the evolutionist says, "The sequence of fossils goes from the simple to the complex. This supports our original prediction that the fossil record should show life becoming more complex through time, and thus the fossil record proves evolution true." The end result is that an assumption (which, by definition, is both unproved and unprovable) is used to "prove" evolutionary theory. This unmistakable logical fallacy has not escaped the attention of even evolutionary scholars. Evolutionist R.R. West observed,

> "Contrary to what most scientists write, the fossil record does not support the Darwinian theory of evolution because it is this theory (there are several) which we use to interpret the fossil record. By doing so, we are guilty of circular reasoning if we then say the fossil record supports this theory" (1968, p. 216, parenthetical comment in orig.).

Such circular reasoning, of course, cannot (and must not) be accepted as a valid argument for evolution. The point to be stressed is that the actual facts of the fossil record must be considered, without recourse to evolutionary-imposed "successions" and/or concepts of long ages. It is obvious (since my opponent in this debate considered "the fossil record" as a proof of evolution) that the fossils are very much a part of the evolutionists' theory of origins. But this certainly does not mean that evolutionists have exclusive rights to the fossil record. Nor does it mean that the inferences they draw from part or all of the fossil record are correct.

The first step must be to separate **scientific facts** from **philosophical presuppositions**. The second step, then, must be to **make decisions based on those facts** (rather than on any presuppositions). The basic question to be asked is this: **Does the fossil record support the theory of macroevolution?** In order to establish neo-Darwinian evolution, its proponents must be able to show intermediate or transitional forms between animals and plants in the major taxonomic subdivisions. This system, first devised by the Swedish biologist Carolus Linnaeus, classifies organisms at several different levels, beginning with the broadest (kingdom), and progressively narrowing through phylum, class, order, family, genus, species, and variety. Evolutionists propose a general sequence at the phylum level beginning with single-celled organisms (e.g., bacteria), and then progressing to "simple" multicellular organisms (e.g., sponges), to mollusks (e.g., scallops), to arthropods (e.g., crabs), and then to chordates (e.g., man). On a more detailed level (say, by classes of animals), the sequence may begin with cartilaginous fishes (e.g., sharks), and then progress to

bony fishes, to amphibians (e.g., frogs), to reptiles (e.g., crocodiles), and then to mammals (e.g., man). In fact, most biology textbooks present an "evolutionary tree of life" that shows these very sequences. Surely such dramatic changes should be manifest in the fossil record.

Truth be told, Charles Darwin himself postulated that there should be "innumerable transitional links" in the fossil record. In the tenth chapter of *The Origin of Species* (titled, "On the Imperfection of the Geological Record"), Darwin argued that, due to the process of natural selection, "the number of intermediate varieties, which have formerly existed, [must] be truly enormous." However, he went on to admit:

> "Geology assuredly does not reveal any such finely graduated organic chain; and this, perhaps, is the most obvious and serious objection which can be argued against this theory. The explanation lies, I believe, in the extreme imperfection of the geological record" (1956, pp. 292-293).

This was indeed a problem for Darwin's theory, and is still a problem for the modern version of neo-Darwinian evolution. After all, is it not a bit ridiculous to expect people to accept a scientific theory as truth when its advocates have to explain why some of the critical evidence does not even exist? It would be like a prosecuting attorney trying a murder case, and saying in his opening speech, "We know that the defendant is guilty of murder, although we cannot find a motive, the weapon, the body, or any witnesses."

It is true, of course, that the fossil record might be imperfect at times, since some potential fossil-containing layers at certain levels in some localities may have been removed or disturbed by erosive or tectonic activities. But Darwin suggested another reason for the imperfection of the fossil record—insufficient searching. In 1859, most fossil collecting had been done in Europe and the United States. However, after more than 150 years of additional paleontological work, Darwin's defense no longer can be upheld. Evolutionary geologist T.N. George of Great Britain correctly stated, "There is no need to apologise any longer for the poverty of the fossil record. In some ways it has become almost unmanageably rich and discovery is outpacing integration" (1960, p. 1). Today, museums worldwide have more fossils than their staffs can successfully integrate into their public collections. If there is a problem with the fossil record in regard to evolution, it definitely is **not** a lack of fossils!

So what, exactly, does an examination of the fossil record actually indicate in regard to evolutionary theory? To answer that question, I would like to examine what the evolutionists themselves have to say on this matter. For example, in a widely heralded defense of the alleged "factual nature" of organic evolution, the July 29, 2002, issue of *U.S. News & World Report* devoted its front cover—and a lengthy accompanying article titled "The New Reality of Evolution" by staff writer Thomas Hayden—to an in-depth examination of current evolutionary theory. In his article, Hayden correctly noted that Darwin held that new species evolve slowly, "the result of countless small changes over many generations" (p. 44). Hayden also correctly noted, however, that "many creatures still appear quite suddenly in the fossil record." He went on to admit that the first animals appearing in the fossil record are "complex animals, including worms, mollusks, and shrimplike arthropods" that "show up some 545 million years ago." "Paleontologists," Hayden continued, "have searched far and wide for fossil evidence of gradual progress toward these advanced creatures but have come up empty" (pp. 44-45). He then quoted paleontologist Whitey Hagadorn of Amherst College, who sheepishly confessed, "Paleontologists have the best eyes in the world. If we can't find the fossils, sometimes you have to think that they just weren't there" (p. 45).

While at first glance the average reader might view this as an amazing, first-of-a-kind admission of defeat, history shows otherwise. The fact of the matter is that this statement—made in 2002—was little more than a dim echo of an identical admission that had been made more than half a century earlier by the eminent evolutionary paleontologist of Harvard, George Gaylord Simpson, who wrote,

> "This **regular absence of transitional forms** is not confined to mammals, but is an almost universal phenomenon, as has long been noted by paleontologists. It is true of almost all orders of all classes of animals, both vertebrate and invertebrate. *A fortiori*, it is also true of the classes, and of the major animal phyla, and it is apparently also true of analogous categories of plants" (1944, p. 105, emp. added).

But that was not all that Dr. Simpson had to say on the matter. Five years later, he confessed,

> "Possibility for such dispute exists because transitions between major grades of organization are seldom

> well recorded by fossils. There is in this respect a tendency toward systematic deficiency in the record of the history of life. **It is thus possible to claim that such transitions are not recorded because they did not exist**…" (1949, p. 231, emp. added).

Non-evolutionists—who adhere to the concept that scientific theories should be based upon the actual **presence** of evidence, rather than on the **absence** of evidence—have long taken exactly such a stance: **transitional forms are not recorded because they did not exist!**

Until fairly recently, an examination of the Precambrian strata of the geologic timetable showed no undisputed evidence of multicellular fossil forms, while the Cambrian layer (the next layer in succession) exhibited a sudden "explosion" of life forms. In years gone by, this was a serious and fundamental problem in evolutionary theory. Today evolutionists suggest that they have found, in the Precambrian era, multicellular animals that had neither shells nor skeletons. Labeled the "Ediacaran fossil complex," these finds include animals resembling jellyfishes, segmented worms, and possible relatives of corals, according to evolutionists. But even with these new finds, the serious, fundamental problem for evolutionists still remains. Geneticist John Klotz explained why.

> "All of the animal phyla are represented in the Cambrian period except two minor soft-bodied phyla (which may have been present without leaving any fossil evidence), and the chordates. Even the chordates may have been present, since an object which looks like a fish has been discovered in Cambrian rock. It is hardly conceivable that all these forms should have originated in this period; and yet there is no evidence for the existence of many of them prior to the Cambrian period" (1972, pp. 193-194).

Since Dr. Klotz's book was published, the chordates have, in fact, been found in Cambrian rocks (see Repetski, 1978). The problem of the "missing ancestors" in Precambrian rocks is as severe as it ever was. As one science text commented,

> "Even theoretically, to make the vast biological leap from primitive organisms to the Cambrian fauna poses enormous problems. A remarkable series of transformations is required to change a single-celled protozoan into a complex animal such as a lobster, crab, or shrimp. The new life-forms appearing in the Cambrian were not simply a cluster of similar cells; they were complex, fully formed animals with many specialized types of cells.... The new Cambrian animals represented an astonishing leap to a higher level of specialization, organization, and integration" (American Scientific Affiliation, 1986, pp. 35, 37).

We are being asked by evolutionists to believe that from such "ancestors" as those found in the Ediacaran complex, ***all of the major animal phyla*** **"evolved" in the time period represented by a jump between the Precambrian and the Cambrian periods**. Such is not only impossible, but also unreasonable.

Writing in *Science News* under the title of "When Earth Tipped, Life Went Wild," Richard Monastersky remarked,

> "Before the Cambrian period, almost all life was microscopic, except for some enigmatic soft-bodied organisms. At the start of the Cambrian, about 544 million years ago, animals burst forth in a rash of evolutionary activity never since equaled. Ocean creatures acquired the ability to grow hard shells, and a broad range of new body plans emerged within the geologically short span of 10 million years. Paleontologists have proposed many theories to explain this revolution but have agreed on none" (1997, 152:52).

Stefan Bengtson, of the Institute of Paleontology, Uppsala University, Sweden, suggested,

> "If any event in life's history resembles man's creation myths, it is this sudden diversification of marine life when multicellular organisms took over as the dominant actors in ecology and evolution. Baffling (and embarrassing) to Darwin, this event still dazzles us and stands as a major biological revolution on a par with the invention of self-replication and the origin of the eukaryotic cell. **The animal phyla emerged out of the Precambrian mists with most of the attributes of their modern descendants**" (1990, 345:765, parenthetical comment in orig., emp. added).

Evolutionist Richard Dawkins of Oxford University wrote,

> "The Cambrian strata of rocks, vintage about 600 million years [evolutionists are now dating the beginning of the Cambrian at about 530 million years], are the oldest in which we find most of the major invertebrate groups. **And we find many of them already in an advanced state of evolution, the very first time they appear. It is as though they were just planted there, without any evolutionary history.** Needless to

> say, this appearance of sudden planting has delighted creationists" (1986, p. 229, bracketed comment in orig.).

Indeed it has. As Stephen J. Gould observed, "Even the most cautious opinion holds that 500 million subsequent years of opportunity have not expanded the Cambrian range, achieved in just five million years. **The Cambrian explosion was the most remarkable and puzzling event in the history of life**" (1994, 271:86, emp. added). Or, as Andrew Knoll had admitted three years earlier, "We now know that the Ediacaran radiation was indeed abrupt and that the geologic floor to the animal fossil record is both real and sharp" (1991, 265:64). This explosion of life that is found in the fossil record all over the world is a serious stumbling block for evolutionists. Perhaps this is what Michael Denton had in mind when he wrote in his book, *Evolution: A Theory in Crisis*,

> "It is still, as it was in Darwin's day, overwhelmingly true that the first representatives of all the major classes of organisms known to biology are already highly characteristic of their class when they make their initial appearance in the fossil record. This phenomenon is particularly obvious in the case of the invertebrate fossil record. **At its first appearance in the ancient paleozoic seas, invertebrate life was already divided into practically all the major groups with which we are familiar today**.... Robert Barnes summed up the current situation: '...the fossil record tells us almost nothing about the evolutionary origin of phyla and classes. Intermediate forms are non-existent, undiscovered, or not recognized'" (1985, pp. 162-163, emp. added).

The "sudden explosion of life" that I mentioned above has been verified throughout the fossil record. But there is more to it than that. Evolutionist Nile Eldredge correctly noted, "We have been looking at the fossil record as a general test of the notion that life has evolved: **to falsify that general idea, we would have to show that forms of life we consider more advanced appear earlier than the simpler forms**" (1982, p. 46).

With Dr. Eldredge's statement in mind that to falsify evolution, "**we would have to show that forms of life we consider more advanced appear earlier than the simpler forms**," I would like to ask you to consider the lowly trilobite, which is an extinct marine arthropod that once inhabited ocean bottoms. The trilobite is so important in the evolutionary scheme of things that it has been designated as an "index fossil" for the Cambrian period (450-500 million years ago, according to the manner in which evolutionists date such things). Trilobites ranged in size from a fraction of an inch to two feet in length. Their segmented bodies were divided into a head, an abdomen, and a tail, with the head sporting compound eyes and antennae. Despite this amazing level of organization, many evolutionists consider trilobites a very primitive sort of animal.

However, I hardly can think of any example of a form of life we consider (to use Dr. Eldredge's words) "more advanced" in certain respects than the trilobite. In fact, one part of this creature in particular poses a tremendous problem for evolutionary theory—its eye. Each trilobite eye possessed a large lens made out of a mineral called calcite. This means that the lens was not flexible, and thus could not adjust for focusing like the lens in our eyes. To compensate for this, the trilobite lens incorporated no less than four complex optical principles in a system known as an "optical doublet," perhaps making it one of the most-sophisticated visual systems known in the biological world. This is amazing for an animal that supposedly died out millions of years before "advanced" eyes like ours first appeared.

A number of years ago, a professional scientific journal, *The Sciences* (which is the official organ of the New York Academy of Sciences), published an article titled "Nature's Most Perfect Eye." But, surprisingly, it was not an article on the eye of the **human**; rather, it was an article on the eye of the **trilobite**! Why so? As it turns out, the trilobite (which, remember, is a fossil from the Cambrian period!) possessed, to quote from *Science News*, "the most sophisticated eye lenses ever produced by nature" (Shawver, 1974, 105:72).

Why is this the case? Riccardo Levi-Setti, a professor at the University of Chicago, one of the world's experts on the trilobites, and the author of the classic scientific text that bears their name (*Trilobites*), put it like this:

> "In fact, this optical doublet is a device so typically associated with human invention that its discovery in trilobites comes as something of a shock. The realization that trilobites developed and used such devices

> half a billion years ago makes the shock even greater. And a final discovery—that the refracting interface between the two lens elements in a trilobite's eye was designed in accordance with optical constructions worked out by Descartes and Huygens in the mid-seventeenth century—borders on sheer science fiction.... **The design of the trilobite's eye lens could well qualify for a patent disclosure**" (1993, pp. 54,57, emp. added).

Dr. Eldredge candidly admitted that, to falsify evolution, "we would have to show that forms of life we considered more advanced appear earlier than the simpler forms." **Exactly!** That task has now been completed. Trilobites **are** far more advanced, and **do** appear much earlier, than numerous "simpler" forms. And that is something from the fossil record that evolution cannot begin to explain.

The Fossil Record of Human Evolution

Let's be blunt about one thing. Of all the branches to be found on that infamous "evolutionary tree of life," the one leading to man should be the best documented. After all, as the most recent evolutionary arrival, pre-human fossils supposedly would have been exposed to natural decay processes for the shortest length of time, and thus should be better preserved and easier to find than any others. [Consider, for example, how many dinosaur fossils we possess, and those animals were supposed to have existed over a hundred million years before man!] In addition, since hominid fossils are of the greatest interest to man (because they are supposed to represent his past), it is safe to say that more people have been searching for them longer than for any other type of fossils. If there are any real transitional forms anywhere in the world, they should be documented most abundantly in the line leading from the first primate to modern man. Certainly, the fossils in this field have received more publicity than in any other. But exactly what does the human fossil record reveal? What is its central message?

The public, of course, generally has no idea just how scarce, and how fragmentary (literally!), the "evidence" for human evolution actually is. Furthermore, it is practically impossible to determine which "family tree" one should accept. Richard Leakey (of the famed fossil-hunting family in Africa) has proposed one. His late mother, Mary Leakey, proposed another. Donald Johanson, while president of the Institute of Human Origins in Berkeley, California, proposed yet another. And Meave Leakey (Richard's wife) has proposed still another. At an annual meeting of the American Association for the Advancement of Science several years ago, anthropologists from all over the world descended on New York City to view hominid fossils exhibited by the American Museum of Natural History. Reporting on this exhibit, *Science News* had this to say:

> "One sometimes wonders whether orangutans, chimps and gorillas ever sit around the tree, contemplating which is the closest relative of man. (And would they want to be?) Maybe they even chuckle at human scientists' machinations as they race to draw the definitive map of evolution on earth. If placed on top of one another, all these competing versions of our evolutionary highways would make the Los Angeles freeway system look like County Road 41 in Elkhart, Indiana" (see "Whose Ape Is It, Anyway?,"1984, p. 361, parenthetical comment in orig.).

How, in light of such admissions, can evolutionary scientists possibly defend the idea of ape/human evolution as a "scientifically proven fact"? This is not a case where science is acting in a "self-correcting" manner. Quite the opposite is true, in fact. In this instance, scientists are looking at the exact same fossil finds and drawing entirely different conclusions about almost all of them!

The primate family (hominidae) supposedly consists of two commonly accepted genera: *Australopithecus* and *Homo*. While it is impossible to present **any** scenario of human evolution upon which even the evolutionists themselves would agree, currently the alleged scenario (gleaned from the evolutionists' own writings) might appear something like this:

> *Aegyptopithecus zeuxis* (28 million years ago) ➔ *Dryopithecus africanus* (20 million) ➔ *Ramapithecus brevirostris* (12-15 million) ➔ *Orrorin tugenensis* (6 million) ➔ *Ardipithecus ramidus* (5.8-4.4 million) ➔ *Kenyanthropus platyops* (3.8 million years) ➔ *Australopithecus anamensis* (3.5 million) ➔ A*ustralopithecus afarensis* (3.4 million) ➔ *Homo habilis* (1.5 million) ➔ *Homo erectus* (2-0.4 million) ➔ *Homo sapiens* (0.3 million-present).

Here, however, is what is wrong with all of this. *Aegyptopithecus zeuxis* has been called by Richard Leakey "the first ape to emerge from the Old World monkey stock" (1978, p. 52). No controversy there; the animal is admittedly nothing more than an ape. *Dryopithecus africanus* is (according to Leakey) "the stock from which all modern apes evolved" (p. 56). But, as evolutionists David Pilbeam and Elwyn Simons have pointed out, *Dryopithecus* already was "too committed to ape-dom" to be the progenitor of man (1971, p. 23). No controversy there; the animal is admittedly an ape. What about *Ramapithecus*? Thanks to additional work by Pilbeam, we now know that *Ramapithecus* was not a hominid at all, but merely another ape (1982, 295: 232). No controversy there; the animal is admittedly an ape. What, then, shall we say of these three "ancestors" that form the tap root of man's family tree? We simply will say the same thing evolutionists have said: all three were nothing but apes.

The 13 fossil fragments that form *Orrorin tugenensis* (broken femurs, bits of lower jaw, and several teeth) were found in the Tugen Hills of Kenya in the fall of 2000 by Martin Pickford and Brigitte Senut of France, and have been controversial ever since. If *Orrorin* were considered to be a human ancestor, it would predate other candidates by around 2 million years. Pickford and Senut, however, in an even more drastic scenario, have suggested that **all the australopithecines**—even those considered to be our direct ancestors—should be relegated to a dead-end side branch in favor of *Orrorin*. Yet paleontologist David Begun of the University of Toronto has stated that scientists can't tell whether *Orrorin* was "on the line to humans, on the line to chimps, a common ancestor to both, or just an extinct side branch" (2001).

In 1994, Tim White and his coworkers described a new species known as *Australopithecus ramidus* (renamed a year later as *Ardipithecus ramidus*), which was dated at 4.4 million years. The August 23, 1999 issue of *Time* contained a feature article, "Up from the Apes," about the creature. When first found (and while still considered an australopithecine), morphologically this was the earliest, most ape-like australopithecine yet discovered, and therefore appeared to be a good candidate for the most distant common ancestor of the hominids. Dr. White eventually admitted, however, that *A. ramidus* no longer could be considered as a missing link because it possessed too many "chimp-like features." A year later, Meave Leakey and colleagues described the 3.5-4.2 million-year-old *Australopithecus anamensis*, a taxon that bears striking similarities to *Ardipithecus* (an admitted chimp) and *Pan* (the actual genus of the chimpanzees). In 1997, researchers discovered another *Ardipithecus—A. ramidus kadabba*—which was dated at 5.8-5.2 million years old. [The original *Ardipithecus ramidu*s then was renamed *A. ramidus ramidus*.] Once again, *Time* ran a cover story on this alleged "missing link" (in its July 23, 2001 issue). What was it that convinced evolutionists that *kadabba* walked upright and was on the road to becoming man? **A single toe bone!**

Then, in the March 22, 2001 issue of *Nature*, Meave Leakey and her co-authors announced the discovery of *Kenyanthropus* platyops ("flat-faced-man of Kenya"). The authors described their finds as "a well-preserved temporal bone, two partial maxillae, isolated teeth, and most importantly a largely complete, **although distorted**, cranium" (410:433, emp. added). Leakey placed a tremendous amount of importance on the flatness of the facial features of this find, due to the widely acknowledged fact that more modern creatures supposedly possessed an admittedly flatter facial structure than their older, more ape-like alleged ancestors. This is no small problem, however, because creatures younger than *K. platyops*, and therefore closer to *Homo sapiens*, have much more pronounced, ape-like facial features. *K. platyops* was dated at 3.5-3.8 million years, and yet has a much flatter face than any other hominid that old. Thus, the evolutionary scenario seems to be moving in the wrong direction. Some have argued that *K. platyops* belongs more properly in the genus *Australopithecus*.

Australopithecus afarensis was discovered by Donald Johanson in 1974 at Hadar, Ethiopia. Dr. Johanson contends that this creature (nicknamed "Lucy") is the direct ancestor of man (see Johanson, 1981). Numerous evolutionists strongly disagree. Lord Solly Zuckerman, the famous British anatomist, published his views in his book, *Beyond the Ivory Tower*. He studied the australopithecines for more than 15 years and concluded that if man descended from an apelike ancestor, he did so without leaving a single trace in the fossil record (1970, p. 64). "But," someone might say, "Zuckerman's work was done before Lucy was discovered." True, but that misses the point. Zuckerman's research—which established conclusively that the australopithecines were nothing but knuckle-walking apes—was performed on fossils **younger** (i.e., closer to man) than Lucy! If more recent finds are nothing but apes, how could an **older** specimen be "more hu-

man"? Charles Oxnard, while at the University of Chicago, reported his multivariate computer analysis, which documented that the australopithecines were nothing but knuckle-walking apes (1975, pp. 389-395). Then, in the April 1979 issue of *National Geographic*, Mary Leakey reported finding footprints—dated even older than Lucy at 3.6-3.8 million years—that she admitted were "remarkably similar to those of modern man" (p. 446). If Lucy gave rise to humans, then how could humans have existed more than 500,000 years before her in order to make such footprints?

You might be asking yourself why this charade was allowed to go on so long. The answer—woven around power, fame, and money—can be found in Johanson's own words.

> "There is no such thing as a total lack of bias. I have it; everybody has it. The fossil hunter in the field has it…. In everybody who is looking for hominids there is a strong urge to learn more about where the human line started. If you are working back at around three million, as I was, that is very seductive, because you begin to get an idea that that is where *Homo* did start. You begin straining your eyes to find *Homo* traits in fossils of that age…. Logical, maybe, but also biased. **I was trying to jam evidence of dates into a pattern that would support conclusions about fossils which, on closer inspection, the fossils themselves would not sustain**" (Johanson and Edey, 1981, pp. 257,258, emp. added).

Dr. Johanson went on to admit, "It is hard for me now to admit how tangled in that thicket I was. But the insidious thing about bias is that it does make one deaf to the cries of other evidence" (p. 277). Questions now are being raised as to whether or not *afarensis* is more primitive than *africanus*, or whether they are one and the same. Some evolutionists have pointed to Lucy's chimp-like features, and have questioned whether this creature ever really walked uprightly. Finally, in the March 1996 issue of *National Geographic*, Donald Johanson himself admitted, "Lucy has recently been dethroned" (189[3]:117). Dr. Johanson's (and Lucy's) fifteen minutes of fame are over. As evolutionist Lee Berger declared, "One might say we are kicking Lucy out of the family tree" (as quoted in Shreeve, 1996). [For extensive discussions and refutations of *Australopithecus afarensis*, see: Gish, 1995, pp. 241-262; and Lubenow, 1992, pp. 45-58.] Isn't it fascinating to see how often the "hominid family tree" must be pruned?

What of *Homo habilis*? J.T. Robinson and David Pilbeam have long argued that *H. habilis* is the same as *A. africanus*. Louis Leakey (Richard's father) even stated: "I submit that morphologically it is almost impossible to regard *Homo habilis* as representing a stage between *Australopithecus africanus* and *Homo erectus*" (1966, 209:1280-1281). Dr. Leakey later reported the contemporaneous existence of *Australopithecus*, *Homo habilis*, and *H. erectus* fossils at Olduvai Gorge (see Mary Leakey, 1971, 3:272), which would make it impossible for one to be leading up to the other, as Lubenow explained when he wrote:

> "When a creationist emphasizes that according to evolution, descendants can't be living as contemporaries with their ancestors, the evolutionist declares in a rather surprised tone, "Why, that's like saying that a parent has to die just because a child is born!" Many times I have seen audiences apparently satisfied with that analogy. But it is a very false one. In evolution, one species (or a portion of it) allegedly turns into a second, better-adapted species through mutation and natural selection. However, in the context of human reproduction, I do not turn into my children; I continue on as a totally independent entity. Furthermore, in evolution, a certain portion of a species turns into a more advanced species because that portion of the species allegedly possesses certain favorable mutations which the rest of the species does not possess. Thus the newer, more advanced group comes into direct competition with the older unchanged group and eventually eliminates it through death…. The analogy used by evolutionists is without logic, and the problem of contemporaneousness remains....
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> "This incontrovertible fact of the fossil record effectively falsifies the concept that *Homo erectus* evolved into *Homo sapiens* and that *Homo erectus* is our evolutionary ancestor. In reality, it falsifies the entire concept of human evolution" (1992, pp. 121,127,129, parenthetical item and emp. in orig.).

And even more startling was Mary Leakey's discovery of the remains of a circular stone hut at the bottom of Bed I at Olduvai Gorge—**beneath** fossils of *H. habilis* in Bed II! Evolutionists have long attributed the deliberate manufacture of shelter only to *Homo sapiens,* yet Dr. Leakey discovered the australopithecines and *H. habilis* together with manufactured housing. As Duane Gish asked:

> "If *Australopithecus, Homo habilis,* and *Homo erectus* existed contemporaneously, how could one have been ancestral to another? And how could any of these creatures be ancestral to Man, when Man's arti-

> facts are found at a lower stratigraphic level, directly underneath, and thus earlier in time to these supposed ancestors of Man?" (1995, p. 271).

And what about *Homo erectus*? Examine a copy of the November 1985 issue of *National Geographic* (pp. 576-577) and see if you can detect any differences between the pictures of *Homo erectus* and *Homo sapiens*. The fact is, there are no recognizable differences. As the late Ernst Mayr, famed evolutionary taxonomist of Harvard remarked, "The *Homo erectus* stage is characterized by a body skeleton which, so far as we know, does not differ from that of modern man in any essential point" (1965, p. 632).

The fossil evidence for evolution (human or otherwise) simply is not there. Apes always have been apes, and humans always have been humans. Evolutionists certainly find themselves in an embarrassing position since they can find neither the transitional forms their theory demands nor the mechanism to explain how the evolutionary process supposedly occurred.

EVOLUTIONIST'S FOURTH/FIFTH ARGUMENTS: SIMILARITIES AMONG ORGANISMS

The fourth and fifth items in the list of ten "proofs of evolution" that my challenger in this debate offered had to do with similarities (such as DNA and enzyme pathways) among various organisms. This represents what is known as "the defense from homology"—a time-worn (and oft'-answered) argument that long ago was laid to rest. However, since my opponent has seen fit to raise the issue again here, I will respond as follows.

Through the years as scientists have worked in fields like anatomy, biochemistry, cytology, embryology, physiology, etc., they frequently have had opportunities to compare one organism to another. At times, basic similarities have arisen between, or among, these organisms. When making comparisons of parts of organisms, scientists commonly speak of **homologous** structures, suggesting that these particular structures go through similar stages of development, have similar attachments, etc. Evolutionists believe that if similarity can be shown between organisms through such things as anatomy, blood chemistry, protein and DNA biochemistry, etc., then evolutionary kinship can be established. In fact, Charles Darwin himself considered the argument from homology as one of the greatest single proofs of organic evolution, and stated as much in *The Origin of Species* when he wrote, "We have seen that the members of the same class, independently of their habits of life, resemble each other in the general plan of their organization.... Is it not powerfully suggestive of true relationship, of inheritance from a common ancestor?" (1962, pp. 434-435). Strausburg and Weimer, in their *General Biology* text, suggested, "The greater the similarity of structure, the closer the relationship, and, wherever close relationship is found, a common ancestry is indicated" (1947, p. 629).

That statement was made in 1947. Decades later, the same kind of thinking still was commonplace among many evolutionists. For example, Michael Denton, in *Evolution: A Theory in Crisis*, showed why such thinking is so prevalent among certain of his fellow evolutionists when he observed that "without underlying homologous resemblance in the fundamental design of dissimilar organisms and organ systems, then evolution would have nothing to explain and comparative anatomy nothing to contribute to evolutionary theory" (1985, p. 145).

At first glance, descent from a common ancestor appears to be a "logical" argument because it seems to make so much sense. After all, isn't that how we explain such similarities as brothers and sisters looking more alike than, say, cousins? They have parents closer in common. And evolutionists have an impressive array of data at their disposal. They are quick to point out such things as the fact that the wing of the bat, the forefoot of the turtle, the forefoot of the frog, and the arm of the man all have the same general structure. They also note, correctly, that the forefoot of the dog, the flipper of the whale, and the hand of the man contain essentially the same bones and muscles. In more recent times, this argument even has been carried to the molecular level as scientists began to compare similarities in blood groups, cytochrome C composition, enzymatic pathways, cellular DNA, and a myriad of other molecular entities. The conclusion we are supposed to draw, of course, is that evolution must be true because we can trace our ancestral lineages to a common ancestor who lived millions of years ago. That, in fact, is exactly what the late evolutionist of Cornell University, Carl Sagan, suggested when he wrote, "The inner workings of terrestrial organisms—from

microbes to men—are so similar in their biochemical details as to make it highly likely that all organisms on the Earth have evolved from a single instance of the origin of life" (see Shklovskii and Sagan, 1966, p. 183).

What is the non-evolutionist's response to all of this? Do similarities exist? And if so, is the evolutionist's explanation the correct, or the only, explanation that fits the facts of the case?

First, let me immediately note that non-evolutionists do **not** deny that such similarities **do** exist. However, it is here that we can learn an extremely valuable lesson in the evolution controversy. That lesson is this: **rarely is it the *data* that are in dispute—it is the *interpretation* placed on the data that is in dispute**. In the cases of basic similarities, whether at the anatomical or biochemical level, denying that such similarities exist serves no good purpose. Evolutionists and non-evolutionists have access to the same data. The evolutionist, however, looks at the data and says that similarity is proof of **common ancestry**. The non-evolutionist, on the other hand, examines the exact same data and suggests that similarity is evidence of **origin due to a common design**. In essence, a stalemate exists. Both sides have an answer to the data at hand. And in many instances, either explanation might appear legitimate.

However, the evolutionists' argument works only if certain portions of the data on homology are presented. If **all** the available data are allowed full exposure, then the evidence from homology fails miserably. Many years ago, evolutionist T.H. Morgan of Columbia University candidly admitted what many evolutionists would rather not become common knowledge: "If, then, it can be established beyond dispute that similarity or even identity of the same character in different species is not always to be interpreted that both have arisen from a common ancestor, the whole argument from comparative anatomy seems to tumble in ruins" (1923, p. 246). Or, as veterinarian R.L. Wysong observed, "If the law of similarity can be used to show evolutionary relationships, then dissimilarities can be used to show a lack of relationship" (1976, pp. 393-394). True enough.

Ferenco Kiss, as dean of the medical faculties at the University of Budapest, once stated that "...it is necessary for the evolutionists—in order to maintain their theory—to collect only the similarities and to neglect the numerous differences" (1949, p. 3). Evolution is an entire cosmogony, and as such, must explain **both** similarities **and** differences within its framework. It is not the **similarities** that present the problem; it is the numerous **differences**. As Sir Alistair Hardy, former professor of zoology at Oxford University, wrote, "The concept of homology is fundamental to what we are talking about when we speak of evolution, yet in truth we cannot explain it all in terms of present-day biological theory" (1965, p. 211).

What did Dr. Hardy mean when he said, more than forty years ago, that "we cannot explain it all in terms of present-day biological theory"? He meant simply this: only when evolutionists are allowed to "pick and choose" similarities that fit their theory can the argument from homology be made to work. When evolutionists are forced to use **all** the data—including those documenting dissimilarity—the argument from homology utterly fails.

His point is well taken—even today. The fact that a sort of "picking and choosing" method exists when it comes to comparative arguments, and that that method has been used to support evolutionary theory, has been exposed, as Lester and Bohlin have pointed out:

> "Another problem is that from the raw data alone, not one single phylogeny emerges, but several. The one that agrees most closely with the traditional phylogeny is **assumed** to be the most 'correct.' This hardly demonstrates the independent confirmation of evolutionary relationships. The combining of several phylogenies from different proteins combines not only strengths but also weaknesses" (1984, p. 173, emp. in orig.).

Evolutionist Vincent Demoulin likewise pointed out the fallacy inherent in this kind of "pick and choose" game when he noted that "the composite evolutionary tree encompasses all the weaknesses of the individual trees" (1979). That is to say, adding up **all** the available data from homology studies makes for an weaker argument than would be present when examining just a few of the data on any given topic.

Homology and the "Rest of the Story"

But please do not take my word on this subject. Evolutionist Michael Denton stated quite succinctly just how valuable all this "proof" from similarity studies really is when he wrote:

> "Invariably, as biological knowledge has grown, common geneology as an explanation for similarity has tended to grow ever more tenuous. Clearly, such a trend carried to the extreme would hold calamitous consequences for evolution, as homologous resemblance is the very *raison d'être* of evolution theory. Without the phenomenon of homology—the modification of similar structures to different ends—there would be little need for a theory of descent with modification....
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> "Like so much of the other circumstantial 'evidence' for evolution, that drawn from homology is not convincing because it entails too many anomalies, too many counter-instances, far too many phenomena which simply do not fit easily into the orthodox picture. The failure of homology to substantiate evolutionary claims has not been as widely publicized as have the problems in paleontology.
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> "The discussion in the past three chapters indicates that the facts of comparative anatomy, and the pattern of nature they reveal, provide nothing like the overwhelming testimony to the Darwinian model of evolution that is often claimed. Simpson's claim that 'the facts simply do not make sense unless evolution is true,' or Dobzhansky's that 'nothing in biology makes sense except in the light of evolution' are simply not true if by the term evolution we mean a gradual process of biological change directed by natural selection....
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> "In the last analysis the facts of comparative anatomy provide no evidence for evolution in the way conceived by Darwin, and even if we were to construe with the eye of faith some 'evidence' in the pattern of diversity for the Darwinian model of evolution, this could only be seen, at best, as indirect or circumstantial....
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> "...the same hierarchic pattern which may be explained in terms of a theory of common descent, also, by its very nature, implies the existence of deep divisions in the order of nature. The same facts of comparative anatomy which proclaim unity also proclaim division; while resemblance suggests evolution, division, especially where it appears profound, is counter-evidence against the whole notion of transmutation" (1985, pp. 154-155).

What did Denton mean when he said that the "evidence" for evolution from homology studies "entails too many anomalies, too many counter-instances, far too many phenomena which simply do not fit easily into the orthodox picture"? The answer to that lies in an examination of the data that have become available during the past several years. For example, Wysong provided an extensive list of such data, among which are the following examples:

1. The octopus eye, pig heart, Pekingese dog's face, milk of the ass, and the pronator quadratus muscle of the Japanese salamander are all very similar to analogous human structures. Do these similarities show evolutionary relationships?
2. The weight of the brain in proportion to body weight is greater in the dwarf monkey of South America, the marmoset, than in man. Since this proportion is used to show relationship between primates and man, is the marmoset, therefore, more evolved than man?
3. The plague bacterium (*Pasteurella pestis* [now known as *Yersinia pestis*]) afflicts only man and rodent. Does this similarity show close relationship?
4. Plant nettle stings contain acetylcholine, 5-hydroxytryptamine and histamine. These chemicals are also found in man. Are man and plant closely related?
5. The root nodules of certain leguminous plants and the crustacean, Daphnia, contain hemoglobin, the blood pigment found in man. Are these organisms closely related to man?
6. If certain specific gravity tests are run on the blood of various animals, the frog and snake are found to be more similar to man than the monkey is to man.
7. If the concentration of red blood cells in animals is compared (millions per cubic millimeter of blood), man is more similar to frogs, fish, and birds than he is to sheep.
8. Since bones are often used to show relationships, bone chemistry should be useful in this regard. If the calcium/phosphorus ratio is plotted against bone carbonate, man proves to be close to the turtle and elephant, the monkey close to the goose, and the dog close to the horse but distant from the cat.
9. The tetrapyrole chemical ring is found in plant chlorophyll, in hemoglobin and other animal respiratory pigments, sporadically as a coloring pigment in molluscan shells, and also in the feathers of some bird species. How does tetrapyrole similarity speak for relationships (1976, pp. 394-395).

After examining examples such as these, it is easy to understand what Dr. Denton meant when he said that there are too many "anomalies," too many "counter-instances," and "too many phenomena which

simply do not fit easily into the orthodox picture." Other writers (both evolutionists and non-evolutionists) have documented this same problem. Such anomalies have caused evolutionists to search for some way to try to salvage the argument from homology. Certain evolutionary scientists have suggested that evidence now is available that can, in fact, perform such a "salvage operation." Bernard Davis of the Bacterial Physiology Unit at Harvard Medical School wrote:

> "In most of its development evolutionary biology has depended on morphological homologies, both in the fossil record and among living species; but this approach has not revealed the continuum of transition forms between species that Darwin predicted. Moreover, while he expected further research in paleontology to fill in the gaps, we no longer entertain that hope. But now, at least, molecular genetics has provided a direct, radically different kind of evidence for such continuity.... Not only does molecular genetics provide the most convincing evidence for evolutionary continuity, but this evidence should impress a public that is well aware of the power of this science in other areas" (1985, 28:252-253).

Notice two important points in Dr. Davis' statement. First, he admits that the approach from morphological homologies "has not revealed the continuum of transition forms that Darwin predicted." In other words, if you look at the data from morphological homologies (i.e., the kind of data examined above), then the result is a dismal failure for evolutionary theory. The required "continuum" simply does not exist. Second, however, Dr. Davis is optimistic that something more powerful as a proof from homology has been found—evidence from molecular (as opposed to morphological) homology. His optimism centers on the hope that, where "proofs" from morphological homologies have failed, perhaps "proofs" from molecular homologies can succeed. Davis' optimism, however, was short lived.

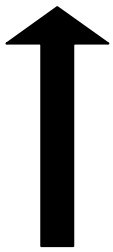
Despite the bright promise that molecular evidences are so strong as to provide almost undeniable proof for evolution, several puzzles have emerged from studies in molecular homologies. For example, in 1981, Colin Patterson (senior paleontologist at the British Museum of Natural History) came to America to speak to several scientific societies. During his various speeches, Dr. Patterson suggested that he had "experienced a shift from evolution as knowledge to evolution as faith." He then presented numerous specific examples documenting the failure of the evolutionary hypothesis of common ancestry. He said that the hypothesis acted as an "anti-theory" and conveyed nothing but "anti-knowledge." Dr. Patterson presented data on amino acid sequences for the alpha hemoglobins of a viper, crocodile, and chicken. Evolutionists "know" (since evolution is assumed to be true) that vipers and crocodiles (two reptiles) should be much more closely related than either is to a bird. But the crocodile and the chicken showed the greatest similarity (17.5% of their amino acids in common) with the viper and the chicken the next most similar (10.5%), and the two reptiles with the **least** similarity (5.6%).

An examination of the amino acids in myoglobin showed that crocodiles and lizards (two reptiles) shared 10.5%, but that a lizard and a chicken (reptile/bird) also shared the same percentage (10.5%). Dr. Patterson then described studies of mitochondrial DNA performed on man and on various primates. He acknowledged that where there should have been a high percentage of similarities, there was a very low percentage. After all his data were presented, Dr. Patterson remarked that "the theory makes a prediction, we've tested it, and the prediction is falsified precisely" (as quoted in Sunderland, 1982).

Homology, DNA, Genes, and Chromosomes

Other molecular studies over the past few years have yielded no better results. For example, within cells of living organisms are found chromosomes that carry the genes responsible for the individual organism's genetic make-up. If there has been a gradual evolution of all creatures—from the simple to the complex, as evolution demands—then the evolutionary scheme would predict that there likewise would be an increase in chromosome number and quality as one moves up the evolutionary scale. Today, however, advanced molecular technology has caused the evolutionary prediction to fall on hard times. Note the following chart comparing the actual chromosome numbers of several organisms with the evolutionary prediction.

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

The chromosome count does not "fit" what one would predict based upon the theory of evolution. Evolutionist Ashley Montagu begrudgingly admitted, "The number of chromosomes does not appear to be associated with the degree of complexity of an organism" (1960, p. 24)—which most assuredly would include the chromosomes, since they are the carriers of the genetic material.

Furthermore, it would make sense that, if humans and chimpanzees (our alleged closest evolutionary ancestor) were 95% genetically the same, then the manner in which they store DNA also would be similar. Yet it is not. DNA, the fundamental blueprint of life, is tightly compacted into chromosomes. All cells that possess a nucleus contain a specific number of chromosomes. Common sense would seem to necessitate that organisms that share a common ancestry would possess the same number of chromosomes. However, chromosome numbers in living organisms vary from 308 in the black mulberry (*Morus nigra*), to 6 in animals such as the mosquito (*Culex pipiens*) or nematode worm (*Caenorhabditis elegans*) [see Sinnot, et al., 1958]. Additionally, complexity does not appear to affect the chromosomal number. The radiolaria (a simple protozoon) has over 800, while humans possess 46. Chimpanzees, on the other hand, have 48 chromosomes. A strict comparison of chromosome numbers would indicate that we are more closely related to the Chinese muntjac (a small deer found in Taiwan's mountainous regions), which also has 46 chromosomes.

This hurdle of differing numbers of chromosomes may appear trivial, but we must remember that chromosomes contain genes, which themselves are composed of DNA spirals. If the blueprint of DNA locked inside the chromosomes codes for only 46 chromosomes, then how can evolution account for the **loss** of two entire chromosomes? The task of DNA is to continually reproduce itself. If we infer that this change in chromosome number occurred through 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 a number of genes, **losing** chromosomes does not make sense physiologically, and almost certainly would prove deadly for new species. No respectable biologist would suggest that by **removing** one chromosome (or more), a new species likely would be produced. To remove even **one** chromosome would potentially remove the DNA codes for millions of vital body factors. Eldon Gardner summed it up as follows: "Chromosome number is probably more constant, however, than any other single morphological characteristic that is available for species identification" (1968, p. 211). To put it another way, humans always have had 46 chromosomes, whereas chimps always have had 48.

Other such "anomalies" abound. Wysong pointed out that human cells contain 7 picograms of DNA/cell, whereas the frog contains more and the African lungfish contains 100 picograms of DNA/cell. According to evolutionary predictions, should the frog and lungfish contain **more** DNA than a man? Or what about amino acid sequences? Cytochrome C, for example, is a coenzyme found in the mitochondria of all aerobic cells and therefore is found in most organisms. As evolutionists have studied amino acid sequences among organisms, they have found many similarities. But what about the many differences? One hears a lot these days about the similarities among organisms in regard to their cytochrome C content, yet numerous dissimilarities exist as well (but rarely are mentioned by evolutionists). Frair and Davis pointed out that 104 amino acids are strung together to build cytochrome C. On the basis of the number of differences in these units, the gray whale has more in common with the duck than with another mammal, the monkey; the bullfrog has more in common with the fruit fly than with the rattlesnake; and the tuna has more in com-

mon with the rabbit than with the dogfish (1983, pp. 45-53). Lester and Bohlin, in their discussion of cytochrome C and many of the dissimilarities associated with it, noted,

> "The most well-known phylogeny is that of cytochrome C, which appears to agree very well with the accepted phylogeny. However, there are exceptions and procedural difficulties of interpretation. There are often large discrepancies between the protein phylogeny and the traditional one. In cytochrome C chickens are more closely related to penguins than to ducks and pigeons, turtles are closer to birds than to snakes (fellow reptiles), and people and monkeys diverge from the mammals before marsupial kangaroos separate from the rest of the mammals" (1984, pp. 172-173, parenthetical item in orig.).

The facts simply do not fit the predictions. And perhaps no one has done a more outstanding job of providing the evidence for that statement than evolutionist Michael Denton. Evolutionists suggest that as one ascends the "tree of life," organisms should become increasingly separated by differences in biochemistry from the "earliest" and most "primitive" organisms. In fact, no evolutionary trend can be observed in the biochemical data—at least none that can be adequately defended. Denton showed that bacteria are as divergent from yeast (69%) as they are from wheat (66%), silkmoths (65%), tuna (65%), pigeons (64%), horses (64%), or humans (65%). There is no gradation from one group to another that would show any kind of evolutionary sequence. Denton's conclusion was that "at a molecular level there is no trace of the evolutionary transition from fish to amphibian to reptile to mammal" (1985, p. 285). He then added, "To those well acquainted with the traditional picture of vertebrate evolution, the result is truly astonishing" (p. 285). Dr. Denton went on to state that "at a molecular level, no organism is 'ancestral' or 'primitive' or 'advanced' compared with its relatives" (p. 290). "Yet," he said, "in the face of this extraordinary discovery the biological community seems content to offer explanations which are no more than apologetic tautologies" (p. 306).

What could be clearer? Homology simply does **not** establish common ancestry. The entire genome of the tiny nematode, *Caenorhabditis elegans*, has been sequenced as a tangential study to the human genome project. Of the 5,000 best-known human genes, 75% have matches in the worm (see "A Tiny Worm Challenges Evolution," n.d.). Does this mean that humans are 75% identical to a nematode worm? Just because living creatures share some genes with humans does not mean there is necessarily a linear ancestry at work. Biologist John Randall admitted this fact 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] 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, emp. added).

Yet textbooks and teachers still continue to proclaim that humans and chimps are 95% genetically identical—while the actual evidence clearly demonstrates vast molecular differences. Elaine Morgan commented on this difference.

> "Considering the very close genetic relationship that has been established by comparison of biochemical properties of blood proteins, protein structure and DNA and immunological responses, **the differences between a man and a chimpanzee are more astonishing than the resemblances**. They include structural differences in the skeleton, the muscles, the skin, and the brain; differences in posture associated with a unique method of locomotion; differences in social organization; and finally the acquisition of speech and tool-using, together with the dramatic increase in intellectual ability which has led scientists to name their own species *Homo sapiens sapiens*—wise wise man. During the period when these remarkable evolutionary changes were taking place, other closely related ape-like species changed only very slowly, and with far less remarkable results. **It is hard to resist the conclusion that something must have happened to the ancestors of *Homo sapiens* which did not happen to the ancestors of gorillas and chimpanzees**" (1989, pp. 17-18, emp. added).

From the genetic material itself (DNA) to the organs that compose the body, gradualistic development is countered by the perpetual discontinuity routinely seen in nature. There is nothing—either in the pro-

posed mechanisms, or resulting from direct observation—to show that Darwin's theory of general evolution is a "fact" of science.

EVOLUTIONIST'S SIXTH ARGUMENT: GENETIC MUTATIONS

The sixth item in the list of ten "proofs of evolution" that my challenger in this debate offered was stated as follows: "the reason new flu virus's [sic.] show up every year." I suspect that most well-read people are familiar with the fact that certain viruses have been known to change over time, thereby frustrating or thwarting our medical efforts to produce vaccines intended to prevent humans or animals from being infected by such viruses. Earlier in this rebuttal, in the section on bacterial antibiotic resistance, I quoted evolutionist Tim Berra who wrote, "Bacteria experience far more mutations because there are so many more individuals and generations. This and the short reproductive cycle allow beneficial mutations to be exploited by natural selection rapidly" (Berra, 1990, p. 54). The same can be said of viruses. And while my evolutionary opponent did not come right out and say so in his one-line "proof" of evolution (that "new flu virus's [sic.] show up every year"), there can be little doubt that the reason he offered up such a scenario has to do with the fact that he wants us to believe that viruses are "selectively adapting" through a mechanism that involves genetic mutations—and that this, in turn, portends great strides for organic evolution. But is this the truth of the matter? No, it is not. Allow me to explain why.

At the end of the nineteenth century, just as Darwin's dogma of natural selection was beginning to fall on hard times (I will have more to say about this as I address the last argument that my evolutionary opponent included in his list of ten "proofs" of evolution), the science of genetics was born. The concepts that had been published in 1865 in a little-known journal by the Moravian monk Gregor Mendel, but which had lain quietly forgotten on dusty library shelves for thirty-five years, were "rediscovered" with an attendant flourish. Some who began to study this fledgling science felt for the first time that they had in their possession the actual mechanism of evolution—genetic mutations. Their suggestion was that species arose by mutations that then somehow were incorporated into the system by natural selection. Today, the alleged mechanism of evolution, therefore, is not merely natural selection, but rather **natural selection plus genetic mutations**. Evolutionist Francis Hitching said in this regard,

> "The theory is that a chance favorable mutation gradually spreads through a population of plants or animals by a process of natural selection of the fittest; and over geological periods of time, a new species emerges. Genetics provides the mechanism that supports Darwin's original insight" (1982, p. 34).

Writing almost twenty years earlier, Ernst Mayr of Harvard wrote in agreement when he said, "The proponents of the synthetic theory maintain that all evolution is due to the accumulation of small genetic changes, guided by natural selection" (1963, p. 586). Simpson and Beck, in their widely used high school biology textbook, *Life: An Introduction to Biology*, concurred by observing that "mutations are the ultimate raw materials for evolution" (1965, p. 430). Evolutionary geneticist Theodosius Dobzhansky commented that "the process of mutation is the only known source of the new materials of genetic variability, and hence of evolution" (1957, p. 385).

Through the years, not much has changed in this regard. In a chapter on the role of mutations in evolution for the 1997 book *Evolution* edited by Mark Ridley, evolutionary biologist Sewall Wright observed,

> "The observed properties of gene mutation—fortuitous in origin, infrequent in occurrence and deleterious when not negligible in effect—seem about as unfavourable as possible for an evolutionary process. Under biparental reproduction, however, **a limited number of mutations which are not too injurious to be carried by the species furnish an almost infinite field of possible variations through which the species may work its way under natural selection**" (see Ridley, 1997, pp. 32-33, emp. added).

In his book, *The Way of the Cell*, evolutionist Franklin M. Harold suggested, "Any alteration in the sequence of DNA, once replicated, is inherited henceforth; that is the chemical basis of mutation, and therefore of much of the genetic variation within populations" (2001, p. 47). Evolutionist Donald Goldsmith noted,

> "During the process of DNA replication, small changes called **mutations** can occur.... Some mutational changes tell the organism to do something additional that proves useful its quest to survive and to repro-

> duce. In that case, provided that the mutation can be passed from ancestors to descendants, the organisms carrying the mutation may come to dominate the local scene, and can eventually produce new types of organisms" (1997, p. 125, emp. in orig.).

In his 2000 volume, *Quantum Evolution: The New Science of Life*, Johnjoe McFadden wrote, "Mutations are therefore the elusive source of the variation that Darwin needed to complete his theory of evolution. They provide the raw material for all evolutionary change" (p. 65). That same year evolutionist Paul Ehrlich penned the following statement in his book, *Human Natures: Genes, Cultures, and the Human Prospect*: "The ultimate source of variation in the DNA—that is, the creation of different kinds of genes—is mutation: the accidental alteration of DNA that changes genes.... In short, genetic variation has its basic source in mutation" (2000, pp. 20-21). Also that same year, the renowned evolutionary geneticist of Stanford University, Luigi Cavalli-Sforza (head of the International Human Genome Diversity Project), remarked in his book, *Genes, Peoples, and Languages*:

> "Evolution also results from the accumulation of new information. In the case of a biological mutation, new information is provided by an error of genetic transmission (i.e., a change in the DNA during its transmission from parent to child).... Natural selection makes it possible to accept the good ones and eliminate the bad ones" (2000, p. 176, parenthetical item in orig.).

Perhaps this would be a good time to ask, "What, exactly, is a mutation?" Simply put, a mutation is an error made when cells copy DNA—usually the loss, insertion, or change of a nucleotide in a DNA molecule (see Wise, 2002, p. 163). As Ariel Roth put it in his book, *Origins*, "A mutation can refer to a variety of genetic changes, such as: a change in a nucleotide base on the DNA chain, an altered gene position, the loss of a gene, duplication of gene, or insertion of a foreign genetic sequence" (1998, p. 85). A.O. Wasserman, in his text, *Biology*, defined a mutation as "a change in the form, qualities, or nature of the offspring from their parent type brought about by a change in the hereditary material from the parents" (1973, p. 803). Geneticist George Burns wrote, "Basically a mutation is a sudden, random alteration in the genotype of an individual. Strictly speaking, it is a change in the genetic material itself..." (1973, pp. 313-314). There are certain other basic facts about genetic mutations that come into play, however, and that need to be mentioned and considered.

Mutations are random. C.H. Waddington, an evolutionary geneticist, once noted, "It remains true to say that we know of no other way other than **random** mutations by which hereditary variation comes into being...." (1962, p. 98, emp. added). Thirty-eight years later, Paul Ehrlich wrote, "A key axiom of modern evolutionary theory is that mutations do not occur in response to the needs of the organism.... Mutations are random" (2000, p. 21). Non-evolutionists concur. Henry Morris, for example, observed, "There is no way to control mutations to make them produce characteristics which might be needed. Natural selection must simply take what comes" (1974, p. 54). In other words, nature is not "selecting" at all. Rather, nature is pressed into accepting whatever appears. The obvious question, then, is: What appears?

Mutations are rare, not common. How often do random mutations occur? Evolutionists themselves frankly and candidly admit what every research biologist knows—mutations occur rarely. Geneticist Francisco J. Ayala of the University of California once remarked, "It is probably fair to estimate the frequency of a majority of mutations in higher organisms between one in ten thousand and one in a million per gene per generation" (1970, p. 3). Lane Lester and Raymond Bohlin, in their book, *The Natural Limits to Biological Change*, commented, "Considering a host of both eukaryotic and prokaryotic organisms, the chances of a single gamete containing a new mutation for a particular gene range from 1/2,000 to 1/1,00,000,000" (1984, p. 59).

Mutations may be good, bad, or neutral. There are, theoretically speaking, at least three types of mutations: good, bad, and neutral. Obviously, the bad mutations (those that cause various diseases such as hemophilia, Duchenne dystrophy, phenylketonuria, galactosemia, etc.) are of no use to evolutionary theory. Neutral mutations likewise are of little use to the evolutionist (see Hitching, 1982, pp. 62-63) because they, then, are dependent on still more mutations in order to be fully expressed and "useful" (in an evolutionary sense). Thus, another obvious question becomes: How often do **good** mutations occur—i.e., "good" in the sense that they can "push evolution forward"?

Good mutations are very, very rare. The late Hermann J. Muller, Nobel laureate in genetics, said, "Accordingly, the great majority of mutations, certainly well over 99%, are harmful in some way, as is to be expected of the effects of accidental occurrences" (1950, 38:35, emp. added). Evolutionary geneticist Theodosius Dobzhansky candidly admitted that favorable mutations amount to less than 1% of all mutations that occur (see Davidheiser, 1969, p. 209). Dr. Dobzhansky even remarked that "most mutants which arise in any organism are more or less **disadvantageous** to their possessors..." (1955, p. 105). Evolutionist C.P. Martin wrote in the *American Scientist*, "Accordingly, mutations are more than just sudden changes in heredity; **they also affect viability, and, to the best of our knowledge, invariably affect it adversely**. Does not this fact show that mutations are really assaults on the organism's central being, its basic capacity to be a living thing?" (1953, p. 102, emp. added).

Almost twenty-five years later, in addressing the rarity of these "good" mutations, one researcher commented, "From the standpoint of population genetics, positive Darwinian selection represents a process whereby advantageous mutants spread through the species. Considering their great importance in evolution, it is perhaps surprising that well-established cases are so scarce" (Kimura, 1976, 138[6]:260). And twenty-five years after that, Harvard's eminent taxonomist, Ernst Mayr, remarked that "...the occurrence of new beneficial mutations is rather rare" (2001, p. 98). Numerous researchers through the years have written in agreement (Winchester, 1951, p. 228; Martin, 1953, p. 100; Ayala, 1968, p. 1436; Morris, 1984, p. 203; Klotz, 1985, p. 181; Margulis and Sagan, 2002, pp. 11-12).

Furthermore, those organisms that ought to show the **most** mutants apparently show the **least**—which is no insignificant problem for the population geneticist. France's preeminent zoologist, Pierre-Paul Grassé ("who is the editor of the 28 volumes of *Traité de Zoologie*, author of numerous original investigations, ex-president of the Academie des Sciences, and whose knowledge of the living world is encyclopedic"—Dobzhansky, 1975, 29:376), lamented,

> "Bacteria, the study of which has formed a great part of the foundation of genetics and molecular biology, are the organisms which, because of their huge numbers, produce the most mutants.... Bacteria, despite their great production of intra-specific varieties, exhibit a great fidelity to their species. The bacillus, *Escherichia coli*, whose mutants have been studied very carefully, is the best example. The reader will agree that it is surprising, to say the least, to want to prove evolution and to discover its mechanisms and then to choose as a material for this study a being which practically stabilized a billion years ago" (1977, p. 87).

Interestingly, the same is true of other species. Consider the lowly fruit fly. "The fruit fly (*Drosophila melanogaster*), the favorite pet insect of the geneticists, whose geographical, biotropical, urban, and rural genotypes are now known inside out, seems not to have changed since the remotest times" (Grassé, p. 130). Dr. Grassé has provided an insightful evaluation, and is absolutely correct in his assessment. We are being asked to believe that organisms that have been in a period of **stasis** (i.e., no change) "somehow" provide the proof of evolution (vast amounts of change). As Roth put it,

> "[T]housands of laboratory experiments with bacteria, plants, and animals witness to the fact that the changes that a species can tolerate have definite limits. There appears to be a tight cohesion of interacting systems that will accept only limited change without inviting disaster. After decades or centuries or decades of experimentation, fruit flies retain their basic body plan as fruit flies, and wool-producing sheep remain basically sheep. Aberrant types tend to be inferior, usually do not survive in nature, and, given a chance, tend to breed back to their original types. Scientists sometimes call this phenomenon genetic inertia (genetic homeostasis)" [1998, pp. 85-86, parenthetical item in orig.].

It bears mentioning here that, as Wise has observed,

> "**Of carefully studied mutations, most have been found to be harmful to organisms, and most of the remainder seem to have neither positive nor negative effec**t. Mutations that are actually beneficial are extraordinarily rare and involve insignificant changes. **Mutations seem to be much more degenerative than constructive**..." (2002, p. 163, emp. added).

Favorable mutations are indeed "extraordinarily rare." It also is a well-known fact that "most mutations are recessive—that is, they will not manifest themselves unless present in both parents. Furthermore, while mutations producing minor changes may survive, **those causing significant modification are especially det-**

rimental and unlikely to persist" (Roth, p. 86, emp. added). Lester and Bohlin also addressed this point when they wrote,

> "Overall, however, mutations would primarily be a constant source of genetic noise and degeneration.... Mutations occur in organisms that are already adapted to their environment. Any large-scale, rapid alteration to the organism will not only be deleterious but most likely lethal" (1984, pp. 171,68).

Good points, those. Mutations in bacteria, to use just one previously mentioned example, may result in antibiotic resistance. But in the end, the resistant microorganisms are still the same species of microorganisms they were **before** the mutations occurred. Alan Hayward was on target when he wrote that

> "...mutations do not appear to bring progressive changes. Genes seem to be built so as to allow changes to occur within certain narrow limits, and to prevent those limits from being crossed. To oversimplify a little: mutations very easily produce new varieties within a species, and might occasionally produce a new (though similar) species, but—despite enormous efforts by experimenters and breeders—**mutations seem unable to produce entirely new forms of life**" (1985, p. 55, parenthetical item in orig., emp. added).

In the end, after mutations have occurred, no macroevolution has taken place. Evolutionary theory requires that mutations occur—in order to add the information needed to push evolution "uphill." But the mutations that we observe generally are neutral (i.e., they do not alter the information or the "message" of the DNA code), or else they go "downhill" (from an informational standpoint), which results in the loss or corruption of information. In addition, the rare "beneficial" mutations that do occur and that do confer some type of survival advantage, still result in the loss of information, and thus are headed in the wrong direction, from an evolutionary vantage point.

But please do not take my word for any of this. Listen instead to evolutionists Lynn Margulis and Dorion Sagan, in their 2002 book, *Acquiring Genomes: A Theory of the Origins of Species*, in which they expressed their strong disagreement with genetic mutations as the alleged mechanism of evolution.

> "We certainly agree that random heritable changes, or gene mutations, occur. We also concur that these random mutations are expressed in the chemistry of the living organism.... The major difference between our view and the standard neodarwinist doctrine today concerns the importance of random mutation in evolution. **We believe random mutation is wildly overemphasized as a source of hereditary variation**. Mutations, genetic changes in living organisms, are inducible; this can be done by X-ray radiation or by addition of mutagenic chemicals to food. Many ways to induce mutations are known but none leads to new organisms. **Mutation accumulation does not lead to new species** or even to new organs or new tissues. If the egg and a batch of sperm of a mammal is subjected to mutation, yes, hereditary changes occur, but as was pointed out very early by Hermann J. Muller (1890-1967), the Nobel prizewinner who showed X-rays to be mutagenic in fruit flies, 99.9 percent of the mutations are deleterious. **Even professional evolutionary biologists are hard put to find mutations, experimentally induced or spontaneous, that lead in a positive way to evolutionary change**" (pp. 11-12, emp. added).

Margulis and Sagan then went on to say,

> "We agree that very few potential offspring ever survive to reproduce and that populations do change through time, and that therefore natural selection is of critical importance to the evolutionary progress. **But this Darwinian claim to explain all of evolution is a popular half-truth whose lack of explicative power is compensated for only by the religious ferocity of its rhetoric**. **Although random mutations influenced the course of evolution, their influence was mainly by loss, alteration, and refinement.... Never, however, did that one mutation make a wing, a fruit, a woody stem, or a claw appear. Mutations, in summary, tend to induce sickness, death, or deficiencies. No evidence in the vast literature of hereditary change shows unambiguous evidence that random mutation itself, even with geographical isolation of populations, leads to speciation**" (pp. 28-29, emp. added).

Adding their combined weight to the testimony of Margulis and Sagan are such eminent evolutionists as the late Pierre-Paul Grassé, who held the position of the Chair of Evolution at the Sorbonne in Paris for over 30 years, and the late Stephen J. Gould of Harvard. Dr. Grassé remarked,

> "The opportune appearance of mutations permitting animals and plants to meet their needs seems hard to believe. Yet the Darwinian theory is even more demanding: a single plant, a single animal would require thousands and thousands of lucky, appropriate events. Thus, miracles would become the rule: events with

> an infinitesimal probability could not fail to occur.... There is no law against day-dreaming, but science must not indulge in it.
>
> "Some contemporary biologists, as soon as they observe a mutation, talk about evolution. They are implicitly supporting the following syllogism: mutations are the only evolutionary variations, all living beings undergo mutations, therefore all living things evolve. This logical scheme, is, however, unacceptable: first, because its major premise is neither obvious nor general; second, because its conclusion does not agree with the facts. No matter how numerous they may be, **mutations do not produce any kind of evolution**" (1977, p. 103, emp. added).

Gould's testimony is no less weighty. In a speech titled, "Is a New and General Theory of Evolution Emerging?," presented at Hobart College on February 14, 1980, Dr. Gould went on record as stating, "A mutation doesn't produce major new raw material. You don't make a new species by mutating the species.... That's a common idea people have; that evolution is due to random mutations. A mutation is not the cause of evolutionary change" (as quoted in Sunderland, 1984, p. 106). Or, as Lester and Bohlin put it,

> "Mutations are mistakes, errors in the precise machinery of DNA replication. Combine this with the rarity and randomness of mutations, and one has a major reason why Neo-Darwinists perceive evolutionary change as being gradual and slow. **Since any specific mutation is rare, and most are deleterious, a mutation that somehow enhances survival is admittedly highly unlikely**..." (1984, p. 67).

Nobel laureate Sir Ernest Chain (credited with purifying penicillin in a way that made it possible to employ it as an antibiotic) wrote in agreement.

> "**To postulate that the development and survival of the fittest is entirely a consequence of chance mutations seems to me a hypothesis based on no evidence and irreconcilable with the facts**. These classical evolutionary theories are a gross oversimplification of an immensely complex and intricate mass of facts, and it amazes me that they are swallowed so uncritically and readily, and for such a long time, by so many scientists without a murmur of protest" (1970, p. 1, emp. added).

Writing in the *Proceedings of the National Academy of Sciences*, D.H. Erwin and J.W. Valentine remarked:

> "Viable mutations with major morphological or physiological effects are exceedingly rare and usually infertile; the chance of two identical rare mutant individuals arising in sufficient propinquity to produce offspring seems too small to consider as a significant evolutionary event" (1984, 81:5482-5483).

"Chances" that are "too small to consider as a significant evolutionary event"? What is **that** all about? It has to do with the mathematical probability of having random mutations account for all we see around us —a probability that is, well, **infinitesimal**. It would require **many** non-harmful mutations to produce the characteristics of just **one** useful structure. The problem is how to get such extremely rare events to occur **simultaneously** in an organism, in order to produce a functional structure that possessed survival value. Evolutionist E.J. Ambrose outlined the problem as follows:

> "The frequency with which a single non-harmful mutation is known to occur is about 1 in 1,000. The probability that two favourable mutations will occur is $1x10^3$ x 10^3, 1 in a million, 1 in a million. Studies of *Drosophila* [fruit fly] have revealed that large numbers of genes are involved in the formation of separate structural elements. There are as many as 30-40 involved in a single wing structure. It is most unlikely that fewer than five genes could ever be involved in the formation of even the simplest new structure previously unknown to the organism. The probability now becomes one in one thousand million million. We already know that mutations in living cells appear once in ten million to once in one hundred thousand million. It is evident that the probability of five favourable mutations occurring within a single life cycle of an organism is effectively zero" (1982, p. 120).

What is the conclusion to be drawn from these facts? George Gaylord Simpson of Harvard admitted that if there was an effective initial breeding population of **100 million** individuals, and if they could produce a new generation **every day**, the likelihood of obtaining good evolutionary results from mutations could be expected only about **once every 274 billion years**! He thus was forced to conclude, "Unless there is an unknown factor tremendously increasing the chance of simultaneous mutations, such a process has played no part whatever in evolution" (1953, p. 96). Little wonder Grassé concluded, "No matter how numerous they may be, **mutations do not produce any kind of evolution**" (1977, p. 103, emp. added).

If evolution does not occur via genetic mutations, how, then, does it occur? Non-evolutionists have been stressing for years that most mutations either are harmful or neutral (neither of which provides the forward thrust required for evolution to occur), and that since mutations are unpredictable random changes in an extremely complex system, any change represents a **mistake**, not an improvement. The practical end result of mutations has been noted time and again by those within the scientific community. The Environmental Mutagenic Society, in a report published in *Science*, warned that "being an error process, mutation consists of all possible changes in the genetic material (excluding recombination and segregation)" and that "most mutations producing effects large enough to be observed are deleterious." Furthermore, the Society stated in its report that "since the vast majority of detectable mutations are deleterious, an artificially increased human mutation rate would be expected to be harmful in proportion to the increase" (Environmental Mutagenic Society, 1975, 187:503-504).

Mutations, represent an undesirable departure from the original. We do not know of mutations that can cause one kind of animal to give rise to another kind of animal, or one kind of plant to give rise to another kind of plant. What we **do** know, and **have** documented, are mutations that damage or destroy what already is present. Mutations are not suggestive of evolution, but instead militate against it.

Returning to the point that my evolutionary opponent raised in regard to "new flu virus's [sic.] showing up every year," I would like to point out that regardless of the fact that viruses (influenza, or any other variety) might have mutated, **the fact remains that they still are exactly the same type of viruses *after* mutating as they were *before* mutating**. No phylogenetic boundary was traversed as a result of the changes in the viral genetic material. This so-called "proof" of evolution, like the bacterial antibiotic resistance that I discussed earlier, turns out to be **not** vertical **macro**evolution but horizontal **micro**evolution (i.e., adaptation). Again, no real "organic evolution" has occurred.

EVOLUTIONIST'S SEVENTH ARGUMENT: THE AGE OF THE EARTH AND ITS GEOLOGICAL HISTORY

The rules set in place to govern this debate, and to which both disputants agreed prior to the beginning of the debate, specifically stated that "arguments regarding the age of the Earth will **not** be a part of this debate due to the limited space available to each disputant, and due to the tangential nature of the age of the Earth in regard to the legitimacy of evolution as a theory of origins." Since both my opponent and I concurred regarding the fact that the age of the Earth was not to be discussed during the debate, I confess to being confused as to why he chose to include that topic in his list of alleged proofs of evolution. But since the rules to which we both agreed specifically eliminated that subject as a topic of discussion, and because it is my intention to adhere to those rules, I will not address my opponent's seventh argument for evolution.

EVOLUTIONIST'S EIGHTH ARGUMENT: VESTIGIAL ORGANS OR STRUCTURES

According to standard dictionaries such as the *American Heritage* or *Merriam Webster*, a "vestige" is "a rudimentary or degenerate, usually nonfunctioning, structure that is the remnant of an organ or part that was fully developed or functioning in a preceding generation or an earlier stage of development; a bodily part or organ that is small and degenerate or imperfectly developed in comparison to one more fully developed in an earlier stage of the individual in a past generation or in a closely related form."

In the original edition of *The Origin of Species*, Charles Darwin cited several cases where animals have diminished or unused parts (1859, pp. 175-179). For instance, the ostrich has wings but cannot fly, and cave fish have eyes but cannot see. Darwin used such examples as evidence that animals have changed over time: ostriches were alleged to have descended from birds with functional wings, while blind cave fish were said to have descended from fish with functional eyes. Later additions to the list of supposedly vestigial organs in animals include such things as the panda's thumb, hip bones in the whale, etc.

It is rare today to find evolutionists who are willing to use vestigial organs as a proof of evolution—for reasons that will become clear as you continue reading. But, every so often an evolutionist sallies forth

in an effort to defend evolution by trotting out supposed vestigial organs or structures. For example, in the November 2004 issue of *National Geographic*, David Quammen authored an article titled, "Was Darwin Wrong?," in which he attempted to defend the theory of organic evolution. To the amazement of many of his evolutionary colleagues (and certainly to non-evolutionists), he offered the following observation:

> "Vestigial characteristics are still another form of morphological evidence, illuminating to contemplate because they show that the living world is full of small, tolerable imperfections. Why do male mammals (including human males) have nipples? Why do some snakes (notably boa constrictors) carry the rudiments of a pelvis and tiny legs buried inside their sleek profiles? Why do certain species of flightless beetles have wings, sealed beneath wing covers that never open? Darwin raised all these questions, and answered them in *The Origin of Species*. Vestigial structures stand as remnants of the evolutionary history of a lineage" (2004, 206[5]:20, parenthetical items in orig.).

What shall we say about such claims? Darwin (and his fellow evolutionists) have witnessed such things as the diminished size of ostrich wings, cave fish with eyes that cannot see, rudimentary legs in certain snakes, hip bones in whales, or thumbs on pandas, and then have assumed (based on evolutionary presuppositions) that such structures serve no obvious or useful purpose. While such structures are (so we are told) of no value to their present-day possessors, they were at some point in the past useful to their evolutionary predecessors. Now, as "evolutionary leftovers," they are viewed as structures that eventually will be weeded out completely through evolutionary processes of natural selection. They thus are designated as "vestigial," and subsequently touted as "proof" of evolution.

But such pronouncements do not tell what the late, well-known newscaster, Paul Harvey, might refer to as "the rest of the story." For example, we know today that the ostrich wing is not vestigial at all, but instead is used in courtship displays, for balance during running, and to shield chicks in a nest from the harmful rays of the Sun. We also know that python legs and whale hip bones provide critical attachment points for certain muscles used in locomotion, reproduction, etc.

And when it comes to the panda's thumb, the story is even more intriguing. In Stephen J. Gould's 1980 book, *The Panda's Thumb*, he went to great lengths to make a case for how poor a structure the thumb is on a panda's "hand." Yet, interestingly, while Dr. Gould was writing about the non-design that he felt was so evident, research was ongoing in regard to the panda's thumb (which Dr. Gould had described as "a somewhat clumsy" appendage that would "win no prize in an engineer's derby"; pp. 20-21). And what, exactly, did that research show? The panda's thumb has now been found to exhibit design for very special functions, as the following information attests.

First, the San Diego Zoo's *Giant Panda Zoobook* states, "In fact, the giant panda is one of the few large animals that can grab things as tightly as a human can" (n.d., p. 6). Second, in 1985 Schaller et al. authored *The Giant Pandas of Wolong*, in which they wrote, "The panda can handle bamboo stems with great precision by holding them as if with forceps in the hairless groove connecting the pad of the first digit and pseudothumb" (n.d., p. 4).

Does the fact that the panda is able to grasp something tightly "with great precision" by using a thumb that is comparable to surgical forceps cause you to think that such a thumb could accurately be described as "a somewhat clumsy" appendage that would "win no prize in an engineer's derby"? I suspect not (me, neither!). Once **all** the data were in, the initial (biased) assessment that Dr. Gould had made of the panda's thumb did not even come close to fitting the available evidence. [Is there a lesson here somewhere?]

Furthermore, when it comes to things like the loss of sight in cave fish, it is quite conceivable (or even likely) that degenerative changes have taken place. For example, genetic mutations are known to have caused the loss of sight in certain cave-dwelling fish (*Astyanax mexicanus*; see Yamamoto and Jeffery, 2000), yet with no subsequent detriment to their survival. **Yet no new speciation occurred due to this mutation**. In fact, *Astyanax mexicanus* can appear in either the "eyed" form or the "eyeless" form. It is refreshing to note that even biological taxonomy plainly supports the fact that the fish with which we began is still a fish. Neither the genus nor the species has changed between the epigean (surface-dwelling) and hypogean (underground) forms.

There also is the principle of **progression** versus **regression** to be considered. Here, information is the key. Evolution demands **pro**gression, and with it there must accompany an increase of **new** information. **Re**gression can be described by the loss or corruption of genetic information. Harvard's Ernst Mayr defined macroevolution as the "evolution above the species level; the evolution of higher taxa and the production of evolutionary novelties, such as new structures" (2001, p. 287). He included in his definition the requirement for the "production of evolutionary novelties, such as new structures." The question then becomes, "**What new structures has the cave fish evolved?**" This is where progression comes to a screeching halt. The cave fish actually falls into the category known as "devolution," which is a category of regression on a downhill slope, where information is being lost—not gained (Wieland, 2001, p. 47). Organic evolution cannot be sustained using examples of "downhill" change. Plus, even if the fish's eyes were considered to be vestigial, they still represent only limited change from the sighted condition (see Frair and Davis, 1983, p. 29). Such vestiges do not support Darwin's claim that lack of use is a driving force in large-scale evolution. The basic tenets needed by evolutionists are not met in this instance, and thus cave fish cannot be touted as a proof of evolutionary theory.

Among *Homo sapiens*, such things as the appendix, hair, wisdom teeth, hair, the coccyx, male nipples, and a number of other organs or structures have been among the items once considered as being remnants of our supposed animal past. The theory of evolution asserts that man has climbed upward out of an animal ancestry to his present plateau of human existence; thus, humans possess within their bodies numerous features that may have served them well during past stages of their brutish backgrounds, but that currently are vestigial. Evolution, however (so we were assured), eventually would eliminate these "useless" structures from our bodies.

Even a quick survey of the scientific literature regarding vestigial organs can produce some fascinating material. For example, under the heading, "Man's Body: A Museum of Evolution," three prominent evolutionists of the past (H.G. Wells, Julian Huxley, and G.P. Wells) penned the following:

> "Our adult human bodies are among the best proofs of Evolution; and the private development of each one of us is an affidavit swearing to the evolutionary history of our race. Wiedersheim, the celebrated German anatomist, enumerated in the body of man no less than one hundred and eighty organs which are vestigial—wholly or almost useless to us, though useful in other species of animals—each one of them a stumbling block to the believer in special creation but an ally to the Evolutionist" (1934, p. 415).

The three evolutionists certainly were correct about Alfred Wiedersheim being a "celebrated German anatomist," as well as about the list of 180 allegedly vestigial human organs that he compiled (see Wiedersheim, 1931). But, as the old saying suggests, "that was then; this is now." With the tremendous advances that have been made in our scientific knowledge over the last hundred years or more, it is difficult to find any "useless" human body part. In fact, it now appears that "vestigial" actually meant simply "of unknown function."

Today the list of supposed vestigial organs in human beings is incredibly short—and getting shorter with each passing day (see Bergman and Howe, 1990). As you consider the following information, I suspect that you will understand why.

Consider, for example, the claim that the human appendix is vestigial. The appendix is a small, worm-shaped tube attached to the cecum at the end of the ascending colon. Various textbooks on anatomy have suggested in the past that it has no function. Evolutionists have contended that it was once part of a much larger cecum in our herbivorous ancestors.

As late as 1997, *Encyclopaedia Britannica* described the appendix in the following manner: "The appendix does not serve any useful purpose as a digestive organ in humans, and it is believed to be gradually disappearing in the human species over evolutionary time" (see *Encyclopaedia Britannica*, "Vestigial Organs," p. 491). We know today, however, that the notion that the human appendix is vestigial is false—and we have known this since the early 1960s when Robert G. Taylor, a specialist in internal medicine, first published the efforts of his research on this topic. He noted, "The function of the thymus and the human appendix are beginning to be understood in the 1960s.... The tonsils and the appendix help us to prevent germs from entering the system" (see Nelson, 1967, pp. 196-197). While all of the appendix's functions may not be fully understood currently, we do know that the appendix has a rich supply of blood,

and that it contains masses of lymphatic tissues in its walls. Today it is difficult to convince a pediatrician to perform an appendectomy on a young child unless absolutely necessary because we recognize that the appendix plays a vital role in our body's immune system, especially in the young.

The importance of this alleged "vestigial organ" was being discussed in medical textbooks as long ago as 1976. As one scientist admitted, "The appendix is not generally credited with significant function; however, current evidence tends to involve it in the immunologic mechanism" (Bockus, 1976, p. 1135). Current, up-to-date medical textbooks generally describe the appendix as a "well-developed lymphoid organ" (Moore, 1992, p. 205) whose "mucosa and submucosa…are dominated by lymphoid nodules" and whose "primary function is as an organ of the lymphatic system" (Martini, 1995, p. 916). The appendix also is believed to boost antibody production in the spleen, and may even play a role in preventing certain types of cancer.

Interestingly, the August 12, 2009, issue of *Journal of Evolutionary Biology* contained an article (written, obviously, by evolutionists) titled, "Comparative Anatomy and Phylogenetic Distribution of the Mammalian Cecal Appendix" (Smith, et al., 2009). The abstract of that article made the following observation: "A recently improved understanding of gut immunity has merged with current thinking in biological and medical science, pointing to an apparent function of the mammalian cecal appendix as a safe-house for symbiotic gut microbes, preserving the flora during times of gastrointestinal infection in societies without modern medicine." This, of course, provides ample evidence of yet another important function of the appendix—documenting once again that it is not vestigial. As the authors of the article conceded, "This function is potentially a selective force for the evolution and maintenance of the appendix, and provides an impetus for reassessment of the evolution of the appendix." "Reassessment" indeed!

Yet, even with this knowledge, the appendix still is mentioned in some evolutionary literature as being vestigial. But this reasoning begs the question: If our ancestors used an appendix in some earlier function, from which ancestral stock did it "devolve"? Neither "old" nor "new" world monkeys possesses an appendix, which leads to the conclusion that they therefore must be more highly evolved than humans. **One cannot help but wonder then, if, according to evolutionary theory, monkeys evolved from humans?** Such reasoning obviously leaves evolutionary theory in utter confusion.

Certain evolutionists also have touted human hair as being vestigial. Is hair strictly vestigial, serving no current worthwhile purpose in human anatomy? Note again the assertions of Wells, Huxley, and Wells.

> "The body-hair of men and women is purely vestigial; it no longer serves to prevent us losing heat. And yet each of these tens of thousands of useless hairs possesses a useless muscle by means of which it can be, quite uselessly, raised. For a furry creature to bristle up its hair when the weather grows cold is useful enough —more air is entangled in its coat, and it loses less heat. In the same circumstances we also erect our futile little hairs; but the resultant goose-flesh condition is of no value whatever—we have performed a vestigial action" (1934, p. 415).

Truth be told, the material in that paragraph is glaringly erroneous. The actual fact of the matter is that human body hair plays a vitally important role in the well-being of the skin. The late Douglas Dewar, a noted scholar and former evolutionist himself, accurately described the importance of hair when he wrote,

> "Each (hair) is embedded in a follicle into which opens the duct of at least one sebaceous gland secreting an oily fluid necessary to keep the skin in good condition. These hairs and the muscles attached to them—the *arrectores pilorum*—have a two-fold function. The muscles, which are situated on the side of the hair toward which it slopes, on contraction diminish the obliquity of the hair follicle and render the hair more erect, and, at the same time compress the sebaceous glands and expel their contents (Cunningham, *Text Book of Anatomy*, 1902, p. 733). The presence of the hair and its movements also prevent the mouth of the follicle from becoming blocked with sebaceous matter. Follicles which have lost their hair sometimes become blocked and this may result in the formation of a sebaceous cyst" (n.d., p. 5).

When a muscle contracts, the hair (working with its associated follicle in the dermis) functions as a sort of lever, thus squeezing nearby sebaceous glands, which then deposit an oily sebum into the upper layer of the epidermis. Marshall and Lazier observed that the sebum keeps the epidermis "soft and pliable

and probably helps to water-proof it" (1946, p. 141). Thus, whenever the phenomenon occurs that we refer to as "goose bumps" or "goose flesh," a person actually is getting (in a very literal fashion!) a lube-job! Human body hair is not a vestige of our animal heritage, but instead performs important functions required for the maintenance of the body.

Certain evolutionists likewise have touted human wisdom teeth as being vestigial. It is undeniable, of course, that some people experience problems with their wisdom teeth (third molars). These teeth sometimes grow improperly, and end up having to be surgically removed. Evolutionists explain this problem by suggesting that the human jaw is getting smaller. As apes (or ape-like creatures), we possessed the proper number of teeth (so we are informed), but now there are too many for our modern jaw. Yet many people have healthy, useful wisdom teeth. The fact that wisdom teeth sometimes can cause problems may be a function of our changing diet. Or perhaps bad wisdom teeth represent a physical weakness, like failing eyesight, or hardening of the arteries. But the fact some people have non-functioning wisdom teeth does not magically negate the fact that many people have wisdom teeth that function quite well. It hardly is good science to point to an organ or structure that does not work in one person (or group of people), and then state that that organ is "vestigial." The fact that wisdom teeth function at all proves beyond any doubt that they are not useless!

Some evolutionists have touted the human coccyx as being vestigial. The coccyx is attached to the lower end of the vertebral column, and consists of three to five (usually four) fused vertebrae. Evolutionists believe the coccyx is basically a rudimentary tail. However, the coccyx serves a very real function as an anchor for muscles and tendons that plan important roles in bowel movements, the birth process, leg movement, and other functions in the lower torso. The coccyx is by no means vestigial.

Some evolutionists have touted human male nipples as being vestigial. Today, however, we know that male nipples have important functions. For example, the nipples release perspiration, whose purpose it is to cool the chest area during times of exertion. They also play an important role in secondary sex characteristics by serving as one of the male erogenous zones (Sloand, 1998; Masters and Johnson, 1966). Male nipples contain an extremely large supply of nervous tissue, and therefore are quite sensitive to touch, much like the sexual organs (Sarhadi et al., 1996, 1997; Sykes, 1969; Wuringer et. al., 1998; Robinson and Short, 1977; Kapdi and Parekh, 1983). Studies have shown that many males find nipple stimulation to be critical in achieving a normal sexual response. Brietzke stated that every expert he interviewed "stressed that" the male nipple is "a central erotic area for men" (1995, p. 13). One study of sexual stimulation reviewed by two Stanford psychologists found that erotic male nipple response appeared in more than half of the cases they studied (Katchadourian and Lunde, 1972, p. 73).

One of the key clues to discovering various functions of the male nipples was the quantity of nervous tissue with which they are endowed (much like one of the key factors in discovering certain functions of the human appendix was the fact that it is endowed with a rich blood supply). Vestigial structures would have no need of either large quantities of nervous tissue or rich blood supplies. The scientific research that is now available regarding male nipples belies the suggestion that they are in any way vestigial, because they most certainly are not.

However, it appears that the endless evolutionary quest for a true human vestigial organ will continue to plague us—at least for a while longer. A review of the medical literature documents one of the last alleged vestigial organs in humans as being the vomeronasal organ (also referred to as Jacobson's organ), which is found on the nasal septae. In the 1970s, this particular organ was regarded as vestigial, but recently was discovered to be more common than previously reported. A study conducted in 1998 found that physicians, using routine nasal examinations, identified the vomeronasal organ in only 16% of the people examined. Yet when nasal endoscopes were employed in the same procedure, the figure jumped to 76% (Gaafar, et al., 1998). Additionally there is now impressive evidence substantiating the fact that this organ has a specific sensory function in humans (Gaafar, et al., 1998; Berliner, et al., 1996).

Another point that needs to be considered is this: Were it ever the case that man at one time possessed 180 vestigial organs (organs that once **were** functional), then in the distant past he would have had **more functioning organs than he now has**. In the past, he would have been developing the organs that he presently possesses, plus he would have had the 180 functional-but-now-vestigial organs. **So the**

farther back we go in time, the more complex the organism becomes (see Wysong, 1976, pp. 398-399). Rather an interesting evolutionary twist, wouldn't you say?

Those evolutionists who keep up with the scientific literature rarely mention vestigial organs any longer for two reasons. First, there is a conspicuous lack of evidence of the transitional stages between functioning organs and useless organs. Second, examples of so-called useless organs or structures would prove **devolution**, not **evolution**, since organisms in the past would have had more organs and structures in their bodies than modern-day organisms do. Evolution is (supposedly) the rise of new, different, and functioning organs, not the wasting away of already-present, complex organs. Thus, non-evolutionists are forced to ask: Where are all of the nascent [new] organs? R.L. Wysong was thus prompted to remark,

> "Not too long ago man was imputed to have 180 vestiges. Organs like the appendix, tonsils, thymus, pineal gland and thyroid gland were on the list. Today, all former vestigial organs are known to have some function during the life of the individual. If the organ has any function at any time, it cannot be called rudimentary or vestigial.... As man's knowledge has increased the list of vestigial organs decreased. So what really was vestigial? Was it not man's rudimentary knowledge of the intricacies of the body?" (p. 397).

Dr. Wysong's point is well made. It turns out that evolutionists actually have used the word "vestigial," not to mean "useless," but instead to say, in reality, "we are ignorant of what this organ's function is at this point in time." Today, we now know there are **no** vestigial organs in the human body. And as our ignorance wanes, so, ironically, does the number of alleged vestigial organs.

Yet in spite of the continually accumulating evidence against so-called vestigial organs, and the fading importance of alleged vestigial structures, some ill-informed evolutionists continue to rely upon the argument from vestigial organs as proof of evolution. My opponent's use of this tired, strained, and irrelevant argument is proof aplenty of that. No structure can be considered vestigial unless it can be proved that it has absolutely no function in any phase of its possessor's existence. I strongly suspect that in the years ahead, the vestigial-organ argument itself will become but another vestige in the relic heap of evolutionary history.

EVOLUTIONIST'S NINTH ARGUMENT: ENDOGENOUS RETROVIRUSES

Because it is likely that some of the people who read the documents produced during this debate may not be scientists, or may not be familiar with some of the technical jargon employed in specific fields of science (such as microbiology in general, or virology specifically), I would like to offer a few general comments and definitions before offering a rebuttal to my opponent's suggestion that endogenous retroviruses provide proof for organic evolution.

As I pointed out in the section of this rebuttal on DNA and heredity, in most organisms the primary genetic material is DNA. However, some viruses (primarily retroviruses) contain only RNA. In addition, the virion (the infectious viral particle that contains the genetic material, and that consists of two proteinaceous structures—a dense core and an envelope around the core) contains an enzyme known as reverse transcriptase. [NOTE: Certain other non-retroviral agents possess some of the above-mentioned characteristics, but none has all three.]

The genome of retroviruses is diploid in nature (meaning that it contains two identical molecules of single-stranded RNA). The reverse transcriptase enzyme in retroviruses allows them to make DNA from the virus' RNA after its successful entry into the host cell. Prior to the discovery of reverse transcriptase, it was believed that DNA could make RNA, but the reverse procedure was unknown. Retroviruses, however, make DNA copies of their genome by using their RNA template. This heretofore-unknown reversal was considered as being somewhat "backward," which explains the reason for the name "retrovirus," meaning "backward virus." Once DNA has been formed from the RNA of the retrovirus (via reverse transcriptase), that DNA then is incorporated into one of the chromosomes of the host cell.

For purposes of our discussion here I will be referring in broad terms to two different groups of retroviruses. **Exogenous retroviruses** (whether pathogenic or non-pathogenic) originate from outside an organism, and infect their hosts via an external environment. Exogenous retroviruses (e.g., HIV, which

causes AIDS) reproduce within **somatic cells** [i.e., body cells, as opposed to germ-line (sex) cells], and generally are spread through a variety of means such as sexual intercourse, contaminated blood or blood products, consumption by a child of breast milk from an infected mother, etc. **Endogenous viruses** (as the term is commonly used in the scientific literature), on the other hand, are alleged to be viruses that have successfully inserted their genetic material into a host's **germ cells** (sperm and eggs), and as a result have been passed down to subsequent generations in a process known as "germline transmission." As Larsson and Anderson noted, "By definition, ERVs are present in the genomes of all cells of an organism. They are transmitted vertically and inherited according to Mendelian expectations by subsequent generations. After integration, the provirval DNA is generally subject to the same biological regulation as 'normal' chromosomal DNA" (1998, 48[4]:329).

Endogenous Retroviruses as an Alleged Proof of Evolution

In the past, evolutionists offered two basic arguments as to why they believed that endogenous retroviruses (ERVs) provided proof of their theory. The first argument suggested that ERVs were part and parcel of a much-larger genetic complement known collectively as "junk DNA" (also known in the literature as "selfish DNA" or "parasitic DNA"). The idea behind such a concept was that the so-called junk DNA was basically a useless leftover that persisted within the genomes of higher organisms solely because such DNA, although it had no known function, was able to reproduce itself within the genome of its host. In essence, then, such portions of an organism's DNA endowment had become a parasite (albeit it an apparently benign one for the most part) that was doing little more than hitching a ride through history. Thus, such junk DNA was viewed as little more than an evolutionary remnant from the distant past. Using that argument, evolutionists were not shy in suggesting to non-evolutionists that since (as was then thought to be the case) 98% of the human genome was "junk," surely that militated strongly against any kind of intelligent Designer. After all (so the argument went) what kind of Designer would create a genetic code that was 98% "junk"?

Today, of course, the argument about junk DNA is a moot point for at least three reasons. First, even the staunchest evolutionists acknowledge that "[i]t is impossible to prove absence of function for any region of DNA" (Max, 2003, Sec. 5.4). Second, as knowledgeable scientists are aware, "recent research has begun to show that many of these useless-looking sequences do have a function" (Walkup, 2000, 14[2]:19). As one writer observed (in speaking of allegedly "useless" pseudogenes, which are part of the so-called "junk DNA suite"), "[E]vidence for function is not limited to generic 'junk DNA', but is now known for representatives of **all** major types of pseudogenes" (Woodmorappe 2000, 14[3]:57, emp. in orig.). The functions of other portions of the junk DNA suite (such as introns) likewise are beginning to be better understood (see Walkup, 2000, for a review), which goes to show how fallacious the evolutionists' argument was about an intelligent Designer supposedly (and improperly) having created a genetic code in humans that was 98% "junk." And third, as I pointed out in the section of this rebuttal on vestigial organs and structures, the fact that scientists do not yet **know** the function of something does not mean it does not **have** a function. As Professor John Mattick of the University of Queensland in Brisbane, Australia, bluntly observed, "The failure to recognise the implications of the non-coding DNA will go down as the biggest mistake in the history of molecular biology" (see Pemberton, 2003). Well put indeed.

Furthermore, today scientists readily acknowledge that ERVs (in both animals and humans) have numerous important functions. The authors of one research paper on benefits of ERVs wrote in regard to human ERVs, "We argue that the activities and expression of HERVs in some cases represent a functional advantage for the host" (Larsson and Andersson, 1998, 48[4]:330). For example, regulation/activation of certain genes during embryonic development of mice has been documented (Peaston, et al., 2004). ERVs also can affect gene expression. Mager, et al. (1999) discovered that the LTRs (long terminal repeats, which are the longer, more-complex repetitive sequences at the ends of certain mobile elements that are required for them to be able to transpose) of two ERVs provide the sequence signal for the polyadenylation of the mRNA of two fairly recently discovered human genes. And, ERVs have been found to be responsible for regulating human genes expressed in the human placenta (e.g. pleiotropin) and somatic tissues (e.g. apolipoprotein C1 in the liver, and β-amylase in the salivary gland) [Coffin, 1996, pp. 1767-

1847; Bannert and Kurth, 2004]. Immunomodulation, including aiding in immunotolerance to self antigens and immunization against exogenous retroviruses, has been described as well (Coffin, 1996, pp. 1767-1847; Bannert and Kurth, 2004).

The HERV-W and HERV-FRD families of ERVs now are known to code for a protein (syncytin) that has the ability to fuse mammalian cells during human trophoblast development (Blond, 2000; Mi, 2000; Frendo, et al., 2003; Mallet, et al., 2004). As one medical doctor (and evolutionist) said of this discovery, "[t]oday there is general acceptance that this HERV-W and its translated product syncytin have important functions in human placental physiology" (Ryan, 2004). ERVs also are known to play other roles, such as in mammalian tissue organization (Matsui, et al., 2006). Plus, as the authors of an article in the *Scandinavian Journal of Immunology* ("Beneficial Role of Human Endogenous Retroviruses: Facts and Hypotheses") concluded,

> "We argue that the activities and expression of HERVs in some cases represent a functional advantage for the host…. The function of ERVs, particularly ERV3, in the placenta has been linked to several ERV activities: (1) provision of immunological protection of the embryo and the fetus; (2) regulation of trophoblast cell growth; (3) protection of the fetus from unwanted maternal material, and (4) protection against infection by a related exogenous retrovirus, i.e. 'germline vaccination'" (Larsson and Andersson, 1998, 48[4]:330,332).

The authors of that article stated at the conclusion of their research that, in their view, the "persistence of endogenous retroviruses in the genomes of eukaryotic cells **reflects their indispensability in important normal functions in specialized cellular environments**" (p. 329, emp. added). Compare that assessment with a statement made as late as 2006 in a review article titled, "The Discovery of Endogenous Retroviruses," in which the author commented, "**ERV appear to be parasitic DNA sequences for which the host has little use**, other than to protect against further retrovirus infection" (Weiss, 2006, emp. added).

Since those two diametrically opposed statements were made, additional information has come to light that shows how correct the assessment of the authors of the 1998 article in the *Scandinavian Journal of Immunology* turned out to be, and how mistaken Weiss was in his 2006 review article. For example, in 2008 researchers reported having identified an important function for a large proportion of the human genome that has been designated as ERVs, and acts as promoters that start transcription at alternative points, thereby enabling different RNA transcripts to be formed from the same DNA sequence. The authors of the paper wrote,

> "We report the existence of 51,197 ERV-derived promoter sequences that initiate transcription within the human genome, including 1,743 cases where transcription is initiated from ERV sequences that are located in gene proximal promoter or 5' untranslated regions (UTRs)…. Our analysis revealed that retroviral sequences in the human genome encode tens-of-thousands of active promoters; transcribed ERV sequences correspond to 1.16% of the human genome sequence, and PET tags that capture transcripts initiated from ERVs cover 22.4% of the genome" (Conley, et al., 2008, 24[14]:1563,1566).

Batten concluded that such data "illustrate the potential of retroviral sequences to regulate human transcription on a large scale consistent with a substantial effect of ERVs on the function and evolution of the human genome" (2006).

Thus, the first part of the evolutionists' argument concerning how ERVs represent proof of organic evolution—i.e., that they are useless vestiges representing an evolutionary past long since gone—can now be placed into the same relic heap of history from which those allegedly useless ERVs were supposed to have originated. Or, as one writer put it, "The molecular taxonomists, who have been drawing up evolutionary histories ('phylogenies') for nearly every kind of life, are going to have to undo all their years of 'junk DNA'-based historical reconstructions and wait for the full implications to emerge before they try again" (Williams, 2007, 21[3]:113).

The second part of the evolutionists' argument regarding ERVs goes something like this. In times past, various strains of ancient exogenous retroviruses infected the germ cells of all vertebrates (and many non-vertebrates). As the retroviruses inserted their genetic material into the host's genome, they became endogenous in nature, thereby ensuring that that genetic material would be passed down to (and through) generations of subsequent offspring. Estimates to date suggest that ERVs make up between 5% (Conley,

et al., 2008) and 8% (Ryan, 2004) of the DNA in a human, or about 10% of the total amount of human DNA that is classified a transposable elements (Thornburg, et al., 2006).

Most ERVs are considered to be "proviral" (meaning that they are a latent form of an original virus that no longer is able to replicate within or infect a host), and therefore are not harmful (at least in a lethal fashion) to the organisms that carry them. [Rare (to date) exceptions have been documented, however. When scientists inoculated an isolated murine leukemia virus (specifically, MuLV, an endogenous mouse retrovirus) into a new host, the proviral material was able to successfully colonize the recipient genome (see Lock, et. al., 1988).]

Because evolutionists believe that ERV integration sites in a host's genome are completely random (at the time of insertion into the germline as an exogenous retroviral infection), and because ERV integration points within host chromosomes frequently are found at identical positions (loci) in species that are alleged by evolutionary theory to be related, the assertion is that those shared loci could have occurred only by common descent. This textbook case of presupposition explains why ERVs are considered by many evolutionary scientists as the "poster children" for biological evolution.

To ensure that I do not misrepresent the evolutionists' position regarding ERVs, I would like to include the following material from Cath Ennis, an evolutionist who writes frequently on the topic of ERVs and how they allegedly support evolutionary theory.

> "What we can show is that ERVs provide evidence in support of the theory of evolution. Let's imagine how ERVs would behave within a model of evolution by common descent. An ancient creature, let's call it the common ancestor of all modern mammals, is infected by a retrovirus that becomes endogenous. All of the animal's descendants (i.e. all mammals) would be expected to carry the same ERV insertion (ERV1) in the same chromosomal location.
>
> "Fast forward in evolutionary time. Different lineages have evolved and diverged from the original common ancestor and there are now many different types of mammal in existence, all carrying ERV1. A small rodent, let's call it the common ancestor of mice and rats, is again infected by a species-specific retrovirus that becomes endogenous. This is ERV2. In a parallel event in a different lineage, the common ancestor of all great apes acquires a third insertion, ERV3.
>
> "Moving forward again, a fourth ERV appears in some of these new-fangled human thingies that are running around in Africa, but not in their hairier relatives who will eventually evolve into modern chimpanzees. The early humans spread out, and a fifth and (don't worry) final ERV arises in a population that is isolated in a discrete geographical location. The infection does not spread to other human populations.
>
> "So what would we expect? Humans, chimps, mice and rats should all possess ERV1. The mouse and rat genomes will also contain ERV2, the virus that infected their common ancestor, but not the primate-specific ERV3, 4 or 5 insertions. All great apes will share an identical ERV3 insertion; all humans will also possess an ERV4 insertion that is not found in chimps or other apes. In addition, some, but not all, humans will carry an insertion of ERV5. The rodent-specific ERV2 insertion will not be found in any primate species.
>
> "Now that several genomes have been sequenced, we have begun to test these predictions. The patterns of ERV insertions observed in modern species exactly match the predictions made by the model described above. Some insertions are shared between humans and mice and represent truly ancient viral infections. Others are found only in primates, and not in other species, obviously derived from an infection of the ancestral primate species after its divergence from other lineages. More modern insertions are found only in humans, while the youngest ERVs of all are found in some humans, but not in all. We do not find any examples of ERV insertions shared by, say, humans and mice, but not by chimps. Insertions are always shared by all species, and only by those species that have a common ancestor. ERV insertions therefore provide excellent support for the theory of evolution by common descent" (Ennis, 2007).

Before commenting on the above material, I first would like to mention another point along these lines that quite frequently appears in the evolutionary literature. In an online article titled "29+ Evidences for Macroevolution," evolutionist Douglas Theobald commented, "In the following list of evidences, 30 major predictions of the hypothesis of common descent are enumerated and discussed" (Theobald, 1999). Included among Dr. Theobald's "30 major predictions" were the following: "Prediction 4.3: Molecular

evidence — Transposons"; and "Prediction 4.5: Molecular evidence — Endogenous Retroviruses." Another avid online defender of ERVs as proof of evolution wrote, "Scientists found the identical Endogenous Retrovirus (ERV) in the identical spot of the same chromosome of different species **just as evolution would have predicted!**" (Anonymous, n.d., emp. added).

When it comes to transposons and ERVs (which represent a subset of transposons, and which some evolutionists prefer to call "retrovirus-like elements" or "remnant sequences" since the genetic material of ERVs is different than that of exogenous retroviruses), someone needs to step forward to point out that **evolutionary theory did not make any such predictions as those claimed by the above-mentioned writers**. Fortunately, "someone" has stepped forward to point out that very thing. In a lengthy response to Theobald's "29+ Evidences for Macroevolution," Ashby Camp addressed the suggestion that evolutionary theory "predicts" such things as transposons or ERVs.

> "It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that the 'same transposon' will exist in the same chromosomal location in two or more species. Evolution does not even predict the existence of transposons, much less that they will be found at the same location in two or more species. Until transposons were discovered in the late 1940s, conventional wisdom was that all genes worked from a stable position along a chromosome, and no one considered that cause for concern. On the contrary, [Nobel-Prize winning microbiologist's Barbara] McClintock's initial claims about transposons were resisted because they were contrary to the prevailing view of genetics. So, while evolutionary theory was able to accommodate transposons, it was quite comfortable with their absence.
>
> "Evolution likewise makes no prediction about how transposons will operate, given their existence. The theory can accommodate any process of transposition, however simple or complex and however chaotic or uniform, and can accommodate the transposed elements remaining at insertion sites for any length of time. Thus, transposons are not confirmation of an evolutionary prediction but observations that are given an evolutionary interpretation
>
> "…Evolutionary theory was considered robust prior to the discovery of ERVs. This is but another example of taking an observation, claiming it as a **prediction** of evolution, and then using the fact that the observation fits the prediction as evidence for the truth of evolution" (2001, emp. in orig.).

Camp is absolutely correct. Evolutionists do not help themselves (or their theory) when, in what is patently (and obviously) an after-the-fact fashion, they attempt to hijack the data in order to boldly claim that those data were "predicted" by evolutionary theory. In the case of ERVs, that most definitely is **not** the case (as the stern opposition to McClintock's suggestion regarding the existence of transposable elements documents all too well).

Problems with ERVs as an Alleged Proof of Evolution

In spite of the bold (and frequent) claims made by evolutionists regarding how strong a case ERVs provide for an evolutionary scenario, the truth of the matter is that there are serious problems with such a concept, as well as possible alternative explanations.

Missing "Must-Have" ERVs in Humans

According to evolution-based phylogenies, chimpanzees are closer relatives to humans than they are to gorillas. If this claim is true, and if evolutionists are correctly interpreting the presence of ERVs in those species' genomes, then gorillas and chimpanzees should not share an ERV unless that ERV also is present in humans. As Ennis (quoted earlier) boasted, "Insertions are always shared by all species, and only by those species that have a common ancestor" (2007).

However, as a paper by Barbulescu, et al., in *Current Biology* explained, there is at least one ERV (HERV-K) that is present in chimps and gorillas, yet not in humans. In reporting the results of their research, the scientists wrote, "We identified a human endogenous retrovirus K (HERV-K) provirus that is present at the orthologous position [where it "should" be if both organisms descended from a common ancestor] in the gorilla and chimpanzee genomes, but not in the human genome," and then went on to state the obvious: "**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" (Barbulescu, 2001, 11:779, emp. and bracketed material added).

This presented the researchers with quite a quandary. It was not possible for them to suggest that the HERV-K had somehow "been eliminated," since "the integration process of a retroviral element *per se* is irreversible" (Bannert and Kurth, 2004, 101:14576). In fact, Barbulescu and colleagues came to the exact same conclusion, writing, "It is highly unlikely that the provirus was deleted in humans, as the retroviral integration process is irreversible" (p. 780). They also noted,

> "There is no small duplication, tandem or otherwise, within the sequenced stretch of any of the genera that might have participated in a putative recombination event to replace the provirus within the human lineage…. The data are consistent with the conclusion that these genera lack an appropriate locus for a putative gene conversion event that could have eliminated the provirus within the human lineage…. Thus, it is unlikely that any nonorthologous sequence in the human genome, L1 repeat or otherwise, existed in recent human evolution that could have served as the source sequence for the putative gene conversion event that replaced the HERV-K-GC1 provirus specifically within the human lineage" (pp. 781,782).

So how did the researchers end up attempting to explain the all-important missing ERV that **should** have been present (a mild understatement!) but was **not**? Here is their explanation:

> "Several possibilities were considered to explain how a provirus could be present in *Gorilla* and *Pan* but be absent in *Homo*…. The presence of HERV-K-GC1 in gorillas and chimpanzees, but not humans, is best explained by the maintenance of the preintegration site in the human lineage since before the time when the provirus formed in the common ancestor of chimpanzees and gorillas…. **The precise details of the nature of the phylogenetic separation of humans from the Africa great apes has remained uncertain**….At positions in the genome where allelism was maintained throughout the period of existence of the human-chimpanzee common ancestor, some of the same alleles that became fixed in the gorilla may also have been fixed in only one of the human or chimpanzee lineages. The HERV-K-CG1 provirus provides a compelling piece of evidence for such a model, as **it is the clearest example to date of a specific location within the genome where chimpanzees and gorillas are more closely related to each other than either is to humans**…. This leads to the conclusion that for some fraction of the genome, the gorilla and chimpanzee genomes are more closely related to each other than either is to humans" (pp. 780,782,783, emp. added).

As one evolutionist noted, "When phylogeny studies are carried out, the phylogeny of broken viruses mirrors that of other phylogenies" (Musgrave, 2006). Oops. Not true!

Closely Related ERVs in Phylogentically Distant Species

But the situation worsens as additional data are examined. Evolutionists hardly would expect ("predict"?) that **closely related** ERVs would appear in **phylogenetically distant** (from an evolutionary viewpoint) species. Yet that is exactly what has been documented. As Baillie and Wilkins reported in the *Journal of Virology*,

> "We have sequenced and characterized an endogenous type D retrovirus, which we have named TvERV(D), from the genome of an Australian marsupial, **the common brushtail possum** (*Trichosurus vulpecula*). Intact TvERV(D) gag, pro, pol, and env open reading frames were detected in the possum genome. TvERV(D) was classified as a type D retrovirus, **most closely related to those of Old World monkeys, New World monkeys, and mice, based on phylogenetic analyses and genetic organization**" (2001, emp. added)

Furthermore, as Weiss (2006, figure 4) pointed out, "two closely related ERV genomes are found in a carnivore (fox) and a ruminant (sheep)." Additionally, as Weiss went on to point out,

> "Gamma-retrovirus was isolated from trophoblastic cells of the **baboon** placenta. This virus was found to be very closely related antigenically and by sequence homology to the endogenous RD114 virus in cats (which is itself unrelated to endogenous FeLV). Benveniste and Todaro observed, like we did for jungle fowl, that only certain species of the **cat** genus, *Felis*, possessed this endogenous genome related to the baboon ERV. In contrast, all species of baboons carry this virus so it would appear to have been present in the germ line of primates much longer than in cats. Thus it seems evident that a horizontal, infectious event occurred to transfer the virus from baboons to cats, whereupon it became endogenous in the new

species" (emp. added)

So-called "convergent evolution" hardly can be expected to account for "two closely related ERV genomes" being found in species as divergent as possums, Old World monkeys, New World monkeys, and mice (or foxes and sheep, or baboons and cats!). Many evolutionists point to ERVs as one of the most-powerful proofs of their theory. As Ennis (quoted above) put it, "We do not find any examples of ERV insertions shared by, say, humans and mice, but not by chimps. Insertions are always shared by all species, and only by those species that have a common ancestor. ERV insertions therefore provide excellent support for the theory of evolution by common descent" (2007). Oops. Not true! This appears to be a textbook case of the old saying, "That which proves too much proves nothing at all."

Non-evolutionists naturally point to the type of documented scientific evidence discussed in this section (and the one above on missing ERVs in humans), and suggest that this type of evidence falsifies evolutionary phylogenies that imply a common ancestor. But, as Camp lamented,

> "The suggestion that the hypothesis of common ancestry would be falsified by the discovery of the same ERV at the same locus in two species that are not believed to have shared a recent common ancestor is incorrect. ERVs simply would join the list of alleged markers for evolution that exhibit homoplasy [correspondence between parts or organs acquired as the result of parallel or convergent evolution]. And given what is known of retrovirus selectivity, I doubt anyone would be surprised" (2001, bracketed material added).

But before you buy into the idea of parallel or convergent evolution as an adequate explanation for closely related ERVs appearing in evolutionary-unrelated species, I invite you to keep reading and consider the remainder of the evidence.

Why Does the Same ERV Transcribe Differently between Supposedly Closely Related Species?

In an article titled, "Lineage-Specific Expansions of Retroviral Insertions within the Genomes of African Great Apes but Not Humans and Orangutans," Yohn, et al., wrote,

> "Based on analysis of finished BAC chimpanzee genome sequence, we characterize a retroviral element (*Pan* troglodytes endogenous retrovirus 1 [PTERV1]) that has become integrated in the germline of African great ape and Old World monkey species but is absent from humans and Asian ape genomes … Six out of ten of these genes, for which there are expression data show significant differences in transcript expression between human and chimpanzee" (Yohn, et al., 2005).

Furthermore, why is PTERV1 present in "great ape and Old World monkey species but **absent** from humans and Asian ape genomes"? Also, as Stengel, et al., observed,

> "Most HERVs are active in at least some tissues, though tissue specificity is common for most elements. We analyzed multiple tissues from several Old World monkeys using retroviral pol-based DNA microarrays and quantitative PCR methods to determine their ERV expression profiles. The results demonstrate that while many ERVs are active in nonhuman primates, overall the tissue expression specificity is unique to each species. Most striking is that while the majority of HERVs analyzed in this study are expressed in human brains, almost none are expressed in Old World monkey brains or are only weakly expressed" (2007).

Why is there such a difference between HERVs in human brains and those in Old World Monkey brains if they evolved from the same common ancestor?

Requiring Far Too Much of "Convergent Evolution"

Convergent evolution (the idea that similar functions or traits evolved separately in unrelated lineages) is a convenient crutch often employed by evolutionists to explain what, at times, is otherwise inexplicable. But when it comes to ERVs (and other transposable elements), evolutionists appear to be requiring far too much of such a construct (and appear to be leaning on a broken reed for support when they do so).

For example, why would unrelated ERVs in unrelated species create practically the same gene? In *The Atlas of Genetics and Cytogenetics in Oncology and Haematology*, the following information can be found:

> "ERVWE1/Synctin-1 and ERVFRDE1/Syncytin-2 are specific to primates, and thus do not exist in other placentae. However, this apparent endogenous retrovirus hijacking for placentation use is not restricted to the primates. Indeed two unique endogenous envelope genes of retroviral origin have been found in the mouse, i.e. Syncytin-A and –B … Altogether the data strongly argue for convergent evolution of endogenous retroviral envelopes to serve for placentation in mammals" (see *Atlas*…).

Or, why would two unrelated ERV LTRs that alleged evolved independently create the same regulatory roles for the same gene? Romanish, et al., wrote,

> "We demonstrate that both the human and rodent neuronal apoptosis inhibitory protein (NAIP) genes, involved in preventing cell death, use different ERV sequences to drive gene expression. Moreover, in each of the primate and rodent lineages, two separate ERVs contribute to NAIP gene expression. **This repeated ERV recruitment by NAIP genes throughout evolution is very unlikely to have occurred by chance**. We offer a number of potential explanations, including the intriguing possibility that it may be advantageous for anti-cell death genes like NAIP to use ERVs to control their expression. These results support the view that not all retroviral remnants in our genome are simply junk DNA" (2007, emp. added).

Non-evolutionists agree: "This repeated ERV recruitment by NAIP genes throughout evolution **is very unlikely to have occurred by chance**."

Additionally, why does the same ERV transcribe differently among different cell types within the same organism? Seifarth, et al., noted,

> "Furthermore, there is evidence that transcription of at least some HERV families may be differentially regulated depending on the cell type. Characterization of promoter activities of HERV-K, HERV-H, HERV-E, ERV9, and HERV-W families, the most intensively studied HERVs, revealed specific cell type preferences for each HERV family, and even individual elements of one family showed significant variation in transcription pattern. In some cases, transcription factor binding sites that interact with cell type-specific nuclear factors could be identified, demonstrating that the expression of HERVs is regulated in a complex and diverse manner comparable to cellular genes" (2005).

A statement like "the expression of HERVs is regulated in a complex and diverse manner comparable to cellular genes" does not lend itself well to purely naturalistic processes, does it?

Why Would Viral-Infected Germline Cells Be "Fit to Survive" (Positive Selection), and How Did ERVs "Evolve" from Being Exogenous Pathogenic Infectious Agents into ERVs that Have the Ability to Protect Hosts from Infections?

From the standpoint of natural selection, why would viral-infected sperm and egg cells be "more fit to survive," and therefore be more (or as) likely to be passed on than their otherwise-healthy counterparts containing no retroviral DNA? Having strong, healthy germ cells is undeniably important in the production of a viable zygote, so why should viral-infected germ cells be "selected for" in nature (and even become more prevalent than non-infected populations)?

Apoptosis (programmed cell death) is a fact of nature, so why wouldn't germ cells containing a viral-infected genome be eliminated by natural selection?

> "Apoptosis, or programmed cell death, is a normal component of the development and health of multicellular organisms. Cells die in response to a variety of stimuli and during apoptosis they do so in a controlled, regulated fashion…. The latter occurs when T-cells recognise damaged or virus infected cells and initiate apoptosis in order to prevent damaged cells from becoming neoplastic (cancerous) or virus-infected cells from spreading the infection" (see "Apoptosis," n.d.)

Evolutionists may claim that retroviruses were in some sort of "dormant cycle" upon insertion into their hosts' genomes (and that the retroviruses may even have remained intact prior to being disabled by mutations, deletions, or recombination). So, before the retroviruses were disabled by such processes, what prohibited them from becoming infectious, thereby weakening or destroying the host embryo, and then also likewise weakening or killing young and/or adult hosts?

Moreover, how did ERVs "evolve" from being **exogenous pathogenic infectious agents** into ERVs that have the ability to **protect hosts from infections**? As Bannert and Kurth noted, "A remarkable fraction [of retrotranscribed and reinserted elements] is derived from ancient exogenous retroviruses that

found their way into the germ line that became for most, if not all, a graveyard" (2004, 101:14578). [Such a comment raises the issue about which evolutionists Larsson and Andersson were bold enough to inquire: "One key question concerns why ERVs have been retained throughout evolution" (1998, 48[4]:330). A thought-provoking question indeed!]

Yet in spite of the fact that when "most, if not all" retroviruses found themselves "in a graveyard" in the human genome, they "somehow" took on (and completed!) amazing tasks, as Ponferrada, Mauck, and Woolley reported when they observed that "HERV-W Env confers host cell resistance to infection by SNV [spleen necrosis virus]. This is the first report of a human endogenous retrovirus gene product blocking infection by any exogenous retrovirus" (2003). Mura, et al., reported in the *Proceedings of the National Academy of Sciences*,

> "A possible biological role hypothesized for ERVs is to help the host resist infections of pathogenic exogenous retroviruses, affording a selective advantage to the host bearing them. For instance, some avian and murine [mouse] ERVs can block infection of related exogenous retroviruses at entry by receptor interference; mouse Fv-1 blocks infection at a preintegration step, also can be viewed as an ERV" (2004, bracketed word added).

One cannot help but wonder how viruses that started out as pathogenic parasites ended up accomplishing so much good "naturally." Geneticists (admittedly intelligent designers) have experienced numerous problems as they have attempted to use retroviruses as vectors in gene-therapy experiments.

> "Scientists have tried to take advantage of the virus's biology and manipulate its genome to remove human disease-causing genes and insert therapeutic genes. However, viruses, while effective, introduce other problems to the body, such as toxicity, immune and inflammatory responses, and gene control and targeting issues" (see "Germline Gene Transfer, n.d.).

This, then, makes one wonder how in "nature" (which, admittedly, is **not** an intelligent designer!) retroviruses could randomly invade healthy germ and somatic cells without damaging them, killing them, or activating apoptosis, and then subsequently be "neutralized" (or even made beneficial in many instances!) by being turned into ERVs.

Furthermore, xenotropic ERVs (i.e., retroviruses that can replicate in cells other than those that would be considered their normal host species) are known to reside in cells that have no receptor for them. Instead, envelope (*env*) proteins of these ERVs bind receptors on the cells of other species. As one scientist asked, "How did these ERVs get into the cell, if they were not built inside?" (Liu, 2006). That question has not been lost on evolutionists, who wrote in the standard go-to manual on retroviruses (eponymously titled *Retroviruses*),

> "It is no surprise to read speculations like this in *Retroviruses*, the 'Bible' of retrovirology: "It is likely that xenotropic viruses originally inserted into the germ line in a host background that encoded their cognate receptor but that the functional xenotropic viral receptor allele was subsequently lost, probably under selective pressure from exogenous xenotropic viruses" (Coffin, et al., 1997, p. 77).

But, as Liu remarked regarding after reading such a suggestion, "The term 'exogenous xenotropic virus' is difficult to conceive, if not self-contradictory" (Liu, 2006). Agreed—100%! [Think about it.]

Why Are There No Examples of an ERV that Has Been "Endogenized" in Modern Times? And Why Are There No Examples of HERVs that Have a Direct Exogenous Counterpart?

As Liu pointed out, "Endogenization of modern exogenous retroviruses is rarely observed in **nature**" (2006, emp. in orig.). According to evolutionists Bannert and Kurth,

> "The rate of new human germ line insertions is presently at an extremely low level compared to earlier periods of evolutionary history or to the rate in some other mammals…. **No current transposition activity of HERVs or endogenization of human exogenous retroviruses has been documented so far**" (2004, 101:14572,14573, emp. added).

Hughes and Coffin wrote, "Most of these elements represent ancient retroviral infections, as evidenced by their wide distribution in primate species, and no infectious counterparts of human endogenous retroviruses (HERVs) are known to exist today" (2004).

In this instance, non-evolutionists agree with their evolutionary counterparts. As Purdom noted, "No exogenous counterparts of HERVs exist." It has been estimated that there are approximately 98,000 different ERV sequences (mostly partial) of some kind or another within the human genome. Why is it that ERVs have supposedly invaded germ cells countless times, but no other virus has been discovered to do the same?

The Controversial Origin of ERVs, and Their Acknowledged Site Specificity

As one writer noted, when it comes to HERVs,

> "We're not just talking about a small scale phenomenon. These ERVs aid transcription in over **one-fifth** of the human genome! This again debunks the idea that 98% of the human genome is junk, and it makes the inserted evolutionary spin look like a tacked-on nod to the evolutionary establishment. These results support the conclusions of the ENCODE project, which found that at least 93% of DNA was transcribed into RNA" (Doyle, 2008).

But what is the specific **origin** of ERVs? The search for an answer to that question has caused considerable discussion (and probably even **more** disagreement!) in the scientific community. There are some scientists (both evolutionists and non-evolutionists) who even have suggested that rather than ERVs starting out by infecting cells as exogenous viruses and then becoming ERVs, the reverse actually occurred. Larsson and Andersson said in this regard:

> "However, there is still controversy concerning the true origin of retroviruses and ERVs. An unresolved issue is whether ERVs originated solely from germline infections or also by reorganization and acquisition of cellular sequences. It would be difficult to clearly discern between these scenarios since many genomes are continuously deleting newly introduced exogenous retroviruses" (1998, 48[4]:330).

Greenwood, et al., added, "Exogenous retroviruses may have originated from ERVs, and ERV-Ls in particular may represent an intermediate between retrotransposons and exogenous viruses" (2004, 4:38). Yet instances in the literature dealing with ERVs show that when non-evolutionists venture to make such a suggestion, it generally is met with great disdain. The point I am attempting to make here is that the specific origin of ERVs is not a matter that, as yet, has been settled (a point that will become quite important toward the conclusion of this discussion).

It also is interesting to note that, in addition to performing certain useful and/or critical functions, some ERVs and other transposable elements (TEs) also exhibit what is known as an "insertion bias." Sverdlov wrote,

> "But although this concept of retrovirus selectivity is currently prevailing, practically all genomic regions were reported to be used as primary integration targets, however, with different preferences. There were identified 'hot spots' containing integration sites used up to 280 times more frequently than predicted mathematically. A recent study of the *de novo* retroviral integration demonstrated also preference for scaffold- or matrix-attachment regions (S/MARs) flanked by DNA with high bending potential. The S/MARs are thought to be important functional sequences of the genome that anchor chromatin loops to the nuclear matrix, subdividing the genome into functional domains. They often neighbor regulatory elements involved in gene expression and DNA replication. A cautious generalization from these findings could be that although TEs can integrate into many sites, and may prefer non-coding regions, the *de novo* integration is frequently targeted at the sites in the vicinity of functionally important elements like transcriptions start points or origins of replication" (Sverdlov, 1998, 428:3).

Purdom hardly overstated the case when she wrote, "It is possible that certain sites are predisposed to the insertion of retroviruses" (2006). Non-evolutionists see this (along with such things as the beneficial functions or roles of many ERVs) as simply one more piece of evidence indicating a finely tuned system. Camp wrote in this regard,

> "Interestingly, one of the ways evolutionists explain how the various kinds of transposons spread from the individuals in whose germline cells they first arose to all members of the species is by appeal to the possibility that each of the transposons wound up close to an advantageous gene that became prevalent in the population by natural selection (Max, 2003, Section 3). In other words, the various transposons are thought to have spread within the originating species by a fortuitous proximity to advantageous genes.

> **One could turn that around and suggest that the transposons were close to genes because they performed a function related to the genes**" (2001, emp. added).

Furthermore, the mobility of transposable elements seems to be highly controlled by the host to provide stability to the host genome (Kazazian, 2004). Most ERVs known today are not actively transposing (i.e., moving or replicating) within the host-cell genome (although some mouse ERVs are capable of moving/expanding within the genomes of their hosts). Human ERVs appear to be fixed in both numbers and positions. This presents somewhat of a quandary for evolutionists, since the mouse ERVs (according to evolutionary theory) would be older, afflicted with more-numerous detrimental mutations, and thus supposedly less functional. Liu undoubtedly had this in mind when, in referring to the fact that ERVs in humans (which are assumed to be younger) are less mobile than their older mouse counterparts, inquired, "Are the human ERVs older, therefore more degenerated and less active? If the human race is younger than the murine race, as evolutionary biologists believe, there is no reason to suppose that the human ERVs are older than those of the mouse" (2006). Good point.

Conclusion

In the introduction to the section of this rebuttal that deals with endogenous retroviruses, I noted that in the past evolutionists employed two arguments related to ERVs. The first argument suggested that ERVs were part of a much-larger genetic complement known collectively as "junk DNA," which was viewed as an evolutionary vestige of a long-distant past. Part of the evolutionists' argument was that such useless genetic remnants militated strongly against any kind of intelligent Designer since no Designer with intelligence would create a genetic code that was filled with junk." Evolutionist Edward Max wrote, "The creationist argument discussed earlier—that similarities in DNA sequence simply reflect the creator's plans for similar protein function in similar species—does not apply to sequences that do not have any function for the organism that harbors them (2003). We now know, of course, that the so-called "junk DNA" is not junk at all—making the evolutionists' argument, at best, moot, and at worst, embarrassingly wrong (what evolutionist John Mattick, quoted earlier, referred to as "the biggest mistake in the history of molecular biology!").

The second part of the evolutionists' argument concerning transposable elements (of which ERVs are a subset) likewise has been shown to be incorrect. First, the assertion was made that "insertions are always shared by all species, and only by those species that have a common ancestor" (Ennis, 2007). We now have scientific evidence showing that is **not** the case. The second assertion was that "when phylogeny studies are carried out, the phylogeny of broken viruses mirrors that of other phylogenies" (Musgrave, 2006). We likewise now know that, too, **not** to be the case. The third assertion was that "a specific retroviral integration site shared by two species is indicative of a common ancestor **because the likelihood of independent integrations at exactly the same locus (insertional homoplasy) is negligible**" (Bannert and Kurth, 2004, 101:14576, emp added). Babulescu, et al., wrote, "Proviruses or solo LTRs [solitary long terminal repeats, which act as promoters and enhancers, and which compose about 85-90% of ERV elements] present at the same site in the genomes of two species are identical by descent, **as the likelihood of independent integrations at the same site (insertional homoplasy) is negligible**" (2001, 11:779; parenthetical item in orig.; emp. and bracketed material added).

Interestingly, however, a few bold souls in the evolutionary camp have stepped forward to reprimand their own colleagues for suggesting that a specific retroviral integration site shared by two species is "negligible." For example, well-known vertebrate paleontologist Maureen O'Leary chastised one fellow evolutionist (Dr. Okada) for rejecting the possibility that various transposons (SINEs and LINEs—short and long interspersed elements) ***could* arise independently in separate lineages**. In an article in *Nature*, Trisha Gura reported Dr. O'Leary's unsettling comments as follows:

> "Okada's studies on SINEs and LINEs, held up by the molecular enthusiasts as their strongest line of evidence, have attracted particular scrutiny. 'It is an outdated method in systematics to assert that one aspect of the organism somehow dictates the true phylogeny,' says O'Leary. 'Okada is approaching this completely backwards by asserting that his retrotransposons are so significant that he cannot imagine a way in which they evolved convergently' " (Gura, 2000, 406:232).

Interestingly, one year after Dr. O'Leary made her critical remarks, a team of molecular geneticists led by Michael Cantrell discovered two "hot spots" where the same SINEs **inserted independently**. They wrote:

> "Vertebrate retrotransposons have been used extensively for phylogenetic analyses and studies of molecular evolution. Information can be obtained from specific inserts either by comparing sequence differences that have accumulated over time in orthologous copies of that insert or by determining the presence or absence of that specific element at a particular site. The presence of specific copies has been deemed to be an essentially homoplasy-free phylogenetic character because the probability of multiple independent insertions into any one site has been believed to be nil. . . . We have identified two hot spots for SINE insertion within *mys-9*, and at each hot spot have **found that two independent SINE insertions have occurred at identical sites**. ***These results have major repercussions for phylogenetic analyses based on SINE insertions, indicating the need for caution when one concludes that the existence of a SINE at a specific locus in multiple individuals is indicative of common ancestry***. Although independent insertions at the same locus may be rare, **SINE insertions are not homoplasy-free phylogenetic markers**." (Cantrell, et al, 2001, 158:769, emp. added).

When non-evolutionists suggest (based on examples like those above in which occurrences of independent integrations at the same site **are** known to occur) that the existence of transposable elements (like ERVs) "at a specific locus in multiple individuals" is not necessarily "indicative of common ancestry," and that such independent insertions in various species "have major repercussions for phylogenetic analyses," they are ignored or ridiculed. Yet that is the very same thing that the evolutionists themselves are now saying!

The bottom line is that the argument based on ERVs turns out to be extremely problematic. If transposons have a function (which we now know that they do), then it goes without saying that an intelligent Designer may well have had a good reason for placing them at the same chromosomal locations in different species. He also may have had a good reason for designing certain transposons with built-in site specificity for certain loci (which we now know is characteristic of various ERVs and other transposons).

Additionally, Todd Wood (2002) has proposed that creatures may have been endowed originally with mobile genetic elements (which Wood has named "altruistic genetic elements") to facilitate diversification. If, as Wood and others have suggested, over time such a diversification system degenerated so that today we see only remnants (and perhaps distorted remnants at that) of its past operation, the fact that we **currently** do not see insertion bias in a particular transposon (to choose just one example) would not necessarily mean that such bias **never existed**. Or, for that matter, the insertion bias that we do observe in certain transposons may no longer be serving its original purpose. In short, it may be that some ERVs are vestiges of an original complex system intended to facilitate diversification within organisms.

One thing is certain (to paraphrase Cantrell, quoted above): The data presented in this section of this rebuttal undoubtedly have major repercussions for evolutionary phylogenetic analyses, and indicate the danger of attempting to conclude that the existence of an ERV (or any other transposon) at a specific locus in multiple individuals is indicative of common ancestry.

EVOLUTIONIST'S TENTH ARGUMENT: NATURAL SELECTION

The tenth item in the list of ten "proofs of evolution" that my challenger in this debate offered was the suggestion that "complexity via natural selection has never been contradicted." As with my opponent's use of several other arguments (e.g., bacterial antibiotic resistance, DNA, and vestigial organs), I was surprised to see him list "complexity via natural selection" as a major argument for evolution. Here's why.

The Origin of Species by Means of Natural Selection was the title of the book authored by Charles Darwin in November 1859. Those last two words, "natural selection," have been discussed frequently within the halls of science. Darwin suggested that "natural selection is daily and hourly scrutinizing every variation, even the slightest; rejecting that which is bad, preserving and adding up all that is good; silently and insensibly working at the improvement of each organic being." And it certainly is no secret that Darwin's concept of "natural selection" (or "survival of the fittest," as it has come to be known) was for many years

at the center of evolutionary thought. According to Darwin, an individual creature with a particular advantage—the "fittest of its kind"—naturally would be selected to pass on the advantage to its offspring. A horse with long legs, for example, would be able to gallop faster than the rest, thus escaping from predators in order to produce heirs. A "fit" creature, therefore, was the one that could best carry out the functions that kept it alive—it was the best adapted to its environment and to its way of life. Paleontologist Kurt Wise defined it as "the preferential survival of those individuals with heritable characters that give advantage to them in the environment in which they find themselves" (2002, p. 165). This is what Darwin meant by "survival of the fittest."

Evolutionist Francis Hitching, in *The Neck of the Giraffe*, noted that natural selection did not become just biology's unifying principle, "but its mantra—a phrase embodying a kind of spiritual power" (1982, p. 83). Harvard's famed taxonomist, Ernst Mayr, compared it to a sculptor. Sir Gavin de Beer called it the "master of ceremonies." George Gaylord Simpson thought it to be like a poet or a builder.

But difficulties with the concept of natural selection soon developed, not the least of which was the fact that natural selection turned out to be little more than a tautology (viz., it is based on circular reasoning; like saying "deafness causes loss of hearing")—something that observant evolutionists themselves have openly acknowledged for many years. T.H. Morgan, the eminent geneticist and pioneer of fruit-fly research, seems to have been one of the first to spot the problem. He wrote early in the twentieth century that "it may be little more than a truism to state that the individuals that are best adapted to survive have a better chance of surviving than those not so well adapted to survive" (as quoted in Bethell, 1976).

Because of the obviously tautological nature of classical Darwinian natural selection (i.e., "survival of the fittest"), the position eventually was redefined as "differential reproduction"—an interpretation that first became popular in the 1950s under the name of "neo-Darwinism." In the neo-Darwinian view, natural selection does not merely select for animals that survive, but for animals that leave the most offspring.

[INTERESTING SIDE NOTE: Darwin viewed nature as a hostile place where there was an overproduction of animals but a limited supply of food. He believed that such conditions led to a vicious struggle for existence—in which only the fittest could survive. This, of course, is why natural selection came to be defined as "survival of the fittest." But take just a moment to think through this line of reasoning. According to Darwin, the reason evolution worked in the first place is that animals leave too many offspring—so there is not enough food to feed them. Yet now we are told by neo-Darwinists that the animals that leave the most offspring will be the ones that ensure the continued survival of their species! Ironically, some neo-Darwinists (like Paul Ehrlich, for example) are now vociferously insisting that we humans must severely limit the number of our offspring if our species is to survive. Go figure!]

British evolutionist Francis Hitching observed, however, that "Darwinism, as Darwin wrote it, could be simply but nonsensically stated: **survivors survive**. Which is certainly a tautology; and tells us nothing about how species originate, as even Darwin's supporters admit" (1982, p. 84, emp. added). Dr. Hitching even went so far as to note that "a tautology (or truism) is a self-evident, circular statement empty of meaning, such as 'Darwin was a man,' or 'biology is studied by biologists.' The trouble with natural selection (and survival of the fittest) is that it seems to fall into this category" (p. 84, parenthetical items in orig.).

Some well-known evolutionists have been trying for years to get their colleagues to concede that natural selection is a tautology. "Somehow," natural selection is supposed to ensure the "survival of the fittest," but the only pragmatic way to define the "fittest" is (you guessed it!): "those that survive." At a professional symposium on Neo-Darwinism, geneticist C.H. Waddington of Edinburgh University opined,

> "The theory of neo-Darwinism is a theory of the evolution of the changing of the population in respect to leaving offspring and not in respect to anything else. Nothing else is mentioned in the mathematical theory of neo-Darwinism. It is smuggled in, and everybody has in the back of his mind that the animals that leave the largest number of offspring are going to be those best adapted also for eating peculiar vegetation, or something of this sort; but this is not explicit in the theory. All that is explicit is that they will leave more offspring. **There, you do come to what is, in effect, a vacuous statement: Natural selection is that some things leave more offspring than others; and you ask, which leave more offspring than others; and it is those that leave more offspring; and there is nothing more to it than that**. The

> whole guts of evolution—which is, how do you come to have horses and tigers and things—is outside the mathematical theory" (as quoted in Moorhead and Kaplan, 1967, p. 14, emp. added).

Waddington is not alone in his assessment of the serious problems facing evolution as a result of natural selection having been shown to be a circular argument. G.A. Peseley joined the ranks of those criticizing natural selection as evolution's mechanism when he stated,

> "One of the most frequent objections against the theory of natural selection is that it is a sophisticated tautology. Most evolutionary biologists seem unconcerned about the charge and make only a token effort to explain the tautology away. The remainder, such as Professors Waddington and Simpson, will simply concede the fact. For them, natural selection is a tautology which states a heretofore unrecognized relation: the fittest—defined as those who will leave the most offspring—will leave the most offspring.
>
> "What is most unsettling is that some evolutionary biologists have no qualms about proposing tautologies as explanations. One would immediately reject any lexicographer who tried to define a word by the same word, or a thinker who merely restated his proposition, or any other instance of gross redundancy; yet no one seems scandalized that men of science should be satisfied with a major principle which is no more than a tautology" (1982, 38:74).

Arthur Koestler, vitalist philosopher and author, incisively described the tautology of natural selection in these words:

> "Once upon a time, it all looked so simple. Nature rewarded the fit with the carrot of survival and punished the unfit with the stick of extinction. The trouble only started when it came to defining fitness.... Thus natural selection looks after the survival and reproduction of the fittest, and the fittest are those which have the highest rate of reproduction.... We are caught in a circular argument which completely begs the question of what makes evolution evolve" (1978, p. 170).

Yet, as Harvard-trained lawyer Norman MacBeth observed, "In the meantime, the educated public continues to believe that Darwin has provided all the relevant answers by the magic formula of random mutations plus natural selection—quite unaware of the fact that random mutations turned out to be irrelevant and natural selection a tautology" (1982, 2:18). James E. Lloyd, editor of the *Florida Entomologist*, condemned evolution with faint praise (while simultaneously attempting to prop up its alleged factuality) when he wrote,

> "Natural selection, though it may be tautological and philosophically a poor theory in the various ways it is usually stated (e.g., 'survival of the fittest'), and perhaps not even capable of being falsified, is nevertheless profound and axiomatic. It provides the most useful insight for problem solving that biological science has, and is the heart and soul of behavioral ecology" (1982, 65:1, emp. added).

The problem for natural selection, however, does not end there. In fact, it gets even more serious. As Stephen J. Gould observed, "The essence of Darwinism lies in a single phrase: natural selection is the creative force of evolutionary change. No one denies that selection will play a negative role in eliminating the unfit. Darwinian theories require that it create the fit as well" (1977, 86[6]:28). Unfortunately, **creating the fit is the one thing natural selection cannot do**. As renowned Dutch botanist Hugo deVries put it, "Natural selection may explain the **survival** of the fittest, but it cannot explain the **arrival** of the fittest" (1905, pp. 825-826). Henry Gee (chief science writer at *Nature*) admitted that "we also have good reason to suspect that to use natural selection to explain long-term trends in the fossil record may not be a valid exercise, because **natural selection is a random, undirected process**, unlikely to work in the same direction for long" (1999, p. 127, emp. added). Evolutionist Richard Lewontin, writing in a special issue of *Scientific American* devoted entirely to evolution, wrote that "natural selection over the long run does not seem to improve a species' chance of survival but simply enables it to 'track,' or keep up with, the constantly changing environment" (1978, [3]:212-230). The late British paleontologist Colin Patterson placed the matter in its proper focus when he commented that "…most of the current argument in neo-Darwinism is about this question: how a species originates. And it is there that natural selection seems to be fading out, and chance mechanisms of one sort or another are being invoked" (as quoted in Sunderland, 1982).

Speaking of "chance mechanisms of one sort or another being invoked" bring to mind David Quammen's explanation (in his November 2004 feature article in *National Geographic*) of how natural selection supposedly works. Quammen wrote,

> "The gist of the concept is that small, random, heritable changes among individuals result in different chances of survival and reproduction—success for some, death without offspring for others—and that this natural culling leads to significant changes in shape, size, strength, chemistry, and behavior among the descendants. Excess population growth drives the competitive struggle. Because less successful competitors produce fewer surviving offspring, the useless or negative variations tend to disappear, whereas the useful variations tend to be perpetuated and gradually magnified throughout a population" (Quammen, 2004, 206[5]:8).

An observant reader who took the time to digest Mr. Quammen's article would immediately have picked up on what he was trying to do. Like a skilled magician who knows exactly how and when to use sleight-of-hand tricks, Quammen employed natural selection as a mechanism for **macro**evolutionary processes ("significant changes in shape, size, strength, chemistry, and behavior"), but suggested as "proof" **only** examples of **micro**evolution. With impressive, full-color photographs, Quammen offered up the tired old argument of Darwin's finches (pp. 26-27,30) as a demonstration of natural selection, citing specifically the scientific studies of Peter Grant from Princeton University who, with his wife Rosemary, has spent the past several decades observing changes in finches' beaks on the Galápagos Islands (p. 30). Yet anyone familiar with the Grants' research knows full well that all they "discovered" was that finches' beaks change. Perhaps this would be a good time to quote once again the definition of macroevolution provided by Harvard's Ernst Mayr when he wrote that the term refers to "**evolution above the species level**" (2001, p. 287, emp. added). **The finches that the Grants studied always remained finches**. They did not turn into something else. Furthermore, when David Quammen referred to variation among dogs (pp. 16-17), he provided another good example of **micro**evolution, but did nothing to establish the factuality of **macro**evolution.

Or, consider another example mentioned in almost all biology textbooks as observable evidence of natural selection—the peppered moth (*Biston betularia*). Peppered moths of that genus and species range in color from mostly white (with a peppering of black specks) to practically all black. At one time in history, the lighter-colored moths were the most numerous because they blended in well with the light-colored bark of the trees they favored. Thus, they were just about invisible to birds (their main predator). But when air pollution from the Industrial Age caused the bark of the trees to darken, that made the lighter moths easier for the birds to see (and eat!). The birds therefore ate the more-visible white variety of moths, leaving behind mostly the darker variety of the species that were camouflaged on the soot-covered trees. To the evolutionist, this represents observable evidence of evolution in action. But while the peppered moths may well be a good example of natural selection, they do not show the evolution of a fundamentally new kind of animal—or, for that matter, even a new species of moth. If such examples are the best that evolutionists have to offer, then their theory is in far worse trouble than they seem to realize.

Or, consider another example—our old friend *Canis familiaris* (the common dog). The American Kennel Club recognizes roughly 150 varieties of dogs that range from the magnificent St. Bernard (which can weigh more than 100 pounds) to the tiny Chihuhua (which can weigh less than 3 pounds). **Yet all 150 or so varieties are the same genus and species**—*Canis familiaris*! Dog breeders have learned that they can breed for things like long legs or short legs (within limits, of course) or long hair or short hair. But they cannot turn a dog into something that uses wings to fly. The reason is that the dog's gene pool does not contain the genes necessary to produce wings (or any of the other numerous specializations required to make flight possible).

Scientists have discovered the genetic parameters that make it possible for creatures within the same species to exhibit an incredible range of variation—yet without changing into a different species (much less a different genus!). Modern-day genetic analysis has shown that the individuals of a species do not share an identical set of genes (except in the case of clones), but instead have a small number of alternative versions of genes known as *alleles*. This is why we say that the individuals of a certain species comprise a *gene pool* from which selection (either artificial or natural) can occur. The important point, of

course, is that natural selection cannot "select" genes that are not within the gene pool of the species (and that might cause us to end up with such things as flying dogs, to choose just one obvious example).

Non-evolutionists have *never* objected to the idea of natural selection as a mechanism for eliminating the unfit, non-adapted organisms. As a matter of fact, non-evolutionists prior to Darwin were advocating natural selection as a conservation principle. Few people are aware, apparently, that natural selection was not Charles Darwin's discovery. A non-evolutionist zoologist/chemist by the name of Edward Blyth (1810-1873) wrote about it in the years between 1835 and 1837, well before Darwin. Some evolutionists, like the late Loren Eiseley (Benjamin Franklin Professor of Anthropology and History of Science at the University of Pennsylvania), even have gone so far as to question the incredible similarity between Blyth's essays and those of Charles Darwin (1959), hinting at plagiarism on Darwin's part. Eiseley wrote that "the leading tenets of Darwin's work—the struggle for existence, variation, natural selection, and sexual selection—are all fully expressed in a paper written by Blyth in 1835" (1979, p. 55). That fact has not been lost on non-evolutionists. Ian Taylor, in his book, *In the Minds of Men*, discussed Darwin's reading of Patrick Matthew's 1831 essay, *Naval Timber and Arboriculture*, which in its appendix contained the phrase "this natural process of selection"—a phrase that Darwin changed slightly to "natural means of selection" and incorporated into his very first essay, published in 1842 (1984, p. 125).

As a screening device for eliminating the unfit, natural selection represents a good plan for preventing harmful mutations from affecting and even destroying the entire species. Furthermore, to employ an old adage, that which says too much says nothing at all. The long neck of the giraffe and the short neck of the hippopotamus are both explicable by natural selection, as are both the dull coloration of the peppered moth and the brilliant colors of the bird of paradise. Natural selection "explains" everything, and therefore really explains nothing. It cannot create new genera, families, phyla, etc. Nor can it explain adaptation. The fact that an organism **is** adapted to its environment tells us absolutely nothing about how it **came to be** adapted. Any organisms not so adapted would not have survived, but this constitutes no proof that those organisms that did survive possessed adaptations produced by evolution. Yet Gould has admitted that natural selection must be able to "create the fit" if it is to be deemed successful in an evolutionary scenario. **This, it cannot do**. And it certainly cannot explain the vast complexity of life around us. Tautologous arguments are not equipped with the power to "explain" such, much less "create" such. As Swedish biologist Søren Løvtrup wrote (mincing no words!):

> "After this step-wise elimination, only one possibility remains: **the Darwinian theory of natural selection**, whether or not coupled with Mendelism, **is false**. I have already shown that the arguments advanced by the early champions were not very compelling, and that there are now considerable numbers of empirical facts which do not fit with the theory. Hence, **to all intents and purposes the theory has been falsified**, so why has it not been abandoned? I think the answer is that current evolutionists follow Darwin's example—they refuse to accept falsifying evidence" (p. 352, emp. added).

Enough said.

[**AUTHOR'S ADDENDUM**: With the rebuttal of my opponent's proposed arguments for evolution now complete, I would like to offer the following comments regarding the importance of the matters that are being discussed within the context of this debate.]

Non-evolutionists happily affirm it, while evolutionists (on occasion) begrudgingly concede it: Everything designed has a designer. As atheist Paul Ricci said in his book on logic, *Fundamentals of Critical Thinking*, "...it's true that everything designed has a designer.... 'Everything designed has a designer' is an analytically true statement" (1986, p. 190). Design, at least in part, has to do with the arrangement of individual components within an object so as to accomplish a functional or artistic purpose. An automobile contains design because its many units, engineered and fitted together, result in a machine that facilitates transportation. A beautiful portrait evinces design when paints of various colors are combined, by brush or knife upon canvas, so as to effect an aesthetic response. Rational individuals instinctively recognize the presence of design—for which there are multiplied thousands of examples within the Universe that we inhabit.

Adding to the force of this argument is the principle known as *a fortiori* reasoning. If something is said to follow in an *a fortiori* fashion, it means that the conclusion can be reached with an even greater logical necessity than another conclusion already accepted. Both a pair of pliers and a computer are tools. If one admits that it took a designer to make the pliers (a conclusion that no rational person would deny), it follows with even greater force that a designer must have been required to make the computer, since the computer is much more complicated than the pliers. Using *a fortiori* reasoning, it can be established that if the lesser (the pliers) requires a designer, then the greater (the computer) **absolutely demands a designer**. Again, this is simple logic.

In this rebuttal, I have had cause to mention such things as the amazing intricacy of bodily organs such as the brain and the eyes, the incredibly complex DNA/RNA genetic code, and numerous other such items. The design inherent in the Universe itself, as well as in the living things that it contains, cannot be explained away or easily (or logically) ignored. My evolutionary opponent in this debate would have us believe that the Universe, plants, animals, and man were conceived accidentally by "Father Chance," and then birthed coincidentally by "Mother Nature." The evidence presented here, however, shows such a conclusion to be as incorrect as it is vacuous. As atheist Ricci also candidly admitted, "...either a divine being exists or he does not; there are no third possibilities, regardless of what the skeptic or agnostic says" (p. 140).

Furthermore, as prominent humanist author Martin Gardner explained in a chapter titled "The Relevance of Belief Systems" in his book, *The New Age: Notes of a Fringe Watcher*, **it does matter what a person believes** (1988, pp. 57-64). This is especially true when it comes to the matter of origins, since in this area we are dealing with complete cosmogonies (i.e., entire world views). Consider the following in this regard.

Anthropologist Jonathan Marks made the following statement in his book, *What It Means to be 98% Chimpanzee*: "**The question of who and what you are is not trivial**" (emp. added). The context in which he made that statement, however, is as important as the statement itself. Here, from his book, are the comments immediately preceding that sentence:

> "Science gives us authoritative ideas about kinship, which force us to reconceptualize our place in the order of things, which is by that very fact disorienting. But it doesn't stick around to explain it to us, to reintegrate us, to give new meaning to our existence. That's the problem with Darwinian theory, of course. It tells us our ancestors were kin to apes, the products of eons of ordinary biological processes of survival and reproduction, and not merely zapped into existence in the Garden of Eden, **but it doesn't tell us what that means or what to do about it**. ***It just walks away from the wreckage***" (2002, p. 222, first emp. in orig., second emp. and italics added).

What "wreckage," exactly, does Dr. Marks have in mind? Let Richard Dawkins, the renowned evolutionist of Oxford University, answer that question. In the 1989 edition of his highly acclaimed 1976 book, *The Selfish Gene*, Dawkins wrote, "**My own feeling is that a human society based simply on the gene's laws of universal ruthless selfishness would be a very nasty society in which to live**. But unfortunately, however much we may deplore something, it does not stop it being true" (pp. 2-3, emp. added). Six years later, in his book, *River Out of Eden*, he continued in the same vein when he wrote,

> "[I]f the universe were just electrons and selfish genes, meaningless tragedies...are exactly what we should expect, along with equally meaningless **good** fortune. Such a universe would be neither evil nor good in its intention. It would manifest no intentions of any kind. In a universe of electrons and selfish genes, blind physical forces and genetic replication, some people are going to get hurt, other people are going to get lucky, and you won t find any rhyme or reason in it, nor any justice. The universe that we observe has precisely the properties we should expect if there is, at bottom, no design, no purpose, no evil and no good, nothing but pitiless indifference" (1995, pp. 132-133, emp. in orig.).

Nobel laureate Steven Weinberg referred to a similar "pitiless indifference" in his classic book on the origin of the Universe, *The First Three Minutes*, when he lamented:

> "It is almost irresistible for humans to believe that we have some special relation to the universe, that human life is not just a more-or-less farcical outcome of a chain of accidents reaching back to the first three minutes, but that we were somehow built in from the beginning. ...It is very hard to realize that this all is

> just a tiny part of an overwhelmingly hostile universe…[which] has evolved from an unspeakably unfamiliar early condition, and faces a future extinction of endless cold or intolerable heat. **The more the universe seems comprehensible, the more it also seems pointless**" (1977, pp. 150,155, emp. added).

Harvard's late, renowned evolutionist, George Gaylord Simpson, argued that "man is the result of a purposeless and materialistic process that did not have him in mind," and therefore concluded, "Discovery that the universe apart from man or before his coming lacks and lacked any purpose or plan has the inevitable corollary that the workings of the universe cannot provide any automatic, universal, eternal, or absolute ethical criteria of right and wrong" (1967, p. 346). Millard Erickson, in his book, *Does It Matter What I Believe?*, wrote that there are numerous reasons

> "...why having correct beliefs is important. Our whole lives are inevitably affected by the real world around us, so what we believe about it is of the utmost importance.... What we believe about reality does not change the truth, nor its effect upon us. Correct belief, however, enables us to know the truth as it is, and then to take appropriate action, so that it will have the best possible effect upon our lives. Having correct beliefs is also necessary because of the large amount and variety of incorrect beliefs which are about" (1992, pp. 12, 13).

It is because of the "large amount and variety of incorrect beliefs which are about," and because I truly believe that it **does** matter what a person believes, that I agreed to participate in this debate on origins. In my estimation, a significant part of the "Darwinian wreckage" has to do with the fact that many people have come to believe, as Richard Leakey and Roger Lewin put it in their book, *Origins* (1977, p. 256), that "…no matter how special we are as an animal," the Universe nevertheless remains (to use Weinberg's one-word assessment) "pointless." As a result, the lives of such individuals are filled with "pitiless indifference"—because they have accepted as correct Charles Darwin's conclusion that "there is no fundamental difference between man and the higher mammals" (as quoted in Francis Darwin, 1898, 1:64). How sad is that?

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