A CRITIQUE OF "29 EVIDENCES FOR MACROEVOLUTION" PART 4
By Ashby L. Camp

PREDICTION 17: FUNCTIONAL MOLECULAR EVIDENCE -- PROTEIN FUNCTIONAL REDUNDANCY

Before the advent of DNA sequencing technology, the amino acid sequences of proteins were used to establish the phylogenetic relationships of species. Sequence studies with functional genes have centered on genes of proteins (or RNAs) that are ubiquitous (i.e. all organisms have them). This is done to insure that the comparisons are independent of the overall species phenotype. . . .

Cytochrome c is an essential and ubiquitous protein found in all organisms, including eukaryotes and bacteria (Voet 1995, p. 24). The mitochondria of cells contain cytochrome c, where it transports electrons in the fundamental metabolic process of oxidative phosphorylation. The oxygen we breathe is used to generate energy in this process (Voet 1995, pp. 577-582).

Using a gene like this, there is no reason to assume that the protein sequence should be the same, unless the two organisms are genealogically related. This is due in part to the functional redundancy of protein sequences and structures. . . .

Only about a third of the 100 amino acids in cytochrome c are necessary to specify its function. Most of the amino acids in cytochrome c are hypervariable (i.e. they can be replaced by a large number of functionally equivalent amino acids) (Dickerson and Timkovich 1975). Most importantly, Hubert Yockey[35] has done a careful study in which he calculated that there are a minimum of 2.3×10^{93} possible functional cytochrome c protein sequences, based on several exhaustive genetic mutational analyses (Hampsey 1986; Hampsey 1988; Yockey 1992, Ch. 6, p. 254). . . . Thus, functional cytochrome c sequences are virtually unlimited in number, and there is no a priori *reason for two different species to have the same, or even mildly similar, cytochrome c protein sequences.*

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then ubiquitous proteins with high functional redundancy will have the same or a similar amino acid sequence in two or more species.

2. Ubiquitous proteins with high functional redundancy have the same or a similar amino acid sequence in two or more species.

[35] This is the same Yockey who concluded (in the very work cited), "The origin of life by chance in a primeval soup is impossible in probability in the same way that a perpetual motion machine is impossible in probability." (Yockey, 257.) The origin of life, however, is beyond the scope of Dr. Theobald's paper.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of neo-Darwinism that ubiquitous proteins with high functional redundancy will have the same or a similar amino acid sequence in two or more species. Evolution can accommodate this phenomenon, but it can also accommodate its absence. If the amino acid sequence in such a protein was not the same or "similar" in two or more species, evolutionists simply would vary the time of divergence and/or the mutation rate, which is claimed to vary for different proteins, to account for the differences. Since neither universal common ancestry nor neo-Darwinism predicts this phenomenon, they cannot be falsified by its absence or confirmed by its presence.

The real argument being made here is theological, not scientific. The claim is that, since God could make cytochrome c with countless arrangements of amino acids, he would not have used an identical or similar series of amino acids in the cytochrome c of separately created species. Of course, even if that were true, it would not establish the claim of universal common ancestry (because, as pointed out above, divine creation is not the only theoretical alternative to universal common ancestry). But more importantly, the claim combines an uncertain factual premise with an unprovable theological assertion.

The allegation that God could employ countless arrangements of amino acids in the construction of cytochrome c (or other proteins) ignores the possibility that the gene coding for cytochrome c may also be involved in the production of numerous other proteins. As noted previously, this possibility was discovered through the recent sequencing of the human genome. Though humans may have as many as 300,000 proteins, they have only about 30,000 genes (see footnote 17). As J. Craig Venter of Celera Genomics explained in the press conference announcing the sequencing of the human genome:

> [O]ur understanding of the human genome has changed in the most fundamental ways. The small number of genes -- some 30,000 -- supports the notion that we are not hard wired. We now know the notion that one gene leads to one protein, and perhaps one disease, is false.
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> One gene leads to many different protein products that can change dramatically once they are produced. We know that some of the regions that are not genes may be some of the keys to the complexity that we see in ourselves. We now know that the environment acting on our biological steps may be as important in making us what we are as our genetic code. (Bethell, 52.)

When asked immediately after the press conference about Venter's suggestion that one gene could give rise to ten proteins, James Watson (of DNA fame) said, "Some genes can give rise to 50 different proteins." (Bethell, 56.) As summed up by the Washington Post, "The way these genes work must therefore be far more complicated than the mechanism long taught." (Bethell, 52.)

If the gene for cytochrome c, for example, does more than code for that particular protein, then its other functions may influence the order of its codons and thus influence the order of amino acids in cytochrome c. Without knowing all that a gene does within an organism and how it accomplishes those functions, one cannot know the gene's design constraints and therefore cannot know the corresponding constraints on amino acid sequences.

Indeed, given the high degree of functional redundancy in the amino acid sequences of cytochrome c, one wonders why those sequences would have been conserved for tens of millions of years by conventional reckoning. For example, humans and rhesus monkeys supposedly diverged from a common ancestor as long as 50 million years ago,[36] but the only difference in their cytochrome c is at position 66, which is isoleucine in humans and threonine in rhesus monkeys. This strong conservation may mean that the gene for cytochrome c is subject to needlessly efficient error correction, but it also may mean that the gene is performing unknown functions that are responsible for or contribute to its conservation.

But even if there were no unknown design constraints on the gene for cytochrome c, how could one be sure that God would not conserve amino acid sequences (or the underlying codons) when creating cytochrome c in separate species? After creating cytochrome c in the first organism, it certainly is conceivable that he would make changes to that blueprint only when necessary for his purpose. In other words, the default in this instance may be similarity rather than dissimilarity. There is no basis for demanding that God introduce novelty for novelty's sake.

From that perspective, it is the differences in cytochrome c that need to be explained, not the similarities. One creationist explanation for those differences is that various cytochrome c molecules were created differently for functional reasons and then diverged further as a result of mutations (whereas the evolutionist attributes the differences entirely to mutation). To repeat a quote from Brand:

> An alternative, interventionist hypothesis is that the cytochrome c molecules in various groups of organisms are different (and always have been different) for functional reasons. Not enough mutations have occurred in these molecules to blur the distinct groupings evident in Fig. 10.1 [the cytochromes percentage of sequence difference matrix]. . . . If we do not base our conclusions on the *a priori* assumption of megaevolution, all the data really tell us is that the organisms fall into nested groups without any indication of intermediates or overlapping groups, and without indicating ancestor/descendant relationships. The evidence can be explained by a separate creation for each group of organisms represented in the cytochrome c data. (Brand, 158-159.)

[36] Encyclopedia Britannica (online edition, in the "molecular evolution" subsection of the "evolution" article) states, "[B]etween humans and rhesus monkeys, who diverged from their common ancestor 50,000,000 to 40,000,000 years ago, [cytochrome c] differs by only one amino-acid replacement." Others would place the split at 30 million years ago or less.

Under this view, the similarities of cytochrome c within groups of organisms are the result of similarities in the biochemistry of those organisms. To use Dr. Theobald's example, the cytochrome c of bats and porpoises is more like that of humans than like that of hummingbirds and sharks, respectively, because the originally created ancestors of these mammals benefited from similar changes to the cytochrome c blueprint.

Dr. Theobald denies implicitly that the originally created ancestors of these mammals could have benefited from similar changes to a cytochrome c blueprint.[37] In his view, if the pattern of similarities in amino acid sequences were attributable to functional considerations, then "we would expect to observe a pattern of sequence similarity correlating with similarity of environment or with physiological requirement." Thus, a bat's cytochrome c sequence should be more like that of a hummingbird than that of a human and a porpoise's sequence should be more like that of a shark than that of a human, neither of which is the case.

The problem is that we do not know every physiological function of an organism for which cytochrome c's performance may be relevant. We thus cannot be certain that the originally created ancestors of bats, porpoises, and humans did not share a physiological function independent of environment and lifestyle for which similar changes to the cytochrome c blueprint would be beneficial. As the recent discovery about genes reminds us, biology is often vastly more complex than we assume.

Moreover, even if the pattern of similarities in cytochrome c could not be attributed to functionally related differences in the original cytochrome c of various groups of organisms, there could be other divine reasons for the pattern. If, for example, ReMine is correct that nested hierarchy is a crucial aspect of the Creator's biotic message, then one would expect that nesting to be expressed at the biochemical as well as the morphological level.

Dr. Theobald repeats the overstatement from Prediction 3 that "the phylogenetic tree constructed from the cytochrome c data *exactly recapitulates* the relationships of major taxa as determined by the completely independent morphological data" (emphasis supplied). I addressed some of the problems with the cytochrome c phylogeny in the discussion of Prediction 3.

The suggestion that the hypothesis of universal common ancestry would be falsified if cytochrome c sequences were "very different from each other" is incorrect. As

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[37] Dr. Theobald addresses the issue in response to an anticipated creationist counter-argument that the sequence similarities of the amino acids are necessary for functional reasons. That is not my argument. I assume the amino acids in cytochrome c have a high functional redundancy, which means that, for the most part, specific sequences are not necessary for the functioning of the protein. Rather, as I indicate, the similarities may be explained by unknown design constraints on the gene, by a default of similarity rather than dissimilarity, and/or by divine purposes unrelated to function, such as the sending of a biotic message. Sequence similarity relates to function only in the claim that at creation similar organisms may have benefited from similar changes to the cytochrome c blueprint.

stated already, if the amino acid sequence in such a protein was not the same or similar in two or more species, evolutionists simply would vary the time of divergence and/or the mutation rate to account for the differences. For example, studies have shown that there are many differences in the proteins of two very similar frog species (Spetner, 69), and no one has abandoned the evolution paradigm because of it.

PREDICTION 18: FUNCTIONAL MOLECULAR EVIDENCE -- DNA CODING REDUNDANCY

Like protein sequence similarity, the DNA sequence similarity of two ubiquitous genes also implies common ancestry. Of course, comprehensive DNA sequence comparisons of conserved proteins such as cytochrome c also indirectly take into account amino acid sequences, since the DNA sequence specifies the protein sequence. However, with DNA sequences there is an extra level of redundancy. The genetic code itself is informationally redundant; on average there are three different codons (a codon is a triplet of DNA bases) that can specify the exact same amino acid (Voet 1995, p. 966). Thus, for cytochrome c there are approximately 3^{104}, or over 10^{49}, different DNA sequences (and, hence, 10^{49} different possible genes) that can specify the exact same protein sequence.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then ubiquitous genes will have the same or a similar codon sequence in two or more species.

2. Ubiquitous genes have the same or a similar codon sequence in two or more species.

Since this is the concept of functional redundancy applied to codons, much of the preceding response is applicable. It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of neo-Darwinism that ubiquitous genes will have the same or a similar codon sequence in two or more species. Evolution can accommodate this phenomenon, but it can also accommodate its absence.

If the codon sequence in such a gene was not the same or "similar" in two or more species, evolutionists simply would vary the time of divergence and/or the mutation rate, which is claimed to vary for different genes, to account for the differences. This is another example of taking a known pattern of life, claiming that pattern as a *prediction* of evolution, and then using the fact the pattern fits the prediction as evidence for the truth of evolution.

Once again, the real argument being made is theological, not scientific. The claim is that, since God could make a gene for a protein with many different codon sequences, he would not have used an identical or similar series of codons in the cytochrome c gene of separately created species. Of course, even if that were true, it would not establish the claim of universal common ancestry (because divine creation is not the only theoretical

alternative to universal common ancestry). But more importantly, the claim combines an uncertain factual premise with an unprovable theological assertion.

As explained above, the allegation that God could employ countless arrangements of codons in the gene for cytochrome c (or other proteins) ignores the recently discovered possibility that the gene may also be involved in the production of numerous other proteins. The gene thus may be subject to design constraints of which we are ignorant.

Indeed, given the functional redundancy of codons and the functional redundancy of the amino acids in cytochrome c, one wonders why only one codon difference has arisen in the five to eight million years since humans and chimps allegedly diverged from a common ancestor. This strong conservation may mean that the gene is subject to needlessly efficient error correction, but it also may mean that the gene is performing unknown functions that are responsible for or contribute to its conservation.

But even if there were no unknown design constraints on the gene for cytochrome c, how could one be sure that God would not conserve codon sequences when creating cytochrome c gene in separate species? After creating the cytochrome c gene in the first organism, it certainly is conceivable that he would make changes to that blueprint only when necessary for his purpose. In other words, the default in this instance may be similarity rather than dissimilarity. Again, there is no basis for demanding that God introduce novelty for novelty's sake.

From that perspective, it is the differences in the cytochrome c gene that need to be explained, not the similarities. One creationist explanation for those differences is that various cytochrome c genes were created differently for functional reasons and then diverged further as a result of mutations (whereas the evolutionist attributes the differences entirely to mutation.)

Under this view, the similarities of cytochrome c genes within groups of organisms are the result of similarities in the biochemistry of those organisms. To use Dr. Theobald's prior example, the cytochrome c gene of bats and porpoises may be more like that of humans than like that of hummingbirds and sharks, respectively, because the originally created ancestors of these mammals benefited from similar changes to the cytochrome c gene blueprint.

And even if the pattern of similarities in cytochrome c genes could not be attributed to functionally related differences in the original genes of various groups of organisms, there could be other divine reasons for the pattern. If, for example, ReMine is correct that nested hierarchy is a crucial aspect of the Creator's biotic message, then one would expect that nesting to be expressed at the biochemical as well as the morphological level.

Thus, the similarity of codon sequences in the cytochrome c gene of humans and chimps does not "make it look exactly like we are genealogically related." That conclusion only follows if one ignores the possibility of unknown design constraints,

insists that God introduce novelty for novelty's sake, and denies that there could be other divine purposes, such as sending a biotic message, for the pattern of similarity.

PREDICTION 19: NONFUNCTIONAL MOLECULAR EVIDENCE -- TRANSPOSONS

Transposons are very similar to viruses. However, they lack genes for viral coat proteins, cannot cross cellular boundaries, and thus they replicate only in the genome of their host. They can be thought of as intragenomic parasites. Except in the rarest of circumstances, the only mode of transmission from one metazoan organism to another is directly by DNA duplication and inheritance (e.g. your transposons are given to your children) (Li 1997, pp. 338-345).

Replication for a transposon means copying itself and inserting the copied DNA randomly somewhere else in the host's genome. . . .

Finding the same transposon in the same chromosomal location in two different species is strong direct evidence of common ancestry, since they insert randomly and generally cannot be transmitted except by inheritance. In addition, once a common ancestor has been postulated that contains this transposition, all the descendants of this common ancestor should also contain the same transposition. A possible exception is if this transposition were removed due to a rare deletion event; however, deletions are never clean and usually part of the transposon sequence remains.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the same transposon will exist in the same chromosomal location in two or more species.

2. The same transposon exists in the same chromosomal location in two or more species.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of neo-Darwinism that the "same transposon"[38] 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, McClintock's initial claims about transposons were resisted because they were contrary to the prevailing view of genetics.

[38] A transposon is "[a] mobile genetic element, known informally as a 'jumping gene,' that can become integrated at many different sites in the genome, either by moving from place to place or by producing copies of itself that insert elsewhere in the genome." (Martin and Hine, 600.) This category includes satellites, various retrotransposons (including LINEs and SINEs), retroviruses, and DNA transposons. (See, Walkup, 21-25.) Transposons need not be identical but only "sufficiently similar" to be considered the same.

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.

Moreover, transposons are inadequate in principle to support Dr. Theobald's claim of universal common ancestry, because they are not shared by all groups of organisms. As Edward Max acknowledges (in Sec. 4.7 of the online article cited by Dr. Theobald), "Another limitation [of this argument] is that there are no examples of 'shared errors' that link mammals to other branches of the genealogic tree of life on earth. . . . Therefore, the evolutionary relationships between distant branches on the evolutionary genealogic tree must rest on other evidence besides 'shared errors.'"

The claim here is that common ancestry is the only viable explanation for the same transposon being at the same locus in separate species. It is based on the premise that transposons are (and always have been) nonfunctional products of genetic accidents that insert randomly into the genome of the host organism. The presumed nonfunctionality of transposons is thought to eliminate the explanation of design (because a Designer could have no purpose in placing nonfunctional sequences at the same locus in separate species). The presumed randomness of transposon insertion is thought to eliminate the explanation of chance (because the DNA "chain" is too long for coincidental insertion at the same locus to be a realistic possibility). That leaves common ancestry as the last explanation standing.

Two considerations undermine this claim. First, it is an unprovable theological assertion that God would not place nonfunctional sequences at the same locus in separate species. God may have a purpose for doing so that is beyond our present understanding. Gibson writes:

> The argument that God would not act in a certain way is a theological argument, and can hardly be addressed by science. The validity of such an argument depends on the kind of God being postulated. The kind of God at issue for most of those involved in this discussion is the God who revealed Himself in the Bible. The question then is: What do the scriptures say about whether God would create structures or DNA sequences for which we can find no use in unrelated organisms? This subject is not addressed in the Bible, leaving us without an answer. We can postulate that God would not do such a thing, but this position would not be based on any evidence other than our own presuppositions, however reasonable they seem. (Gibson, 100.)

The suggestion that God *could not* place nonfunctional sequences at the same locus in separate species because that would make him guilty of deception is patently theological. It is also incorrect. God cannot be charged fairly with deception when we choose to draw conclusions from data that contradict what he has revealed in Scripture. To quote Gibson again:

> The Scriptures do state clearly that God does not deceive us (Titus 1:2), but they also make it clear that we are naturally prone to make wrong conclusions (Romans 11:33-36). The Scriptures reveal the truth about history. When God tells us in Scripture that he created in a certain way, we need not be deceived by what we believe to be appearances to the contrary. (Gibson, 100.)

Second, even the staunchest critics of creation theory recognize that "[i]t is impossible to prove absence of function for any region of DNA."[39] As molecular biologist Carl Schmid puts it, "We know there's a lot of DNA that we don't know its function. The fact that we don't know its function doesn't mean it doesn't have a function."[40] The recent indication from the Human Genome Project that the way genes work is "far more complicated than the mechanism long taught" only increases the possibility that seemingly useless DNA has an unknown function.

The issue of function is, of course, much more complex than determining whether a given sequence codes for a product in a laboratory. To repeat a quote from Jerlstrom:

> Failure to observe a pseudogene coding for a product under experimental conditions is no proof that they never do so inside an organism. It is also impossible to rule out protein expression based solely on sequence information, as DNA messages can be altered by, e.g. editing the transcribed RNA, skipping parts of the sequence, etc. Moreover, the inability to code for a protein useful to an organism hardly exhausts other possible functions pseudogenes may have. (Jerlstrom, 15.)

Walkup says of transposons (and the other major kinds of "junk DNA"), "Recent research has begun to show that many of these useless-looking sequences do have a function." (Walkup, 19.) According to Woodmorappe, who cites a forthcoming paper by Paul Nelson and others, "[E]vidence for function is not limited to generic 'junk DNA', but is now known for representatives of *all* major types of pseudogenes."[41] (Woodmorappe 2000, 57.)

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[39] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 5.4 (available online at http://talkorigins.org/faqs/molgen). Dr. Max believes, however, that nonfunctionality is the only reasonable conclusion that can be drawn for most transposons in light of current scientific evidence.

[40] Edie Lau, "Much DNA just 'junk' -- or is it? Human Genome Project spurs new look at mystery material," *Sacramento Bee* (March 19, 2001) (available online at http://www.sacbee.com/news/news/local01_20010319.html).

Regarding the Alu element cited by Dr. Theobald as an illustrative transposon, Jerlstrom writes, "[T]here is a growing body of evidence that Alu (a SINE) sequences are involved in gene regulation, such as in enhancing and silencing gene activity, or can act as a receptor-binding site -- this is surely a precedent for the functionality of other types of pseudogenes." (Jerlstrom, 15.) Woodmorappe reports that "[t]he functionality of Alu units has long been suspected, and recently confirmed." (Woodmorappe 2000, 57; see also, Walkup, 23.)

Of course, if transposons have a function, then God may have had a functional reason for initially placing them at the same chromosomal location in separately created species. He also may have had a functional reason for designing certain transposons with an insertion bias for certain loci.

As mentioned previously, geneticist Todd Wood proposes that God endowed creatures with mobile genetic elements (which he calls Altruistic Genetic Elements) to facilitate diversification within created kinds (see, Walkup, 26-27).[42] Since the Fall, this complex diversification system is believed to have degenerated so that only remnants and distortions of its past operation are available to us today. If that is correct, the fact we do not see insertion bias in a particular transposon, for example, does not mean that it never existed. And the insertion bias that we do observe in some transposons (see, e.g., Walkup, 25; Woodmorappe 2000, 63-64) may no longer be serving its original purpose.

The evolutionary belief that transposons have remained recognizable for eons supports the view that they are (or have been) functional. Woodmorappe writes, "[O]rthologous SINEs have now been found in different *phyla*, and the cited researchers recognize that the (evolutionary) maintenance of a close correspondence between such phylogenetically-distant organisms is very difficult to explain if SINEs are of no use to their carriers." (Woodmorappe 2000, 58.) To repeat another quote from Jerlstrom:

> The persistence of pseudogenes [including transposons] is in itself additional evidence for their activity. This is a serious problem for evolution, as it is expected that natural selection would remove this type of DNA if it were useless, since DNA manufactured by the cell is energetically costly. Because of the lack of selective pressure on this neutral DNA, one would also expect that 'old' pseudogenes should be scrambled beyond recognition as a result of accumulated random mutations. Moreover, a removal mechanism for neutral DNA is now known. (Jerlstrom, 15.)

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[41] As used in the article, the term pseudogene "encompasses both the classical and retroposited varieties, the latter of which includes interspersed repeats, most notably SINEs and LINEs." (Woodmorappe 2000, 55.)

[42] Woodmorappe mentioned the possible role of transposons in the post-Flood world in *Noah's Ark: A Feasability Study* (Santee, CA: Institute for Creation Research, 1996), 201-202.

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.[43] 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. Indeed, the proximity of Alu elements to genes is accepted as evidence that the Alu elements are *functional*.

> [Eric] Lander [a geneticist at M.I.T.] said that in 1998, Carl Schmid, a molecular biologist at the University of California at Davis, advanced what seemed like a nutty idea to explain Alu's unusual affinity for genes. Schmid suggested Alu sequences resided near genes because they weren't junk, but rather a mechanism to help cells repair themselves.
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> With the entire genome map in front of them, showing so many instances of Alu sequences around genes, scientists are beginning to take Schmid seriously. "It looks pretty convincing," [Francis] Collins said.[44]

One need not be a creationist to doubt the claim that shared transposons are sufficient to establish common ancestry. Regarding the very transposons cited by Dr. Theobald as proof of the common ancestry of whales, hippos, and ruminants, noted vertebrate paleontologist Maureen O'Leary recently rebuked Okada for rejecting the possibility that SINEs and LINEs could arise independently in separate lineages (i.e., evolve convergently). Gura reports:

> 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, 232.)

Even more recently, a team of molecular geneticists discovered two "hot spots" where the same SINEs inserted *independently*. They write:

> Vertebrate retrotransposons have been used extensively for phylogenetic analyses and studies of molecular evolution. Information can be obtained

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[43] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 3.

[44] Tom Abate, "Genome Discovery Shocks Scientists," *San Francisco Chronicle* (February 11, 2001) (available online at http://www.sfgate.com/cgi-bin/article.cgi?file=/chronicle/archive/2001/02/11/MN180850.DTL)

> 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 and others, 769.)

PREDICTION 20: NONFUNCTIONAL MOLECULAR EVIDENCE -- PSEUDOGENES

Other nonfunctional molecular examples that provide evidence of common ancestry are pseudogenes. Pseudogenes are very closely related to their functional counterparts (in primary sequence and often in chromosomal location), except that either they have faulty regulatory sequences or they have internal stops that keep the protein from being made. They are functionless and do not affect an organism's phenotype when deleted. Pseudogenes, if they are not vestigial (like the examples in proof 7), are created by gene duplication and subsequent mutation. There are many observed processes that duplicate genes, including transposition events, chromosomal duplication, and unequal crossing over of chromosomes. Like transpositions (c.f. prediction 19), gene duplication is a rare and random event and, of course, any duplicated DNA is inherited. Thus, finding the same pseudogene in the same chromosomal location in two species is strong evidence of common ancestry.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the same pseudogene will exist in the same chromosomal location in two or more species.

2. The same pseudogene exists in the same chromosomal location in two or more species.

Since this is the concept of "shared errors" applied to pseudogenes,[45] much of the preceding response is applicable. It is not a prediction of the hypothesis of universal

[45] Pseudogenes proper are sequences of nucleotides in DNA that resemble a functional gene but which lack one or more of the elements necessary for transcription, i.e., necessary for the sequence information to be transferred to messenger RNA (and ultimately to be synthesized into a protein). Some use the term "pseudogene" more loosely to include other categories of allegedly nonfunctional DNA (such as interspersed repeats). From his comments and examples, Dr. Theobald appears to be referring to classic duplicated pseudogenes rather than processed pseudogenes, which presumably are included in his discussion of transposons.

common ancestry or the more specific hypothesis of neo-Darwinism that the same pseudogene will exist in the same chromosomal location in two or more species. Evolution does not even predict the existence of pseudogenes, much less that they will be found at the same location in two or more species. After all, pseudogenes were not discovered until recently, the first published report being in 1977. (Gibson, 92.) Evolutionary theory managed just fine without them for more than a century. Thus, pseudogenes are not confirmation of an evolutionary prediction but observations that are given an evolutionary explanation.

Moreover, pseudogenes are inadequate in principle to support Dr. Theobald's claim of universal common ancestry, because they are not shared by all groups of organisms. To repeat the quote from Dr. Max, "Another limitation [of this argument] is that there are no examples of 'shared errors' that link mammals to other branches of the genealogic tree of life on earth. . . . Therefore, the evolutionary relationships between distant branches on the evolutionary genealogic tree must rest on other evidence besides 'shared errors.'"

The claim here is that common ancestry is the only viable explanation for "finding the same pseudogene in the same chromosomal location in two species." But classic duplicated pseudogenes "are usually found within clusters of similar, functional sequences on the same chromosome." (Gibson, 93.) That is, they are found close to the genes of which they are believed to be duplicates. So if the same gene (or a member of the gene family) were duplicated independently in separate species, it would not be surprising to find it at the same chromosomal location.

Dr. Theobald apparently considers it too unlikely that the same gene (or a member of the gene family) would be duplicated in separate species because he believes that "gene duplication is a rare and random event." According to Dr. Max, however, the presumed duplication of blocks of sequences has been observed *frequently* in the DNA of a variety of species.[46] Indeed, gene duplication is the most popular explanation for the formation of the new genes believed necessary to fuel evolution, so evolutionary theory is committed to the frequency of the process.

The issue with classic pseudogenes (both singular and duplicated varieties) is not that they have the same chromosomal location in separate species but that they sometimes differ from the presumed original gene in identical ways at identical nucleotide positions. The claim is that identical nucleotide changes could not occur independently, given the random nature of those changes, so they must be the result of common ancestry.

Again, it is an unprovable theological assertion that God would not place the same nonfunctional sequences at the same locus in separate species. He may have a purpose for doing so that is beyond our present understanding. The objection that placing

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[46] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 2.2.1.b.

nonfunctional sequences at the same locus in separate species would make God guilty of deception is ill founded. God cannot be charged fairly with deception when we choose to draw conclusions from data that contradict what he has revealed in Scripture (see Gibson's comments from the preceding section).

But even if one assumes that God would not place the same nonfunctional sequences in different species, it is by no means certain that pseudogenes are nonfunctional. Even the staunchest critics of creation theory recognize that "[i]t is impossible to prove absence of function for any region of DNA."[47] The recent indication from the Human Genome Project that the way genes work is "far more complicated than the mechanism long taught" only increases the possibility that pseudogenes are functioning in some way we do not appreciate.

Back in 1994 Gibson reported that "[s]ome pseudogenes are believed to function as sources of information producing genetic diversity [citations omitted], possibly involving a process similar to gene conversion. It is thought that partial pseudogene sequences are copied into functional genes, producing variants of the functional sequence." (Gibson, 102.) He also noted that "[s]ome pseudogenes have been implicated in gene regulation" [citations omitted]. (Gibson, 103.) Just last year, Petrov and Hartl wrote, "The problem is that generally one does not know whether a pseudogene has any noncoding phenotypic effect and whether the effect is deleterious or advantageous." (Petrov and Hartl, 222.)

Moreover, the "[f]ailure to observe pseudogenes coding for a product under experimental conditions is no proof that they never do so inside an organism." (Jerlstrom, 15.) In fact, there are indications that "some pseudogenes may produce small amounts of polypeptides in specific tissues" [citations omitted]. (Gibson, 101.) Mighell (and others) noted recently that "there are genes that have many features of pseudogenes, but which are functional, and a separate group of genes that are currently considered as pseudogenes, but with the recognition that these genes are potentially functional." (Mighell and others, 113.)

Consider Dr. Theobald's example of the eta globin (psi beta globin) pseudogene. Gibson's description of the beta globin gene cluster is helpful background and gives a hint of the complexity that is involved:

> The beta globin gene cluster consists of five somewhat-similar functional genes and one pseudogene. The five functional genes are arranged on the chromosome in a sequence that corresponds to the sequence of timing of their respective functions during growth and development. The first gene in the series is the "epsilon globin" gene, which helps form hemoglobin molecules early in embryonic development. The second and third genes are called "gamma-G" and "gamma-A." They

[47] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 5.4 (available online at http://talkorigins.org/faqs/molgen). Dr. Max believes, however, that nonfunctionality is the only reasonable conclusion that can be drawn for most transposons in light of current scientific evidence.

help form hemoglobin molecules later during fetal development. The "eta globin" pseudogene is next in sequence, followed by the "delta" globin gene which is produced at a low rate in adults. The last gene in the series is the "beta" globin gene, which produces most of the adult beta globin, and gives the gene family its name. As the adult globin genes become functional, the fetal genes are turned off. The fact that the sequence of the genes of the chromosome matches the sequence of their activity in the developing organism seems unlikely to be the result of chance, and can easily be interpreted as the result of intelligent design. (Gibson, 95.)

Several researchers have suggested that the eta globin pseudogene may function in gene regulation of the beta globin gene family, but that suspicion has not been confirmed. (Gibson, 102.) Gibson writes:

> The fact the eta globin pseudogene is located between the fetal and adult genes suggests that it could play a role in gene switching -- turning off the fetal gamma genes and turning on the adult beta gene. There is evidence that gene switching in human beta globin genes depends in some way on the sequence lying between the fetal and adult genes [citation omitted], although it is not known whether the eta globin sequence itself is involved. Some pseudogenes have been implicated in gene regulation [citations omitted]. Such a role could involve competition for regulatory proteins, production of signal RNA molecules, or perhaps some other mechanism [citation omitted]. (Gibson, 102-103.)

The possibility that the eta globin pseudogene has an undiscovered function is supported by the fact the "exons" of the pseudogene (meaning those sequences that correspond to exons of the assumed parent gene -- gamma A) differ in humans and chimpanzees less than do the other allegedly nonfunctional sequences (the introns of both gamma A and the eta globin pseudogene). (Gibson, 103.) In other words, mutations of the eta globin pseudogene "exons" appear to be constrained, as would be expected if they were functional.

In addition, according to the standard evolutionary scenario, "the eta globin pseudogene has been maintained for more than 70 million years without being converted into a useful gene and without being eliminated." (Gibson, 98-99.) If it were functionless and thus not subject to selective pressure, "one would expect that [it] should be scrambled beyond recognition as a result of accumulated random mutations." (Jerlstrom, 15.)

The blanket assertion that pseudogenes "do not affect an organism's phenotype when deleted" is unproven. As Gibson says regarding the eta globin pseudogene:

> Several hemoglobin beta globin abnormalities are known, but none of them is associated specifically with the eta globin pseudogene [citation omitted]. This is interpreted as supporting the assertion that the

> pseudogene has no function. However, this argument is quite weak. The same result could occur for lethal mutations. No defective individuals would be observed because they do not survive long enough to be observed. Individuals with defective pseudogene sequences have been reported, but their abnormal hemoglobins were attributed to deleted portions outside the pseudogene sequence. It would be helpful to know whether normal individuals exist without the pseudogene sequence. Unless more information is available, the argument that the eta globin pseudogene has no effect on health cannot be said to have been demonstrated. (Gibson, 101-102.)

Establishing such a thing has only been made even more difficult by the discovery that gene function may be far more complicated than previously believed. If we do not know all that a gene does within the life of an organism, we are in no position to declare unequivocally that its absence can have no consequences.

Of course, if pseudogenes have a function, then God may have had a functional reason for initially placing them in separately created species. As Woodmorappe states:

> If pseudogenes are functional, they are no different from any other homologous structure found in nature. These all reflect the fact that God used the same 'blueprint' or 'art form' repeatedly when constructing different living things. In this case, the orthologous placement of pseudogenes, and their respective differences, are moot. (Woodmorappe 2000, 56.)

Finally, even if one could be certain that the existence of the same pseudogene in separate species had no functional explanation, it is possible that the same gene was inactivated by the same mutation occurring independently. The evolutionists' reply that this suggestion is too improbable to take seriously depends on the assumption that the mutation in question occurs randomly. But if there is a mechanism of mutation that favors certain locations in the gene, the odds against an independent occurrence of the mutation drop according to the strength of that bias.

As in the case of possible functions for pseudogenes, we simply do not know enough to assess definitively the odds against the independent occurrence of inactivating mutations (because we lack complete knowledge of all mechanisms of mutation). For example, molecular biologist Michael Brown believes there is evidence for the existence of either viral or enzymatic activity that creates mutations.

> So I think there is a mechanistic process that has produced many of the Pseudogenes that we have, rather than a random process. If the Pseudogene is truly defective and if the mutations are truly found in patterns (not random), then the idea that it's a common mechanism is possible. Viruses have enzymes that, under the same conditions, do repeatable reactions.

> If the DNA in Humans, Chimps, Monkeys, etc., are very similar, then if they are all infected by the same virus, would we expect the virus to do the same thing in the different species? I think so.[48]

Another possibility is the lateral transfer of a pseudogene from one species to another. Though Dr. Max admits that viral transfer of a pseudogene between species "seems superficially plausible," he concludes that "[f]or the present, the evidence argues against [it] as a general mechanism to explain shared pseudogenes/retroposons."[49] But if the mechanism of lateral transfer has not always been the same, if the mechanism we see today represents degeneration of a complex system that was designed to facilitate variation within created kinds (per Wood), then our judging of past possibilities by today's observations is flawed.

In other words, maybe lateral gene transfers occurred in the past through a mechanism that targeted a specific location in recipient cell DNA and that did not leave viral sequences near the inserted pseudogenes. Perhaps this mechanism is no longer operating, as a result progressive degeneration, and the viral action we see today is a distorted remnant of that originally designed process.

Many claims have been made in the past that a certain type of genetic data provided definitive proof of common ancestry, only to have further research reveal that the situation was more complicated than assumed. David Hillis's brief historical review is a useful reminder of the need for approaching such data with caution:

> What of the claim that the SINE/LINE insertion events are perfect markers of evolution (i.e., they exhibit no homoplasy)? Similar claims have been made for other kinds of data in the past, and in every case examples have been found to refute the claim. For instance, DNA-DNA hybridization data were once purported to be immune from convergence, but many sources of convergence have been discovered for this technique. Structural rearrangements of genomes were thought to be such complex events that convergence was highly unlikely, but now several examples of convergence in genome rearrangements have been discovered. Even simple insertions and deletions within coding regions have been considered to be unlikely to be homoplastic, but numerous examples of convergence and parallelism of these events are now known. Although individual nucleotides and amino acids are widely acknowledged to exhibit homoplasy, some authors have suggested that widespread simultaneous convergence in many nucleotides is virtually impossible. Nonetheless, examples of such convergence have been demonstrated in experimental evolution studies. (Hillis, 9980.)

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[48] These comments are from an email posted at Dr. Brown's website (http://www.mhrc.net/pseudogene.htm).

[49] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 5.10.

PREDICTION 21: NONFUNCTIONAL MOLECULAR EVIDENCE -- ENDOGENOUS RETROVIRUSES

Endogenous retroviruses are molecular remnants of a past parasitic viral infection. Occasionally, copies of a retrovirus genome are found in its host's genome, and these retroviral gene copies are called endogenous retroviral sequences. Retroviruses (like the AIDS virus or HTLV1, which causes a form of leukemia) make a DNA copy of their own viral genome and insert it into their host's genome. If this happens to a germ line cell (i.e. the sperm or egg cells) the retroviral DNA will be inherited by descendants of the host. Again, this process is rare and fairly random, so finding retrogenes[50] in identical chromosomal positions of two different species indicates common ancestry.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the same endogenous retrovirus (ERV) will exist in the same chromosomal location in two or more species.

2. The same ERV exists in the same chromosomal location in two or more species.

Since this is the concept of "shared errors" applied to endogenous retroviruses (and since retroviruses are a type of transposon), much of the two preceding responses is applicable. It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of neo-Darwinism that the same ERVs will exist in the same chromosomal location in two or more species. Evolution does not even predict the existence of ERVs, much less that they will be found at the same location in two or more species. After all, 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 the observation fits the prediction as evidence for the truth of evolution.

Moreover, ERVs are inadequate in principle to support Dr. Theobald's claim of universal common ancestry, because they are not shared by all groups of organisms. To quote Dr. Max once again, "Another limitation [of this argument] is that there are no examples of 'shared errors' that link mammals to other branches of the genealogic tree of life on earth. . . . Therefore, the evolutionary relationships between distant branches on the evolutionary genealogic tree must rest on other evidence besides 'shared errors.'"

The claim here is that common ancestry is the only viable explanation for "finding [ERVs] in identical chromosomal positions of two different species." It is based on the premise that ERVs are (and always have been) nonfunctional products of retroviral infection that have, for the most part, inserted randomly into the genome of the host organism. The presumed nonfunctionality of ERVs is thought to eliminate the explanation of design (because a Designer could have no purpose in placing

[50] The term "retrogene" is usually applied to a retrotransposed gene that has acquired promoter sequences and is thus actively transcribed.

nonfunctional sequences at the same locus in separate species). The presumed randomness of ERV insertion is thought to eliminate the explanation of chance (because the DNA "chain" is too long for coincidental insertion at the same locus to be a realistic possibility). That leaves common ancestry as the remaining explanation.

Again, it is an unprovable theological assertion that God would not place the same nonfunctional sequences at the same locus in separate species. He may have a purpose for doing so that is beyond our present understanding. The objection that placing nonfunctional sequences at the same locus in separate species would make God guilty of deception is ill founded. God cannot be charged fairly with deception when we choose to draw conclusions from data that contradict what he has revealed in Scripture (see Gibson's comments in the discussion of Prediction 19).

In any event, not all ERVs are nonfunctional. Some are transcriptionally active, and studies have revealed ERV protein expression in humans. (Sverdlov, 1.) We simply do not know all that ERVs (or other transposons) may be doing in an organism or what roles they may have played in the past. Sverdlov writes:

> [S]ometimes the hosts exploit the capacity of TEs [transposable elements] to generate variations for their own benefit. The retroelements can come out as traveling donors of sequence motifs for nucleosome positioning, DNA methylation, transcriptional enhancers, poly(A) addition sequences, splice sites, and even amino acid codons for incorporation into open reading frames of encoded proteins.
>
> The number of described cases in which retroelement sequences confer useful traits to the host is growing. Retropositions can therefore be considered as a major pacemaker of the evolution that continues to change our genomes. In particular HERV [human endogenous retrovirus] elements could interact with human genome through (i) expression of retroviral genes, (ii) human genome loci rearrangement following the retroposition of the HERVs or (iii) the capacity of LTRs [long terminal repeats that are common to ERVs] to regulate nearby genes. A plethora of solitary LTRs comprises a variety of transcriptional regulatory elements, such as promoters, enhancers, hormone-responsive elements, and polyadenylation signals. Therefore the LTRs are potentially able to cause significant changes in expression patterns of neighboring genes. (Sverdlov, 1-2.)

The functionality of ERV LTRs is suggested by the fact some elements within them are highly conserved. This means that "[t]here probably exists a kind of selection protecting the elements from mutational erosion. . . . It supports the idea that the LTRs (and perhaps other TEs) are of importance for some genomic purposes." (Sverdlov, 5.) The bottom line is that "[w]e do not know how important the involvement of LTRs is in genome functioning." (Sverdlov, 5.)

Of course, if ERV sequences have a function, then God may have had a functional reason for initially placing them at the same chromosomal location in separately created species. He also may have had a functional reason for designing a system to favor the insertion of certain ERV sequences at certain loci. In other words, maybe retroviruses are a corruption of an original complex system that was designed to facilitate diversification within kinds (per Wood). What was designed as an "altruistic genetic element," now shows only vestiges of that original benevolent purpose. In that case, the fact ERVs (and other transposons) now have mostly deleterious effects is because the original system has degenerated as a result of the Fall, not because they arose by random processes.

In that regard, it is interesting that, in addition to evincing certain functions, some ERVs (and other transposons) also exhibit an insertion bias. Perhaps this is another remnant of a more finely tuned system. Sverdlov writes:

> 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, 3.)

In addition, LTRs associated with HERVs frequently coincide with genes. This raises the possibility that they are somehow related functionally to those genes.

> We found frequent coincidences in positions of HERV-K LTRs and mapped genes on human chromosome 19 where the situation with mapped genes is slightly better. Although it would be premature to interpret this result as the indication of the regulatory interplay between closely located LTRs and genes, still some the the coincidences seem interesting. Most striking is the frequent coincidence of the LTRs with Zn-finger or Zn-finger-like genes scattered all over the chromosome. . . . Among other interesting coincidences, the LTRs were often detected in the vicinity of a number of genes (*RRAS*, *EPOR*, *JAK3* etc.) implicated at different stages of Jak-Strat signal transduction pathway. The frequent coincidences of the LTRs with the genes of similar or concerted functions

might suggest either functional involvement of the LTRs in the expression of the genes or their evolutionary relations. (Sverdlov, 4.)

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. And given what is known of retrovirus selectivity, I doubt anyone would be surprised.