A Critique of Douglas Theobald's
"29 Evidences for Macroevolution"

by Ashby Camp

Part 1

"One True Phylogenetic Tree"

In "29 Evidences for Macroevolution," Douglas Theobald sets forth the evidence that he believes proves scientifically that all living organisms descended from "one original living species." He does so by listing what he claims are 29 potentially falsifiable predictions of the hypothesis of universal common ancestry and presenting the evidence that he believes confirms each of those predictions.

Dr. Theobald does not address the origin of the first living thing or the mechanism by which that first organism diverged into every life form that has ever existed. His thesis is expressly restricted to the affirmation of universal common ancestry. In other words, he argues that, without knowing anything about how the first life arose or how it diversified, one can still be certain that all living things descended from the same ancestor. He states in the introduction (emphasis supplied):

In this treatise, I consider only macroevolution [which he labels a "virtual synonym" for universal common descent]. I do not consider microevolutionary theories, such as natural selection, genetic drift, sexual selection, theories of speciation, etc., *which biologists use as mechanistic theories to explain macroevolution.* Neither do I consider abiogenesis; I take it as axiomatic that an original self-replicating life form existed in the distant past.

In the conclusion, he says (emphasis supplied):

These previous points are all proofs of macroevolution alone; the evidences and the conclusion are *independent of any explanatory mechanism.* This is why scientists call macroevolution the "fact of evolution." None of the 29 predictions directly address how macroevolution has occurred; nevertheless, *the validity of the macroevolutionary conclusion does not depend on whether Darwinism, Lamarckism, or something else is the true mechanism of evolutionary change or not. The macroevolutionary conclusion still stands, regardless.*

Dr. Theobald understandably seeks to free the claim of universal common ancestry from the debate about the sufficiency of evolutionary mechanisms, particularly the debate about Neo-Darwinism. It should not go unnoticed, however, that a bare claim of universal common ancestry is compatible with *all* mechanisms of common descent, including divine direction. So if God chose to have a reptile give birth to a bird, for

example, that would be consistent with an “amechanistic” argument for universal common ancestry.[1]

The fact that Dr. Theobald leaves the mechanism of descent completely open does not make his claim trivial. On the contrary, the claim of universal common ancestry is incompatible with the belief that God separately created more than one living thing. It therefore challenges the convictions of biblical creationists, progressive creationists, and all who believe that mankind was created separately from animals.

I address Dr. Theobald’s predictions in the order in which he presented them. The italicized paragraphs following the predictions are quotations from his article. I quote only the prediction portion (or what I deem the relevant parts of it), not the alleged confirmations and potential falsifications. That would require me to duplicate the entire article. The accuracy of my references to the alleged confirmations or potential falsifications can be verified by consulting Dr. Theobald’s article.

I appreciate the civility with which Dr. Theobald argued his case and hope that my response is in kind. I also appreciate his candor in acknowledging that “science can never establish ‘truth’ or ‘fact’ in the sense that a scientific statement can be made that is formally beyond question.” (That may seem obvious to those attuned to the philosophy of science, but I suspect it will come as a surprise to many.) So however much weight one assigns to the evidences adduced by Dr. Theobald, they cannot “prove” universal common ancestry in the sense of rendering its rejection illogical.[2] That being said, the focus of this response is on the weight to which the evidences are entitled.

I include here for convenient reference Dr. Theobald’s Figure 1, which he labels “The standard phylogenetic tree.”

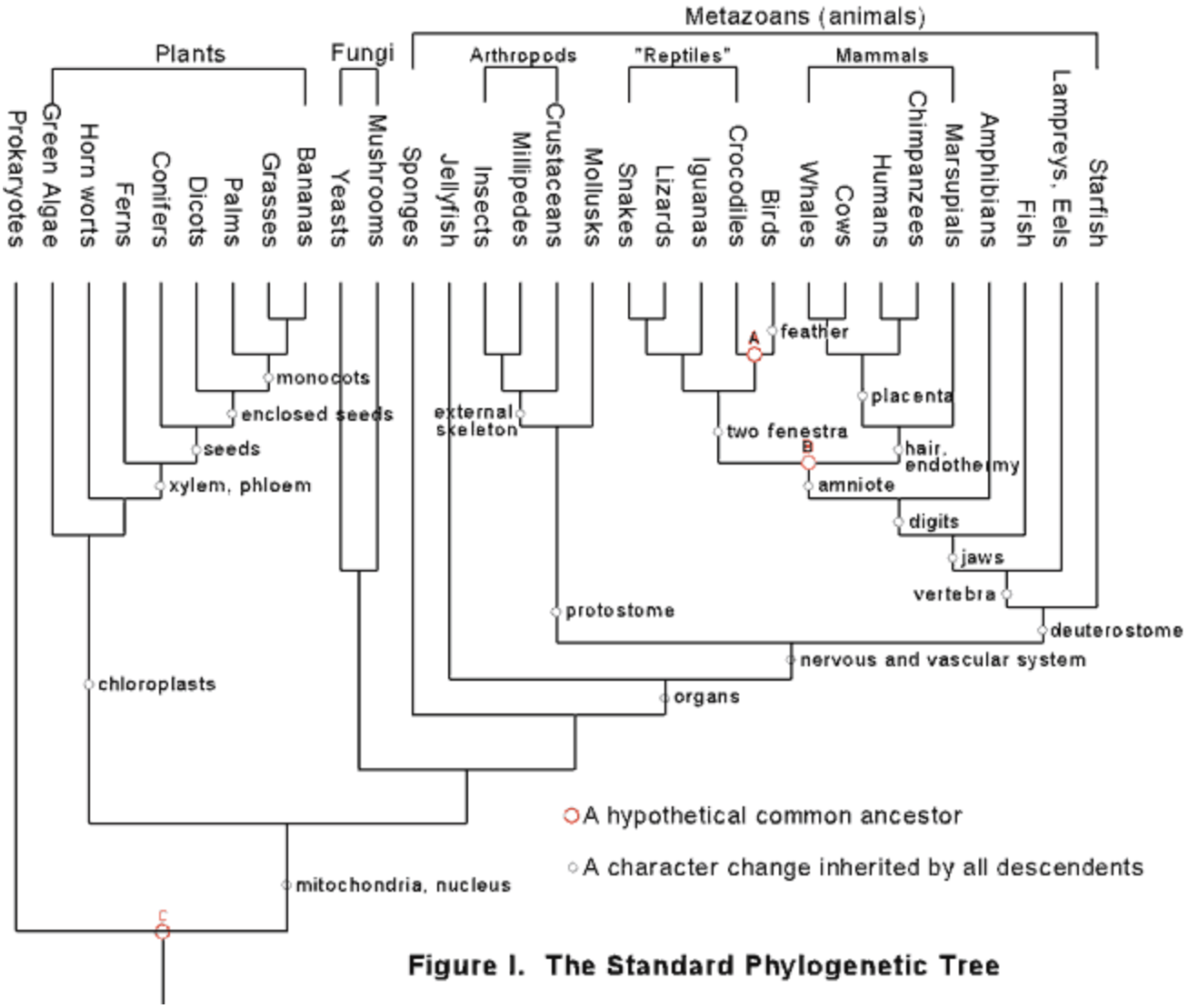

Figure I. The Standard Phylogenetic Tree

PREDICTION 1: THE FUNDAMENTAL UNITY OF LIFE

According to the theory of common descent, modern living organisms, with all their incredible differences, are the progeny of one single species in the distant past. In spite of the extensive variation of form and function among organisms, several fundamental criteria characterize all life. Some of the macroscopic properties that characterize all of life are (1) replication, (2) information flow in continuity of kind, (3) catalysis, and (4) energy utilization (metabolism). At a very minimum, these four functions are required to generate a physical historical process that can be described by a phylogenetic tree.

If every living species descended from an original species that had these four obligate functions, then all living species today should necessarily have these functions. Most importantly, they should have inherited the structures that perform these functions. The genealogical relatedness of all life predicts that organisms should be very similar in the particular mechanisms and structures that execute these basic life processes.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then all organisms will have one or more traits in common.
2. All organisms have one or more traits in common.

Unless one inserts an additional premise imposing a limit on the degree to which descendants can vary (which would require specification of a mechanism of descent), the claim of common ancestry does not require that all of the descendants share one or more traits. There is no *logical* reason why completely novel organisms could not arise in one or more lineages. Absent specification of a mechanism of descent, which Dr. Theobald purposefully avoids, there is no way to tether the traits of the descendants to those of the common ancestor.

The belief that evolution predicts biologic universals is "one of evolution's major illusions." (ReMine, 92.) As Walter ReMine says:

First, evolution does not predict that life would arise precisely once on this planet. If there were two or more unrelated systems of life, then evolutionary theory would effortlessly accommodate that situation.[3]
Second, even if life originated precisely once, then evolutionary theory would still not predict biologic universals. Shortly after life's origin, nothing prevented life from branching and leading separate lineages to higher life forms entirely lacking the known biologic universals.
Third, evolutionary loss and replacement processes could prevent biologic universals. If one organism is a distant ancestor to another, then nothing in evolution predicts the two must share similarities. If evolution were true, then distant ancestors and descendants (as well as sister groups) can be totally different.
Evolution never did predict biologic universals, it merely accommodated them. (ReMine, 92-93.)

Biophysicist Cornelius G. Hunter concurs. He writes:

There is yet another reason that the universality of the genetic code is not strong evidence for evolution. Simply put, the theory of evolution does not predict the genetic code to be universal (it does not, for that matter, predict the genetic code at all). In fact, leading evolutionists such as Francis Crick and Leslie Orgel are surprised that there aren't multiple codes in nature.
Consider how evolutionists would react if there were in fact multiple codes in nature. What if plants, animals, and bacteria all had different codes? Such a finding would not falsify evolution; rather, it would be incorporated into the theory. For if the code is arbitrary, why should there be just one? The blind process of evolution would explain why there are multiple codes. In fact, in 1979 certain minor variations in the code were found, and evolutionists believe, not surprisingly, that the variations were caused by the continuing evolution of the universal genetic code. Of course, it would not be a problem for such an explanation to be extended if it were the case that there were multiple codes.

There is nothing wrong with a theory that is comfortable with different outcomes, but there is something wrong when one of those outcomes is then claimed as supporting evidence. If a theory can predict both A and not-A, then neither A nor not-A can be used as evidence for the theory. When it comes to the genetic code, evolution can accommodate a range of findings, but it cannot then use one of those findings as supporting evidence. (Hunter, 38.)

The fact that some leading evolutionists believe early life forms were biochemically distinct from modern forms confirms that evolution does not predict biologic universals. Robert Shapiro, for example, entertains the possibility of finding living relics of an original protein-based life form that lacked DNA and RNA. (Shapiro, 293-295.) Likewise, A. G. Cairns-Smith thinks that descendants of ancient crystalline clay organisms may be all around us. He states: "Evolution did not start with the organic molecules that have now become universal to life: indeed I doubt whether the first organisms, even the first evolved organisms, had any organic molecules in them at all." (Cairns-Smith, 107.)

On the other hand, ReMine argues that biologic universals are a prediction of his message theory of creation, which "says all life was constructed to look like the unified work of a single designer." (ReMine, 94.) So evolution does not predict the unity of living things, but at least one theory of creation does.

Of course, the biochemical similarity of living things fits easily within a creation framework. As biochemist Duane Gish explains:

A creationist would also expect many biochemical similarities in all living organisms. We all drink the same water, breathe the same air, and eat the same food. Supposing, on the other hand, God had made plants with a certain type of amino acids, sugars, purines, pyrimidines, etc.; then made animals with a different type of amino acids, sugars, purines, pyrimidines, etc.; and, finally, made man with a third type of amino acids, sugars, etc. What could we eat? We couldn't eat plants; we couldn't eat animals; all we could eat would be each other! Obviously, that wouldn't work. All the key molecules in plants, animals, and man had to be the same. The metabolism of plants, animals, and man, based on the same biochemical principles, had to be similar, and therefore key metabolic pathways would employ similar macromolecules, modified to fit the particular internal environment of the organism or cell in which it must function. (Gish, 277.)

As for the alleged fulfillment, I do not doubt that all living things have carried out the basic functions of life in similar ways, but there are many organisms, past and present, about which we know nothing. It is impossible to be certain that none of these organisms is (or was) biochemically unique (witness the speculations of Shapiro and Cairns-Smith). The claim that all organisms have one or more traits in common is true in the sense that all living things necessarily have the traits by which life is defined, but that is simply a tautology—living things all have the traits of living things.

PREDICTION 2: A "NESTED" HIERARCHY OF SPECIES

As you can see from the phylogeny in Figure 1, the predicted pattern of organisms at any given point in time can be described as "groups within groups." This nested hierarchical organization of species contrasts sharply with the continuum of "the great chain of being" and the continuum predicted by Lamarck's theory of organic progression. Few other natural processes would predict a nested hierarchical classification. Real world examples that cannot be classified as such are elementary particles (which are described by quantum chromodynamics), the elements (whose organization is described by quantum mechanics and illustrated by the periodic table), the planets in our Solar System, books in a library, or specially designed objects like buildings, furniture, cars, etc. That certain organisms merely are similar to each other is not enough to support macroevolution; the nested classification pattern that satisfies the macroevolutionary process is very specific.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then organisms will be classifiable in a nested hierarchy.
2. Organisms are classifiable in a nested hierarchy.

It is not a corollary of the hypothesis of common descent that organisms will have features by which they can be classified as groups within groups. Common descent can explain or accommodate nested hierarchy (though not without difficulty in the specific case of Neo-Darwinism), but it does not predict it. There are mechanisms of descent from a common ancestor that would yield a different pattern. If common descent can yield either nested hierarchy or something else, then the presence of nested hierarchy does not count as evidence of common descent.

Hunter puts it this way:

It has been known since Aristotle that species tend to cluster in a hierarchical pattern, and in the eighteenth century Linnaeus saw it as a reflection of the Creator's divine plan. Obviously this pattern does not force one to embrace evolution. Also, Darwin's law of natural selection does not predict this pattern. He had to devise a special explanation—his principle of divergence—to fit this striking pattern into his overall theory. To be sure, evolution can accommodate this hierarchical pattern, but the pattern is not necessarily implied by evolution. (Hunter, 108.)

Even a mechanism of descent that includes branching events does not ensure a nested pattern. As ReMine explains:

The pattern of descent depends on the extent that evolved characters are later lost. Suppose losses are significant, and characters are replaced at a high rate. Then there is no reason to expect a nested pattern. Descendants could be totally different from their

distant ancestors and sister groups, with little or no semblance of nested similarities linking them. (ReMine, 343.)
Evolution does not predict a hierarchical pattern. Simple processes of loss, replacement, anagenesis, transposition, unmasking, or multiple biogenesis would prohibit such a pattern. Since hierarchical patterns (such as cladograms or phenograms) are not predicted by evolution they are not evidence for evolution. (ReMine, 444.)

In fact, nested hierarchy raises some difficult issues within a Neo-Darwinian framework. As Michael Denton observes:

In the final analysis the hierarchic pattern is nothing like the straightforward witness for organic evolution that is commonly assumed. There are facets of the hierarchy which do not flow naturally from any sort of random undirected evolutionary process. If the hierarchy suggests any model of nature it is typology[4] and not evolution. How much easier it would be to argue the case for evolution if all nature's divisions were blurred and indistinct, if the *systema naturalae* was largely made up of overlapping classes indicative of sequence and continuity. (Denton 1986, 136-137.)

The notion that the nested hierarchy of organisms is incompatible with creation is based, not on science, but on the unprovable theological assumption that if God created life he would do it in some other way. As biologist Leonard Brand explains:

The hierarchical arrangement of life illustrated in Fig. 9.6 has been used by Futuyma (1983) and others as evidence that life must have evolved. They believe that if life were created, the characteristics of different organisms would be arranged chaotically or in a continuum, not in the hierarchy of nested groups evident in nature. If we think of that concept as a hypothesis, how could it be tested? Actually, to state how a Creator would do things and then show that nature is or is not designed that way is an empty argument. Such conjecture depends on the unlikely assumption that we can decide what the Creator would be like and how he would function. (Brand, 155.)

It may be that the nested hierarchy of living things simply is a reflection of divine orderliness. It also may be, as Walter ReMine suggests, that nested hierarchy is an integral part of a message woven by the Creator into the patterns of biology. (See, e.g., ReMine, 367-368, 465-467.) The point is that the hierarchical nature of life can be accommodated by creation theory as readily as by evolution. Accordingly, "[i]t is not evidence for or against either theory." (Brand, 155.)

Dr. Theobald's claim that "specially designed objects like buildings, furniture, cars, etc." cannot be classified in a nested hierarchy requires elaboration. In terms of mere classification, it is incorrect. Buildings and vehicles have both been used as examples of nesting (Ridley 1993, 52-54; Fastovsky and Weishampel, 51-53; Brand, 165-166). According to Mark Ridley:

Any set of objects, whether or not they originated in an evolutionary process, can be classified hierarchically. Chairs, for instance, are independently created; they are not

generated by an evolutionary process: but any given list of chairs could be classified hierarchically, perhaps by dividing them first according to whether or not they were made of wood, then according to their colour, by date of manufacture, and so on. The fact that life can be classified hierarchically is not, in itself, an argument for evolution. (Ridley 1985, 8.)

PREDICTION 3: CONVERGENCE OF INDEPENDENT PHYLOGENIES

If there is one true historical phylogenetic tree, all separate lines of evidence should converge on the same tree, our standard phylogenetic tree.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then phylogenies constructed from any comparisons of organisms will "converge" on the standard phylogenetic tree.
2. Phylogenies constructed from comparisons of certain biological molecules in organisms "converge" on the standard phylogenetic tree.

There is an obvious disconnect between the alleged prediction and fulfillment. The fulfillment refers to only one basis of comparison (biological molecules), not all bases of comparison, and it refers to only some comparisons on the selected basis (some biological molecules), not all comparisons.

The alleged prediction could, of course, be amended to conform to the statement of fulfillment. The important point is that it is not a prediction of the hypothesis of common ancestry that phylogenies[5] constructed from comparisons of biological molecules will match phylogenies constructed from comparisons of morphology. This is obvious from the fact molecular and morphological phylogenies often are inconsistent, and yet the hypothesis of common descent is not considered falsified. The discordant data are simply accommodated by the theory.

The conflict between molecular and morphological phylogenies is a notorious problem in systematics. In fact, it was the focus of a recent article in Nature, subtitled: "Evolutionary trees constructed by studying biological molecules often don't resemble those drawn up from morphology. Can the two ever be reconciled, asks Trisha Gura." (Gura, 230.) Ms. Gura states in the article:

When biologists talk of the 'evolution wars', they usually mean the ongoing battle for supremacy in American schoolrooms between Darwinists and their creationist opponents. But the phrase could also be applied to a debate that is raging within systematics. On one side stand traditionalists who have built evolutionary trees from decades of work on species' morphological characteristics. On the other lie molecular

systematists, who are convinced that comparisons of DNA and other biological molecules are the best way to unravel the secrets of evolutionary history. . . .
Battles between molecules and morphology are being fought across the entire tree of life. Perhaps the most intense are in vertebrate systematics, where molecular biologists are challenging a tradition that relies on studies of fossil skeletons and the bones and soft tissue of living species. . . .
So can the disparities between molecular and morphological trees ever be resolved? Some proponents of the molecular approach claim there is no need. The solution, they say, is to throw out morphology, and accept their version of the truth. "Our method provides the final conclusion about phylogeny," claims Okada. Shared ancestry means a genetic relationship, the molecular camp argues, so it must be better to analyse DNA and the proteins it encodes, rather than morphological characters that can end up looking similar as a result of convergent evolution in unrelated groups, rather than through common descent. But morphologists respond that convergence can also happen at the molecular level, and note there is a long history of systematists making large claims based on one new form of evidence, only to be proved wrong at a later date. (Gura, 230, 232.)

These conflicts have long been recognized. In 1986, biochemist Christopher Schwabe wrote:

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

The incongruities of the molecular evidence led Schwabe to conclude that there were *multiple evolutionary trees* stemming from many separate origin-of-life events. In other words, he thought the evidence favored the existence of different genealogies instead of a unique one, i.e., polyphyletic evolution rather the traditional view of monophyletic evolution (universal common ancestry). He opined, "The quirks that will not submit to the neo-darwinian hypothesis are telling us that life had countless origins and that the chemistry of the origins of life has produced the diversity that has become a substrate for the evolution of biological complexity." (Schwabe, 282.)

Two years earlier, Schwabe and Gregory Warr were equally blunt in their criticism of molecular phylogenies. They saw the field of molecular evolution as being mired in subjectivity driven by an *a priori* commitment to universal common ancestry. They wrote:

We believe that it is possible to draw up a list of basic rules that underlie existing molecular evolutionary models:

1. All theories are monophyletic, meaning that they all start with the *Urgene* and the *Urzelle* which have given rise to all proteins and all species, respectively.

2. Complexity evolves mainly through duplications and mutations in structural and control genes.
3. Genes can mutate or remain stable, migrate laterally from species to species, spread through a population by mechanisms whose operation is not fully understood, evolve coordinately, splice, stay silent, and exist as pseudogenes.
4. Ad hoc arguments can be invented (such as insect vectors or viruses) that can transport a gene into places where no monophyletic logic could otherwise explain its presence.

This liberal spread of rules, each of which can be observed in use by scientists, does not just sound facetious but also, in our opinion, robs monophyletic evolution of its vulnerability to disproof, and thereby its entitlement to the status of a scientific theory.

The absolute, explicit and implicit, adherence to all the monophyletic principle and consequently the decision to interpret all observations in the light of this principle is the major cause of incongruities as well as for the invention of all the genetic sidestepping rules cited above. (Schwabe and Warr, 467.)

In 1993, Patterson, Williams, and Humphries scientists with the British Museum, reached the following conclusion in their review of the congruence between molecular and morphological phylogenies:

As morphologists with high hopes of molecular systematics, we end this survey with our hopes dampened. Congruence between molecular phylogenies is as elusive as it is in morphology and as it is between molecules and morphology. . . .
Partly because of morphology's long history, congruence between morphological phylogenies is the exception rather than the rule. With molecular phylogenies, all generated within the last couple of decades, the situation is little better. Many cases of incongruence between molecular phylogenies are documented above; and when a consensus of all trees within 1% of the shortest in a parsimony analysis is published (e.g. 132, 152, 170), structure or resolution tends to evaporate. (Patterson and others, 180.)

Citing many recent examples, Laura Maley and Charles Marshall wrote in 1998: "Animal relationships derived from the new molecular data sometimes are very different from those implied by older, classical evaluations of morphology. Reconciling these differences is a central challenge for evolutionary biologists at present." (Maley and Marshall, 505.) An important issue is the nature of the assumptions under which this reconciliation will be pursued.

The following year, biologist Carl Woese, an early pioneer in constructing rRNA-based phylogenetic trees, wrote: "No consistent organismal phylogeny has emerged from the many individual protein phylogenies so far produced. Phylogenetic incongruities can be seen everywhere in the universal tree, from its root to the major branchings within and among the various taxa to the makeup of the primary groupings themselves." (Woese, 6854.)

It should be noted that molecular phylogenies are constructed on the basis of certain evolutionary assumptions. The tree that is presented is chosen from a forest of alternatives, typically on the assumption of maximum parsimony. That is, the tree that is selected is the one that reflects the least amount of presumed evolutionary change. But if the assumption of maximum parsimony fails to fit the data, it can be jettisoned in favor of another. (Hunter, 40-41.)[6] The availability of such ad hoc adjustments for resolving incongruities makes the claim of falsifiability an illusion. Any result can be accommodated by the theory by revising one or more of the underlying assumptions.

Even if a morphological phylogeny was matched closely by multiple molecular phylogenies, that would not prove that the groups in question descended from a common ancestor.[7] The molecular differences could be linked to the morphological differences for some other reason. Hunter illustrates the point this way:

Penny[8] obtained his trees by culling those that were most parsimonious—that is, he selected the trees that showed the least amount of evolutionary change to represent the history of life. The first problem is that Penny's method works perfectly fine on things we know did not come about via Darwinian evolution. For example, Penny's method would also claim that automobiles evolved from one another. Consider a group of vehicles, beginning with a small economy car and increasing in size to larger cars and to minivans and large-sized vans. One could quantify several aspects of the vehicle designs, such as tire size, steering mechanism, engine size, number of seats and so forth. Presupposing the evolutionary paradigm and searching for parsimonious relationships, we would find that most of the design measures suggest the same relationship. The smaller vehicles have smaller tires, manual steering, smaller engines, and fewer seats. The larger vehicles have larger tires, power steering, larger engines, and more seats. In other words, the groupings suggested by the different design measures (tire size, steering mechanism, engine size, etc.) tend to be similar. But of course, the family of automobiles did not evolve from one another via random mutations. The groupings of the design measures are a natural result of engineering and have nothing to do with Darwinian evolution. How then can Penny's results provide "strong support" for evolution? (Hunter, 40.)

As Gish explains, it would not be surprising from a creation perspective to find that biochemical similarities increase in relation to other similarities of the creatures being compared. He writes:

We know, for instance, that man is more similar to a chimpanzee than he is to a bat; that he is more similar to either a chimpanzee or a bat than he is to a crocodile or a flea. Man, chimpanzee, and the bat are mammals. The creationist would expect, therefore, that his protein, DNA, and RNA molecules, those macromolecules that are among the most important molecules in metabolism, would be more similar to those of the chimpanzee and to those of the bat than to those of the crocodile or the flea. . . . Creationists believe that all normal genes, the genes that account for the normal, healthy differences in plants and animals, were created. Each basic type of plant and animal was created with a sufficient genetic potential or variability (or gene pool, as geneticists say) to permit

sufficient variability within the circumscribed boundaries of each kind, in order to adapt to various environments and conditions. (Gish, 277-278.)

Biologist Leonard Brand concurs. "Anatomy is not independent of biochemistry. Creatures similar anatomically are likely to be similar physiologically. Those similar in physiology are, in general, likely to be similar in biochemistry, whether they evolved or were designed." (Brand, 156.) He makes the same point with specific reference to phylogenies based on cytochrome c.

An alternate, 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 grouping 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 of 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.)

Of course, failure to discern a relationship between morphology and a particular biological molecule does not prove the nonexistence of such a relationship. It may mean simply that the relationship is beyond our present understanding. The possibility of our ignorance is obvious, but even if it was not, earlier proclamations that most DNA is functionless "junk" illustrate the point. "Recent research has begun to show that many of these useless-looking sequences do have a function." (Walkup, 19.)

The cytochrome c data on which Dr. Theobald relies present some puzzles from a Neo-Darwinian perspective. First, the cytochromes of all the higher organisms (yeasts, plants, insects, fish, amphibians, reptiles, birds, and mammals) exhibit an almost equal degree of sequence divergence from the cytochrome of the bacteria *Rhodospirillum.* In other words, the degree of divergence does not increase as one moves up the scale of evolution but remains essentially uniform. The cytochrome c of other organisms, such as yeast and the silkworm moth, likewise exhibits an essentially uniform degree of divergence from organisms as dissimilar as wheat, lamprey, tuna, bullfrog, snapping turtle, penguin, kangaroo, horse, and human. (See matrices in Brand, 157 and Davis and Kenyon, 37.)

Why would the sequence divergence of cytochrome c between bacteria and horses be the same as the divergence between bacteria and insects? The presumed evolutionary lineage from the ancestral cell to a modern bacterium differs radically from the presumed evolutionary lineage from the ancestral cell to a modern horse, both of which differ radically from the presumed evolutionary lineage from the ancestral cell to a modern insect. How could a uniform rate of divergence have been maintained through such radically different pathways? According to Michael Denton, a molecular biology researcher, "At present, there is no consensus as to how this curious phenomenon can be explained." (Denton 1998, 291.)

Moreover, the notion that the rates of divergence remain uniform regardless of evolutionary pathway does not fit all of the cytochrome c data. For example, referring to Dr. Theobald's Figure 1 (reproduced above), lampreys, carp, and bullfrogs allegedly shared a common ancestor above the node labeled "vertebra." Since that time, the branch leading to carp and bullfrogs evolved independently of the branch leading to lampreys. If the rates of cytochrome c divergence remain uniform regardless of evolutionary pathway, then the degree of sequence variance between the cytochrome c of lampreys and carp would be essentially the same as the degree of variance between the cytochrome c of lampreys and bullfrogs. That is not the case. The variance between the cytochrome c of lampreys and carp is 12%, whereas the variance between lampreys and bullfrogs is 20%. (See matrix in Davis and Kenyon, 37.)

Second, the sequences of cytochrome c sometimes differ inversely to the presumed evolutionary proximity of the organisms being compared. For example, turtles and rattlesnakes, both being reptiles, are presumed to have shared a common ancestor with each other more recently than they shared a common ancestor with humans. So the evolutionist would expect the cytochrome c of a rattlesnake to be more similar to that of a turtle than to that of a human. That, however, is not the case. The cytochrome c of the rattlesnake varies in 22 places from that of the turtle but only in 14 places from that of a human. (See matrix in Brand, 134.)

Humans and horses, both being placental mammals, are presumed to have shared a common ancestor with each other more recently than they shared a common ancestor with a kangaroo (a marsupial). So the evolutionist would expect the cytochrome c of a human to be more similar to that of a horse than to that of a kangaroo. Yet, the cytochrome c of the human varies in 12 places from that of a horse but only in 10 places from that of a kangaroo. (See matrix in Brand, 134.)

Such discrepancies between traditional phylogenies and those based on cytochrome c are well known. Even Ayala could only bring himself to say that "[t]he overall relations agree *fairly well* with those inferred from the fossil record and other sources" (emphasis supplied). (Ayala, 68.) He then acknowledged:

The cytochrome c phylogeny disagrees with the traditional one in several instances, including the following: the chicken appears to be related more closely to the penguin than to ducks and pigeons; the turtle, a reptile, appears to be related more closely to birds than to the rattlesnake, and man and monkeys diverge from the mammals before the marsupial kangaroo separates from the placental mammals. (Ayala, 68.)

PREDICTION 4: POSSIBLE MORPHOLOGIES OF PREDICTED COMMON ANCESTORS

Any fossilized animals found should conform to the standard phylogenetic tree. Every node shared between two branches represents a predicted common ancestor; thus there are ~30 common ancestors predicted from the tree shown in Figure 1. Our standard tree shows that the bird grouping is most closely related to the reptilian grouping, with a node

linking the two (A in Figure 1); thus we predict the possibility of finding fossil intermediates between birds and reptiles. The same reasoning applies to mammals and reptiles (B in Figure 1). However, we predict that we should never find fossil intermediates between birds and mammals.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then all fossilized animals will "conform"[9] to the standard phylogenetic tree.
2. All fossilized animals "conform" to the standard phylogenetic tree.

Universal common ancestry affirms only that all creatures descended from the same ancestor. There is nothing about that affirmation that requires conformity to the standard phylogenetic tree. A phylogenetic tree is merely a diagram that reflects current evolutionary thinking about the relationships of the taxa included. Branches are arranged on the tree on the assumption of evolution and according to perceived similarities in selected traits.[10] The relationships of some branches are viewed more dogmatically than the relationships of others, but none of the branches are set in stone.

Since phylogenies are by nature provisional, the suggestion that the hypothesis of common descent would be falsified by "[a]ny finding of mammal/bird intermediates" is mistaken. Should a strikingly birdlike mammal be discovered, the standard tree simply would be modified to accommodate the new creature, after wrangling over its placement in the schema.

The ease with which this precise adjustment could occur was illustrated two decades ago, when "[t]he reality of the 'mammal-bird,' a hypothetical common ancestor of birds and mammals, [was] a contentious issue in modern systematics." (Mike Benton, 18.) Brian Gardiner's cladistic analysis indicated that birds were most closely related to mammals, which relationship was supported by two Cambridge scientists' analysis of molecular data. That view was readily accepted by some, even to the point that one French paleontologist "published a restoration of the hypothetical common ancestor between birds and mammals—a sort of warm-blooded, hairy/feathery climbing insect eater!" (Mike Benton, 18.) Branches can be rearranged, even between mammals and birds, without skipping a beat in terms of commitment to common ancestry.

Of course, the discovery of a strikingly birdlike mammal would not necessarily force a shift in thinking about the relationship of mammals and birds (a placing of their branches next to each other). The birdlike features could be attributed to convergent evolution. Many organisms are believed by evolutionists to have evolved similar traits independently. (In fact, some experts believe that the birdlike features of dromaeosaurids, the dinosaurs considered by most experts to be the sister group to birds, arose independently rather than by inheritance from the ancestor of birds.) If the mammal's birdlike traits were judged to be the result of convergent evolution, the species would be shown on the phylogenetic tree as a subset or side branch of mammals that was unrelated to birds.

The shift in thinking over the last 30 years about the relationship of dinosaurs and birds is an example of a generally accepted phylogenetic adjustment, albeit at a lower taxonomic level. From the publication of Gerhard Heilmann's *The Origin of Birds* in 1926, it was a matter of textbook orthodoxy that birds were more closely related to thecodonts (an order of reptiles) than to theropods (a suborder of a different order of reptiles). Thus, the discovery in 1964 of the birdlike theropod *Deinonychus* was contrary to phylogenetic expectations. Today, however, the standard phylogeny shows birds more closely related to theropods than to thecodonts.

The assertion that all fossilized animals conform to the standard phylogenetic tree is unprovable, because one can never be sure that all fossilized animals have been discovered. But more importantly, the premise turns out to be merely a restatement of the claim of nested hierarchy. It adds nothing to the case for common ancestry.

Conformity and nonconformity to the standard phylogenetic tree are defined in the article in terms of "intermediates." It is stated that, given the standard phylogeny, one would expect "intermediates" between reptiles and birds and between reptiles and mammals (because these pairs are shown as sharing hypothetical common ancestors, A and B in Figure 1), but one would not expect "intermediates" between mammals and birds. It is then alleged that the fossils conform to this expectation, and thus "conform to the standard phylogenetic tree," in that "intermediates" have been found between reptiles and birds (citing mainly dromaeosaurids) and between reptiles and mammals (citing synapsids) but not between mammals and birds.

But according to the definition of "intermediate" given in the article, dromaeosaurids are *not* reptile-bird intermediates and synapsids are *not* reptile-mammal intermediates. An "intermediate form" is defined as "[a] fossil or modern species that displays characters *definitive* of two or more different taxa" (emphasis supplied). Dromaeosaurids do not display characters that are *definitive* of both reptiles and birds (which is why they are not considered birds), and synapsids do not display characters that are *definitive* of both reptiles and mammals (which is why they are not considered mammals).

On the other hand, under the given definition, *all* taxa qualify as "intermediates" between themselves and the taxa in which they are shown as nested.[11] For example, *all* mammal species, including all monotremes and marsupials, are reptile-mammal "intermediates" because they all possess the traits that are definitive of both Reptilia and Mammalia.[12] That is, they are all amniotes with the definitive traits of Mammalia. (Reptilia is defined simply as amniotes that are not birds or mammals [Carroll, 193].) Likewise, *all* bird species, including the Kiwi (called an "honorary mammal"), are reptile-bird "intermediates" because they all possess the traits that are definitive of both Reptilia and Aves.

But if taxa are intermediate by virtue of being nested, the existence of intermediates is not a separate argument for common ancestry. It is the argument of nested hierarchy under a different label. And if there are no intermediates between non-nested taxa, that means only that nested hierarchy is a pattern to which there are no known exceptions. As

previously explained, that result could be accommodated by the theory of common descent, but it is not evidence for it.

In citing dromaeosaurids as reptile-bird intermediates and mammal-like reptiles as reptile-mammal intermediates, Dr. Theobald is apparently defining "intermediates" as organisms that are morphologically between alleged ancestors and descendants (rather than using the specified definition of organisms that possess the *definitive* traits of the two relevant taxa). But if intermediates can occur by definition only between alleged ancestors and descendants, then they can occur by definition only in conformity to the phylogenetic tree.

Consider the striking similarities between some marsupials and placentals. If the consensus were that a marsupial wolf evolved into a placental wolf, then the marsupial wolf would qualify as an intermediate under the definition being considered. That is, it would be morphologically between its alleged ancestor (an earlier marsupial) and descendant (the placental wolf). But since the consensus (which is reflected in the standard phylogeny) is that marsupial wolves and placental wolves arose independently, the marsupial wolf cannot qualify as a marsupial-placental intermediate, whatever its morphology. Conformity with the standard phylogeny is guaranteed by the definition.

The assertions that there are "no morphological gaps" in the alleged dinosaur-to-bird transition and that there is an "exquisitely complete series of fossils" for the alleged reptile-to-mammal transition are debatable, to say the least. I have elsewhere tried to point out some of the limitations of those claims (see, "On the Alleged Dinosaurian Ancestry of Birds" and "Reappraising the Crown Jewel").

But even if one granted that reptiles evolved into a bird and a mammal, that would not establish that reptiles and all other organisms descended from a common ancestor, which is the proposition being argued. The difference between a bacterium and a reptile, not to mention the other organisms, is considerably greater than the difference between a reptile and a bird or a reptile and a mammal. So the fact a reptile could evolve into a bird or a mammal would not mean that a bacterium could evolve into a reptile and everything else. In fact, granting that reptiles evolved into a bird and a mammal would not even establish that all birds and all mammals descended from a reptile. That would be an assumption.

PREDICTION 5: CHRONOLOGICAL ORDER OF PREDICTED COMMON ANCESTORS

Fossilized intermediates should appear in the correct general chronological order based on the standard tree. Any phylogenetic tree predicts a relative chronological order of hypothetical common ancestors and intermediates between these ancestors. For instance, in our current example, the reptile/mammal common ancestor (B) [from Figure 1] and intermediates should be older than the reptile/bird common ancestor (A) [from Figure 1] and intermediates.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then fossil intermediates will appear in the "general chronological order" reflected in the standard phylogenetic tree;
2. Fossil intermediates appear in the "general chronological order" reflected in the standard phylogenetic tree;

As pointed out above, "intermediate" is defined in the article as "[a] fossil or modern species that displays characters *definitive* of two or more different taxa" (emphasis supplied). Since, under that definition, a taxon is intermediate by virtue of being nested within another, the alleged prediction is that fossils will appear in the order of nesting reflected in Figure 1. In other words, a prokaryotic organism would appear first, followed successively (in the fungi/metazoan direction) by organisms with nuclei, multicellularity, organs, nervous and vascular system, and so on up the deuterostomic and protostomic branches.

There is nothing about the hypothesis of universal common ancestry that requires organisms to have descended in the pattern depicted in the standard phylogeny. Common ancestry does not even require nested hierarchy, let alone any particular pattern of nesting. A phylogeny is simply a depiction of the order in which evolutionists believe taxa arose, not the order in which they were *required* to arise. (And even if it was believed that universal common descent could occur in only one way, that is an assertion about the mechanism of descent, a subject Dr. Theobald purposefully excluded from his case.)

Moreover, while ancestral taxa must have existed before any taxa that descended from them, that does not mean the appearance of their fossilized forms must correspond to that order of existence. However unlikely the claim may be, it remains possible for a proponent of common descent to assert that select taxa appear in the fossil record contrary to the order in which they came into existence.

Witness the fact dromaeosaurids, which are offered by Dr. Theobald as "reptile-bird intermediates,"[13] first appear in the fossil record some 25 million years *after* the first fossil bird. (If one accepts *Protoavis*, rather than *Archaeopteryx*, as the first fossil bird, the gap in appearance increases to about 100 million years.) Rather than disqualifying dromaeosaurids in Dr. Theobald's eyes as "reptile-bird intermediates," which he argues must appear in the order suggested by the standard phylogeny, it is simply assumed that dromaeosaurids lived tens of millions of years before there is any evidence of their existence. (The ambiguity of "*general* chronological order" prevents such nonconformities from falsifying the claim.)

This same strategy could be employed if dromaeosaurids turned up in strata older/lower than that in which synapsids first appear. That is, it could be assumed that pelycosaurs and therapsids actually predated dromaeosaurids but for some reason did not appear in the fossil record until later. So the suggestion that the hypothesis of universal common

ancestry would be falsified if dromaeosaurids first appeared in the fossil record before synapsids reptiles is incorrect.

The fact synapsids appear before dromaeosaurids hardly constitutes proof (confirms the "prediction") that "fossilized intermediates" appear in the general chronological order indicated in the standard phylogeny. They are only two data points. But more importantly, one must bear in mind that Figure 1 is of necessity a simplified and fragmentary phylogeny. The picture changes significantly when the scope of inquiry is broadened.[14] According to one Harvard-trained paleontologist:

[T]he correspondence between phylogeny and the fossil record is not as strong as it might first seem. When the order of all kingdoms, phyla and classes is compared with the most reasonable phylogenies, over 95 percent of all the lines are not consistent with the order in the fossil record. The only statistically significant exceptions are the orders of first appearances of the phyla of plants and the classes of vertebrates and arthropods. Yet these three lineages also order organismal groups from sea-dwellers to land dwellers. The land-plant phyla, for example, are in a simple sequence from plants that need standing water to survive (e.g., algae and bryophytes) to those that can survive extreme desiccation (e.g., the cacti). The vertebrate classes go from sea-dwellers (fish) to land/sea creatures (amphibians) to land creatures (reptiles/mammals), to flying creatures (birds). The arthropod classes go from sea-dwellers (e.g., trilobites, crustaceans) to land dwellers (e.g., insects). So it's not clear that macroevolution is a truly good explanation for the order of fossil first appearances of major groups of life. Such a radical idea as a global flood, for example, which gradually overcame first the sea and then the land, actually explains the primary order of major groups in the fossil record (sea to land) better than macroevolutionary theory. (Wise, 225-226.)

Notes for Part 1

[1] Dr. Theobald contradicts his claim to argue for common ancestry without regard to any particular mechanism by including in his definition of macroevolution the requirement of gradualness. He states, "Macroevolution, as I will use it, is the theory of common descent with gradual modification" (emphasis supplied). He states further that "[g]radualness concerns genetically probable organismic changes between two consecutive generations, i.e., those changes that are within the range of normal variation observed within modern populations." There is no reason to believe that the range of variation observed within modern populations can account for the creation of new organs, structures, and systems as required by the hypothesis of universal common ancestry. In restricting the mechanism of macroevolution to observable degrees of genetic variation, Dr. Theobald lets in the back door the very debate about mechanism that he tossed out the front. He thereby assumes the burden of proving that accumulated observable variation can account for universal common ancestry. Since he makes no attempt to meet that burden but rather repeatedly disavows the relevance of any particular mechanism of modification, I assume he did not intend to specify accumulated observable variation as the mechanism of macroevolution, despite what his definitions may suggest.

[2] This limitation is evident, from a philosophical perspective, by the fact the evidences, if offered as formal proofs, are in the form "If A (universal common ancestry), then B; B therefore A." This argument is a non sequitur, known more specifically as the fallacy of affirming the consequent. [3] Dr. Theobald

assumes a single origin of life, so this comment is beyond the scope of his paper. I include it to provide context for the remainder of the quote.

[4] "Typology" views organisms as variations of *distinct* archetypes. See, Denton (1986), 93-118. With reference to nested hierarchy, Denton writes:

The sort of evolution [pre-Darwinian typologists] conceived was the creative derivation of all the members of a class from the hypothetical archetype which existed in the mind of God. When typologists drew up branching tree diagrams to illustrate the relationships between different species, this did not imply that the members of a class had been derived by natural descent from a common ancestor. None of the nodes or branches of such trees had any real empirical existence; they were 'links' but only in an abstract and ideal sense. As Agassiz in his essay on classification maintained:
What we call branches expresses, in fact, a purely ideal connection between animals, the intellectual conception which unites them in creative thought. It seems to me that the more we examine the true significance of this kind of group, the more we shall be convinced that they are not founded upon material relations. [emphasis added] (Denton 1986, 132.)

[5] Strictly speaking, these are not "phylogenies" but "phenograms" and "cladograms." "[A] lineage is a recognizable line of ancestry with identifiable ancestors and descendants. A *phylogeny* is merely discrete segments of lineage connected to an identifiable tree-structure of ancestry." (ReMine, 259.) A "phenogram" is a tree-structured diagram based on the overall similarities between the objects being classified. A "cladogram" is a tree-structured diagram based on the distribution of particular characters throughout the objects being classified. Neither phenograms nor cladograms specify ancestors, whereas a true phylogeny does. (ReMine, 265-268.) I am aware that most writers do not observe these distinctions, but they are still worth keeping in mind.

[6] By appealing to molecular phylogenies, Dr. Theobald is appealing implicitly to their assumption about the manner of descent. However reasonable any given assumption may be from a Neo-Darwinian perspective, Dr. Theobald's stated objective is to establish universal common descent without regard to any explanatory mechanism. He is thus precluded from assuming particular mechanisms of descent (e.g., one that excludes widespread lateral gene transfers) to make his case.

[7] Of course, to have relevance for Dr. Theobald's thesis of *universal* common ancestry, the analyses would need to include all groups of living things.

[8] "David Penny reconstructed the phylogeny for a group of eleven species, using five protein molecules. The proteins were used one at a time, independently of the other four, yet they suggested similar phylogenies." (Hunter, 40.) Hunter points out that "though Penny found the trees to be 'very similar,' there were significant differences. For example, some of his trees show the dog relatively far from the human (nine species out of a possible ten), whereas others show the dog relatively close to the human (three species distant out of ten)." (Hunter, 40.)

[9] By "conform" to the standard phylogenetic tree, Dr. Theobald appears to mean having traits that are definitive of two taxa that are shown on the phylogeny as ancestral and descendant (e.g., reptiles and birds). Nonconformity to the standard phylogenetic tree is having traits that are definitive of two taxa that are shown on the phylogeny as having arisen independently of each other (e.g., birds and mammals).

[10] Brand is worth quoting at length here.

The process used in constructing phylogenetic trees begins with the collection of data on the characteristics of the groups being studied. If we study the relationships between several orders of mammals, we compare many characters of these orders, perhaps beginning with tooth and skeletal anatomy to determine which orders have canine teeth and which have a complete postorbital bar behind the eyes. Many additional

characters would be added. Then we tell the computer to compare these groups, to determine the similarities (homologies) between them, and to generate phylogenetic trees.

Determining which characteristics are primitive (ancestral) and which are derived is called polarization. This is usually accomplished by including an outgroup in the analysis for comparison. The outgroup is a group that is closely related to but is outside of the groups that are being studied. For example, a study of the orders of mammals might use reptiles as an outgroup. The mammalian order with the fewest differences from the outgroup is considered the most primitive order, closest to the common ancestor of mammals. (Brand, 162-163.)

When we first put the data into the computer, it does not produce a tree; it has no way to determine which one of the groups is the ancestor or closer to the ancestor. It can only produce an uprooted tree, showing which groups are more similar (D in Fig. 10.4). An outgroup must be added before it can produce a tree. However, we have no reason to introduce an outgroup unless we first assume evolution of the two groups from a common ancestor. A study of mammals, using reptiles as the outgroup, is based on the assumption that they both evolved from a common ancestor. If we make that assumption, then the computer looks for the order of mammals with the most characters in common with the outgroup. Now the computer makes the mammalian group the root of the tree that it can construct. It cannot even construct a tree unless the researcher first makes the assumption of megaevolution by adding an outgroup. (Brand, 164.)

[11] Theobald's definition thus approaches that of Cracraft, who wrote, "Each species, then is an intermediate in some sense of the word; all species possess primitive and derived characters." (Cracraft, 146.)

[12] Figure 1 is misleading here in that it uses the label "Reptiles" only for diapsids. Anapsida, the group believed to have given rise to both diapsids and synapsids (and eventually mammals), is a subclass of Reptilia.

[13] Dromaeosaurids are considered a sister group to birds, meaning they are believed to have shared with birds a most recent common ancestor. They are not believed to have been in the actual lineage of birds. In fact, they possess certain specializations, such as the stiffened tail, that make them ill suited as ancestors. Of course, the presumed common ancestor of birds and dromaeosaurids is thought to have been quite dromaeosaurid-like. As already noted, dromaeosaurids do not qualify as reptile-bird intermediates according to Dr. Theobald's definition.

[14] But even Figure 1, which highlights plants, vertebrates, and arthropods, is not free of incongruities. For example, the first appearance of Cnidaria, the phylum to which jellyfish are assigned, is earlier/lower than (or possibly contemporaneous with) the first appearance of Porifera, the phylum to which sponges are assigned.

A Critique of Douglas Theobald's
"29 Evidences for Macroevolution"

by Ashby Camp

Part 2

"Past History"

PREDICTION 6: ANATOMICAL VESTIGIAL STRUCTURES

Some of the more renowned evidences for evolution are the explanations it provides for nonfunctional or rudimentary vestigial characters, both anatomical and molecular. Throughout macroevolutionary history, functions necessarily have been gained and lost; thus, we predict vestigial structures, which are structural evidence of lost functions. Since there is no apparent reason for their existence, nonfunctional characters of organisms are especially puzzling. So are rudimentary structures, which have different and relatively minor functions compared to the same more developed structures in other organisms. Consequently, evolutionary explanations for vestigial characters are strong proofs.

Explanations are not evidence; they are attempts to explain evidence. So the first and last sentences of the quoted paragraph are at best overstatements. The question is whether the evidence of "vestigial" structures favors the explanation of universal common ancestry, and if so, how strongly.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some organisms will have structures the function of which was lost or diminished in the course of the organism's evolutionary history ("vestiges" of the organism's evolutionary history).
2. Some organisms have structures the function of which was lost or diminished in the course of the organism's evolutionary history.

Vestigial structures are not a necessary result of all possible mechanisms of universal common descent. They are understandable within a Neo-Darwinian framework of random mutation and natural selection, but since Dr. Theobald has chosen to argue for common ancestry without regard to any mechanism of descent, he cannot offer as evidence data that can be explained only by particular mechanisms of descent.

Moreover, even Neo-Darwinism does not demand vestigial structures; it simply accommodates them. They can exist or not exist with equal ease under the theory and can appear with any frequency. Any result can be explained by appeal to the randomness of mutation and the uncertainty of the selective pressures that were at work in any given lineage.

In any event, vestigial structures provide no support for the claim of universal common ancestry. A bona fide vestigial structure says only that the organism in which it is found descended from an earlier organism that possessed the structure in fully functional form. It says nothing about how that earlier organism came to exist, whether it descended from a universal common ancestor, descended from one of many independently created organisms, or was itself created independently.[15] Since vestigial structures can arise in unconnected lineages as well as in lineages that are rooted in a common ancestor, they do not count as evidence for universal common ancestry.

Of course, the identification of bona fide vestigial structures is fraught with difficulty. Dr. Theobald defines a vestigial character as "a character that *for all intents and purposes* has no *obvious* or *important* function, yet is structurally *similar* to functional characters in other species" (emphasis supplied). He elaborates: "If the character appears reduced and rudimentary compared to the same structure in other organisms, and the structure has obvious important functions in the majority of other organisms, then it is considered a vestigial structure."

The problems are illustrated by Dr. Theobald's use of the human coccyx (which he describes as "the four fused tail vertebrae of humans") as an example of a vestigial structure. It has long been known that the coccyx serves as a point of attachment for ligaments and several important muscles. So why think the coccyx was not specially designed by a Creator to fulfill that function?

The answer, from Dr. Theobald's definition, is twofold. First, the function of the coccyx must be judged "unimportant" (given that the function is obvious). That, however, is a grossly subjective assessment. It is also clearly theological. How does one determine when a function is important enough to make it plausible that a Creator would specially design a structure to fulfill it?

This particular trap is avoided if vestigial structures are defined as those that have *no* function (rather than those that have no *important* function). But that definition stumbles over the fact one can never really be certain that an apparently functionless structure is really functionless. It may be that we lack the knowledge necessary to appreciate its function. As S. R. Scadding pointed out 20 years ago:

I would suggest that the entire argument that vestigial organs provide evidence for evolution is invalid on two grounds, one practical, the other more theoretical. The practical problem is that of unambiguously identifying vestigial organs, i.e., those that have no function. The analysis of Wiedersheim's list of vestigial organs points out the difficulties. . . . Wiedersheim could list about one hundred in humans; recent authors

usually list four or five. Even the current short list of vestigial structures in humans is questionable. . . . (Scadding, 173.)[16]

Second, the coccyx must be judged "similar" to a functional structure in another organism. But how similar must the structures be and how is that similarity to be measured? It is a vague concept that can be shaped easily by one's presuppositions. Moreover, there is no reason why a Creator could not adapt similar designs for different purposes. To conclude that one structure is too similar to another to have been separately designed to fulfill a function requires an assumption about the Creator's modus operandi. It is, therefore, a theological assessment.

In his recent book, Hunter spotlights the metaphysical nature of such arguments. After citing comments by evolutionists about various alleged vestigial structures, he writes:

Behind this argument about why the patterns in biology prove evolution lurks an enormous metaphysical presupposition about God and creation. If God made the species, then they must fulfill our expectations of uniqueness and good engineering design. We might say that God was supposed to have optimized the design of each species. Evolutionists have no scientific justification for these expectations, for they did not come from science. They are part of a personal religious belief and as such are not amenable to scientific debate. In fact, evolutionists rely on a rather narrow metaphysical target in their attacks on creation. The evolutionist's notion of God and divine creation is, for many people, just a straw man—an overly simplified metaphysic that conveniently supports their views. (Hunter, 49.)

The suggestion that universal common ancestry would be falsified by finding "vestigial structures" in an organism that were not present in that organism's alleged ancestors, as depicted in the standard phylogeny, is incorrect (in that it is based on a false premise). To use one of Dr. Theobald's examples, if a fish species were discovered with a relatively small, nonfunctional leg or pelvis, it would only be labeled a "vestigial structure" if that species was judged to have "evolved back" from a tetrapod (i.e., if its branch on the phylogeny was relocated). Otherwise, it would be hailed as an example of a nascent structure, that is, a structure that is on its way "in" rather than on its way "out." Rather than being the death knell of common descent, it would be touted as evidence that tetrapods evolved from fish. Dr. Theobald sorely underestimates the flexibility of the theory he is asserting.

In fact, the absence of nascent structures poses a problem for Neo-Darwinian common descent. If, as Dr. Theobald says, "functions necessarily have been gained and lost" throughout evolutionary history, why does one find evidence only of degeneration? As Wise says:

The absence of [nascent] organs would seem to argue that although we have evidence of degeneration from an earlier, more optimal design, we lack evidence of a move toward a new optimal design. It would seem that if an Intelligent Designer created optimal designs in the past and life's history has been a move away from that optimum, the presence of

vestigial organs and the absence of nascent organs would be better explained by intelligent design than by evolutionary theory. (Wise, 223.)

PREDICTION 7: MOLECULAR VESTIGIAL CHARACTERS

Vestigial characters should also be found at the molecular level. Humans do not have the capability to synthesize ascorbic acid (otherwise known as Vitamin C), and the unfortunate consequence can be the nutritional deficiency called scurvy. However, the predicted ancestors of humans had this function (as do all animals except primates and guinea pigs). Therefore, we predict that humans, primates, and guinea pigs should carry evidence of this lost function as a molecular vestigial character.

Just for the record, it is not true that all animals except primates and guinea pigs have the ability to synthesize ascorbic acid. That ability is lacking in some species of fish, birds, and bats and is present in some species of primates.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some organisms will have genes the function of which was lost in the course of the organism's evolutionary history.
2. Some organisms have genes the function of which was lost in the course of the organism's evolutionary history.

Since this is the concept of vestigial structure applied to genes, the preceding response is largely applicable. Vestigial genes are not a necessary result of all possible mechanisms of universal common descent, and since Dr. Theobald has chosen to argue for common ancestry without regard to any mechanism of descent, he cannot offer as evidence data that can be explained only by particular mechanisms of descent.

Moreover, even Neo-Darwinism does not demand vestigial genes; it simply accommodates them. If they did not exist, it would mean that an incapacitating mutation never occurred or never occurred in an environment that was selectively neutral in terms of the gene's function. If they did exist, it would mean the opposite. Any result can be fit within the scheme.

In any event, vestigial genes provide no support for the claim of universal common ancestry. A bona fide vestigial gene says only that the organism in which it is found descended from an earlier organism that possessed the gene in functional form. It says nothing about how that earlier organism came to exist, whether it descended from a universal common ancestor, descended from one of many independently created organisms, or was itself created independently.

Consider Dr. Theobald's primary example, the L-gulano-g-lactone oxidase gene, which is one of the genes required for the synthesis of vitamin C. Assuming this is a bona fide pseudogene in humans, meaning a nonfunctional version of a gene that was functional at

some point in the human lineage, it says nothing about the origin of the ancestor that possessed the functioning gene. That ancestor could have been independently created or could have descended from a creature that had been independently created. So this entire line of argument cannot do what Dr. Theobald needs it to do.

As with other vestigial structures, it is difficult to identify bona fide vestigial genes. We simply do not know enough to be able to declare definitively that any given series of nucleotides has absolutely no function. As molecular biologist Pierre Jerlstrom recently noted:

Pseudogenes are often referred to in the scientific literature as nonfunctional DNA, and are regarded as junk. But more scientists are now conceding that this is far from true for many pseudogenes. Failure to observe pseudogenes 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.)

The possibility of an undiscovered function has become even greater with the recent sequencing of the human genome. Though humans may have as many as 300,000 proteins, it turns out that they have only about 30,000 genes.[17] Thus, the genome is even more complex than previously believed. 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.

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.)

Indeed, the evolutionists' claim that pseudogenes are still present and recognizable tens of millions of years after they supposedly ceased functioning suggests that they serve some kind of purpose. Otherwise, they should have been removed or altered beyond recognition by the accumulation of mutations. Jerlstrom writes:

The persistence of pseudogenes is in itself 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 expect that 'old' pseudogenes would be scrambled beyond recognition as a result of accumulated random mutations. Moreover, a removal mechanism for neutral DNA is now known. (Jerlstrom, 15.)

Granting the possibility that pseudogenes have a function, the claim that they are a vestige of evolutionary history reduces to the notion that a Creator would not fulfill a function in one organism by using a series of nucleotides that are similar to a series of nucleotides that fulfill a different function in another organism. That, however, is a theological argument. Hunter makes the point well:

A pseudogene is a DNA sequence that resembles a gene but appears to be nonfunctional. In evolutionary lore, these are vestigial organs at the molecular level. And just as the vestigial organ argument for evolution relies on the assumption of full knowledge about the organism, so too the pseudogene argument assumes that we can be sure they are not useful. They are assumed to be the byproduct of useless, but not terribly harmful, mutations. Gray writes:

Further analysis shows that this gene is a pseudogene, i.e., it looks like a real gene, but it is not expressed due to a mutation in the gene itself. Now we could argue that in God's inscrutable purpose he placed that vitamin C synthesis look-alike gene in the guinea pig or human DNA or we could admit the more obvious conclusion, that humans and primates and other mammals share a common ancestor.

Here Gray makes a negative theological argument. He seems comfortable in assuming just what God would have done when it comes to designing the genotype. Gray states unequivocally that the pseudogene is a result of mutation, but this is nothing more than evolutionary speculation. More important, he then claims that God obviously would not have an inscrutable purpose for having the nonstandard gene there. For our purposes the point is not that pseudogenes do or do not have function or that God must have or must not have designed them. The point is simply that, like evolutionists, theistic evolutionists need Darwin's negative theology. (Hunter, 168-169.)

Even if one could be certain that a gene was functionless (a pseudogene) and had been rendered such by a specific mutation, finding that same gene and mutation in another species would not mean that those species had descended from a common ancestor. The same gene could have been 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 (or was) 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 past and present mechanisms of mutation). So even conclusions about common ancestry that are based on the presence of similar, bona fide pseudogenes must remain tentative.[18]

For example, molecular biologist Michael Brown believes there is evidence for the existence of either viral or enzymatic activity that creates mutations. He writes:

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.[19]

The point is not that Brown's opinion necessarily is correct but that it (or something analogous) may be correct. Our understanding is just too rudimentary to permit us to say with certainty that similar pseudogenes were not caused independently by a nonrandom mechanism.

The fact some pseudogene-derived phylogenies disagree is consistent with the suggestion that something other than common descent is involved in the phenomenon. Phylogenies based on several pseudogene sequences have yielded conflicting results with regard to the human-chimp-gorilla trichotomy. (Woodmorappe 2000, 62-63.) Of course, evolutionists have ways of accommodating these discordant data, but their presence remains noteworthy.

PREDICTION 8: ONTOGENY AND DEVELOPMENT OF ORGANISMS

Embryology and developmental biology have provided some fascinating insights into evolutionary pathways. Since the cladistic morphological classification of species is generally based on derived characters of adult organisms, embryology and developmental studies provide a nearly independent body of evidence.

The ideas of Ernst Haeckel greatly influenced the early history of embryology; however, his ideas have been superseded by those of Karl Ernst van Baer, his predecessor. Van Baer suggested that the embryonic stages of an individual should resemble the embryonic stages of its ancestors (rather than resembling its adult ancestors, a la Haeckel). The final adult structure of an organism is the product of numerous cumulative developmental processes; for species to evolve, there necessarily must have been change in these developmental processes. The modern developmental maxim is the inverse of Haeckel's

biogenetic law. "Phylogeny recapitulates Ontogeny," not the opposite. Walter Garstang stated even more correctly that ontogeny creates phylogeny. What this means is that once given knowledge about an organism's ontogeny, we can confidently predict certain aspects of the historical pathway that was involved in this organism's evolution (Gilbert 1997, pp. 912-914). Thus, embryology can provide confirmations and predictions about evolution.

Two different concepts seem to be mixed here. On the one hand, there is the suggestion that descendant ontogenies tend to recapitulate ancestral ontogenies (Garstang's notion of paleogenesis). This is the claim that all vertebrates, for example, are very similar at an early stage of embryological development, with noticeable differences coming only in later stages. The more closely related the species being compared, the longer their embryos will develop similarly. The more distantly related the species, the sooner their embryos will diverge in appearance.

On the other hand, there is the suggestion that the embryos of organisms develop in ways that exhibit aspects of the organism's evolutionary history.[20] Thus, Dr. Theobald points to the fact certain reptile jaw bones and marsupial middle ear bones develop from the same embryological structures as evidence that the middle ear bones of mammals evolved from the jaw bones of reptiles.

The alleged prediction and fulfillment under the first concept are:

1. If universal common ancestry is true, then all ontogenies will begin similarly, and the ontogenies of more closely related species will remain similar longer than will the ontogenies of more distantly related species.
2. All ontogenies begin similarly, and the ontogenies of more closely related species remain similar longer than do the ontogenies of more distantly related species.

The alleged prediction and fulfillment under the second concept are:

1. If universal common ancestry is true, then certain aspects of an organism's evolutionary history will be exhibited in its ontogeny.
2. Certain aspects of an organism's evolutionary history are exhibited in its ontogeny.

There is nothing about the hypothesis of universal common ancestry that requires any particular manner of reproduction, let alone one in which embryos either recapitulate the ontogenies of their ancestors or pass through stages representative of their evolutionary history. Common ancestry can accommodate such phenomena, but it certainly does not predict it. And if it does not predict the phenomena, it cannot be falsified by their absence or confirmed by their presence.

Even if one could rightly claim these as predictions of the hypothesis of universal common ancestry, they are too general to be scientifically meaningful. How does one measure objectively the similarities of various ontogenies? What specific aspects of an organism's evolutionary history will be reflected in its ontogeny and why those aspects and not others?

And even if these ambiguities could be nailed down, views about "closely related species" and "evolutionary history" are tentative. So if all other avenues of accommodating the embryological data should fail, the option of revising phylogenies is always available. Falsifiability is again merely an illusion.

As for the first concept, the claim that ontogenies of organisms begin similarly and then progressively diverge in accordance with their alleged evolutionary proximity is false. Developmental biologist Jonathan Wells explains:

Although it is true that vertebrate embryos are somewhat similar at one stage of their development, at earlier stages they are radically dissimilar. After fertilization, animal embryos first undergo a process called cleavage, in which the fertilized egg divides into hundreds or thousands of separate cells. During cleavage, embryos acquire their major body axes (e.g., anterior-posterior, or head to tail, and dorsal-ventral, or back to front). Each major group of animals follows a distinctive cleavage pattern; among vertebrates, for example, mammals, birds, fishes, and reptiles cleave very differently.

Animal embryos then enter the gastrulation stage, during which their cells move relative to each other, rearranging themselves to generate basic tissue types and establish the general layout of the animal's body. The consequences of this process are so significant that embryologist Lewis Wolpert has written that "it is not birth, marriage, or death, but gastrulation which is truly the important event in your life" (Wolpert 1991, 12). Like cleavage patterns, gastrulation patterns vary markedly among the major groups of animals, including the different classes of vertebrates (Elinson 1987).

Only after gastrulation do the embryos of mammals, birds, fishes and reptiles begin to resemble each other. In the pharyngula stage, every vertebrate embryo looks vaguely like a tiny fish, with a prominent head and a long tail. (Wells 1998, 59.)

Reviewing the notion that "during their ontogenies the members of twin taxa follow the same course up to the stage where they diverge into separate taxa," embryologist Wolfgang Dohle wrote:

Everybody who is even slightly acquainted with ontogenetic facts knows that there are hundreds of examples to which this theorem does not apply. In many polychaete and prosobranch genera one species develops through a planktonic larva, whereas another species has direct development. The telolecithal cephalopod eggs cleave in a bilateral manner without any similarity to the spiral cleavage of other related Mollusca. Triclad eggs have a blastomeric anarchy, whereas the adults very closely resemble the polyclads which show spiral cleavage. This list could easily be elongated. (Dohle, 285.)

Wells points out that this ontogenetic pattern of early differences followed by similarities and then differences again "is quite unexpected in the context of Darwinian evolution." He adds, "Instead of providing support for Darwin's theory, the embryological evidence presents it with a paradox." (Wells 2000, 99.) Of course, attempts are being made to explain the paradox by proposing that early development evolves more easily than expected, but as Wells notes, "it is clear that [these proposed explanations] start by assuming Darwinian evolution, then read that back into the embryological evidence." (Wells 2000, 99.)

Of course, this is the exact opposite of basing evolutionary theory on embryological evidence. If one were to start with the evidence and then follow Darwin's reasoning about the implications of development for evolution, one would presumably conclude that the various classes of vertebrates are not descended from a common ancestor, but had separate origins. Since this conclusion is unacceptable to people who have already decided that Darwin's theory is true, they cannot take the embryological evidence at face value, but must re-interpret it to fit the theory. (Wells 2000, 101.)

Even if one ignores the paradoxical hourglass pattern of vertebrate ontogenies (the fact they start out looking very different, converge in appearance midway through development, and then increasingly diverge toward adulthood) and focuses only on the latter half of development, common ancestry is not the only explanation for the gradual divergence of different species. As Brand explains:

[A] home builder builds the foundation first (homes that look very different when complete can have similar foundations) and adds the unique features of the home later. An engineer attempting to design the developmental stages of all these organisms would very possibly find that it is most efficient to follow a basic plan for all and add special features later in the process, as needed for each animal. (Brand, 150.)

In addition, if Dr. Theobald's assertion that "[t]he final adult structure of an organism is the product of numerous *cumulative* developmental processes" (emphasis supplied) is correct, then organisms that are thought to be more evolutionarily derived would take longer to develop. But as Wise points out, that is not the case. "[O]rganisms that are thought to be more evolutionarily derived don't seem to have longer development." (Wise, 216.)

As for the second concept, the claim that "certain aspects" of an organism's evolutionary history are exhibited in its ontogeny (such as the alleged evolution of the mammalian middle ear from reptile jaw bones) is nothing more than an opinion. Common descent is not the only explanation for the fact separate structures in adults of different species develop from an analogous embryological structure. It is certainly possible for a designer to fabricate different structures from similar elements. A manufacturer, for example, can make the tops of one kind of footwear and the laces of another from the same nylon.

The notion that God would not employ an ontogeny in which the middle ear bones of mammals develop from the embryological structure that is analogous to that from which

reptile jaw bones develop is a theological assessment. Those who reject that assessment are rightly unimpressed by evidence that draws its weight from it.

Moreover, it is unclear in what sense the ontogenetic development of mammalian middle ear bones from a structure analogous to that from which certain reptile jaw bones develop can be said to exhibit the evolution of the mammalian middle ear from the reptile jaw. The evolutionary claim is that the quadrate and articular bones of the reptilian jaw were (on two separate occasions) gradually transformed into ear ossicles through many random steps, each of which provided a significant enough advantage to be established in the population. There is no need, however, for the intermediate stages of an embryo's development to be progressively advantageous, as they are part of a directed development program that unfolds in a protective environment. The two thus seem quite unrelated.

PREDICTION 9: PRESENT BIOGEOGRAPHY

Because species divergence happens not only in the time dimension, but also in spatial dimensions, common ancestors originate in a particular geographical location. Thus, the spatial and geographical distribution of species should be consistent with their predicted genealogical relationships. The standard phylogenetic tree predicts that new species must originate close to the older species from which they are derived. Closely related contemporary species should be close geographically, regardless of their habitat or specific adaptations. If they are not, there had better be a good explanation, such as extreme mobility (cases like sea animals, birds, human mediated distribution, etc.), continental drift, or extensive time since their divergence. In this sense, the present biogeographical distribution of species should reflect the history of their origination.

A reasonable nonevolutionary prediction is that species should occur wherever their habitat is. However, macroevolution predicts just the opposite—there should be many locations where a given species would thrive yet is not found there, due to geographical barriers (Futuyma 1998, pp. 201-203).

The standard phylogenetic tree does not "predict that new species must originate close to the older species from which they are derived." A phylogeny is simply a diagram that depicts current evolutionary thinking about the genealogical relationships of the taxa included. It does not address questions of geographical proximity.

It is unclear (at least to me) what is being claimed here. On the one hand, there is the suggestion that the geographical distribution of species should be consistent with their believed (not "predicted") genealogical relationships. Thus the statement, "Closely related contemporary species should be close geographically, regardless of their habitat or specific adaptations."

On the other hand, there is the suggestion in the second quoted paragraph that only macroevolution (which Dr. Theobald labels a "virtual synonym" for universal common descent) can explain why some species live only in certain areas, despite the existence of

similar habitat elsewhere. This point relates solely to the location of "given species," not to their distance from "closely related contemporary species."

It is not spelled out how these two propositions translate into support for the hypothesis of universal common ancestry. Presumably they are to be merged into something like the following: only universal common ancestry can explain the fact groups of similar species are often restricted to a particular geographic region. In that case, the alleged prediction and fulfillment can perhaps best be expressed as:

1. If universal common ancestry is true, then groups of similar species will often be restricted to a particular geographic region.
2. Groups of similar species are often restricted to a particular geographic region.

The fact groups of similar species are often restricted to a particular geographic region is not evidence of universal common ancestry. At best, it suggests that the similar species arose in that region from a common ancestor. It says nothing about whether that regional common ancestor descended from a universal common ancestor, descended from one of many independently created organisms, or was itself created independently. It therefore does not do what Dr. Theobald needs it to do.

In fact, the degrees of evolution suggested by biogeography are quite limited. As biologist L. James Gibson notes, "Geographical distributions indicate common ancestry only for lower taxonomic categories. Endemic groups are most common at the Family level or lower. Few Orders are endemic to a particular region. Thus common ancestry is suggested primarily among members of families and genera."[21]

Wise makes the point this way:

There are two sorts of biogeographical evidences. One type is the claim that very similar species are often found near one another, as if they evolved from one another. This type of biogeography, which I call *microbiogeography*, has many supporting examples. Microbiogeography is evidence for microevolution (the evolution of populations) and the origin of species, however, not for macroevolution of the origin of major groups. What I call *macrobiogeography* is the claim that major types of organisms tend to be associated with one another.

There are very few examples of macrobiogeographical evidences for macroevolution, and none of them is very strong. (Wise, 223.)

The best known claim of macrobiogeographical evidence is the one cited by Dr. Theobald—the concentration of marsupials in Australia. But as Wise explains, "there are several reasons that marsupials in Australia are actually a poor example."

First, all marsupials are not in Australia. The Virginia opossum of North America, for example, is a marsupial. It is thought to have come from South America, not Australia.

Thus, not all similar organisms are in the same area. Second, in the fossil record marsupials are known from every continent. Third, marsupials are the oldest fossil mammals known from Africa, Antarctica and Australia—in that order. The fossil record seems to show a migration of marsupials from somewhere around the intersection of the Eurasian and African continents and then a survival in only the continents farthest from their point of origin (South America and Australia). The same major groups of marsupials (opossums) are found in both South America and Australia. Macroevolutionists claim that these major groups of marsupials are together because they evolved from a common ancestor, but the evidence can be at least as well explained as similar organisms (fit for similar environments and with similar capabilities) traveling more or less together to similar environments. (Wise, 223.)

All of Dr. Theobald's other examples involve endemic groups at lower taxonomic categories (species of lungfishes, ratite birds, leptodactylid frogs, alligators, giant salamanders, magnolias, cacti, and pineapples). Of course, creationists of all stripes accept speciation or diversification within created kinds (understanding that both "species" and "created kinds" are nebulous concepts).

Dr. Theobald acknowledges that species of alligators, giant salamanders, and magnolias occur half a world apart, but he still counts them as "close" species because it is hypothesized that Eastern North America and East Asia were once close together. There are many other seemingly anomalous geographical occurrences of the same or similar species that are explained with speculative hypotheses.[22]

The flexibility of biogeography is well illustrated by the fact it was used as support for evolution prior to the acceptance of plate tectonics and continental drift. As ReMine explains:

One reason for this erosion [in the assessment of the importance of biogeography as evidence for evolution] was the 1960s development of plate tectonics and continental drift. This development radically changed the picture, and forced evolutionists to rapidly restructure biogeographical ideas away from the fixed continents axiom of Darwin and Wallace. The biogeographers tried to reconcile their data with the new concepts of movable paleogeography. The plasticity of evolutionary biogeography was nakedly revealed by its ability to suddenly adapt to the dramatic shift in geologic understanding. (ReMine, 437.)

Though biogeography has from the earliest days of the theory been touted as strong evidence for evolution, Nelson and Platnick wrote in 1981: "We conclude, therefore, that biogeography (or geographical distribution of organisms) has not been shown to be evidence for or against evolution in any sense." (Nelson and Platnick, 223.) Perhaps that explains why "Mark Ridley has an entire chapter on biogeography in his evolution textbook [see bibliography] but does not use biogeography as one of his evidences for evolution." (Hunter, 184 n.64.)

The assertion in the second quoted paragraph that under nonmacroevolutionary theories species should exist wherever there is suitable habitat for them is groundless. It not only ignores the room nonmacroevolutionary theories can leave for microevolution and ignores the complexity of factors that can shape the distribution of species, it is apparently based on a theological presupposition. Hunter is again worth quoting on the subject:

And for Michael Ruse, God cannot be reconciled with the facts of biogeography, so we must turn to evolution. He argues, "Given an all-wise God, just why is it that different forms appear in similar climates, whereas the same forms appear in different climates? It is all pointless without evolution." According to Edward Dodson and Peter Dodson, if God created the species, then they should be distributed uniformly around the globe. They write, "had all species been created in the places where they now exist, then amphibian and terrestrial mammals should be as frequent on oceanic islands as on comparable continental areas. Certainly, terrestrial mammals should have been created on these islands as frequently as bats were." It is remarkable how often evolutionists feel free to dictate what God should and shouldn't do. (Hunter, 113.)

Finally, the suggestion that universal common ancestry would be falsified by finding elephants or amphibians on remote islands is incorrect. Their presence would be explained in a way consistent with evolutionary convictions. Ideas of continental drift, land bridges, human involvement, storm rafts, unknown swimming ability, some form of hitchhiking, etc. would be invoked and judged more likely than the alternative. In fact, many amphibians exist on many islands, and their presence is not viewed as a threat to the evolutionary hypothesis.

PREDICTION 10: PAST BIOGEOGRAPHY

Past biogeography, as recorded by the fossils that are found, must also conform to the standard phylogenetic tree. As on [sic] example, we conclude that fossils of the hypothetical common ancestors of South American marsupials and Australian marsupials should be found dating from before these two landmasses separated.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the geographic distribution of fossil species will conform to the standard phylogenetic tree.
2. The geographic distribution of fossils conforms to the standard phylogenetic tree.

As I have pointed out, it is not a prediction of the hypothesis of universal common ancestry that organisms, past or present, will conform to the standard phylogenetic tree. Rather, the standard phylogenetic tree is a depiction of current evolutionary thinking about the genealogical relationship of the taxa included. Phylogenies are provisional, evolutionary constructs of data, including biogeography. If facts develop that make some

aspect of a phylogeny untenable, adjustments will be made within the evolutionary framework and a new orthodoxy will be established.

Judging by the example given, the geographic distribution of fossil species is deemed to conform to the standard phylogenetic tree if the date and location of alleged ancestors do not render impossible the claim of descent. Thus, if Australian marsupials originated on another continent, one would expect to find marsupials on that continent prior to the time Australia is believed to have become geographically isolated.

The presence of similar marsupials in South America, Antarctica, and Australia may be evidence for the claim that the continents were at one time contiguous, but it is not evidence for the hypothesis of universal common ancestry. It is not even evidence for the claim that all Australian marsupials arose from a common ancestor.

"There is no direct evidence to document when marsupials first entered Australia." (Carroll, 431.) When they first appear in the fossil record of Australia in the late Oligocene, "the major groups had already differentiated," and "the interrelationships of the various lineages have not been satisfactorily established." (Carroll, 431, 440.) According to Carroll, marsupials may have entered Australia "at least 40 million years" before there is a record of their presence. (Carroll, 435.)

There is thus no way to tell what marsupials entered Australia or when they did so. If representatives of the major groups of Australian marsupials were included among the immigrants, Australia would at most bear witness to diversification at lower taxonomic levels.

Finding marsupial fossils in Antarctica establishes that there were once marsupials on that continent, but it says nothing about their having evolved from some nonmarsupial stock or even about their having given rise to distinct marsupial suborders. If South America, Antarctica, and Australia were once contiguous and if one hypothesized that marsupials radiated from South America into Australia (or vice versa), then one would predict that marsupials once existed in Antarctica. Finding that to be the case certainly is not "an astounding macroevolutionary confirmation."

Determining the place of origin and direction of marsupial dispersal is not a simple matter of tracking the date of known fossils (i.e., they arose in North American and migrated to Australia). As Carroll's statement about the entry of marsupials into Australia makes clear, the absence of fossils is not necessarily evidence of the absence of marsupials.[23] That is why "[t]he place of origin and direction of dispersal of marsupials in the southern continents is subject to continuing debate." (Carroll, 431.)

The uncertainty surrounding this issue is apparent from the remarks of Clemens, Richardson, and Baverstock:

Against this background of ignorance and uncertainty, no definitive evaluation of the competing hypotheses concerning the time and place of the origin of the marsupials can

be presented. Almost every continent on which marsupials have existed has been nominated as an area of origin of the group. . . .

Currently, hypotheses suggesting origin and early radiation of marsupials in the southern continents, particularly Australia, Antarctica and South America are favoured by many workers. (Clemens and others, 542.)

Under the heading "Potential Falsification," Dr. Theobald writes, "We confidently predict that fossils of recently evolved animals like apes and elephants should never be found on South America, Antarctica, or Australia (excepting, of course, the apes that travel by boat)." But finding fossil apes or elephants on one or more of those continents would not falsify universal common ancestry.

Any such find would, of course, face an extreme standard of proof in terms of both identification and dating (the more recent the date the more readily the explanation of human involvement would be accepted). Assuming the problem could not be avoided by denying the identification or dating, a devotee of common ancestry could revise his theory of continental drift or historical geography, propose temporary land bridges, reconsider when apes or elephants arose, suggest parallel or convergent evolution, etc. However problematic any of these proposals may be, they would be considered more likely than the alternative, i.e., that universal common ancestry is false.

The fact geography does not rule out the possibility that the modern horse descended from *Hyracotherium* provides no support for the claim of universal common ancestry. Assuming that *Equus* descended from *Hyracotherium,* that is merely diversification within the family Equidae. The change from *Hyracotherium* to *Equus* is trivial compared to the changes required by the theory of universal common ancestry.

Again, the claim under the heading "Potential Falsification" that it would be "macroevolutionarily devastating" to find an equid in South America before about 12 million years ago underestimates the theory's flexibility. It has great capacity for accommodating seemingly discordant data. Various hypotheses for fitting the data within an evolutionary framework would circulate, and the issue would be considered "a problem" until a consensus was reached regarding the solution. It would not, however, be judged a threat to (and certainly not a falsification of) the evolutionary paradigm.

Notes for Part 2

[15] Creationists, for example, agree that bona fide vestigial structures exist, but they believe those structures are strictly the result of degenerative changes within created kinds (e.g., blind salamanders and fish, flightless birds and beetles).

[16] Scadding's more theoretical objection is that one cannot arrive scientifically at the negative assertion that an organ has no function.

[17] There is some uncertainty about this number, but the fact two competing teams came up with roughly the same estimate (30,000 - 40,000) supports its accuracy. A recent letter published in Cell argues that the figure is too low. While Michael Cooke, one of the authors of the letter, believes 30,000 is too low, "he estimates the total is probably not more than 60,000," which is only slightly less amazing. Researchers who came up with the original figure are sticking with it. Daniel Q. Haney, "Researchers Question Report on Genes," Washington Post (August 23, 2001).

[18] For a fuller and more technical discussion of these and other issues, see the articles by Gibson, Walkup, and Woodmorappe (2000) listed in the bibliography. These articles are also available online at http://www.grisda.org/origins/21091u.htm, http://www.answersingenesis.org/home/area/magazines/tj/tjv14n2_junk_dna.pdf, and http://www.answersingenesis.org/Home/Area/Magazines/tj/docs/tj14_3-jw_pseudo.pdf, respectively.

[19] These comments are from an email posted at Dr. Brown's website.

[20] In saying that one can "predict" certain aspects of an organism's evolutionary history from its ontogeny, Dr. Theobald means that the embryos of organisms develop in ways that exhibit aspects of the organism's evolutionary history. This is clear from what he cites as fulfillment.

[21] From L. James Gibson, "Creation and Evolution: A Look at the Evidence".

[22] For example, three of the four species of tapirs are restricted to Latin America (southern Mexico to Brazil), whereas the fourth species inhabits Burma, Thailand, Malaya, and Sumatra. Giant tortoises are found on the Galapagos islands and on the island of Aldabra (near Madagascar). Species of coelacanths are known only in Indonesia and the western Indian Ocean. Lungless salamanders live in the Western Hemisphere and southern Europe. The list could be extended easily.

[23] This is also apparent from the fact marsupial presence in southeastern Asia is known from only one fossil, which was reported in 1992 (see, S. Ducrocq and others).

A Critique of Douglas Theobald's
"29 Evidences for Macroevolution"

by Ashby Camp

Part 3

"Evolutionary Opportunism"

PREDICTION 11: ANATOMICAL PARALOGY

One major consequence of evolutionary opportunism is the predicted existence of paralogy. Paralogy, as I use the term here, is similarity of structure despite difference in function. When one species branches into two species, one or both of the species may acquire new functions. Since the new species must recruit and modify preexisting structures to perform these new functions, the same structure shared by these two species will now perform a different function in each of the two species. This is paralogy. It follows that paralogous structures have a history that should be explicable from other lines of evolutionary evidence, since derived characteristics (which is what these new functions and structures now are) have evolved from more primitive (i.e. older) structures. Consequently, detailed and explicit predictions can be made about the possible morphologies of fossil intermediates.

An equivalent way of stating the principle of evolutionary paralogy is that the predicted phylogenetic tree must have structural continuity, as opposed to functional continuity. Structures and patterns are inherited, but not necessarily functions. As one follows the line from ancestor through descendants, the functions and forms can come and go, but the underlying structures must grade from one into another, relatively unchanged.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some species will have structures that are similar to structures in other species but which perform different functions in the other species.
2. Some species have structures that are similar to structures in other species but which perform different functions in the other species.

"Paralogous structures"[24] are not a necessary result of all possible mechanisms of universal common descent. They are understandable within a Neo-Darwinian framework of random mutation and natural selection, but since Dr. Theobald has chosen to argue for common ancestry without regard to any mechanism of descent, he cannot offer as evidence data that can be explained only by particular mechanisms of descent.

Moreover, even Neo-Darwinism does not demand paralagous structures; it simply accommodates them. Putting aside the ambiguity of "similar," which can span even the gulf between reptilian and avian respiration systems, if hands, wings, flippers, and legs (to cite Dr. Theobald's examples) did *not* share a "similar" pattern of construction, it simply would produce a different evolution story. Various reasons would be offered as to why the basic limb structure was not conserved in these lineages. I can imagine the structural differences being touted as proof of the blindness of the evolutionary process. What I cannot imagine is this lack of conservation causing evolutionists to lose any sleep.

One cannot take a known pattern of life, claim that pattern as a *prediction* of evolution, and then use the fact the pattern fits the prediction as evidence for the truth of evolution. To be of evidentiary value, the predictions must derive from the hypothesis itself, not be read back into the hypothesis from present knowledge. This is an especially daunting task for Dr. Theobald, since he disavows reliance on any particular mechanism of descent.

Even if one granted that "paralagous structures" indicate a common ancestry for those possessing them, they say nothing about the origin of that common ancestor, whether it descended from a universal common ancestor, descended from one of many independently created organisms, or was itself created independently. Therefore, the argument does not advance the proposition of universal common ancestry. It is like arguing that everyone at a party in Los Angeles came from New York by showing that groups of them arrived from various cities between the two.

In any event, common ancestry certainly is not the only explanation available for "paralagous structures." As biologists Percival Davis and Dean Kenyon explain:

[T]he existence of homologous structures[25] merely raises questions of relationship, but it cannot answer them. This is why Stephen Gould remarked that homology supports common design as well as it does common ancestry. [S. J. Gould, *Natural History*, January 1987, 14.] Both Darwinists and design proponents can explain the existence of homologies within their respective frameworks of interpretation. Because of this, neither side can disprove the other's interpretation of homology, and neither view stands solely on its own interpretation of homology. (Davis and Kenyon, 133.)

The standard creationist understanding of the phenomenon is summarized by Brand:

How did the limb bones of those four mammals (the human, seal, bat, and dog in Fig. 9.7) develop the way they did? An engineer devising different kinds of machines wouldn't start from scratch for every machine. The data indicate that if an intelligent Creator designed the limb system for vertebrates, He developed a flexible general plan which could be adapted for the lifestyle of each animal. The result is a series of homologies from the work of a common Designer who created all of these animals in an organized fashion. (Brand, 156-157.)

It is interesting that the forelimbs and hindlimbs of a terrestrial vertebrate are strikingly similar in design,[26] but no evolutionist attributes that similarity to common descent. Rather, they believe the two patterns arose independently from the pectoral and pelvic fins of a fish. The creationist links the similar limb designs through a common Creator, whereas the evolutionist is left to appeal to chance and unspecified selective pressures.

The suggestion that a Creator would not use similar designs for different functions is blatantly theological. Those who do not share that opinion will be unimpressed by evidence that draws its weight from it. To repeat a previous quote:

Behind this argument about why the patterns in biology prove evolution lurks an enormous metaphysical presupposition about God and creation. If God made the species, then they must fulfill our expectations of uniqueness and good engineering design. We might say that God was supposed to have optimized the design of each species. Evolutionists have no scientific justification for these expectations, for they did not come from science. They are part of a personal religious belief and as such are not amenable to scientific debate. In fact, evolutionists rely on a rather narrow metaphysical target for their attacks on creation. The evolutionist's notion of God and divine creation is, for many people, just a straw man—an overly simplified metaphysic that conveniently supports their views. (Hunter, 49.)

In fact, ReMine argues that use of similar designs for different functions is actually evidence for creation in that it is an essential part of the Creator's biotic message. He believes life was designed both for survival and for communicating a message about where life came from. The content of that biotic message is that living objects were constructed by a single source and that they did not result from naturalistic processes. Nested hierarchy is an important part of the message for a number of reasons (see, ReMine, 368 and 465 for a summary),[27] and that makes it necessary for similar designs sometimes to be used for different functions.

The answer [to why a designer would sometimes use the same design to accomplish a different purpose] is simple. The designer must use the same design to accomplish different purposes because the nested pattern requires it. The nested pattern places demands on the occasions when a biomessage sender must use shared design, and when shared design is prohibited. Therefore, on occasion the designer is forced to use the same design for different purposes. (ReMine, 364.)

Regardless of whether one accepts ReMine's thesis, it offers a glimpse into the potential complexity of creative purposes. It is a weak argument indeed that assumes complete comprehension of those purposes.

The claim that "[t]he fossil record shows a chronological progression of intermediate forms between theropod dinosaurs and modern birds, in which theropod structures were modified into modern bird structures" is incorrect. No dinosaur with particularly avian affinities is known before the Late Jurassic, making them contemporaries of

Archaeopteryx, and those with the most birdlike characteristics do not occur until much later.[28]

The suggestion that the hypothesis of universal common ancestry would be falsified if the fossil record showed "a chronological progression in which bird wings are gradually transformed into reptilian arms" is incorrect. If evolution can make a reptile into bird, there is no reason it cannot modify wings into reptilian arms. In fact, some experts have actually suggested that coelurosaurs were derived from primitive birds. (Fallow, 108-110; Feduccia, 90.)

Neither would the hypothesis of universal common ancestry be falsified if it were demonstrated that the "primitive structures of an organism's predicted ancestors could not be reasonably modified into the modern organism's derived structures." That is precisely what happened in the case of *Coelurosauravus,* and yet evolutionists did not view it as a threat to their theory.[29] Evolution can accept the development of novel structures and does so whenever necessary to accommodate the data.

Contrary to Dr. Theobald's assertion, it is not a prediction of universal common ancestry (or Neo-Darwinism) "that we should never find birds with both wings and arms, or mollusks harboring chloroplasts." If those things were found, they would be explained within the evolutionary framework. As ReMine says, "The frustrating thing about natural selection is that its theorists can refuse to be ingenious at the necessary places. They take a structure *known not* to exist, then they say that natural selection predicts it could not exist. This sudden lack of imagination is too convenient." (ReMine, 149.)

PREDICTION 12: MOLECULAR PARALOGY

The concept of paralogy applies equally to both the macroscopic structures of organisms and structures on the molecular level.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some species will have biological molecules that are similar in structure to biological molecules in other species but which perform different functions in the other species.
2. Some species have biological molecules that are similar in structure to biological molecules in other species but which perform different functions in the other species.

Since this is the concept of "paralagous structures" applied to biological molecules, much of the preceding response is applicable. Paralogous biological molecules are not a necessary result of all possible mechanisms of universal common descent. They are understandable within a Neo-Darwinian framework of random mutation and natural selection, but since Dr. Theobald has chosen to argue for common ancestry without

regard to any mechanism of descent, he cannot offer as evidence data that can be explained only by particular mechanisms of descent.

Moreover, even Neo-Darwinism does not demand paralagous biological molecules; it simply accommodates them. To use Dr. Theobald's example, if lysozyme and alpha-lactalbumin were *not* "similar," it simply would be assumed that alpha-lactalbumin arose by some other evolutionary path. Indeed, lactalbumin was long considered to have arisen de novo in the ancestor of mammals, and this caused no consternation in evolutionist ranks. If the similarities between lysozyme and lactalbumin had not been discovered, the old script would have continued in service of the evolution paradigm.

In any event, universal common ancestry is not the only explanation available for paralogous biological molecules. On what basis does one assert that a Creator would not use similar amino acid sequences and similar folds in the design of two separate proteins? Even if it was possible biochemically to design a protein that had all the functions of lactalbumin but was unlike lactalbumin in structure, a Creator could still opt to work from a lysozyme-like template. If, on the other hand, the structure of lactalbumin is dictated largely by its function, it could not be created without similarity to lysozyme. Either way, there is no basis for insisting that the similarity of these proteins is the result of common descent.

The example of the genetic comparisons between the baker's yeast (*Saccharomyces cerevisiae*) and the worm (*Caenorhabditis elegans*) is much the same. The opinion that the genes unique to *C. elegans* were derived from genes it shared with *S. cerevisiae* is driven by a commitment to evolution and a theological assumption. As Hunter said of a similar argument, "Ultimately it comes down to [the] belief that a Creator would not have created species with commonality—at bottom it is a religious argument." (Hunter, 100.)

The suggestion that universal common ancestry would be falsified if proteins performing more recently evolved functions did not "have homologues with proteins performing core functions" is incorrect. As already mentioned, lactalbumin was long considered to have arisen de novo in the ancestor of mammals, and evolutionists were unfazed. As with anatomical structures, evolution can accommodate both the presence and the absence of novel proteins.

The suggestion that universal common ancestry would be falsified "if we had found that genes involved in multicellular functions . . . were *more ancient* than the core function genes" (emphasis supplied) is trivial. By "core function genes" Dr. Theobald means "genes dealing directly with core biochemical functions *that all organisms must perform"* (emphasis supplied). If these genes are essential for life, then obviously no genes can predate them, as genes exist only in living things.

The suggestion that universal common ancestry would be falsified "if we had found that genes involved in multicellular functions were more deeply rooted in their phylogenies" is incorrect. It is already believed that some genes are rooted in an organism's phylogeny

more deeply than is the feature the gene is believed to control,[30] and yet evolutionists remain committed to their theory.

PREDICTION 13: ANATOMICAL CONVERGENCE

A corollary of the principle of evolutionary opportunism is convergence. Convergence is the case where different structures perform the same or similar functions in different species. Two distinct species have different histories and different structures; if both species evolve the same new function, they may recruit different structures to perform this new function. Convergence also must conform to the principle of structural continuity; convergence must be explained in terms of the structures of predicted ancestors.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some species will have structures that perform the same or a similar function performed by different structures in other species.
2. Some species have structures that perform the same or a similar function performed by different structures in other species.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that different species will have different structures that perform the same function.[31] As ReMine observes, "Evolutionary theory does not predict any adaptations, much less convergent adaptations." (ReMine, 141.) This is but 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. "Evolutionists merely *claim* that extensive convergence is virtually inevitable. They do this because 'convergence' is abundant in nature." (ReMine, 168.)

Moreover, "convergence" by definition involves two separate starting points ("distinct species") that "converge" on a given structure and/or function. It says nothing about how those starting points came to be separate. So whatever else one may make of the phenomenon, convergence is not evidence for universal common ancestry, the proposition being argued by Dr. Theobald.

The plasticity of evolutionary theory with regard to convergence is laid bare by ReMine:

Again, there is a difference between what evolutionary theory says (which is nothing coherent), and what evolutionists claim the theory says (which they conveniently choose to match the data). Evolutionary theory is flexible to whatever problem is at hand.

Here is an example.

Thus we should expect to see *many* different genetic solutions to *any* adaptive problem. (Futuyma, 1983, p. 126, my italics)

Indeed *many* of the environmental problems have *only a limited number* of genetic solutions . . . (Newell, 1982, p. 193, my italics)

Futuyma is explaining the plentiful existence of adaptive *variation* (called diversity). Newell is explaining the plentiful existence of adaptive *similarity* (called convergence). Therefore, they selected different explanations from the evolutionary smorgasbord. The two explanations happen to contradict each other.

The major "adaptation" in evolution is the way evolutionists adapt their theory to fit the data. Evolutionary theory can be easily adapted because it is untestable; its bonds are few, its flexibility is great. It is bent and molded by the needs of evolutionary storytelling. (ReMine, 142.)

In fact, convergence poses quite a challenge to Neo-Darwinism, which is why "evolutionists avoid the convergence explanation when they can." (ReMine, 269.) As Wise explains:

[A]nalogies[32] are being found to be a very common feature of life. Every tree that takes into account at least a couple dozen features and includes several major groups of organisms seems to encounter several noninherited similarities.

Considering the fact that organisms are composed of millions or even billions of features, the true number of analogies is likely to be extremely high. Yet this does not seem to be consistent with evolutionary theory. In an evolutionary scenario, analogies are features formed independently in two different organismal groups. The pathway that evolution takes is thought to be fraught with unpredictable events that the likelihood that two separate evolutionary pathways will end up at the same place is thought to be very low. This is the major theme of Stephen Jay Gould's book *Wonderful Life.* If the evolutionary process were run over again, one would not expect to get the same organisms again. Only when a feature is extremely advantageous to the organism and easy to produce naturalistically can it be considered reasonable that it could have evolved more than once. Most features, however, are so very complex that it is not clear that any of them could be so easily produced as to make even one a probable event. In evolutionary theory analogies would be expected to be a very uncommon feature of life. (Wise, 213.)

Biophysicist Lee Spetner has attempted to quantify the magnitude of the challenge convergence poses to evolutionary theory. In a nutshell, he claims that, if evolutionists ascribe a positive selective value to enough potential mutations to make speciation conceivable, then it becomes essentially impossible for similar traits to arise independently (because the number of potential pathways is too great). (Spetner, 85-124.) In his words, "if the variation arises from random copying errors, convergent evolution is impossible." (Spetner, 110.) A summary of Spetner's argument is available online.

[T]he explanations [of convergence offered by evolutionists] go no further than sweeping generalities about how evolution "in due course selects the most efficient design for the

animal's or plant's lifestyle in the particular set of the environmental circumstances." In fact, evolutionists seem to be quite content with this explanation. As Berra concludes: "Such close similarities in very unrelated groups are easily explained as a result of convergent evolution." Perhaps too easily. Though evolution is a blind process that produces a broad menagerie of species and designs, it is also supposed to produce striking similarities. (Hunter, 31.)

On the other hand, if "the diversity of life is due to an Intelligent Designer's creating a number of distinct organisms, analogies should be common, as is observed, and as will be more commonly recognized with time." (Wise, 213.) Brand puts it this way:

Different kinds of wings are analogous because the Designer gave different kinds of organisms some of the same abilities. He made insects with a body plan different from mammals and birds, but some representatives of each group were made to fly. Because of their different underlying structural organization, their flight mechanisms are analogous, not homologous. (Brand, 156.)

ReMine argues that convergences are a crucial aspect of the Creator's biotic message. They serve the goals of that message in that they unify life, thwart phylogeny, and resist naturalistic explanation. (ReMine, 261-262, 264, 351, 354, 367.) He writes:

To successfully send the biotic message, a designer must not indiscriminately use the same design repeatedly. Therefore, the designer is forced to sometimes use different designs to perform the same function. The wings of bats, birds, and pterodactyls make a classic example. . . .

The bat's wing is made by lengthening four fingers, while the pterodactyl's wing is made by lengthening only one finger (what would be our little finger), and the bird's wing is made by diminishing the hand and providing it with feathers. Evolutionists claim these are evidences of evolution. They claim that a capable designer would not experiment with different designs.

The evolutionists are mistaken. A biomessage sender has every reason to design this way:

- The similarity of these three organisms cannot be denied. They are variations on a theme, and possess a common body plan. This sends the unifying message.
- These organisms are systematically placed (regarding all other organisms) so their common possession of wings cannot be explained by common descent. This sends the non-naturalistic message.
- The wing designs are sufficiently different that they cannot be explained by transposition. This sends the non-naturalistic message. This is precisely their difficulty for

evolution. If the wings were identical, then there would be no trouble rationalizing them by transposition.

The biomessage sender made these organisms difficult for evolutionists to explain. Evolutionists are left to account for the evolution of wings (and flight!) separately for each case. Rather than being evidence for evolution, these organisms are clean evidence for message theory. (ReMine, 354.)

The suggestion that universal common ancestry would be falsified by the discovery of a "close mammalian relative" that possessed gills is incorrect. It would be explained within the evolutionary framework, either by assuming the creature was related to mammals more distantly or by appealing to the amazing transforming powers of natural selection. To repeat ReMine's comment, "The frustrating thing about natural selection is that its theorists can refuse to be ingenious at the necessary places. They take a structure known not to exist, then they say that natural selection predicts it could not exist. This sudden lack of imagination is too convenient." (ReMine, 149.)

PREDICTION 14: MOLECULAR CONVERGENCE

Like paralogy, convergence should be represented on both macroscopic and molecular levels.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some species will have biological molecules that perform the same function performed by different biological molecules in other species.
2. Some species have biological molecules that perform the same function performed by different biological molecules in other species.

This is the concept of anatomical convergence applied to biological molecules, so much of the preceding response is applicable. Again, it is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that different species will have different biological molecules that perform the same function. To paraphrase ReMine, evolutionary theory does not predict any particular biological molecules, let alone convergent biological molecules. Since the theory does not predict the phenomenon, it cannot be falsified by its absence or confirmed by its presence.

As already noted, "convergence" by definition involves two separate starting points (distinct molecules) that "converge" on a given structure and/or function. It says nothing about how those starting points came to be separate, whether by separate creation or common descent. Therefore, it is not evidence for universal common ancestry, the proposition being argued by Dr. Theobald.

This is another attempt to make the hypothesis of common ancestry more attractive by arguing against the alternative of divine creation (rather than arguing *for* common ancestry). Such negative theology is especially ill suited for Dr. Theobald's case because, as pointed out in the introduction, divine creation (via common descent) is compatible with the "amechanistic" theory of common ancestry he is asserting. Furthermore, divine creation is not the only theoretical alternative to universal common ancestry, so eliminating it as a possibility would not establish the truth of universal common ancestry.

More importantly, however, the argument is based on the unprovable theological premise that a Creator would not use different structures to accomplish similar functions in different species. If two or more robots at a science fair solved a sophisticated problem in different ways, it would be considered a tribute to human ingenuity. If it were revealed that the same person had designed all the robots, he or she would be considered a creative genius. Perhaps multiple solutions to some biological functions are an analogous display of creativity.

The retort that an intention to display creativity in biology is contradicted by the fact organisms often exhibit similarity assumes that a Creator cannot have a reason for employing both diversity and similarity selectively. This too is a theological claim, not a scientific one. Those who believe that God may have reasons for things that are beyond our searching out will rightfully discount evidence that draws its weight from a rejection of that belief.

ReMine's biotic message theory provides one possible purpose for a Creator to employ diversity and similarity selectively. He writes:

These problems [of untestability and seemingly contradictory intentions] are resolved by message theory, which identifies a specific self-consistent goal the designer had in mind. The designer created life as a biotic message: to look like the product of a single designer and unlike all other interpretations. Similarity and diversity both have a role in the biotic message. Similarity makes life look like the work of one designer, while diversity makes life difficult to explain by naturalistic processes. Similarity and diversity are the alphabet for the biotic message. (ReMine, 37.)

It is also quite possible that different biological molecules that perform the same function in different species are also performing other functions of which we are not aware. In that case, the differences in structure could be explainable on functional grounds. The assumption that we have complete knowledge of how convergent molecules function within an organism is unwarranted.

PREDICTION 15: SUBOPTIMAL ANATOMICAL FUNCTION

Another consequence of evolutionary opportunism is the existence of apparent suboptimal function. As stated before, in evolving a new function, organisms must make do with what they already have. Thus, functions are likely to be performed by structures that would be arranged differently (e.g. more efficiently) if the final function were known from the outset. "Suboptimal function" does not mean that a structure functions poorly. It simply means that a structure with a more efficient design (usually with less superfluous complexity), could perform the same final function equally as well. Structures with suboptimal function should have a gradualistic historical evolutionary explanation, based on the opportunistic recruitment of ancestral structures, if this history is known from other evidence (e.g. if this history is phylogenetically determined by closely related organisms or fossil history).

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some organisms will have structures that are not designed most efficiently for the function they perform.
2. Some organisms have structures that are not designed most efficiently for the function they perform.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that organisms will have structures that are not designed most efficiently for the function they perform. Evolution can accommodate suboptimal design, but it can also accommodate its absence. As ReMine says:

[T]he argument from imperfection is not evidence for evolution. Neither perfection nor imperfection is evidence for evolution, since evolution is so vacuous it could accommodate both situations. Perfection is not immune from the facile just-so stories of natural selection, even Gould admits this.

But perfection could be imposed by a wise creator or evolved by natural selection. (Gould, 1984a, p 122 my italics) (ReMine, 26.)

Even Neo-Darwinism does not maintain that "in evolving a new function, organisms *must* make do with what they already have" (emphasis supplied). (This error is repeated under "Potential Falsification," where Dr. Theobald writes, "The *only* 'fixing' that is allowed evolutionarily is relatively minor modification of what already exists" [emphasis supplied].) I have already shown that Neo-Darwinism is flexible on that proposition. The development of novel structures is accepted whenever necessary to fit the data within the evolution paradigm (see the examples in the discussion of Prediction 11).

Once again, this is an attempt to make the hypothesis of common ancestry more attractive by arguing against the alternative of divine creation (rather than arguing *for* common ancestry). "The argument from imperfection never was evidence for evolution, instead it

was used as evidence *against* a designer." (ReMine, 26.) As noted above, such negative theology is especially inappropriate in Dr. Theobald's case (because an "amechanistic" theory of common ancestry is compatible with divine design). Furthermore, divine creation is not the only theoretical alternative to universal common ancestry, so eliminating it as a possibility would not establish the truth of universal common ancestry.

The argument against divine creation is: (a) God would never create a structure that was not designed most efficiently for the performance of its task; (b) some structures in organisms are not designed most efficiently for the performance of their task; (c) therefore, God did not create those structures (and hence did not create those organisms). There are several difficulties with this argument.

First, it is an unprovable theological premise that God would never create a structure that was not designed most efficiently for the performance of its task. Brand remarks:

The use of the Panda's thumb [a frequently-cited example of imperfect design] as a scientific argument against interventionism is valid only if we have objective data to support the hypothesis that a Creator would not use such a design; otherwise, it is only a philosophical argument. The data show us that if there is a Creator, He used a hierarchical design for life. How can we be sure that He would not use the genetic patterns of other bears to fashion a thumb for the panda, rather than interjecting a feature from some other animal into the bear's already cohesive genetic system? (Brand, 166.)

ReMine argues that the placement of such odd arrangements and funny solutions within living creatures was done deliberately by a Creator to send a message. As he explains:

Evolutionists have seen "odd arrangements and funny solutions" in nature and they insist these are paths a sensible designer would never tread. They are mistaken. Not only is it sensible, but message theory *absolutely requires it*, though at first it will seem paradoxical.

We expect a designer of life to create perfect designs. Yet this expectation itself constrains a biomessage sender to do the unexpected. A world full of perfect optimal designs would form an ambiguous message. In fact, it would not look like a message at all. It would provide no clues of an intentional message. It would look precisely as expected from a designer having no such intentions. Life's designer created life to look like a message, and therefore had to accept an astonishing design constraint: life *must* incorporate odd designs. . . .

It is not enough for a biomessage sender to merely include odd designs. All the designs together must form a pattern attributable only to a single designer. Life on earth has such a pattern.

Suppose we examined many separate handwritten documents. How would we recognize they all had the same author? Answer: By the overall pattern, especially the funny quirks and odd imperfections. It is the same thing with living organisms.

The quirks and imperfections play a key role in the pattern. They unite all organisms into a unified whole, while looking unlike the product of multiple designers. They give life the distinctive look of a single designer. They also make the pattern look like an intentional message, rather than an ordinary design effort. . . .

The concept of "perfection" is loaded with different meanings. Anti-creationists thought of it solely as engineering elegance. Yet message theory indicates a slightly different standard is needed. Biological designs serve a dual role: (1) as instruments of survival; and (2) as conveyors of a message. There is some range between 'elegant engineering designs' and 'designs sufficient for survival.' Life's designer used this range to incorporate a message. In this sense, life's designs are neither imperfect nor non-optimal. They are more aptly described as odd and curious. The argument from imperfection falls down because it used the wrong concept of perfection. (ReMine, 27-28.)

God also may have allowed an originally perfect creation to degenerate as a result of human rebellion. In that context, organisms may have evolved adaptations (within their created kind) that were less than optimally efficient. Also, optimally efficient original designs may have become flawed by the introduction of unreliability into the performance of system components. For example, the intersecting of the trachea and esophagus is problematical only if the epiglottis fails to close during swallowing. If that would not occur in a pre-Fall world, the original design could not even be accused of being suboptimal.

Other divine considerations, such a beauty or whimsy, may have guided some design choices. If, for example, the trachea were connected directly to the nostrils, what additional organs would be required for speech? The teeth and tongue could not simply be relocated, as they are used in chewing. And how large would the nasal passage need to be to accommodate highly aerobic activity (the kind that now causes us to breathe through our mouths)? We are in no position to judge the aesthetic price of such changes in the eyes of a Creator.

The second problem with this argument against divine creation is the difficulty in determining whether biological structures are in fact suboptimally designed. It may be that we are just too ignorant to appreciate the wisdom of a particular design.

[W]e are far from understanding the complexity of individual organisms, let alone the entire ecosystem in which that organism lives. What appears to be less than optimal design to us with our limited knowledge may actually be an optimal design when the entire system is considered. Consider the thickness of armor plating on the side of a warship. Since the purpose of such plating is to protect the ship from the puncture of an incoming warhead, it is advantageous to make the plating as thick as possible. Yet the plating on actual warships is much thinner than it could be made. The reason is, of course, that an increase in plating thickness makes the ship heavier, and thus slower. A less mobile ship is more likely to get hit more often and less likely to get where it is needed when it is needed. The actual thickness of armor on a warship is a tradeoff—not

so thin as to make the ship too easily sinkable, and not so think as to make the ship too slow. We know too little about the complexity of organisms and the environment in which they live to conclude that any one particular feature is actually less than optimal. (Wise, 221-222.)

This seems to be the case with the claim that the vertebrate retina could be designed more efficiently. As one examines the structure in detail, the design appears exquisite.[33] At the very least, the charge of suboptimality is debatable.

Acknowledging (*arguendo*) the possibility that the vertebrate retina may be optimally designed for land creatures, Dr. Theobald asserts that such a retina in fish is undoubtedly suboptimal (needlessly complex) because "the more elegant, more efficient, less complex cephalopod eye could perform underwater functions equally as well." But, as the cited articles make clear, it is by no means certain that a verted retina could perform underwater functions equally as well as an inverted retina. Perhaps the cephalopod eye is deliberately designed for less acute vision for larger ecological purposes.

A system of life has many requirements. Not every organism needs the most perfect vision, wings, or hands. Not every organism can be at the top of the food chain. A system of life requires organisms with different capabilities and different positions in the overall scheme of things. Perhaps a system of life requires, for its survival, some organisms that are "imperfect"? (ReMine, 27.)

The analysis of optimality is further complicated by the possibility the niches and ecosystems occupied by creatures today may not correspond to the original niches and ecosystems for which they were created. What was an optimum design in one world may not be appreciated as such in another.

The suggestion that universal common ancestry would be falsified by "the discovery of a mammal without crossed gastrointestinal and respiratory tracts, or a reptile or mammal without blindspots in its eyes" is incorrect. It is another example of taking what is known not to exist and claiming that evolution predicts it could not exist.

A theory that can tolerate the creation of novel structures and accommodate the radical alteration of a reptilian respiratory system into an avian system[34] can surely handle the segregation of an airway and the removal of a blind spot. If push came to shove, one could always argue that Mammalia and/or Vertebrata were not monophyletic. Whatever difficulties an evolutionary explanation may pose, it would be considered more reasonable than denying "the fact of evolution."

PREDICTION 16: MOLECULAR SUBOPTIMAL FUNCTION

The principle of imperfect design should apply to biomolecular organization as well.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then some organisms will have biological molecules that are not designed most efficiently for the function they perform.
2. Some organisms have biological molecules that are not designed most efficiently for the function they perform.

Since this is the concept of suboptimal design applied to biological molecules, 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 organisms will have biological molecules that are not designed most efficiently for the function they perform. Evolution can accommodate suboptimal design, but it can also accommodate its absence.

This is more negative theology in the guise of science. The claim is that God would never create in such a way as to leave the kinds of inefficiently designed biological molecules that we find today. Of course, even if that were true, it would not mean that all living things descended from a common ancestor, which is the proposition being argued.

In any event, the only example of a suboptimally designed biological molecule offered by Dr. Theobald is DNA. He claims it is suboptimally designed because the vast majority of an organism's DNA purportedly is "junk," sequences that have no function. So the argument against divine creation is: (a) God would never create DNA that contained nonfunctional sequences; (b) the DNA of all organisms contains nonfunctional sequences; (c) therefore, God did not create the DNA of organisms (and hence did not create living things). There are several problems with this argument.

First, the assertion that God would never create DNA that contained nonfunctional sequences is unprovable. Even if the nonfunctional sequences were present in the original creation, the most one could say is that one cannot perceive a reason for God's having created them. One's assessment of the likelihood of there being an inscrutable purpose behind the phenomenon will depend largely on one's concept of God.

Second, if God endowed organisms with an ability to adapt (within limits) to changing environments and/or if he allowed an originally perfect creation to degenerate as a result of human rebellion, then useless sequences in the DNA of modern organisms would be consistent with divine creation. In that case, one who claims suboptimality must retreat to the assertion that DNA which allows the accumulation of nonfunctioning sequences is less "efficiently designed" than DNA which includes a mechanism for the complete elimination of those sequences. But that is nothing more than an opinion.

Third, it is by no means clear that the DNA sequences alleged to be nonfunctional are in fact nonfunctional. Several years ago, Denton noted that "the idea that [most DNA] is really junk is now under increasing attack." (Denton 1998, 290.) More recently, molecular geneticist Linda Walkup wrote:

'Junk' DNA is thought by evolutionists to be useless DNA leftover from past evolutionary permutations. According to the selfish or parasitic DNA theory, this DNA persists only because of its ability to replicate itself, perhaps because it has randomly mutated into a form advantageous to the cell. The types of junk DNA include introns, pseudogenes, and mobile repetitive DNAs. But now many of the DNA sequences formerly relegated to the junk pile have begun to obtain new respect for their role in genome structure and function, gene regulation and rapid speciation. (Walkup, 18.)[35]

Evidence of functionality in "junk DNAs" supports geneticist Todd Wood's hypothesis that, at one time, repetitive and mobile DNA elements served to facilitate rapid diversification within created kinds. Walkup describes his theory this way:

Since these elements are capable of rapid change of the genome, and can even be transmitted horizontally between species, [Wood] proposes that God designed them to move about or recombine in the genomes of organisms to allow the rapid intrabaraminic diversification seen in the 500 years or so after the Flood. He sees their role as being designed to act for a limited period of time, after which they would be inactivated by mutation or repression by other regulatory elements. He proposes that such elements should be renamed Altruistic Genetic Elements (AGEs) to emphasize that their purpose is different than that proposed for 'selfish' DNA.

The AGEs are hypothesized to work by activating dormant genes or inactivating active genes, or by horizontally transferring genetic information between species or possibly baramins with AGEs in the form of mobile elements. The phenotypic changes would be primarily cosmetic, such as variations in size and coloration, or would involve activation of a complex of genes needed to utilize a new environmental niche, like the Arctic fox's adaptation to cold. . . .

If, for example, the proposed AGEs were at work in the diversification of equines, we have the testable prediction that differences in size, morphology and coloration could be traced back to the genetic level by mobile or repetitive DNA elements located near genes controlling coloration. Pseudogenes and relic retroviral sequences could then be the result of the action of an AGE gone wrong after its designed activity began to fail. (Walkup, 27.)

Walkup's conclusion points out the plasticity of evolutionary theory with regard to "junk DNA." She writes:

The fact that functions are being found for junk DNAs fits in well with creation science, but was not predicted by evolutionary theory, though of course the theory is being adjusted to accommodate the data. The intricate flexibility and specificity of these 'junk'

DNA sequences are a strong testimony to a Creator who plans and provides for the future of his creation. (Walkup, 28.)

Notes for Part 3

[24] "Paralogy" is commonly used in the context of molecular biology to refer to the presence of gene copies within the same organism. Dr. Theobald defines "paralogy" as "[s]imilarity of structure despite difference of function; the opposite of convergence." He defines "convergence" as "[t]he case of similar function despite different structures; the opposite of paralogy." In conventional parlance, "Characters that are *similar in structure and function* but have arisen separately rather than from a common ancestor are termed convergent" (emphasis supplied). (Carroll, 7.) They are presumed to have become similar structurally through the pressure to perform similar functions (e.g., the forelimbs of sharks, penguins, and porpoises). Parallelism (or parallel evolution), which is apparently not what Dr. Theobald means by paralogy, has been distinguished from convergence as follows: "Convergence is the development of similar characters separately in two or more lineages without a common ancestry pertinent to that similarity. Parallelism is the development of similar characters separately in two or more lineages of common ancestry on the basis of, or channeled by, characteristics of that ancestry." (Carroll, 7.) A standard example of parallel evolution is the wing patterns of moths and butterflies. Some question whether parallel evolution is a genuine phenomenon, claiming that all evolution is ultimately convergent or divergent (adaptive radiation).

[25] Homologous structures are parts of different organisms that are similar in structure and are assumed to have arisen by common descent. They may serve the same or different functions in the organisms. Dr. Theobald's "paralogous structures" are thus a specific case of homology (in that he includes only those structures that serve a different function).

[26] "The proximal part of both the fore- and hindlimb is composed of one main bone, humerus in the arm, femur in the leg. The next section of the limbs is composed of two bones, radius and ulna in the arm, tibia and fibula in the leg." (Denton 1986, 152.) The next section has a cluster of small bones, carpals in the arm, tarsals in the leg. This section is followed by metacarpals and phalanges in the arm, which correspond to metatarsals and phalanges in the leg.

[27] Prior to ReMine, Denton emphasized that nested hierarchy "implies the absence of any sort of natural sequential relationship among the objects grouped by the scheme." (Denton 1986, 121.) Instead, it "implies artificial logical relationships of a non-sequential sisterly kind." (Denton, 1986, 122.) Like ReMine, he recognized that "direct evidence for evolution only resides in the existence of unambiguous sequential arrangements," whereas "these are never present in ordered hierarchic schemes." (Denton 1986, 131.)

[28] The earliest known coelurosaurs, Compsognathus, Coelurus, and Ornitholestes, are all from the Late Jurassic. The earliest known dromaeosaurids, Sinornithosaurus and an unidentified species, are from the middle Early Cretaceous, some 25 million years later. (Xing and others; Qiang and others.) Deinonychus, the next oldest dromaeosaurid, is from the late Early Cretaceous. With the exception of the two Yixian specimens, the most birdlike dromaeosaurids are from the Late Cretaceous, some 75 million years after Deinonychus. (Feduccia, 90; Padian and Chiappe, 78; Hutchinson and Padian, 132; M. J. Benton, 699, 702.)

[29] Prior to 1997, it was believed that coelurosauravids had greatly elongated ribs that were connected to tissue to form a gliding surface comparable to that of the living lizard Draco. (Carroll, 220.) It was discovered, however, that the hollow, rodlike wing bones of Coelurosauravus were not extensions of the

ribs. In fact, they were not attached to any part of the skeleton! Rather, they formed in the skin, making them completely unlike the bones of any other tetrapod. (Frey and others.) The pteroid bone of pterosaurs and the carapace of turtles are other examples of novel structures in vertebrates.

[30] The homeotic gene Pax-6 is believed to be a master control gene for eye morphogenesis and to be universal among multicellular animals. The universality of the gene is thought to be the result of its presence in a common ancestor, yet this common ancestor is not thought to have had eyes. Evolutionists assume that this gene evolved by encoding primitive adaptations that remain to be discovered. (Wells 1998, 56-58.)

[31] What are believed to be convergently evolved features are often very similar in structure, but since they are not identical, they warrant the label "different." For example, squid eyes are so similar to human eyes that they are often dissected in biology classes to help students understand human eyes (Wise, 212), but they are not identical.

[32] "Analogies" are similar features believed to have evolved convergently (i.e., independently).

[33] See, for example, Ayoub, "On the Design of the Vertebrate Retina" ; Gurney, "Is Our 'Inverted' Retina Really 'Bad Design'?"; and Bergman, "Inverted Human Eye a Poor Design?". **[RETURN TO TEXT]**

[34] Denton describes the differences this way:

No lung in any other vertebrate species is known which in any way approaches the avian system. Just how such a different respiratory system could have evolved gradually from the standard vertebrate design without some sort of direction is, again, very difficult to envisage, especially bearing in mind that the maintenance of respiratory function is absolutely vital to the life of the organism. Moreover, the unique function and form of the avian lung necessitates a number of additional unique adaptations during avian development. As H. R. Dunker, one of the world's authorities in this field, explains, because first, the avian lung is fixed rigidly to the body wall and cannot therefore expand in volume and, second, because the small diameter of the lung capillaries and the resulting high surface tension of any liquid within them, the avian lung cannot be inflated out of a collapsed state as happens in all other vertebrates after birth. In birds, aeration of the lung must occur gradually and starts three to four days before hatching with a filling of the main bronchi, air sacs, and parabronchi with air. Only after the main air ducts are already filled with air does the final development of the lung, and particularly the growth of the air capillary network, take place. The air capillaries are never collapsed as are the alveoli of other vertebrate species; rather, as they grow into the lung tissue, the parabronchi are from the beginning open tubes filled with either air or fluid. (Denton 1998, 361.)

[35] The article is available online. See also, Richard Deem, "When 'Junk' DNA Isn't Junk" and Jaan Suurkula, "Junk DNA".

A Critique of Douglas Theobald's
"29 Evidences for Macroevolution"

by Ashby Camp

Part 4

"Molecular Evidence"

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[36] has done a careful study in which he calculated that there are a minimum of 2.3 x 1093 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.

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 under Prediction 7 [Part 2]). 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.

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,[37] 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.)

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.[38] 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 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 3104, or over 1049, different DNA sequences (and, hence, 1049 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"[39] 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. 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."[40] 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."[41] 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."[42] (Woodmorappe 2000, 57.)

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).[43] 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.)

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.[44] 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.

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.[45]

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 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,[46] 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 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.[47] 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 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."[48] 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 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.[49]

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."[50] 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.)

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[51] 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 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.

Notes for Part 4

[36] 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.

[37] 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.

[38] 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.

[39] 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.

[40] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 5.4 . Dr. Max believes, however, that nonfunctionality is the only reasonable conclusion that can be drawn for most transposons in light of current scientific evidence.

[41] Edie Lau, "Much DNA just 'junk'—or is it? Human Genome Project spurs new look at mystery material," Sacramento Bee (March 19, 2001).

[42] 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.)

[43] 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.

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

[45] Tom Abate, "Genome Discovery Shocks Scientists," San Francisco Chronicle (February 11, 2001)

[46] 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.

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

[48] Edward E. Max, "Plagiarized Errors and Molecular Genetics," Sec. 5.4. Dr. Max believes, however, that nonfunctionality is the only reasonable conclusion that can be drawn for most transposons in light of current scientific evidence.

[49] These comments are from an email posted at Dr. Brown's website.

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

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

A Critique of Douglas Theobald's

"29 Evidences for Macroevolution"

by Ashby Camp

Part 5

"Change"

PREDICTION 22: GENETIC CHANGE

The genetic information specifies everything about an organism and its potential. Genotype specifies possible phenotypes, therefore, phenotypic change follows genetic change. This obviously should be one of the areas where evolutionary change is seen, and genetic change is truly the most important for understanding evolutionary processes.

For the record, it is most doubtful that genetic information specifies everything about an organism. "According to a small but growing number of biologists, there is considerable evidence that genes do *not* control development." (Wells 1999, 51.) This evidence includes the following: (1) replacing an egg's genes with those of another species does not change the developmental pattern of the egg into an embryo; (2) mutations induced in developmental genes often lead to death or deformity but never alter the endpoint of embryonic development (they cannot even change the species); (3) strikingly different cell types arise in the same animal, even though all of them contain the same DNA; (4) similar developmental genes are found in animals as different as worms, flies, and mammals.

No one knows all the nongenetic factors involved in development, but they appear to include patterns in the egg cell membrane (that help to route gene products) and patterns in microtubules (microscopic fibers that are continually arranging themselves to give the cell its shape and to transport molecules within it). There is good evidence that both of these patterns are heritable apart from DNA. (Wells 1999, 52-53.) Wells concludes:

This does not mean that we now understand developmental programs. Far from it! But it is quite clear that they cannot be reduced to genetic programs, written in the language of DNA sequences. It would be more accurate to say that a developmental program is written into the structure of the entire fertilized egg—including its DNA, microtubule arrays, and membrane patterns—in a language of which we are still largely ignorant. (Wells 1999, 53.)

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the DNA of organisms will be capable of change.
2. The DNA of organisms is capable of change.

It is not a prediction of the hypothesis of universal common ancestry that the DNA of organisms will be capable of change. Common ancestry does not even predict the existence of DNA. It has no stake in any particular mechanism of descent.

If one adds to the hypothesis the more specific claim that all organisms arose from a common ancestor by accumulated alterations of DNA, the issue is not simply whether DNA can change but whether the kinds of changes required for universal common ancestry can and did occur. From a naturalistic perspective, meaning without some kind of intelligent design or intervention, that seems impossible.

Renowned French zoologist Pierre-Paul Grasse made no secret of his skepticism:

What gambler would be crazy enough to play roulette with random evolution? The probability of dust carried by the wind reproducing Durer's "Melancholia" is less infinitesimal than the probability of copy errors in the DNA molecule leading to the formation of the eye; besides, these errors had no relationship whatsoever with the function that the eye would have to perform or was starting to perform. There is no law against daydreaming, but science must not indulge in it. (Grasse, 104.)

In 1967 a group of internationally known biologists and mathematicians met to consider whether random mutations and natural selection could qualify as the mechanism for evolution. The answer of the mathematicians was "No." In the words of Murray Eden of M.I.T., "What I am claiming is that without some constraint on the notion of random variation, in either the properties of the organism or the sequence of the DNA, there is no particular reason to expect that we could have gotten any kind of viable form other than nonsense." (Moorehead and Kaplan, 14.)

Mathematicians/astronomers Sir Fred Hoyle and Chandra Wickramasinghe concur.[52] Summarizing his and Hoyle's analysis of the alleged mechanism of evolution, Wickramasinghe states:

We found that there's just no way it could happen. If you start with a simple micro-organism, no matter how it arose on earth, primordial soup or otherwise, then if you just have that single organizational, informational unit and you said that you copied this sequentially time and again, the question is does that accumulate enough copying errors, enough mistakes in copying, and do these accumulations of copying errors lead to the diversity of living forms that one sees on earth. That's the general, usual formulation of the theory of evolution. . . . We looked at this quite systematically, quite carefully, in numerical terms. Checking all the numbers, rates of mutation and so on, we decided that there is no way in which that could even marginally approach the truth." (Varghese, 28.)

Biophysicist Spetner has likewise concluded that the probability of getting the necessary mutations through random copying errors is far too small to make Neo-Darwinism a feasible explanation for all the diversity of life. A summary of his argument is available here. See also, Spetner's "Evolution, Randomness, and Hashkafa".

In any event, the fact the DNA of organisms is capable of change does nothing to advance the claim of universal common ancestry. That datum is fully compatible with the claim that multiple lineages were created independently and endowed with a degree of genetic adaptability.

PREDICTION 23: MORPHOLOGICAL CHANGE

Cladistic classification, and thus, phylogenetic reconstruction, is largely based on the various distinguishing morphological characteristics of species. Macroevolution requires that organisms' morphologies have changed throughout evolutionary history; thus, we should observe morphological change and variation in modern populations.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then morphological change and variation will be observable in modern populations.
2. Morphological change and variation are observable in modern populations.

Again, it is not a prediction of the hypothesis of universal common ancestry that morphological change and variation will be observable in modern populations. The hypothesis does not predict any particular mechanism of diversification. Thus, populations could be morphologically uniform and change in one generation outside our viewing frame. There is not even a requirement that the mechanism of universal common ancestry still be operating today.

If one adds to the hypothesis the more specific claim that all organisms arose by a continuing process of selection from morphological variation within existing populations, it raises the issue of the source of the continuing variations on which selection supposedly operated.[53] That leads straight to the preceding discussion. The revised hypothesis also raises the issue of the nature of the selection and its adequacy for accomplishing the results attributed to it.

In any event, the morphological changes and variations that are observed in modern populations certainly do not justify the conclusion of universal common ancestry. They are fully compatible with the claim that multiple lineages were created independently and endowed with a degree of genetic adaptability. In fact, the experimental data suggest that there are natural limits to the extent to which species can change.

As science commentator Jeremy Rifkin (and many others) has noted:

The fruit fly has long been the favorite object of mutation experiments because of its fast gestation (twelve days). X rays have been used to increase the mutation rate in the fruit fly by 15,000 percent. All in all, scientists have been able to "catalyze the fruit fly evolutionary process such that what has been seen to occur in *Drosophila* (fruit fly) is the equivalent of many millions of years of normal mutations and evolution." Even with this tremendous speedup of mutations, scientists have never been able to come up with anything other than a fruit fly. More important, what all these experiments demonstrate is that the fruit fly can vary within certain upper and lower limits but will never go beyond them. (Rifkin, 134.)

The same holds true for the extensive genetic experiments done on *E. coli* bacteria. According to geneticists Lane Lester and Ray Bohlin:

The study of bacteria has been profoundly at the center of studies of mutations. This is because they reproduce rapidly, producing large populations and large numbers of mutants. They are also easily maintained and their environments are easily manipulated in the laboratory. Despite all their advantages, never has there arisen in a colony of bacteria a bacterium with a primitive nucleus. Never has a bacterium in a colony of bacteria been observed to make a simple multicellular formation. Although hundreds of strains and varieties of *Escherichia coli* have been formed, it is still *Escherichia coli* and easily identifiable as such. (Lester and Bohlin, 88.)

PREDICTION 24: FUNCTIONAL CHANGE

One of the major differences between organisms is their capacity for various functions. The ability to occupy one niche over another is invariably due to differing functions. Thus, functional change must be extremely important for macroscopic macroevolutionary change.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the acquisition of new capabilities will be observable in modern populations.
2. The acquisition of new capabilities is observable in modern populations.

It is not a prediction of the hypothesis of universal common ancestry that the acquisition of new capabilities will be observable in modern populations. The hypothesis does not predict any particular mechanism of diversification. Thus, the capabilities of populations could be constant within our viewing frame. There is not even a requirement that the mechanism of universal common ancestry still be operating.

But more importantly, the functional changes observed in species do nothing to advance the claim of universal common ancestry. They are fully compatible with the claim that multiple lineages were created independently and endowed with a degree of genetic adaptability.

Interestingly, most if not all of the functional changes observed in species point away from random mutation as the explanation. They do so in two ways. First, some of the changes are produced by a *loss* of information. That raises the question of how the information that was lost arose in the first place. Spetner writes:

We have seen that there are some point mutations that, under the right circumstances, do give the organism and advantage. There are point mutations that make bacteria resistant to antibiotics. There are some that make insects resistant to insecticides. There are some that increase quantitative traits in farm plants and animals. But all these mutations reduce the information in the gene by making the protein less specific. They add no information, and they add no new molecular capability. Indeed, all mutations studied destroy information. None of them can serve as an example of a mutation that can lead to the large changes of macroevolution.

The Neo-Darwinian would like us to believe that large evolutionary changes can result from a series of small events if there are enough of them. But if these events all lose information they can't be the steps in the kind of evolution the [Neo-Darwinian theory] is supposed to explain, no matter how many mutations there are. Whoever thinks macroevolution can be made by mutations that lose information is like the merchant who lost a little money on every sale but thought he could make it up on volume. (Spetner, 159-160.)

Second, some of the changes appear to be nonrandom responses to the environment, suggesting that the genome was "set up" for an adaptive change to be triggered by a cue from the environment. (See, Spetner, 175-208.) That raises the question of how the genome came to be in that prepared state.

The 1982 study by Barry Hall cited by Dr. Theobald provides a good illustration. Hall prepared a strain of *E. coli* that lacked a gene necessary for the metabolizing of lactose. But in the presence of lactose, *two mutations* were found in the same bacterium (one to a dormant and previously unknown structural gene and the other to its control gene) that in combination permitted it to metabolize lactose. By Hall's calculation, he should have had to wait 100,000 years to see these double mutations, but in the presence of lactose he found about 40 of them in a few days. "These results suggest that lactose in the environment induced these mutations." (Spetner, 188.) Spetner observes:

Darwinian evolutionists see the nonrandom interpretation of these experimental results as obviously incorrect because they contradict the Neo-Darwinian dogma. I, on the other hand, see this interpretation as confirming, on the bacterial level, the nonrandom variation indicated by many examples in plants and animals—examples that Darwinian evolutionists have largely ignored because they do not fit in. Resistance to the nonrandom-variation interpretation stems from a refusal to abandon the Darwinian agenda that evolution must confirm that life arose and developed spontaneously. With that agenda, nonrandom adaptive variation, arising from an environmental signal turning ON an already present set of genes, is hard to account for. . . .

The several examples cited above indicate that the phenomenon, if it is indeed vindicated, may be widespread in bacteria. Just as these bacteria contain "cryptic" genes which encode for enzymes that are needed in some environments, so I suggest that other organisms also may have latent parts of their genome dedicated to be adaptive to a certain set of environmental conditions that may arise. The environment can then supply a cue that will turn ON the latent section that will make the organism adaptive. (Spetner, 191-192.)[54]

PREDICTION 25: EARTH'S STRANGE PAST AND THE FOSSIL RECORD

A very general conclusion made from the theory of common descent is that life, as a whole, was different in the past. The predicted evolutionary pattern is that the farther back we look back in time, the more different life should appear from the modern biosphere. More recent fossils should be more similar to contemporary life forms than older fossils.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then life forms will differ increasingly from modern forms as one moves down the fossil record.
2. Life forms differ increasingly from modern forms as one moves down the fossil record.

It is not a prediction of the hypothesis of universal common ancestry that life forms will differ increasingly from modern forms as one moves down the fossil record. Of course, if all life forms arose from a common ancestor, then there necessarily was a period of diversification. But an "amechanistic" theory of universal common ancestry says nothing about how quickly modern forms were achieved, and it does not require a fossil record of that diversification to have been made, preserved, or discovered.

In other words, if the fossil record began with modern forms, that fact alone would not falsify the hypothesis of universal common ancestry. The proponent of common ancestry could make the same kind of argument that is made currently regarding the "Cambrian Explosion," i.e., massive diversification went undocumented or undiscovered. See, e.g., Meyer, Nelson, and Chien, "The Cambrian Explosion: Biology's Big Bang".

But more importantly, universal common ancestry is not the only possible explanation for why life forms differ increasingly from modern forms as one moves down the fossil record. In fact, the types of differences that are seen actually weigh against the claim of universal common ancestry. As paleontologist Wise points out:

As one goes back in time, organismal groups tend not to converge in morphology, but remain distinct. Most major groups remain identifiable by modern characters and distinct from their supposed ancestors all the way back to their oldest fossil representatives. This

would seem to imply that the branching event of one major group from another never did occur. (Wise, 219-220.)

So, at best, the increasing divergence from modern life forms that is observed as one moves down the fossil record can be claimed to support evolution only within a multitude of major groups, not evolution from a universal common ancestor. And even that claim is weak, as significant gaps exist in alleged fossil lineages within all the major groups. I have elsewhere attempted to point out some of the gaps in purported mammal lineages, which are considered an evolutionary showcase (see, "Reappraising the Crown Jewel").[55]

Some creationists explain the change in life forms in the fossil record by proposing that God created new species intermittently over vast ages. Biblical creationists, however, believe the fossil order is largely an artifact of a complex and unique cataclysmic process, the details of which are obscured by its uniqueness and by our ignorance of the ancient biosphere. Wise writes:

The general features of the fossil record that *are* explained by evolutionary theory are at least as well explained by other theories. The existence of a Creator who introduced organisms on earth in a particular order could explain the general change in organisms through the record, but so could the effect of a global flood as it successively sampled from a biogeographically zoned distribution of organisms. The general change in organisms through time can be predicted by any one or all of these three theories (macroevolution, progressive creation, global deluge). On the other hand, the rarity or absence of evidence for transitions between major groups and the fact major groups do not converge on one another as one goes back in the fossil record seem to argue that major groups were introduced in the fossil record only *after* they were fully formed. This is more consistent with creative order and global deluge theories than with macroevolutionary theory. As for the linear relationship of species similarity above and below a particular level in the geologic column, this can be just as well explained by global deluge theory or progressive creation theory as it is by macroevolution. In deluge theory, different species are found in different preflood environments and get mixed with species from adjacent environments, providing the species similarity relationship. Continual introduction of species whether by evolution or creation would produce the same relationship. In short, all fossil-record order can be at least as well explained by order of creation decided by creative fiat or ocean-to-land burial of organisms in a diverse world overcome by global deluge as it is by macroevolution. (Wise, 226.)[56]

PREDICTION 26: STAGES OF SPECIATION

The most useful definition of species (which does not assume evolution) for sexual metazoans is the Biological Species Concept: species are groups of actually or potentially interbreeding natural populations that are reproductively isolated from other such groups (Mayr 1942).

If branching of existing species into new species occurred gradually in the past, we should see all possible degrees of speciation or genetic isolation today, ranging from fully interbreeding populations, to partially interbreeding populations, to populations that interbreed with reduced infertility or with complete infertility, to completely genetically isolated populations.

Presumably, the alleged prediction and fulfillment are:

1. If universal common ancestry is true, then all the stages of the process of speciation will be observable today.
2. All the stages of the process of speciation are observable in species today.

Gradual branching of species into new species is not an element of the hypothesis of universal common ancestry, as that hypothesis says nothing about the mechanism of descent. Rather, it is an element of the more specific hypothesis of Neo-Darwinism. Since Dr. Theobald purports to establish universal common ancestry apart from any particular mechanism of descent, he cannot assume a particular mechanism of descent in making his case.

Moreover, the hypothesis of universal common ancestry does not require that its processes be continuing and thus does not require that all its stages be present today. So even if the hypothesis of universal common ancestry entailed speciation, it could accommodate a failure to observe the various stages of that process.

But most importantly, evidence of speciation does nothing to advance the claim of universal common ancestry. Speciation is fully compatible with the claim that multiple lineages were created independently and endowed with a degree of genetic adaptability. The fact one species can give rise to another similar species does not mean there are no limits to the process, that a bacterium can give rise to a human. On the contrary, the experimental data cited previously suggests the opposite.

One need not be a creationist to question the extrapolation from speciation to universal common ancestry. As Brand notes, "Some scientists are beginning to doubt that the microevolutionary process extrapolated over time is adequate to produce more significant changes. They suggest that larger scale evolution must involve a different mechanism than microevolution and that it happens rapidly. (Ridley 1993, p. 523-525)." (Brand, 120.)

PREDICTION 27: SPECIATIONS

The standard phylogenetic tree illustrates countless speciation events; each common ancestor also represents at least one speciation event. Thus we should be able to observe actual speciation, if even only very rarely. Current estimates from the fossil record and measured mutational rates place the time required for full reproductive isolation in the wild at ~3 million years on average (Futuyma 1998, p. 510). Consequently, observation

of speciation in nature should be a possible but rare phenomenon. However, evolutionary rates in laboratory organisms can be much more rapid than rates inferred from the fossil record, so it is still possible that speciation may be observed in common lab organisms (Gingerich 1983).

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then speciation will be observable today in laboratory organisms.
2. Speciation is observable today in laboratory organisms.

The preceding claim was that snapshots of the various stages of speciation will be present in nature. This claim is that one or more speciation events can be induced in a laboratory.

The response is the same. Dr. Theobald impermissibly assumes a particular mechanism of descent in arguing for an "amechanistic" theory of common ancestry. Moreover, the hypothesis of universal common ancestry does not require that its processes be continuing, so even if it entailed speciation, it could not be falsified by a failure to induce speciation in a laboratory.

Most importantly, however, evidence of speciation does nothing to advance the claim of universal common ancestry. Speciation is fully compatible with the claim that multiple lineages were created independently and endowed with a degree of genetic adaptability.

PREDICTION 28: MORPHOLOGICAL RATES OF CHANGE

Observed rates of evolutionary change in modern populations must be greater than or equal to rates observed in the fossil record.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the calculated rates of evolutionary change in modern populations will be greater than or equal to the calculated rates of evolutionary change in the fossil record.
2. The calculated rates of evolutionary change in modern populations are greater than or equal to the calculated rates of evolutionary change in the fossil record.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that the rates of evolutionary change in modern populations will be equal to or greater than the rate believed to have prevailed in the past. The hypothesis says nothing about the constancy of the rate of evolutionary change.

So if rates of change in modern populations were not sufficient to generate all living things from a universal common ancestor, it would not falsify the hypothesis. It simply would be assumed that the rate had slowed down.

After all, according to Dr. Theobald, evolutionists believe that the average evolutionary rate from historical colonization events is 616 times faster than the average rate in the fossil record. If the theory can accommodate faster modern rates, it can also accommodate slower modern rates.

In any event, one cannot simply assume that the minor changes occurring in modern populations could continue beyond certain natural limits so as to create new orders, classes, and phyla. In fact, there is good reason for doubting that assumption. As biologists Davis and Kenyon point out:

Over the years, numerous investigations have explored the questions of whether species are "infinitely plastic," capable of unlimited change, or whether change is limited. Darwin advocated the unlimited change view. The accumulated evidence to date, however, severely questions Darwin on this. For example, the Bumpus study of birds (Chapter 2) showed a remarkable tendency for birds to vary within limits. Hermann J. Muller labored for many years conducting mutation experiments with fruit fly *Drosophila* to demonstrate unlimited change, and found the same tendency: change occurs only within definite limits. Others have tried, as well. Such attempts have all met with uniform lack of success, and ultimately died a quite death. Hardly anyone is still trying to furnish an observable basis for Darwin's view of unlimited change. . . .

The Darwinist, however, believes species have unlimited potential for change even if scientists have not been able to experimentally produce it. Darwinian theory holds that the diversity of contemporary species arose through descent from a common ancestor. According to Darwinists, we must regard lack of experimentally induced, unlimited change as a problem in need of research, not a basis to doubt macroevolution. (Davis and Kenyon, 78-79.)

If, as asserted by Dr. Theobald (from Gingerich), a change rate of 400 darwins sustained over 10,000 years is sufficient to turn a mouse into an elephant, then the alleged average laboratory change rate of 60,000 darwins would be sufficient to accomplish that task in just under 67 years. This makes it all the more remarkable that decades of laboratory experiments have produced such meager results, nothing approaching the dramatic levels of transformation predicted by these figures. It is not surprising that "[s]ome scientists are beginning to doubt that the microevolutionary process extrapolated over time is adequate to produce more significant changes." (Brand, 120.)

Of course, estimates of rates of change in the fossil record are loaded with assumptions. One first must assume that two specimens are ancestor and descendant. Since evolutionists often insist that only sister groups can be identified, not actual ancestors, there is an additional level of speculation. One must prescribe the morphology of the hypothetical common ancestor and then quantify the degree to which it differs from the

alleged descendant. One also must make assumptions about when the lineage in question split from the assumed ancestor and when the alleged descendant first arose.

PREDICTION 29: GENETIC RATES OF CHANGE

Rates of genetic change, as measured by nucleotide substitutions, must also be consistent with the rate required from the time allowed in the fossil record and the sequence differences observed between species.

The alleged prediction and fulfillment are:

1. If universal common ancestry is true, then the current rate of nucleotide substitution in the nonfunctioning DNA of two species will be sufficient to account for the nucleotide differences in the nonfunctioning DNA of those species, given the assumed date of their divergence from a common ancestor.
2. The current rate of nucleotide substitution in nonfunctioning DNA of various species is sufficient to account for the nucleotide differences in the nonfunctioning DNA of those species, given the assumed date of their divergence from a common ancestor.

It is not a prediction of the hypothesis of universal common ancestry or the more specific hypothesis of Neo-Darwinism that the current rate of nucleotide substitution in the nonfunctioning DNA of two species will be sufficient to account for the nucleotide differences in the nonfunctioning DNA of those species. The hypotheses say nothing about the constancy of the rate of nucleotide substitution or about the dates on which species diverged from a common ancestor.

So if the rate of substitution in modern populations were not sufficient to account for the nucleotide differences within the time prescribed, it would not falsify the hypotheses. It simply would be assumed that the rate had slowed down and/or that the date of divergence was earlier than previously believed.

In addition, the fact current substitution rates are sufficient to account for the nucleotide differences within the assumed time frames (from the alleged dates of divergence) does nothing to advance the claim of universal common ancestry. It does not even advance the claim that the particular species being compared descended from a more recent common ancestor. That must be *assumed*. A contrary assumption could easily be accommodated.

It should be pointed out that nucleotide substitution rates in presumably nonfunctional DNA are not always in easy agreement with current phylogenies. Woodmorappe writes:

It is interesting to note that the inferred nucleotide-substitution rate in pseudogenes shows only crude correspondence with primate phylogeny, for which reason it has to be

manipulated *post hoc* by up to tenfold in order to contrive an agreement between the timing of different episodes of primate evolution. (Woodmorappe 2000, 86.)

CONCLUSION

In the words of the great detective Sherlock Holmes:

"Circumstantial evidence is a very tricky thing," answered Holmes thoughtfully; "it may seem to point very straight to one thing, but if you shift your point of view a little, you may find it pointing in an equally uncompromising manner to something entirely different" . . . "There is nothing more deceptive than an obvious fact".[57]

Dr. Theobald and many other bright and well-educated evolutionists are certain that the evidence of nature points ineluctably to the conclusion of universal common ancestry. I once shared that opinion of history, but having shifted my point of view, I find that the same evidence points to something entirely different.

I have explained in this paper the way Dr. Theobald's evidence looks from my perspective. I have argued that what he labels falsifiable predictions of the hypothesis of universal common ancestry are in fact mere observations that have been read back into a plastic theory and claimed as predictions. His hypothesis accommodates these observations, but since it could also accommodate contrary ones, that fact has little or no probative value. As Hunter says, "There is nothing wrong with a theory that is comfortable with different outcomes, but there is something wrong when one of those outcomes is then claimed as supporting evidence. If a theory can predict both A and not-A, then neither A nor not-A can be used as evidence for the theory." (Hunter, 38.)

I have shown how Dr. Theobald's evidence can be accommodated by alternative hypotheses. I have also highlighted instances where his interpretation of the evidence is driven by theological assumptions. One who rejects those underlying assumptions is justified in rejecting the conclusions that follow from them.

Since this is a critique of Dr. Theobald's article, evidence for creation has been presented only when relevant to the discussion of one of his alleged predictions. Nothing has been said about the immense difficulty in accounting for the origin of life, with its vast information content, by purely naturalistic processes.[58] And little if anything has been said about the mind-boggling complexity that exists at a variety of levels: subcellular processes and bodies, tissues, bodily organs and systems, symbiotic systems, ecosystems, and even astronomical arrangements. As Wise notes:

Each of these levels features a complexity that is staggering to the human mind—a complexity greater than any that in our experience can be produced by [a] nonintelligent natural cause. If we follow the principle of appealing only to principles that are reasonable in our experience, then the complexity of any one of these levels seems to require an appeal to an intelligent cause. However, the *total* complexity is at least the sum of the complexities of each level. If the complexity of each level suggests an

intelligent cause, the total complexity screams for an intelligent cause. Macroevolutionary theory has never successfully explained the acquisition of any level of this complexity, let alone the total complexity. (Wise, 229-230.)

Likewise, little if anything has been said about the equally mind-boggling integration of these amazingly complex items and events. To quote Wise again:

As if the basic complexity of things were not enough, the integration of that complexity is truly astounding. Not only do subcellular chemical processes involve a large number of complex molecules and chemical steps, but those items and events are connected in a well-balanced and well-timed series of items and steps to produce a well-integrated process. Similarly, the workings of subcellular organelles, cells in tissues, tissues in organs, organs in systems, systems in bodies, organisms with other organisms, organisms in communities, and communities in the biosphere all show staggering integration. As with the complexity of these items and events on any given level, such a level of integration has never been observed to arise from nonintelligent natural law and process. Integration seems to argue for intelligent cause.

In addition, the integration that is so striking *within* levels is even more striking *between* levels. Not only do subcellular organelle systems and chemical processes show integration, but the chemical and organelle systems are themselves linked together, and must be for the cell to survive. Even more impressive, a similar integration exists between all levels. Once again, this level of integration is unexplained by evolutionary theory but is addressable by intelligent cause theory. (Wise, 230.)

"For since the creation of the world God's invisible qualities—his eternal power and divine nature—have been clearly seen, being understood from what has been made, so that men are without excuse." Rom. 1:20 (NIV)

Notes for Part 5

[52] Hoyle was a professor at Cambridge and the former head of the Institute of Theoretical Astronomy at that university. Wickramasinghe is the chairman of the Department of Applied Mathematics and Astronomy at University of Cardiff. They published a booklet titled Why Neo-Darwinism Does Not Work (Cardiff: University College Cardiff Press, 1982), which they describe as a "simple and decisive disproof of the 'Darwinian' theory." See also, Hoyle's Mathematics of Evolution (Memphis, TN: Acorn Enterprises, 1999).

[53] As philosopher of biology Paul Nelson notes, "Whether [favorable] variations have, or could have, occurred are factual questions to which selection is helpless to speak." (Nelson, 63.)

[54] Spetner describes two other kinds of nonrandom variations of phenotype, one that is heritable and one that is not. (Spetner, 192-197.)

[55] For one committed to universal common ancestry, no gaps, however large or numerous, are sufficient to put a claim of descent in doubt. Gaps are assumed to be merely the absence of evidence, not evidence of the absence of lineages. Creationists, however, are committed to the proposition that numerous kinds of living things were created independently. From their perspective, gaps can be an indication that hypothesized lineages are imaginary.

[56] Wise would be the first to admit that there are questions for which current deluge theories lack answers, but that is true for all explanations of earth history. For a more detailed discussion of one flood model, see Brand, 171-179, 209-318. Some worthwhile online resources are Austin and others, "Catastrophic Plate Tectonics: Global Flood Model of Earth History"; Brand and Florence, "Stratigraphic Distribution of Vertebrate Fossil Footprints Compared with Body Fossils"; Wise, "Punq Eq Creation Style"; and Gibson, "Fossil Patterns: A Classification and Evaluation".

[57] Quoted in Denton 1986, 155.

[58] I realize that the bare hypothesis of universal common ancestry does not address the origin of life and is consistent with intelligently directed descent. But to the extent its advocates insist on strictly natural causes, it is appropriate to cite as evidence of a Creator the extreme improbability of generating life and of achieving the complexity and integration exhibited in nature.

A Critique of Douglas Theobald's
"29 Evidences for Macroevolution"

by Ashby Camp

Part 6

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