



THE TOP TEN SCIENTIFIC PROBLEMS WITH BIOLOGICAL AND CHEMICAL EVOLUTION

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“There are no weaknesses in the theory of evolution.”¹ Such was professed by Eugenie Scott, the de facto head of the Darwin lobby, while speaking to the media in response to the Texas State Board of Education’s 2009 vote to require students to learn about both the scientific evidence for and against neo-Darwinian evolution.

For those who follow the debate over origins, Dr. Scott’s words are as unsurprising as they are familiar. It seems that almost on a daily basis, we find the news media quoting evolutionary scientists declaring that materialist accounts of biological and chemical evolution are “fact.” Students who take college-preparatory or college-level courses on evolution are warned that doubting Darwinism is tantamount to committing intellectual suicide—you might as well proclaim the Earth is flat.² Such bullying is enough to convince many that it’s much easier on your academic standing, your career, and your reputation to just buy into Darwinism. The few holdouts who remain are intimidated into silence.

But is it true that there are “no weaknesses” in evolutionary theory? Are those who express doubts about Darwinism displaying courage, or are they fools that want to take us back to the dark ages and era of the flat Earth?³ Thankfully, it’s very easy to test these questions: all one must do is examine the technical scientific literature and inquire whether there are legitimate scientific challenges to chemical and biological evolution.

This chapter will review some of this literature, and show that there are numerous legitimate scientific challenges to core tenets of Darwinian theory, as well as predominant theories of chemical evolution. Those who harbor doubts about Darwinism need not be terrified by academic bullies who pretend there is no scientific debate to be had.

**PROBLEM 1:
NO VIABLE MECHANISM
TO GENERATE A PRIMORDIAL SOUP.**

According to conventional thinking among origin of life theorists, life arose via unguided chemical reactions on the early Earth some 3 to 4 billion years ago. Most theorists believe that there were many steps involved in the origin of life, but the very first step would have involved the production of a primordial soup—a water-based sea of simple organic molecules—out of which life arose. While the existence of this “soup” has been accepted as unquestioned fact for decades, this first step in most origin-of-life theories faces numerous scientific difficulties.

In 1953, a graduate student at the University of Chicago named Stanley Miller, along with his faculty advisor Harold Urey, performed experiments hoping to produce the building blocks of life under natural conditions on the early Earth.⁴ These “Miller-Urey experiments” intended to simulate lightning striking the gasses in the early Earth’s atmosphere. After running the experiments and letting the chemical products sit for a period of time, Miller discovered that amino acids—the building blocks of proteins—had been produced.

For decades, these experiments have been hailed as a demonstration that the “building blocks” of life could have arisen under natural, realistic Earthlike conditions,⁵ corroborating the primordial soup hypothesis. However, it has also been known for decades that the Earth’s early atmosphere was fundamentally different from the gasses used by Miller and Urey.

The atmosphere used in the Miller-Urey experiments was primarily composed of reducing gasses like methane, ammonia, and high levels of hydrogen. Geochemists now believe that the atmosphere of the early Earth did not contain appreciable amounts of these components. (Reducing gasses are those which tend to donate electrons during chemical reactions.) UC Santa Cruz origin-of-life theorist David Deamer explains this in the journal *Microbiology & Molecular Biology Reviews*:

This optimistic picture began to change in the late 1970s, when it became increasingly clear that the early atmosphere was probably volcanic in origin and composition, composed largely of carbon dioxide and nitrogen rather than the mixture of reducing gases assumed by the Miller-Urey model. Carbon dioxide does not support

the rich array of synthetic pathways leading to possible monomers...⁶

Likewise, an article in the journal *Science* stated: “Miller and Urey relied on a ‘reducing’ atmosphere, a condition in which molecules are fat with hydrogen atoms. As Miller showed later, he could not make organics in an ‘oxidizing’ atmosphere.”⁷ The article put it bluntly: “the early atmosphere looked nothing like the Miller-Urey situation.”⁸ Consistent with this, geological studies have not uncovered evidence that a primordial soup once existed.⁹

There are good reasons to understand why the Earth’s early atmosphere did not contain high concentrations of methane, ammonia, or other reducing gasses. The earth’s early atmosphere is thought to have been produced by outgassing from volcanoes, and the composition of those volcanic gasses is related to the chemical properties of the Earth’s inner mantle. Geochemical studies have found that the chemical properties of the Earth’s mantle would have been the same in the past as they are today.¹⁰ But today, volcanic gasses do not contain methane or ammonia, and are not reducing.

A paper in *Earth and Planetary Science Letters* found that the chemical properties of the Earth’s interior have been essentially constant over Earth’s history, leading to the conclusion that “Life may have found its origins in other environments or by other mechanisms.”¹¹ So drastic is the evidence against pre-biotic synthesis of life’s building blocks that in 1990 the Space Studies Board of the National Research Council recommended that origin of life investigators undertake a “reexamination of biological monomer synthesis under primitive Earthlike environments, as revealed in current models of the early Earth.”¹²

Because of these difficulties, some leading theorists have abandoned the Miller-Urey experiment and the “primordial soup” theory it is claimed to support. In 2010, University College London biochemist Nick Lane stated the primordial soup theory “doesn't hold water” and is “past its expiration date.”¹³ Instead, he proposes that life arose in undersea hydrothermal vents. But both the hydrothermal vent and primordial soup hypotheses face another major problem.

CHEMICAL EVOLUTION IS DEAD IN THE WATER

Assume for a moment that there was some way to produce simple organic molecules on the early Earth. Perhaps they did form a

“primordial soup,” or perhaps these molecules arose near some hydrothermal vent. Either way, origin of life theorists must then explain how amino acids or other key organic molecules linked up to form long chains (polymers) like proteins (or RNA).

Chemically speaking, however, the last place you’d want to link amino acids into chains would be a vast water-based environment like the “primordial soup” or underwater near a hydrothermal vent. As the National Academy of Sciences acknowledges, “Two amino acids do not spontaneously join in water. Rather, the opposite reaction is thermodynamically favored.”¹⁴ In other words, water breaks protein chains back down into amino acids (or other constituents), making it very difficult to produce proteins (or other polymers) in the primordial soup.

Materialists lack good explanations for these first, simple steps which are necessary to the origin-of-life. Chemical evolution is literally dead in the water.

PROBLEM 2:

UNGUIDED CHEMICAL PROCESSES

CANNOT EXPLAIN THE ORIGIN OF THE GENETIC CODE.

Let’s assume, again, that a primordial sea filled with life’s building blocks did exist on the early Earth, and somehow it formed proteins and other complex organic molecules. Origin of life theorists believe that the next step in the origin of life is that—entirely by chance—more and more complex molecules formed until some began to self-replicate. From there, they believe Darwinian natural selection took over, favoring those molecules which were better able to make copies. Eventually, they assume, it became inevitable that these molecules would evolve complex machinery—like that used in today’s genetic code—to survive and reproduce.

Have modern theorists explained how this crucial bridge from inert nonliving chemicals to self-replicating molecular systems took place? The most prominent hypothesis for the origin of the first life is called the “RNA world.” In living cells, genetic information is carried by DNA, and most cellular functions are carried out by proteins. However, RNA is capable of both carrying genetic information and catalyzing some biochemical reactions. As a result, some theorists postulate the first life might have used RNA alone to fulfill all these functions.

But there are many problems with this hypothesis.

For one, the first RNA molecules would have to arise by unguided, non-biological chemical processes. But RNA is not known to assemble without the help of a skilled laboratory chemist intelligently guiding the process. New York University chemist Robert Shapiro critiqued the efforts of those who tried to make RNA in the lab, stating: “The flaw is in the logic—that this experimental control by researchers in a modern laboratory could have been available on the early Earth.”¹⁵

Second, while RNA has been shown to perform many roles in the cell, there is no evidence that it could perform all the necessary cellular functions currently carried out by proteins.¹⁶

Third, the RNA world hypothesis does not explain the origin of genetic information.

RNA world advocates suggest that if the first self-replicating life was based upon RNA, it would have required a molecule between 200 and 300 nucleotides in length.¹⁷ However, there are no known chemical or physical laws that dictate the order of those nucleotides.¹⁸ To explain the ordering of nucleotides in the first self-replicating RNA molecule, materialists must rely on sheer chance. But the odds of specifying, say, 250 nucleotides in an RNA molecule by chance is about 1 in 10^{150} —below the universal probability boundary, or events which are remotely possible to occur within the history of the universe.¹⁹ Shapiro puts the problem this way:

The sudden appearance of a large self-copying molecule such as RNA was exceedingly improbable. ... [The probability] is so vanishingly small that its happening even once anywhere in the visible universe would count as a piece of exceptional good luck.²⁰

Fourth—and most fundamentally—the RNA world hypothesis does not explain the origin of the genetic code itself. In order to evolve into the DNA / protein-based life that exists today, the RNA world would need to evolve the ability to convert genetic information into proteins. However, this process of transcription and translation requires a large suite of proteins and molecular machines—which themselves are encoded by genetic information. This poses a chicken-and-egg problem, where essential enzymes and molecular machines are needed to perform the very task that constructs them.

THE CHICKEN AND THE DVD

To appreciate this problem, consider the origin of the first DVD and DVD player. DVDs are rich in information, but without the machinery of a DVD player to read the disk, process its information, and convert it into a picture and sound, the disk would be useless. But what if the instructions for building the first DVD player were only found encoded on a DVD? You could never play the DVD to learn how to build a DVD player. So how did the first disk and DVD player system arise? The answer is obvious: a goal directed process—intelligent design—is required to produce both the player and the disk at the same time.

In living cells, information-carrying molecules (e.g. DNA or RNA) are like the DVD, and the cellular machinery which reads that information and converts it into proteins are like the DVD player. Just like the DVD analogy, genetic information can never be converted into proteins without the proper machinery. Yet in cells, the machines required for processing the genetic information in RNA or DNA are encoded by those same genetic molecules—they perform and direct the very task that builds them.

This system cannot exist unless both the genetic information and transcription / translation machinery are present at the same time, and unless both speak the same language. Biologist Frank Salisbury explained this problem in a paper in *American Biology Teacher* not long after the workings of the genetic code were first uncovered:

It's nice to talk about replicating DNA molecules arising in a soupy sea, but in modern cells this replication requires the presence of suitable enzymes. ... [T]he link between DNA and the enzyme is a highly complex one, involving RNA and an enzyme for its synthesis on a DNA template; ribosomes; enzymes to activate the amino acids; and transfer-RNA molecules. ... How, in the absence of the final enzyme, could selection act upon DNA and all the mechanisms for replicating it? It's as though everything must happen at once: the entire system must come into being as one unit, or it is worthless. There may well be ways out of this dilemma, but I don't see them at the moment.²¹

Despite decades of work, origin-of-life theorists are still at a loss to explain how this system arose. In 2007, Harvard chemist George Whitesides was given the Priestley Medal, the highest award of the American Chemical Society. During his acceptance speech, he offered

this stark analysis, reprinted in the respected journal, *Chemical and Engineering News*:

The Origin of Life. This problem is one of the big ones in science. It begins to place life, and us, in the universe. Most chemists believe, as do I, that life emerged spontaneously from mixtures of molecules in the prebiotic Earth. How? I have no idea.²²

Similarly, the aforementioned article in *Cell Biology International* concludes: “New approaches to investigating the origin of the genetic code are required. The constraints of historical science are such that the origin of life may never be understood.”²³ That is, they may never be understood unless scientists are willing to consider goal-directed scientific explanations like intelligent design.

But there is a much deeper problem with theories of chemical evolution, as well as biological evolution. This pertains not just to the ability to process genetic information via a genetic code, but the origin of that information itself.

**PROBLEM 3:
RANDOM MUTATIONS
CANNOT GENERATE THE GENETIC INFORMATION
REQUIRED FOR IRREDUCIBLY COMPLEX STRUCTURES**

According to evolutionary biologists, once life got started, Darwinian evolution took over and eventually produced the grand diversity we observe today. Under the standard view, a process of random mutation and natural selection built life’s vast complexity one small mutational step at a time. All of life’s complex features, of course, are thought to be encoded in the DNA of living organisms. Building new features thus requires generating new information in the genetic code of DNA. Can the necessary information be generated in the undirected, step-by-step manner required by Darwin’s theory?

Most everyone agrees that Darwinian evolution tends to work well when each small step along an evolutionary pathway provides some survival advantage. Darwin-critic Michael Behe notes that “if only one mutation is needed to confer some ability then Darwinian evolution has little problem finding it.”²⁴ However, when multiple mutations must be present simultaneously to gain a functional advantage, Darwinian evolution gets stuck. As Behe explains, “If more than one [mutation] is

needed, the probability of getting all the right ones grows exponentially worse.”²⁵

Behe, a professor of biochemistry at Lehigh University, coined the term “irreducible complexity” to describe systems which require many parts—and thus many mutations—to be present—all at once—before providing any survival advantage to the organism. According to Behe, such systems cannot evolve in the step-by-step fashion required by Darwinian evolution. As a result, he maintains that random mutation and unguided natural selection cannot generate the genetic information required to produce irreducibly complex structures. Too many simultaneous mutations would be required—an event which is highly unlikely to occur.

Observation of this problem is not limited to Darwin-critics. A paper by a prominent evolutionary biologist in the prestigious journal *Proceedings of the U.S. National Academy of Science* acknowledges that “simultaneous emergence of all components of a system is implausible.”²⁶ Likewise, University of Chicago evolutionary biologist Jerry Coyne—a staunch defender of Darwinism—admits that “natural selection cannot build any feature in which intermediate steps do not confer a net benefit on the organism.”²⁷ Even Darwin intuitively recognized this problem, as he wrote in *Origin of Species*:

If it could be demonstrated that any complex organ existed, which could not possibly have been formed by numerous, successive, slight modifications, my theory would absolutely break down²⁸

Evolutionary scientists like Darwin and Coyne claim they know of no real-world case where Darwinian selection gets blocked in this manner. But they would agree, at least in principle, that there are theoretical limits to what Darwinian evolution can accomplish: If a feature cannot be built by “numerous, successive, slight modifications,” and if “intermediate steps do not confer a net benefit on the organism,” then Darwinian evolution will “absolutely break down.”

The problems are real. Modern biology continues to uncover more and more examples where biological complexity seems to outstrip the information-generative capacity of Darwinian evolution.

MOLECULAR MACHINES

In his book *Darwin's Black Box*, Michael Behe discusses molecular machines which require multiple parts to be present before they could function and confer any advantage on the organism. Behe's most famous example is the bacterial flagellum—a micromolecular rotary-engine, functioning like an outboard motor on bacteria to propel it through liquid medium to find food. In this regard, flagella have a basic design that is highly similar to some motors made by humans containing many parts that are familiar to engineers, including a rotor, a stator, a u-joint, a propeller, a brake, and a clutch. As one molecular biologist writes in the journal *Cell*, “[m]ore so than other motors, the flagellum resembles a machine designed by a human.”²⁹ However the energetic efficiency of these machines outperforms anything produced by humans: the same paper found that the efficiency of the bacterial flagellum “could be ~100%.”³⁰

There are various types of flagella, but all use certain basic components. As one paper in *Nature Reviews Microbiology* acknowledges, “all (bacterial) flagella share a conserved core set of proteins” since “Three modular molecular devices are at the heart of the bacterial flagellum: the rotor-stator that powers flagellar rotation, the chemotaxis apparatus that mediates changes in the direction of motion and the T3SS that mediates export of the axial components of the flagellum.”³¹ As this might suggest, the flagellum is irreducibly complex. Genetic knockout experiments have shown that it fails to assemble or function properly if any one of its approximately 35 genes are missing.³² In this all-or-nothing game, mutations cannot produce the complexity needed to provide a functional flagellar rotary engine one incremental step at a time, and the odds are too daunting for it to assemble in one great leap. Indeed, the aforementioned *Nature Reviews Microbiology* paper admitted that “the flagellar research community has scarcely begun to consider how these systems have evolved.”³³

Yet the flagellum is just one example of thousands of known molecular machines in biology. One individual research project reported the discovery of over 250 new molecular machines in yeast alone.³⁴ The former president of the U.S. National Academy of Sciences, Bruce Alberts, wrote an article in the journal *Cell* praising the “speed,” “elegance,” “sophistication,” and “highly organized activity” of these “remarkable” and “marvelous” molecular machines. He explained what inspired those words: “Why do we call the large protein assemblies that underlie cell function protein machines? Precisely

because, like machines invented by humans to deal efficiently with the macroscopic world, these protein assemblies contain highly coordinated moving parts.”³⁵ Biochemists like Behe and others believe that with all of their coordinated interacting parts, many of these machines could not have evolved in a step-by-step Darwinian fashion.

But it’s not just multi-part machines which are beyond reach of Darwinian evolution. The protein-parts themselves which build these machines would also require multiple simultaneous mutations in order to arise.

RESEARCH CHALLENGES THE DARWINIAN MECHANISM

In 2000 and 2004, protein scientist Douglas Axe published experimental research in the *Journal of Molecular Biology* on mutational sensitivity tests he performed on enzymes in bacteria.³⁶ Enzymes are long chains of amino acids which fold into a specific, stable, three-dimensional shape in order to function. Mutational sensitivity experiments begin by mutating the amino acid sequences of those proteins, and then testing the mutant proteins to determine whether they can still fold into a stable shape, and function properly. Axe’s research found that amino acid sequences which yield stable, functional protein folds may be as rare as 1 in 10^{74} sequences’ suggesting that the vast majority of amino acid sequences will not produce stable proteins, and thus could not function in living organisms.

Because of this extreme rarity of functional protein sequences, it would be very difficult for random mutations to take a protein with one type of fold, and evolve it into another, without going through some non-functional stage. Rather than evolving by “numerous, successive, slight modifications,” many changes would need to occur *simultaneously* to “find” the rare and unlikely amino acid sequences that yield functional proteins. To put the matter in perspective, Axe’s results suggest that the odds of blind and unguided Darwinian processes producing a functional protein fold are less than the odds of someone closing his eyes and firing an arrow into the Milky Way galaxy, *and hitting one pre-selected atom.*³⁷

Proteins commonly interact with other molecules through a “hand-in-glove” fit, but these interactions often require multiple amino acids to be ‘just right’ before they occur. In 2004, Behe, along with University of Pittsburgh physicist David Snoke, simulated the Darwinian evolution of such protein-protein interactions. Behe and Snoke’s calculations found that for multicellular organisms, evolving a simple protein-

protein interaction which required two or more mutations in order to function would probably require more organisms and generations than would be available over the entire history of the Earth. They concluded that “the mechanism of gene duplication and point mutation alone would be ineffective...because few multicellular species reach the required population sizes.”³⁸

Four years later during an attempt to refute Behe’s arguments, Cornell biologists Rick Durrett and Deena Schmidt ended up begrudgingly confirming he was basically correct. After calculating the likelihood of two simultaneous mutations arising via Darwinian evolution in a population of humans, they found that such an event “would take > 100 million years.” Given that humans diverged from their supposed common ancestor with chimpanzees only 6 million years ago, they granted that such mutational events are “very unlikely to occur on a reasonable timescale.”³⁹

Now a defender of Darwinism might reply that these calculations measured the power of the Darwinian mechanism only within multicellular organisms where it is less efficient because these more complex organisms have smaller population sizes and longer generation times than single-celled prokaryotic organisms like bacteria. Darwinian evolution, the Darwinian notes, might have a better shot when operating in organisms like bacteria, which reproduce more rapidly and have much larger population sizes. Scientists skeptical of Darwinian evolution are aware of this objection, and have found that even within more-quickly evolving organisms like bacteria, Darwinian evolution faces great limits.

In 2010, Douglas Axe published evidence indicating that despite high mutation rates and generous assumptions favoring a Darwinian process, molecular adaptations requiring more than six mutations before yielding any advantage would be extremely unlikely to arise in the history of the Earth.

The following year, Axe published research with developmental biologist Ann Gauger regarding experiments to convert one bacterial enzyme into another closely related enzyme—the kind of conversion that evolutionists claim can easily happen. For this case they found that the conversion would require a minimum of at least *seven* simultaneous changes,⁴⁰ exceeding the six-mutation-limit which Axe had previously established as a boundary of what Darwinian evolution is likely to accomplish in bacteria. Because this conversion is thought to be relatively simple, it suggests that more complex biological features

would require more than six simultaneous mutations to give some new functional advantage.

In other experiments led by Gauger and biologist Ralph Seelke of the University of Wisconsin, Superior, their research team broke a gene in the bacterium *E. coli* required for synthesizing the amino acid tryptophan. When the bacteria's genome was broken in just one place, random mutations were capable of “fixing” the gene. But even when only two mutations were required to restore function, Darwinian evolution seemed to get stuck, with an inability to regain full function.⁴¹

These kind of results consistently suggest that the information required for proteins and enzymes to function is too great to be generated by Darwinian processes on any reasonable evolutionary timescale.

DARWIN SKEPTICS ABOUND

Drs. Axe, Gauger, and Seelke are by no means the only scientists to observe the rarity of amino acid sequences that yield functional proteins. A leading college-level biology textbook states that “even a slight change in primary structure can affect a protein's conformation and ability to function.”⁴² Likewise, evolutionary biologist David S. Goodsell writes:

[O]nly a small fraction of the possible combinations of amino acids will fold spontaneously into a stable structure. If you make a protein with a random sequence of amino acids, chances are that it will only form a gooey tangle when placed in water.⁴³

Goodsell goes on to assert that “cells have perfected the sequences of amino acids over many years of evolutionary selection.” But if functional protein sequences are rare, then it is likely that natural selection will be unable to take proteins from one functional genetic sequence to another without getting stuck in some maladaptive or non-beneficial intermediate stage.

The late biologist Lynn Margulis, a well-respected member of the National Academy of Sciences until her death in 2011, once said “new mutations don't create new species; they create offspring that are impaired.”⁴⁴ She further explained in a 2011 interview:

[N]eo-Darwinists say that new species emerge when mutations occur and modify an organism. I was taught over and over again that the

accumulation of random mutations led to evolutionary change-led to new species. I believed it until I looked for evidence.⁴⁵

Similarly, past president of the French Academy of Sciences, Pierre-Paul Grasse, contended that "[m]utations have a very limited 'constructive capacity'" because "[n]o matter how numerous they may be, mutations do not produce any kind of evolution."⁴⁶

Many other scientists feel this way. Over 800 Ph.D. scientists have signed a statement agreeing they "are skeptical of claims for the ability of random mutation and natural selection to account for the complexity of life."⁴⁷ Indeed, two biologists wrote in *Annual Review of Genomics and Human Genetics*: "it remains a mystery how the undirected process of mutation, combined with natural selection, has resulted in the creation of thousands of new proteins with extraordinarily diverse and well optimized functions. This problem is particularly acute for tightly integrated molecular systems that consist of many interacting parts..."⁴⁸ Perhaps it would be less mysterious if the theoretical conceptions could be expanded beyond unguided evolutionary mechanisms like random mutation and natural selection to explain the origin of complex biological features.

PROBLEM 4: NATURAL SELECTION STRUGGLES TO FIX ADVANTAGEOUS TRAITS INTO POPULATIONS.

In 2008, 16 biologists from around the world convened in Altenberg, Austria to discuss problems with the modern neo-Darwinian model of evolution. The journal *Nature* covered this "Altenberg 16" conference, quoting leading scientists saying things like:

- "[T]he origin of wings and the invasion of the land . . . are things that evolutionary theory has told us little about."⁴⁹
- "You can't deny the force of selection in genetic evolution . . . but in my view this is stabilizing and fine-tuning forms that originate due to other processes."
- "The modern synthesis is remarkably good at modeling the survival of the fittest, but not good at modeling the arrival of the fittest."

In Problem 3, we learned that mutations cannot generate many complex traits in living organisms on reasonable evolutionary timescales. But mutations are only part of the standard evolutionary mechanism—there is also natural selection. And Darwinian evolution not only commonly fails to explain the “arrival of the fittest” via mutations, but also often struggles to explain the “survival of the fittest” via natural selection.

Evolutionary biologists often assume that once mutations produce a functionally advantageous trait, it will easily spread (become “fixed”) throughout a population by natural selection. For example, imagine a population of brown-haired foxes which lives in a snowy region. One fox is born with a mutation which turns its fur coat white, rather than brown. This fox now has an advantage in hunting prey and escaping predators, because its white fur provides it with camouflage in the snow-filled environment. The white fox survives, passing its genes on to its offspring, which are also adept at surviving and reproducing. Over time, the white-haired trait spreads throughout the population.

This is how it’s supposed to work—in theory. In the real world, however, merely generating a functionally advantageous trait does not guarantee it will persist, or become fixed. For example, what if by chance the white fox trips, breaks a leg, and gets eaten by a predator—never passing on its genes? Random forces or events can prevent a trait from spreading through a population, even if it provides an advantage. These random forces are lumped together under the name “genetic drift.” When biologists run the mathematics of natural selection, they find that unless a trait gives an extremely strong selective advantage, genetic drift will tend to overwhelm the force of selection and prevent adaptations from gaining a foothold in a population.

This underappreciated problem has been recognized by some evolutionary scientists who are skeptical of the ability of natural selection to drive the evolutionary process. One of those scientists is Michael Lynch, an evolutionary biologist at Indiana University, who writes that “random genetic drift can impose a strong barrier to the advancement of molecular refinements by adaptive processes.”⁵⁰ He notes that the effect of drift is “encouraging the fixation of mildly deleterious mutations and discouraging the promotion of beneficial mutations.”⁵¹ Likewise, Eugene Koonin, a leading scientist at the National Institutes of Health, explains, genetic drift leads to “random fixation of neutral or even deleterious changes.”⁵²

COMPLEX REDUNDANCY

In Lynch's view, there are many cellular systems which aid in survival, but are redundant. As a result, they serve as backup mechanisms that are only used when a highly effective primary system fails. Because they are only seldom used, these systems are only occasionally exposed to the sieve of selection. Yet these systems can be extremely complex and efficient. How can a system which is only rarely used, or only occasionally needed, evolve to such a high and efficient level of complexity? After observing the many "layers" of complex cellular mechanisms which are involved in processes like DNA replication, Lynch poses a crucial question:

Although these layered lines of defense are clearly advantageous and in many cases essential to cell health, because the simultaneous emergence of all components of a system is implausible, several questions immediately arise. How can selection promote the establishment of additional layers of fitness-enhancing mechanisms if the established primary lines of defense are already highly refined?⁵³

Lynch doesn't believe natural selection is up to the task. In a 2007 paper in *Proceedings of the U.S. National Academy of Sciences* titled "The frailty of adaptive hypotheses for the origins of organismal complexity," he explains that among evolutionary biologists, "What is in question is whether natural selection is a necessary or sufficient force to explain the emergence of the genomic and cellular features central to the building of complex organisms."⁵⁴ Using similar language, a paper in the journal *Theoretical Biology and Medical Modelling* concludes that "it is important for biologists to realistically appraise what selection can and cannot do under various circumstances. Selection may neither be necessary nor sufficient to explain numerous genomic or cellular features of complex organisms."⁵⁵ Lynch is clear in his views: "there is no compelling empirical or theoretical evidence that complexity, modularity, redundancy or other features of genetic pathways are promoted by natural selection."⁵⁶

DAMNED IF YOU APPEAL TO SELECTION, DAMNED IF YOU DON'T

In place of natural selection, however, evolutionary biologists like Lynch propose random genetic drift to explain the origin of complex biological features. According to Lynch, "many aspects of complexity at

the genomic, molecular and cellular levels in multicellular species are likely to owe their origins to these non-adaptive forces, representing little more than passive outcomes...”⁵⁷ But he recognizes that these “nonadaptive forces of evolution are stochastic in nature.”⁵⁸

Stochastic, of course, means random. Can a strictly random force—which has no reason to preserve features that might provide some advantage—explain the highly complex biological features—like DNA replication or bioluminescence—which appear finely tuned to perform useful biological functions? Biologist Ann Gauger is skeptical of Lynch’s explanation, as she observes that he “offers no explanation of how non-adaptive forces can produce the functional genomic and organismal complexity we observe in modern species.”⁵⁹ Jerry Coyne similarly points out the major deficiency in appeals to genetic drift:

Both drift and natural selection produce genetic change that we recognize as evolution. But there’s an important difference. Drift is a random process, while selection is the anti-thesis of randomness. ... As a purely random process, genetic drift can’t cause the evolution of adaptations. It could never build a wing or an eye. That takes nonrandom natural selection. What drift can do is cause the evolution of features that are neither useful nor harmful to the organism.⁶⁰

Coyne further observes: “The influence of this process on important evolutionary change, though, is probably minor, because it does not have the molding power of natural selection. Natural selection remains the only process that can produce adaptation.”⁶¹ But in a sense agreeing with Lynch, even he recognizes that “genetic drift is not only powerless to create adaptations, but can actually *overpower* natural selection.”⁶²

The debate over whether natural selection, or genetic drift, is more influential in evolution will undoubtedly continue. But there is little reason to believe that whichever side wins this debate, a viable materialistic solution will be offered. Evolutionary biology now finds itself facing a catch-22:

- Natural selection is too inefficient a mechanism to overcome random forces and fix the sort of complex adaptations we observe in populations because it is easily overpowered by random forces like genetic drift.

- Life is full of highly complex and efficient adaptations, but random genetic drift offers no justifiable reason to believe that such features will have any reason to arise.

In essence, genetic drift is like invoking the “mutation-selection” mechanism, but minus all of the selection. This subjects drift to all of the difficulties we saw in Problem 3, where random mutations were unable to build biochemical features like functional proteins, or simple protein-protein interactions, because multiple coordinated mutations were required to produce those traits. Absent selection, there is no reason for random mutations alone—i.e. genetic drift—to produce anything useful.

Unfortunately, the public is rarely made aware of these problems or this debate. According to Lynch, natural selection is typically portrayed as an “all powerful (without any direct evidence)”⁶³ mechanism that can build complex biological features. He warns that “the myth that all of evolution can be explained by adaptation continues to be perpetuated by our continued homage to Darwin’s treatise in the popular literature.”⁶⁴ The reality is that neither non-random forces like natural selection, nor random forces like genetic drift, can explain the origin of many complex biological features.

PROBLEM 5: ABRUPT APPEARANCE OF SPECIES IN THE FOSSIL RECORD DOES NOT SUPPORT DARWINIAN EVOLUTION.

The fossil record has long-been recognized as a problem for evolutionary theory. In *Origin of Species*, Darwin explained that his theory led him to believe that “[t]he number of intermediate varieties, which have formerly existed on the earth, [must] be truly enormous.”⁶⁵ However, he recognized that the fossil record did not document these “intermediate” forms of life, asking, “Why then is not every geological formation and every stratum full of such intermediate links?”⁶⁶ Darwin’s answer showed the tenuous nature of the evidence backing his ideas: “Geology assuredly does not reveal any such finely graduated organic chain; and this, perhaps, is the most obvious and gravest objection which can be urged against my theory.”⁶⁷

Today, some 150 years later, out of thousands of species known from the fossil record, only a small fraction are claimed to be candidates for Darwin’s intermediate forms. Fossil evidence of evolutionary

intermediates is generally lacking, as the late evolutionary paleontologist Stephen Jay Gould admitted: “The absence of fossil evidence for intermediary stages between major transitions in organic design, indeed our inability, even in our imagination, to construct functional intermediates in many cases, has been a persistent and nagging problem for gradualistic accounts of evolution.”⁶⁸

Darwin attempted to save his theory of gradual evolution by maintaining that intermediate fossils are not found because of “the extreme imperfection of the geological record.”⁶⁹ Even Gould noted that Darwin’s argument that the fossil record is imperfect “persists as the favored escape of most paleontologists from the embarrassment of a record that seems to show so little of evolution directly.”⁷⁰ But in the last few decades, this excuse has lost credibility.

Paleontologists today generally recognize that while the fossil record is *imperfect*, it is still *adequate* to assess questions about evolution. One study in *Nature* reported that “if scaled to the ... taxonomic level of the family, the past 540 million years of the fossil record provide uniformly good documentation of the life of the past.”⁷¹ Another paper in *Paleobiology* evaluated our knowledge of the fossil record and concluded that “our view of the history of biological diversity is mature.”⁷² Paleontologists now increasingly recognize that “jumps” between species, without intermediates, are not simply the result of an incomplete record. Niles Eldredge, an evolutionary paleontologist and curator at the American Museum of Natural History, puts it this way with Ian Tattersal: “The record jumps, and all the evidence shows that the record is real: the gaps we see reflect real events in life’s history -- not the artifact of a poor fossil record.”⁷³ This conclusion did not come easily, as one scientist who studied under Gould felt the need to implore his colleagues that “[e]volutionary biologists can no longer ignore the fossil record on the ground that it is imperfect.”⁷⁴

A PATTERN OF EXPLOSIONS

The eventual realization that the fossil record is not entirely incomplete has forced evolutionary biologists to accept that the record shows *a pattern of explosions, not gradual evolution of living organisms*. One biology textbook explains this:

Many species remain virtually unchanged for millions of years, then suddenly disappear to be replaced by a quite different, but related, form. Moreover, most major groups of animals appear abruptly in

the fossil record, fully formed, and with no fossils yet discovered that form a transition from their parent group.⁷⁵

Probably the most famous instance of abrupt appearance is the Cambrian explosion, where nearly all of the major living animal phyla appear in the Cambrian period. An invertebrate biology textbook explains this:

Most of the animal groups that are represented in the fossil record first appear, ‘fully formed’ and identifiable as to their phylum, in the Cambrian, some 550 million years ago. These include such anatomically complex and distinctive types as trilobites, echinoderms, brachiopods, molluscs, and chordates. ... The fossil record is therefore of no help with respect to the origin and early diversification of the various animal phyla...⁷⁶

Evolutionary scientists acknowledge that they cannot explain this rapid appearance of diverse animal body plans by classical Darwinian processes, or other known material mechanisms. Robert Carroll, a paleontologist at McGill University, argues in *Trends in Ecology and Evolution* that “The extreme speed of anatomical change and adaptive radiation during this brief time period requires explanations that go beyond those proposed for the evolution of species within the modern biota.”⁷⁷ Another paper likewise maintains that “microevolution does not provide a satisfactory explanation for the extraordinary burst of novelty during the Cambrian Explosion” and concludes “the major evolutionary transitions in animal evolution still remain to be causally explained.”⁷⁸ Likewise a 2009 paper in *BioEssays* concedes that “elucidating the materialistic basis of the Cambrian explosion has become more elusive, not less, the more we know about the event itself.”⁷⁹

But the Cambrian explosion is by no means the only explosion of life recorded in the fossil record. Regarding the origin of major fish groups, former Columbia University geoscientist Arthur Strahler writes that, “This is one count in the creationists’ charge that can only evoke in unison from paleontologists a plea of *nolo contendere* [no contest].”⁸⁰ A paper in *Annual Review of Ecology and Systematics* explains that the origin of land plants “is the terrestrial equivalent of the much-debated Cambrian ‘explosion’ of marine faunas.”⁸¹ Regarding the origin

of angiosperms (flowering plants), paleontologists have discovered a “big bloom” type of explosion event. As one paper states:

In spite of much research and analyses of different sources of data (e.g., fossil record and phylogenetic analyses using molecular and morphological characters), the origin of the angiosperms remains unclear. Angiosperms appear rather suddenly in the fossil record... with no obvious ancestors for a period of 80-90 million years before their appearance.⁸²

In a similar way, many orders of mammals appear in an explosive manner. Niles Eldredge explains that “there are all sorts of gaps: absence of gradationally intermediate ‘transitional’ forms between species, but also between larger groups—between, say, families of carnivores, or the orders of mammals.”⁸³ There is also a bird explosion, with major bird groups appearing in a short time period.⁸⁴ One paper in *Trends in Ecology and Evolution* titled “Evolutionary Explosions and the Phylogenetic Fuse” explains:

A literal reading of the fossil record indicates that the early Cambrian (c. 545 million years ago) and early Tertiary (c. 65 million years ago) were characterized by enormously accelerated periods of morphological evolution marking the appearance of the animal phyla, and modern bird and placental mammal orders, respectively.⁸⁵

Of course there are a handful of examples where evolutionary scientists believe they have found transitional fossils documenting gradual Darwinian evolution. The origin of whales has been called a “poster child for macroevolution,”⁸⁶ where it is believed that around 55 million years ago, certain land mammals lost their hind-limbs and evolved into fully aquatic whales. In particular, it is claimed there are fossil land-mammals with ear-bones similar to whales, and fossil whale-like mammals that retain their hindlimbs.

Even though vertebrate and whale expert Phillip Gingerich admits that we only have “fossils illustrating three or four steps that bridge the precursor of whales to today's mammals,”⁸⁷ let's assume for a moment that a full sequence of fossils exists. Is this enough to demonstrate that this transition occurred? Even if there are fossils that look like potential intermediate forms, if the overall evolutionary story does not make sense, then the fossils cannot be transitional. In this case, the Darwinian

evolution of whales from land-mammals faces serious mathematical challenges from population genetics.

Many changes would have been necessary to convert a land-mammal into a whale, including:

- Emergence of a blowhole, with musculature and nerve control
- Modification of the eye for permanent underwater vision
- Ability to drink sea water
- Forelimbs transformed into flippers
- Modification of skeletal structure
- Ability to nurse young underwater
- Origin of tail flukes and musculature
- Blubber for temperature insulation⁸⁸

Many of these necessary adaptations would require multiple coordinated changes. But as we saw in Problem 3, such simultaneous mutations require extremely long periods of time to arise via the Darwinian mechanism. Whale evolution now runs into a severe problem. The fossil record requires that the evolution of whales from small land mammals would have to have taken place in less than 10 million years.⁸⁹ That may sound like a long time, but it actually falls dramatically short, especially given that whales have small population sizes and long generation times.⁹⁰ Biologist Richard Sternberg has examined the requirements of this transition mathematically and puts it this way: “Too many genetic re-wirings, too little time.”⁹¹

Whale origins thus provides an interesting case study of evolutionary transitions: On a rare occasion where there actually are fossils that potentially show intermediate traits, unguided neo-Darwinian evolution is invalidated by the short amount of time allowed by the fossil record. If this “poster child” of macroevolution doesn’t hold up to scrutiny, what does this tell us about other cases where evolutionists tout supposed transitional fossils?

HUMAN ORIGINS AND THE FOSSIL RECORD

Indeed, the public is commonly told that there are fossils documenting the evolution of humans from ape-like precursors, but a closer look at the technical literature tells a different story. Hominid fossils generally fall into one of two groups: ape-like species and human-like species, with a large, unbridged gap between them. In 2004, the famed

evolutionary biologist Ernst Mayr recognized the abrupt appearance of humans:

The earliest fossils of *Homo*, *Homo rudolfensis* and *Homo erectus*, are separated from *Australopithecus* by a large, unbridged gap. How can we explain this seeming saltation? Not having any fossils that can serve as missing links, we have to fall back on the time-honored method of historical science, the construction of a historical narrative.⁹²

In light of such evidence, a paper in the *Journal of Molecular Biology and Evolution* called the appearance of *Homo sapiens* “a genetic revolution” where “no australopithecine species is obviously transitional.”⁹³ The lack of fossil evidence for this hypothesized transition is confirmed by Harvard paleoanthropologists Daniel E. Lieberman, David R. Pilbeam, and Richard W. Wrangham:

Of the various transitions that occurred during human evolution, the transition from *Australopithecus* to *Homo* was undoubtedly one of the most critical in its magnitude and consequences. As with many key evolutionary events, there is both good and bad news. First, the bad news is that many details of this transition are obscure because of the paucity of the fossil and archaeological records.⁹⁴

As for the “good news,” they still admit: “although we lack many details about exactly how, when, and where the transition occurred from *Australopithecus* to *Homo*, we have sufficient data from before and after the transition to make some inferences about the overall nature of key changes that did occur.”⁹⁵ In other words, the fossil record provides ape-like australopithecines (“before”), and human-like *Homo* (“after”), but not fossils documenting a transition between them. In the absence of intermediates, we’re left with “inferences” of a transition based strictly upon the assumption of Darwinian evolution. One commentator proposed the evidence implies a “big bang theory” of the appearance of our genus *Homo*.⁹⁶ This does not make for a compelling evolutionary account of human origins.⁹⁷

Rather than showing gradual Darwinian evolution, the history of life shows a pattern of explosions where new fossil forms come into existence without clear evolutionary precursors. Evolutionary anthropologist Jeffrey Schwartz summarizes the problem:

[W]e are still in the dark about the origin of most major groups of organisms. They appear in the fossil record as Athena did from the head of Zeus—full-blown and raring to go, in contradiction to Darwin’s depiction of evolution as resulting from the gradual accumulation of countless infinitesimally minute variations. . . .”⁹⁸

This poses a major challenge to Darwinian evolution, including the view that all animals are related through common ancestry.

**PROBLEM 6:
MOLECULAR BIOLOGY
HAS FAILED TO YIELD A GRAND “TREE OF LIFE.”**

When fossils failed to demonstrate that animals evolved from a common ancestor, evolutionary scientists turned to another type of evidence—DNA sequence data—to demonstrate a tree of life. In the 1960s, around the time the genetic code was first understood, biochemists Émile Zuckerkandl and Linus Pauling hypothesized that if DNA sequences could be used to produce evolutionary trees—trees which matched those based upon morphological or anatomical characteristics—this would furnish “the best available single proof of the reality of macro-evolution.”⁹⁹ Thus began a decades-long effort to sequence the genes of many organisms and construct “molecular” based evolutionary (“phylogenetic”) trees. The ultimate goal has been to construct a grand “tree of life,” showing how all living organisms are related through universal common ancestry.

THE MAIN ASSUMPTION

The basic logic behind building molecular trees is relatively simple. First, investigators choose a gene, or a suite of genes, found across multiple organisms. Next, those genes are analyzed to determine their nucleotide sequences, so the gene sequences of various organisms can then be compared. Finally, an evolutionary tree is constructed based upon the principle that the more similar the nucleotide sequence, the more closely related the species. A paper in the journal *Biological Theory* puts it this way:

[M]olecular systematics is (largely) based on the assumption, first clearly articulated by Zuckerkandl and Pauling (1962), that degree of overall similarity reflects degree of relatedness.¹⁰⁰

This assumption is essentially an articulation of a major feature of the theory – the idea of universal common ancestry. Nonetheless, it's important to realize that it is a *mere assumption* to claim that genetic similarities between different species necessarily result common ancestry.

Operating strictly within a Darwinian paradigm, these assumptions flow naturally. As the aforementioned *Biological Theory* paper explains, the main assumption underlying molecular trees “derives from interpreting molecular similarity (or dissimilarity) between taxa in the context of a Darwinian model of continual and gradual change.”¹⁰¹ So the theory is assumed to be true to construct a tree. But also, if Darwinian evolution is true, construction of trees using different sequences should reveal a reasonably consistent pattern across different genes or sequences.

This makes it all the more significant that efforts to build a grand “tree of life” using DNA or other biological sequence data have not conformed to expectations. The basic problem is that one gene gives one version of the tree of life, while another gene gives a highly different, and conflicting, version of the tree. For example, as we'll discuss further below, the standard mammalian tree places humans more closely related to rodents than to elephants. But studies of a certain type of DNA called microRNA genes have suggested the opposite—that humans were closer to elephants than rodents. Such conflicts between gene-based trees are extremely common.

The genetic data is thus not painting a consistent picture of common ancestry, showing the assumptions behind tree-building commonly fail. This leads to justifiable questions about whether universal common ancestry is correct.

CONFLICTS IN THE BASE OF THE TREE OF LIFE

Problems first arose when molecular biologists sequenced genes from the three basic domains of life—bacteria, archaea, and eukarya—but those genes did not allow these basic groups of life to be resolved into a treelike pattern. In 2009, the journal *New Scientist* published a cover story titled, “Why Darwin was wrong about the tree of life” which explained these quandaries:

The problems began in the early 1990s when it became possible to sequence actual bacterial and archaeal genes rather than just RNA. Everybody expected these DNA sequences to confirm the RNA

tree, and sometimes they did but, crucially, sometimes they did not. RNA, for example, might suggest that species A was more closely related to species B than species C, but a tree made from DNA would suggest the reverse.¹⁰²

This sort of data led biochemist W. Ford Doolittle to explain that “Molecular phylogenists will have failed to find the ‘true tree,’ not because their methods are inadequate or because they have chosen the wrong genes, but because the history of life cannot properly be represented as a tree.”¹⁰³ *New Scientist* put it this way: “For a long time the holy grail was to build a tree of life ... But today the project lies in tatters, torn to pieces by an onslaught of negative evidence.”¹⁰⁴

Many evolutionists sometimes reply that these problems arise only when studying microorganisms like bacteria—organisms which can swap genes through a process called “horizontal gene transfer,” thereby muddying the signal of evolutionary relationships. But this objection isn’t quite true, since the tree of life is challenged even among higher organisms where such gene-swapping is not prevalent. Carl Woese, a pioneer of evolutionary molecular systematics, explains:

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.¹⁰⁵

Likewise, the *New Scientist* article notes that “research suggests that the evolution of animals and plants isn't exactly tree-like either.”¹⁰⁶ The article explains what happened when microbiologist Michael Syvanen tried to create a tree showing evolutionary relationships using 2000 genes from a diverse group of animals:

He failed. The problem was that different genes told contradictory evolutionary stories. ... the genes were sending mixed signals. ... Roughly 50 per cent of its genes have one evolutionary history and 50 per cent another.¹⁰⁷

The data were so difficult to resolve into a tree that Syvanen lamented, “We’ve just annihilated the tree of life.”¹⁰⁸ Many other papers in the technical literature recognize similar problems.

CONFLICTS BETWEEN HIGHER BRANCHES

A 2009 paper in *Trends in Ecology and Evolution* notes that, “A major challenge for incorporating such large amounts of data into inference of species trees is that conflicting genealogical histories often exist in different genes throughout the genome.”¹⁰⁹ Similarly, a paper in *Genome Research* studied the DNA sequences in various animal groups and found that “different proteins generate different phylogenetic tree[s].”¹¹⁰

A June, 2012 article in *Nature* reported that short strands of RNA called microRNAs “are tearing apart traditional ideas about the animal family tree.” Dartmouth biologist Kevin Peterson who studies microRNAs lamented, “I’ve looked at thousands of microRNA genes, and I can’t find a single example that would support the traditional tree.” According to the article, microRNAs yielded “a radically different diagram for mammals: one that aligns humans more closely with elephants than with rodents.” Peterson put it bluntly: “The microRNAs are totally unambiguous ... they give a totally different tree from what everyone else wants.”¹¹¹

CONFLICTS BETWEEN MOLECULES AND MORPHOLOGY

Not all phylogenetic trees are constructed by comparing molecules like DNA from different species. Many trees are based upon comparing the form, structure, and body plan of different organisms—also called “morphology.” But conflicts between molecule-based trees and morphology-based trees are also common. A 2012 paper studying bat relationships made this clear, stating: “Incongruence between phylogenies derived from morphological versus molecular analyses, and between trees based on different subsets of molecular sequences has become pervasive as datasets have expanded rapidly in both characters and species.”¹¹²

This is hardly the only study to encounter conflicts between DNA-based trees and trees based upon anatomical or morphological characteristics. Textbooks often claim common descent is supported using the example of a tree of animals based upon the enzyme *cytochrome c* which matches the traditional evolutionary tree based upon morphology.¹¹³ However, textbooks rarely mention that the tree based upon a different enzyme, *cytochrome b*, sharply conflicts with the standard evolutionary tree. As one article in *Trends in Ecology and Evolution* observed:

[T]he mitochondrial cytochrome b gene implied . . . an absurd phylogeny of mammals, regardless of the method of tree construction. Cats and whales fell within primates, grouping with simians (monkeys and apes) and strepsirhines (lemurs, bush-babies and lorises) to the exclusion of tarsiers. Cytochrome b is probably the most commonly sequenced gene in vertebrates, making this surprising result even more disconcerting.¹¹⁴

Strikingly, a different article in *Trends in Ecology and Evolution* concluded, “the wealth of competing morphological, as well as molecular proposals [of] the prevailing phylogenies of the mammalian orders would reduce [the mammalian tree] to an unresolved bush, the only consistent [evolutionary relationship] probably being the grouping of elephants and sea cows.”¹¹⁵ Because of such conflicts, a major review article in *Nature* reported, “disparities between molecular and morphological trees” lead to “evolution wars” because “[e]volutionary trees constructed by studying biological molecules often don’t resemble those drawn up from morphology.”¹¹⁶

Finally, a study published in *Science* in 2005 tried to use genes to reconstruct the relationships of the animal phyla, but concluded that “[d]espite the amount of data and breadth of taxa analyzed, relationships among most [animal] phyla remained unresolved.” The following year, the same authors published a scientific paper titled, “Bushes in the Tree of Life,” which offered striking conclusions. The authors acknowledge that “a large fraction of single genes produce phylogenies of poor quality,” observing that one study “omitted 35% of single genes from their data matrix, because those genes produced phylogenies at odds with conventional wisdom.” The paper suggests that “certain critical parts of the [tree of life] may be difficult to resolve, regardless of the quantity of conventional data available.” The paper even contends that “[t]he recurring discovery of persistently unresolved clades (bushes) should force a re-evaluation of several widely held assumptions of molecular systematics.”¹¹⁷

Unfortunately, one assumption that these evolutionary biologists aren’t willing to re-evaluate is the assumption that universal common ancestry is correct. They appeal to a myriad of *ad hoc* arguments—horizontal gene transfer, long branch attraction, rapid evolution, different rates of evolution, coalescent theory, incomplete sampling, flawed methodology, and convergent evolution—to explain away inconvenient data which doesn’t fit the coveted treelike pattern. As a

2012 paper stated, “phylogenetic conflict is common, and frequently the norm rather than the exception.”¹¹⁸ At the end of the day, the dream that DNA sequence data would fit into a nice-neat tree of life has failed, and with it a key prediction of neo-Darwinian theory.

PROBLEM 7:

CONVERGENT EVOLUTION CHALLENGES DARWINISM AND DESTROYS THE LOGIC BEHIND COMMON ANCESTRY.

In Problem 6, we saw that the *main assumption* underlying all phylogenetic trees is that biological similarity is the result of inheritance from a common ancestor. The problem for evolutionary biologists faced with conflicting evolutionary trees is that biological similarity often appears in places not predicted by common descent. In other words, everyone recognizes that biological similarities often appear among species in cases where they *cannot be explained* as the result of inheritance from a common ancestor. This means the main assumption fails.

We also saw at the end of Problem 6 that when biologists are unable to construct phylogenetic trees, they often make *ad hoc* appeals to other processes to explain away data that won't fit a treelike pattern. One of these explanations is convergent evolution, where evolutionary biologists postulate that organisms acquire the same traits independently, in separate lineages, and not through inheritance from a common ancestor. Whenever evolutionary biologists are forced to appeal to convergent evolution, it reflects a breakdown in the *main assumption*, and an inability to fit the data to a treelike pattern. Examples of this abound in the literature, but a few will suffice.

GENETIC CONVERGENT EVOLUTION

A paper in the *Journal of Molecular Evolution* found that molecule-based phylogenies conflicted sharply with previously established phylogenies of major mammal groups, concluding that this anomalous tree “is not due to a stochastic error, but is due to convergent or parallel evolution.”¹¹⁹

A study in *Proceedings of the U.S. National Academy of Sciences* explains that when biologists tried to construct a phylogenetic tree for the major groups of birds using mitochondrial DNA (mtDNA), their results conflicted sharply with traditional notions of bird relationships. They even found “convergent” similarity between some bird mtDNA and the

mtDNA of distant species such as snakes and lizards. The article suggests bird mtDNA underwent “multiple independent originations,” with their study proposing “multiple independent origins for a particular mtDNA gene order among diverse birds.”¹²⁰

A 2005 paper in *Nature Immunology* observed that plants and animals have a highly similar biochemical organization of their respective innate immune systems, but their common ancestor didn’t have such an immune system:

Although it seems to be generally accepted that the innate immune responses of plants and animals share at least some common evolutionary origins, examination of the available data fails to support that conclusion, despite similarities in the overall ‘logic’ of the innate immune response in diverse multicellular [organisms].¹²¹

According to the paper, common descent cannot explain these “unexpectedly similar” systems, “suggesting independent evolutionary origins in plants and animals.” The paper is forced to conclude that such complex similarities make for a “compelling case for convergent evolution of innate immune pathways.”¹²²

Another famous example of convergent evolution is the ability of bats and whales to use echolocation, even though their distant common ancestor did not have this trait. Evolutionary biologists long-believed this was a case of morphological convergence, but an article in *Current Biology* explains the “surprising” finding that echolocation in bats and whales also involves *genetic* convergence:

Only microbats and toothed whales have acquired sophisticated echolocation, indispensable for their orientation and foraging. Although the bat and whale biosonars originated independently and differ substantially in many aspects, we here report the surprising finding that the bottlenose dolphin, a toothed whale, is clustered with microbats in the gene tree constructed using protein sequences encoded by the hearing gene *Prestin*.¹²³

One paper called this data, “one of the best examples of convergent molecular evolution discovered to date”¹²⁴ But again, these are hardly isolated examples. In 2010, a paper in *Trends in Genetics* explained:

The recent wide use of genetic and/or phylogenetic approaches has uncovered diverse examples of repeated evolution of adaptive traits including the multiple appearances of eyes, echolocation in bats and dolphins, pigmentation modifications in vertebrates, mimicry in butterflies for mutualistic interactions, convergence of some flower traits in plants, and multiple independent evolution of particular protein properties.¹²⁵

Biochemist and Darwin-skeptic Fazale Rana reviewed the technical literature and documented over 100 reported cases of convergent genetic evolution.¹²⁶ Each case shows an example where biological similarity—even at the *genetic* level—is *not* the result of inheritance from a common ancestor. So what does this do to the main assumption of tree-building that biological similarity implies inheritance from a common ancestor? With so many exceptions to the rule, one has to wonder if the rule itself holds merit.

THE EARTH IS ROUND, BUT IS COMMON ANCESTRY TRUE?

One evolutionary scientist tried to pressure his readers into accepting Darwinism by claiming “biologists today consider the common ancestry of all life a fact on par with the sphericity of the earth.”¹²⁷ But are such categorical statements even helpful, much less true?

Proponents of neo-Darwinian evolution are forced into reasoning that biological similarity implies common ancestry, *except for when it doesn't*. And in the many cases where it doesn't, they appeal to all sorts of *ad hoc* rationalizations to save common ancestry.

Tellingly, the one assumption rarely questioned is the overall assumption of common ancestry itself. But perhaps the reason why different genes are telling different evolutionary stories is because the genes *have wholly different stories to tell*, namely stories that indicate that all organisms are *not* genetically related. There is some hope for a different story more attuned to the data, as Michael Syvanen dared to suggest in *Annual Review of Genetics* in 2012, that “life might indeed have multiple origins.”¹²⁸ In other words, universal common ancestry may in fact, not be true.

PROBLEM 8:
DIFFERENCES BETWEEN VERTEBRATE EMBRYOS
CONTRADICT THE PREDICTIONS OF COMMON ANCESTRY.

Another area where evolutionary biologists claim powerful evidence for common ancestry is the patterns of development of vertebrate embryos. Biology textbooks typically portray the embryos of different groups of vertebrate as starting off development in a highly similar fashion, reflecting their common ancestry.¹²⁹ However, such claims overstate the degree of similarity between the early stages of vertebrate embryos.

Biologists who investigate these questions have found considerable variability among vertebrate embryos from their earliest stages onward, contradicting what we are told to expect from common ancestry.¹³⁰ As a paper in *Nature* stated, “Counter to the expectations of early embryonic conservation, many studies have shown that there is often remarkable divergence between related species both early and late in development.”¹³¹ Or, as another article in *Trends in Ecology and Evolution* stated, “despite repeated assertions of the uniformity of early embryos within members of a phylum, development *before* the phylotypic stage is very varied.”¹³²

But most embryologists who acknowledge that vertebrate embryos start development differently will still claim embryos pass through a highly similar stage midway through development, called the “phylotypic” or “pharyngula” stage. These theorists propose an “hourglass model” of development, where it is claimed that similarities between embryos during this midpoint stage provide evidence for common ancestry. One critical biologist explains how this concept is viewed: “It is almost as though the phylotypic stage is regarded as a biological concept for which no proof is needed.”¹³³

But when biologists have looked for evidence supporting the existence of a phylotypic or pharyngula stage, they found the data points in the opposite direction. One comprehensive study in *Anatomy and Embryology* surveyed the characteristics of many vertebrates during this purportedly similar stage, and found that embryos show differences in major traits, including:

- body size,
- body plan,
- growth patterns, and
- timing of development.¹³⁴

The researchers conclude that the evidence is “[c]ontrary to the evolutionary hourglass model” and “difficult to reconcile” with the existence of a pharyngula stage.¹³⁵ Likewise, a paper in *Proceedings of the Royal Society of London* found the data was “counter to the predictions of the [phylotypic stage]: phenotypic variation between species was highest in the middle of the developmental sequence.” It concluded that a “surprising degree of developmental character independence argues against the existence of a phylotypic stage in vertebrates.”¹³⁶

While vertebrate development shows wide variation, evolutionary embryologists seek to force-fit evolutionary interpretations to the data. When every rule is stymied by exceptions, a better way is to simply let the data speak for itself. A non-evolutionary approach to embryology would more easily acknowledge that differences exist between vertebrate embryos at all stages of development, and that vertebrate embryos show some similarities—but also significant differences—during the purported phylotypic stage.

PROBLEM 9:

NEO-DARWINISM STRUGGLES TO EXPLAIN THE BIOGEOGRAPHICAL DISTRIBUTION OF MANY SPECIES.

Biogeography is the study of the distribution of organisms in time and space both in the present and past on Earth. It is often contended that biogeography strongly supports neo-Darwinian theory. For example, the National Center for Science Education (NCSE), a pro-Darwin advocacy group, claims that “consistency between biogeographic and evolutionary patterns provides important evidence about the continuity of the processes driving the evolution and diversification of all life,” and “[t]his continuity is what would be expected of a pattern of common descent.” However, the NCSE dramatically overstates its case and ignores the many instances where biogeography does not show the sort of “continuity” that would be expected under a pattern of common descent.

Evolutionary explanations of biogeography fail when terrestrial (or freshwater) organisms appear in a location (such as an island or a continent) where there is no standard migratory mechanism for them to have arrived there from some ancestral population. In other words, when we find two populations of organisms, Darwinian evolution claims that if we go back far enough, they must be linked by common descent. But sometimes it’s virtually impossible to explain how these

populations could have arrived at their respective geographical locations on the globe from some ancestral population.

For example, one of the most severe biogeographical puzzles for Darwinian theory is the origin of South American monkeys, called “platyrrhines.” Based upon molecular and morphological evidence, New World platyrrhine monkeys are thought to be descended from African “Old World” or “catarrhine” monkeys. The fossil record shows that monkeys have lived in South America for about the past 30 million years.¹³⁷ But plate tectonic history shows that Africa and South America split off from one another between 100 and 120 million years ago (mya), and that South America was an isolated island continent from about 80 - 3.5 mya.¹³⁸ If South American monkeys split off from African monkeys around 30 mya, proponents of neo-Darwinism must somehow account for how they crossed hundreds, if not thousands, of kilometers of open ocean to end up in South America.

This problem for evolutionary biologists has been recognized by numerous experts. A Harper Collins textbook on human evolution states: “The origin of platyrrhine monkeys puzzled paleontologists for decades. ... When and how did the monkeys get to South America?”¹³⁹ Primatologists John G. Fleagle and Christopher C. Gilbert put it this way in a scientific volume on primate origins:

The most biogeographically challenging aspect of platyrrhine evolution concerns the origin of the entire clade. South America was an island continent throughout most of the Tertiary...and paleontologists have debated for much of this century how and where primates reached South America.¹⁴⁰

Primate specialist Walter Carl Hartwig is similarly blunt: “The platyrrhine origins issue incorporates several different questions. How did platyrrhines get to South America?”¹⁴¹ Such basic, vexing questions certainly don’t lend credence to the NCSE’s claims of “consistency between biogeographic and evolutionary patterns.”

For those unfamiliar with the sort of arguments made by neo-Darwinian biogeographers, responses to these puzzles can be almost too incredible to believe. A Harper Collins textbook explains: “The ‘rafting hypothesis’ argues that monkeys evolved from prosimians once and only once in Africa, and ... made the water-logged trip to South America.”¹⁴² And of course, there can’t be just one seafaring monkey, or the monkey will soon die leaving no offspring. Thus, at least two

monkeys (or perhaps a single pregnant monkey) must have made the rafting voyage.

Fleagle and Gilbert observe that the rafting hypothesis “raises a difficult biogeographical issue” because “South America is separated from Africa by a distance of at least 2600 km, making a phylogenetic and biogeographic link between the primate faunas of the two continents seem very *unlikely*.”¹⁴³ But they are wedded to an evolutionary paradigm, meaning that they are obligated to find such a “link” whether it is likely or not. They argue that in light of “[t]he absence of any anthropoids from North America, combined with the considerable morphological evidence of a South American-African connection with the rodent and primate faunas” that therefore “the rafting hypothesis is the most *likely* scenario for the biogeographic origin of platyrrines.”¹⁴⁴

In other words, the “unlikely” rafting hypothesis is made “likely” only because we know common descent *must* be true.

Indeed, the rafting hypothesis faces serious problems, as mammals like monkeys have high metabolisms and require large amounts of food and water.¹⁴⁵ Fleagle and Gilbert thus admit that “over-water dispersal during primate evolution seems truly amazing for a mammalian order,” and conclude, “[t]he reasons for the prevalence of rafting during the course of primate evolution remain to be explained.”¹⁴⁶ Or, as Hartwig puts it, “The overwhelming evidence for the late Cretaceous-Pliocene isolation of South America renders the mechanical aspect of platyrrhine dispersal virtually irresolvable” for “any late Eocene origins model must invoke a transoceanic crossing mechanism that is implausible (rafting) or suspect ... at best.”¹⁴⁷

And there are deeper problems: monkeys apparently made the journey from Africa to South America, but other smaller African primates never colonized the New World. If it was so easy for monkeys to raft across the proto-Atlantic ocean, why didn't these lower primates also make the voyage? The reason we're given by Fleagle and Gilbert is that there is no reason, and it all comes down to sheer chance. In their own words, rafting is “clearly a chance event” and “[o]ne can only speculate that by a stroke of good luck anthropoids were able to ‘win’ the sweepstakes while lorises and galagos did not.”¹⁴⁸

This is not the only case that appeals to rafting or other speculative mechanisms of “oceanic dispersal” to explain away biogeographical conundrums that challenge neo-Darwinism. Examples include the presence of lizards and large caviomorph rodents in South America,¹⁴⁹

the arrival of bees, lemurs, and other mammals in Madagascar,¹⁵⁰ the appearance of elephant fossils on “many islands,”¹⁵¹ the appearance of freshwater frogs across isolated oceanic island chains,¹⁵² and numerous similar examples.¹⁵³ This problem also exists for extinct species, as a paper in *Annals of Geophysics* notes the “still unresolved problem of disjointed distribution of fossils on the opposite coasts of the Pacific.”¹⁵⁴ A 2005 review in *Trends in Ecology and Evolution* explains the problem:

A classic problem in biogeography is to explain why particular terrestrial and freshwater taxa have geographical distributions that are broken up by oceans. Why are southern beeches (*Nothofagus spp.*) found in Australia, New Zealand, New Guinea and southern South America? Why are there iguanas on the Fiji Islands, whereas all their close relatives are in the New World?¹⁵⁵

After reviewing a number of “unexpected” biogeographical examples that require oceanic dispersal, the review concludes: “these cases reinforce a general message of the great evolutionist [Darwin]: given enough time, many things that seem unlikely can happen.”¹⁵⁶

Thus, neo-Darwinian evolutionists are forced to appeal to “unlikely” or “unexpected” migration of organisms, in some cases requiring the crossing of oceans to account for the biogeographical data. This kind of data may not necessarily absolutely falsify Darwinism, but at the least it challenges the simplistic argument that biogeography supports universal common descent through congruence between migration pathways and evolutionary history. In many cases, the congruence is simply not there.

**PROBLEM 10:
NEO-DARWINISM HAS A LONG HISTORY OF
INACCURATE DARWINIAN PREDICTIONS
ABOUT VESTIGIAL ORGANS AND “JUNK DNA.”**

For decades, evolutionists have claimed that our bodies and genomes are full of useless parts and genetic material—“vestigial” organs—showing life is the result of eons of unguided evolution. During the Scopes trial in 1925, evolutionary biologist Horatio Hackett Newman contended that there are over 180 vestigial organs and structures in the human body, “sufficient to make of a man a veritable walking museum of antiquities.”¹⁵⁷

Over time, however, these predictions of vestigial body parts and useless DNA have not held true. As scientists have learned more and more about the workings of biology, important functions and purpose have been discovered for these so-called vestigial structures. Indeed, in 2008 the journal *New Scientist* reported that, since the days of Professor Newman, the list of vestigial organs “grew, then shrank again” to the point that today “biologists are extremely wary of talking about vestigial organs at all.”¹⁵⁸ Structures that were previously—and incorrectly—considered to be vestigial include:

- *The tonsils*: At one time, they were routinely removed. Now it’s known they serve a purpose in the lymph system to help fight infection.¹⁵⁹
- *The coccyx (tailbone)*: Many evolutionists still claim this is a hold-over from the tails of our supposed primate ancestors,¹⁶⁰ but it’s actually a vital part of our skeleton, used for attaching muscles, tendons, and ligaments that support the bones in our pelvis.
- *The thyroid*: This gland in the neck was once believed to have no purpose, and was ignored or even destroyed by medical doctors operating under false Darwinian assumptions. Now scientists know that it is vital for regulating metabolism.
- *The appendix*: Darwinian scientists have claimed the appendix is a “vestige of our herbivorous ancestry,”¹⁶¹ and over eons of evolution its function in humans has been diminished, or lost. But it’s now known that the appendix performs important functions, such as providing a storehouse for beneficial bacteria, producing white blood cells, and playing important roles during fetal development.¹⁶² In light of this evidence, Duke University immunologist William Parker observed that “Many biology texts today still refer to the appendix as a ‘vestigial organ’” but “it’s time to correct the textbooks.”¹⁶³

Despite the poor track record of claiming organs were vestigial, evolutionary biologists have applied this same kind of thinking to our genomes. Many have postulated that the random nature of mutations would fill our genomes with useless genetic garbage, dubbed “junk

DNA.” This hypothesis was seemingly confirmed when it was discovered that only 2% of the human genome coded for proteins, leaving the other 98% unexplained. Many scientists who serve as spokespersons for evolutionary biology have claimed this evidence provides case-closed evidence for Darwinian evolution:

- Brown University evolutionary biologist Kenneth Miller argues that “the human genome is littered with pseudogenes, gene fragments, ‘orphaned’ genes, ‘junk’ DNA, and so many repeated copies of pointless DNA sequences that it cannot be attributed to anything that resembles intelligent design.”¹⁶⁴
- Richard Dawkins likewise writes that “creationists might spend some earnest time speculating on why the Creator should bother to litter genomes with untranslated pseudogenes and junk tandem repeat DNA.”¹⁶⁵
- In his 2006 book *The Language of God*, Francis Collins claimed that some “45 percent of the human genome” is made up of “genetic flotsam and jetsam.”¹⁶⁶ (Flotsam and jetsam, of course, is useless trash floating in the ocean.) Sounding much like Dawkins, he makes the implications clear: “Unless one is willing to take the position that God has placed [shared functionless repetitive DNA] in these precise positions to confuse and mislead us, the conclusion of a common ancestor for humans and mice is virtually inescapable.”¹⁶⁷

The problem with these arguments isn’t so much theological as it is scientific: Numerous examples of function have been discovered for so-called junk DNA.

Biologist Richard Sternberg surveyed the literature and found extensive evidence of function for repetitive DNA. Writing in the *Annals of the New York Academy of Sciences*, he found that functions for repeats include forming higher-order nuclear structures, centromeres, telomeres, and nucleation centers for DNA methylation. Repetitive DNA was found to be involved in cell proliferation, cellular stress responses; gene translation, and DNA repair.¹⁶⁸ Sternberg concluded that “the selfish [junk] DNA narrative and allied frameworks must join the other ‘icons’ of neo-Darwinian evolutionary theory that, despite

their variance with empirical evidence, nevertheless persist in the literature.”¹⁶⁹

Other research has continued to uncover functions for various types of repetitive DNA, including SINE,¹⁷⁰ LINE,¹⁷¹ and *Alu* elements.¹⁷² One paper even suggested that repetitive *Alu* sequences might be involved in “the development of higher brain function” in humans.¹⁷³ Numerous other functions have been discovered for various types of non-protein-coding DNA, including:

- repairing DNA¹⁷⁴
- assisting in DNA replication¹⁷⁵
- regulating DNA transcription¹⁷⁶
- aiding in folding and maintenance of chromosomes¹⁷⁷
- controlling RNA editing and splicing¹⁷⁸
- helping to fight disease¹⁷⁹
- regulating embryological development¹⁸⁰

Sternberg, along with University of Chicago geneticist James Shapiro, predicted in 2005 in the journal *Cytogenetic and Genome Research* that “one day, we will think of what used to be called ‘junk DNA’ as a critical component of truly ‘expert’ cellular control regimes.”¹⁸¹

The day foreseen by Sternberg and Shapiro may have come sooner than they expected. In September, 2012, the journal *Nature* reported the results of a years-long research project, involving over 400 international scientists studying the functions of non-coding DNA in humans. Called the ENCODE Project, its set of 30 groundbreaking papers reported that the “vast majority” of the genome has function. The lead paper reporting ENCODE’s results stated:

These data enabled us to assign biochemical functions for 80% of the genome, in particular outside of the well-studied protein-coding regions.¹⁸²

Ewan Birney, ENCODE’s lead analysis coordinator commented in *Discover Magazine* that since ENCODE looked at only 147 types of cells, and the human body has a few thousand, “It’s likely that 80 percent will go to 100 percent.”¹⁸³ The same article quoted Tom Gingeras, a senior scientist with ENCODE, noting that, “Almost every nucleotide is associated with a function of some sort or another, and we now know

where they are, what binds to them, what their associations are, and more.”¹⁸⁴ Another *Nature* commentary noted that “80% of the genome contains elements linked to biochemical functions, dispatching the widely held view that the human genome is mostly ‘junk DNA’.”¹⁸⁵ *Discover Magazine* put it this way: “The key point is: It’s not ‘junk’.”¹⁸⁶

While there’s still much we don’t know about the genome, the trendline of the research is clearly pointing in one direction: the more we study the genome, the more we detect function for non-coding DNA. Yet the now-dubious “junk-DNA” paradigm was born and bred inside the evolutionary paradigm based upon the idea that our genome was built through random mutations. Yes, a few rogue biologists dared to seek function for non-coding DNA, but the Darwinian “junk DNA” view of genetics has generally hindered the progress of science, as was admitted by a 2003 article in *Science*:

Although catchy, the term ‘junk DNA’ for many years repelled mainstream researchers from studying noncoding DNA. Who, except a small number of genomic clochards, would like to dig through genomic garbage? However, in science as in normal life, there are some clochards who, at the risk of being ridiculed, explore unpopular territories. Because of them, the view of junk DNA, especially repetitive elements, began to change in the early 1990s. Now, more and more biologists regard repetitive elements as a genomic treasure.¹⁸⁷

Despite widespread Darwinian assumptions to the contrary, the paper concluded that “repetitive elements are not useless junk DNA but rather are important, integral components”¹⁸⁸ of animal genomes. Studies suggest that these long stretches of non-coding DNA between genes “constitute an important layer of genome regulation across a wide spectrum of species.”¹⁸⁹

Like repetitive elements, another kind of “junk” DNA for which function is being discovered is pseudogenes. Pseudogenes are thought to be copies of once-functional genes that have been inactivated through mutations. One paper in *Science Signaling* observes that “pseudogenes have long been dismissed as junk DNA,”¹⁹⁰ but notes:

Recent advances have established that the DNA of a pseudogene, the RNA transcribed from a pseudogene, or the protein translated from a pseudogene can have multiple, diverse functions and that

these functions can affect not only their parental genes but also unrelated genes. Therefore, pseudogenes have emerged as a previously unappreciated class of sophisticated modulators of gene expression, with a multifaceted involvement in the pathogenesis of human cancer.¹⁹¹

Indeed, functions for many pseudogenes have already been discovered;¹⁹² the ENCODE project alone found over 850 pseudogenes that are “transcribed and associated with active chromatin.”¹⁹³ But what exactly are these pseudogenes doing? A 2011 paper in the journal *RNA* again argues they can regulate the expression of genes:

Pseudogenes have long been labeled as ‘junk’ DNA, failed copies of genes that arise during the evolution of genomes. However, recent results are challenging this moniker; indeed, some pseudogenes appear to harbor the potential to regulate their protein-coding cousins.¹⁹⁴

Likewise, a 2012 paper in the journal *RNA Biology* similarly stated that “Pseudogenes were long considered as junk genomic DNA” but “pseudogene regulation is widespread”¹⁹⁵ in complex multicellular organisms. The paper proposed that “[t]he high abundance and conservation of the pseudogenes in a variety of species indicate that selective pressures preserve these genetic elements, and suggest that they may indeed perform important biological functions.”¹⁹⁶

Pseudogenes serve as another good example of how Darwinian biologists have assumed that a type of non-coding DNA they didn’t understand was functionless genetic junk, and thus ignored their functions. Indeed, the aforementioned paper in *RNA Biology* explains that one reason why evolutionists have been so slow to abandon the assumption that pseudogenes are junk is because their functions are difficult to detect. The authors observe that “almost all pseudogenes that exhibit significant biological activity are expressed in specific tissue or cell lines,” meaning only specific tissues or cell lines may use a given pseudogene for some function. Additionally, it’s difficult to detect function for pseudogenes because we have lacked the research tools to understand how they influence gene expression. The paper predicts that “more and more functional pseudogenes will be discovered as novel biological technologies are developed in the future,” and concludes

“The study of functional pseudogenes is just at the beginning.”¹⁹⁷ Indeed, two leading biologists writing in *Annual Review of Genetics* reported that “pseudogenes that have been suitably investigated often exhibit functional roles.”¹⁹⁸

Many evolutionary biologists are wedded to the view that our genomes are full of junk, and resist the interpretation that virtually all DNA has function. Indeed, a 2012 evolution textbook teaches that “Over half of the genome is composed of neither genes, nor vestiges of human genes, nor regulatory regions. Instead, it is made up of parasite-like segments of DNA...”¹⁹⁹ Meanwhile, the evidence continues to point in the opposite direction. While much remains to be learned about the workings of our genome, the research trendline is unambiguous: the more we study non-coding DNA, the more we are finding evidence of widespread function.

BONUS PROBLEM:

HUMANS DISPLAY MANY BEHAVIORAL AND COGNITIVE ABILITIES THAT OFFER NO APPARENT SURVIVAL ADVANTAGE.

In recent years, evolutionary biologists have tried to explain the origin of human moral, intellectual, and religious abilities in terms of Darwinian evolution. Harvard University evolutionary psychologist Marc Hauser has promoted the increasingly common hypothesis that “people are born with a moral grammar wired into their neural circuits by evolution.”²⁰⁰

Humans do appear hard-wired for morality, but were we programmed by unguided evolutionary processes? Natural selection cannot explain extreme acts of human kindness. Regardless of background or beliefs, upon finding strangers trapped inside a burning vehicle, people will risk their own lives to help them escape—with no evolutionary benefit to themselves. For example, evolutionary biologist Jeffrey Schloss explains that Holocaust rescuers took great risks which offered no personal biological benefits:

The rescuer’s family, extended family and friends were all in jeopardy, and they were recognized to be in jeopardy by the rescuer. Moreover, even if the family escaped death, they often experienced deprivation of food, space and social commerce; extreme emotional distress; and forfeiture of the rescuer’s attention.²⁰¹

Francis Collins gives the example of Oskar Schindler, the German businessman who risked his life “to save more than a thousand Jews from the gas chambers.”²⁰² As Collins points out, “That’s the opposite of saving his genes.”²⁰³ Schloss adds other examples of “radically sacrificial” behavior that “reduces reproductive success” and offers no evolutionary benefit, such as voluntary poverty, celibacy, and martyrdom.²⁰⁴

In spite of the claims of evolutionary psychologists, many of humanity’s most impressive charitable, artistic, and intellectual abilities outstrip the basic requirements of natural selection. If life is simply about survival and reproduction, why do humans compose symphonies, investigate quantum mechanics, and build cathedrals?

Natural Academy of Sciences member Philip Skell explained why evolutionary psychology does not adequately predict human behavior:

Darwinian explanations for such things are often too supple: Natural selection makes humans self-centered and aggressive—except when it makes them altruistic and peaceable. Or natural selection produces virile men who eagerly spread their seed—except when it prefers men who are faithful protectors and providers. When an explanation is so supple that it can explain any behavior, it is difficult to test it experimentally, much less use it as a catalyst for scientific discovery.²⁰⁵

Contrary to Darwinism, the evidence indicates that human life isn’t about mere survival and reproduction. But in addition to our moral uniqueness, humans are also distinguished by their use of complex language. As MIT professor and linguist Noam Chomsky observes:

Human language appears to be a unique phenomenon, without significant analogue in the animal world. If this is so, it is quite senseless to raise the problem of explaining the evolution of human language from more primitive systems of communication that appear at lower levels of intellectual capacity. ... There is no reason to suppose that the “gaps” are bridgeable.²⁰⁶

Finally, humans are also the only species that seeks to investigate the natural world through science. In fact, the next time someone tries to break down the differences between humans and apes, remind them

that it's humans who write scientific papers studying apes, not the other way around.

SCIENCE VS. RELIGION?

This chapter has cited dozens of papers from the technical scientific literature and by credible scientists which, taken together, pose strong scientific challenges to modern evolutionary theory. Yet defenders of neo-Darwinism commonly assert that legitimate scientific objections to their viewpoint do not exist, and that the only criticisms which remain are based upon religion. Clearly, this is not true. In fact, the attempt to re-label criticisms of neo-Darwinian evolution as religion is typically a ploy to dismiss scientific criticisms without addressing them.

The balance of this book, of course, raises both religious and scientific arguments supporting the progressive creation view that God created life on earth over the course of millions of years. This viewpoint has both religious and scientific dimensions, and for that reason is different from the strictly scientific approach taken in this chapter.

The fact that some arguments in this book may be based upon religion, in no way changes the fact that there are strong scientific challenges to neo-Darwinian theory. Likewise, the fact that there are important religious dimensions to this debate does not mean that materialists can ignore the scientific weaknesses in their own arguments. Until those scientific problems are addressed, scientists will continue to grow skeptical of evolutionary theory.

1. Eugenie Scott, quoted in Terrence Stutz, "State Board of Education debates evolution curriculum," Dallas Morning News (January 22, 2009), also requested in Ed Stoddard, "Evolution gets added boost in Texas schools," Reuters.com at <http://blogs.reuters.com/faithworld/2009/01/23/evolution-gets-added-boost-in-texas-schools/>
2. Karl W. Giberson, *Saving Darwin: How to be a Christian and Believe in Evolution*, p. 53 (HarperOne, 2008) ("biologists today consider the common ancestry of all life a fact on par with the sphericity of the earth").
3. In any case, it's largely a myth that Western Civilization once believed in a flat earth. See Jeffrey Burton Russell, "The Myth of the Flat Earth," at <http://www.veritas-ucsb.org/library/russell/FlatEarth.html>
4. See Stanley L. Miller, "A Production of Amino Acids under Possible Primitive Earth Conditions," *Science*, 117: 528-529 (May 15, 1953).
5. See Jonathan Wells, *Icons of Evolution: Why Much of What We Teach About Evolution Is Wrong*, (Washington D.C.: Regnery, 2000); Casey Luskin, "Not Making the Grade: An Evaluation of 19 Recent Biology Textbooks and Their Use of Selected Icons of Evolution," Discovery Institute (September 26, 2011), at http://www.evolutionnews.org/DiscoveryInstitute_2011TextbookReview.pdf
6. David W. Deamer, "The First Living Systems: a Bioenergetic Perspective," *Microbiology & Molecular Biology Reviews*, 61:239 (1997).
7. Jon Cohen, "Novel Center Seeks to Add Spark to Origins of Life," *Science*, 270: 1925-1926 (December 22, 1995).
8. Ibid.
9. Antonio C. Lasaga, H. D. Holland, and Michael J. Dwyer, "Primordial Oil Slick," *Science*, 174: 53-55 (October 1, 1971).
10. Kevin Zahnle, Laura Schaefer, and Bruce Fegley, "Earth's Earliest Atmospheres," *Cold Spring Harbor Perspectives in Biology*, 2(10): a004895 (October, 2010) ("Geochemical evidence in Earth's oldest igneous rocks indicates that the redox state of the Earth's mantle has not changed over the past 3.8 Gyr"); Dante Canil, "Vanadian in peridotites, mantle redox and tectonic environments: Archean to present," *Earth and Planetary Science Letters*, 195:75-90 (2002).
11. Dante Canil, "Vanadian in peridotites, mantle redox and tectonic environments: Archean to present," *Earth and Planetary Science Letters*, 195:75-90 (2002) (internal citations removed).
12. National Research Council Space Studies Board, *The Search for Life's Origins* (National Academy Press, 1990).
13. Deborah Kelley, "Is It Time To Throw Out 'Primordial Soup' Theory?," NPR (February 7, 2010).
14. Committee on the Limits of Organic Life in Planetary Systems, Committee on the Origins and Evolution of Life, National Research Council, *The Limits of Organic Life in Planetary Systems*, p. 60 (Washington D.C.: National Academy Press, 2007).
15. Richard Van Noorden, "RNA world easier to make," *Nature* news (May 13, 2009), <http://www.nature.com/news/2009/090513/full/news.2009.471.html>.
16. See Stephen C. Meyer, *Signature in the Cell: DNA and the Evidence for Intelligent Design*, p. 304 (New York: HarperOne, 2009).

17. Jack W. Szostak, David P. Bartel, and P. Luigi Luisi, "Synthesizing Life," *Nature*, 409: 387-390 (January 18, 2001).
18. Michael Polanyi, "Life's Irreducible Structure," *Science*, 160 (3834): 1308-1312 (June 21, 1968).
19. See William A. Dembski, *The Design Inference: Eliminating Chance through Small Probabilities* (Cambridge University Press, 1998).
20. Robert Shapiro, "A Simpler Origin for Life," *Scientific American*, pp. 46-53 (June, 2007).
21. Frank B. Salisbury, "Doubts about the Modern Synthetic Theory of Evolution," *American Biology Teacher*, 33: 335-338 (September, 1971).
22. George M. Whitesides, "Revolutions In Chemistry: Priestley Medalist George M. Whitesides' Address," *Chemical and Engineering News*, 85: 12-17 (March 26, 2007).
23. J.T. Trevors and D.L. Abel, "Chance and necessity do not explain the origin of life," *Cell Biology International*, 28: 729-739 (2004).
24. See Michael Behe, "Is There an 'Edge' to Evolution?" at <http://www.faithandevolution.org/debates/is-there-an-edge-to-evolution.php>.
25. *Ibid.*
26. Michael Lynch, "Evolutionary layering and the limits to cellular perfection," *Proceedings of the U.S. National Academy of Sciences*, www.pnas.org/cgi/doi/10.1073/pnas.1216130109 (2012).
27. Jerry Coyne, "The Great Mutator (Review of *The Edge of Evolution*, by Michael J. Behe)," *The New Republic*, pp. 38-44, 39 (June 18, 2007).
28. Charles Darwin, *Origin of Species* (1859), Chapter 6, available at <http://www.literature.org/authors/darwin-charles/the-origin-of-species/chapter-06.html>.
29. David J. DeRosier, "The turn of the screw: The bacterial flagellar motor," *Cell*, 93: 17-20 (1998).
30. *Ibid.*
31. Mark Pallen and Nicholas Matzke, "From The Origin of Species to the Origin of Bacterial Flagella," *Nature Reviews Microbiology*, 4:788 (2006).
32. These experiments have been done on flagella in *E. coli* and *S. typhimurium*. See Transcript of Testimony of Scott Minnich, pp. 103–112, *Kitzmiller et al. v. Dover Area School Board*, No. 4:04-CV-2688 (M.D. Pa., Nov. 3, 2005). Other experimental studies have identified over 30 proteins necessary to form flagella. See Table 1. in Robert M. Macnab, "Flagella," in *Escheria Coli and Salmonella Typhimurium: Cellular and Molecular Biology Vol 1*, pp. 73-74, Frederick C. Neidhart, John L. Ingraham, K. Brooks Low, Boris Magasanik, Moselio Schaechter, and H. Edwin Umbarger, eds., (Washington D.C.: American Society for Microbiology, 1987).
33. Mark Pallen and Nicholas Matzke, "From The Origin of Species to the Origin of Bacterial Flagella," *Nature Reviews Microbiology*, 4:788 (2006).
34. See "The Closest Look Ever at the Cell's Machines," ScienceDaily.com (January 24, 2006), at <http://www.sciencedaily.com/releases/2006/01/060123121832.htm>
35. Bruce Alberts, "The Cell as a Collection of Protein Machines: Preparing the Next Generation of Molecular Biologists," *Cell*, 92:291 (February 6, 1998).
36. Douglas A. Axe, "Estimating the Prevalence of Protein Sequences Adopting Functional Enzyme Folds," *Journal of Molecular Biology*, 341: 1295-1315 (2004);

- Douglas A. Axe, "Extreme Functional Sensitivity to Conservative Amino Acid Changes on Enzyme Exteriors," *Journal of Molecular Biology*, 301: 585-595 (2000).
37. See Stephen C. Meyer, *Signature in the Cell: DNA and the Evidence for Intelligent Design*, p. 211 (Harper One, 2009).
 38. Michael Behe and David Snoke, "Simulating Evolution by Gene Duplication of Protein Features That Require Multiple Amino Acid Residues," *Protein Science*, 13: 2651-2664 (2004).
 39. Rick Durrett and Deena Schmidt, "Waiting for Two Mutations: With Applications to Regulatory Sequence Evolution and the Limits of Darwinian Evolution," *Genetics*, 180:1501-1509 (2008). For a more detailed discussion of this argument, see Ann Gauger, Douglas Axe, Casey Luskin, *Science and Human Origins* (Discovery Institute Press, 2012).
 40. Ann Gauger and Douglas Axe, "The Evolutionary Accessibility of New Enzyme Functions: A Case Study from the Biotin Pathway," *BIO-Complexity*, 2011 (1): 1-17.
 41. Ann Gauger, Stephanie Ebnet, Pamela F. Fahey, and Ralph Seelke, "Reductive Evolution Can Prevent Populations from Taking Simple Adaptive Paths to High Fitness," *BIO-Complexity*, 2010 (2): 1-9.
 42. Neil A. Campbell and Jane B. Reece, *Biology*, p. 84 (7th ed., 2005).
 43. David S. Goodsell, *The Machinery of Life*, pp. 17, 19 (2nd ed., Springer, 2009).
 44. Lynn Margulis, quoted in Darryl Madden, UMass Scientist to Lead Debate on Evolutionary Theory, Brattleboro (Vt.) Reformer (February 3, 2006).
 45. Lynn Margulis quoted in "Lynn Margulis: Q + A," *Discover Magazine*, p. 68 (April, 2011).
 46. Pierre-Paul Grassé, *Evolution of Living Organisms: Evidence for a New Theory of Transformation* (Academic Press: New York NY, 1977).
 47. See "A Scientific Dissent from Darwinism" at <http://www.dissentfromdarwin.org/>
 48. Joseph W. Thornton and Rob DeSalle, "Gene Family Evolution and Homology: Genomics Meets Phylogenetics," *Annual Review of Genomics and Human Genetics*, 1:41-73 (2000).
 49. Scott Gilbert, Stuart Newman, and Graham Budd quoted in John Whitfield, "Biological theory: Postmodern evolution?" *Nature*, 455: 281-284 (September 17, 2008).
 50. Michael Lynch, "Evolutionary layering and the limits to cellular perfection," *Proceedings of the U.S. National Academy of Sciences*, www.pnas.org/cgi/doi/10.1073/pnas.1216130109 (2012).
 51. Michael Lynch, "The frailty of adaptive hypotheses for the origins of organismal complexity," *Proceedings of the U.S. National Academy of Sciences*, 104: 8597-8604 (May 15, 2007).
 52. Eugene V. Koonin, "Darwinian evolution in the light of genomics," *Nucleic Acids Research* (2009): 1-24, doi:10.1093/nar/gkp089
 53. Ibid.
 54. Michael Lynch, "The frailty of adaptive hypotheses for the origins of organismal complexity," *Proceedings of the U.S. National Academy of Sciences*, 104: 8597-8604 (May 15, 2007).

55. Chase W. Nelson and John C. Sanford, "The effects of low-impact mutations in digital organisms," *Theoretical Biology and Medical Modelling*, 8:9 (2011).
56. Michael Lynch, "The evolution of genetic networks by non-adaptive processes," *Nature Reviews Genetics*, 8:803-813 (October, 2007).
57. Ibid.
58. Michael Lynch, "The frailty of adaptive hypotheses for the origins of organismal complexity," *Proceedings of the U.S. National Academy of Sciences*, 104: 8597–8604 (May 15, 2007).
59. Ann Gauger, "The Frailty of the Darwinian Hypothesis, Part 2," *Evolution News & Views* (July 14, 2009), at http://www.evolutionnews.org/2009/07/the_frailty_of_the_darwinian_h_1022911.html
60. Jerry A. Coyne, *Why Evolution is true*, p. 123 (Viking, 2009).
61. Ibid., p. 13.
62. Ibid., p. 124.
63. Michael Lynch, "The frailty of adaptive hypotheses for the origins of organismal complexity," *Proceedings of the U.S. National Academy of Sciences*, 104: 8597–8604 (May 15, 2007).
64. Ibid.
65. Charles Darwin, *The Origin of Species* (1859), p. 292 (reprint, London: Penguin Group, 1985).
66. Ibid.
67. Ibid.
68. Stephen Jay Gould, "Is a new and general theory of evolution emerging?" *Paleobiology*, 6(1): 119-130 (1980).
69. Charles Darwin, *The Origin of Species* (1859), p. 292 (reprint, London: Penguin Group, 1985).
70. Stephen Jay Gould, "Evolution's erratic pace," *Natural History*, 86(5): 12-16, (May, 1977).
71. M. J. Benton, M. A. Wills, and R. Hitchin, "Quality of the fossil record through time," *Nature*, 403: 534-536 (Feb. 3, 2000).
72. Mike Foote, "Sampling, Taxonomic Description, and Our Evolving Knowledge of Morphological Diversity," *Paleobiology*, 23: 181-206 (Spring, 1997).
73. Niles Eldredge and Ian Tattersall, *The Myths of Human Evolution*, p. 59 (New York: Columbia University Press, 1982).
74. David S. Woodruff, "Evolution: The Paleobiological View," *Science*, 208: 716-717 (May 16, 1980).
75. C.P. Hickman, L.S. Roberts, and F.M. Hickman, *Integrated Principles of Zoology*, p. 866 (Times Mirror/Moseby College Publishing, 1988, 8th ed).
76. R.S.K. Barnes, P. Calow and P.J.W. Olive, *The Invertebrates: A New Synthesis*, pp. 9-10 (3rd ed., Blackwell Sci. Publications, 2001).
77. Robert L. Carroll, "Towards a new evolutionary synthesis," *Trends in Ecology and Evolution*, 15(1):27-32 (2000).
78. Jaume Bagaña and Jordi Garcia-Fernández, "Evo-Devo: the Long and Winding Road," *International Journal of Developmental Biology*, 47:705-713 (2003) (internal citations removed).

79. Kevin J. Peterson, Michael R. Dietrich and Mark A. McPeck, "MicroRNAs and metazoan macroevolution: insights into canalization, complexity, and the Cambrian explosion," *BioEssays*, 31 (7):736-747 (2009).
80. Arthur N. Strahler, *Science and Earth History: The Evolution/Creation Controversy*, pp. 408-409 (New York: Prometheus Books, 1987).
81. Richard M. Bateman, Peter R. Crane, William A. DiMichele, Paul R. Kenrick, Nick P. Rowe, Thomas Speck, and William E. Stein, "Early Evolution of Land Plants: Phylogeny, Physiology, and Ecology of the Primary Terrestrial Radiation," *Annual Review of Ecology and Systematics*, 29: 263-292 (1998).
82. Stefanie De Bodt, Steven Maere, and Yves Van de Peer, "Genome duplication and the origin of angiosperms," *Trends in Ecology and Evolution*, 20:591-597 (2005).
83. Niles Eldredge, *The Monkey Business: A Scientist Looks at Creationism* (New York: Washington Square Press, 1982), 65.
84. See Alan Cooper and Richard Fortey, "Evolutionary Explosions and the Phylogenetic Fuse," *Trends in Ecology and Evolution*, 13 (April, 1998): 151-156; Frank B. Gill, *Ornithology*, 3rd ed. (New York: W.H. Freeman, 2007), 42.
85. Alan Cooper and Richard Fortey, "Evolutionary Explosions and the Phylogenetic Fuse," *Trends in Ecology and Evolution*, 13: 151-156 (April, 1998).
86. J.G.M. Thewissen and Sunil Bajpai, "Whale Origins as a Poster Child for Macroevolution," *BioEssays*, 51: 1037-1049 (December, 2001).
87. Philip Gingerich, "Fossils and the Origin of Whales," ActionBioScience.org (December, 2006), <http://www.actionbioscience.org/evolution/gingerich.html>
88. List provided courtesy of Dr. Richard Sternberg.
89. Alan Feduccia, "'Big bang' for tertiary birds?," *Trends in Ecology and Evolution*, 18: 172-176 (2003).
90. See Walter James ReMine, *The Biotic Message: Evolution Versus Message Theory* (Saint Paul: MN, Saint Paul Science, 1983).
91. Private communication with Richard Sternberg.
92. Ernst Mayr, *What Makes Biology Unique?*, p. 198 (Cambridge University Press, 2004).
93. John Hawks, Keith Hunley, Sang-Hee Lee, and Milford Wolpoff, "Population Bottlenecks and Pleistocene Human Evolution," *Journal of Molecular Biology and Evolution*, 17(1):2-22 (2000).
94. Daniel E. Lieberman, David R. Pilbeam, and Richard W. Wrangham, "The Transition from *Australopithecus* to *Homo*," *Transitions in Prehistory: Essays in Honor of Ofer Bar-Yosef*, p. 1 (John J. Shea and Daniel E. Lieberman eds., Oxbow Books, 2009) (internal citations removed).
95. Ibid.
96. "New study suggests big bang theory of human evolution," (January 10, 2000) at <http://www.umich.edu/~newsinfo/Releases/2000/Jan00/r011000b.html>
97. For a more detailed discussion of the fossil evidence and human origins, see Casey Luskin, "Human Origins and the Fossil Record," pp. 45-83 in *Science and Human Origins* (Discovery Institute Press, 2012).
98. Jeffrey Schwartz, *Sudden Origins: Fossils, Genes, and the Emergence of Species*, p. 3 (Wiley, 1999).
99. Zuckerkandl and Pauling, "Evolutionary Divergence and Convergence in Proteins," 101.

100. Jeffrey H. Schwartz, Bruno Maresca, "Do Molecular Clocks Run at All? A Critique of Molecular Systematics," *Biological Theory*, 1(4):357-371, (2006).
101. *Ibid.*
102. Graham Lawton, "Why Darwin was wrong about the tree of life," *New Scientist* (January 21, 2009).
103. W. Ford Doolittle, "Phylogenetic Classification and the Universal Tree," *Science*, 284:2124-2128 (June 25, 1999).
104. Partly quoting Eric Baptiste, in Lawton, "Why Darwin was wrong about the tree of life," (internal quotations omitted).
105. Carl Woese "The Universal Ancestor," *Proceedings of the National Academy of Sciences USA*, 95:6854-9859 (June, 1998) (emphasis added).
106. Graham Lawton, "Why Darwin was wrong about the tree of life," *New Scientist* (January 21, 2009).
107. Partly quoting Michael Syvanen, in Lawton, "Why Darwin was wrong about the tree of life," (internal quotations omitted).
108. Michael Syvanen, quoted in Lawton, "Why Darwin was wrong about the tree of life."
109. James H. Degnan and Noah A. Rosenberg, "Gene tree discordance, phylogenetic inference and the multispecies coalescent," *Trends in Ecology and Evolution*, 24 (2009): 332-340.
110. Arcady R. Mushegian, James R. Garey, Jason Martin and Leo X. Liu, "Large-Scale Taxonomic Profiling of Eukaryotic Model Organisms: A Comparison of Orthologous Proteins Encoded by the Human, Fly, Nematode, and Yeast Genomes," *Genome Research*, 8 (1998): 590-598.
111. Elie Dolgin, "Rewriting Evolution," *Nature*, 486: 460-462 (June 28, 2012).
112. Liliana M. Dávalos, Andrea L. Cirranello, Jonathan H. Geisler, and Nancy B. Simmons, "Understanding phylogenetic incongruence: lessons from phyllostomid bats," *Biological Reviews of the Cambridge Philosophical Society*, 87:991-1024 (2012).
113. For example, see *BSCS Biology: A Molecular Approach* (Glencoe/McGraw Hill, 2006), 227; Sylvia S. Mader, Jeffrey A. Isaacson, Kimberly G. Lyle-Ippolito, Andrew T. Storfer, *Inquiry Into Life*, 13th ed. (McGraw Hill, 2011), 550.
114. See Michael S. Y. Lee, "Molecular Phylogenies Become Functional," *Trends in Ecology and Evolution*, 14: 177 (1999).
115. W. W. De Jong, "Molecules remodel the mammalian tree," *Trends in Ecology and Evolution*, 13(7), pp. 270-274 (July 7, 1998).
116. Trisha Gura, "Bones, Molecules or Both?," *Nature*, 406 (July 20, 2000): 230-233.
117. Antonis Rokas & Sean B. Carroll, "Bushes in the Tree of Life," *PLoS Biology*, 4(11): 1899-1904 (Nov., 2006) (internal citations and figures omitted).
118. Liliana M. Dávalos, Andrea L. Cirranello, Jonathan H. Geisler, and Nancy B. Simmons, "Understanding phylogenetic incongruence: lessons from phyllostomid bats," *Biological Reviews of the Cambridge Philosophical Society*, 87:991-1024 (2012).
119. Ying Cao, Axel Janke, Peter J. Waddell, Michael Westerman, Osamu Takenaka, Shigenori Murata, Norihiro Okada, Svante Pääbo, Masami Hasegawa, "Conflict Among Individual Mitochondrial Proteins in Resolving the Phylogeny of Eutherian Orders," *Journal of Molecular Evolution*, 47 (1998): 307-322.

120. David P. Mindell, Michael D. Sorenson, and Derek E. Dimcheff, "Multiple independent origins of mitochondrial gene order in birds," *Proceedings of the National Academy of Sciences USA*, 95 (September, 1998): 10693-10697.
121. Frederick M Ausubel, "Are innate immune signaling pathways in plants and animals conserved?," *Nature Immunology*, 6 (10): 973-979 (October, 2005).
122. Ibid.
123. Ying Li, Zhen Liu, Peng Shi, and Jianzhi Zhang, "The hearing gene *Prestin* unites echolocating bats and whales," *Current Biology*, 20(2):R55-R56 (January, 2010) (internal citations removed);
124. Gareth Jones, "Molecular Evolution: Gene Convergence in Echolocating Mammals," *Current Biology*, 20(2):R62-R64 (January, 2010); Yong-Yi Shen, Lu Liang, Gui-Sheng Li, Robert W. Murphy, Ya-Ping Zhang, "Parallel Evolution of Auditory Genes for Echolocation in Bats and Toothed Whales," *PLoS Genetics*, 8 (6): e1002788 (June, 2012).
125. Pascal-Antoine Christin, Daniel M. Weinreich, and Guillaume Besnard, "Causes and evolutionary significance of genetic convergence," *Trends in Genetics*, 26(9):400-405 (2010) (internal citations omitted).
126. See Fazale Rana, *The Cell's Design: How Chemistry Reveals the Creator's Artistry*, pp. 207-214 (Baker Books, 2008).
127. Karl W. Giberson, *Saving Darwin: How to be a Christian and Believe in Evolution*, p. 53 (HarperOne, 2008).
128. Michael Syvanen, "Evolutionary Implications of Horizontal Gene Transfer," *Annual Review of Genetics*, 46:339-356 (2012).
129. For example, see Colleen Belk and Virginia Borden Maier, *Biology: Science for Life*, p. 234 (Benjamin Cummings, 2010) ("Similarity among chordate embryos. These diverse organisms appear very similar in the first stages of development (shown in the top row), evidence that they share a common ancestor that developed along the same pathway"); Neil. A. Campbell and Jane B. Reece, *Biology*, p. 449 (Benjamin Cummings, 7th ed., 2005) ("Anatomical similarities in vertebrate embryos. At some stage in their embryonic development, all vertebrates have a tail located posterior to the anus, as well as pharyngeal (throat) pouches. Descent from a common ancestor can explain such similarities"); Holt Science & Technology, *Life Science*, p. 183 (Holt, Rinehart, and Winston, 2001) ("Early in development, the human embryos and the embryos of all other vertebrates are similar. These early similarities are evidence that all vertebrates share a common ancestor. ... They embryos of different vertebrates are very similar during the earliest stages of development").
130. For example, one paper states "Recent workers have shown that early development can vary quite extensively, even within closely related species, such as sea urchins, amphibians, and vertebrates in general. By early development, I refer to those stages from fertilization through neurulation (gastrulation for such taxa as sea urchins, which do not undergo neurulation). Elinson (1987) has shown how such early stages as initial cleavages and gastrula can vary quite extensively across vertebrates." Andres Collazo, "Developmental Variation, Homology, and the Pharyngula Stage," *Systematic Biology*, 49 (2000): 3 (internal citations omitted). Another paper states, "According to recent models, not only is the putative conserved stage followed by divergence, but it is preceded by

- variation at earlier stages, including gastrulation and neurulation. This is seen for example in squamata, where variations in patterns of gastrulation and neurulation may be followed by a rather similar somite stage. Thus the relationship between evolution and development has come to be modelled as an 'evolutionary hourglass.'" Michael K. Richardson *et al.*, "There is no highly conserved embryonic stage in the vertebrates: implications for current theories of evolution and development," *Anatomy and Embryology*, 196:91-106 (1997) (internal citations omitted).
131. Kalinka *et al.*, "Gene expression divergence recapitulates the developmental hourglass model," *Nature*, 468:811 (December 9, 2010) (internal citations removed).
132. Brian K. Hall, "Phylogenic stage or phantom: is there a highly conserved embryonic stage in vertebrates?," *Trends in Ecology and Evolution*, 12(12): 461-463 (December, 1997).
133. Michael K. Richardson *et al.*, "There is no highly conserved embryonic stage in the vertebrates: implications for current theories of evolution and development," *Anatomy and Embryology*, 196:91-106 (1997).
134. Michael K. Richardson *et al.*, "There is no highly conserved embryonic stage in the vertebrates: implications for current theories of evolution and development," *Anatomy and Embryology*, 196:91-106 (1997). See also Steven Poe and Marvalee H. Wake, "Quantitative Tests of General Models for the Evolution of Development," *The American Naturalist*, 164 (September, 2004): 415-422; Michael K. Richardson, "Heterochrony and the Phylogenic Period," *Developmental Biology*, 172 (1995): 412-421; Olaf R. P. Bininda-Emonds, Jonathan E. Jeffery, and Michael K. Richardson, "Inverting the hourglass: quantitative evidence against the phylogenic stage in vertebrate development," *Proceedings of the Royal Society of London, B*, 270 (2003): 341-346;
135. Michael K. Richardson *et al.*, "There is no highly conserved embryonic stage in the vertebrates: implications for current theories of evolution and development," *Anatomy and Embryology*, 196:91-106 (1997).
136. Olaf R. P. Bininda-Emonds, Jonathan E. Jeffery, and Michael K. Richardson, "Inverting the hourglass: quantitative evidence against the phylogenic stage in vertebrate development," *Proceedings of the Royal Society of London, B*, 270:341-346 (2003) (emphases added). See also Steven Poe and Marvalee H. Wake, "Quantitative Tests of General Models for the Evolution of Development," *The American Naturalist*, 164 (3):415-422 (September 2004).
137. Alfred L. Rosenberger and Walter Carl Hartwig, "New World Monkeys," *Encyclopedia of Life Sciences* (Nature Publishing Group, 2001).
138. Carlos G. Schrager and Claudia A. M. Russo, "Timing the origin of New World monkeys," *Molecular Biology and Evolution*, 20(10):1620--1625 (2003); John J. Flynn and A.R. Wyss, "Recent advances in South American mammalian paleontology," *Trends in Ecology and Evolution*, 13(11):449-454 (November, 1998); C. Barry Cox & Peter D. Moore, *Biogeography: An Ecological and Evolutionary Approach*, p. 185 (Blackwell Science, 1993).
139. Adrienne L. Zihlman, *The Human Evolution Coloring Book*, pp. 4-11 (Harper Collins, 2000).

140. John G. Fleagle and Christopher C. Gilbert, "The Biogeography of Primate Evolution: The Role of Plate Tectonics, Climate and Chance," in *Primate Biogeography: Progress and Prospects*, pp. 393-394 (Shawn M. Lehman and John G. Fleagle, eds., Springer, 2006) (emphasis added).
141. Walter Carl Hartwig, "Patterns, Puzzles and Perspectives on Platyrrhine Origins," in *Integrative Paths to the Past: Paleoanthropological Advances in Honor of F. Clark Howell*, p. 69 (Edited by Robert S. Corruccini and Russell L. Ciochon, Prentice Hall, 1994).
142. Adrienne L. Zihlman, *The Human Evolution Coloring Book*, pp. 4-11 (Harper Collins, 2000).
143. John G. Fleagle and Christopher C. Gilbert, "The Biogeography of Primate Evolution: The Role of Plate Tectonics, Climate and Chance," in *Primate Biogeography: Progress and Prospects*, p. 394 (Shawn M. Lehman and John G. Fleagle, eds., Springer, 2006) (emphasis added).
144. *Ibid.* at 394-395 (emphasis added).
145. *Ibid.* at 404.
146. *Ibid.* at 403-404.
147. Walter Carl Hartwig, "Patterns, Puzzles and Perspectives on Platyrrhine Origins," in *Integrative Paths to the Past: Paleoanthropological Advances in Honor of F. Clark Howell*, pp. 76, 84 (Edited by Robert S. Corruccini and Russell L. Ciochon, Prentice Hall, 1994).
148. John G. Fleagle and Christopher C. Gilbert, "The Biogeography of Primate Evolution: The Role of Plate Tectonics, Climate and Chance," in *Primate Biogeography: Progress and Prospects*, p. 395 (Shawn M. Lehman and John G. Fleagle, eds., Springer, 2006).
149. John C. Briggs, *Global Biogeography*, p. 93 (Elsevier Science, 1995).
150. Susan Fuller, Michael Schwarz, and Simon Tierney, "Phylogenetics of the allodapine bee genus *Braunsapis*: historical biogeography and long-range dispersal over water," *Journal of Biogeography*, 32:2135--2144 (2005); Anne D. Yoder, Matt Cartmill, Maryellen Ruvolo, Kathleen Smith, and Rytas Vilgalys, "Ancient single origin of Malagasy primates." *Proceedings of the National Academy of Sciences USA*, 93:5122-- 5126 (May, 1996); Peter M. Kappeler, "Lemur Origins: Rafting by Groups of Hibernators?," *Folia Primatol*, 71:422--425 (2000); Christian Roos, Jürgen Schmitz, and Hans Zischler, "Primate jumping genes elucidate strepsirrhine phylogeny," *Proceedings of the National Academy of Sciences USA*, 101: 10650--10654 (July 20, 2004); Philip D. Rabinowitz & Stephen Woods, "The Africa--Madagascar connection and mammalian migrations," *Journal of African Earth Sciences*, 44:270--276 (2006); Anne D. Yoder, Melissa M. Burns, Sarah Zehr, Thomas Delefosse, Geraldine Veron, Steven M. Goodman, & John J. Flynn, "Single origin of Malagasy Carnivora from an African ancestor," *Nature*, 421:734-777 (February 13, 2003).
151. Richard John Huggett, *Fundamentals of Biogeography*, p. 60 (Routledge, 1998).
152. G. John Measey, Miguel Vences, Robert C. Drewes, Ylenia Chiari, Martim Melo, and Bernard Bourles, "Freshwater paths across the ocean: molecular phylogeny of the frog *Ptychadena newtoni* gives insights into amphibian colonization of oceanic islands," *Journal of Biogeography*, 34: 7-20 (2007).

153. Alan de Queiroz, "The resurrection of oceanic dispersal in historical biogeography," *Trends in Ecology and Evolution*, 20(2): 68-73 (February 2005). For a more detailed discussion, see Casey Luskin, "The Constitutionality and Pedagogical Benefits of Teaching Evolution Scientifically," *University of St. Thomas Journal of Law & Public Policy*, VI (1): 204-277 (Fall, 2009).
154. Giancarlo Scalera, "Fossils, frogs, floating islands and expanding Earth in changing-radius cartography – A comment to a discussion on Journal of Biogeography," *Annals of Geophysics*, 50(6):789-798 (December, 2007).
155. Alan de Queiroz, "The resurrection of oceanic dispersal in historical biogeography," *Trends in Ecology and Evolution*, 20(2):68-73 (February 2005).
156. Ibid.
157. Horatio Hackett Newman, quoted in *The World's Most Famous Court Trial: Tennessee Evolution Case*, 2nd ed. (Dayton, TN: Bryan College, 1990), 268. See also Robert Wiedersheim, *The Structure of Man: An Index to His Past History* (London: MacMillan and Co, 1895; reprinted by Kessinger, 2007).
158. Laura Spinney, "Vestigial organs: Remnants of evolution," *NewScientist*, 2656 (May 14, 2008), at <http://www.newscientist.com/article/mg19826562.100-vestigial-organs-remnants-of-evolution.html>.
159. Sylvia S. Mader, *Inquiry into Life*, 10th ed. (McGraw Hill, 2003), 293.
160. Laura Spinney, "The Five things humans no longer need," *NewScientist* (May 19, 2008), at <http://www.newscientist.com/article/dn13927-five-things-humans-no-longer-need.html>.
161. Douglas Theobald, "29+ Evidences for Macroevolution," TalkOrigins.org, at <http://www.talkorigins.org/faqs/comdesc/section2.html>.
162. See Loren G. Martin, "What is the function of the human appendix? Did it once have a purpose that has since been lost?," *Scientific American* (October, 21, 1999), at <http://www.scientificamerican.com/article.cfm?id=what-is-the-function-of-t>.
163. William Parker quoted in Charles Q. Choi, "The Appendix: Useful and in Fact Promising," *LiveScience* (August 24, 2009).
164. Miller, "Life's Grand Design," 24-32. Miller cites "orphaned genes" but these are not normally understood to be functionless genes. Rather, orphan genes are functional genes that have no known homology to any other gene. Such orphan genes provide evidence for intelligent design because there is no plausible source for their information.
165. Richard Dawkins, "The Information Challenge," *The Skeptic*, 18 (December, 1998).
166. Francis Collins, *The Language of God: A Scientist Presents Evidence for Belief* (New York: Free Press, 2006), 136-37.
167. Francis Collins, *The Language of God*, pp. 134-137 Free Press, 2006).
168. Richard Sternberg, "On the Roles of Repetitive DNA Elements in the Context of a Unified Genomic- Epigenetic System," *Annals of the New York Academy of Sciences*, 981 (2002): 154-88.
169. Ibid.
170. Sternberg, "On the Roles of Repetitive DNA Elements in the Context of a Unified Genomic- Epigenetic System," 154-88.
171. Tammy A. Morrish, Nicolas Gilbert, Jeremy S. Myers, Bethaney J. Vincent, Thomas D. Stamato, Guillermo E. Taccioli, Mark A. Batzer, and John V. Moran,

- “DNA repair mediated by endonuclease-independent LINE-1 retrotransposition,” *Nature Genetics*, 31 (June, 2002): 159-65.
172. Galit Lev-Maor, Rotem Sorek, Noam Shomron, and Gil Ast, “The birth of an alternatively spliced exon: 3’ splice-site selection in Alu exons,” *Science*, 300 (May 23, 2003): 1288-91; Wojciech Makalowski, “Not junk after all,” *Science*, 300 (May 23, 2003): 1246-47.
173. Nurit Paz-Yacova, Erez Y. Levanonc, Eviatar Nevod, Yaron Kinare, Alon Harmelin, Jasmine Jacob-Hirscha, Ninette Amariglioa, Eli Eisenbergg, and Gideon Rechavi, “Adenosine-to-inosine RNA editing shapes transcriptome diversity in primates,” *Proceedings of the National Academy of Sciences USA*, 107 (July 6, 2010): 12174-79.
174. Morrish et al., “DNA repair mediated by endonuclease-independent LINE-1 retrotransposition,” 159-65; Annie Tremblay, Maria Jasin, and Pierre Chartrand, “A Double-Strand Break in a Chromosomal LINE Element Can Be Repaired by Gene Conversion with Various Endogenous LINE Elements in Mouse Cells,” *Molecular and Cellular Biology*, 20 (January, 2000): 54-60; Ulf Grawunder, Matthias Wilm, Xiantuo Wu, Peter Kulesza, Thomas E. Wilson, Matthias Mann, and Michael R. Lieber, “Activity of DNA ligase IV stimulated by complex formation with XRCC4 protein in mammalian cells,” *Nature*, 388 (July 31, 1997): 492-95; Thomas E. Wilson, Ulf Grawunder, and Michael R. Lieber, “Yeast DNA ligase IV mediates non-homologous DNA end joining,” *Nature*, 388 (July 31, 1997): 495-98.
175. Richard Sternberg and James A. Shapiro, “How repeated retroelements format genome function,” *Cytogenetic and Genome Research*, 110 (2005): 108-16.
176. Jeffrey S. Han, Suzanne T. Szak, and Jef D. Boeke, “Transcriptional disruption by the L1 retrotransposon and implications for mammalian transcriptomes,” *Nature*, 429 (May 20, 2004): 268-74; Bethany A. Janowski, Kenneth E. Huffman, Jacob C. Schwartz, Rosalyn Ram, Daniel Hardy, David S. Shames, John D. Minna, and David R. Corey, “Inhibiting gene expression at transcription start sites in chromosomal DNA with antigene RNAs,” *Nature Chemical Biology*, 1 (September, 2005): 216-22; J. A. Goodrich, and J. F. Kugel, “Non-coding-RNA regulators of RNA polymerase II transcription,” *Nature Reviews Molecular and Cell Biology*, 7 (August, 2006): 612-16; L.C. Li, S. T. Okino, H. Zhao, H., D. Pookot, R. F. Place, S. Urakami, H. Enokida, and R. Dahiya, “Small dsRNAs induce transcriptional activation in human cells,” *Proceedings of the National Academy of Sciences USA*, 103 (November 14, 2006): 17337-42; A. Pagano, M. Castelnovo, F. Tortelli, R. Ferrari, G. Dieci, and R. Cancedda, “New small nuclear RNA gene-like transcriptional units as sources of regulatory transcripts,” *PLoS Genetics*, 3 (February, 2007): e1; L. N. van de Lagemaat, J. R. Landry, and D. L. Mager, P. Medstrand, “Transposable elements in mammals promote regulatory variation and diversification of genes with specialized functions,” *Trends in Genetics*, 19 (October, 2003): 530-36; S. R. Donnelly, T. E. Hawkins, and S. E. Moss, “A Conserved nuclear element with a role in mammalian gene regulation,” *Human Molecular Genetics*, 8 (1999): 1723-28; C. A. Dunn, P. Medstrand, and D. L. Mager, “An endogenous retroviral long terminal repeat is the dominant promoter for human B1,3-galactosyltransferase 5 in the colon,” *Proceedings of the National Academy of Sciences USA*, 100 (October 28, 2003): 12841-46; B. Burgess-Beusse, C.

- Farrell, M. Gaszner, M. Litt, V. Mutskov, F. Recillas-Targa, M. Simpson, A. West, and G. Felsenfeld, "The insulation of genes from external enhancers and silencing chromatin," *Proceedings of the National Academy of Sciences USA*, 99 (December 10, 2002): 16433-37; P. Medstrand, Josette-Renée Landry, and D. L. Mager, "Long Terminal Repeats Are Used as Alternative Promoters for the Endothelin B Receptor and Apolipoprotein C-I Genes in Humans," *Journal of Biological Chemistry*, 276 (January 19, 2001): 1896-1903; L. Mariño-Ramírez, K.C. Lewis, D. Landsmana, and I.K. Jordan, "Transposable elements donate lineage-specific regulatory sequences to host genomes," *Cytogenetic and Genome Research*, 110 (2005):333-41.
177. S. Henikoff, K. Ahmad, H. and S. Malik "The Centromere Paradox: Stable Inheritance with Rapidly Evolving DNA," *Science*, 293 (August 10, 2001): 1098-1102; C. Bell, A. G. West, and G. Felsenfeld, "Insulators and Boundaries: Versatile Regulatory Elements in the Eukaryotic Genome," *Science*, 291 (January 19, 2001): 447-50; M.-L. Pardue and P.G. DeBaryshe, "Drosophila telomeres: two transposable elements with important roles in chromosomes," *Genetica*, 107 (1999): 189-96; S. Henikoff, "Heterochromatin function in complex genomes," *Biochimica et Biophysica Acta*, 1470 (February, 2000): O1-O8; L. M.Figueiredo, L. H. Freitas-Junior, E. Bottius, Jean-Christophe Olivo-Marin, and A. Scherf, "A central role for *Plasmodium falciparum* subtelomeric regions in spatial positioning and telomere length regulation," *The EMBO Journal*, 21 (2002): 815-24; Mary G. Schueler, Anne W. Higgins, M. Katharine Rudd, Karen Gustashaw, and Huntington F. Willard, "Genomic and Genetic Definition of a Functional Human Centromere," *Science*, 294 (October 5, 2001): 109-15.
178. Ling-Ling Chen, Joshua N. DeCervo, and Gordon G. Carmichael, "*Alu* element-mediated gene silencing," *The EMBO Journal* 27 (2008): 1694-1705; Jerzy Jurka, "Evolutionary impact of human *Alu* repetitive elements," *Current Opinion in Genetics & Development*, 14 (2004): 603-8; G. Lev-Maor *et al.* "The birth of an alternatively spliced exon: 3' splice-site selection in *Alu* exons," 1288-91; E. Kondo-Iida, K. Kobayashi, M. Watanabe, J. Sasaki, T. Kumagai, H. Koide, K. Saito, M. Osawa, Y. Nakamura, and T. Toda, "Novel mutations and genotype-phenotype relationships in 107 families with Fukuyama-type congenital muscular dystrophy (FCMD)," *Human Molecular Genetics*, 8 (1999): 2303-09; John S. Mattick and Igor V. Makunin, "Non-coding RNA," *Human Molecular Genetics*, 15 (2006): R17-R29.
179. M. Mura, P. Murcia, M. Caporale, T. E. Spencer, K. Nagashima, A. Rein, and M. Palmarini, "Late viral interference induced by transdominant Gag of an endogenous retrovirus," *Proceedings of the National Academy of Sciences USA*, 101 (July 27, 2004): 11117-22; M. Kandouz, A. Bier, G. D. Carystinos, M. A Alaoui-Jamali, and G. Batist, "Connexin43 pseudogene is expressed in tumor cells and inhibits growth," *Oncogene*, 23 (2004):4763-70.
180. K. A. Dunlap, M. Palmarini, M. Varela, R. C. Burghardt, K. Hayashi, J. L. Farmer, and T. E. Spencer, "Endogenous retroviruses regulate periimplantation placental growth and differentiation," *Proceedings of the National Academy of Sciences USA*, 103 (September 26, 2006):14390-95; L. Hyslop, M. Stojkovic, L. Armstrong, T. Walter, P. Stojkovic, S. Przyborski, M. Herbert, A. Murdoch, T. Strachan, and M. Lakoa, "Downregulation of NANOG Induces Differentiation of Human

- Embryonic Stem Cells to Extraembryonic Lineages,” *Stem Cells*, 23 (2005): 1035-43; E. Peaston, A. V. Evsikov, J. H. Graber, W. N. de Vries, A. E. Holbrook, D. Solter, and B. B. Knowles, “Retrotransposons Regulate Host Genes in Mouse Oocytes and Preimplantation Embryos,” *Developmental Cell*, 7 (October, 2004): 597-606.
181. Richard Sternberg and James A. Shapiro, “How repeated retroelements format genome function,” *Cytogenetic and Genome Research*, 110 (2005): 108-16.
182. The ENCODE Project Consortium, “An integrated encyclopedia of DNA elements in the human genome,” *Nature*, 489:57-74 (September 6, 2012).
183. Ewan Birney, quoted in Ed Yong, “ENCODE: the rough guide to the human genome,” *Discover Magazine* (September 5, 2012), at <http://blogs.discovermagazine.com/notrocketscience/2012/09/05/encode-the-rough-guide-to-the-human-genome/>
184. Tom Gingeras, quoted in Ed Yong, “ENCODE: the rough guide to the human genome,” *Discover Magazine* (September 5, 2012), at <http://blogs.discovermagazine.com/notrocketscience/2012/09/05/encode-the-rough-guide-to-the-human-genome/>
185. Joseph R. Ecker, “Serving up a genome feast,” *Nature*, 489:52-55 (September 6, 2012).
186. Ed Yong, “ENCODE: the rough guide to the human genome,” *Discover Magazine* (September 5, 2012), at <http://blogs.discovermagazine.com/notrocketscience/2012/09/05/encode-the-rough-guide-to-the-human-genome/>
187. Makalowski, “Not Junk After All,” 1246-47.
188. *Ibid.*
189. David R. Kelley and John L. Rinn, “Transposable elements reveal a stem cell specific class of long noncoding RNAs,” *Genome Biology*, 13:R107 (2012).
190. Laura Polisenio, “Pseudogenes: Newly Discovered Players in Human Cancer,” *Science Signaling*, 5 (242) (September 18, 2012).
191. *Ibid.*
192. See for example D. Zheng and M. B. Gerstein, “The ambiguous boundary between genes and pseudogenes: the dead rise up, or do they?,” *Trends in Genetics*, 23 (May, 2007): 219-24; S. Hirotsune *et al.*, “An expressed pseudogene regulates the messenger-RNA stability of its homologous coding gene,” *Nature*, 423 (May 1, 2003): 91-96; O. H. Tam *et al.*, “Pseudogene-derived small interfering RNAs regulate gene expression in mouse oocytes,” *Nature*, 453 (May 22, 2008): 534-38; D. Pain *et al.*, “Multiple Retropseudogenes from Pluripotent Cell-specific Gene Expression Indicates a Potential Signature for Novel Gene Identification,” *The Journal of Biological Chemistry*, 280 (February 25, 2005):6265-68; J. Zhang *et al.*, “NANOGP8 is a retrogene expressed in cancers,” *FEBS Journal*, 273 (2006): 1723-30.
193. The ENCODE Project Consortium, “An integrated encyclopedia of DNA elements in the human genome,” *Nature*, 489:57-74 (September 6, 2012).
194. Ryan Charles Pink, Kate Wicks, Daniel Paul Caley, Emma Kathleen Punch, Laura Jacobs, and David Paul Francisco Carter, “Pseudogenes: Pseudo-functional or key regulators in health and disease?,” *RNA*, 17 (2011): 792-98.

195. Yan-Zi Wen, Ling-Ling Zheng, Liang-Hu Qu, Francisco J. Ayala and Zhao-Rong Lun, "Pseudogenes are not pseudo any more," *RNA Biology*, 9(1):27-32 (January, 2012).
196. Yan-Zi Wen, Ling-Ling Zheng, Liang-Hu Qu, Francisco J. Ayala and Zhao-Rong Lun, "Pseudogenes are not pseudo any more," *RNA Biology*, 9(1):27-32 (January, 2012).
197. Ibid.
198. Evgeniy S. Balakirev and Francisco J. Ayala, "Pseudogenes, Are They 'Junk' or Functional DNA?," *Annual Review of Genetics*, 37 (2003): 123-51.
199. Carl Zimmer and Douglas Emlen, *Evolution: Making Sense of Life*, p. 132 (Roberts and Company, 2012).
200. Nicholas Wade, "An Evolutionary Theory of Right and Wrong," *The New York Times* (October 31, 2006), accessed April 28, 2012, <http://www.nytimes.com/2006/10/31/health/psychology/31book.html>.
201. Jeffrey P. Schloss, "Evolutionary Accounts of Altruism & the Problem of Goodness by Design," in *Mere Creation: Science, Faith & Intelligent Design*, ed. William A. Dembski (Downers Grove, IL, Intervarsity Press, 1998), 251.
202. Francis Collins quoted in Dan Cray, "God vs. Science," *Time Magazine* (November 5, 2006), accessed April 28, 2012, <http://www.time.com/time/printout/0,8816,1555132,00.html>.
203. Ibid.
204. Jeffrey P. Schloss, "Emerging Accounts of Altruism: 'Love Creation's Final Law?'," in *Altruism and Altruistic Love: Science, Philosophy, & Religion in Dialogue*, eds. Stephen G. Post, Lynn G. Underwood, Jeffrey P. Schloss, and William B. Hurlbut (Oxford: Oxford University Press, 2002), 221.
205. Philip S. Skell, "Why do we invoke Darwin?," *The Scientist*, 19 (August 29, 2005): 10.
206. Noam Chomsky, *Language and Mind*, 3rd ed. (Cambridge: Cambridge University Press, 2006), 59.