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CHAPTER ONE Human Origins

1.3 THE NINETY-EIGHT PERCENT CHIMPANZEE?
Ninety-Eight Percent Similarity for Written Language

Ninety-eight percent similarity amounts to identity for written language. To see this, we present here Hamlet's famous soliloquy as originally written by Shakespeare along with a 98 percent similar variant. This original text is about 1200 characters in length (including spaces and punctuation). Thus, a text at least 98 percent similar to Hamlet's soliloquy will introduce no more than 24 character changes. Even if those changes are entirely random, they do not alter the ability of human readers to decipher it. To see this, look at the following two versions of Hamlet's soliloquy, the original as well as a modified version that introduces 24 random changes (signified by boldface Xs). Except for one or two words that might be compromised, Hamlet's actual soliloquy can be readily recovered from the version with random Xs. Redundancy and context in written language enable us to make sense of a text even when it has been corrupted.

Hamlet's Soliloquy:

To be, or not to be: that is the question:
Whether 'tis nobler in the mind to suffer
The slings and arrows of outrageous fortune,
Or to take arms against a sea of troubles,
And by opposing end them? To die: to sleep;

No more; and by a sleep to say we end
The heart-ache and the thousand natural shocks
That flesh is heir to, 'tis a consummation
Devoutly to be wish'd. To die, to sleep;
To sleep: perchance to dream: ay, there's the rub;
For in that sleep of death what dreams may come
When we have shuffled off this mortal coil,
Must give us pause: there's the respect
That makes calamity of so long life;
For who would bear the whips and scorns of time,
The oppressor's wrong, the proud man's contumely,
The pangs of despised love, the law's delay,
The insolence of office and the spurns
That patient merit of the unworthy takes,
When he himself might his quietus make

With a bare bodkin? who would fardels bear,
To grunt and sweat under a weary life,
But that the dread of something after death,
The undiscover’d country from whose bourn
No traveller returns, puzzles the will
And makes us rather bear those ills we have
Than fly to others that we know not of?
Thus conscience does make cowards of us all;
And thus the native hue of resolution
Is sicklied o’er with the pale cast of thought,
And enterprises of great pith and moment
With this regard their currents turn awry,
And lose the name of action. - Soft you now!
The fair Ophelia! Nymph, in thy orisons
Be all my sins remember’d.

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98 Percent of Hamlet's Soliloquy
(changes marked with bold Xs):
To be, or not to be: that is the question:
Whether 'tis nobler in the mind to suffer
The slings and arrows of outrageous fortune,
Or to take arms against a sea of troXbleX,
And by oppXsing end them? To die: tX sleep;
No more; and by a sXeep to say we end
The heart-ache and the thousand natural shocks
That flesh is heir to, 'tis a consummatioX
Devoutly to be wish'd. To die, to sleep;
To sleep: perchance to dream: ay, there's the rub;
For in that sleep of death what dreams mXyXcome
When wX have shuffled off this mortal coil,
Must give us pause: there's the respect
That makes calamity of so long life;
For who would bear the whips and scorns of time,
The oppresXor's wroXg, the proud man's contumely,
The pangs of despised love, the law's delay,
The insolence of oXfice and the spurns
That patient merit of the unworthy takes,
When he hiXself mighX his quietus make
With a bare bodkin? who would fardels bear,
To grunt Xnd sweat under a weary life,
But that the dread of something after dXatX,
The undiscover'd country from whose bXurn
No traveller returns, puzzles the will
And makes us rather beXr those ills we have
Than fly to others thaX we know not of?
Thus conscience does make cowards of us all;
And thus the native hue of resXlution
Is sicklied o'er with the pale cast of thought,
And enterprises of great pith and moment
With this regard their currents turn awry,
And lose the name of action. – Soft you noX!
The fair Ophelia!XNymph, in thy orisons
Be Xll my sins remember'd.

Genetic Differences Between Humans and Chimpanzees

To see how the genetic differences between humans and chimpanzees are more complex than the claim of 98-percent similarity suggests, consider the publication of the chimpanzee chromosome 22 sequence in Nature.1 Consistent with past

studies, the overall sequence divergence between chimpanzees and humans was found to be

1.44 percent. Nevertheless, the researchers were surprised to find 68,000 places where the sequences of humans and chimpanzees could not

be aligned because the corresponding place was simply missing in one species or the other. These regions are called insertions/deletions, or indels. They represent places where DNA is present or absent in one of the organisms compared. The original DNA annealing experiments by Sibley and Ahlquist could not detect indels and therefore underestimated the true amount of divergence between humans and chimpanzees. Roy Britten, one of the pioneers of the DNA annealing process, argues that the true genetic divergence between humans and chimpanzees is closer to 95 percent once indels are accounted for.2

Not only is there more sequence divergence than expected between human and chimpanzee genomes (due to indels), but divergence affects both the amino-acid sequences of proteins and the ways proteins are regulated. A direct comparison of protein coding regions of 231 genes shared between humans and chimpanzees revealed that 47 differed significantly in amino-acid sequences they produced:

- 15 contain insertions or deletions of amino acids;
- 32 have different translation start or stop signals; many genes made several different RNA transcripts, and these transcripts varied between humans and chimpanzees;
- some transcripts present in humans were lacking altogether in chimpanzees, suggesting that the genes are being utilized differently in the two species.

Overall, the paper reports that 20.3 percent of proteins are substantially different between humans and chimpanzees.3 This shows that the

1The International Chimpanzee Chromosome 22 Consortium, "DNA Sequence and Comparative Analysis of Chimpanzee Chromosome 22," Nature 429 (27 May 2004): 383–388.

2R. Britten, "Divergence between Samples of Chimpanzee and Human DNA Sequences Is 5%, Counting Indels," Proceedings of the National Academy of Sciences 99(21) (15 October 2002): 13633–13635.

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assumption of a simple, one-to-one correspondence between genes and proteins is false and that the seemingly few genetic differences between humans and chimpanzees disproportionately affect protein sequences and regulation.

A study in Science by researchers at the Max Planck Institute for Evolutionary Anthropology illustrates in detail how the genetic differences between humans and chimpanzees result from different uses of the same gene in each organism. The researchers used gene chip technology to measure expression levels of different genes in humans and chimpanzees.4 They found little difference in genes expressed in blood cells and liver cells of the two species, but when brain cells were compared, massive differences were evident. The difference was so great that if humans and chimpanzees shared a common ancestor, humans needed to accumulate 5.5 times the changes that accumulated in chimpanzees over the same time period. In other words, the rate of change would have been

5.5 times faster in humans than in chimpanzees. The study also separated proteins from human and chimpanzee brains using a 2-dimensional gel electrophoresis technique. The researchers were able to separate the proteins on the basis of their size and charge. Two kinds of data can be gathered: qualitative (differences in types of proteins) and quantitative (differences in amounts of proteins). The researchers compared the proteins expressed in two mouse species' brains and found the relative measures were approximately the same: qualitative and quantitative data both showed around a 7-percent difference between the two species. For humans and chimpanzees, the researchers also found around a 7-percent qualitative difference, but the quantitative difference was found to be over four times higher than expected, namely, 31 percent.

This differing pattern of protein quantity reflects the vastly different patterns of gene expression in the neuronal cells of humans versus chimpanzees.

In other words, even though the genes are remarkably similar, they are used in very different ways in each species. Indeed, the researchers found specific gene regulatory sites that differ between humans and chimpanzees, suggesting that genes are expressed differently because they are regulated by different transcription factors.

The different use of genes in humans and chimpanzees suggests that two fundamental types of genetic changes would be required to evolve them from a common primate ancestor:

(1) re-wiring of portions of the gene regulatory network (i.e., changing how genes regulate each other) and (2) modifying how key regulators interact with their targets. In (1) the topology of the network is fundamentally altered whereas in (2) the connections stay the same but the connection strengths are altered in ways that ramify throughout the network.
For example, a key repressor protein might be altered so that it no longer represses a host of downstream targets. As a result, the associated genes are activated (upregulated), which may in turn regulate other genes. The study of chimpanzee chromosome 22 illustrates precisely this point: protein sequences showed significantly more differences than expected, potentially altering their ability to regulate other genes; and regulatory sites themselves also exhibited variations, showing directly that the regulatory network was different.

The full extent to which the regulatory network wiring of humans and chimpanzees differs is not known. Nonetheless, the holistic nature of the gene expression system means that large-scale reworking of the network would require more than the trial-and-error tinkering characteristic of standard evolutionary theory. Rather, it would require multiple coordinated changes to produce the current systems. Such changes strongly suggest intelligent planning.

3Ibid.

4W. Enard, P. Khaitovich, J. Klose, S. Zollner, F. Heissig, P. Giavalisco, K. Nieselt-Struwe, E. Muchmore, A. Varki, R. Ravid,

G. Doxiadis, R. Bontrop, and S. Paabo, “Intra- and Interspecific Variation in Primate Gene Expression Patterns,” Science 296 (12 April 2002): 340-343.
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1.5 THE BENEFITS OF SMALLER BRAINS

The Brains of Miniature Humans

Miniature humans have normal intelligence. In 2004, the remains of at least seven humans of about three feet in stature, who lived about 18,000 years ago, were discovered on the Indonesian island of Flores. Homo floresiensis, as they were called, has a skull that holds 380 cubic centimeters, whereas typical humans have a skull that holds between 1,300 and 1500 cubic centimeters. However, the Flores people seem to have been of normal intelligence; they made delicate stone tools, with which they hunted dwarf elephants. Accounts vary as to why they were so tiny. Some have argued for genetic dwarfing; others have pointed to the fact that, in general, isolated island species are smaller than mainland species.

As a result of deficiencies in growth hormone, some individual humans today do not reach sizes larger than Homo floresiensis. However, these proportionate dwarfs (formerly called "midgets") typically enjoy normal intelligence. Proportionate dwarfs are relatively rare in societies with access to modern medicine because growth hormone treatment enables them to attain average sizes.

1.6 LANGUAGE AND INTELLIGENCE

The Chimpanzee's Neural Endowment

To protect primate apes from habitat loss and exploitation, some scientists have sought to "humanize" them as much as possible. For example, astronomer and popular science commentator Carl Sagan (1934–1996) writes, regarding the attempts to teach language to primate apes, "What sort of culture, what kind of oral tradition would chimpanzees establish after a few hundred or a few thousand years of communal use of a complex gestural language?" He speculated that the chimpanzees would see the pioneer primatologists as gods.5 Indeed, Sagan seriously considered the possibility that the only reason that chimpanzees

do not now have a complex language is that humans "systematically exterminated" intelligent chimpanzees.6

But in the face of such claims, hopes, and accusations, we must raise an obvious question: Do chimpanzees have the neural capacity to process complex thought? In addressing this question, Andrew Newberg et al. note,

A rudimentary version of the parietal lobe is present in our close evolutionary relative, the chimpanzee. While chimps are smart enough to master simple mathematical concepts and develop non-verbal language skills, their brains appear to lack the neural complexity needed to formulate any significant kind of abstract thought, which is the type of thought that leads to the formation of cultures, art, mathematics, technology, and myths.7

In that case, there is a strict limit on what chimpanzees can accomplish, no matter how much coaching they receive from sympathetic humans.

Evolutionary Psycholog y's Abuse of Evidence—
A Double Standard

When evolutionary psychologists look to animal cognition for evidence of evolution, proof only flows in one direction, that is, to confirm evolutionary relationships that are already taken for granted. Suppose, for instance, a chimpanzee does something that a human could do (e.g., letting you know he wants a cracker.) For evolutionary psychologists, that shows chimpanzees are close evolutionary relatives of humans. Now, suppose a parrot can similarly let you know that he wants a cracker? Would this suggest that the tree of life should be reorganized to place parrots closer to humans? Of course not.

Alternatively, consider a task that a chimpanzee cannot do but a human can do. For instance,

5Carl Sagan, The Dragons of Eden: Speculations on the Nature of Human Intelligence (New York: Random House, 1977), 123–24.
6Ibid., 124.
7Andrew Newberg, Eugene D'Aquili, and Vince Rause, Why God Won't Go Away: Brain Science and the Biology of Belief

(New York: Ballantine Books, 2001), 65.

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chimpanzees show little aptitude for determining what humans are thinking from human facial expressions, and that despite chimpanzee faces being structurally similar to human faces. On the other hand, dogs, which reportedly are less intelligent than chimpanzees, are more adept at determining what humans are thinking from their facial expressions, and that despite being faced with learning a facial language that is not at all like theirs.8 Would this suggest that the tree of life should be reorganized to place dogs closer to humans? Of course not.

In both cases no reorganization of the tree of life occurs because the only significant evidence for human–chimpanzee similarity comes from morphology and genome mapping. Even if parrots or dogs were far more intelligent than they are, evolutionists would not rethink their basic classifications of life, on which all claims for common ancestry are based. Cognitive similarities support evolution when they are found but do nothing to undermine it when they are absent. This double standard makes clear that evolutionary psychology is not a scientific theory but rather an ideological project to undermine human uniqueness.

Explaining Human Mathematical Ability— Three Evolutionary Hypotheses

Humans have many unique cognitive abilities apart from language. Evolutionary theorists have proposed three main types of hypotheses for how these abilities might have evolved: the adaptationist hypothesis, the byproduct hypothesis, and the sexual selection hypothesis. Let’s consider these hypotheses in turn with respect to a specific cognitive ability, namely, mathematics.

The adaptationist hypothesis. How did humans acquire their talent for mathematics? According to the adaptationist hypothesis, mathematical ability conferred a selective advantage on our evolutionary ancestors. Those with better mathematical abilities were as a result better able to survive and reproduce. In other words, they were better able to “adapt” to their environments (hence the term “adaptationist hypothesis”). This hypothesis has a certain plausibility when it comes to the acquisition of rudimentary mathematical abilities like simple arithmetic.

For example, if one of our hunter-gatherer ancestors counted five lions earlier in the day but now sees four of them dead (killed by him and his fellow

hunters), a knowledge of basic arithmetic will warn him that one lion is still on the loose. He will thus know to act cautiously, which will translate into a survival and reproductive advantage. But rudimentary mathematical abilities are one thing; developing four-dimensional Riemannian geometries that describe a curved spacetime manifold, as Albert Einstein did, is quite another. It is hardly plausible that abstract mathematics, such as the Einstein Field Equations,

8pGT ab , 9

Gab + .gab = _____
c4

confers any immediate survival and reproductive advantage. Moreover, future survival and reproduction is ruled out because evolution does not "look ahead." So the adaptationist hypothesis breaks down, and other hypotheses are required.

The byproduct hypothesis. According to the byproduct hypothesis, higher cognitive functions like mathe

8See the work of Vilmos Csányi: "Chimpanzees, our closest relatives, have been shown to follow a human's gaze, but they do very poorly in a classic experiment that requires them to extract clues by watching a person. In that test, a researcher hides food in one of several containers out of sight of the animal. Then the chimp is allowed to choose one container after the experimenter indicates the correct choice by various methods, such as staring, nodding, pointing, tapping, or placing a marker. Only with considerable training do chimps and other primates manage to score above chance. Dogs, however, performed marvelously, and even outdoor dogs with no particular master could solve the problem immediately. (The researchers controlled for the scent of the food.)" Colin Woodward, "Clever Canines: Did Domestication Make Dogs Smarter," Chronicle of Higher Education, April 15, 2005, http://chronicle.com/free/v51/i32/32a01201.htm (last accessed June 7, 2006).

9Here Gab is the Einstein tensor, § is the cosmological constant, gab is the metric tensor, c is the speed of light in a vacuum, G is the gravitational constant, and Tab is the energy tensor. This form of the Einstein Field Equations employs abstract index notation, i.e., a and b are indices.

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matics are not evolutionary adaptations at all. Instead, they are unintended byproducts of traits that are adaptive. Spectacular mathematical abilities are thus said to piggyback on adaptive traits. Pascal Boyer offers such an argument. According to him, some rudimentary ability to count and add is adaptive, but the capacity to do higher-level mathematics is a byproduct of this rudimentary ability. The higher-level capacity is not adaptive by itself; rather, it emerges as a free rider on abilities that are adaptive. But how, exactly, does rudimentary quantitative ability turn into the ability to develop curved spacetime Riemannian geometries or mathematical theories of comparable sophistication? Boyer doesn't say.10

This is always the weakness of byproduct hypotheses, namely, bridging the gap between what can be explained in standard evolutionary terms (adaptations) and the unexpected "freebies" (byproducts) that come along for the ride. Some free lunches are just too good to be true. And precisely when they are too good to be true, they require explanation. That's especially true of mathematics: Here we have a human capacity that not only emerges, according to the byproduct hypothesis, from other capacities, but also provides fundamental insights into the structure of the physical universe (mathematics is, after all, the language of physics).11 How could a capacity like that arise as the byproduct of a blind evolutionary process, unguided by any intelligence? It is not a sufficient explanation here simply to say that it could have happened that way. Science does not trade in sheer possibilities. If our mathematical ability is the byproduct of other evolved traits, then the connection with those traits needs to be made explicit. To date, it has not been.

The sexual selection hypothesis. Finally, we turn to the sexual-selection hypothesis. Sexual selection is

Darwin's explanation for how animals acquire traits that have no direct adaptive value. Consider a stag whose antlers are so large that they are more deadweight than defense. Or a peacock whose large colored tail makes it easy prey. How do such structures evolve? According to Darwin, they evolve because they help to attract mates—they are a form of sexual display. Thus, even though these features constitute a disadvantage for survival in the greater environment, the reproductive advantage they provide in attracting mates more than adequately compensates for this disadvantage and provides an evolutionary explanation for the

formation of these features.

Geoffrey Miller has applied Darwin's idea of sexual selection to explain the formation of our higher cognitive functions.12 According to him, extravagant cognitive abilities like those exhibited by mathematical geniuses are essentially a form of sexual display. Once a capacity begins to attract mates, it acts like a positive feedback loop, continually reinforcing itself. In the case of cognitive functions, such a positive feedback loop can run unchecked because there are no environmental constraints to impose limits: unlike stag antlers or peacock tails, which can only get so large before their adaptive disadvantage outweighs their ability to attract mates, higher cognitive functions can essentially increase without limits. This, for Miller, is the origin of our higher cognitive functions, and our talent for mathematics in particular.

The fundamental weakness of these evolutionary hypotheses.

Leaving aside whether mathematical ability really is a form of sexual display (most mathematicians would be surprised to learn as much), there is a fundamental problem with these hypotheses. To be sure, they presuppose that the traits in question evolved, which in itself is problematic. The main

10Boyer makes this argument in Religion Explained: The Evolutionary Origins of Religious Thought (New York: Basic Books, 2001). In attempting to account for higher cognitive functions, Boyer is concerned not just with mathematics but also with art, religion, and ethics. For another byproduct approach to higher cognitive functions, see Steven Mithen, The Prehistory of the Mind: The Cognitive Origins of Art, Religion, and Science (London: Thames & Hudson, 1996). Mithen sees higher-level functions like mathematics as the byproducts of a "cognitive fluidity" that is adaptive in the sense that it facilitates the coordination and communication of various lower-level cognitive modules.

11See especially Mark Steiner, The Applicability of Mathematics as a Philosophical Problem (Cambridge, Mass.: Harvard University Press, 1999).

12See his book The Mating Mind: How Sexual Choice Shaped the Evolution of Human Nature (New York: Doubleday, 2000).

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problem, however, is that none of them provides a detailed, testable model for assessing its validity. If spectacular mathematical ability is adaptive, as the adaptationist hypothesis claims, how do we determine that? What precise evolutionary steps would be needed to achieve that ability? If it is a byproduct of other abilities, as the byproduct hypothesis claims, of which abilities exactly is it a byproduct and how do these other abilities facilitate it? If it is a form of sexual display, as the sexual selection hypothesis claims, how exactly did the ability become a criterion for mate selection?

In short, the main difficulty with all three hypotheses is that they attempt to account for an existing state of affairs without hard evidence of the factors that brought it about, only speculation. In the case of mathematics in particular, that is an especially severe deficit because higher mathematics is not obviously useful when it first emerges. The fact that uses are sometimes found later is, on conventional evolutionary grounds, irrelevant to its emergence. It becomes relevant only if one is justified in thinking that there is purpose in nature.

Intelligent design? Certainly, if evolution is true, then one of these hypotheses or some combination of them is likely to account for our ability to do mathematics. But even if evolution is true, in the absence of a detailed, testable model of how various higher-level cognitive functions emerged, these hypotheses are scientifically sterile. On the other hand, from an intelligent design perspective, mathematics is readily viewed as an inherent feature of intelligence and rationality. Moreover, the fact that the mathematical theorems we prove

mirror the deep structure of physical reality suggests that intelligence is fundamental to nature and not merely an accidental or historical byproduct of blind material forces. The intelligence underlying nature as reflected in mathematics is a theme explored by Eugene Wigner, who referred to the “unreasonable effectiveness” of mathematics in elucidating nature.13

Number Sense in Animals

Many animals have a basic ability to know the difference between more and less, or many and few. Rhesus monkeys and chimpanzees appear to pay more attention to a quantity if it has changed than if it hasn’t. According to M. D. Hauser, captive rhesus monkeys have been taught to understand

ordinal relations from 1 to 9, but only after hundreds of training trials in conditions that are not duplicated in the wild.14 Essentially, after six months of training, some rhesus monkeys were accurate 50 percent of the time in identifying an ascending or descending order from 1 to 9.15 A weakness of this research is the high level of human interference, a point often overlooked in evolutionary literature (though not by Hauser). The monkeys develop this skill under intensive training by humans. It is unlikely that they would do so otherwise, because almost any non-destructive use of the average wild monkey's time would be better and more immediately rewarded in nature. This fact tells against an adaptationist hypothesis in explaining even the most basic arithmetic skills, never mind abstract mathematical skills that typically only find a use after they have emerged apart from any survival goal.

13See Eugene P. Wigner, "The Unreasonable Effectiveness of Mathematics in the Natural Sciences," Communications in Applied Mathematics 13 (1960): 1. For a deeper exploration of this theme, see Steiner, The Applicability of Mathematics as a Philosophical Problem.

14M. D. Hauser, "What Do Animals Think about Numbers?" American Scientist 88 (2) (2000): 144–51.

15Beth Azar, "Monkeying Around with Numbers," Monitor on Psycholog y: Science Watch 31(1) (January 2000): available online at http://www.apa.org/monitor/jan00/sc4.html (last accessed June 7, 2006).

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CHAPTER TWO Genetics and Macroevolution

2.1 DARWIN'S THEORY

The Conserving Power of Natural Selection

Although natural selection's creative role in originating new forms of life is widely contested, its role as a conservative force for maintaining organisms is not in doubt. One particularly interesting example of natural selection's conservative role comes from the work of evolutionist Hermon Bumpus. Bumpus collected the dead remains of English sparrows (Passer domesticus) killed in a severe winter storm. He then collected a sample of survivors so he could compare their characteristics with those of the dead sparrows.

He found that the birds that died tended to be more extreme in their physical characteristics. They were either heavier or lighter or in some other significant way deviated from the norm. Individuals that varied from the norm could survive under moderate conditions, but when the going got tough, only a narrow range of variation was tolerated. This suggests that there might be an optimal body type for this species of bird and that any bird that departs too much from this type will eventually be weeded out.

Bumpus's observations suggest that each species might be associated with an optimal body type that maximizes its function in a particular habitat. Accordingly, the ability of organisms to undergo small-scale adaptive changes due to the Darwinian mechanism of random variation and natural selection could itself be viewed as part of the original design of life. Such a result, though consistent with intelligent design, would not strictly speaking be necessitated by it because intelligent design can accommodate substantial evolutionary change. Nevertheless, apart from intelligent guidance of the evolutionary process, there is no known mechanism for driving large-scale evolutionary change. Biologists acknowledge stabilizing selection, where selection functions as a weeding-out mechanism to maintain the cohesion and stability of species. But that is a far cry from attributing creative power to natural selection.

2.2 MENDEL ON INHERITANCE

The Hardy-Weinberg Law

One of the most important questions for

proponents of both intelligent design and Darwinian theory concerns the behavior of the genetic world over time. Is it a world of stability? Do traits and the genes that produce them remain relatively unchanged in expression and frequency from generation to generation? Or is it a world of constant change where traits may be easily modified, lost, or altered in frequency?

Mendel demonstrated that the units we now call genes, which determine the inheritance of traits, are stable and retained from generation to generation. For example, when Mendel crossed a pea plant having wrinkled seeds with one having round seeds, all of the offspring in the first generation had round seeds. Was the gene for wrinkled seeds then lost or changed? No. In the second generation the trait reappeared in one-fourth of the plants. The gene for wrinkled seeds was present even in the first generation. It was merely suppressed by the dominant gene for the round trait.

One misconception held by many biologists during the late nineteenth and early twentieth century was that traits determined by dominant genes would become increasingly common, while traits determined by recessive genes would become less common. In other words, it was thought that the frequency of a trait (and of the gene that produces it) is constantly changing in a population.

What better evidence could there be for Darwinian change than such shifting in frequencies of genes and corresponding traits? Godfrey Hardy, an English mathematician, and Wilhelm Weinberg, a German physician studied this question independently. Hardy worked out the effects of random mating on the frequencies of individual traits (and thus members of gene pairs) in large populations. Here is what Hardy concluded: “In a word, there is not the slightest foundation for the idea that a dominant character should show a tendency to

spread over a whole population, or that a recessive should tend to die out."1

Hardy showed that there is not only stability in a gene itself but also in the frequency of its occurrence. Weinberg independently came to the same conclusion. The Hardy-Weinberg Law describes mathematically how genes are distributed in a population. Notwithstanding, it provides no evidence for macroevolution. It states that in the absence of selection or other outside forces, random mating keeps the proportions of genes in a population the same from generation to generation. This claim is consistent with Mendel's finding that genes can be shuffled around within a population but that the gene pool itself is remarkably stable. The Hardy-Weinberg Law makes precise this finding of Mendel's. To see how the law works over successive generation, see figure 2.8.

PARENTAL GENERATIONfreq. of M = 0.6 freq. of N = 0.4X1st GENERATION.36MM .48MN .16NNfreq. of M = 0.6 still freq. of N = 0.4 stillX2nd GENERATION.36MM .48MN .16NNfreq. of M = 0.6 still freq. of N = 0.4 still
Figure 2.13 The Hardy-Weinberg Law predicts gene frequencies in subsequent generations given an initial distribution of gene frequencies. Given initial gene frequencies M = 0.6 and N = 0.4, the next two generations of offspring will show the frequencies indicated here. Each subsequent generation shows the same gene frequencies again. In particular, frequencies of the three genotypes (i.e., MM, MN, NN) are stabilized at the same genotype frequencies as in the first generation of offspring.

The evidence of genetics confirms that an organism's genetic instruction set permits limited variation but is essentially stable and resilient. Without this inherent stability, a species would soon cease to exist. Mendel's principles and the Hardy-Weinberg Law provide theoretical support for this genetic stability. By contrast, Darwinism requires the operation of change factors that act on genes to produce new traits. At the same time, however, these change factors must not act too radically lest populations of a given species cease to exist (in which case there could be no origin of species properly so-called). According to Darwinists, genetic material must have some means of changing dramatically enough to result in entirely new organisms, but not so dramatically as to throw the living world into chaos. In section 2.4 we examine the genetic change factors that neo-Darwinists cite as responsible for macroevolution, namely, mutations.

2.4 THE MOLECULAR BASIS FOR GENES AND EVOLUTION

How DNA Codes for Protein

Building on the work of Crick and Watson, scientists determined that three consecutive bases, called a triplet or codon, code for each specific amino acid in a protein molecule. This is how the genetic messages of a species (instructions influencing various features of an organism) are written out. Sections of the genetic message are carried to various work-stations in the cell by a slightly different code (using RNA), which can be thought of as a dialect. The RNA code uses a uracil base (U) in place of the thymine (T) of the DNA code. The RNA code transfers the messages of the DNA with amazing accuracy. Both codes, however, are redundant in the sense that some amino acids are coded for by more than one triplet. The entire genetic code for RNA is given in table 2.1.

The protein-assembling information in DNA is recorded as a sequence of nucleotide triplets known as codons so that, except for start and stop codons, each triplet corresponds to a given amino acid. This information is transferred via a process known as transcription to a shorter sequence of

1Godfrey H. Hardy, “Mendelian Proportions in a Mixed Population,” Science 28 (1908): 49–50.

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AGUAGCSerineAGAAGGArginineTHOSE WITH
CENTRAL UTHOSE WITH
CENTRAL CTHOSE WITH
CENTRAL ATHOSE WITH
CENTRAL GUUUUUC
UUAUUGCUUCUCCUACUGAUUAUCAUAGUUGUCGUAGUGAUGLeucineIsoleucineMethionine or StartValineValine or StartPhenylalanineUCUUCCUCAUCG
SerineCCUCCCCCACCGProlineACUACCACAACGThreonineCGUCGCCGACGGArginineGCUGCCG
CAGCGAlanineGGUGGCGGAGGGGlycineUAUUAC TyrosineUAAUAG StopCAUCAC
HistidineCAACAG GlutamineAAUAACAsparagineAAAAAGLysineGAUGACAspartic acidGAAGAGGlutamic acidUGUUGCCysteineUGAUGGStopTryptophan
Figure 2.14 The genetic code for RNA. Sixty-four possible triplets can be formed from the four letters, or bases. The code's redundancy is seen in that more than one triplet is associated with a given amino acid (several triplets map onto

the same amino acid).

RNA (see figure 2.8). Successive bases along the DNA chain record the genetic information. Taken three at a time, these bases code for the specific sequence of amino acids in protein as it is synthesized. The complete coding information unique to each species is called its genotype.

Section by section, information in DNA is transferred to mRNA (messenger RNA), which is then carried to the protein assembling machinery known as the ribosome. The information carried from DNA to the ribosome via mRNA directs the building of each protein out of just the right combination and sequence of twenty naturally-occurring amino acids. The resulting amino acid sequences in the newly manufactured proteins, in turn, help determine both the orchestrated development and integrated structure of the organism. But where did these sequences come from? Consider figure 2.4. On the "messages side" we see DNA, with its coded information. On the

"products side" we see functional structures (proteins) that are constructed on the basis of DNA messages.

The DNA bases and their helical arrangement occur in all organisms. The information contained in the sequencing of DNA bases, however, varies

MESSAGESDNA(coded information)
PRODUCTSPROTEINS(functional structure)
= Functional Information
Figure 2.15 The mRNA copy is then taken from the nucleus to a ribosome, which is where cells manufacture proteins.

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from organism to organism. This is like the print in a book. Books are pretty much the same in shape and placement of print on the page. Moreover, within a given language convention, books display the same alphabetic characters, and even the same words and phrases. Nevertheless, the sequencing of alphabetic characters (like the sequencing of DNA bases) varies considerably from book to book.

Not all DNA codes for proteins. Although non-coding DNA is not well understood, some regions of DNA thought to be noncoding have turned out to perform functions.2 Taken together, the total DNA for a given organism is known as its genome. A single strand of human DNA is about 50 million times longer than it is wide. A train ten feet wide and of comparable proportions would be about a hundred thousand miles long!

The information in the sequence of base pairs is crucial to understanding an organism. One of the main tasks of the multibillion dollar Human Genome Project is to record this sequence information for human genomes. But even bacteria have astonishingly complex genomes. Many bacteria contain a genome with at least three million base pairs. For the much studied E. coli bacterium the genome consists of 4.7 million base pairs. The Cambridge biochemist Frederick Sanger received his second Nobel prize for determining the base sequences for a bacteria-attacking virus called a bacteriophage. Bacteriophages are much simpler than the bacteria they attack but still immensely complex. Their genetic material consists of between 5,000 and 500,000 nucleotide bases.

Problems with Making Mutation the Basis for Macroevolution

If the proportion of gene sequences that are biologically useful were large, there might be reason to think that point or chromosome mutations could be helpful in achieving the novel biological structures required by macroevolution. But all the evidence points to biologically useful gene sequences being exceedingly rare (see Chapter 7). It’s therefore highly unlikely that point and

chromosome mutations can transform a duplicated gene into a novel functional gene.

Genetic sequence space (i.e., the set of all possible genetic sequences) is functionally sparse (i.e., the

overwhelming majority of genetic sequences don't, and indeed can't, do anything biologically useful or significant). As a consequence, navigating genetic sequence space by undirected means is no help getting from one island of functionality (i.e., one region of biologically useful or significant genetic sequences) to the next. For instance, there is no evidence that conventional evolutionary mechanisms, such as natural selection, can evolve a gene in one region of genetic sequence space with one set of functions into a gene in a far distant region of genetic sequence space with another set of functions (distance here being measured in terms of sequence similarity). In the language of mathematical biology, genetic sequence space gives no indication of being highly interconnected by functional pathways that continuously connect genes with one function to genes with another (which would be required if natural selection, say, were to assist a duplicated gene in transforming into a novel gene).

But there are still more problems with trying to make mutation the basis for macroevolution. For point and chromosome mutations to account for macroevolutionary change, it is not enough for individual genes to be transformed into novel genes that exhibit novel functions. Rather it is required that a whole suite of novel genes be produced through the coordinated transformation of existing genes. This is because for new biological structures to evolve (as required by macroevolution), many genes will have to change.

But it has not been demonstrated that mutations can produce the highly coordinated protein parts required for many biological structures. These are the structures that macroevolution would need to produce. Till now, however, there is no evidence for the coordinated macromutations required for macroevolution. The closest evidence cited in textbooks includes increased immunity to malaria associated with the mutation for sickle-cell anemia

2John W. Bodnar, Jeffrey Killian, Michael Nagle, and Suneil Ramchandani, "Deciphering the Language of the Genome," Journal of Theoretical Biology 189, 1997: 183–193.

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and the resistance to antibiotics by mutant strains of bacteria.

In no such case, however, do we see a coordinated set of mutations that lead to complex novel structures. Sickle-cell anemia, for instance, is induced by a single point mutation that leads to single change in an amino acid (a valine is substituted for a glutamic acid in a hemoglobin molecule). Point mutations like this might enable organisms to stabilize and maintain themselves in the face of severe environmental pressure.

In most instances, however, novel traits induced by such mutations do not continue to benefit the organisms when the environmental pressure is removed. Apart from environmental pressure, such mutations can even be deleterious. For instance, when an individual is heterozygous for sickle-cell anemia, the mutation provides an advantage for surviving the threat of malaria. It does so, however, at the expense of inflicting on homozygous individuals an anemia that impairs the transport of oxygen to the body's cells. Indeed, sickle-cell anemia is often lethal.

Antibiotic Resistance and Mutation

The vast majority of clinically significant cases of bacterial antibiotic resistance have nothing at all to do with mutations. Resistance to penicillin, for example, is typically due to a very complex enzyme known as penicillinase. Nobody knows the origin of penicillinase. Some bacterial cells have it and some don't, though it can be passed from one cell to another on a plasmid (a tiny circular piece of DNA containing the gene for the enzyme). If penicillin is applied to a population of bacteria, those without the enzyme die and those with it survive and multiply. Other examples of non-mutational resistance include complex molecular systems possessed by some bacteria that pump the antibiotic out of a bacterial cell before it can do any damage.

Nevertheless, there are a few examples of antibiotic resistance due to recently-occurring mutations. What's not clear, however, is whether the mutations were present before the antibiotic was applied or arose in response to it. Darwinists insist on the first option, in which mutations arise at random and

not in response to environmental changes. Nonetheless, the second option, which is

Lamarckism, has not been experimentally ruled out. Indeed, mutations that confer antibiotic resistance give every appearance of resulting from a "programmed defense mechanism."

Resistance mutations typically involve alterations in the molecule inside a bacterial cell that a particular antibiotic targets for poisoning. The most famous example is streptomycin, which targets bacterial ribosomes, the molecular complexes responsible for translating RNA into proteins. In rare instances, a mutation can deform the surface of the ribosome so that streptomycin cannot recognize it. The resulting bacterial cell is handicapped in its protein-synthesizing machinery, but it can survive in the presence of streptomycin.

A "fitness cost" is associated with such mutations so that resistant organisms are likely to be eliminated by hardier unmutated ones after the antibiotic is removed. In other words, mutated cells are fitter only while the antibiotic is present. So, even if one concedes that antibiotic resistance in such cases results from a Darwinian process of natural selection acting on random variations, it would become fixed in the population only if streptomycin remained a permanent feature of the environment. Clearly, such cases of antibiotic resistance are too unstable to play any role in macroevolution.

For this reason, antibiotic resistance is not properly ascribed to the interaction of natural selection and genetic mutation. Minor changes in existing species were well-known (and even produced artificially by breeders) for many centuries before Darwin. Darwin took them for granted and proposed a new theory that proposed much more radical changes. After all, he titled his magnum opus On the Origin of Species, not How Existing Species Change Over Time. Had Darwin merely written about how species change, his book would now be gathering dust along with scores of other outdated books on breeding—some of which Darwin himself had read. Yet, Darwin's followers have never observed the origin of a new species by natural selection, with or without mutation.

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To observe the origin of new species, one would expect to find it most readily in bacteria. That's because bacteria can be easily mutated with chemicals and radiation in the laboratory, they take up very little space, and they have very short generation times. Indeed, thousands of mutations, billions of organisms, and thousands of generations can be studied by a single scientist. Yet, bacteriologists have never witnessed the origin of a new species. (Some new plant species have been observed to originate through hybridization, but the combining of two species to make a third is the opposite of the Darwinian process of splitting one species into two.)

For mutations to contribute to evolution, they must benefit the organism. If a mutation harms the organism, it will tend to be eliminated, rather

than favored, by natural selection. The only beneficial mutations that have ever been observed, in bacteria or in any other kind of organism, have been biochemical. That is, they affect only single molecules (such as the target molecule for streptomycin). There are no known beneficial mutations affecting morphology, or shape. All known morphological mutations are either neutral (i.e., they don't have any noticeable effect on the organism's fitness), or they are harmful—and the bigger their effect the more harmful they are. Yet, Darwinian evolution (i.e., the origin of new species, new organs, and new body plans) clearly requires changes in shape. So, there is no evidence for (and indeed a lot of evidence against) a role for mutations as providing raw materials for Darwinian evolution.

Figure 2.16 The conserved pattern of expression of homeotic genes. The colored bands distributed along
the fruit fly chromosome represent homeotic genes. Their lineal sequence on the chromosome results in
the lineal axis, or head-to-tail pattern, of the adult, as reflected by the color code. The same (or similar)
sequences of homeotic genes appear on four chromosomes in the mouse and other mammals, and is
expressed in the same axial pattern in the adults.

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Homeotic Genes

One of the most famous homeotic mutations is Antennapedia (Antp), so called because fruit flies that possess it grow legs on their heads where there would normally be antennae. The DNA sequence of Antp was determined in 1983, and it turned out to include a 180-base pair segment that was about 75 percent identical to segments in several other homeotic genes that were sequenced at about the same time. This segment was dubbed a "homeobox," and it encodes a protein subunit called a "homeodomain" that binds to DNA. Apparently, homeotic genes affect development by regulating the expression of DNA.

For many biologists, this molecular characterization of homeotic genes confirmed the neo-Darwinian proposition that a program encoded in the DNA controls development. Developmental biologist Walter Gehring, co-discoverer of the homeobox, confidently reported in 1987 that "organisms develop according to a precise developmental program that specifies their body plan in great detail and also determines the sequence and timing of the developmental events. This developmental information is stored in the nucleotide sequences of the DNA." 3

The eight principal homeotic genes in fruit flies (one of which is Antp) are differentially expressed along the anterior-posterior axis of the embryo. In other words, the product of one is found in the anterior part of the head, the product of a second is found just behind that, the product of a third behind the second, and so on. Apparently, this differential expression pattern specifies the identities of cells along the body axis, and when the pattern is disrupted by homeotic mutations, cells assume incorrect identities (such as forming into legs rather than antennae). By 1990, it was determined that these eight homeotic genes in the

fruit fly are located on a single chromosome where (remarkably) they are arranged in the same order as their expression pattern in the embryo. Together, the eight genes in Drosophila have been dubbed the homeotic gene complex (HOM-C).4

In the past few years, similar genes have been found in many other kinds of animals. Surprisingly, the similarities often include not only the DNA sequences of individual genes, but also their order on the chromosome and their expression patterns in the embryo. Vertebrates, for

example, have a cluster of genes (called the Hox complex) similar to the HOM-C of Drosophila, arranged in the same order on the chromosome and expressed in the same order along the anterior-posterior axis of the embryo. Although the correspondence is not exact, the similarities are striking. For instance, by interfering with the expression of Hox-6 (the vertebrate counterpart of Antp), the anterior spinal cord in frogs can be morphologically transformed into hindbrain structures,5 and Hox-6 from a mouse can mimic some of the functions of Antp when it is artificially transferred to a fly embryo.6

There are other homeotic genes which are not in the HOM/Hox complexes. One such gene is Pax-6, which primarily affects eye development. Pax-6 made the news when Walter Gehring and his colleagues used it to induce eyes in unusual parts of the fly, such as the antennae and legs. Since it had previously been shown that Pax-6 is similar in flies and mammals (including humans), and that sequences similar to the DNA-binding domain of Pax-6 are present even in worms and squids,7 Gehring and his colleagues concluded that they had found "the master control gene for eye morphogenesis" which is "universal" among multi-cellular animals.8

3Walter J. Gehring, "Homeo Boxes in the Study of Development," Science 236 (1987): 1245.
4D. Duboule, ed., Guidebook to the Homeobox Genes (Oxford: Oxford University Press, 1994).
5C. V. E. Wright, K. W. Y. Cho, J. Hardwicke, R. H. Collins, and E. M. De Robertis, "Interference with Function of a

Homeobox Gene in Xenopus Embryos Produces Malformations of the Anterior Spinal Cord," Cell 59 (1989): 81–93.

6Scott F. Gilbert, Developmental Biology, 6th ed. (Sunderland, Mass.: Sinauer Associates, 2000).

7R. Quiring, U. Walldorf, U. Kloter, and W. J. Gehring, "Homology of the Eyeless Gene of Drosophila to the Small Eye Gene

in Mice and Aniridia in Humans," Science 265 (1994): 785–789.

8G. Halder, P. Callaerts, and W. J. Gehring, "Induction of Ectopic Eyes by Targeted Expression of the Eyeless Gene in Drosophila," Science 267 (1995): 1792.

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Evo-devo proponents were at the time quite excited about these advances. Not only did developmental geneticists succeed in characterizing genes that have dramatic effects in embryogenesis, but they also provided convincing evidence that these genes are present in all multicellular animals. According to biologist Sean Carroll, "One of the most important biological discoveries of the past decade is that arthropods and chordates, and indeed most or all other animals, share a special family of genes, the homeotic (or Hox) genes, which are important for determining body pattern."9

Surely here was decisive empirical confirmation of the neo-Darwinian proposition that "novel morphological forms in animal evolution result from changes in genetically encoded programs of developmental regulation." 10 Alas, no. The very universality of the homeotic genes means that they cannot account for the pattern of diversification required for macroevolution. To explain macroevolution is to explain the vast differences in organisms that started out alike. Those differences are not explained by noting that these organisms preserve the same genes. According to neo-Darwinism, different genes or different ways of regulating the homeotic genes presumably account for the differences. But then those genetic differences that influence development and lead to different organisms need themselves to be accounted for. It's here that evo-devo has no answer.11

What Besides DNA Controls Development?

If DNA does not control development, what does?

Actually, there is good evidence for the involvement of at least two other factors in the developing egg: the cytoskeleton and the membrane. Every animal cell contains a network of microscopic fibers called a cytoskeleton. These fibers include microtubules, which are known to be involved in patterning embryos. For example, one of the gene products involved in head-to-rear patterning of fruit fly embryos is delivered to its proper location by microtubules; if the microtubules are experimentally disrupted, the gene product doesn't reach its proper destination and the embryo is grossly deformed.

Microtubules consist of many identical protein subunits, and each subunit is produced according to a template in the organism's DNA. What matters in development is the organization of microtubule arrays, and the organization of a microtubule array

is not determined by its subunits any more than the layout of a house is determined by its bricks. Instead, microtubule arrays are formed by organelles called centrosomes, which are inherited independently of an organism's DNA. Centrosomes play a central role in development: a frog egg can be induced to develop into a frog merely by injecting a sperm centrosome—no sperm DNA is needed.

Another non-genetic factor involved in development is the membrane pattern of the egg cell. Cell membranes are not merely featureless bags, but highly complex structures. For example, a membrane contains specialized channels that pump molecules in and out of the cell, enabling it to control its interactions with the external

9Sean B. Carroll, "Homeotic Genes and the Evolution of Arthropods and Chordates," Nature 376 (1995): 479.

10E. H. Davidson, K. J. Peterson, and R. A. Cameron, "Origin of Bilaterian Body Plans: Evolution of Developmental Regulatory Mechanisms," Science 270 (1995): 1319.

11Biologists Marc Kirschner and John Gerhart's proposal of "facilitated variation" does not avoid this problem or provide an answer. It requires that living things be biased toward useful variations. This is either a teleological proposal (which they claim it is not) or it is a case of materialistic mystery mongering, attributing to purely material forces an unanalyzed tendency to promote useful variations despite the utter absence of an explicitly identified mechanism capable of facilitating variation. To be sure, they invoke known developmental mechanisms. But these mechanisms don't do what Krischner and Gerhart need them to do: none of their mechanisms in fact accounts for the bias toward useful variations that their proposal demands. See "What's New?", a review by Jonathan Wells of Gerhart and Kirschner's The Plausibility of Life in Books & Culture, September-October 2006.

Note that in proposing their theory, they pit themselves against Darwinism, asserting that Darwin's theory is radically incomplete and suggesting that their own theory provides the necessary completion. So, here we see an admission by utterly mainstream scientists (Kirschner is at Harvard, Gerhart at Cal Berkeley) that Darwinism has deep problems; and then we are given a proposal that is supposed to tie together all the loose ends. People of a skeptical bent might wonder why history waited till now finally to shore up Darwin's theory. They might also wonder why high school and college students continue to be taught neo-Darwinism as settled truth if it requires something like facilitated variation to answer its unresolved questions. See Marc W. Kirschner and John C. Gerhart, The Plausibility of Life: Resolving Darwin's Dilemma (New Haven: Yale, 2005).

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environment. An egg cell membrane also contains "targets" which ensure that molecules synthesized in the nucleus reach their proper destinations in the embryo. The gene product mentioned earlier, which is involved in head-to-rear patterning of fruit fly embryos and which depends on microtubules to deliver it to its proper location, also needs a target molecule to keep it in place after it arrives. The target is already there, embedded in the membrane.

Experiments with single-celled animals show that membrane patterns are determined by pre-existing membranes, not by DNA. Like microtubule subunits, the proteins embedded in a membrane are produced according to templates in the organism's

DNA; but like the form and location of microtubule arrays, the patterns of those embedded proteins are inherited independently of the organism's DNA. So the control exercised by microtubule arrays and membrane patterns over embryonic development is not encoded in DNA sequences.

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

CHAPTER THREE The Fossil Record

3.1 READING THE FOSSIL RECORD
Reasoning with Circumstantial Evidence

In general, theories of biological origins cannot be confirmed by direct empirical tests as can be done for theories that describe repeatable phenomena. This means that theories of origins have to be tested indirectly and therefore in light of circumstantial evidence. To be sure, circumstantial evidence can be airtight (or reasonably so). For example, Smith may be accused of shooting Jones with a .38 revolver because

1. Smith's fingerprints were found on a .38 revolver beside Jones's body;
2. Smith threatened to kill Jones the night before

the murder;
3. Smith had been arrested in the past for stalking Jones; and
4. Smith was seen in the vicinity of Jones at the time of the murder.

Even without eyewitnesses actually catching Smith in the act, the case for murder against him would be compelling.

But not all circumstantial evidence is so convincing. Often circumstantial evidence points in several directions at once. Sometimes, it even appears to point simultaneously in opposite directions. When that happens, contradictory ways of interpreting the evidence need to be carefully weighed. Darwin himself agreed. As he put it: "A fair result can be obtained only by fully stating and balancing the facts and arguments on both sides of each question."1 A case built on circumstantial evidence is not a strict logical proof that rules out all possibility of error. In place of such proof, it aims at plausibility. As a consequence,

1Charles Darwin, On the Origin of Species, facsimile 1st ed. (1859; reprinted Cambridge, Mass.: Harvard University Press, 1964), 2.

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subjective factors often play a role in assessing circumstantial evidence.

Our legal system is sensitive to this fact. For instance, a jury's belief that Smith is guilty may be more a product of subjective factors than the jurors realize.2 That is why Smith is entitled to defense counsel, so that the evidence for Smith's guilt, as well as for his innocence, can both receive fair consideration. Smith's fate will typically depend on how the defense attorney marshals the evidence. Usually, it's not the evidence itself that is in dispute but patterns exhibited by evidence and how one interprets those patterns. The job of the defense attorney, therefore, is to draw attention to salient patterns in the evidence and thereby to sketch a plausible scenario suggesting Smith's innocence. Can the case against Smith be shaken, or even overturned, and replaced with a scenario in which Smith's innocence appears plausible?

A lawyer who has to marshal circumstantial evidence is in the position of a card player dealt a hand of cards: a lot depends on the cards that are dealt, but a lot also depends on how the cards are played. Circumstantial evidence against a client is sometimes so overwhelming that the defense attorney has no chance of getting the client declared innocent. In that case, rather than let the case go to trial, the attorney can do no better than to plea-bargain. So too, cards dealt may be so pitiful that a poker player stands no chance of winning (short of bluffing). In general, then, with circumstantial evidence, a lot depends on the evidence of the case, but a lot also depends on how skillfully the evidence can be arranged into a compelling picture of what actually happened.

3.3 MAJOR FEATURES OF THE FOSSIL RECORD

Sidestepping the Challenge of the Cambrian Explosion

Despite the severe challenge that the Cambrian Explosion poses to conventional evolutionary theory, some Darwinists contend that it is not really a problem for their theory. For instance, Alan Gishlick, Nicholas Matzke, and Wesley Elsberry criticize the view that "the Cambrian Explosion represented an actual sudden origin of higher taxa; that these taxa (such as phyla) are 'real' and not an artifact of human retrospective classification; and that morphological disparity coincides with phyletic categories." According to Gishlick et al.,

paleontologists long ago abandoned these obsolete views in favor of "more useful realms of research," and they cite a 2000 article by Budd and Jensen to support their view.3

Budd and Jensen argue that the notions of "phylum" and "body plan" that most biologists employ can actually be a "hindrance" to understanding how animals evolved. These authors consider the evolutionary relatedness of animals a logical necessity, and they propose a phylogenetic criterion for identifying body plans that incorporates evolution and common ancestry. In making this proposal, they attempt to redefine the problem so that the apparently sudden origin of multiple body plans in the Cambrian Explosion does "not require particular explanation."

Redefining the problem, however, does not make it go away. The fundamental differences in body structure between a clam and a starfish, or between an insect and a frog, cannot be eliminated by a

2See, for instance, the work of forensic psychologist Elizabeth Loftus, especially her book Eyewitness Testimony (Cambridge, Mass.: Harvard University Press, 1996).

3Alan Gishlick, Nicholas Matzke and Wesley R. Elsberry, "Meyer's Hopeless Monster," The Panda's Thumb (August 24, 2004), http://www.pandasthumb.org/pt-archives/000430.html. Graham E. Budd and Soren Jensen, "A Critical Reappraisal of the Fossil Record of the Bilaterian Phyla," Biological Reviews of the Cambridge Philosophical Society 75 (2000): 253–295. For the article that Gishlick et al. are criticizing, see Stephen C. Meyer, "The Origin of Biological Information and the Higher Taxonomic Categories," Proceedings of the Biological Society of Washington 117(2) (2004): 213–239.

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change in terminology. Of course, human classification systems include an element of subjectivity. But there are real differences here that cannot be explained away or papered over by verbal gymnastics.

In denying that "the lack of transitional fossils prior to the Cambrian explosion indicates a lack of ancestors," Gishlick et al. also cite an article by Chen et al.4 This article reports the discovery of microscopic, soft-bodied bilaterians (i.e., worms with bilateral symmetry, or similar left and right sides) 40 to 55 million years before the Cambrian. Since evolutionary theory requires a common ancestor for the animal phyla of the Cambrian Explosion, a small worm-like bilaterian is as good a candidate as any for this role. These fossilized organisms may be the long sought-after common ancestor of the Cambrian animal phyla. And then again they may not. Given the paucity of evidence, there is no way to decide.

Chen et al. provide no evidence that these worms were ancestral to the Cambrian animal phyla. The fossils they describe might simply have been worms that never evolved into anything else. All the evidence really shows is that bilaterians existed 40 to 55 million years before the Cambrian Explosion. There is no evidence how these worms might have transformed into other organisms of the Cambrian. Since pre-Cambrian trace fossils (i.e., fossil burrows presumably made by tiny worms) have been known for years, the fossils described by Chen et al. may simply be direct evidence of a pre-Cambrian worm phylum for which indirect evidence was already available.

Darwin himself conceded in The Origin of Species: "If the theory be true, it is indisputable that before the lowest [Cambrian] stratum was deposited long periods elapsed . . . [in which] the world swarmed

with living creatures." Yet he acknowledged that "several of the main divisions of the animal kingdom suddenly appear in the lowest known fossiliferous rocks"—that is, the Cambrian. Darwin called this a "serious" problem which "at present must remain inexplicable; and may be truly urged as a valid argument against the views here entertained." 5

Many of Darwin's followers have tried to minimize or explain away the fossil evidence of the Cambrian Explosion. One way to do so is to imply, as Gishlick et al. do, that the solution to

Darwin's problem might lie with "near-microscopic, soft-bodied ancestors of the Cambrian animals" that "aren't readily preserved." Yet the article by Chen et al. cited by Gishlick et al. describes small, soft-bodied fossils at the time of or before the Cambrian. If these animals could fossilize, why not the innumerable ancestors needed by Darwin's theory? As paleontologists (and experts on the Cambrian Explosion) James Valentine and Douglas Erwin have written: The "explosion is real; it is too big to be masked by flaws in the fossil record." 6 It is therefore entirely reasonable to conclude that the persistent lack of fossil evidence for common ancestors of the animal phyla suggests that the ancestors never existed.

3.6 PUNCTUATED EQUILIBRIUM
Is Punctuated Equilibrium an Argument from Silence?

Punctuated equilibrium is a widely cited approach for explaining the gaps in the fossil record. According to it, major evolutionary changes in small populations take place rapidly (over several thousands of years) rather than slowly (over several millions of years). Conventional evolutionary theory, by contrast, holds that evolutionary change

4J-Y. Chen, D. J. Bottjer, P. Oliveri, S. Q. Dornbos, F. Gao, S. Ruffins, H. Chi, C.-W. Li, and E. H. Davidson, "Small Bilaterian Fossils from 40 to 55 Million Years Before the Cambrian," Science 305 (2004): 218–222.

5Darwin, Origin of Species, 307–308.

6James W. Valentine and Douglas H. Erwin, "Interpreting Great Developmental Experiments: The Fossil Record," in

R. A. Raff and E. C. Raff, eds., Development as an Evolutionary Process (New York: Alan R. Liss, 1987), 71–107. Or consider the following more recent remark by Valentine and Jablonski: "Unfortunately there is thus no direct fossil evidence of the morphological features of the earliest members of the bilaterian clades; the bodyplans of the phyla come to us ready-made." Quoted from James W. Valentine and David Jablonski, "Morphological and Developmental Macroevolution: A Paleontological Perspective," International Journal of Developmental Biology 47 (2003): 519–520.
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takes place slowly. Yet, according to the theory of punctuated equilibrium, when an organism assumes a new lifestyle by inhabiting a new ecological niche, major adaptations must occur early and rapidly. If they don't, the organism is in danger of being displaced by other, better-adapted competitors. The survivors will be those that most quickly and thoroughly adapt to the demands of the new lifestyle.

After an initial burst of evolutionary change (according to the theory), further refinements are likely to be minor and to occur slowly. As genes become fixed, the population will exhibit less and less variability and will lose its ability to adapt further. Such a population, though too well adapted to be threatened by competitors, is nevertheless a "sitting duck" for extinction should its ecological niche be seriously threatened. Although the reason dinosaurs became extinct is still a matter for debate, some evidence suggests their extinction resulted from environmental changes to which the dinosaurs were unable to adapt, rather than from competition with birds and mammals.

The theory of punctuated equilibrium makes two main assumptions: (1) rapid initial adaptation to a niche, and (2) extinction due to factors other than competition. If true, these assumptions could account for extensive gaps in the fossil record. The earliest population in a lineage of organisms would, according to this theory, adapt quickly to a new lifestyle and therefore evolve quickly. If the changes took place quickly enough, the probability would be low that any of the intermediate forms would fossilize. Instead, the fossil record would be most likely to contain examples from the long history of well-adapted later forms, which, according to the theory, would show little or no evolutionary change. In addition, the theory of punctuated equilibrium makes a third assumption: (3) rapid evolution is most likely to take place in small, isolated populations. The most rapid changes in gene frequencies would be due to the bottleneck and founder effects (see Chapter 4). But for such random factors to operate effectively in a population, the population must be small.

Suppose, therefore, that such a small population breaks free from its ancestral population, becomes geographically isolated, and evolves rapidly in

adapting to a new ecological niche. Later, after the population has thoroughly adapted to its new lifestyle and completed its evolution, it might even

rejoin the ancestral population. In that case, it would no longer be geographically isolated, but it would have become genetically isolated from the ancestral population—there would be two species in place of one. Those two species, adapted as they are to their environmental niches, would be unlikely to evolve further and thus would be likely to fossilize. On the other hand, the evolutionary changes resulting in intermediate forms would only have occurred in small populations. In consequence, few such intermediates would ever have existed and they would be unlikely to have found their way into the fossil record. As we shall see in the next chapter, however, the scenario proposed here is entirely theoretical, and is based mainly on the absence of evidence.

Figure 3.14

Some think the extinction of the dinosaurs occurred because they didn't have the genetic diversity to adapt to environmental changes.

The theory of punctuated equilibrium therefore actually predicts the existence of gaps in the fossil record. One might even be tempted to view these gaps as evidence for the theory. Nevertheless, there are serious difficulties with taking gaps of any sort as evidence. Proponents of intelligent design have long known that gaps in the fossil record cannot by themselves count as evidence for (or against) intelligent design. The problem is that the lack of anything (like gaps in the fossil record) is by itself merely an

argument from silence. Just as gaps cannot support the theory of intelligent design, so too they cannot support the theory of punctuated equilibrium.

3.7 ABRUPT EMERGENCE
Generative Transmutation and the Hopeful Monster

The idea of generative transmutation was for a time vigorously debated within the evolutionary biology literature. In the 1930s, paleontologist Otto Schindewolf argued that the intermediates missing from the fossil record were missing not because they hadn't or couldn't be found, but because they never existed at all. Schindewolf proposed a saltational view of evolution in which all major evolutionary changes occur in large single steps. Thus, for instance, he proposed that the evolutionary transition from reptile to bird occurred when a reptile laid an egg from which a bird then hatched (in contrast to punctuated equilibrium, the saltations here are massive).

In the 1940s, geneticist Richard Goldschmidt championed this view. As a geneticist, however, Goldschmidt wanted a mechanism for the saltations that Schindewolf claimed were responsible for major evolutionary changes. Goldschmidt found his answer in embryological monsters. Occasionally, animals give birth to monsters—offspring with two heads or missing limbs or some other striking deformity or abnormality. Ordinarily, such monsters do not survive to reproduce. Most monsters are therefore "hopeless." But what if a monster had features that actually were beneficial, facilitating survival and reproduction? Such "hopeful monsters" could account for the saltations that Schindewolf thought were necessary to explain the fossil record. Goldschmidt's hopeful monster theory, which is a form of generative transmutation, has little evidence to support it. Indeed, most evolutionists ridiculed the hopeful monster theory at the time it was proposed (though Goldschmidt countered that the evidence for hopeful monsters was no worse than the evidence for gradual evolution).

Obstacles Facing Symbiogenic Reorganization

As a general solution to the problem of explaining abrupt emergence, symbiogenic reorganization faces two serious obstacles:

1. It can at best reorganize existing structures. By itself it cannot originate novel structures. Symbiogenic

reorganization, apart from intelligent design, produces not coherent organisms but kludges or chimeras. Chimeras were mythical hodge-podge creatures, like the triple-bodied monster in Homer's Iliad—"lion before, serpent behind, she-goat in the middle." Indeed, biochemist Radhey Gupta even introduced "chimera" as a technical term to describe organisms that result from symbiogenic reorganization.
2 Even in reorganizing existing structures, symbiogenic reorganization is severely limited unless it can adapt and coordinate existing parts into synthetic wholes. A laptop computer, to use one of Lynn Margulis's examples, is a synthetic whole that combines a television screen and a typewriter keyboard (among other things). But getting these to work together as a synthetic whole requires intelligence to carefully coordinate one structure with another. Margulis regards natural selection as a designer-substitute that can do the necessary adapting and coordinating to make symbiogenic reorganization successful in biology. But instead of showing that natural selection has such causal powers, she merely presupposes that it does. As a consequence, all the design problems raised against natural selection in other chapters of this book (e.g., how to account for the functional information in organisms by natural selection) apply as well to her account of symbiogenic reorganization.

The actual evidence for symbiogenic reorganization falls in quite limited patterns and seems inadequate to account for the full range of biological complexity and diversity that any theory of abrupt emergence requires. Biological hodge-podges, of which there are many, may be accounted for in terms of symbiogenic reorganization without the need for significant intelligent input. But carefully refined structures that are complex, integrated, and universal throughout the living world give no indication of being the result of undirected symbiogenic reorganization (e.g., the ribosome, the genetic code, irreducibly complex biochemical machines, and various ubiquitous proteins).

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4.2 SPECIES AS REPRODUCTIVELY ISOLATED POPULATIONS

Species in the Making?

Can the founder effect and genetic drift lead to the formation of new species? The answer is, maybe. Consider the Hawaiian fruit flies. These flies may have diversified into a number of sibling and near-sibling species mainly by genetic drift and the founder effect. Here is what might have happened. In fruit flies, mating depends on the exact performance of an inborn courtship behavior pattern. All elements of the ritual must be performed with precision in order for mating to follow. This complex series of behaviors is associated with various genes. If a male fruit fly is missing one of these genes, he will fail to perform some element of the ritual. If this male tries to mate with a female from the ancestral population, she will have nothing to do

with him. If, on the other hand, a female also lacks the same gene and is willing to accept him, the two might be able to mate and a new species might become established.

Speciation via such mechanisms may now be happening in some organisms. A certain species of butterfly, Heliconius erato, living in the Amazonian and Central American rain forests, provides an interesting example. These rain forests constitute an immense jungle region that once stretched over an area the size of the European continent. At one time this species of butterfly probably had a continuous distribution over this vast region, but numerous subpopulations now exist with highly varied wing markings. All of the subpopulations are connected to each other by a breeding chain (see figure 4.8). Although adjacent subpopulations

661728710611459Figure 4.8 The distribution of Heliconius erato. The numbered zones indicate the distribution of various subpopulations of Heliconius erato. All adjacent subpopulations can interbreed with one another.

General Notes

can interbreed with each other, subpopulations found at the opposite edges of the rain forest have less interfertility and might, over time, lose their ability to interbreed.

Two populations of squirrels separated by the Grand Canyon might also represent species in the making. The Kaibab squirrel lives to the north of the canyon whereas the Abert squirrel lives to the south. It seems likely that the two descended from one original population. Rarely, however, can squirrels from both populations meet, and thus they tend not to interbreed. Biologists are still not sure whether to classify these two populations of squirrels as separate species or merely as separate varieties. Even so, it seems plausible that in this case a small group of founders acquired gene frequencies that differed from the ancestral population. Thereafter, different environmental conditions on the two sides of the Grand Canyon

could have selected certain genotypes to the exclusion of others. The result was a new variety—which may eventually become a new species—of squirrel.

According to some evolutionary biologists, reproductive isolation resulting in speciation can also occur in the absence of geographic isolation. Consider a jungle. Not all jungle is the same. Areas within a given rain forest can exhibit significant environmental differences due to varying altitude, soil types, water supplies, species of plants, etc. The optimal fit between genetic makeup and environmental conditions will therefore vary considerably depending on which part of the jungle an organism inhabits (see figure 4.9). As a consequence, natural selection might eliminate individuals carrying certain genes so that the subpopulation in an area would become restricted to an environmental niche and thus reproductively isolated from adjacent subpopulations.

Figure 4.9 Subpopulations of many insects inhabit specific trees. Just this difference can bring about reproductive isolation from nearby subpopulations.

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Ornithologist W. Oliver tells the story of the flightless wrens of Stephen's Island near New Zealand. In 1894 a lighthouse was placed on the island. That year the lighthouse keeper's cat brought in several specimens of flightless wrens. No more specimens have ever been found, so the cat that discovered the species may have also exterminated it. Conceivably, the founder effect and genetic drift gave rise to the flightless wrens in the first place, then strong selection in the form of a cat caused the wrens to go extinct.

Finally, consider the case of the Madeira rabbits. These descendants of ordinary domestic European hares were brought to the Madeira Islands by colonists in the late Middle Ages. Yet today the Madeira rabbits are quite different from other European hares in both appearance and behavior, and no longer interbreed with them. Like the flightless Stephen's Island wrens, the Madeira rabbits might be examples of the founder effect.

4.3 ALLEGED INSTANCES OF OBSERVED SPECIATION

Hybridization and Secondary Speciation

Although polyploidy can be physically or chemically induced without hybridization, secondary speciation occurs commonly in plants by hybridization and subsequent polyploidy. For instance, early in the twentieth century, Swedish scientist Arne Müntzing hybridized two plant species. As the hybrid underwent the most familiar form of polyploidy, chromosome doubling, it produced hempnettle, a member of the mint family. Since hempnettle had already been found in nature, Müntzing concluded that hempnettle had formed in nature through the same process of hybridization that he had intentionally employed.1

The only well supported instances of speciation by hybridization are confined to plants. In 2006, Nature published a report of speciation by hybridization (without polyploidy) in Central American butterflies. If corroborated, this would be the first case of observed speciation in animals. But speciation by hybridization (with or without polyploidy) is secondary speciation, not the primary speciation required by Darwin's theory. Darwinian evolution requires that one species split into two that continue to diverge, not that two species combine to make a third with intermediate characteristics. Thus, even if it were discovered that new animal species could form by hybridization,

this would not confirm Darwinism.2

Despite that, the news media tend to exaggerate the significance of secondary speciation, promoting hybridization as a way of originating species even when the scientific evidence for actual speciation is weak. For example, on June 9, 2004, the BBC reported: "Scientists see new species born." 3 But the scientific article on which the BBC based its report was about two existing species of fruit fly that can hybridize, though not with fully fertile offspring. In contrast to the BBC's confident report about the birth of a new species, the scientists' actual conclusions were far more tentative: "Hybrid male sterility does not have a simple basis," and "earlier Drosophila speciation studies probably tell only a partial story." 4

4.5 SPECIATION AND INTELLIGENT DESIGN

The Original Species

If species are the product of intelligent design, would the originally designed species have remained as they were when first formed? Influenced by Plato's concept of unchanging

1Arne Müntzing, "Cytogenetic Investigations on Synthetic Galeopsis tetrahit," Hereditas 16 (1932): 105–154. Justin Ramsey and Douglas W. Schemske, "Neopolyploidy in Flowering Plants," Annual Review of Ecology and Systematics 33 (2002): 589–639.

2Jesus Mavarez et al., "Speciation by Hybridization in Heliconius Butterflies," Nature 441 (2006): 868–871.

3David Whitehouse, "Scientists See New Species Born," BBC News, June 9, 2004, available online at http://news.bbc.co.uk/2/hi/science/nature/3790531.stm (last accessed January 16, 2007). See also the June 2004 press release from the University of Arizona, available online at http://www.newswise.com/p/articles/view/505399 (last accessed January 16, 2007).

4Laura K. Reed and Therese A. Markow, "Early Events in Speciation: Polymorphism for Hybrid Male Sterility in Drosophila," Proceedings of the National Academy of Sciences USA 101 (June 15, 2004): 9009–9012, available online at http://www.pnas.org/cgi/reprint/101/24/9009 (last accessed January 16, 2007).

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essences or types, some eighteenth and nineteenth century proponents of intelligent design believed not only that species were unchangeable but also that they were inextinguishable. Today's proponents of intelligent design know that material mechanisms (such as random variation and natural selection) can change species within limits. Moreover, they recognize that many species have become (and are becoming) extinct. The crucial question, therefore, is not whether species change or go extinct, but the degree to which they can change as a consequence of material mechanisms and, in particular, the limits of such change.

Does this mean that proponents of intelligent design are committed to species being suddenly or specially created from scratch, with all evolutionary change taking place subsequent to such special creations and limited strictly to small-scale, within-species change? No. Intelligent design is compatible with the creationist idea of species being suddenly created from scratch. But it is also compatible with the Darwinian idea of new species arising from old through successive generations of offspring gradually diverging from a parental type, or what Darwin called "descent with modification." What separates intelligent design from materialistic accounts of evolution is not whether organisms evolved, but what was responsible for their evolution—purely material mechanisms or the activity of intelligence.

Proponents of materialistic evolution believe that material mechanisms alone are responsible for evolution (the chief of these being the Darwinian mechanism of random variation and natural selection). Proponents of intelligent design, by contrast, hold that material mechanisms are capable of producing only limited evolutionary change and that any substantial evolutionary change would require input from a designing intelligence. Moreover, design proponents hold that the input of intelligence into biological systems is empirically detectable, that is, detectable by observation through the methods of science. For intelligent design, the crucial question therefore is not whether organisms emerged through an evolutionary process or suddenly from scratch, but whether a designing intelligence made a discernible difference.

This raises another question: How often and at what places did a designing intelligence intervene

in the course of natural history to produce those biological structures that are beyond the power of

material mechanisms? One of the criticisms of intelligent design is that it draws an unreasonable distinction between material mechanisms and designing intelligences, claiming that material mechanisms are fine most of the time but then on rare (or perhaps not so rare) occasions a designing intelligence is required to get over some hump that material mechanisms can't quite manage. This criticism is misconceived. The proper question is not how often or at what places a designing intelligence intervenes, but rather at what points do signs of intelligence first become evident.

To understand the difference, imagine a computer program that outputs alphanumeric characters on a computer screen. The program runs for a long time and throughout that time outputs what look like random characters. Then, abruptly, the output changes and the program outputs the most sublime poetry. Now, at what point did a designing intelligence intervene in the output of the program? Clearly, this question misses the mark because the program is deterministic and simply outputs whatever the program dictates. There may have been no intervention at all that changed the output of the program from random gibberish to sublime poetry. And yet, the point at which the program starts to output sublime poetry is the point at which we recognize that the output is designed and not random. Moreover, it is at that point that we recognize the program itself is designed. But when and where was design introduced into the program? Although that is an interesting question, it is ultimately irrelevant to the more fundamental question whether there was design in the program and its output in the first place.

Intelligent design is not a theory about the frequency or locality at which a designing intelligence intervenes in the material world. It is not an interventionist theory at all. Indeed, intelligent design is perfectly compatible with the idea of "front-loading"—that all design in the world was introduced at the beginning (say at the Big Bang) and then expressed subsequently over the course of natural history, much as a computer program's output becomes evident only when the program is run. This actually is an old idea, and one that Charles Babbage, the inventor of the

digital computer, explored in the 1830s in his Ninth Bridgewater Treatise (twenty years before Darwin's Origin of Species).

Let's be clear, however, that such preprogrammed evolution would be very different from evolution as it is now conceived. Evolution, as currently presented in textbooks, is blind: nonpurposive material mechanisms run the show. Within this materialistic conception of evolution, the origin of any species gives no evidence of actual design because mindless material mechanisms do all the work. Within a preprogrammed conception of evolution, by contrast, the origin of some species and biological structures would give evidence of actual design and demonstrate the inadequacy of material mechanisms to do such design work. Thus, materialist evolution and preprogrammed evolution would have different empirical content and be distinct scientific theories.

Of course, such preprogrammed evolution or front-loaded design is not the only option for the theory of intelligent design. Intelligent design is also compatible with discrete interventions at intermittent times and diverse places. Intelligent design is even compatible with what philosophers call an occasionalist view in which everything that occurs in the world is due to the active intervention of a designing intelligence, though only some of those outcomes may exhibit clear signs of being designed. In that case the distinction between natural causes and intelligent causes would have to

do with how we make sense of the world rather than with how the world actually is (or, as philosophers would say, the distinction would be epistemological rather than ontological).

We may never be able to tell how often or at what places a designing intelligence intervened in the world. But that's okay. What's crucial for the theory of intelligent design is the ability to locate signs of intelligence in the world—and in the biological world in particular—and thus conclude that a designing intelligence played an indispensable role in the formation of some object or the occurrence of some event. That is the start. Often in biology there will be clear times and locations where we can say that design first became evident. But whether that means a designing intelligence actually intervened at those points will require further investigation and may indeed not be answerable. As the computer analogy indicates, the place and

time at which design first becomes evident need have no connection with the place and time at which design was actually introduced.

In the context of evolution, this means that design can be real and discernible in evolutionary change without requiring an explicit "design event," such as a special creation, miracle, or supernatural intervention. At the same time, however, for evolutionary change to exhibit actual design would mean that material mechanisms were inadequate by themselves to produce that change.

CHAPTER FIVE Similar Features

5.4 DARWINISM'S REDEFINITION OF HOMOLOGY

Independent Evidence for Common Ancestry: DNA Sequences

Molecular phylogenies attempt to trace out the evolutionary history of a group of organisms based on molecular sequencing data. To construct molecular phylogenies, biologists compare DNA sequences

(or their RNA or protein products) in different organisms. Since DNA sequences are copied directly from other DNA sequences through the process of replication, molecular phylogeneticists assume that sequence similarities are more likely to indicate an ancestor-descendant relationship than morphological similarities, which are produced by a complex series of events in the embryo rather than inherited directly from parents.

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DNA is like a written language. In general, it's a lot easier to trace the derivation of one piece of writing from another than to determine whether one invention, say, is the source for another. With an invention, whether a machine or artifact, it might simply have been reinvented. Thus, with inventions, it's often not clear whether there is an "evolutionary descent with modification" from some other invention. But with written texts, if the precise phraseology is preserved, it's clear that some form of copying or plagiarism occurred. In such cases, the evidence for an "evolutionary transmission" from one text to the other can seem irresistible. That's why molecular phylogenies initially held such promise for reconstructing evolutionary histories and providing independent evidence for evolution.

That promise, however, has gone unfulfilled. As it turns out, molecular sequence comparisons face as many difficulties as morphological comparisons. First, the meaning of "homology" in molecules is no less problematic than it is in anatomical features. As molecular biologist David Hillis wrote in 1994, "The word homology is now used in molecular biology to describe everything from simple similarity (whatever its cause) to common ancestry (no matter how dissimilar the structures)." Thus, "molecular biologists may have done more to confound the meaning of the term homology than have any other group of scientists." 1

Second, identifying homologous sequences is as difficult as identifying homologous organs. According to Hillis: "Some proponents of molecular techniques have claimed that molecular biology 'solves the problem of homology' . . . [but] the difficulties of assigning homology to molecules parallel many of the difficulties of assigning homology to morphological structures." 2 Here are some of the difficulties: Where do you start and stop comparing two DNA sequences? What do

you do when the sequences don't match up precisely (which they almost never do)? How are such divergences to be explained? Even if the match-up is identical, why should this be taken as independent evidence of evolution? What if, for instance, the match is identical because the organisms share identical functional requirements?

Finally, molecular homology generates at least as many conflicting results as the more traditional approach. "Congruence between molecular phylogenies," wrote British biologists Colin Patterson,

David Williams and Christopher Humphries in 1993, "is as elusive as it is in morphology." 3 As we have seen, comparisons of different genes in the same organisms can lead to different phylogenies. Comparisons of the same genes in the same organisms but performed in different laboratories can lead to different phylogenies. Thus, when molecular phylogenies conflict, the only way to choose among them is to have independent knowledge of common ancestry, and this leads right back into the very circular reasoning that molecular comparisons were supposed to avoid.

Independent Evidence for Evolution: Fossils

Some biologists have argued the best way to determine evolutionary relationships would be to trace the similarities in two or more organisms back through an unbroken chain of fossil organisms to their common ancestor. Unfortunately, comparing fossils is no more straightforward than comparing live specimens. As Sokal and Sneath pointed out in 1963, "Even when fossil evidence is available, this evidence itself must first be interpreted" by comparing similar features. Any attempt to infer evolutionary relationships among fossils based on homology-ascommon-ancestry "soon leads to a tangle of circular arguments from which there is no escape." 4

1David M. Hillis, "Homology in Molecular Biology," 339–368, in B. K. Hall (ed.), Homology: The Hierarchical Basis of Comparative Biology (New York: Academic Press, 1994), 339–341

2Ibid., 359.

3Colin Patterson, David M. Williams, and Christopher J. Humphries, "Congruence Between Molecular and Morphological Phylogenies," Annual Review of Ecology and Systematics 24 (1993): 153–188. See also Colin Patterson, "Homology in classical and Molecular Biology," Molecular Biology and Evolution 5 (1988): 603–625; and Michael S. Y. Lee, "Molecular Phylogenies Become Functional," Trends in Ecology and Evolution 14 (1999): 177–178. On the growing problems with DNA sequence comparisons, see W. Ford Doolittle, "Uprooting the Tree of Life," Scientific American 282 (February 2000): 90–95.

4Sokal and Sneath, Principles of Numerical Taxonomy, 56–57.

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In fact, inferring evolutionary relationships from the fossil record is more difficult than inferring them from live specimens because the record is fragmentary and because fossils do not preserve all relevant features. As biologist Bruce Young wrote in 1993, "If anything, fossils are of less value in establishing homologues since they normally include far fewer characters" than living organisms.5

But, as we saw in Chapter 3, even if the fossil record were complete and even if it preserved all the desired characters, it would not establish that homology is due to common ancestry (much less that it is due to an unguided materialistic evolutionary process). Biologist Timothy Berra inadvertently illustrated this problem in a 1990 book defending Darwinian evolution against creationist critics. Berra compared the fossil record to a series of automobile models: "If you compare a 1953 and a 1954 Corvette, side by side, then a 1954 and a 1955 model, and so on, the descent with modification is overwhelmingly obvious. This is what [paleontologists] do with fossils, and the evidence is so solid and comprehensive that it cannot be denied by reasonable people." 6

1953
1957
1963
1969
Berra's analogy actually spotlights the problem of using a sequence of similarities as evidence for Darwinian evolution. We all know that automobiles are manufactured according to archetypes (in this case, plans drawn up by engineers), so it is clear that there can be other explanations for a sequence of similarities besides an evolutionary descent with modification. In fact, most pre-Darwinian biologists would have explained such sequences by something akin to automobile manufacturing—that is, creation by design. Even though Berra believed he was defending Darwinian evolution against creationist explanations, he unwittingly showed that the fossil evidence is compatible with either. Law professor and Darwin critic Phillip Johnson refers to this mistake as "Berra's Blunder." 7

1978
Figure 5.16 The "evolution" of the Corvette.

5Bruce A. Young, "On the Necessity of an Archetypal Concept in Morphology: With Special Reference to the Concepts of

'Structure' and 'Homology'," Biology and Philosophy 8 (1993): 231. See also Elliott Sober, Reconstructing the Past (Cambridge, Mass.: MIT Press, 1988), 20.

6Tim Berra, Evolution and the Myth of Creationism (Stanford, Calif.: Stanford University Press, 1990), 117–119, emphasis in the original.

7Phillip E. Johnson, Defeating Darwinism by Opening Minds (Downers Grove, Ill.: InterVarsity Press, 1997), 65–63.

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Berra's Blunder demonstrates that a mere succession of similar forms does not explain the origin of the forms. Something more is needed, namely, a suitable cause, be it an intelligent agent or a mindless material mechanism. In the case of Corvettes, the cause is an agent (human manufacturing), and it can be directly observed. But in a succession of fossils, the cause cannot be directly observed. According to Darwin, the cause of modification and evolution is his mechanism of natural selection and random variation. But why should we think that this mechanism is up to the task? In the ordinary process of reproduction, like always produces like. Can natural selection and random variation so alter the process by which embryos develop that like sometimes produces not-so-like? Darwin didn't know enough about embryonic development to answer the question.

Independent Evidence for Evolution: Developmental Pathways

In 1982, University of Chicago evolutionary biologist Leigh Van Valen wrote that the key to explaining homology lies in understanding the "continuity of information." 8 An embryo contains information, inherited from its parents, that directs its development. Until we understand the nature of that information, we cannot understand how it might be modified. Developmental information could be in the form of "developmental pathways," that is, the patterns of cell division, cell movement, and tissue differentiation by which embryos produce adult structures. Or it could be encoded in genes that affect the development of the embryo. But neither developmental pathways nor developmental genetics has solved the problem of what causes homology, much less confirmed that natural selection and random variation are the mechanism of evolution.

The theory that homologous structures are products of similar developmental pathways does not fit the

evidence, and biologists have known this for over a century. "It is a familiar fact," said American embryologist Edmund B. Wilson in 1894, "that parts which closely agree in the adult, and are undoubtedly homologous, often differ widely in larval or embryonic origin either in mode of formation or in position, or in both."9 More than sixty years later, after reviewing the embryological evidence that had been amassed since Wilson's time, British biologist Gavin de Beer agreed: "The

fact is that correspondence between homologous structures cannot be pressed back to similarity of position of the cells in the embryo, or of the parts of the egg out of which the structures are ultimately composed, or of developmental mechanisms by which they are formed." 10

De Beer's assessment is still accurate. It is "the rule rather than the exception," developmental biologist Pere Alberch wrote in 1985, that "homologous structures form from distinctly dissimilar initial states." 11 Evolutionary developmental biologist Rudolf Raff, who studies two species of sea urchin that develop by radically different pathways into almost identical adult forms, restated the problem in 1999: "Homologous features in two related organisms should arise by similar developmental processes . . . [but] features that we regard as homologous from morphological and phylogenetic criteria can arise in different ways in development." 12

Homology and developmental pathways fail to match up not only in general, but also in the particular case of vertebrate limbs. The classic example of this problem is salamanders. In most vertebrate limbs, development of the digits proceeds from posterior to anterior—that is, in the tail-to-head direction. This accurately describes frogs, but their fellow amphibians, salamanders, do it differently. In salamanders, development of the digits proceeds in the opposite direction, from head to tail. The difference is so striking that some

8Leigh M. Van Valen, "Homology and Causes," Journal of Morphology 173 (1982): 305–312.
9Edmund B. Wilson, "The Embryological Criterion of Homology," 101–124, in Biological Lectures Delivered at the Marine

Biological Laboratory of Wood's Hole in the Summer Session of 1894 (Boston: Ginn and Company, 1895), 107.
10Gavin de Beer, Embryos and Ancestors, 3rd ed. (Oxford: Clarendon Press, 1958), 152.
11Pere Alberch, "Problems with the Interpretation of Developmental Sequences," Systematic Zoology 34 (1985): 46–58.
12Rudolf Raff, "Larval Homologies and Radical Evolutionary Changes in Early Development," 110–121, in Homology,

Novartis Symposium 222 (Chichester, UK: John Wiley & Sons, 1999), 111.

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Figure 5.17 Different developmental pathways converging on homologous structures.

biologists have argued that the evolutionary history of salamanders must have been different from all other vertebrates, including frogs.

Thus, homologous features, even in vertebrate limbs, are often not produced by similar developmental pathways. As further evidence for this claim, consider that skeletal patterns in vertebrate limbs initially form as cartilage, which later turns into bone. If the development of vertebrate limbs reflected their origin in a common ancestor, one might expect to see a common ancestral cartilage pattern early in vertebrate limb development. But this is not the case. Cartilage patterns correspond to the form of the adult limb from the beginning, not only in salamanders, but also in frogs, chicks, and mice. According to British zoologists Richard Hinchliffe and P. J. Griffiths, the idea that vertebrate limbs develop from a common ancestral pattern in

the embryo "has arisen because investigators have superimposed their preconceptions" on the evidence.13

Independent Evidence for Evolution: Developmental Genetics

Since homologies do not in general arise from similar developmental pathways, perhaps they arise from similar developmental genes. According to neo-Darwinian theory, the information that directs embryological development and that Leigh Van Valen regards as the key to explaining homology resides in DNA sequences, or genes. Genes carry information from one generation to the next and, according to the theory, direct the development of the embryo. Therefore, the neo-Darwinian explanation for homologous features is that they are programmed by similar genes inherited from a common ancestor. If it could be shown that homologous structures in different organisms

13J. R. Hinchliffe and P. J. Griffiths, "The Prechondrogenic Patterns in Tetrapod Limb Development and Their Phylogenetic Significance," 99–121, in B. C. Goodwin, N. Holder, and C. C. Wylie (eds.), Development and Evolution (Cambridge: Cambridge University Press, 1983), 118.

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require similar genes and that non-homologous structures require different genes, then we would have evidence for the "continuity of information" referred to by Van Valen.

But that is not the case, and biologists have known it for decades. In 1971, Gavin de Beer wrote, "Because homology implies community of descent from . . . a common ancestor it might be thought that genetics would provide the key to the problem of homology. This is where the worst shock of all is encountered . . . [because] characters controlled by identical genes are not necessarily homologous . . . [and] homologous structures need not be controlled by identical genes." De Beer concluded that "the inheritance of homologous structures from a common ancestor . . . cannot be ascribed to identity of genes."14

To illustrate his point that homologous structures can arise from different genes, de Beer cited one experiment involving eye development in fruit flies. Other examples have since been found. One involves segment formation in insects. Fruit fly embryos require the gene even-skipped for the proper development of body segments. But other insects, such as locusts and wasps, form segments without using this gene. Since all insect segments are considered homologous (whether defined in terms of structural similarity or common ancestry), this shows that homologous features need not be controlled by identical genes. Another example is Sex-lethal, a gene that is required for sex-determination in fruit flies. Sex-determination in other insects occurs without Sex-lethal.

The opposite point that non-homologous structures can arise from identical genes is both more striking and more common. Geneticists have found that many of the genes required for proper development in fruit flies are similar to genes in mice, sea urchins, and even worms. In fact, gene transplant experiments have shown that developmental genes from mice (and humans) can functionally replace their counterparts in flies. If genes control structure, and the developmental genes of mice and flies are

so similar, why doesn't a mouse embryo develop into a fly, or a fly embryo into a mouse? Clearly, something besides genes is influencing development.

Genes and structures fail to match up not only for entire organisms, but also for limbs. One developmental gene shared by several different types of

animals is Distal-less, so named because a mutation in it blocks limb development in fruit flies ("distal" refers to structures away from the main part of the body). A gene with a very similar DNA sequence has been found in mice. In fact, genes similar to Distal-less have been found in sea urchins, spiny worms (members of the same phylum as earthworms), and velvet worms (another phylum entirely)—see figure 5.18.

Figure 5.18 The gene Distal-less is involved in the development of appendages in all five of these animals, yet the appendages are not homologous either by similar structure or by common ancestry. The animals, each in a different phylum, are (counterclockwise from top): mouse, spiny worm, butterfly, sea urchin (its limbs are tube feet underneath its body), and velvet worm.

14Gavin de Beer, Homology: An Unsolved Problem (London: Oxford University Press, 1971), 15–16. On homologous features

not due to homologous genes, see Gregory A. Wray and Ehab Abouheif, "When Is Homology Not Homology?" Current Opinion

in Genetics & Development 8 (1998): 675–680.

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In all these animals, Distal-less influences the development of appendages, yet the appendages of these five groups of animals are not structurally or evolutionarily homologous. “These similarities are puzzling,” noted the biologists who reported them in 1997, because the “appendages have such vastly different anatomies and evolutionary histories.” 15 In 1999 Gregory Wray found “surprising” the association between Distal-less and “what are superficially similar, but non-homologous structures.” He concluded, “This association between a regulatory gene and several non-homologous structures seems to be the rule rather than the exception.” 16

Not only Distal-less but also entire networks of genes involved in limb development have been found to be similar in insects and vertebrates. Clifford Tabin, Sean Carroll, and Grace Panganiban, who described these networks in 1999, noted that “there has been no [evolutionary] continuity of any structure from which the insect and vertebrate appendages could be derived, i.e., they are not homologous structures. However, there is abundant evidence for continuity in the genetic information” involved in their development.17

Evolutionary biologists maintain that the striking similarity of developmental genes in such a wide variety of animal phyla points to their common ancestry. But if so, the same problems we encountered earlier in this section with molecular phylogenies surface again. And that means in particular that the problem of explaining how homologous structures arise remains unresolved. Once again we’ve come full circle. In 1971, Gavin de Beer wrote: “What mechanism can it be that results in the production of homologous organs, the same ‘patterns’, in spite of their not being controlled by the same genes? I asked this question in 1938, and it has not been answered.” 18 Today, seventy years

after it was first asked, evolutionary biology has still not answered de Beer’s question.

5.6 VESTIGIALITY: THE BEST EVIDENCE FOR EVOLUTION?

Shared Errors

To support the use of shared errors as evidence for common ancestry, biochemist Edward Max offers the following argument:

One way to distinguish between copying

and independent [origination] is suggested by analogy to the following two cases from the legal literature. In 1941 the author of a chemistry textbook brought suit charging that portions of his textbook had been plagiarized by the author of a competing textbook (Colonial Book Co, Inc. v. Amsco School Publications, Inc., 41 F. Supp.156

(S.D.N.Y. 1941), aff ’d 142 F.2d 362 (2nd Cir. 1944)). In 1946 the publisher of a trade directory for the construction industry made similar charges against a competing directory publisher (Sub-Contractors Register, Inc. v. McGovern’s Contractors & Builders Manual, Inc., 69 F.Supp. 507, 509 (S.D.N.Y. 1946)). In both cases, mere similarity between the contents of the alleged copies and the originals was not considered compelling evidence of copying. After all, both chemistry textbooks were describing the same body of chemical knowledge (the books were designed to “function similarly”) and both directories listed members of the same industry, so substantial resemblance would be expected even if no copying had occurred. However, in both cases errors present in the “originals” appeared in the alleged copies. The courts judged that it was inconceivable that

15Grace Panganiban et al., “The Origin and Evolution of Animal Appendages,” Proceedings of the National Academy of Sciences USA 94 (1997): 5162–5166.

16Gregory Wray, “Evolutionary Dissociations between Homologous Genes and Homologous Strctures,” 189–203, in Homology, Novartis Symposium 222 (Chichester, UK: John Wiley & Sons, 1999), 195–196.

17Clifford J. Tabin, Sean B. Carroll, and Grace Panganiban, “Out on a Limb: Parallels in Vertebrate and Invertebrate Limb Patterning and the Origin of Appendages,” American Zoologist 30 (1999): 560–663.

18de Beer, Homology: An Unsolved Problem, 16.

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the same errors could have been made independently by each plaintiff and defendant, and ruled in both cases that copying had occurred. The principle that duplicated errors imply copying is now well established in copyright law. (In recognition of this fact, directory publishers routinely include false entries in their directories to trap potential plagiarizers.) Can "errors" in modern species be used as evidence of "copying" from ancient ancestors? In fact, the answer to this question appears to be "yes." 19

What sorts of shared errors does Max cite as evidence of copying from common ancestors? All of his examples derive from molecular genetics. Although the DNA copying mechanism is very accurate, occasionally errors will creep in. Yet precisely because the copying mechanism is so accurate, once an error has crept in, it will tend to persist. One type of error is a deletion mutation in which a specific block of DNA subunits is removed from a functioning gene, thereby rendering it nonfunctional. For instance, goats and cows have a pseudogene for hemoglobin with the exact same deletion.20 Since deletions are rare, Max argues that the occurrence of exactly the same deletion mutation in both organisms establishes their common ancestry.

Retroposons provide perhaps the most persuasive evidence for common ancestry based on shared errors. Retroposons are floating pieces of DNA formed from an RNA template (often viral RNA mapped onto DNA via a reverse transcriptase enzyme). Consider now the following scenario: Suppose a gene has an intron (an intron is a segment of DNA found within a gene that does not code for the gene's protein product). Introns begin with a clear start signal and end with a clear stop signal that tell the cell's protein synthesizing machinery not to transcribe the DNA sequence in between them. Suppose now that a retroposon gets inserted within an intron. There is no known

mechanism for removing it. If there is in fact no such mechanism, the retroposon will tend to be copied faithfully generation after generation (subject, of course, to rare copying errors). What's more, if the retroposon is long enough and has a sufficiently distinctive sequence of bases, it can be used to track evolutionary relationships.

Take, for instance, the SINE CHR-1 retroposon, which consists of 120 DNA bases. This retroposon

is found in the same place in the same gene of even-toed ungulates such as cows, sheep, deer, and giraffes. But it is also found in whales and dolphins, which biologists had previously speculated to be closely related to even-toed ungulates. At the same time, this retroposon is not found in camels and pigs. The SINE CHR-1 retroposon appears to be a useless hanger-on, merely going along for the ride and not doing the organisms that possess it any good. Indeed, why should cows and whales need this retroposon but not camels and pigs? Many geneticists interpret the SINE CHR-1 retroposon as having been inserted millions of years ago into the genome of an animal that was ancestral to cows and whales but not to camels and pigs.

Or take the HERV retroposons (HERV stands for human endogenous retrovirus). These retroposons have been used to connect humans and other primates to common ancestors. According to molecular geneticists Welkin Johnson and Jon Coffin, "The genomes of modern humans are riddled with thousands of endogenous retroviruses (HERVs), the proviral remnants of ancient viral infections of the primate lineage. Most HERVs are nonfunctional, selectively neutral loci. This fact, coupled with their sheer abundance in primate genomes, makes HERVs ideal for exploitation as phylogenetic markers." 21 For instance, old world monkeys and apes have the virus insertion HERVK64, but new world monkeys do not. Treating HERV-K64 as a shared error, molecular geneticists therefore interpret this virus insertion as indicating

19Edward Max, "Plagiarized Errors and Molecular Genetics," available online at http://www.talkorigins.org/faqs/molgen, last accessed October 21, 2003.

20S. G. Shapiro and M. Moshirfar, "Structure of the Goat Psi Beta Y Beta-Globin Pseudogene: Analysis of Goat Pseudogene Evolutionary Patterns," Journal of Molecular Biology 209(2) (1989): 181–189.

21Welkin E. Johnson and Jon M. Coffin, "Constructing Primate Phylogenies from Ancient Retrovirus Sequences," Proceedings of the National Academy of Sciences 96 (1999): 10254–10260.

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that there was an ancestor common to apes and old world monkeys but not to new world monkeys. Similarly, the HERV-K18 virus insertion, which occurs in apes but not in old world monkeys, seems to point to an ancestor common to apes but not to old world monkeys.

What are we to make of such shared-error arguments? How powerfully do they argue for common ancestry? Are they knock-down arguments that conclusively demonstrate the common ancestry of organisms sharing a common error? These arguments, of course, depend on whether the errors truly are errors. If they are not errors but instead serve some useful function for the organism (a function that for now remains undiscovered), then obviously they can't be shared errors and any argument based on their being shared errors dissolves. But there's another way that shared errors can fail to establish common ancestry: if relatively similar organisms (such as even-toed ungulates) are susceptible to similar genetic accidents, then the errors will match up because of this common susceptibility and not because of common ancestry.

Edward Max responds to such objections as follows:

> [Some argue that] we know too little about these newly discovered DNA features to be confident that function will not be discovered for them in the future. [But] imagine a defendant at a murder trial defending himself—against overwhelming incriminating evidence—with the parallel argument: that since some convicted criminals have later been exonerated, he (the current defendant) should therefore be acquitted now, because someday in the future, evidence might be found to clear him. . . . Scientists (and juries) must draw their conclusions based on the best evidence available at the time. It is true that later evidence may exonerate a convicted criminal or overturn a scientific theory, . . . but it should not dissuade us from drawing the most reasonable conclusions from the data at hand. Our present knowledge supports the interpretation that most shared
>
> pseudogenes/retroposons are evidence for common descent and macroevolution.22

In responding to Max's argument, let's begin by noting that a significant proportion of the intelligent

design community finds his argument persuasive and accepts common descent. For intelligent design, common descent is not the crux of the matter. Rather, the crux is whether organisms and the functionally complex structures they comprise are the product of intelligence. Darwinian theory assumes that evolution is a blind, purposeless process from which any actual design is absent. Intelligent design, by contrast, is perfectly compatible with a directed form of evolution in which design plays a substantive and empirically significant role.

That said, Max's argument is not conclusive. For one thing, shared-error arguments based on pseudogenes/retroposons work only below the phylum level. Darwinists argue that this is simply because animal phyla emerged in the Cambrian and are now separated by over 500 million years of evolution. Since pseudogenes and retroposons are supposed to be vestigial DNA and therefore useless, they won't be preserved by natural selection. Hence, Darwinists argue, the only thing that could preserve them is generative inertia resulting from the accuracy of the DNA copying mechanism. But this mechanism, though highly accurate in the short term, can't copy DNA accurately over 500 million years in the absence of selection pressure. Thus, according to neo-Darwinism, we should not expect to find shared errors across distinct phyla. To be sure, this line of reasoning is persuasive within the context of neo-Darwinism, but the fact remains that shared-error arguments do not extend to the phylum level. Accordingly, shared-error arguments cannot underwrite the full-scale macroevolution required by Darwinism.

Also, Max's analogy with a murder trial is flawed. A murder is a designed event. We can have positive evidence of a murder that is truly overwhelming. But a shared-error purports to be an accident. Now the problem with accidental or random events is that we can never be sure that they are indeed accidental, the result of random forces.

22Max, "Plagiarized Errors."

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Instead, we may simply have failed to detect the patterns that clearly mark the events as nonrandom. There is a vast difference between showing that a murder isn't an accident and showing that an accident isn't a murder. If Frank is lying in a pool of blood with numerous stab wounds and the phrase "Die, Frank, Die!" inscribed on his chest, then we have solid evidence that Frank was indeed murdered. What's more, additional forensic analysis may tell us who the murderer is. But if Frank gives all appearance of having died by accident, how do we know that Frank wasn't in fact murdered?

Edward Max would say that the burden of evidence is on the one who wants to claim that Frank was murdered to show that this was in fact the case. And he is right when it comes to the law, where the presumption of innocence trumps the presumption of guilt. But in biology, things are not so straightforward. We know a lot about murders. But we actually know rather little about how the cell works or about the sources of information in the cell (genes coding for proteins are just the tip of the iceberg). Like vestigial organs subsequently found to possess functions and like "silent" mutations subsequently found to be not so silent, pseudogenes may possess undiscovered functions. Granted, the preponderance of evidence at this point seems to indicate otherwise, especially for certain retroposons appearing inside introns. But for now the possibility that pseudogenes possess undiscovered functions needs to be kept open.

Edward Max, by contrast, thinks this possibility needs to be closed off more firmly: "We know enough about how [vestigial DNA sequences] arise that we do not need to postulate any . . . unknown function to explain them." But what we actually know is certain genetic models for how pseudogenes might arise and spread through a population. We also know of specific deletions and insertions in the DNA of living organisms. What we don't know are detailed evolutionary histories that track such genetic changes from a common ancestor through a diverging macroevolutionary process leading to significantly diverse organisms that share a pseudogene. Evolutionary history is inferred

from genes that, ostensibly, have lost their function; it is not verified independently of them. To be sure, the inference has merit, but it must remain tentative; there is not enough evidence at this time to decisively rule out alternative possibilities.

5.7 RECAPITULATION
Darwinian Evolution vs. the Embryological Evidence

Darwinian biologists have long expected ontogeny to provide evidence of phylogeny. Recapitulation in some sense is a logical consequence of Darwinian evolution. The question is: In what sense? In discussions of development and evolution, two views keep recurring. Both are found in Darwin's Origin of Species:

1. The earliest stages of embryos are more similar than their later stages. In Darwin's words: "The embryos of the most distinct species belonging to the same class are closely similar, but become, when fully developed, widely dissimilar." 23 This idea was first articulated by the Prussian/ Estonian zoologist Karl Ernst von Baer, and is often referred to as von Baer's law (though von Baer rejected Darwinian evolution).
2. Embryos pass through the adult forms of their ancestors as they develop. In Darwin's words: "With many animals the embryonic or larval stages show us, more or less completely, the condition of the progenitor of the whole group in its adult state."24 The German zoologist Ernst Haeckel further articulated this idea, and it is referred to as Haeckelian recapitulation or the Biogenetic Law.

Both views are now known to be false—they have been disconfirmed empirically. To see this, let us consider the earliest stages of embryonic development (those substantially preceding the point midway through development on which Haeckel focused). When an animal egg is fertilized, it first undergoes a process called "cleavage," during which it subdivides into hundreds or thousands of separate cells. At the end of cleavage, the cells

23Charles Darwin, On the Origin of Species, 6th ed. (London: John Murray, 1872), 387.
24Ibid., 395.

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Figure 5.19 Early Stages in Vertebrate Embryos. Drawings of early embryonic stages in five classes of vertebrates.
©Jody F. Sjogren 2000Figure 5.19 Early Stages in Vertebrate Embryos. Drawings of early embryonic stages in five classes of vertebrates.
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The stages are (top to bottom): fertilized egg; early cleavage; end of cleavage; gastrulation; and Haeckel's "first" stage.
The fertilized eggs are drawn to scale relative to each other, while the scales of the succeeding stages are normalized to
facilitate comparisons. The embryos are (left to right): bony fish (zebrafish), amphibian (frog), reptile (turtle), bird
(chick), and mammal (human).

begin to move and rearrange themselves in a insect or vertebrate) and for generating basic tissue
process known as "gastrulation." Gastrulation, types and organ systems (e.g., skin, muscles, and
even more than cleavage, is responsible for gut). According to British embryologist Lewis
establishing the animal's general body plan (e.g., Wolpert, "It is not birth, marriage, or death, but

25Lewis Wolpert, The Triumph of the Embryo (Oxford: Oxford University Press, 1991), 12.

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gastrulation which is truly ‘the important event in your life’.” 25

Yet only after cleavage and gastrulation does a vertebrate embryo reach the stage that Haeckel labeled the “first.” If it were true (as Darwin and Haeckel claimed) that vertebrates are most similar in the earliest stages of their development, then the various classes would be most similar during cleavage and gastrulation. Yet a survey of five classes (bony fish, amphibian, reptile, bird, and mammal) reveals that this is not the case (see figure 5.19).

Differences among the five classes are evident even in the fertilized eggs. Zebrafish and frog eggs are about a millimeter in diameter; turtles and chicks start out as discs 3 or 4 millimeters in diameter that rest on top of a large yolk; while the human egg is only about a tenth of a millimeter in diameter (see figure 5.19, top row). The earliest cell divisions in zebrafish, turtle, and chick embryos are somewhat similar, but in most frogs they penetrate the yolk. Mammals are completely different, however, since one of the second cleavage planes is at a right angle to the other (see figure 5.20, second row). Continued cleavage in the other four classes produces a stable arrangement of cells, but mammalian embryos become a jumbled mass.

At the end of cleavage, the cells of the zebrafish embryo form a large cap on top of the yolk; in the frog they form a ball with a cavity; in the turtle and chick they form a thin, two layered disc on top of the yolk; and in humans they form a disc within a ball (see figure 5.20, third row). Cell movements during gastrulation are very different in the five classes: in zebrafish the cells crawl down the outside of the yolk; in frogs they move as a coherent sheet through a pore into the inner cavity; and in turtles, chicks, and humans they stream through a furrow into the hollow interior of the embryonic disc (see figure 5.20, fourth row). Whatever pattern can be

discerned here, it is certainly not a pattern in which the earliest stages are the most similar and later stages are more different (as held by Darwin and Haeckel).

Biologists have known about the striking dissimilarities among early vertebrate embryos for over a century. Embryologist Adam Sedgwick pointed out in 1894 that early similarity and later difference is “not in accordance with the facts of development.” Comparing a dogfish with a fowl (i.e., a chicken), Sedgwick wrote: “There is no stage of development

in which the unaided eye would fail to distinguish between them with ease." Even more to the point: "If [recapitulation] has any meaning at all, surely it must imply that animals so closely allied as the fowl and duck would be indistinguishable in the early stages of development; . . . yet I can distinguish a fowl and a duck embryo on the second day." It is "not necessary to emphasize further these embryonic differences," Sedgwick continued, because "every embryologist knows that they exist and could bring forward innumerable instances of them. I need only say with regard to them that a species is distinct and distinguishable from its allies from the very earliest stages all through the development."26

Modern embryologists confirm this. William Ballard wrote in 1976 that it is "only by semantic tricks and subjective selection of evidence," by "bending the facts of nature," that one can argue that the cleavage and gastrulation stages of vertebrates "are more alike than their adults." 27 The following year Erich Blechschmidt noted, "The early stages of human embryonic development are distinct from the early development of other species."28 And in 1987 Richard Elinson reported that frogs, chicks, and mice "are radically different in such fundamental properties as egg size, fertilization mechanisms, cleavage patterns, and [gastrulation] movements." 29

26Adam Sedgwick, "On the Law of Development Commonly Known as von Baer's Law; and on the Significance of Ancestral Rudiments in Embryonic Development," Quarterly Journal of Microscopical Science 36 (1894): 35–52. Emphasis in the original.
27William W. Ballard, "Problems of Gastrulation: Real and Verbal," BioScience 26 (1976): 36–39.
28Erich Blechschmidt, The Beginnings of Human Life, trans. Transemantics (New York: Springer-Verlag, 1977), 29–30.
29Richard P. Elinson, "Change in Developmental Patterns: Embryos of Amphibians with Large Eggs," in R. A. Raff and E. C. Raff, eds., Development as an Evolutionary Process, vol. 8 (New York: Alan R. Liss, 1987), 3.

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Figure 5.20 The Developmental Hourglass. The vertical axis represents developmental time, from top to bottom; the horizontal axis represents morphological diversity. Vertebrate embryos start out looking very different, then superficially converge midway through development at the "pharyngula" or "phylotypic" stage, before diverging into their adult forms.

Surprisingly, after developing quite differently in their early stages, vertebrate embryos become somewhat similar midway through development. It is this midway point that Haeckel chose as the "first" stage for his drawings. Although he greatly exaggerated the similarities at this stage, some similarities are there. Classical embryologists called this midpoint the "tailbud stage." In 1981 William Ballard called it the "pharyngula" because of the paired ridges and pouches on either side of

the pharynx.30 Klaus Sander proposed in 1983 to call it the "phylotypic stage," since it is here that the various classes first exhibit the characteristics common to all vertebrates.31

So vertebrate embryos start out looking very different, converge in appearance midway through development (though not at the same time), then become increasingly different as they continue toward adulthood. Rudolf Raff describes this pattern

30William W. Ballard, "Morphogenetic Movements and the Fate Maps of Vertebrates," American Zoologist 21 (1981): 391–399.

31Klaus Sander, "The Evolution of Patterning Mechanisms: Gleanings from Insect Embryogenesis and Spermatogenesis," in

B. C. Goodwin, N. Holder, and C. C. Wylie, eds., Development and Evolution, 6th Symposium of the British Society for Developmental Biology (Cambridge: Cambridge University Press, 1983), 140.
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(see figure 5.20) as a "developmental hourglass." 32 According to Darwin, it was the similarity of embryos in their earliest stages that provided evidence for common descent. The actual pattern—early differences, followed by similarities, and then followed by differences again—is quite unexpected in the context of Darwinian evolution. Instead of providing support for Darwin's theory, the embryological evidence presents it with a paradox.

Recently, some embryologists have sought to explain the paradox by proposing that early development evolves much more easily than anyone expected. According to Gregory Wray, differences in early development indicate that "profound changes in developmental mechanisms can evolve quite rapidly." 33 Rudolf Raff suggests "the evolutionary freedom of early ontogenetic stages is significant in providing novel developmental patterns and life histories."34

Whatever the merit of such proposals may be, it is clear that they start by assuming Darwinian evolution and then read it back into the embryological evidence. Of course, this is the exact opposite of basing evolutionary theory on embryological evidence. If one were to start with the evidence and follow Darwin's reasoning about the implications of development for evolution, one would conclude that the various classes of vertebrates are not descended from a common ancestor but had separate origins.

Reflexively looking for evolutionary relationships where none exist, some biologists claim to find evidence for evolution in "gill pouches" or "gill slits" that occur in the embryological development of humans. These are supposed to demonstrate our

genealogical relatedness to fish. The problem is that these "gill slits" are not gills. Midway through development, all vertebrate embryos possess a series of folds in the neck region, or pharynx. The convex parts of the folds are called pharyngeal "arches" or "ridges," and the concave parts are called pharyngeal "clefts" or "pouches." But pharyngeal folds are not gills. They're not even gills in pharyngulastage fish embryos.

In a fish, pharyngeal folds later develop into gills, but in a reptile, mammal, or bird they develop into other structures entirely (such as the inner ear and parathyroid gland). In reptiles, mammals, and birds, pharyngeal folds are never even rudimentary

gills; they are never "gill-like" except in the superficial sense that they form a series of parallel lines in the neck region. According to British embryologist Lewis Wolpert, "A higher animal, like the mammal, passes through an embryonic stage when there are structures that resemble the gill clefts of fish. But this resemblance is illusory and the structures in mammalian embryos only resemble the structures in the embryonic fish that will give rise to gills." 35

In other words, there is no embryological reason to call pharyngeal pouches "gill-like." As Swiss embryologist Günter Rager explains, "The concept 'pharyngeal arches' is purely descriptive and ideologically neutral. It describes folds which appear [in the neck] region. . . . In man, however, gills do never exist." 36 The only way to see "gill-like" structures in human embryos is to read evolution into development. But once this is done, development cannot be used as evidence for evolution without plunging into circular reasoning—like that used to infer common ancestry from the neo-Darwinian conception of homology (see

32Rudolf A. Raff, The Shape of Life: Genes, Development, and the Evolution of Animal Form (Chicago: University of Chicago Press, 1996), 197.

33Gregory Wray, "Punctuated Evolution of Embryos," Science 267 (1995): 1115–1116.

34Raff, Shape of Life, 211.

35Wolpert, Triumph of the Embryo, 185.

36Günter Rager, "Human Embryology and the Law of Biogenesis," Rivista di Biologia 79 (1986): 449–465.

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section 5.4).

6.2 MICHAEL BEHE'S DANGEROUS IDEA

Cumulative Complexity

Irreducible complexity may be distinguished from cumulative complexity. A system is cumulatively complex if the parts of the system can be arranged sequentially so that the successive removal of parts never leads to the complete loss of function. An example of a cumulatively complex system is a city. It is possible to successively remove people and services from a city until one is down to a tiny village—all without losing the sense of community, which in this case constitutes the city's basic function. If we now think of the successive removal of citizens and services from a city as running a videotape backwards, then by changing the videotape direction and running it forwards we see the gradual evolution of a city.

A similar removal process could operate in biology: the successive removal of components from a cumulatively complex biological system might correspond, in reverse, to the gradual buildup of complexity via a Darwinian evolutionary process. Note that for such a process to be truly Darwinian, at each step in the removal process the system's function has to be preserved. It follows that the Darwinian selection mechanism can, at least in principle, account for the evolution of cumulative complexity. Accounting for the evolution of irreducible complexity, however, is another matter: the Darwinian selection mechanism shows no aptitude in producing irreducibly complex biological systems.

Goal-Directed Selection and Irreducible Complexity

Can a selection mechanism account for irreducible complexity? If selection acts with reference to a goal, then there is no difficulty for selection to produce irreducible complexity. Take the old-fashioned pocket watch considered earlier. Given the goal of constructing a functioning timepiece, one can specify a goal-directed selection process that in turn selects a spring, a face, an hour hand, a minute hand, and all the other indispensable parts required for the pocket watch to keep time, and at

the end puts all these parts together to form a

functional watch. Similarly, one can imagine an organism forming a new structure over the course of several generations by successively introducing components (perhaps by random variation), setting them aside (by a goal-directed selection process), and then, once all the components are in place, putting them together to form that new structure. Given a prespecified goal and given the ability to identify and set aside parts needed to accomplish the goal, selection has no difficulty producing irreducibly complex systems.

This line of reasoning can't be extended to biology, however. The selection operating in biology is Darwinian natural selection, and this form of selection operates without goals, plans, or purposes. Natural selection looks not to the future but only to the present. It asks what will benefit the organism now rather than at some future date or in some future offspring. It is interested only in immediate gratification, not delayed gratification. It is an opportunist rather than a strategist. These characteristics of natural selection at once limit it but also account for its appeal among mechanistically inclined biologists who prefer to understand the emergence of biological complexity as the result of undirected material processes and thus apart from design. Yet, by making selection an undirected process, Darwin severely restricted the type of complexity that biological systems could manifest. According to Darwin's theory, biological systems should readily exhibit cumulative complexity. But, as we shall see, Darwin's theory has a much harder time accounting for irreducible complexity.

6.4 COEVOLUTION AND CO-OPTION

Scaffolding and Roman Arches

Besides coevolution and co-option, scaffolding and Roman arches are also used to argue against irreducible complexity. As with all such objections, the point is to show that an irreducibly complex system could, on closer examination, have been produced by gradual increments apart from design. According to the scaffolding objection, for

evolution to produce an irreducibly complex system, first some nonirreducibly complex system needs to arise by mutation and selection incrementally adding components. Then, at some point, a subsystem arises that is able to function autonomously (i.e., without the rest of the system). Since it can function autonomously, the other components are now vestigial and drop away. When all have dropped away, we have a system that is irreducibly complex. In short, what appears to be a qualitative difference is really only the result of a lot of small quantitative changes.

The scaffolding objection thus claims that eliminating functional redundancy is a plausible route to irreducible complexity. If you will, instead of evolution achieving irreducible complexity from the bottom up by gradually adding components to a system, irreducible complexity is supposed to arise from the top down by taking a system and removing redundant components. For instance, there are situations in which, according to Thomas Schneider, "a functional species can survive without a particular genetic control system but . . . would do better to gain control ab initio."1 In such situations, Schneider continues,

Any new function must have this property until the species comes to depend on it, at which point it can become essential if the earlier means of survival is lost by atrophy or no longer available. I call such a situation a "Roman arch" because once such a structure has been constructed on top of scaffolding, the scaffold may be removed, and will disappear from biological systems when it is no longer needed. Roman arches are common in biology, and they are a natural consequence of evolutionary processes.2

To build a Roman arch requires a scaffold. So long as the scaffold is in place, pieces of the arch can be shifted in and out of position. But once all the pieces of the arch are in position and the scaffold is removed (i.e., redundancy is eliminated), each of the pieces of the arch becomes indispensable and the arch itself forms an irreducibly complex system.

Figure 6.4 Roman arch.

But there are two problems here. First, strictly speaking a Roman arch is not irreducibly complex. Yes, each of the pieces of the arch is indispensable

in the sense that if you remove a part, the remaining parts cannot be rearranged to form an arch. But a Roman arch is simplifiable—a single, solid piece of rock can be made into the same shape as the arch, thereby performing the same function as the arch and doing so in essentially the same manner. Even so, one might argue that the failure of a Roman arch to be, strictly speaking, irreducibly complex is not all that serious. A Roman arch, after all, is functionally integrated, and so the question remains whether scaffolds constitute a plausible route to functionally integrated systems generally, and thus perhaps to irreducibly complex systems in particular.

1Thomas D. Schneider, "Evolution of Biological Information," Nucleic Acids Research 28(14) (2000): 2794.
2Ibid.

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Notwithstanding, there is a more serious problem with the scaffolding objection. Consider what it would mean for Darwinian evolution to produce an irreducibly complex system such as the bacterial flagellum by means of a scaffold. The Darwinian selection mechanism acts by taking advantage of, or selecting for, an existing function. What's more, an irreducibly complex system such as the bacterial flagellum obviously exhibits a basic function that is selectable. It follows that the bacterial flagellum plus any putative scaffold exhibits that same basic function, though the scaffold, by now being redundant, is destined to be eliminated by natural selection. So let's ask the following question: In building up to the aggregate system of irreducibly complex system plus scaffold, when did the basic function arise? With a bacterial flagellum plus scaffold, for instance, when did bidirectional rotary motion for propelling the bacterium through its watery environment arise?

Scaffolding does nothing to change the fact that the basic function of an irreducibly complex system arises, by definition, only after all the core components of that system are in place. Given an irreducibly complex system to be explained by scaffolding, the challenge for the Darwinist is to identify a sequence of gradual functional intermediaries leading to it. These need to start from some initial simple system and eventually lead to an irreducibly complex system plus scaffold, whereupon natural selection then discards the scaffold once it becomes redundant. Even though the scaffold can help build the irreducibly complex system, the scaffold is specifically adapted to the basic function of the system it is helping to construct (e.g., the flagellum). What's more, the only evidence of that basic function is from the irreducibly complex system itself. Thus, for the Darwinian mechanism to produce an irreducibly complex system by means of a scaffold, the system plus scaffold must have served a different function up until all the core components of the final irreducibly complex system became available, snapped into place, and formed a functional system. But in that case, the scaffold metaphor becomes inappropriate—a scaffold, after all, is for constructing a structure serving a definite function and not for evolving structures whose functions are likewise evolving.

Intermediates Between the TTSS and the Flagellum

To explain the evolution of the bacterial flagellum, Darwinists typically posit the type three secretory system (TTSS) as an evolutionary precursor to the bacterial flagellum.3 Some even go so far as to posit a few intermediate structures by which the TTSS is supposed to have evolved into the bacterial flagellum.4 But as evolutionary precursors to the bacterial flagellum, such intermediate structures are on even shakier ground than the TTSS. Unlike the TTSS, they exist only in the imaginations of evolutionary biologists. They do not exist in nature or in the laboratory, and evolutionary biologists never define them with enough specificity to be able to recognize them should they actually encounter them. In positing such intermediates, Darwinists purport to provide transitional steps that could lead from the TTSS to the bacterial flagellum. Some even claim that in providing such imaginary intermediates they have provided a "detailed, testable, step-by-step" Darwinian account for the formation of the bacterial flagellum.5 But this is wishful thinking.

One such reconstruction proposes the following transitional steps leading to the bacterial flagellum:

(1) Posit a bacterium that possesses "an ancestral TTSS" to start the evolutionary ball rolling. (2) Next, suppose this bacterium evolves a pilus or hair-like filament that extrudes through the TTSS; this pilus will later become the "propeller" that drives the fully evolved flagellum. (3) Next, suppose this pilus experiences "rapid improvements . . .

3Kenneth R. Miller, "The Flagellum Unspun: The Collapse of 'Irreducible Complexity'," in W. Dembski and M. Ruse, eds., Debating Design: From Darwin to DNA (Cambridge: Cambridge University Press, 2004), 81–97. Though compare Michael Behe's essay in this same volume: "Irreducible Complexity: Obstacle to Darwinian Evolution," 352–370.

4Ian Musgrave, "Evolution of the Bacterial Flagellum," in M. Young and T. Edis, eds., Why Intelligent Design Fails: A Scientific Critique of the New Creationism (Piscataway, N.J.: Rutgers University Press, 2004).

5Nicholas Matzke, "Evolution in (Brownian) Space: A Model for the Origin of the Bacterial Flagellum," published online at http://www.talkreason.org/articles/flag.pdf (last accessed January 19, 2007).

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under selection for increased strength, minimizing breakage, increased speed of assembly, etc." (4) Next, suppose the pilus, though originally involved in adhesion, evolves motility that initially is quite crude, being nondirectional and simply for "random dispersal." (5) Next, suppose this "crudely functioning protoflagellum" gets a chemotaxis and switching system tacked on so that motility becomes directional and interactive with the environment. (6) And finally, suppose this entire system gets refined through natural selection, which evolves a hook and additional axial components and thereby forms a modern flagellum.6

To justify such a model, Darwinists need to show that each step in it is reasonably likely to follow from the previous one. This requires being able to assess the probability of transitioning from one step to the next. And this in turn presupposes that the biological structures at each step are described in sufficient detail so that it is possible to assess the probabilities of transitioning between steps. Darwinism is a theory about connecting points in biological configuration space. It says that you can connect point A to point B in biological configuration space provided that you can take small enough steps where each step is fitness enhancing (or at least fitness neutral). The steps need to be small because Darwinism is a theory of gradual incremental change where each step along the way is reasonably probable. As Darwin put it in his Origin, for his theory to succeed it must explain biological complexity in terms of "numerous, successive, slight modifications." 7 Anything else would cause his theory to founder on the rocks of improbability.

Are the transitions from one step to the next in the preceding model reasonably probable? Does each step constitute, as Darwin required, only a "slight modification"? And is each such modification advantageous or at least selectively neutral? There's no way even to begin to answer such questions because this model is not sufficiently detailed. Evolutionary biologists have empirical evidence

for only one possible precursor to the modern bacterial flagellum, namely, the modern TTSS. They have no empirical evidence for the intermediates that this model posits or for the ancestral TTSS that supposedly starts this model off. They don't know what these intermediates look like. They don't know what mutations are needed to go from one intermediate to the next. They don't have

precise biochemical specifications for the intermediates. They don't know if the intermediates that the model hypothesizes would work. They don't know the environments within which those intermediates would excel or even whether the succession of environments is conducive to the survival and reproduction of the intermediates. They have no way of determining how easy or hard it is for the Darwinian mechanism to bridge the steps in this model.

Evolutionary biologists typically invoke gene duplications and mutations at key points where the Darwinian mechanism is supposed to effect transitions that are reasonably probable. But what gene exactly is being duplicated? And what locus on which gene is being mutated? Evolutionary biologists never say. Indeed, the steps in these models are so unspecific and bereft of detail that these questions are unanswerable. But unless we know detailed answers to such questions, there's no way to know whether the transitions these models describe are reasonably probable and therefore of the type required by Darwin's theory. It follows that such models are untestable. To actually test such models requires being able to evaluate the likelihood of transitioning from one step in the model to the next. Yet because the intermediate systems described at the various transitional steps are so lacking in detail (they are hypothetical; they do not, as far as we know, currently exist in nature; they are not available in any laboratory; and researchers for now have no experimental procedures for generating them in the laboratory), the models offer no way to carry out this evaluation. These models of the evolution of the flagellum are therefore sheer speculation.

6Ibid.
7Darwin, Origin of Species, 189.

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6.5 THE ARGUMENT FROM IRREDUCIBLE COMPLEXITY

Darwinism of the Gaps

A standard move by critics of intelligent design is to charge that the argument from irreducible complexity constitutes an argument from ignorance. In other words, intelligent design is merely capitalizing on gaps in our existing knowledge of how irreducibly complex systems arose, gaps that evolutionary theorists will be sure to fill in as they continue their scientific investigations. One way to formulate this criticism is to say, "Absence of evidence is not evidence of absence." But as with so many overused expressions, this one requires careful scrutiny. Certainly, this expression appropriately characterizes many everyday circumstances. Imagine, for instance, someone feverishly hunting about the house for a missing set of car keys, searching under every object, casing the house, bringing in reinforcements, and then the next morning, when all hope is gone, finding them on top of the car outside. In that case, the absence of evidence prior to finding the car keys was not evidence of absence. Yet with the car keys there was independent evidence of their existence in the first place.

But what if we weren't sure that there even were any car keys? The situation in evolutionary biology is even more extreme than that. One might not be sure our hypothetical set of car keys exist, but at least one has the reassurance that car keys exist generally. Indirect Darwinian pathways that account for irreducible complexity are more like the leprechauns supposedly hiding in a child's room. Precisely because the absence of evidence for

the existence of leprechauns is complete, it is unreasonable to cite "Absence of evidence is not evidence of absence" as a reason for taking leprechauns seriously. And yet that, essentially, is what evolutionary theory counsels concerning the to-date utterly fruitless search for credible indirect Darwinian pathways that account for irreducible complexity.

If after repeated attempts looking in all the most promising places you don't find what you expect to find, and if you never had any evidence that the thing you were looking for existed in the first place, then you have reason to think that the thing you are looking for doesn't exist at all. That's the argument from irreducible complexity's point

about indirect Darwinian pathways. It's not just that we don't know of such a pathway for, say, the bacterial flagellum (the irreducibly complex biochemical machine that has become the mascot of the intelligent design community). It's that we don't know of such pathways for any such systems. The absence of evidence here is pervasive and systemic. That's why critics of Darwinism such as Franklin Harold and James Shapiro (neither of whom is an intelligent design proponent) argue that positing as-yet undiscovered indirect Darwinian pathways for such systems constitutes "wishful speculations." 8 It follows that appealing to the Darwinian mechanism to explain irreducibly complex molecular machines does itself constitute an argument from ignorance: from the absence of evidence for how such machines arose, Darwinists conclude that they must nonetheless have evolved by Darwinian means. This is Darwinism of the gaps.

8Franklin Harold, The Way of the Cell: Molecules, Organisms and the Order of Life (Oxford: Oxford University Press, 2001),

205. James Shapiro, "In the Details . . . What?" National Review (16 September 1996): 62–65.

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CHAPTER SEVEN Specified Complexity

7.1 THE MARK OF INTELLIGENCE

Designer Rocks

If you drive through the Black Hills of southwestern South Dakota, you'll encounter a rock formation that bears unmistakable marks of intelligent design. The formation is Mount Rushmore. Mount Rushmore is a huge relief showing the heads of George Washington, Thomas Jefferson, Abraham Lincoln, and Theodore Roosevelt. There is no question that it was designed. Not only is the circumstantial evidence for its design overwhelming, but we also have direct evidence of its design. In fact, we even know who designed it, namely, the sculptor Gutzon Borglum.1

On the other hand, if you drive through the northern part of Arizona, you'll come across a rock formation that bears no evident marks of intelligent design but only those of wind and erosion. The formation is the Grand Canyon. The Grand Canyon is a huge ravine carved out by material forces, notably, the flow of the Colorado River. Remarkable though the Grand Canyon is, it is reasonable to attribute its formation to blind material forces.

Not all rock formations are so clearly designed as Mount Rushmore or so clearly undesigned as the Grand Canyon. Consider the most famous geological feature in the White Mountains of New Hampshire, namely, the "Profile" or the "Old Man of the Mountains." Nathaniel Hawthorne referred to it as the "Great Stone Face," and the formation became widely known through his short story by the same name. Hawthorne's description of it is as follows:

> The Great Stone Face, then, was a work of Nature in her mood of majestic playfulness, formed on the perpendicular side of a mountain by some immense rocks, which had been thrown together in such a position as, when viewed at a proper distance,
>
> precisely to resemble the features of a human countenance. It seemed as if an enormous giant, or a Titan, had sculpted his own likeness on the precipice.2

When viewed at just the right angle from the just the right distance, the Old Man of the Mountains looks designed. But it really isn't. Material forces

acting blindly and without intelligence or foresight just happened to produce it.

These are three quite different types of rock formations: one that looks designed and in fact is designed (Mount Rushmore), one that does not look designed and in fact is not designed (the Grand Canyon), and one that looks designed but in fact is not designed (the Old Man of the Mountains). That leaves still one possibility we haven't considered: a rock formation that does not look designed but in fact is designed. Finding such objects is the job of archeologists. Archeologists have trained their eyes to see ancient artifacts where most of us see the results of blind material forces. For instance, ancient arrowheads and burial mounds can look like undesigned material objects to untrained eyes but are in fact the product of intelligent design.

Figure 7.8 Mount Rushmore (as it is today)

1See the National Park Service's official website on Mount Rushmore and its history: http://www.nps.gov/moru/historyculture/people.htm (last accessed March 19, 2007).

2N. H. Pearson, ed., The Complete Novels and Selected Tales of Nathaniel Hawthorne (New York: Random House, 1937), 1171.

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Figure 7.11 The Grand Canyon
Figure 7.9 Mount Rushmore (photo of it during construction)

Figure 7.12 The Old Man of the Mountains

Figure 7.10 Gutzon Borglum—the sculptor (designer) of Mount Rushmore

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Recent satellite shot of naturally formed hills in Alberta, Canada that look like a "native American face." Enter 50° 0'38.20"N 110° 6'48.32"W in Google Earth (see http://earth.google.com).

Figure 7.13 Arrowheads and Burial Mounds that don't look like anything special; contrast these with arrowheads that are clearly arrowheads and burial mounds that are clearly burial mounds (e.g., the pyramids).

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Figure 7.14 Stonehenge, the Rosetta Stone, and some natural rock formations from Arches National Park near Moab, Utah.

Consequently, there are four types of rock formations that we might encounter. We can summarize them in the following table:

	Designed	Undesigned
Design-like	Mount Rushmore	Old Man of the Mountains
Undesign-like	Burial Mound	Grand Canyon

In this table “Design-like” denotes objects that look designed, and “Undesign-like” denotes objects that look undesigned. On the other hand, “Designed” denotes objects that actually are designed, and “Undesigned” denotes objects that actually did result apart from design (e.g., as the result of blind material forces).

Table 7.1

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A Reliable Criterion for Detecting Design

As a marker of design, specified complexity functions as a criterion for determining whether an object is the product of intelligence. How so? The word criterion (the plural is criteria) comes from the Greek word for judgment or decision. A criterion is a method for forming a judgment or reaching a decision. In practice, criteria employ observational features of objects to identify some underlying reality of those objects. A common criterion is the medical test. A medical test, by coming up positive, is supposed to indicate the presence of a disease and, by coming up negative, is supposed to indicate its absence. Thus a medical test can be represented schematically as follows:

	Sick	Well
Positive test	I	II
Negative test	III	IV

Table 7.2

If the test comes up positive, we judge the person to be sick (quadrant I). If it comes up negative, we judge the person to be well (quadrant IV).

A perfectly reliable medical test would detect the presence of a disease whenever it is indeed present (quadrant I) and fail to detect the disease whenever it is indeed absent (quadrant IV). Unfortunately, no medical test is perfectly reliable, and so the best we can do is try to keep the proportion of false positives and false negatives as low as possible.3 A false positive occurs when the test says that someone is sick but the person is actually well (quadrant II). A false negative occurs when the test says that someone is well but the person is actually sick (quadrant III).

All criteria, and not just medical tests, face the problem of false positives and false negatives. A criterion attempts to classify individuals with respect to a target group (in the case of medical tests, those who have a certain disease). When the criterion places an individual who should not be there in the target group, it commits a false positive. Alternatively, when the criterion fails to place an

individual who should be there in the target group, it commits a false negative.

The problem of false positives and false negatives also arises in detecting design. Any criterion for detecting design will look as follows:

Designed Undesigned
Design-like I II
Undesign-like III IV
Table 7.3

The target group here consists of all actually designed objects. The problem of false positives arises when objects look designed but really are not (quadrant II). The problem of false negatives arises when objects look as though they are not designed (e.g., the result of blind material forces) but really are designed (quadrant III).

According to Darwinism, all attributions of design to biological systems commit the error of a false positive (quadrant II). Thus, regardless of how designed a biological system looks, in fact it resulted from a blind evolutionary process and therefore apart from any actual design. Accordingly, all criteria indicating that biological systems are designed are said to be unreliable and must give way to evolutionary mechanisms such as natural selection and random variation.

In the past, this view was easier to maintain because many of the criteria used to uncover biological design were unreliable. Consider, for instance, William Paley's main criterion for determining design: the adaptation of means to ends. Organisms certainly exhibit means adapted to ends. For instance, the human eye is adapted to enable sight. But undesigned objects can also exhibit means adapted to ends. Consider a river. A river is adapted to its terrain for delivering water to the sea. But clearly we don't want to say that a river is designed. As a consequence, the criterion of means adapted to ends does not reliably identify design. Other criteria that have been employed historically to identify biological design have

3For the statistics behind medical tests, see Charles H. Hennekens and Julie E. Buring, Epidemiology in Medicine (Boston: Little, Brown and Company, 1987), ch. 13.

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proven similarly unreliable. For instance, geometric symmetry has been invoked as a criterion for detecting design but mistakenly identifies snow crystals as designed.4

Specified complexity, by contrast, serves as a reliable criterion for detecting design. The target group for this criterion comprises all things that are intelligently caused or, as we usually say, designed. How accurate is this criterion at correctly assigning things to this target group and correctly omitting things from it? The things we are trying to explain have causal histories. In some of those causal histories intelligent causation is indispensable whereas in others it is dispensable. An inkblot can be explained without appealing to intelligent causation; ink arranged to form meaningful text on a page cannot. When the criterion assigns something to the target group because it exhibits specified complexity, can we be confident that it actually is designed? If not, we have a problem with false positives. On the other hand, when this criterion fails to assign something to the target group, can we be confident that no intelligent cause underlies it? If not, we have a problem with false negatives.

Consider first the problem of false negatives. When the criterion fails to detect design in a thing, can we be sure that no intelligent cause underlies it? No, we cannot. To determine that something is not designed, this criterion does not work. False negatives are a problem for it. In fact, design-detection criteria in general have no way of resolving this problem. One difficulty is that intelligent causes can mimic blind material causes. A bottle of ink may by chance fall off a cupboard and spill onto a sheet of paper. Alternatively, a human agent may deliberately take a bottle of ink and pour it over a sheet of paper. The resulting inkblot may look identical in both instances, but in one case results from blind material causes, in the other by design.

Another difficulty is that detecting design requires background knowledge. It takes an intelligent cause to recognize an intelligent cause. But if we do not know enough, we will miss it. Consider a spy listening in on a communication channel whose messages are encoded. Unless the spy knows how to break the code used by the parties on whom she is eavesdropping, any messages passing the communication channel will be unintelligible and might in fact be meaningless and the result of chance. Consequently, any design in those messages

will go undetected.

The problem of false negatives therefore arises either when an intelligent agent has acted (whether consciously or unconsciously) to conceal his or her actions, or when an intelligent agent, in trying to detect design, has insufficient background knowledge to determine whether design actually is present. Detectives face this problem all the time. A detective confronted with a murder needs first to determine whether a murder has indeed been committed. If the murderer was clever and made it appear that the victim died by accident, then the detective will mistake the murder for an accident. So too, if the detective misses certain obvious clues, the detective will mistake the murder for an accident. In either case, the detective commits a false negative. Contrast this, however, with a detective facing a murderer intent on revenge and who wants to leave no doubt that the victim was intended to die. In that case, the problem of false negatives is unlikely to arise.

Intelligent causes can do things that unintelligent causes cannot do and can make their actions evident. When, for whatever reason, an intelligent cause fails to make its actions evident, we may miss it. But when an intelligent cause strives to make its actions evident, we often see it. This is why false negatives do not invalidate specified complexity as a criterion for detecting design. This criterion is

4The issue here is the direct design of snow crystals. One could argue that the laws of nature and matter were themselves so designed as to make water organize itself to form snow crystals. Such an argument, however, would not look to specified complexity in the patterns exhibited by snow crystals. Yet then again, specified complexity need hardly exhaust our ability to legitimately find design in the world. Specified complexity is a limited tool with a limited range of application. Was Henry David Thoreau arguing for the design of snow crystals when he remarked, "How full of the creative genius is the air in which [snow crystals] are generated! I should hardly admire more if real stars fell and lodged on my coat"? See Henry David Thoreau, The Journal of Henry David Thoreau, Bradford Torrey and Francis Allen, eds. (Boston: Houghton Mifflin, 1906), 8:87–88.

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fully capable of detecting intelligent agents intent on making their presence evident. Masters of stealth intent on concealing their actions may successfully evade the criterion. But masters of self-promotion, on the other hand, depend on this criterion to make sure that they get credit for their actions.

This brings us to the problem of false positives. Even though specified complexity is not a reliable criterion for eliminating design, it is, we argue, a reliable criterion for detecting design. Think of the criterion of specified complexity as a net. Things that are designed will occasionally slip past the net. We would prefer that the net catch more than it does, omitting nothing due to design. But given the ability of design to mimic unintelligent causes and the power of ignorance to obscure design, this problem cannot be remedied. Nevertheless, we want to be very sure that whatever the net does catch includes only what we intend it to catch—namely, things that are designed. Only things that are designed had better end up in the net. If this is the case, we can have confidence that whatever the criterion of specified complexity attributes to design is indeed designed. On the other hand, if things end up in the net that are not designed, the criterion is undermined.

How can we see that specified complexity is a reliable criterion for detecting design? Alternatively, how can we see that this criterion successfully avoids false positives—that whenever it attributes design, it does so correctly? The justification for this claim is a straightforward inductive generalization: In every instance where specified complexity is exhibited and where the underlying causal story is known (i.e., where we are not just dealing with circumstantial evidence, but where, as it were, the video camera is running and any alleged designer would be caught red-handed), it turns out design actually is present. Therefore, design actually is present whenever the specified-complexity is exhibited. Indeed, concerted efforts by the scientific community to show that this criterion can mistakenly identify design have failed. In particular, none of the proposed counterexamples attempting to show that this criterion commits false positives has

held up. That is to say, there is no known instance of something that is both complex (i.e., highly improbable) and specified (i.e., low descriptive complexity) but not also designed.

Most of the proposed counterexamples look to the Darwinian mechanism to produce structures that are not designed but nonetheless are supposed to exhibit specified complexity. But because the Darwinian mechanism takes a divide and conquer approach to seemingly improbable events, reducing them to a sequence of events with manageable probabilities, it never actually produces highly improbable structures. The great selling point of the Darwinian mechanism is that it is supposed to render probable what only appears improbable. True specified complexity is therefore beyond the reach of this mechanism—specified complexity requires actual improbability and not merely the appearance of improbability. As a consequence, specified complexity establishes not only the design of biological systems but also the insufficiency of Darwinian processes to generate it.

To sum up, the criterion of specified complexity assumes the following form (here SC abbreviates "specified complexity"):

	Designed	Undesigned
Exhibits SC	I	II (no counterexample)
Does not	III	IV (perhaps exhibit SC designed)

Table 7.4

As usual, quadrant I is unproblematic: If something exhibits specified complexity (SC) and in fact is designed, then specified complexity has accurately identified the presence of design. Nor is quadrant II a problem since the criterion of specified complexity does not commit false positives: There is no known counterexample of something exhibiting specified complexity and yet plausibly being accounted for in terms of blind material forces. As for objects that do not exhibit specified complexity, these can either be designed or undesigned—it does not matter. The reliability of specified complexity as a criterion for detecting design is unaffected by false negatives (quadrant III).

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7.7 THE ORIGINATION INEQUALITY

The Drake Equation

The origination inequality parallels the Drake equation, which comes up in the search for extraterrestrial intelligence (SETI). In 1960, astrophysicist Frank Drake organized the first SETI conference. At that conference, he introduced the now-famous Drake equation:

N = Nx
f p x
n x
f 1 x
f i x
f x
f L. 5

e c

*

Here is what the terms of this equation mean:

N
The number of technologically advanced

civilizations in the Milky Way Galaxy capable

of communicating with Earth.

N*
The number of stars in the Milky Way Galaxy.

f p
The fraction of stars that have planetary systems.

n
The average number of planets per star

e

capable of supporting life.

f 1
The fraction of life-supporting planets where life evolves and thus actually becomes present.

f i
The fraction of planets with life where intelligent life evolves.

f

The fraction of planets with intelligent

c

life where civilizations arise and develop

advanced communications technology.

f L
The fraction of a planetary lifetime during
which communicating civilizations exist.

The Drake equation gauges how likely SETI researchers are to find signs of intelligence from distant space: the bigger N, the more likely they are to succeed; the smaller N, the less likely they are to succeed.

Just as there are seven terms on the right of the origination inequality, so there are seven terms on the right of the Drake equation. Just as these terms

in the origination inequality determine its applicability, so with the Drake equation. Moreover, in both the origination inequality and the Drake equation, the seven terms on the right occur in a particular order, reflecting the dependence of terms on previous terms. For instance, the fraction of planets on which intelligent life evolves depends on the fraction of planets on which life as such evolves.

Despite these and other similarities between the Drake equation and the origination inequality—not least that both are used for discovering signs of intelligence—there is also a notable difference. For the Drake equation to convince us that the search for extraterrestrial intelligence is likely to succeed, none of the terms on the right side of that equation must get too small. Only then will SETI researchers stand a reasonable chance of discovering signs of extraterrestrial intelligence. By contrast, with the origination inequality, to determine the specified complexity, and therefore design, of an irreducibly complex system, it is enough to show that even one term on the right side of the inequality is sufficiently small. That’s because all the terms in this inequality are probabilities and therefore cannot exceed 1.

This difference greatly diminishes the applicability of the Drake equation compared to the origination inequality. Because most of its terms cannot be estimated, the Drake equation is difficult, if not

impossible, to apply. As Michael Crichton noted in a widely publicized Caltech lecture,

> The only way to work the [Drake] equation is to fill in with guesses. And guesses—just so we're clear—are merely expressions of prejudice. Nor can there be "informed guesses." If you need to state how many planets with life choose to communicate, there is simply no way to make an informed guess. It's simply prejudice. As a result, the Drake equation can have any value from "billions and billions" to zero. An expression that can mean anything means nothing. . . . I take the hard view

5See Carl Sagan, Cosmos (New York: Random House, 1980), 299.

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that science involves the creation of testable hypotheses. The Drake equation cannot be tested. . . . There is not a single shred of evidence for any other life forms, and in forty years of searching, none has been discovered.6

Yet even if the terms of the Drake equation can be estimated (as opposed to merely guessed at), the equation itself tends more readily to point up the failure of the search for extraterrestrial intelligence than its success. For the Drake equation to undercut the likelihood of finding an extraterrestrial intelligence, it's enough that even one of the equation's terms be estimated and turn out to be small. On the other hand, for the Drake equation actually to confirm the likelihood of finding an extraterrestrial intelligence, all its terms have to be estimated (which, for now, they cannot) and turn out to be large.

By contrast, the origination inequality confirms an intelligence active in the formation of irreducibly complex biological structures provided that even one of its terms can be estimated and turns out to be small. That's because as soon as even one term on the right side of the origination inequality shows itself to be small, the product of terms on the right side (as a product of probabilities) must be at least as small. Consequently, the origination probability, which is bounded above by this product of probabilities, must also be at least that small. In this way, the origination inequality is better suited than the Drake equation for discovering signs of intelligence.

7.8 NOT TOO COMPLEX, NOT TOO SIMPLE, JUST RIGHT

Arguments from Imagination

In place of detailed, testable scenarios for how complex structures such as the eye could have evolved, Darwinists propose imaginative stories devoid of biological specifics. Consider the following account by Richard Dawkins of how the

human eye is supposed to have evolved. In reading it, ask yourself what, if any, specialized biological knowledge from genetics, embryology, or neurophysiology (each of which must play a key role in any thoroughgoing account of the evolution of the eye) was needed to come up with this "evolutionary explanation":

Some single-celled animals have a light-sensitive spot with a little pigment screen behind it. The screen shields it from light coming from one direction, which gives it some "idea" of where the light is coming from. Among many-celled animals, various types of worm and some shellfish have a similar arrangement, but the pigment-backed light-sensitive cells are set in a little cup. This gives slightly better direction-finding capability, since each cell is selectively shielded from light rays coming into the cup from its own side. In a continuous series from flat sheet of light-sensitive cells, through shallow cup to deep cup, each step in the series, however small (or large) the step, would be an optical improvement. Now, if you make a cup very deep and turn the sides over, you eventually make a lensless pinhole camera. . . .

When you have a cup for an eye, almost any vaguely convex, vaguely transparent or even translucent material over its opening will constitute an improvement, because of its slight lens-like properties. It collects light over its area and concentrates it on a smaller area of retina. Once such a crude proto-lens is there, there is a continuously graded series of improvements, thickening it and making it more transparent and less distorting, the trend culminating in what we would recognize as a true lens [as in the human eye].7

Such "arguments from imagination" are, to be sure, entertaining, but they are also misleading.

6Michael Crichton, "Aliens Cause Global Warming," Caltech Michelin Lecture, January 17, 2003, available online at http://www.crichton-official.com/speeches/spee-ches_quote04.html (last accessed February 18, 2007).

7Richard Dawkins, The Blind Watchmaker (New York: Norton, 1987), 85–86.

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They lull us into thinking that we are making scientific progress and gaining scientific insight when in fact we are merely speculating—in the vaguest of terms—about what adaptive changes might have led to the evolution of one structure into another. Science needs to hold itself to a higher standard. What genetic changes were needed for a lens to form within a pinhole camera? What changes in embryological development were required for a light-sensitive sheet to turn into a light-sensitive cup? What neurological changes were needed for a spot to become innervated and thereby light-sensitive? Dawkins does not say. But such questions must be answered before Darwin's theory can properly be said to explain the evolution of the eye.

Yet, rather than admit that such arguments from imagination are scientifically useless, Dawkins turns the tables and charges those who doubt the power of Darwinian processes to produce the eye as guilty of arguing from personal incredulity. 8 In an argument from personal incredulity, one concludes that a proposition is false because one personally cannot think of a good reason for how it could be true. But the issue here is quite different. It's not a failure of imagination that in this instance is justifying doubt in Darwin's theory. Rather, it's that imagination, rather than scientific evidence, is being used to justify acceptance of Darwin's theory. Dawkins, we might say, is arguing from personal credulity, too readily believing in the validity of Darwin's theory based on arguments from imagination that, far from being scientific, are in fact evidence-free speculations.

Note that arguments from imagination do not become scientific by giving them a veneer of technical sophistication. For instance, computer simulations are widely supposed to have shown how the vertebrate eye could have evolved by

Darwinian processes. The principal work cited to prove this point is that of Dan-E. Nilsson and Susanne Pelger.9 But in fact, Nilsson and Pelger never performed a computer simulation of the eye's evolution. Rather, they made some loose calculations based on questionable mathematical models concerning the number of steps it would take for light sensitive cells to arrange themselves into the shape of a sphere (thus resembling an eyeball). The myth that that they did more than this may be credited to Richard Dawkins. As David Berlinski notes:

Nilsson and Pelger's paper has gained currency in both the popular and the scientific press because it has been misrepresented as a computer simulation, most notably by Richard Dawkins. . . . Subsequent references to Nilsson and Pelger's work have ignored what they actually wrote in favor of that missing computer simulation, in a nice example of a virtual form of virtual reality finally displacing the real thing altogether.10

Bottom line: Arguments from imagination, whatever form they take, do not constitute scientific evidence and are useless for deciding whether complex structures evolved by Darwinian processes.

Is the Eye Badly Designed?

Even if Darwinian biologists were to admit that they don't know how the vertebrate eye evolved, they would be reluctant to attribute it to design. That's because the eye is, in their view, badly designed. The problem with the vertebrate eye, according to Darwinian biologists, is that it has an inverted retina. In other words, the photoreceptors in the eye are oriented away from incoming light and situated behind nerves through which light must pass before reaching the photoreceptors. No

8Ibid., 38.

9Dan-E. Nilsson and Susanne Pelger, "A Pessimistic Estimate of the Time Required for an Eye to Develop," Proceedings of the Royal Society of London 256 (1994): 53–58.

10See "A Scientific Scandal? David Berlinski & Critics," Commentary 116 (July-August 2003), available online at http://www.discovery.org/scripts/viewDB/ index.php?command=view&id=1509 (last accessed February 21, 2007).

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self-respecting designer, we are told, would design the eye that way.11

But in fact, there are good functional reasons for this construction. A visual system needs three things: speed, sensitivity, and resolution. The inverse wiring does not affect speed. Nor does it affect resolution (except for a tiny blind spot, which neural processing in the brain readily overcomes). Indeed, there is no evidence that the cephalopod retina of squids and octopuses, which is said to be "correctly wired" by having receptors facing forwards and nerves tucked behind, is any better at resolving objects in its visual field.

As for sensitivity, however, there are good functional reasons in favor of an inverted retina. Retinal cells require the most oxygen of any cells in the human body, so they need a copious blood supply. Retinal cells that face the incoming light would have to be covered by blood vessels, but blood absorbs light strongly. By facing away from the light, retinal cells can be nourished by blood vessels that do not block the light. The result is a design that is so sensitive it can respond to single photons, the smallest unit of light.

Why is the eye so efficient at capturing and processing light? Research published in 2007 and conducted at the Paul-Flechsig-Institute of Brain Research shows that "living optical fibers" create a clear passage for light to the light-sensitive cells at the back of the eye. Concerning his research in this

area, Andreas Reichenbach remarks, "Nature is so clever. This means there is enough room in the eye for all the neurons and synapses and so on, but still the Müller cells can capture and transmit as much light as possible."12

Is this finding more consistent with intelligent design or Darwinism? Darwinists have for years been saying that no competent designer would have wired our retinas the "wrong" way. Instead, we now find optic fibers inside the eye that transmit light with 100 percent efficiency through the layers of "bad stuff " in front (i.e., the wiring) to the "good stuff " in back (i.e., the light-sensitive cones and rods). It appears, then, that the eye is far better designed than previously imagined.

Can the eye be improved? Is it less than optimal? Even if the answer to such questions should turn out to be yes (for now, we just don't know), simply

drawing attention to the inverted retina is no reason to think that eyes with that structure are suboptimal. The design may be counterintuitive, but many clever designs are. And with the discovery of "living optical fibers," the eye's design seems clever indeed. As it is, there are no concrete proposals on the table for how the eye might be improved that also preserve its present speed, sensitivity, and resolution. And even with such a proposal on the table, it would show that the design of the eye could be improved, not that design as such is absent.

11The supposed poor design of the vertebrate retina is widely mentioned in both the technical and popular evolutionary literature. In the technical literature, see William M. Thwaites, "Design: Can We See the Hand of Evolution in the Things It Has Wrought?" Evolutionists Confront Creationists: Proceedings of the 63rd Annual Meeting of the Pacific Division, AAAS 1(3) (1992): 206–213 and George C. Williams, Natural Selection: Domains, Levels, and Challenges (Oxford: Oxford University Press, 1992).
In the popular literature, see Jared Diamond, "Voyage of the Overloaded Ark," Discover (June 1985): 82–92 and Dawkins, The Blind Watchmaker, 93. Thus Dawkins writes, "Any engineer would naturally assume that the photocells would point towards the light, with their wires leading backwards towards the brain. He would laugh at any suggestion that the photocells might point away from the light, with their wires departing on the side nearest the light. Yet this is exactly what happens in all vertebrate eyes. Each photocell is, in effect, wired in backwards, with its wires sticking out on the side nearest to the light. This means that the light, instead of being granted an unrestricted passage to the photocells, has to pass through a forest of connecting wires, presumably suffering at least some attenuation and distortion (actually probably not much but, still, it is the principle of the thing that would offend any tidy-minded engineer!)."

12Reported by Lucy Sherriff, "Living Optical Fibers Found in the Eye: Moving Light Past All Those Synapses," The Register (May 1, 2007): available online at http://www.theregister.co.uk/2007/05/01/eye_eye (last accessed May 2, 2007).

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Figure 7.15 Human and cephalopod eyes.

7.9 VARIATION AND SELECTION OUT OF SYNC From Axe's One in 1064 Improbability to Specified Complexity

Douglas Axe estimated at one in 1064 the improbability of evolving a working ß-lactamase domain. How does this improbability demonstrate that this domain exhibits specified complexity? As we've noted throughout this chapter, brute improbability is not enough to guarantee specified complexity (and therefore design). What's required, in addition, is that the highly improbable outcome in question also conforms to an independently given pattern, or what we called a specification. Now, we noted in section 7.5 that irreducibly complex systems are always specified in virtue of their biological function, and this holds as well for Axe's working ß-lactamase domain. But specifications, as defined in section 7.2, come with a descriptive complexity and presuppose specificational resources. The higher that descriptive complexity, the greater the specificational resources, and hence the smaller the probability needs to be if the system is going to exhibit specified complexity and implicate design.

With Axe's ß-lactamase domain, however, the problem of high descriptive complexity and numerous specificational resources does not arise. That's because working domains with the hydropathic signature of Axe's ß-lactamase domain are not just specified but also uniquely specified—in other words, domains with this hydropathic signature cannot fold and function differently

from Axe's original TEM-1 ß-lactamase domain. To appreciate what's at stake here, imagine that a bullet is shot at a wall and hits an unlucky fly sitting on it. How can we rule out that the bullet happened just by chance to hit the fly? Perhaps most of the wall is just covered with flies, and a random bullet was bound to hit one. But what if the

local area surrounding the unlucky fly was empty of other flies and what if hitting that particular fly by chance within that

local area was highly improbable (think of the local area as a large target surrounding a tiny bull's-eye, namely, the unlucky fly)? In that case, it does not matter if the wall in question is the Wall of China and if all but the local area surrounding the unlucky fly is carpeted with flies. Indeed, it does not matter what the global density distribution of flies is on the wall but only the density distribution in the local area surrounding the unlucky fly.13

This example precisely captures what's happening probabilistically in Axe's experiment and why his one in 1064 improbability is so devastating for Darwinian evolutionary theory. The working domains with the hydropathic signature of Axe's ß-lactamase domain correspond to the unlucky fly on the wall. The complete set of nonworking domains with this signature corresponds to the local area surrounding the fly. Because this local area so dwarfs the working domains, the working domains are extremely improbable. Moreover, because domains with this signature can work in one and only one way (they either function as a working ß-lactamase domain or not at all), they are uniquely specified. Thus, in this analogy, we might say that there are no other insects in the local area surrounding the fly whose demise might engender a design inference if a bullet happened to hit them. In estimating at one in 1064 the improbability of evolving a working ß-lactamase domain, Axe has therefore also established that this domain exhibits specified complexity.

13John Leslie describes this fly-on-the-wall example in Universes (London: Routledge, 1989), 156–162.

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CHAPTER EIGHT The Origin of Life

8.1 WHAT NEEDS TO BE EXPLAINED?

Must Life Evolve?

Is evolution part of what it means to be alive? Does life presuppose evolution? In redefining life to make its origin easier to explain, origin-of-life researchers are now increasingly emphasizing the ability of life to evolve. Because lots of things can evolve, and thus be counted as "alive," without satisfying the more stringent conditions ordinarily demanded of life, this shift in emphasis expands the concept of life to include a lot more than it should.1 The minimal functional requirements for cellular life include reproduction, growth, metabolism, homeostasis, well-defined internal organization, maintenance of boundaries, stimulus-response repertoire, and goal-directed interaction with the environment. Notably absent from this list is evolution. By contrast, geophysicist and originof-life researcher Robert Hazen gives pride of place to evolution in his definition of life: "Most experts agree that life can be defined as a chemical phenomenon possessing three fundamental attributes: the ability to grow, the ability to reproduce, and the ability to evolve." 2

To be sure, in the reproduction of known living forms, offspring always differ, however slightly, from parents. Yet such variation is hardly what is meant by evolution. Indeed, if that's all that was meant by the term, there would be no point to including evolution in the definition of life. To say that the ability to evolve is an essential feature of life must therefore mean, at a minimum, that such changes resulting from reproduction can carry over from one generation to the next, cumulating and thereby bringing about novel species. But how is such an ability to evolve evident simply from inspecting the abilities and functions of actual living forms? It is possible that living forms might vary within such strict limits that no evolution,

in the sense of speciation, could occur. It is even conceivable that asexual forms might reproduce so precisely (suppose, for instance, the copying mechanisms inside these cells were so exact as to rule out copying errors) that offspring were always identical to parents. Granted, such systems might have difficulty adapting to changing environments and thus be more likely to go extinct. But they would, at least for the time, be alive.

The ability to evolve is not a prerequisite for life but an additional property that living forms may or may not possess. If they possess this ability, they exercise it, according to conventional evolutionary theory, not by planning variations to optimize offspring but essentially by rolling dice. And how could life have acquired such an ability to evolve? By "evolving" it from a "proto-life form" that lacked the ability to evolve? Or was the ability to evolve there from the start? And if so, what form did it initially take? Even to raise such questions makes clear that evolvability can never be justified by simply presupposing that organisms must have the ability to evolve. Ascribing to life the ability to evolve thus needs to be a conclusion reached from an exact scientific knowledge of the life-forms in question—and thus based on empirical evidence.

8.2 OPARIN'S HYPOTHESIS

Spontaneous Generation

In the centuries before Oparin formulated his hypothesis, people believed that full-fledged animals could originate suddenly, without parents, from mud, rags, or decaying organic matter such as rotting meat. Today the idea appears no more than a superstition, but at one time both observation and commonsense seemed to confirm it. Leave dirty rags in the corner of a shed, and doesn't it soon become a nest of mice? Leave rotting meat out,

1The field of "artificial life," which had its heyday in the 1980s and 90s, and seems now to have run out of steam, emphasized

evolvability of virtual organisms in virtual environments to the exclusion of the actual functional requirements of actual living

systems. For artificial life's swan song, see Christoph Adami, Introduction to Artificial Life (New York: Springer, 1999).

2Robert M. Hazen, Genesis: The Scientific Quest for Life's Origin (Washington, D.C.: Joseph Henry Press, 2005), 189.

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and isn't it quickly covered with maggots? Decaying meat seemed always to be covered with swarming flies. As a consequence, it appeared that they had originated from it. Thus, it was widely believed that animals could arise on their own, full-blown, from nonliving matter. The belief was called spontaneous generation.

With the rise of modern science, belief in spontaneous generation began to wane. In 1668 Francesco Redi conducted an experiment to determine whether worms arose spontaneously in decaying food. He placed similar samples of raw meat in two sets of jars. One set he covered with a muslin screen, the other he left open. After several days, the muslin screen covering the first sample was sprinkled with fly eggs, but there were none on the meat itself. The meat in the open jar was covered with eggs, which soon hatched into maggots. Redi had shown that maggots were not simply small worms that arose spontaneously but rather were fly larvae. Redi's experiment cast doubt on the spontaneous generation of macroscopic organisms (i.e., organisms large enough to be visible to the naked eye).

After the invention of the microscope, scientists could observe bacteria, and some thought that these originated spontaneously from nonliving chemicals. The idea of spontaneous generation not only seemed to explain the appearance of new individual organisms, but was also used by some to explain the origin of the first life on Earth.

This matched up nicely with Darwin's theory. Darwin's Origin of Species was published in 1859. In that book Darwin hypothesized how species might evolve from already existing species. Darwin's theory purported to explain how life could have become gradually more complex starting from one or a few simple forms. Nevertheless, it did not explain, nor did it attempt to explain, how life had arisen in the first place.

Darwin speculated on the origin of life in one place—an unpublished letter written in 1871 to Joseph Hooker. In that letter he sketched how life

Figure 8.13 Illustration of cover of Darwin's Origin of Species.

might have originated through a series of chemical reactions:

It is often said that all the conditions for the first production of a living organism are now present, which could ever have been present. But if (and oh! what a big if!) we could conceive in some warm little pond, with all sorts of ammonia and phosphoric salts, lights, heat, electricity, etc. present, that a proteine [sic] compound was chemically formed ready to undergo still more complex changes, at the present day such matter would be instantly devoured or absorbed, which would not have been the case before living creatures were formed.3

3Charles Darwin, Letter to Joseph Hooker (1871), in Francis Darwin, ed., The Life and Letters of Charles Darwin, in 3 volumes (London: John Murray, 1887), III:18.

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In the 1870s and 1880s evolutionary thinkers such as Ernst Haeckel and Thomas Henry Huxley also began to speculate about how life originated. Haeckel and Huxley thought that the problem of resolving life's origin would be fairly simple because they assumed that life was in essence a chemically simple substance, which they called "protoplasm." Thus, according to Haeckel, the cell was essentially an enclosed blob of Jell-O or, as he called it, a "homogeneous globule of plasm."4 Both Haeckel and Huxley thought protoplasm could be easily constructed by combining and recombining simple chemicals such as carbon dioxide, oxygen, and nitrogen.

Thomas Henry Huxley (left) and Ernst Haeckel (right)

Over the next sixty years biologists and biochemists gradually revised their view of the nature of life. By the 1930s most biologists had come to see the cell as a complex metabolic system. Origin-of-life theories of the time reflected this increasing appreciation of cellular complexity. Nineteenth-century theories of life's origin envisioned life as arising almost instantaneously via a one- or two-step chemical process. In effect, they proposed that the simplest life forms were capable of forming by spontaneous generation. Yet, by the twentieth century it became evident that not even the simplest life forms could be produced by spontaneous generation.

Even in the nineteenth century, the last outpost of spontaneous generation was the world of microscopic life. Microscopic creatures were so small

and appeared to be so simple that it was not difficult to believe they arose spontaneously from nonliving matter. After all, if bits of straw were left to rot in a pan of water, the water was soon swarming with bacteria. And bacteria were, according to the science of the day, just blobs of protoplasm.

Notwithstanding, Louis Pasteur showed that even here spontaneous generation failed. In the early 1860s, two centuries after Francesco Redi had effectively challenged the spontaneous generation of macroscopic organisms, Pasteur effectively challenged the spontaneous generation of microscopic organisms. In a famous set of experiments, Pasteur showed that water could be kept free of bacteria by boiling it and then exposing it only to purified air. By doing so, he demonstrated that the

microscopic life that mysteriously appeared to
make straw rot in water consisted of airborne bacteria.
Pasteur's elegant experiments showed that
the growth of microbes in otherwise sterile media
was due to contamination by preexisting microbes.

Louis Pasteur, whose experiments challenged the
spontaneous generation of life.

4Ernst Haeckel. The Wonders of Life, trans. J. McCabe (London: Watts, 1905), 111. Compare Thomas Henry Huxley, "On the Physical Basis of Life," The Fortnightly Review 5 (1869): 129–45.

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Although Pasteur's experiments sounded the death knell for the view that microbes could generate spontaneously, it was left to physicist William Tyndall to administer the coup de grâce. Tyndall thought it odd that people with broken ribs that had pierced their lungs but left their skin intact (i.e., the damage was purely internal) did not develop infections even though they were breathing nonsterile air. In 1876 Tyndall therefore decided

to replicate the sterilizing effects of lungs in the laboratory, employing a simple device that became known as a Tyndall box. He coated the insides of a black-painted box with a thin layer of sticky glycerin. When the box was put in a quiet place, all the floating particles inside it soon settled out or collided with the sides and became trapped. The air inside the box could be seen to be completely transparent when a beam of light was shone through it. At this point, sterile solutions of any sort that were exposed inside the box would remain sterile indefinitely. Tyndall boxes were put on display at the Royal Society in London, where they convinced everyone who saw them. . . . The battle of Tyndall's boxes was the last skirmish in a war that had lasted three hundred years, from the time of Francesco Redi. . . . By quite literally settling the dust of this final skirmish, Tyndall managed to resolve the matter.5

Tyndall boxes disposed of an idea that had been held, in one form or another, for thousands of years. Thereafter scientists rejected spontaneous generation as an explanation for the abrupt appearance of microscopic organisms.

In the decades following Pasteur and Tyndall, science's understanding of the complexity of cells greatly increased. Advances in cell biology and biochemistry during the rest of the nineteenth and early years of the twentieth century provided additional reasons for ruling out the abrupt origin of

life from nonlife. During this period, the view that life comes only from preexisting life was universally accepted. Nearly every scientist agreed that cells come only from cells and that even the simplest cell was not generated spontaneously. The idea of spontaneous generation appeared all but dead.

Yet if Darwinian evolution were correct, complex

forms of life ought to have evolved by materialistic processes from simpler ancestors. Redi, Pasteur, and Tyndall had shown that full-blown organisms—whether mice, maggots, or microbes—do not arise from nonliving matter. Nevertheless, Darwinism, the dominant view of evolution, seemed to point to a purely materialistic origin for life. To be sure, Darwin's actual theory focused on the formation of new from existing organisms by purely material processes. But the question whether the origin of life might come about by purely material processes was not far behind. Indeed, without a materialistic explanation of life's origin, Darwin's explanation of the origin of species remains fundamentally incomplete.6

Scientists therefore continued to search for a materialistic explanation of life's origin. During the first two decades of the twentieth century, many advances were made in the study of viruses and the chemistry of living matter. For instance, colloid chemistry became during this period an important field of study. Colloids are particles that make up gels. The size of many colloids is approximately that of large cells, and colloids seem to share some features with cells. Colloids were therefore thought to offer insight into the origin of life. At the same time, amino acids and other simple building blocks of cells were synthesized in the laboratory much as sugars had been synthesized in the nineteenth century. Through these and other studies, scientists gained increasing insight into the chemical makeup of cells. Origin-of-life research thus became a program for showing how the chemical building blocks of life could have originated and organized themselves into living forms by purely material processes.

5Christopher Wills and Jeffrey Bada, The Spark of Life: Darwin and the Primeval Soup (New York: Perseus, 2000), 24–25.

6Richard Dawkins has remarked that "Darwin made it possible to be an intellectually fulfilled atheist." Yet without a materialistic account of life's origin, one cannot be a completely intellectually fulfilled atheist. Richard Dawkins, The Blind Watchmaker: Why the Evidence of Evolution Reveals a Universe without Design (New York: Norton, 1987), 6.

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8.6 THE RNA WORLD

SELEX

Even if RNA were a viable first biomolecule, the RNA world has yet to suggest a self-consistent, self-contained materialistic account of life's origin. Consider a standard experiment from the ribozyme engineering literature: SELEX. The acronym SELEX stands for "Systematic Evolution of Ligands by EXponential enrichment." In 1990 the laboratories of J. W. Szostak (Boston), L. Gold (Boulder), and G. F. Joyce (La Jolla) independently developed this technique, which permits the simultaneous screening of more than a thousand trillion (i.e., 1015) polynucleotides for different functionalities (polynucleotides are sequences of DNA or RNA).7

A typical SELEX experiment starts with a random pool of RNAs that cannot do much of anything and ends with RNAs that can perform a particular function, such as catalyzing a specific reaction or binding to a specific molecule. Consequently, there appears to be a net increase in biologically useful information over the course of the experiment. Moreover, the molecules one gets at the end of the experiment do not match any blueprint identifiable in advance. Thus, the experimenter cannot predict the precise molecular structures that emerge. An extensive effort usually follows a SELEX experiment to characterize the evolved RNA. The RNA must be sequenced, and in some cases it is crystallized for the structure to be solved. Only then does the scientist know what was created and how it performs its function.

SELEX experiments mimic Darwinian evolution in the sense that RNAs that approximate some function get selected and then preferentially duplicated. Do SELEX experiments therefore demonstrate the power of purely materialistic forces to evolve biologically significant RNA structures under realistic prebiotic conditions? Not at all. Intelligent intervention by the experimenter is indispensable. In SELEX experiments large pools of randomized RNA molecules are formed by

intelligent synthesis and not by chance—there is no natural route to RNA (in fact, the chemical processes in nature that facilitate the formation of nucleotide bases undercut the formation of RNA's sugar-phosphate backbone and vice versa). The artificially synthesized molecules are then sifted chemically by various means to select for catalytic

function. What's more, the catalytic function is specified by the investigator. Those molecules showing some activity are isolated and become templates for the next round of selection. And so on, round after round.

At every step in SELEX and ribozyme (catalytic RNA) engineering experiments, the investigator is carefully arranging the outcome, even if he or she does not know the specific sequence that will emerge. It is simply irrelevant that the investigator learns the identity or structure of the evolved ribozyme only after the experiment is over. The investigator first had to specify a precise catalytic function. Next, the investigator had to specify a fitness measure gauging degree of catalytic function for a given polynucleotide. And finally, the investigator had to run an experiment to optimize the fitness measure. Only then does the investigator obtain a polynucleotide exhibiting the catalytic function of interest. In all such experiments the investigator is inserting crucial information at every step. Ribozyme engineering is engineering. Indeed, there is no evidence that material processes as found in nature can do their own ribozyme engineering without the aid of human intelligence.

8.8 MOLECULAR DARWINISM
When All Else Fails—Panspermia

According to some origin-of-life researchers, the conditions on the early Earth were so inhospitable that life did not originate here. Rather, it originated elsewhere in the universe and then was seeded to Earth from outer space. Theories in which life comes to Earth by being seeded from outer space are known as panspermia theories, and they come in two forms.

7See S. Klug and M. Famulok, "All You Wanted to Know about SELEX," Molecular Biology Reports 20 (1994): 97–107.

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In one form, spores that are able to withstand harsh conditions travel through space on dust or asteroids, land on Earth, and thereby seed it with the first life.8 Once life is here, the Darwinian mechanism is supposed to kick in and life is supposed to evolve. There are serious difficulties with this proposal. Out of all the diversity of life forms, few, if any, could withstand the radiation or extremes of heat and cold found in space for periods like those necessary for transport between solar systems. Moreover, the distances between stars are immense. It seems unlikely that spores released near one star would be intercepted by a planet orbiting another.

To circumvent these difficulties, Francis Crick proposed a modification of the panspermia idea known as directed panspermia: intelligent aliens who travel in spaceships come to Earth and seed it intentionally with life (in an interstellar version of "Johnny Appleseed").9 The spaceships protect the life forms and thus circumvent the difficulties associated with the "undirected" panspermia theories.

The chief problem with both undirected and directed panspermia theories is that they explain only the appearance of life on Earth, but not how life originated in the first place. All panspermia theories therefore merely shift the problem of life's origin to another location. In other words, they pass the buck.

8.11 A REASONABLE HYPOTHESIS
The Law of Biogenesis, the Origin of Life, and Universal Common Ancestry

The Law of Biogenesis states that all life comes from life (in Latin, omne vivum ex vivo). In our ordinary experience, this law appears to hold without exception. Nevertheless, it faces an obvious

exception in the origin of life: since life has not always existed, there must come a point (or points) at which life arose from nonlife. Accordingly, the Law of Biogenesis does not hold universally but only after life originates (regardless of whether it originates by strictly material mechanisms or by intelligent design). Given that this law must be suspended whenever life arises from nonlife, one may ask whether in the history of life it was suspended only once or more than once. In evolutionary terms, this is to ask whether the history of life can be represented as a single tree (i.e., universal common ancestry) or as multiple trees. By itself,

the Law of Biogenesis says nothing about the number of starting points for life. That requires an independent assumption. Many materialistic biologists, by regarding it as highly unlikely that the same genetic code could originate more than once, assume that life began only once. But this assumption is becoming increasingly controversial, with molecular evolutionists such as Carl Woese and W. Ford Doolittle holding to multiple origins of life.10 Bottom line: one cannot derive universal common ancestry from the Law of Biogenesis.

Thinking Outside the Box

By limiting themselves to materialistic accounts of life's origin, origin-of-life researchers artificially restrict their problem-solving abilities in a field that requires maximal resources for solving problems. Precisely because the problem of life's origin is so difficult, the full range of theoretical options for resolving it needs to be on the table. As Paul Davies notes: "We are a very long way from comprehending the how [of life's origin]. This gulf in understanding is not merely ignorance about certain technical details, it is a major conceptual lacuna. . . . My personal belief, for what it is worth, is that a fully satisfactory theory of the origin of life demands some radically new ideas." 11

8Fred Hoyle and Chandra Wickramasinghe, Astronomical Origins of Life—Steps Towards Panspermia (Dordrecht: Kluwer, 2000).

9Francis Crick and Leslie Orgel, "Directed Panspermia," Icarus 19 (1973): 341–346. Crick also wrote a book on directed panspermia titled Life Itself (New York: Simon & Schuster, 1981).

10Kalin Vetsigian, Carl Woese, and Nigel Goldenfeld, "Collective Evolution and the Genetic Code," Proceedings of the National Academy of Sciences 103(28) (July 11, 2006): 10696–10701. W. Ford Doolittle and Eric Bapteste, "Pattern Pluralism and the Tree of Life Hypothesis," Proceedings of the National Academy of Sciences 104(7) (February 13, 2007): 2043–2049.

11Paul Davies, The Fifth Miracle: The Search for the Origin and Meaning of Life (New York: Simon & Schuster, 1999), 17.

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Yet to entertain radically new ideas, one must think outside the box. And this, it seems, is where origin-of-life research leaves much to be desired. Wedded to an outdated materialist dogma that rejects design-based hypotheses out of hand, origin-of-life research has been spinning its wheels for the last fifty-plus years. Successful problem-solving requires two forms of ingenuity: (1) the ingenuity of selecting the appropriate reference frame within which to solve the problem and (2) the ingenuity of working adeptly within that frame to find an effective solution. It seems that where origin-of-life research has gone off course is in limiting itself to a materialistic reference frame that consistently fails to provide fruitful insights into the problem of life's origin.

The two-fold ingenuity required for successful problem solving may be illustrated with a classic problem from the field of cognitive psychology. Consider nine dots are arranged in the form of a square as follows:

What is the minimum number of line segments needed to connect all nine dots if they are joined continuously?

Many people assume that the line segments joining the dots have to be confined to the square implicitly outlined by the dots. But, of course, this assumption is entirely gratuitous—the statement of the problem says nothing about confining the line segments to

this implicit square. Given this faulty assumption, one can connect the dots in no fewer than five continuous line segments. But once this assumption is abandoned, and the possibility of drawing line segments outside the implicit square is taken seriously, the solution becomes straightforward:

Thus we see that four continuously joined line segments are sufficient to connect the nine dots.

Although it is too early to tell how successful a design-based approach to life's origin may ultimately prove to be, expanding the reference frame for the problem of life's origin can do no harm and may actually do a lot of good in resolving this problem. In the nine-dots problem, moving to the unrestricted reference frame does not invalidate any of the candidate solutions proposed with respect to the more restrictive

reference frame. It’s just that the restrictive frame offers fewer candidate solutions and, as it is, does not contain the actual solution. The lesson here for origin-of-life research is clear: expanding the range of permissible hypotheses to include design-based explanations of life’s origin is not to rule out materialistic approaches but rather to take away their monopoly so that other solutions, which might be better, have a fair chance to succeed.

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