

# The Evolutionary Gospel According to Sean B. Carroll

A Review of Sean B. Carroll's *The Making of the Fittest: DNA and the Ultimate Forensic Record of Evolution* (W.W. Norton, 2006).

By Casey Luskin<sup>1</sup>

March 16, 2007. Version 1.5.

Copyright © Casey Luskin 2007. All Rights Reserved.

## (1) Praise Darwin and Preach the Gospel

In November 2006, University of Wisconsin-Madison biologist Sean B. Carroll starkly admitted in a biology journal that “[t]he recurring discovery of persistently unresolved clades (bushes) should force a re-evaluation of several widely held assumptions of molecular systematics.”<sup>2</sup> Unfortunately, one of the assumptions he does not recommend re-evaluating is that of common descent itself. This is probably because Carroll believes, as he states in his recent book, *The Making of the Fittest: DNA and the Ultimate Forensic Record of Evolution*, that Neo-Darwinian evolution is true “beyond any reasonable doubt.”<sup>3</sup> However, it would seem that even “reasonable doubt” is not enough for Carroll, as he later admits that it is “a major purpose” of his book to “eliminate *any* doubt” because “there should be *no doubt* about the reality of natural selection.”<sup>4</sup>

Technically, Carroll is right to say that there is “no doubt about the reality of natural selection.” Even skeptics of Darwin readily acknowledge that natural selection is a real force in nature that can effect at least some small-scale changes within species. But Carroll wants us to believe in much more than the mere existence of natural selection. He wants us to accept that all life is related through descent from a common set of microbial ancestors, and that the blind, unguided mechanisms of neo-Darwinism created the diversity of life. When Carroll says “evolution,” he doesn’t just mean changing gene frequencies within a population; he wants us to accept the “evolution of complex bodies and body parts,” as he claims that “direct evidence has poured in as to how complex structures, particularly those of animals, are made and evolved.”<sup>5</sup>

To ensure the reader adopts his own view of evolution, Carroll resorts to scare tactics. After a bleak discussion of the potentially disastrous consequences of global warming, Carroll explains that “acceptance of [evolutionary biology’s] facts” is not “a matter that should be open to political or philosophical debate.”<sup>6</sup> Carroll, who interestingly *always capitalizes the term* “Nature,”<sup>7</sup> quotes Peter Medawar, saying that “the alternative to thinking in evolutionary terms is not to think at all.”<sup>8</sup> Carroll seeks to “focus on the significance of evolution and the importance of adhering to the scientific process in our responsibility to as the stewards of our planet.”<sup>9</sup> For Carroll, the salvation of the human species hangs upon acceptance of neo-Darwinism, and there’s no room for dissenting

---

<sup>1</sup> The author welcomes feedback and may be contacted at [cluskin@discovery.org](mailto:cluskin@discovery.org)

<sup>2</sup> Antonis Rokas & Sean B. Carroll, "Bushes in the Tree of Life," *PLOS Biology*, Vol 4(11): 1899-1904 (Nov., 2006) (internal citations and figures omitted).

<sup>3</sup> Sean B. Carroll, *The Making of the Fittest: DNA and the Ultimate Forensic Record of Evolution*, pg. 17 (W. W. Norton, 2006).

<sup>4</sup> *Id.* at 192 (emphases added).

<sup>5</sup> *Id.* at 192-193.

<sup>6</sup> *Id.* at 39.

<sup>7</sup> For example, see Carroll, *The Making of the Fittest*, at 142, 243.

<sup>8</sup> *Id.* at 39.

<sup>9</sup> *Id.* at 247.

viewpoints (i.e. “debate”) or “any doubt,” and if you don’t accept the facts of Darwin, we’ll all spend eternity in extinction. One might call it the gospel of evolution according to Sean B. Carroll.

Part of Carroll’s rhetorical strategy is to play up Charles Darwin and praise the accomplishments of science. He says that Darwin’s arguments for evolution are “brilliantly constructed, supported by a dazzling breadth of facts” and are “the product of a heroic individual.”<sup>10</sup> In Carroll’s eyes, Darwin can apparently do little wrong. In case the reader misses the message, he notes that Darwin’s discussion of pigeons in *Origin of Species* was merely “the first of many brilliant masterstrokes.”<sup>11</sup> Showing little restraint or reflection upon the uses and misuses of evolution, he heaps praise upon the scientific accomplishments of evolution and expresses his complete inability to understand why anyone would disagree with his viewpoint and support intelligent design:

The argument for design by some external intelligence is eviscerated. It is hard to imagine how anyone in command of these facts could harbor any reasonable doubt. These facts are derived from the very same science and technology that has deciphered the genetic cause of hundreds of diseases, invented dozens of new gene-derived medicines, and revolutionized forensics and agriculture.<sup>12</sup>

Despite his denigration of those who disagree with him, Carroll does have the ability to admit arguments which may challenge his views. For example, he admits that before the “new molecular age” we somehow knew, based upon the fossil record and comparative anatomy, that “the outcome was the survival of the fittest, but we did not know *how the fittest are made*.”<sup>13</sup> In other words, the evidence for evolution no longer comes from fossils or comparative anatomy, it now comes from genes. For an author who is willing to admit that “[d]espite the amount of data and breadth of taxa analyzed, relationships among most metazoan phyla remained unresolved,”<sup>14</sup> he appears extremely confident that the entire future of the world depends on accepting all his arguments. To those, we must now turn.

## **(2) How Carroll Supports Natural Selection**

Carroll offers many arguments in support of evolution. Some of them are familiar canards, such as animal-breeding (pg. 44), peppered moths (pg. 52), or junk-DNA (pgs. 75-76), while others, dealing with opsin proteins, are less common. We’ll begin with the most familiar examples.

### ***Animal Breeding***

Carroll discusses many historical examples of animal breeding which has selected for specific traits in various species. He observes that “[s]ome patterns were intermediate between the patterns of the original breeding stocks, but others were more extreme, *beyond* the limits of the original variation present.”<sup>15</sup> As if this is supposed to be impressive, he points us to a diagram of 13 rats which have different black, white, and gray coloration patterns, all selected through breeding. But he neglects the obvious point that they are all still highly similar rats. Thus, the classic rejoinder to citation of animal breeding is appropriate. Animal breeders are always stopped by impregnable barriers of the amount of change they can effect in a species. Moreover, these examples at best show artificial (intelligent) selection, not natural selection.

---

<sup>10</sup> *Id.* at 31.

<sup>11</sup> *Id.* at 43.

<sup>12</sup> *Id.* at 212-213.

<sup>13</sup> *Id.* at 34.

<sup>14</sup> Antonis Rokas, Dirk Krüger, Sean B. Carroll, “Animal Evolution and the Molecular Signature of Radiations Compressed in Time” *Science*, Vol. 310:1933-1938 (Dec. 23, 2005).

<sup>15</sup> Carroll, *The Making of the Fittest*, at 47.

### ***Peppered Moths***

Carroll tells the standard peppered moth story as an example of natural selection, except he asserts that “[t]he agents of natural selection on the peppered moth are birds.”<sup>16</sup> But Carroll’s assertion is controversial, because the reports of Jonathan Wells indicate that the agent of selection is the missing element in the story.<sup>17</sup> As Wells writes in *The Scientist*:

Most textbook pictures of peppered moths show specimens that have been manually placed on tree trunks. Since 1980, however, it has become clear that peppered moths do not normally rest there. K. Mikkola observed that "the normal resting place of the Peppered Moth is beneath small, more or less horizontal branches (but not on narrow twigs), probably high up in the canopies, and the species probably only exceptionally rests on tree trunks." He noted that "night-active moths, released in an illumination bright enough for the human eye, may well choose their resting sites as soon as possible and most probably atypically." Thus "the results of Kettlewell (1955, 1956) fail to demonstrate the qualitative predation of the morphs of the Peppered Moth by birds or other predators in natural conditions."<sup>18</sup>

### ***Junk-DNA:***

Carroll extensively uses the classic Darwinist icon of junk-DNA, claiming that he will “ignore this junk”:

In humans, and many other complex species, genes occupy only a small fraction of all of the DNA, and are separated by long intervals of noncoding DNA. Some of this noncoding DNA functions in the control of how genes are used, but a lot of it is what is called “junk.” This junk accumulates by various mechanisms and often contains long repetitive tracts with no informational content; it is not purged unless it has adverse effects. I will generally ignore this junk, but it is worth mentioning in order to have a picture of the structure of our genomes as archipelagos of islands (genes) separated by vast areas of open sea (junk DNA).<sup>19</sup>

Time will tell if Carroll was right to “ignore” the “junk DNA,” but at least he has made a concrete admission that, as a die-hard neo-Darwinist, he ignores it.

One type of alleged junk-DNA he discusses extensively is the “pseudogene.” Carroll’s rule of thumb is that when it comes to DNA, you “[u]se it or lose it.”<sup>20</sup> Carroll gives various examples of “pseudogenes,” which he also calls “fossil genes”—claiming they are useless stretches of DNA that used to be functional genes but acquired deleterious mutations due to misuse that caused the original gene to stop working. He cites the bacterial pathogen that causes leprosy, *Mycobacterium leprae*, as having 1600 functional genes and 1100 “fossil genes.” Carroll’s explanation is that it can survive with the 1100 genes “fossilized” because it acts as a parasite, living off of its hosts, and no longer needs them. Perhaps Carroll is right and *M. leprae*’s “fossil genes” really are just non-functional junk. *But Carroll’s “use it or lose it” rule also implies that if something has not been lost, perhaps it is still being used.* Maybe the reason this bacteria species has not completely lost its “fossil genes”

---

<sup>16</sup> *Id.* at 52.

<sup>17</sup> See Jonathan Wells, *Icons of Evolution* (Regnery, 2000).

<sup>18</sup> Jonathan Wells, Second Thoughts about Peppered Moths: This classical story of evolution by natural selection needs revising," *The Scientist*, Vol:13(11):13 (May 24, 1999) (internal citations omitted).

<sup>19</sup> Carroll, *The Making of the Fittest*, at 76.

<sup>20</sup> *Id.* at 123.

is because it's still using them for something. Indeed, he recounts one pseudogene in the coelacanth, a species which, from what paleontologists can tell, has remained unchanged for 360 million years.<sup>21</sup> Could a pseudogene remain unerased for so long if it were truly non-functional? Instead of considering this possibility, Carroll always assumes that these "fossil genes" truly have no function.

Similarly, Carroll claims that half the human genes for smell (olfactory receptor genes) are now "pseudogenes" and do not make functional receptors. I see three possible explanations:

- (1) They have function and were designed or were not designed;
- (2) They don't have function but originally did, and that function was either designed or not, and was lost through natural processes; or
- (3) They don't have function and they were never actually designed in the first place.

Carroll is forced to choose option 3 by default. Not only does he reject the other options outright, he actually makes an extensive argument against ID based upon "pseudogenes":

The fossilization and loss of genes are powerful arguments against notions of "design" or intent in the making of species. In the evolution of the leprosy bacterium, for example, we don't see evidence that this pathogen was designed. Rather, we see that the organism is a stripped down version of a mycobacterium, which still carries around a thousand useless, broken genes that are vestiges of its ancestry. Similarly, we carry around the vestige of an olfactory system that was once much more acute than it is today.<sup>22</sup>

While one might think that humans are the only mammals with such "fossil" olfactory genes, Carroll finds that essentially all mammals have many "fossil" olfactory receptor genes: "In mice, lemurs, and New World monkeys that lack full color vision about 18 percent of olfactory receptor genes are fossilized."<sup>23</sup> If this is true, then these species probably inherited those "pseudogenes" from a common ancestor, which humans also shared. Under his "use it or lose it" rule, what does that imply?

Carroll claims that the mutation rate for mice is  $2 \times 10^{-9}$  per base pair per generation,<sup>24</sup> and other sources indicate that mouse generation time is 3 months.<sup>25</sup> This means that a non-functional mouse "pseudogene" should be completely rewritten in about 125 million years. According to Neo-Darwinists, humans and mice supposedly shared a common ancestor between 75 and 125 million years ago,<sup>26</sup> which means that any such shared "pseudogenes" could have been 60%-100% rewritten by neutral mutations. Could we still recognize a "pseudogene" if it were 60% rewritten? 75%? 100%? I'm not sure, but further investigation on this topic could shed light on whether these "fossil" olfactory genes are truly non-functional "fossils" or functional stretches of DNA for which we simply need to find the function. Indeed, I find it suspicious that all mammals have the same types of "fossil" olfactory "pseudogenes," making me wonder if perhaps these are necessary genetic

---

<sup>21</sup> *Id.* at 119, 123. Of course Darwinists would contend the Coelacanth pseudogene may be a recent change in the Coelacanth species. But we have no way of knowing that apart from assuming that it is non-functional, and therefore must be recent or it would have been lost. Given that our only hard data is the stasis of the Coelacanth species from paleontological data, perhaps that is a questionable assumption indeed.

<sup>22</sup> *Id.* at 136

<sup>23</sup> *Id.* at 128.

<sup>24</sup> *Id.* at 60.

<sup>25</sup> <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=150244&tools=bot>

<sup>26</sup> <http://archives.cnn.com/2002/TECH/science/12/04/coolsc.coolsc.mousegenome/index.html>

components in the mammalian genome which performs an important function. Perhaps this could be an active area of research. But Carroll explores none of these arguments which indirectly would imply that these “pseudogenes” do indeed have some function.

Regardless, it is interesting to see Carroll claim that “pseudogenes” are an argument against design, as that would imply that intelligent design makes testable predictions.

### ***Antarctic Icefish AntiFreeze Genes***

Throughout the book, Carroll uses Antarctic “icefish” antifreeze proteins as one of his favorite examples of how genes evolve. He calls this a “prime example of how evolution works”<sup>27</sup> but admits that the gene has a “simple structure” because it is “made up of 4 to 55 repeats of just three amino acids.”<sup>28</sup> Most genes don’t consist solely of repeats of amino acids, and have a much more complicated sequence structure. So his “prime example” of evolution is from an atypically simple gene. Here is Carroll’s vague explanation of how the gene evolved:

Where in the world did antifreeze come from? Chi-Hing Cheng, Arthur DeVries, and colleagues at the University of Illinois discovered that the antifreeze genes arose from a part of another, entirely unrelated gene. The original gene encoded a digestive enzyme. A little piece of its code broke off and relocated to a new place in the fish genome. From this simple nine-letter piece of DNA code, a new stretch of code evolved for making the antifreeze protein. The origin of the antifreeze proteins stands out as a prime example of how evolution works more often by tinkering with materials that are available—in this case a little piece of another gene’s code—rather than by designing new things from scratch.<sup>29</sup>

This “prime example of how evolution works” seems awfully vague. Carroll’s just-so story of this gene’s evolution has numerous deficiencies.

First, evolving a new functional gene is not remotely as simple as “presto—duplicate 9 nucleotides encoding 3 amino acids and you have a functional gene.” Assuming Carroll’s description of the nature of this gene is accurate, creating a functional antifreeze gene would still require the simultaneous duplication of the 9-letter stretch plus the evolution of a start sequence, a stop sequence, and any introns and regulatory domains associated with the gene. Indeed, the paper Carroll cites to explain “[t]he origin of icefish antifreeze”<sup>30</sup> recognizes these requirements and states that the evolution of this gene required more than the mere duplication of the “nine-letter piece”:

The primordial AFGP gene apparently arose through recruitment of the 5\* and 3\* ends of an ancestral trypsinogen gene, which provided the secretory signal and the 3\* untranslated region, respectively, plus de novo amplification of a 9-nt Thr-Ala-Ala coding element from the trypsinogen progenitor to create a new protein coding region for the repetitive tripeptide backbone of the antifreeze protein.<sup>31</sup>

---

<sup>27</sup> *Id.* at 26.

<sup>28</sup> *Id.* at 25.

<sup>29</sup> *Id.* at 26.

<sup>30</sup> *Id.* at 271.

<sup>31</sup> Liangbiao Chen, Arthur L. DeVries, and Chi-Hing C., “Cheng, Evolution of antifreeze glycoprotein gene from a trypsinogen gene in Antarctic notothenioid fish,” *Proc. Natl. Acad. Sci. USA*, Vol. 94:3811–3816 (April, 1997).

The primary literature shows that the beginning and ending sequences of this gene, as well as its intron, share many similarities with a trypsinogen gene.<sup>32</sup> But these similarities only go so far, and Carroll doesn't discuss the complexities which would be associated with converting a duplicated trypsinogen gene into the antifreeze gene which consists largely of tandem repeats. He just asserts that it happened. But there are other, much greater complexities to this gene.

The second—and most egregious—problem with Carroll's explanation of the evolution of this gene is that he drastically understates the real complexity of this gene. Carroll claims that the proteins are merely “made up of 4 to 55 repeats of just three amino acids.”<sup>33</sup> Perhaps that's true for the final peptide product, but the process by which these small peptides are generated requires a much more complicated interaction of various genes.

The icefish antifreeze genes are actually polyproteins that do not merely contain repeats of 3 amino acids. One of these polyproteins uses at least 10 amino acids—Methionine, Lysine, Leucine, Alanine, Isoleucine, Glycine, Threonine, Proline, Phenylalanine, Valine, Asparagine, Arginine.<sup>34</sup> A polyprotein is a type of protein which, “after synthesis, is cleaved to produce several functionally distinct polypeptides.”<sup>35</sup> In other words, these antifreeze proteins are complex many-in-one proteins designed to be cut into many pieces of specific lengths, each of which performs an important antifreeze function.

In these antifreeze polyproteins, the different segments are separated by special separator markers and cleaved by a “chymotrypsin-like protease.”<sup>36</sup> According to other literature, this polyprotein is unique and may require multiple “cleaver” proteases in order to be broken into its constituent segments:

There are two unique features in the AFGP 8 polyprotein. First, the polyproteins identified to date are of low peptide copy number, up to 8 (36). The AFGP 8 polyprotein contains 46 copies, the highest peptide copy number known so far. Second, most of the bioactive peptide domains in the known polyproteins are flanked by pairs of basic amino acids (Lys-Arg, Lys-Lys, or Arg-Arg), suggesting that trypsin-like proteases are involved in the cleavage reaction (36). Of the 45 3-residue spacers in the AFGP 8 polyprotein, 43 are Leu/Phe-Xaa-Phe, and 2 are Cys-Asn-Phe, suggesting that a chymotrypsin-like protease is the cleavage enzyme. Presumably, a carboxypeptidase-like protease can remove the remaining single (Leu or Phe) or two (Cys-Asn) residues to produce the mature AFGP peptides.<sup>37</sup>

To evolve a unique and functional polyprotein would require the simultaneous evolution of both the polyprotein and its associated cleaver protease enzyme. Michael Behe has discussed the irreducibly complex nature of protease - substrate interactions and the difficulties involved in their evolution

---

<sup>32</sup> Liangbiao Chen, Arthur L. DeVries, and Chi-Hing C. Cheng, “Convergent evolution of antifreeze glycoproteins in Antarctic notothenioid fish and Arctic cod,” *Proc. Natl. Acad. Sci. USA*, Vol. 94:3817–3822 (April, 1997).

<sup>33</sup> Carroll, *Making of the Fittest*, at 25.

<sup>34</sup> *Id.*

<sup>35</sup> <http://www.biology-online.org/dictionary/Polyprotein>

<sup>36</sup> See Figure 2B in Liangbiao Chen, Arthur L. DeVries, and Chi-Hing C. Cheng, “Convergent evolution of antifreeze glycoproteins in Antarctic notothenioid fish and Arctic cod,” *Proc. Natl. Acad. Sci. USA*, Vol. 94:3817–3822 (April, 1997).

<sup>37</sup> K. Hsiao, C.C. Cheng, I.E. Fernandes, H.W. Detrich, and A.L DeVries, “An Antifreeze Glycopeptide Gene from the Antarctic Cod *Notothenia coriiceps neglecta* Encodes a Polyprotein of High Peptide Copy Number,” *Proc. Natl. Acad. Sci. USA*, Vol. 87:9265–9269 (Dec., 1990).

elsewhere.<sup>38</sup> This does not sound like a “simple”<sup>39</sup> protein at all. When this gene’s complexity is considered realistically, it is clear that the just-so story Carroll tells is woefully inadequate to explain how this gene evolved.

### ***Opsin Proteins***

Carroll relies upon opsin proteins in order to argue that new proteins can evolve with only a few small mutations. Opsin proteins are light-sensitive molecules in animal eyes which respond to particular wavelengths of light to permit vision. Carroll finds that two opsin proteins in humans and other primates are highly similar, such that changes in their function could be mostly accounted for by merely 3 amino acid changes. Because this provides Carroll with a good example of how a new protein might evolve, it is when discussing opsins that Carroll encourages his readers to extrapolate from his examples to accept the grander claims of evolution:

Much of the resistance to Darwin’s theories was or is based on doubts about the validity of such extrapolations (e.g., not accepting the “adding up” of effects over vast periods of time). To this point I, too, have implied a degree of extrapolation. I have shown how, for example, given an eye equipped with visual pigments, one or a few changes in pigment proteins alter their properties and help organisms adapt to different light environments.<sup>40</sup>

Carroll does not consider the possibility that the opsins are similar due to common design. However, common design may be suggested by the fact that some opsin proteins among widely divergent species show “convergence,” even at the molecular level. This will be discussed in the next section.

### **(3) Problems for Neo-Darwinism Part 1: Convergent Molecular Evolution**

Carroll provides a fascinating discussion of what evolutionists would term “molecular convergence.” Essentially, he finds that it is not uncommon to see the same amino acid sequences in the genes of widely unrelated organisms where their common ancestor was not thought to have that sequence.

Carroll’s first example deals with rhodopsin, a light-sensitive opsin protein which aids vision. He explains that distantly related deep-water species, such as deep-sea eels (fish), dolphins and whales (both mammals) each have similar amino acids at critical positions in their rhodopsin proteins. In a similar fashion, fresh-water eels have similar amino acids to mammals which use light near the surface of the water or above water (like cows, humans, or manatees). Carroll writes that these similarities must have evolved “independently”:

But eels are fish, whose lines of evolution split off from other kinds of vertebrates several hundred million years ago. This means that the same precise differences in the deep- and shallow-water eels and terrestrial and marine mammals evolved *independently*.<sup>41</sup>

Of course Carroll attributes this sort of “convergence” to natural selection and the presence of similar selection pressures, and ignores the possibility of common design:

---

<sup>38</sup> See Michael Behe, *In Defense of the Irreducibility of the Blood Clotting Cascade: Response to Russell Doolittle, Ken Miller and Keith Robison*, <http://www.discovery.org/scripts/viewDB/index.php?command=view&id=442> and Michael Behe & David W. Snoke, "Simulating evolution by gene duplication of protein features that require multiple amino acid residues," *Protein Science*, Vol. 13 (2004).

<sup>39</sup> Carroll, *The Making of the Fittest*, at 26.

<sup>40</sup> *Id.* at 192.

<sup>41</sup> *Id.* at 109 (emphasis in original).

The DNA record also reveals that evolution can and does repeat itself. Similar or identical adaptations have occurred by the same means in species as different as butterflies and humans. This is powerful evidence that, confronted with the same challenges or opportunities, the same evolution can arise at entirely different times and places in life's history.<sup>42</sup>

However, if genetic similarities can tell Darwinists both that two organism are both closely related and distantly unrelated, what is the explanatory power of neo-Darwinian systematics? Of course ID can accommodate either relatedness or unrelatedness, but independent convergence (if selective pressures are demonstrably insufficient to achieve the observed similarities) constitutes evidence for ID and evidence against neo-Darwinism. Indeed, Carroll admits that this molecular convergence is “[t]he most profound surprise of all”:

The *most profound surprise* of all, though, is how evolution repeats itself (chapter 6). When species that have independently gained or lost similar traits are compared, we often find that evolution has repeated itself, at the level of the same gene, sometimes right down to the very same letter in the code of the same gene.<sup>43</sup>

At the DNA level, such different structural details are expected to involve evolutionary changes in different genes. What makes the howler case so remarkable, as well as many of the other examples I will describe in this chapter, is that the recurring events in different species involve the same genes, and sometimes the very same letters of DNA code.<sup>44</sup>

Despite his post-hoc appeals to similar selection pressures, Carroll admits that these examples of molecular convergence may not have been “expected” under his neo-Darwinian mindset. Other examples he gives of molecular convergence include similar digestive enzymes in monkeys and cows, body pigmentation genes in mice, birds, jaguars and bears, and antifreeze proteins in Arctic and Antarctic fish. The antifreeze proteins are of particular interest due to the unique but highly similar functional nature of those proteins. Some other potential examples of molecular convergence will be discussed in part (5).

While Carroll is surprised at this data, it would have been completely expected under a theory of common design, where the same genetic blueprint might be re-used in different organisms. In fact, molecular similarities among widely different organisms are precisely what one would expect if an intelligent designer were involved.

#### **(4) Problems for Neo-Darwinism Part 2: Phylogenetic Trees**

Carroll is upfront about the problems encountered when constructing phylogenetic trees. He never questions neo-Darwinism, but he does admit that contradictions between morphological and fossil-based trees existed even before molecular data was analyzed:

For the larger part of biology's history, trees were constructed based upon the outward appearance of living species, as well as those of species in the fossil record. The resemblances or lack of resemblances were, however, often discovered to be

---

<sup>42</sup> *Id.* at 16.

<sup>43</sup> *Id.* at 36-37 (emphasis added).

<sup>44</sup> *Id.* at 141.

misleading, or at least a source of contention and disagreement among biologist such that many, many different trees have been drawn of virtually all groups.<sup>45</sup>

Carroll also directly acknowledges the fact that a “pattern of treelike evolution” is challenged by the molecular data:

The possibilities for building species trees based upon DNA, RNA, and protein sequences were quickly recognized by scientists (such as Francis Crick, Emile Zuckerkandl, and Linus Pauling), as soon as protein sequencing began to reveal the similarities and differences in proteins shared among groups of species. ... Darwin described the genealogy of species as trees, with speciation producing ramifying branches. But in the world of microbes, unknown to Darwin, some events happen that violate the pattern of treelike evolution. Microbes exchange genes, and some microbes live within the host species in a process called endosymbiosis. These processes enable the transfer of genes between very distant relatives, and thus confuse the family tree.<sup>46</sup>

This inability to resolve species relationships into a tree is most apparent when dealing with the three fundamental domains of life, commonly thought to be the base of the tree of life: “The resulting base of the tree of life is then not a trunk, but a ring from which our tree ascends and branches.”<sup>47</sup> Indeed, Carroll notes that such conflicts are common, stating that, “I do not want to give the impression that every gene will give the same, correct, or clear answer. So, one way that potential trees are tested further is by examining whether the same trees are obtained with different sequences, and with larger amounts of sequences.”<sup>48</sup> As noted, however, Carroll has elsewhere stated that “[d]espite the amount of data and breadth of taxa analyzed, relationships among most metazoan phyla remained unresolved,”<sup>49</sup> and also, “certain critical parts of the [tree of life] may be difficult to resolve, regardless of the quantity of conventional data available.”<sup>50</sup> So despite his many admissions about problems constructing phylogenetic trees, his book may be painting a rosier picture than he admits when writing before biologists.

### **(5) How are the fittest made?**

Chapter 8, “The Making and Evolution of Complexity,” promised to be the most important of the book, which after all is titled *The Making of the Fittest*. We are forewarned at the beginning of this chapter that we may have to sometimes employ “extrapolation”<sup>51</sup> from his examples in order to understand how evolution works. That’s an understatement, given the paucity of the examples he provides.

Carroll’s primary example is the fact that the gene *Pax-6* is used in widely different organisms—from vertebrates to mollusks to arthropods to echinoderms—to build eyes. He has a useful diagram on page 195 showing how the amino acid sequence of *Pax-6* is nearly identical between fruit flies, mice, and humans, even though mammals and insects have completely different types of eyes.

---

<sup>45</sup> *Id.* at 98.

<sup>46</sup> *Id.* at 85.

<sup>47</sup> *Id.* at 88.

<sup>48</sup> *Id.* at 99.

<sup>49</sup> Antonis Rokas, Dirk Krüger, Sean B. Carroll, "Animal Evolution and the Molecular Signature of Radiations Compressed in Time," *Science*, Vol. 310:1933-1938 (Dec. 23, 2005).

<sup>50</sup> *Id.*

<sup>51</sup> Carroll, *The Making of the Fittest*, at 192.

Carroll recounts how it was originally thought, due to the widely different types of eyes among animals, that eyes evolved independently many different times—perhaps between 40 and 65 times. When it was discovered that *Pax-6* controlled eye development in so many diverse types of eye-bearing animals, Carroll explains that “it is very unlikely that each happened upon the use of *Pax-6* by accident.”<sup>52</sup> Evolutionists thus assume that the first eye-bearing animals *must* have used *Pax-6* and that the usage was passed down to all of their descendants.

But where did those first eyes come from? Carroll warns against assuming that the simplest eyes are really “simple”: “But do not be fooled by these eyes’ simple construction and appearance. They are built with and use many of the ingredients used in fancier eyes.”<sup>53</sup> These ubiquitous eye similarities include the use of *Pax-6*, opsin proteins, and other visual pigments. Thus, the earliest eyes had the same basic functional mechanisms as the most advanced eyes known. Carroll expects us to think this explains the evolution of complexity, but he never explains where the first eye came from. The infamous simple “light sensitive spot” is not simple at all, and it requires an explanation which Carroll does not provide. How are the fittest made?

Given a light sensitive spot, Carroll asserts that further eye evolution “is a matter of just arranging larger numbers of the same types of eye cells in three-dimensional space—the same building materials, a different organization.”<sup>54</sup> As evidence that this is possible, he asserts that “[c]omputer modeling by Dan Nilsson and Susanne Pelger at the University of Lund in Sweden has suggested that selection on small variations could, in 2000 steps over as few as 500,000 years, produce a camera eye from a *simple* prototype.”<sup>55</sup> But wait—I thought Carroll just told us the earliest eyes weren’t actually so “simple.” And what of this “computer modeling”? According to mathematician David Berlinski, the computer model of eye evolution is a Darwinist urban legend, which was widely promulgated by Richard Dawkins. Berlinski writes:

**The facts:** Nilsson & Pelger’s study, which was widely considered a computer simulation, contained *no* computer simulation whatsoever. It contained, in fact, no *computer* analysis at all, perhaps because it contained no *analysis* at all. It was Richard Dawkins who conveyed the widespread impression to the contrary, writing about a computer simulation that did not exist with the excitement of a man persuaded that he had seen a digital vision. As, indeed, he had. Commentators at the time came to Dawkins defense with a gratifyingly prompt display of personal generosity, so that what was, in fact, a complete fabrication took on the aspects of an understandable but trivial error. Any man, after all, might mistake nothing for something.<sup>56</sup>

Berlinski goes on to explain that Nilsson and Pelger’s study lacks calculations and details vital to the claims they are making:

Moreover, Nilsson and Pelger do not calculate the “visual acuity” of any structure, and certainly not over the full 1,829 steps of their sequence. They suggest that various calculations have been made, but they do not show how they were made or tell us

---

<sup>52</sup> *Id.* at 196.

<sup>53</sup> *Id.* at 197.

<sup>54</sup> *Id.* at 197.

<sup>55</sup> *Id.* at 199 (emphasis added).

<sup>56</sup> David Berlinski, “The Vampire’s Heart, at <http://www.discovery.org/scripts/viewDB/filesDB-download.php?command=download&id=1061>

where they might be found. At the very best, they have made such calculations for a handful of data points, and then joined those points by a continuous curve.<sup>57</sup>

All of this aside, it appears that ideas about eye evolution have undergone major revisions since the discovery of *Pax-6*. The old phylogeny has been discarded. No longer are eyes thought to have evolved independently, because the odds are far too small that so many organisms would independently employ the same gene—*Pax-6*—for eye construction. Under neo-Darwinism, that trait must be primary. Unfortunately for neo-Darwinists, there is some molecular data which conflicts with this new hypothesis:

One of the other deep puzzles of eye evolution concerns the different kinds of photoreceptors founding our eyes versus those found in squid or fly eyes. In human and other vertebrate eyes, the rod and cone photoreceptor cells are of the so-called ciliary type, while in squids and flies, the photoreceptor cells are called rhabdomeric. The distinction has to do with how the membranes of each type of photoreceptor cell are enlarged in order to pack them with opsins. This difference was one of the key pieces of cellular evidence cited for the “independent” origin of vertebrate and other animal eyes.<sup>58</sup>

Under a design-paradigm, we might expect a designer to re-use similar parts in different organisms. Under his Darwinian paradigm, Carroll calls these unexpected similarities “puzzles,” but if Carroll were willing to consider the possibility of common genetic design, perhaps these “puzzles” would make more sense.

Carroll finds that the re-usage of common genes in widely different organisms is not restricted to eyes. He writes that discoveries “have also revealed that common genetic tools are used to build the very different hearts, digestive tracts, muscles, nervous systems, and limbs of all sorts of animals.”<sup>59</sup>

Finally, Carroll gives a few other examples in his chapter explaining “the making and evolution of complexity.” The first involves the fact that single mutations in various genes can abolish eyes or the pelvis in fish. These are simple mutations which turn off regulatory genes, thereby preventing an organ structure from forming. His second example deals with the loss of wing spots on butterfly wings. Again, the mechanism is a simple mutation which turns off the wing-spot genes. ***These examples all invoke loss of function by turning off pre-existing genes.*** Exactly *how are the fittest made?* Carroll’s examples don’t answer that question.

It is from these examples that Carroll asserts that “[t]he argument from design by some external intelligence is eviscerated.” If the loss of function by turning off genes, and the usage of the same genes to build organs in vastly diverse organisms—a fact cited by design-proponents as supporting common design<sup>60</sup>—are the best facts he can muster against design, then it would appear that ID has very little to fear from the discoveries of evo-devo. To reiterate, after reading this chapter, I fear that I am still left wondering, “How are the fittest made?”

## (6) Carroll’s Projections onto Darwin-Skeptics

---

<sup>57</sup> *Id.*

<sup>58</sup> Carroll, *The Making of the Fittest*, at 201.

<sup>59</sup> *Id.* at 202.

<sup>60</sup> See Paul Nelson and Jonathan Wells, “Homology in Biology: Problem for Naturalistic Science and Prospect for Intelligent Design,” in *Darwinism, Design, and Public Education* (John Angus Campbell and Stephen C. Meyer, eds., Michigan State University Press, 2003).

Carroll's chapter on "the denial of evolution"<sup>61</sup> focuses primarily upon arguments from young earth creationists, such as Henry Morris. He can't imagine how anyone could doubt the science, and therefore concludes that it *must* be the result of "religious ideology": "My fundamental premise is that the denial of evolution, like the other instances of denial, is not about the science. It can't be. It is about ideology, in this case religious ideology."<sup>62</sup> As noted in the beginning of this review, Carroll is so entrenched in his own mindset that he thinks that no one can have rational scientific reasons for disagreeing with his own viewpoint. After his excessively weak effort in the linchpin chapter on how complexity evolves, perhaps Carroll needs to apply some healthy skepticism to his own perspective. Carroll gives six rationalizations to explain why people doubt evolution. As discussed below, Carroll engages in much projection, so perhaps a little more self-scrutiny would not hurt:

1. "Doubt the science": Actually, this "tacti[c]"<sup>63</sup> disproves what Carroll said earlier about Darwin-skepticism not being about science. Carroll gives quotes from Darwin skeptics who, although they are creationists, make some legitimate points. He quotes creationist Henry Morris discussing how evolution does not produce new genetic information, or how there are few transitional forms. Though the quote he provides from Morris leaves room for attack, the paucity of transitional forms in the fossil record can be a legitimate criticism of neo-Darwinism when framed in the right manner.
2. "Question the Motives and Integrity of Science": Carroll claims that Darwin-skeptics are always attacking the motives of scientists who promote evolution. But it is ironic that Carroll tries to expose this "tactic" because he himself states prior to scrutinizing Darwin-skeptics that "[w]e have to pierce those screens to understand the motives behind them."<sup>64</sup> Carroll himself thus attacks the motive of Darwin-skeptics and seems to be projecting this tactic on to Darwin-skeptics.
3. "Magnify Disagreements Among Scientists and Cite Gadflies as Authorities": Carroll complains about people who "mine" quotes from Darwinists. I'm sure I'll be accused of this because I cite Carroll so much in this review. Perhaps the first part of this objection stems more from Carroll's personal frustration than any legitimate complaints. One always has to be careful when citing opponents, but when they make a stark admission, no fault can be charged against those who cite that authority. And given how often Darwin-skeptics commonly cite the highest authorities, like Stephen Jay Gould, Bruce Alberts, or Charles Darwin himself, I see little basis for Carroll's charge that Darwin-skeptics "cite gadflies as authorities." Unless Carroll considers himself a gadfly, I assume he will have no objections when I cite him discussing problems the molecular data pose for constructing phylogenetic trees.
4. "Exaggerate the Potential Harm": Carroll cites Biblical creationists trying to tie Darwinism to various horrors of the 20<sup>th</sup> century, such as Nazism or communism. While their tactics may not always be productive or appropriate, again, we see Carroll is projecting. Just as Carroll claims Darwin-skeptics "exaggerate the potential harm" of accepting evolution, Carroll himself suggests that failing to accept evolution could cause the downfall of the whole biosphere: And to what does he compare the coming horror if we fail to accept

---

<sup>61</sup> Carroll, *The Making of the Fittest*, at 233.

<sup>62</sup> *Id.* at 234.

<sup>63</sup> *Id.* at 234

<sup>64</sup> *Id.* at 234.

evolution? He compares the fight against Darwin-skeptics to the fight against “Nazism” and “Soviet-style communism”<sup>65</sup>:

I realize that this chapter is ending our afterdinner conversation on a most downbeat note. But I believe this is absolutely necessary. I am a professional biologist, yet I was largely unaware of the dire statistics I have just presented until I did the research for this chapter. You can at least be thankful that I did not examine trends in the rain forests or other habitats here. Winston Churchill’s words are the most apt parting message I can offer. The future of nature at present looks terribly gloomy, much as the state of geopolitics looked to Churchill in 1935. He saw the threat of fascism, Nazism, and Soviet-style communism and issued countless warnings that went unheeded for years. Then, as now, most of the West’s leaders were in denial ... Yet here we are at the opening of the twenty-first century, with the benefit of two centuries of evolutionary science, still debating the existence of evolution. And with more than two centuries of experience of the consequences of overfishing, overhunting, and pollution, chasing what few fish are left.<sup>66</sup>

Carroll tries to lump ID-theorists with global warming skeptics, a lumping which I stridently oppose. The claim that design theorists are both anti-science and anti-conservation doubly fails. I am all for conservation and protecting the environment. But it is odd that Carroll accuses Darwin-skeptics of “exaggerating the potential harm” when he makes the outlandish argument that “the future of nature” is bleak unless we stop doubting Darwin.

5. “Appeal to Personal Freedom”: Carroll argues against teaching any evidence against Darwinism in public schools. This sounds fairly dogmatic, and indeed Carroll acknowledges that he’s not on the side of “personal freedom” in this debate. He cites a now-defunct court ruling that removed a sticker-disclaimer from Cobb County, Georgia biology textbooks which simply suggested that students approach evolution “with an open mind, studied carefully, and critically considered.” It’s revealing that Carroll would oppose such innocuous language.

Carroll claims that when Judge Clarence Cooper struck down the stickers, the Judge was troubled that the policy failed to explain why evolution “is the only theory being isolated.” In fact, this badly misrepresents the ruling. In the two sentences of the ruling following the section quoted by Carroll, Judge Cooper explains that *he accepted the school board’s singling out of evolution*:

The School Board members convincingly testified at trial that they believed all scientific theories should be critically considered, and they also stated that they singled out evolution ***because it was the topic causing the controversy at the time.*** *The Court finds the School Board’s explanation to be rational and does not declare the Sticker to violate the purpose prong of Lemon.*<sup>67</sup>

Singling out evolution was appropriate precisely because evolution *was controversial!* Carroll is not a lawyer, so perhaps he did not read the case carefully. (Judge Cooper went on

---

<sup>65</sup> *Id.* at 267.

<sup>66</sup> *Id.* at 267-268.

<sup>67</sup> *Selman v. Cobb County Board of Education*, 390 F. Supp. 2d 1286, 1309 (N.D. Ga. 2005) (emphases added).

to strike down the stickers on the effect prong due to a bizarre and unorthodox line of reasoning which threatened the political rights of religious Americans, but that's a story for another day.)

6. "Acceptance Repudiates Key Philosophy": As his final reason for why people oppose evolution, Carroll explains that evolution "is viewed to be at odds with matters of faith that are not open to scientific evidence."<sup>68</sup> He criticizes Biblical creationists at length, but then affirmatively promotes pro-evolution theology by quoting from various religious organizations that accept evolution. It would be interesting to find out if Carroll, who capitalizes "Nature" throughout the book, holds metaphysical views which would be refuted if evolution were false. Perhaps he should scrutinize his own motives and beliefs rather than simply attacking those of others.

Finally Carroll turns to intelligent design. He wrongly characterizes ID as merely a negative argument against evolution. However, Carroll does treat Michael Behe's ideas about a designer possibly using "'pre-formed' genes" as a real scientific hypothesis. Carroll argues that this is an unlikely explanation because of the "use it or lose it rule." Early organisms holding unused potential genes would lose them to do lack of use, and they would get overwritten by neutral mutations due to a lack of selection to preserve unused genes. This sounds like a legitimate objection from Carroll, and proponents of ID who hold to a "pre-formed" genes model should take it seriously. As one who has never favored the "pre-formed" genes hypothesis, I agree with Carroll here.

Carroll also tries to refute intelligent design by appealing to "pseudogenes." He asks, "What Designer designs partial, non-functional genes?"<sup>69</sup> Again, that is a legitimate question to ask, and it proves that ID can make testable claims: designers make things which are functional. As I stated earlier, "pseudogenes" really present three options:

- (1) They have function and were designed or were not designed;
- (2) They don't have function but originally did, and that function was either designed or not, and was lost through natural processes; or
- (3) They don't have function and they were never actually designed in the first place.

Carroll always chooses option (3), but I think that there could be modes of exploring options (1) and (2). ID would retain its testability because it would typically be held that even if the gene were originally designed, it did not arrive at its non-functional state through design, but rather through natural processes. "Pseudogenes" may indeed provide a subject for study to learn about what happens when an object has experienced both intelligent design and natural decay.

What is most revealing about all of this is that while Carroll claims ID is wrong, he treats it as if it is a testable hypothesis. For all of the talk about ID lacking a model, Carroll most certainly treats the "pre-formed" gene hypothesis like it is a testable ID-model. Unfortunately, Carroll then contradicts himself by later suggesting that ID is "not science."<sup>70</sup> I suppose ID just doesn't fit with his gospel message.

## Conclusion

---

<sup>68</sup> Carroll, *The Making of the Fittest*, at 240.

<sup>69</sup> *Id.* at 241.

<sup>70</sup> *Id.* at 245.

Sean B. Carroll's book *The Making of the Fittest: DNA and the Ultimate Forensic Record of Evolution* makes large promises but fails to deliver. He claims that science will remove "any doubt" about evolution, and he hopes his scare-tactics about a coming environmental apocalypse will convince people to just accept evolution and save the planet. But there's no valid reason to argue that one must be a Darwinist to accept responsibility for protecting the environment. As a conservationist myself, I don't need, as Carroll taunts me, to "*accept evolution or you won't 'think at all'*" in order to understand the importance of conserving our natural resources.

While Carroll is a good writer who makes science easy to understand, his book has a politically oriented *gospel message* which is simple: just believe evolution without any doubt, and we may be saved from environmental catastrophe. But Carroll's scientific arguments fail to back up his big talk. Carroll's examples of natural selection's creative power—animal breeding, peppered moths, or loss of function—fail to impress. His repetition of Darwinist urban legends about computer studies of eye-evolution and heavy reliance upon vague just-so stories about icefish give little reason to turn the head of the informed Darwin-skeptic. Carroll's discussions of junk-DNA and pseudogenes are interesting, but it is disconcerting that Carroll never mentions that his "use it or lose it" rule implies that if a stretch of DNA has not been lost, then perhaps it's still being used. His observation that widely diverse organisms often use the same or similar proteins only serves to further confirm my suspicions of common design in biology. Incredibly, Carroll uses these examples to claim he has "eviscerated" intelligent design. The ID-proponent who reads this book will feel very encouraged about the strength of her own position, for Carroll failed to provide any compelling explanations for the primary subject of his book: *the evolutionary making of the fittest*.