Florid forensic fable

A review of
The Making of the Fittest: DNA and the Ultimate Forensic Record of Evolution
by Sean B. Carroll
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Sean Carroll has emerged in the last two years as as the premier next-generation apologist for evolution, following the likes of Gould and Dawkins. He has by no means won the fight (though he claims to have) but he unveils the latest research in easy to understand language, and makes it a powerful (superficial) argument in favour of evolution. However, he makes numerous logical blunders, and his claims to 'victory' are largely bluff. Creationists need to read his evidences and be prepared to refute them. There is a bewildering new array of fascinating molecular detail out there to learn, and to deal with.

Introduction

'Of all the scientists in the world today, there is no one with whom Charles Darwin would rather spend and evening than Sean Carroll,' according to philosopher of science Michael Ruse on the back cover. I agree. His ground-breaking book from 2005, *Endless Forms Most Beautiful: The New Science of Evo Devo,* is still on my desk from last year's review,[1] because it is so accessible and so full of useful information. *The Making of the Fittest* will surely be remembered for years to come as the book that turned neo-Darwinism from a theoretical exercise into a practical molecular 'reality'. I suspect that many lay people who read this book will sadly be converted to his cause.

Contents

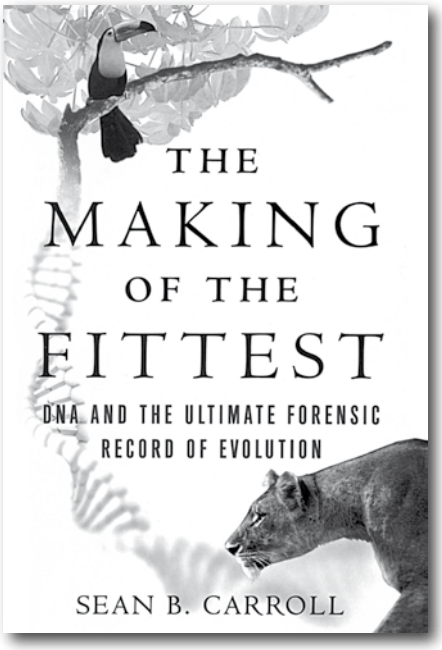
He opens with a moving tale of the power of DNA analysis of crime scenes to exonerate the innocent and convict the guilty, and then argues that it can do the same thing for the history of life. His three-fold aim in the book is to illustrate the grandeur of evolution, to focus on the best examples of the evolutionary process, and to 'vaporize' the arguments of anti-evolutionists 'beyond any reasonable doubt' (p. 17).

Chapter 1: Introduction—the bloodless fish of Bouvet Island

Antarctic ice fish have lost their red hemoglobin and myoglobin because they don't need them in the below-freezing southern waters. There is far more oxygen dissolved in such cold water, and they have many cardiovascular compensating mechanisms for getting it into their bodies, and 'many more genes that have been modified so that all sorts of vital processes can occur in the subfreezing climate' (p. 25). Furthermore, they invented an 'antifreeze' glycoprotein to stop their blood (and thus body) freezing solid. This new invention, we are told, was the result of some random 'tinkering' with an old gene. So, by 'an improvised series of many steps, including invention of some new code, the destruction of some very old code, and the modification of much more' the 'icefish has managed to change its whole engine *while the car was still running* [emphasis in original]' (p. 26).

Chapter 2: The everyday math of evolution—chance, selection and time

A brief history of genetics and Darwinism is complemented with the algebra of compound interest, selection coefficients, population sizes, mutation rates and generation times. He asserts that all mutations are random, and that



Kimura's neutral theory of evolution provides a baseline for how DNA should change over time if no other forces (e.g. natural selection) are at work.

Chapter 3: Immortal genes—running in place for eons

Discovery of the three domains of life (Archaea (the extremophile bacteria), Bacteria, Eukaryotes (everything other than bacteria) and their 500 genes in common, revolutionized our view of evolution. Theoretically, mutation should obliterate any vestige of ancestral relationships after about 100 Ma, but the earliest life emerged 2 (or 3) Ga ago. The answer can only be

> 'Natural selection. There is no other explanation' (p. 81).
> 'This pattern of the strong preservation of the protein sequences at most sites … in any group of species … is the predominant pattern of evolution in the DNA record' (p. 83).

Chapter 4: Making the new from the old

Tri-color vision is said to have arisen via duplication of an opsin gene in organisms with bi-color vision, followed by fine-tuning of the extra

opsin molecule to the frequency of red light. It is so easy to do (he implies) that in some cases only one amino acid needs changing. 'Gene duplication is one important way in which information is increased in DNA' (p. 97). Accidental insertions of 'junk DNA sequences near genes' (LINES—long interspersed elements, and SINES—short interspersed elements) are 'perfect tracers of genealogy' because 'there is no active mechanism for removing them' and 'their presence in the DNA of two species can be explained only by the species sharing a common ancestor' (p. 99).

Chapter 5: Fossil genes—broken pieces of yesterday's life

When genes fall into disuse they quickly decay by mutation because natural selection is no longer weeding out the mutants. Carroll shows how corrupted opsin genes provide a 'fossil record' of past evolutionary events, and cites both the coelacanth and the cetaceans (whales) having dispensed with color vision in the deep sea. These independent events (whales are closer to hippos than to fish) occurred on the same gene, and in fact it also happened in owl monkeys, slow loris, bush baby and blind mole rat, and we know this because each species' lesion is in a different place in the 'fossil' gene. Humans are not immune—about half of our olfactory receptor genes have become fossilized (p. 128). But the champion fossilizer is the parasitic leprosy bacterium which has 1,600 functional genes and almost 1,100 fossil genes—compared with its free-living cousin the tuberculosis bacterium which has about 4,000 intact and only about 6 fossil genes (p. 131). Fossil genes, he concludes, 'are powerful arguments against design' (p. 136).

Chapter 6: Déjà vu—how and why evolution repeats itself

Howler monkeys are the only New World monkeys with tri-color vision, and it evolved independently of the Old World primates which all have tri-color vision.

> 'We know this because the size of the DNA region that was duplicated was different in each event … [and] … In all Old World primates the texts of the two opsin genes differ by more than 5%, while in the howler the two genes differ by just 2.7%. This indicates that the duplication of the howler genes occurred more recently than the duplication of the Old World genes [which] is consistent with geological evidence of the more recent evolution of New World monkeys' (pp. 144–145).

Numerous other examples of 'convergent evolution' are given and then the argument is sealed with calculations of mutation rates and population sizes showing that 'given sufficient time, identical or equivalent mutations will arise repeatedly by chance, and their fate … will be determined by [natural] selection' (p. 155).

Chapter 7: Our flesh and blood—arms races, the human race, and natural selection

At first I was puzzled by Carroll's choice of human diseases as his 'conclusive proof' of human evolution. I then realized, at the genetic level, that is the *only* evidence available! His prize exhibit is sickle cell anemia having arisen at least five separate times in human populations, and its benefit to carriers in reducing mortality from malaria (p. 174–179).[2] Then a brief look at cancer and its genetic causes and potential cures. 'The power of these particular examples is that they run so counter to our notions of progress and design' (p. 186).

Chapter 8: The making and evolution of complexity

Eyes are easy to make, we are told. All you need is two cells to begin with (a light sensitive pigment cell and a photoreceptor cell) plus a 500-Ma-old gene called Pax-6. Then 'complexity in this case is a matter of just arranging larger numbers of the same types of eye cells in three-dimensional space—the same building materials [in each case, but] a different organization' (pp. 193–203). The threespine stickleback fish in North America has evolved from the spiny marine form to the spine-reduced freshwater form by switching off the *Pitx1* gene during pelvis development. Then the *pièce de resistance*—how the fruit fly got its spots. 'A seemingly endless variety of patterns can be generated using the same tool kit of body-building and body-painting genes, by tinkering with genetic switches' (p. 210). He then deals his death blow: 'The argument for design by some external intelligence is eviscerated' (p. 212).

Chapter 9: Seeing is believing

Carroll now moves away from science into psychology, because 'the reasons for doubt [anti-evolutionism] could not be, and are not, scientific' (p. 213). They lie in the reluctance of people in general to believe in what they cannot see, as illustrated historically by (a) resistance to the germ theory of disease, (b) Lysenko (and Soviet) rejection of genetics, and (c) chiropractic rejection of vaccination. Then, using six lessons from these examples, he rips into anti-evolutionists. Henry Morris and Ken Ham feature, but only as quotable stooges. And there is a separate section on Michael Behe and the Intelligent Design movement, which he dismisses because 'It has produced no insights into any scientific question and it is inconsistent with rigorously tested knowledge' (p. 245).

Chapter 10: The palm trees of Wyoming

In this final chapter, he addresses the question of why evolution matters. The tragic tale of the recent collapse of the cod fishing industry introduces this lament: 'A perfect storm is brewing—of overfishing, pollution, and man-made climate change—that threatens to extinguish ecosystems beyond any chance of recovery' while 'we are in massive denial' and 'still debating the existence of evolution' (pp. 263–268).

There are 15 pages of source references and further reading, and an ample index.

Discussion

Carroll's scientific authority and his clear and persuasive writing style will likely convince all but the vigilant. But here are some of the 'holes' in his arguments.

The 'forensic science' fallacy

His opening equation between DNA evidence for evolution and DNA evidence in forensic work is a fundamental error of logic that permeates the whole book. The only thing that forensic DNA analysis can 'prove' is whose DNA was found at the scene—or even more narrowly, whose DNA was present in the laboratory at the time of testing. There are always other possible reasons for a person's DNA being present. Clever thieves may plant false DNA evidence. Corrupt officials can switch samples. Technicians can bungle the crime scene investigation, sample preparation, sample handling and/or DNA analysis.

Fundamental realities in forensic science are crucially absent in evolutionary studies. In forensic work a crime *has* occurred, while evolution is only an *assumption* of the evolutionist. In forensic work there is a co-incidence in time that allows for cause-and-effect argument—the offender is (or was) alive and active at the time the crime was committed. In evolution, time frames, gaps in the record and phylogenetic reconstructions have potential errors of millions of years so there can be no certainty to support a cause-and-effect argument. Juries, lawyers, witnesses and confessions have all worked towards a consensus in forensic work that DNA analysis is (usually) effective, but no such corroboration is possible in evolution because there were no witnesses and no suspect to confess or describe the events. Forensic work always uses much more than DNA evidence, so Carroll is inconsistent in pinning all his hopes on DNA evidence for evolution.

The 'assumption-of-evolution-to-prove-evolution' fallacy

Without even mentioning the problem of circular reasoning, Carroll again and again presents evolutionary trees as the basis for his conclusions about evolution. Moreover, all of the DNA evidence he gives is interpreted in the framework of cladistics, a method of evolutionary tree generation that assumes evolution has occurred.

The fallacy of affirming the consequent

Carroll's evidence is presented in the logical format 'evolution implies *A*, and *A* is observed, therefore evolution is true.' This is the 'fallacy of affirming the consequent'[3] and is invalid because other causes (which he never investigates) could potentially produce the same result. At no point does Carroll attempt to engage with creationist or intelligent design reasoning regarding the nature or structure of life. All of his evidence is used to affirm evolution and then he simply ridicules design ideas as an afterthought.[4]

The 'every-change-is-a-random-error' fallacy

Carroll affirms on many occasions that all of the changes he cites were the result of random copying errors. At no point does he consider that relevant changes may have been designed, or that the many enzyme-mediated changes he calls 'mutations' (as opposed to errors that occur *despite* error correction mechanisms) could have been planned as part of a natural variation mechanism. For example, antibody production involves a short section of the genome that is *designed* to mutate rapidly to sample a wide variety of possible antigens.[5]

The 'junk DNA' fallacy

Carroll asserts at the beginning of the book that most of our DNA consists of 'junk' leftovers of the past, and this forms the basis for his strong conclusions in Chapter 4. This is very surprising because several lines of reasoning infer a role in gene regulation, and the 'junk' label is (now) recognized as something of a misnomer, and many prefer the more neutral term 'non-coding DNA' According to Prof. John Mattick of the University of Queensland in Brisbane, Australia:

> '… the failure to recognise the implications of the non-coding DNA will go down as the biggest mistake in the history of molecular biology.'[6]

The 'all change is evolution' fallacy

Carroll's 'best' experimental evidence is (a) switching off the *pitx1* gene reduced pelvic spine size in threespine sticklebacks, and (b) gain and loss of wing spots in *Drosophila* is the result of 'tinkering with switches' in a pre-existing complete tool kit of body-building and body-painting genes. But (a) is *loss* of structure, and (b) requires an unexplained complex new device (gene switch). The evidence best fits Creation and the Fall!

The information bluff

On page 97 Carroll says that gene duplication increases information content in genomes, but neither here nor anywhere else does he attempt to explain or justify this crucial statement. As it stands, the statement is false,

Image by Alex Williams

Illustration of blood cells. Sickle cell anaemia is caused by a mutation in the hemoglobin molecule, which then changes red blood cells from the normal round shape (above) to the sickle shape (below). If sickled blood cells are infected with the malaria parasite, they die and kill the parasite. The mutated condition thus mitigates the severity of malaria and naturally selected for in areas of high malaria rates. As evidence for microbe-to-human evolution it is useless because it is a disease that causes loss of useful structure and function.

because a duplicated gene is like a photocopied page from a book—it is just an extra copy of the *same* information.[7] Since any theory of origins must explain the new information required to turn a microbe into a microbiologist, this attempt to 'touch on' where new information comes from is nothing more than bluff.

The oversimplification fallacy

Many of Carroll's examples imply that apparently simple changes (e.g. a single amino acid substitution can make an eye) is all it takes to initiate some profound step forward in evolution. What actually happens is that pre-existing and very complex structure and metabolic machinery includes the 'simple change' as just one of its essential parts, and without it the machinery does not work. There is also no point in changing just the absorption spectrum of the pigment without changing the processing machinery to interpret the novel signals.

Or perhaps the 'small change' initiates a re-orientation or shift in a pre-existing mechanism, as it was designed to, in order to produce useable variations. To be fair, Carroll does on *one* occasion, admit this:

> 'The dramatic effects of Pax-6—the loss of eyes when it is inactivated, thet induction of eyes where it is active—are due to its effects on many other genes and steps in development' (pp. 204–205).

However, page 204 is too late for the naïve reader because Carroll has likely won him over already with his earlier 'simple' arguments. Without the necessary background knowledge of molecular biology, the crucial and global significance of this one caveat to Carroll's argument would be lost.

Overstatement

> 'The whales, turtles, fish, crabs, and corals of the [Great] barrier reef are complex, but they all start life as a fertilized egg and in a matter of just days, weeks, or months, a complete individual, with its many complex parts, is built by processes that *we are now understanding in great detail* [emphasis added]' (p. 212).

Carroll implies that he knows how life works and can explain it all in great detail by chance, time and natural selection. He then concludes: '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.' This may persuade many readers, but in fact neither Carroll nor anyone else fully understands the life processes of a single living cell,[8] let alone a complex multi-cellular organism. Carroll knows this. And he surely knows that there is a huge difference between the fertilized egg of a complex organism that *has the information pre-programmed* to develop into the adult, and the hypothetical first living cell that would lack this.

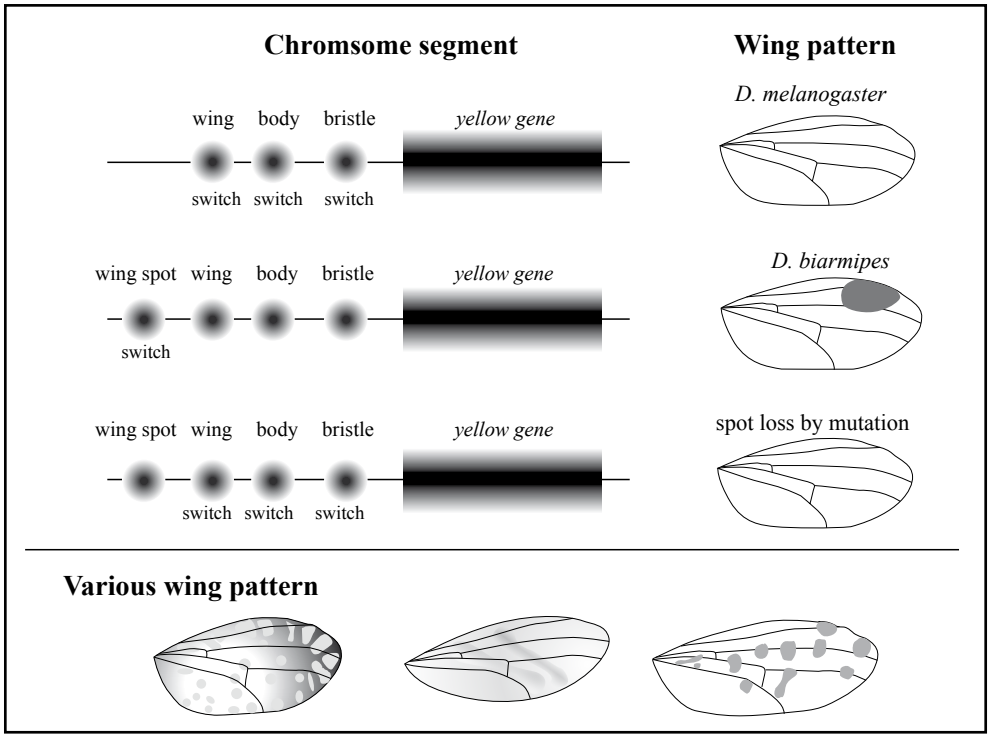


Image by Alex Williams

Wing spot development in the fruit fly *Drosophila*. The gene called *yellow* in *D. melanogaster* (top panel) is involved in three stages during development (wing, body and bristle, for each of which there is an associated switch near the gene) but there are no spots on the wing. But in *D. biarmipes* a wing spot switch has been added to the gene, and a spot develops on the wing. In some descendent species of the spotted kind, the wing spot switch is turned off by mutation and spot does not develop. Just three of many different wing patterns in the lower panel illustrate the variety that can be produced by different switching combinations amongst this and other 'paint-brush' genes. This is useless as evidence for microbe-to-human evolution because gene switches are as complex as GPS satellite navigation devices and are better explained by creation, while the malfunction in the mutated switch is clear evidence that mutations destroy rather than add new information to the genome.

Icefish illusionism

There is certainly a lot of genome decay in the loss of hemoglobin and myoglobin, so this is just more loss of information. But in the highly oxygenated water they live in, natural selection would not work against this loss. Thus it is no different in principle from examples long ago addressed by creationists, such as wingless beetles that survive on windy islands because they can't fly, so won't be blown into the sea,[9] and animals in dark caves with shriveled eyes that are less prone to damage.[10]

The antifreeze glycoprotein doesn't require high information content. Any non-volatile solute (e.g. ethylene glycol or automotive 'antifreeze' will depress the freezing point of its solvent, a well known colligative property (depending on the concentration not the nature of the solute). This fish antifreeze protein is many times more powerful because it works in a non-colligative manner by inhibiting the growth of ice crystals. It has a hydrophilic face that binds to the fastest growing surfaces of the ice crystal, and a hydrophobic surface that inhibits the approach of more water molecules.

Since half the 20 universal amino acids that comprise proteins are hydrophilic and half are hydrophobic, many proteins/peptides could act as antifreeze; they do not have to have much specificity. And many proteins exist that already have these properties—many membrane-embedded proteins, for example, are hydrophobic at the membrane end and hydrophilic on the other end. Some degenerative changes in such a protein such that it no longer embeds in the membrane, plus a loss of control over the synthesis so that large quantities are produced, could easily generate an antifreeze. So such a 'gain-of-function' change is informationally downhill, involving loss of specificity and control.

Ignorance of opposing views

If Carroll had done his homework, he would know that a crucial part of biblical creation involves the Fall, and the consequent decay of creation. Apparent 'design flaws' (e.g. decay of the leprosy genome as it became a parasite, malaria and cancer evidences) are not evidence against the biblical worldview but strong evidence *for* it.[11]

Remaining unknowns

There is only one place in the whole text where Carroll used just *one* word to hint at the great area which still remains largely unknown. Most people would, unfortunately, miss it:

> 'Complexity in this case [eye varieties] 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* [emphasis added]' (p. 197).

Consider an analogy by substituting 'metal, plastic, wire, electrons and semi-conductors' for 'eye cells'. We could use these same materials, organized in various ways, to make anything from airplanes to submarines to children's toys to high explosives. The crucial difference between all these items is the way the material is *organized*. In particular, how the wiring makes the components function. For example, a vacuum cleaner and a hair dryer consist of similar components, but one is wired up to suck, and the other is wired up to blow. All living things consist of essentially the same materials—proteins, carbohydrates, lipids, nucleic acids—the grand question of life's variety lies in the fundamentally different ways these materials are organized. At this stage, we do not have the complete 'wiring diagram' for even a single celled organism, but Carroll does not devote even *one sentence* to the topic! He simply attributes everything to evolution. Like other evolutionists before him, Carroll claims victory over the argument for design without even addressing the question.

References

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2. He ignores the fact that one of the world's leading experts on sickle-cell anemia is a staunch opponent of evolution—see Sarfati, J., Exposing evolution's icon: interview with Dr Felix Konotey-Ahulu, *Creation* **29**(1):16–19, 2006.
3. Sarfati, J., Loving God with all your mind, *Journal of Creation* **12**(2):142–151, 1998; <www.creationontheweb.com/logic>.
4. Karl Popper's logically valid method of evaluating a scientific theory is to use 'denial of the consequent'—that is, evolution (or creation) implies *A*. If *A* is shown to be *false*, then the proposition 'evolution (or creation) is true' can be credibly denied. In practice, however, this approach is rarely implemented because the creation/evolution controversy is fundamentally about presuppositions rather than evidence. If one evolutionary scenario is shown to be false then another is sought to replace it.
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