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BIOLOGY'S BLIND SPOT: TWO VIEWS ON HUMAN ORIGINS

a feature-length book review of

Ancestors in Our Genome: The New Science of Human Evolution

by Eugene E. Harris

(Oxford University Press, 2015)

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Ancestors in Our Genome: The New Science of Human Evolution by Dr. Eugene Harris, a molecular anthropologist and professor at City University of New York, is a thorough exposition of current scientific opinion concerning our evolutionary history. This book covers a difficult and technical subject, and Harris simplifies and illustrates the ideas behind the book's claims quite well; however, one assumption makes much of what he says no better than hypothetical.

The assumption that I am talking about is *common descent*—the idea that we are descended from a common ancestor going back to the dawn of life. Common descent as a theory has substantial evidentiary support, but there is also significant evidence against it. If it's wrong, everything in evolutionary biology is wrong, including current theories about human evolution.

MISLEADING FOSSILS

Harris begins his book with a chapter on fossils called "Looks Can Be Deceiving." Here he describes how apparent similarity of fossil shape or structure can be misleading and

can make fossil evidence unable to answer key questions. (Take note: we will encounter parallel arguments based on *DNA* similarity in a bit. The same caveats apply there as well as here.)

Are Any Fossils Our Ancestors? Anthropologists dispute among themselves which fossils are our ancestors or even *if* any of them are our ancestors[1] because it is difficult to determine which fossil traits are most significant. For example, the shape of the pelvis might indicate that certain fossils are closely related to us, while the shape of the teeth might point to another group of fossils.[2]

By some measures, the famous fossil "Lucy" may have walked upright, but by other measures she may have spent her time in the trees. Her diverse traits tell different stories about how she may have lived; even physical anthropologists disagree on the matter.[3] She may or may not be related to us—no one agrees.

Silent Bones. Bones can only reveal that they look alike, but not why they look alike. They might look alike because they came from the same ancestor or because they had a common lifestyle. Harris uses the example of fish and dolphins. They both have sleek bodies with fins to stabilize and tails to propel. Yet their similarity is due to environment, not ancestry. The technical term used by biologists to describe the widespread phenomenon is *homoplasy*. Homoplasy occurs at both anatomical[4] and molecular levels.[5]

As another example of homoplasy, Harris describes how baboons and mandrills were once grouped together because of the shape of their skulls. Molecular phylogenetic studies gave a different picture, however, indicating that they are more closely related to other monkeys. If the DNA evidence is correct, mandrills and baboons arrived at the same shape skulls independently.

Overlooked Problems with the DNA Data

Despite his clear statement of the problems of fossil interpretation, Harris does not see that many of the same problems apply to the DNA data. His assumption of common descent leads him and other evolutionary biologists to many *ad hoc* evolutionary explanations for how things happened. *Ad hoc* explanations are hypotheses added to a theory in order to prevent its falsification. Because common descent is assumed to be indisputably true, when contradictory evidence is found, new hypotheses—new ways of explaining the data—are added to preserve the idea of common descent. In the case of DNA comparisons, there are actually two hidden assumptions that must be

preserved. One is common descent and the other is that we are what our DNA makes us to be. What follows from these two assumptions is the conclusion that DNA similarity indicates common descent.

For mandrills and baboons, when the similar shape of their skulls disagreed with the DNA data, the two assumptions were preserved by pitching out the skull data and claiming that baboons and mandrills arrived at their skull shapes independently. The DNA data had to take precedence.

Mixed-Up DNA. Harris reports that about 30 percent of our DNA shows similarity to gorilla DNA, 1 percent orangutan DNA, and 60 percent chimp DNA. This was not expected. Scientists argued *ad hoc* that orangutans, chimps, gorillas, and humans came from a large population of ancestral apelike creatures that gradually separated into four species.[6] This argument was necessary to preserve the ideas of common ancestry and DNA similarity as a signal of common ancestry. We couldn't share DNA with chimps, gorillas, and orangutans unless we came from a large population with interbreeding and time for slow separation of the population into distinct species. The alternate hypothesis, that chimps, gorillas, orangutans, and humans have unique origins with different amounts of shared DNA for functional reasons, could not be considered without tossing out the assumption that DNA similarity proves common descent.

Estimates of the size of that proposed original group range from five to fifty or sixty thousand individuals.[7] The calculations are done by comparing sequences from multiple versions of the same DNA and modeling their evolutionary history. This can be done only if initial assumptions, including common descent and DNA similarity as its signal, are true and certain other conditions concerning our early history are met.[8]

These estimates of population size matter because a large population size at our origin is the main argument used against a historical Adam and Eve—a point of great significance. But the estimates also matter because they affect the science—their consequences flow downstream.

Population Size and Natural Selection. According to population genetics, four forces shape us—mutation, recombination, random genetic drift, and natural selection (see the glossary for definitions).[9] Only natural selection is directional—it tends to preserve good traits and discard bad ones. All the other forces act without regard for what's good or bad.

In small populations, random genetic drift overwhelms natural selection, while in large populations, the reverse is true. Imagine a population of a hundred rare birds—

a single disease or bad winter can wipe them out. It's not because they aren't fit—just unlucky. In contrast, one single event is unlikely to kill all in a population of several million.

Here's the surprise: the math of population genetics says that the human population would have to be very large—larger than our human population historically has been—for natural selection to have shaped us instead of random genetic drift.[10] Chance events—accidents of fate—determine which weakly beneficial mutations become universal (fixed) in our population and which weakly deleterious mutations are lost over the course of time.[11] As a consequence, natural selection cannot be invoked as the cause of most things in our evolution, especially things that require many mutations before a benefit is achieved. Many evolutionary biologists, including Harris, forget this and attribute everything from our hairlessness to our large brains to natural selection.[12] Population genetics says the opposite: we evolved because of random events—luck—that just happened to lead to creatures like us. Chance seems entirely implausible, though, as an explanation for our existence, so alternate explanations that do not rely on either natural selection or chance are necessary.

Homoplasy. The expectation based on common descent is that gene and species trees should show the same relationships. Yet gene trees drawn often disagree with species trees—a version of molecular homoplasy that is common. We call such trees discordant. Evolutionary scientists offer an *ad hoc* explanation: variant genes in a large population that predate speciation can be inherited independently of speciation. This is similar to the explanation for why we had to come from a large initial population.

Imagine having a large bag[13] of marbles (genes) with different colors—cat eyes, bluesies, aggies, spirals, and so on. The players (species) pick two at random from the bag of marbles. Marbles and players will have no particular pattern of inheritance. Alternatively, if each player (species) starts with his own distinct kind of marbles (genes) but then plays games (exchanges genetic information) with other players, relationships between marbles and players will be scrambled.

If common descent is true, we expect genes to have the same tree of relatedness as the species they come from. This expectation is not met in many cases: gene and species trees are discordant. Whether or not the explanations given above are true is not known—they have been advanced ad hoc to protect the idea of common descent. An alternative not requiring common descent is also possible—genes were assigned in the beginning to different species according to need (design).

The remainder of the book discusses how, when, and where we originated.

Who? A number of fossils have been found with varying similarity to us, starting with *Sahelanthropus*, then progressing to *Australopithicus*, *Paranthropus*, *Homo ergaster*, and *Homo erectus*. Are any of these creatures related to us? Are they even related to each other? Harris gives a tidy tree showing our relationship to the species classed as Homo, avoiding the mess that precedes the Homo lineage. Still, as he admits, any tree of relatedness for these fossil creatures is hypothetical because fossils are poor sources of information.[14]

When and Where? In current scientific thought, modern man (people with skulls like ours) appeared about 100,000 to 70,000 years ago, probably in Africa. His culture advanced rapidly. By about 40,000 years ago, he was making artistic cave paintings in Europe and sophisticated tools. Definitely human.

How? How did we evolve large brains? How did we lose our body hair? How have we acquired the diseases we are afflicted with? The book contains interesting speculation about these topics and more. I give Harris credit for acknowledging where the arguments are conflicting or weak. In particular, he admits that there is no direct evidence that natural selection is responsible. Explanations of our origin using natural selection are therefore *ad hoc*.

Designed Arrival. In fact, we simply *could not have evolved* in the time scientists assign to our split from chimps, about six million years ago. Many coordinated changes are required to turn an ape into a human. Harris acknowledges, "With complex features like skeletal traits, we expect there to be many DNA changes responsible for them, and we expect that these DNA changes will not have occurred only in a single gene but in numerous different genes."[15]

Most scientists believe that the important mutations occurred in DNA sites that regulate how much product genes make. From Harris: "We have also learned from laboratory studies that *genetic alterations* [mutations] underlying complex and continuously varying traits *often occur within regions of the genome that regulate how genes are expressed*, rather than within the protein-coding portions of genes [emphasis added]."[16]

Mathematicians Richard Durrett and Deena Schmidt calculated that to get a *single mutation in such a regulatory site* would take six million years,[17] as much time as we've got to evolve from ape to us. Even if every gene evolved simultaneously, all those mutations would need to work together to create helpful traits. Since mutations are blind to benefit, mostly harmful, and typically work against each other, there simply isn't enough time for it to have happened.[18]

What Now? Where does this leave Adam and Eve? The short answer is the science is so uncertain that an original first pair cannot be ruled out. In fact, a strong case can be made that we didn't evolve at all, which makes the question of our first parents easy to answer.

Is common descent true? There are enough arguments against common descent that the scientific community needs to acknowledge there is a problem. It matters tremendously because without common descent, evolutionary theory implodes.

In summary, the book is a good review of current scientific literature concerning our origins, but with a blind spot. If assumptions of common descent and the power of natural selection are false, if problems of homoplasy and discordant gene and species trees are common, and if estimates of how long it would take to get a single regulatory mutation are true, then the ancestors hidden in our genome may not be the ancestors Harris thinks they are.

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GLOSSARY OF TERMS

allele: a particular version of a gene.

gene: DNA that specifies how to make something.

genetic drift: gain or loss of alleles because of random events.

genome: the entire complement of an organism's DNA.

homoplasy: the phenomenon when two unrelated things look alike.

mutation: a change to the DNA.

natural selection: the survival of organisms with beneficial alleles is favored over others.

phylogenetics: the study of evolutionary relatedness using DNA comparisons.

population genetics: the study of phenomena affecting gene frequencies in populations.

recombination: shuffling the DNA into new combinations of alleles.

NOTES

1 Ann Gauger, Douglas Axe, and Casey Luskin, *Science and Human Origins* (Seattle: Discovery Institute Press, 2012), chap. 3.

2 Ibid.

3 Eugene E. Harris, *Ancestors in Our Genome: The New Science of Human Evolution* (Oxford: Oxford University Press, 2015), chap. 1; Gauger, *Science and Human Origins*, chap. 3.

4 Harris, *Ancestors in Our Genome*, chap. 1; David B. Wake, Marvalee H. Wake, and Chelsea D. Specht, "Homoplasy: From Detecting Pattern to Determining Process and Mechanism of Evolution," *Science*, February 2011, 1032–35.

5 Antonis Rokas and Sean B. Carroll, "Bushes in the Tree of Life," *PLoS Biology* (2006) 4:e352. Accessed March 19, 2015, doi:10.1371/journal.pbio.0040352; Antonis Rokas and Sean B. Carroll, "Frequent and Widespread Parallel Evolution of Protein Sequences," *Molecular Biology and Evolution* 25 (2008): 1943–53.

6 Harris, *Ancestors in Our Genome*, chap. 2.

7 Ibid., chap. 3.

8 P. Sjödin et al., "On the Meaning and Existence of an Effective Population Size," *Genetics* 169 (2005): 1061–70.

9 Michael Lynch, "The Frailty of the Adaptive Hypothesis for the Origins of Organismal Complexity," *Proceedings of the National Academy of Sciences USA* 104 (May 2007): 8597–8604.

10 Michael Lynch, "Rate, Molecular Spectrum, and Consequences of Human Mutation," *Proceedings of the National Academy of Sciences USA* 107 (January 2010): 961–68.

11 Strong selection for a particular mutation in one part of the world can lead to local predominance of that mutation, but it is highly unlikely for it to become fixed in the population as a whole, unless the strong selection persists and is universally beneficial. Examples are sickle cell trait and lactose tolerance, both of which provide local benefits but not universal ones, and therefore neither is universally present in our genomes.

12 Harris, *Ancestors in Our Genome*, chap. 7.

13 If the bag is small, the chances of a player drawing the same color twice are increased.

14 Harris, *Ancestors in Our Genomes*, chap. 7.

15 Ibid., 59.
16 Ibid., 109.
17 Richard Durrett and Deena Schmidt, "Waiting for Regulatory Sequences to Appear," *Journal of Applied Probability* 17 (2007): 1–32.
18 Gauger, *Science and Human Origins*, chap. 1.