CRI
CHRISTIAN RESEARCH INSTITUTE
PO Box 8500, Charlotte, NC 28271

Feature Article: **JAF9351**

JUNK DNA: EVIDENCE FOR EVOLUTION OR DESIGN?

by Fazale Rana

This article first appeared in the CHRISTIAN RESEARCH JOURNAL, volume 35, number 01 (2012). For further information or to subscribe to the CHRISTIAN RESEARCH JOURNAL, go to: http://www.equip.org/christian-research-journal/.

My wife enjoys looking for antiques (and, to my horror, buying them). For Amy, antiques are priceless treasures. For me, these relics from the past are just old junk.

Traditionally, biochemists thought a vast proportion of most organisms' genomes consisted of DNA sequences that once had value but eventually decayed into nonfunctional DNA elements.

According to this view, undirected biochemical processes and random chemical and physical events transform functional DNA into useless "junk." These nonfunctional elements often bear structural similarity to the functional counterparts from which they putatively originated, lending support for this mechanistic account of their origin.

For skeptics, the existence of noncoding (junk) DNA is incompatible with God's direct involvement in the origin of life. Evolutionary biologists consider it one of the most potent pieces of evidence for biological evolution.[1]

Evolutionists highlight the fact that in many instances identical (or near identical) segments of junk DNA appear in a wide range of related organisms. Frequently the identical segments reside in corresponding locations in these genomes.

According to the evolutionary perspective, shared junk DNA sequences among related organisms would be expected if the junk DNA segment arose prior to the time that the organisms diverged from their shared evolutionary ancestor. Junk DNA remains part of an organism's genome solely because of its attachment to functional DNA. In this way, the molecular artifact persists from one generation to the next.

Skeptics, then, rightly wonder why a Creator would purposely introduce nonfunctional, junk DNA at the exact same location in the genomes of different, but seemingly related, organisms.

PREDICTED FUNCTION FOR JUNK DNA

Until the past few years, creation/intelligent design proponents were unable to offer a robust response to this legitimate challenge. Yet many, myself included, felt confident that science would eventually uncover functional significance for junk DNA sequences.

In the past half-decade or so, this prediction has been satisfied. Molecular biologists and geneticists have discovered function for virtually every class of junk DNA. My books *Who Was Adam?* (NavPress, 2005) and *The Cell's Design* (Baker, 2008) describe the importance of all the various types of junk DNA. Here I will focus on one of the more significant examples: functional pseudogenes.

PSEUDOGENES

Evolutionary biologists consider pseudogenes the dead, useless remains of once-functional genes. According to this view, severe mutations destroyed the capacity of the cell's machinery to read and process the information contained in these genes. Still, pseudogenes possess the telltale signatures that allow molecular biologists to recognize them as genes, albeit nonfunctional ones.

Experts recognize several classes of pseudogenes. Duplicated pseudogenes are the largest class. Scientists posit that these DNA segments arose when the gene(s) underwent duplication in the genome, after which the duplicated copies experienced severe mutations, rendering them unrecognizable as a functional gene by the cell's machinery. Loss of duplicated gene function has little, if any, effect on an organism's fitness since an intact copy of the functional gene still exists.

FUNCTIONING PSEUDOGENES

About five years ago, molecular biologists began to suspect that pseudogenes play a vital role in regulating gene expression.[2]

A recent study confirmed this suspicion by demonstrating how pseudogenes control gene expression by acting as decoys that protect gene products from destruction.[3]

The researchers who made this discovery focused on PTEN, a gene that suppresses tumors. When this gene is functionally impaired, it leads to the onset of several different types of cancer. The same result occurs if mutations arise in PTEN's corresponding pseudogene, PTENP1. This finding means that the PTENP1 pseudogene must be functional.

Researchers discovered that expression of PTEN can be reduced by small RNA molecules (miRNA). The tiny molecules bind to messenger RNA produced from PTEN. The miRNA then directs the breakdown of the messenger RNA, and prevents the PTEN protein from carrying out its cellular role. Left unchecked, the miRNA molecules would direct the breakdown of most of the PTEN messenger RNA, thus suppressing the activity of PTEN gene products and leading to the onset of cancer.

However, the similarity between PTEN and PTENP1 allows the PTENP1 messenger RNA to also bind miRNA molecules. This means PTENP1's messenger RNA breaks down instead of PTEN's. In other words, the gene product of the PTENP1 pseudogene operates as a decoy, which allows for the appropriate levels of PTEN messenger RNA's production.

The researchers also uncovered similar relationships for a number of gene/pseudogene pairs, including the KRAS gene and KRAS1P pseudogene, which are also implicated in the invasion of certain cancers.

Advocates of the evolutionary paradigm offer this rejoinder: yes, some pseudogenes are functional, but the vast majority lack function; they are junk. These proponents maintain that a small number of pseudogenes acquire function through a process called neofunctionalization. According to this idea, evolutionary mechanisms occasionally generate functional pseudogenes after the genes initially arise through mutational events.

Molecular biologists from China recently discovered a large number of functional pseudogenes in *Trypanosoma brucei*, a parasitic protist (a single-celled organism) that causes sleeping sickness in humans.[4] This organism has about nine thousand genes and about nine hundred pseudogenes in its genome. Nearly all of the pseudogenes, it seems, pair to an intact gene and regulate its expression.

The molecular biologists' work indicates that the occurrence of functional pseudogenes is widespread among all eukaryotic organisms (i.e., organisms whose cells possess a clearly defined nucleus, among other things) because, from an evolutionary standpoint, protists anchor the evolutionary tree that led to plants, fungi, and animals. Therefore, if functional pseudogenes are present in protists, they must be present in all eukaryotic organisms.

PSEUDOGENES AND THE CASE FOR INTELLIGENT DESIGN

The widespread occurrence of functional pseudogenes and the likely universal distribution of functional pseudogene decoys among eukaryotic organisms make it difficult to believe that neofunctionalization (i.e., a gene's evolutionary acquisition of a novel function) can explain the emergence of pseudogene function. It is one thing to say that pseudogenes occasionally acquired function via undirected natural processes. But it is another thing entirely to say this happened over and over again, until virtually every pseudogene in the genome possessed function.

The importance of functional pseudogenes (as well as other classes of DNA) undermines the best argument for evolution. If functional, then junk DNA sequences in genomes do make sense from a design standpoint. So, too, does the corresponding location of junk DNA sequences in the genomes of related organisms. The common location likely reflects functional significance and could be understood as reflecting shared design instead of shared ancestry. In the case of pseudogenes, similarity to the "real" gene could be understood to reflect its functional role, not necessarily its evolutionary origin. Like an antique hunter finding a priceless treasure at a yard sale, biochemists have found that these tiny biological components are extremely valuable molecular treasures.

Fazale "Fuz" Rana, Ph.D., is the vice president of research and apologetics at Reasons to Believe. He has authored and coauthored the books *Origins of Life* (NavPress, 2004), *Who Was Adam?* (NavPress, 2005), *The Cell's Design* (Baker Books, 2008), and *Creating Life in the Lab* (Baker Books, 2011).

NOTES

1 For example, Edward E. Max, "Plagiarized Errors and Molecular Genetics," Talk Origins, http://www.talkorigins.org/faqs/molgen/ accessed October 10, 2011.

2 Deyou Zheng and Mark B. Gerstein, "The Ambiguous Boundary between Genes and Pseudogenes: The Dead Rise Up, or Do They?" *Trends in Genetics* 23 (2007): 219–24.

3 Laura Poliseno et al., "A Coding-Independent Function of Gene and Pseudogene mRNAs Regulates Tumour Biology," *Nature* 465 (2010): 1033–40.

4 Yan-Zi Wen et al., "Pseudogene-Derived Small Interference RNAs Regulate Gene Expression in African *Trypanosoma brucei*," *Proceedings of the National Academy of Sciences, USA* 108 (2011): 8345–60.