

Are Some Mutations Directed? A Response to “From Atoms to Traits” in *Scientific American*

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Scientific American has devoted the first issue of 2009 to promoting evolution as the “most powerful idea in science.” The cover lists “How life invents complex traits” as one of the topics covered. In “From Atoms to Traits,” David M. Kingsley describes the molecular basis for a number of different traits.¹ Does the evidence he presents really support the notion that life arose by chance and has developed through random mutations in DNA?

What Is Evolution?

Darwin proposed that variations arise in organisms and natural selection tends to weed out less desirable traits. Over time this process may produce new species. Darwin contrasted this to the unbiblical idea of species fixity. If this was all there was to Darwin’s theory, there would be no controversy. After all, biblical creationists believe in speciation. For example, from the two equines on the Ark, we now have donkeys, zebras, and horses.

However, Darwin was also proposing that all life on earth descended from one or a few original forms that began to change and diversify by completely natural means. Thus, the organs (for example, heart, brain, and eyes), structures (for example, limbs and digits), and complex biochemical pathways seen in animals today are believed to have arisen gradually over time by natural processes from an original organism that lacked them. This is in contrast with the biblical account that states God created all life according to their kinds and humans distinct from all other animals.

Kingsley’s article follows the typical pattern of promoting evolution: the concept of genetic change over time is automatically assumed to support the idea that all life has arisen through natural processes. Yet change alone does not support the idea that organs, structures, and complex biochemical pathways can arise without an intelligent source. One must examine the patterns of genetic change to determine if they best fit the biblical creation or evolutionary model for the origin of life.

What Pattern of Change Are We Looking For?

The evolutionary model requires the overall pattern of change to be “upward and onward” or information-gaining. Without this strong overall direction, evolution cannot truly account for the origin of complex traits.² Kingsley states that “[o]nly a tiny fraction of these changes are likely to improve, rather than degrade, the original hereditary information and the trait that derives from it.” This is a serious problem for evolutionists, since the changes are not going the right direction. Evolutionists tend to believe that natural selection can overcome this problem, yet in all the examples given, which would have been subject to selection, there is no clear example of an information-gaining mutation.

The biblical creation model allows for several different types of changes. Since mankind sinned, suffering and death has entered the world.³ The whole world is described as being in bondage to decay, so degenerative changes can certainly occur.⁴ Mutations that cause disease are the most obvious example of this.

However, a second type of change is consistent with the biblical record. God is a provider who cares for His creation even in its present fallen condition.⁵ He created life to fill the earth and intends for the earth to be inhabited.⁶ Therefore, providential changes may also occur.⁷ These may include adaptive changes as illustrated by the example of stickleback fish, which have “evolved myriad forms to suit diverse environments” in a relatively short period of time.

The example of humans developing lactose tolerance also fits very well within the creation model. Originally humans were designed to produce lactase, the enzyme necessary to digest milk, only during early childhood. This excellent design prevents wasteful production of the enzyme during adulthood. However, the lactase gene was designed to allow for change. Kingsley explains how mutations in the regulatory region of this gene have allowed humans to develop cultures where milk consumption continues in adulthood.

Providential changes include examples where plants or animals can become more productive for the benefit of mankind. Kingsley gives an excellent example in his discussion of how maize differs from its weedy ancestor, teosinte. Again, the plant was designed to vary in ways that allowed for a few changes to produce significant practical results. Other changes can add beauty or variety, such as changes in pigment genes. Yet in all these providential changes, there is no pattern of information building on the molecular level. Instead, we see some genes were designed to allow for disruptions that have potentially beneficial results.

What Causes the Changes?

Because of the atheistic assumptions (that is, life arose by natural processes, not by an intelligent designer) of evolution, Kingsley portrays these genetic changes as being errors or accidents. The most widely known mutations are associated with disease, so many people have accepted accidents as the explanation for all mutations. Yet as detailed DNA sequence data rapidly accumulates, it is clear that many genetic changes do not directly cause disease.

There were some early experiments with bacteria that suggested some mutations arise in a statistically random pattern. However, statistical randomness is used by computer programmers in applications to minimize worst-case scenarios; so, such patterns cannot be invoked to imply that there was not an intelligent designer. A recent article by a creationist programmer discusses how statistically random variation may provide for the survival of life in adverse environments.⁸

Creationists are not confined to the idea that all mutations must be accidents or happenstance occurrences. Research in an important pigment gene, the melanocortin 1 receptor (MC1R) gene, suggests that some genetic changes in animals may be directed. One example involves repeated nucleotides in the DNA. Kingsley describes similar patterns as “very prone to copying errors during the process of DNA replication.” In the MC1R gene, these patterns appeared in a region prone to deletions, usually resulting in a black phenotype.⁹ Since similar patterns of rearrangement and mutations appear in very diverse animals and the changes in the gene produce interesting variety, it is easy to question whether these changes are really purely chance events acted on by selection.¹⁰ It has been suggested that these repeat patterns may actually format the genome for future potentially adaptive changes.¹¹

One of the most difficult patterns for evolutionists to explain by purely naturalistic processes is one that involves human skin color. Humans with ancestry farther from the equator typically have fairer skin. Much of this is the result of mutations in the MC1R that impair the signal for production of the darker form of melanin. As Kingsley mentions, lighter skin tones in humans allow for more UV light absorption required for vitamin D synthesis. This is an advantage at higher latitudes where sun exposure is more limited. There are over 60 variants in this gene in humans, and they vary in how strongly they impair MC1R function. However, this impaired function is not an advantage near the equator where sun exposure is higher and a darker complexion gives some protection against several forms of skin cancer.

Given that mutations in the MC1R gene are so common in humans overall, it was surprising to researchers that out of more than 100 Africans tested, none carried a mutation that changed the amino acid sequence of the MC1R.¹² The statistical tests they used suggested that very strong selection had occurred; so, the researchers claimed natural selection was a plausible cause for the pattern.¹³

The problem is that mutations in this gene are very unlikely to result in the death of a person before they have children. The average age of onset for malignant melanoma, the deadliest of the skin cancers, is 57 years. Not all people carrying MC1R mutations will develop skin cancer, though it would certainly be more common near the equator with the greater sun exposure. This glaring discrepancy again suggests that it is time to question the underlying assumption that mutations are essentially random. It appears more plausible that certain environmental clues may bias the appearance of mutations so that people and animals can adapt to new environments while minimizing the risk of disease.

Does Evolution Explain the Origin of Anything?

The types of changes we see may explain the origin of species within created kinds, but they certainly don't explain the origin of life by naturalistic processes. There is still no plausible explanation for a naturalistic origin of complex biological molecules, the code used in living things, or the information encoded on the DNA.

As discussed here, many genes are designed to allow for potentially useful mutations and some mutations appear to be directed. All of these observations are compatible with the biblical concept of life having an infinitely wise Designer. So, while scientific investigation has certainly shed light on how things change, only the Bible sheds light on their ultimate origin. Clearly it is God who designed complex traits and enables these traits to vary so that the earth will be inhabited as He created it to be.

Footnotes

1. Kingsley, D.M., 2009. From atoms to traits. *Scientific American* **300**(1):52–59.
2. Spetner, L., 1998. *Not by chance! Shattering the modern theory of evolution*, pp.70–74, 128–160. New York: The Judaica Press.
3. Genesis 3.
4. Romans 8:20–21.
5. Psalm 147:8, 9; Matthew 6:25–34.
6. Genesis 1:20–22, 24–28; 8:15–19; Isaiah 45:18.
7. The term *providential change* is not meant to necessarily imply a miracle *per se*. The mere fact that creatures were designed to respond to their environment in a way that promotes survival is considered providential. This is in contrast with the atheistic notion that such abilities could arise by chance.
8. Bartlett, J., 2008. Statistical and philosophical notions of randomness in creation biology. *Creation Research Society Quarterly* **45**(2):91–99.
9. Deletions in this region result in a black animal in jaguarundi, golden-headed lion tamarin, and jaguar, but not in mustelids. It is curious how these deletions could be common in mustelids even though there is no obvious phenotype that can be selected for. Even more curious is that these are all “in frame” deletions, which should account for no more than a third of all deletion events. Since loss of function mutations are not lethal and do exist in some populations, natural selection does not appear to be able to explain this biased pattern.
10. Actually, it is the same changes occurring in mustelids and not being associated with an obvious selectable phenotype that argues strongly against “natural selection acting on random mutations” as accounting for the pattern. Instead, there appears to be an inherent bias toward certain types of changes, even in the mustelids where these changes don’t alter phenotype as expected. See above footnote.
11. Lightner, J.K., 2008. Genetics of coat color I: The melanocortin 1 receptor (MC1R). *Answers Research Journal* **1**:109–116.
12. There were actually five haplotypes that involved silent, third base pair substitutions in these African populations. This variability in the absence of any nonsynonymous changes would be expected only if the mutation is under strong selection pressure.
13. Harding, R.M., E. Healy, A.J. Ray, N.S. Ellis, E. Flanagan, C. Todd, C. Dixon, A. Sajantila, I.J. Jackson, M.A. Birch-Machin, and J.L. Rees, 2000. Evidence for variable selective pressures at MC1R. *American Journal of Human Genetics* **66**:1351–1361.