

New and Improved?

Is belief in beneficial mutations scientific?

For years, engineers who designed Mercedes-Benz cars refused to offer electric windows as an option to standard hand crank windows. In characteristic German fashion, they argued that the simple structure of the hand crank mechanism functioned perfectly for the task, had few parts, and almost never needed service. By contrast, the switches, wiring, and electric motor were overall heavier, more complicated, and less reliable. In the end, it was the expectation of the consumer, not the engineer, which drove the adoption of the more complicated electric window design.

By contrast, the driving force for the incredible increase in complexity and genetic information that would have accompanied the evolution of the first living cell into plants, animals, and, eventually, people is not at all clear. Natural selection is not the driving force because selection and chance events act to reduce, not increase, the genetic variation in a population. Nor does selection create information. So where could new and improved genes, proteins, and structures come from?

Kenneth Miller, author of *Finding Darwin's God*¹ says that evolution does not start from scratch but from existing genes which are shuffled, spliced, duplicated, inverted, and mutated in random ways. These new genes may or may not be an improvement on the old genes. If selection judges the genes to be both new and improved, the organism survives with more genetic material and possibly a more complex structure. Simple. So simple, in fact, that Miller claims that increased biological complexity is actually predicted by evolution.

To illustrate this, Miller narrates how easily these mechanisms can account for the evolution of the human blood clotting system from a hypothetical 600-million-year-old marine invertebrate ancestor.² Unfortunately,

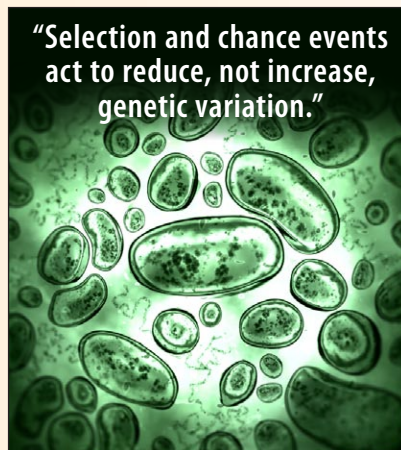
this process involves reading the present into the past. Without the actual animal to examine, Miller is forced to assume that this ancient ancestor is much like modern invertebrates. But this begs a question. If its descen-

dents still use essentially the same system today, there must have been little genetic or selective pressure to change. So what process of genetic change and selection would have forced one part of the population to turn into increasingly complex, land-dwelling vertebrates with a complex clotting system built from more than two dozen gene products, while at the same time forcing another part to keep the same clotting system for 600 million years? As much as Miller wants to claim a factual, scientific

basis for the evolution of vertebrate blood clotting, all he can offer is faith in a story he already assumes to be true.

An important assumption in Miller's argument is the existence of beneficial mutations. But what if mutations are almost exclusively harmful and actually reduce an organism's "fitness"? Recently, the effects of accumulating genetic mutations have been modeled by John Sanford³ who concludes that, rather than increasing genetic complexity and improving fitness, genetic load does just the opposite. He calls this process "genetic entropy" and suggests that it, and not the evolutionary story of "new and improved" genetics, is the real universal law of genetic change. 📌

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"Selection and chance events act to reduce, not increase, genetic variation."

1 Kenneth R. Miller, *Finding Darwin's God* (New York, NY: Cliff Street Books, Harper Collins, 2000).

2 Kenneth R. Miller, *The Evolution of Vertebrate Blood Clotting*, <http://www.millerandlevine.com/km/evol/DI/clot/Clotting.html>

3 John C. Sanford, *Genetic Entropy & The Mystery of the Genome*. (Lima, NY: Elim Publishing, 2005).