Evolution's Fast Lane

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BIG BIRDS

An unforgettable replica of New Zealand's largest bird, the giant moa, looms three meters tall in the Auckland Museum's natural history gallery. Created in 1913, the replica stands with its head erect, as it was imagined to look before scientists realized moa had S-shaped necks that kept their heads at back level. The display is a vivid reminder that New Zealand was once a remote, richly forested land dominated entirely by birds.

Before the arrival of humans and rats around AD 1100, New Zealand had approximately 245 bird species; moa (10 species) were the dominant herbivores, and the giant eagle (*Harpagornis moorei*) was the dominant predator. From the markings left on some moa skeletons, scientists have reconstructed how the 10- to 15-kilogram (kg) *H. moorei* struck and killed moa 15 times its size.

In a study published in the January issue of *PLOS Biology*, University of Oxford biologists Michael Bunce and Alan Cooper, New Zealand paleontologist Richard Holdaway, and their colleagues analyzed mitochondrial DNA from two *Harpagornis* specimens and compared these sequences with those for 16 living eagle species.

Their surprising conclusion is that New Zealand's giant eagle is not, as previously thought, related to the 4.5-kg wedge-tailed eagle (*Aquila audax*) from Australia. Instead, *H. moorei* shares a common ancestor with two much smaller eagles (genus *Hieraaetus*), each about 1 kg. They are so similar genetically that they may have diverged as recently as 0.7 million to 1.8 million years ago.

The rapid increase in the giant eagle's size is unprecedented, even for a large predatory bird without mammalian competitors. "*H. moorei*," the authors conclude, "therefore represents an extreme example of how freedom from competition on island ecosystems can rapidly influence morphological adaptation and speciation."

DOMESTICATED DOGS

When it comes to rapid changes in body plan, artificial selection reveals what's possible. The wide range of sizes and shapes of dog breeds, from 1- to 5-kg Chihuahuas to 50- to 90-kg Great Danes, shows the power of selective breeding. Where does the plasticity that gives breeders the ability to generate such changes in dog form come from?

John Fondon and Harold Garner have attempted to answer this complex question (*Proceedings of the National Academy of Sciences,* 28 December 2004). They were particularly interested in genes that determine skull shape and snout length, so they sequenced polymorphic regions of 17 developmental genes—orthologs of known human transcription factors—in 142 dogs representing 92 breeds and compared the sequence variability with morphometric measurements of the dogs' skulls.

What they discovered was that variation in the length of repeat-containing regions of these developmental genes results in changes in skull shape—without point mutation. The way it works is that the repeat domain length influences the activity of the developmental gene products. Longer repeats containing polyglutamine tend to drive transcription of target genes, whereas longer polyalanine repeats repress transcription. In the case of one particular gene, *Runx-2* (runt-related transcription factor 2), the target is "several genes whose function is to drive osteoblast differentiation and bone development," Fondon explains.

How universal is this mechanism? Fondon says, "I believe that in some circumstances, for some traits, repeat mutation will be the primary mode of change; in others, mutation of cis-regulatory elements; and in still others, yet undiscovered modes of evolutionary change will be responsible.

"What this tells us is that we don't know nearly what we thought we did about all the tricks that evolution has in its bag."

THE HUMAN BRAIN

Another wonder of rapid evolutionary change is the human brain. What kinds of genetic or developmental changes made brains of this size and complexity possible, especially given the similarity of human and chimpanzee genomes?

Researchers at the Howard Hughes Medical Institute, led by Bruce Lahn at the University of Chicago, published a study (*Cell,* 29 December 2004) examining evolutionary rates of a large number of genes known to be active in mammalian nervous systems. By looking at the divergence of 214 human and macaque genes and comparing the rates of change in coding sequences with those for orthologous genes in rats and mice, they determined that nervous-system genes evolved more quickly in humans.

The study included a similar comparison of 95 housekeeping genes—a subset of the genes considered essential for basic functions in all cells—which exhibited similar rates of change in primates and rodents. This result demonstrates that the rapid changes seen in the nervous-system genes of primates are not occurring throughout the genome. Furthermore, when genes of the nervous system are classified into functional groupings (developmental, physiological, or unclassified), it is the developmental genes of the nervous system that show the highest evolutionary rates, particularly in the lineage leading to humans.

For so many genes to be evolving rapidly in concert is remarkable, but it stands to reason that the genes underpinning human cognitive and behavioral adaptations would be a crucial component of evolutionary change.

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