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EVOLUTION

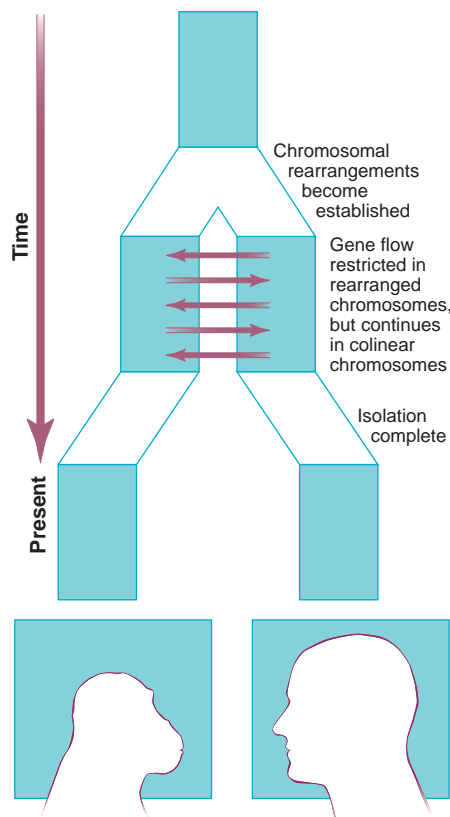
Chromosomal Speciation in Primates

Loren H. Rieseberg and Kevin Livingstone

The divergence of humans from the great apes highlights two of the most debated issues in speciation theory. First, modern humans and their closest relative, the chimpanzee, differ in both gene sequence and chromosome structure, and it is not clear which kind of change was the initial cause of reproductive isolation. Second, early humans and chimpanzees are likely to have lived in the same part of Africa, which bears on the larger question of whether speciation often occurs in the absence of geographic isolation. In a report on page 321 of this issue, Navarro and Barton (1) address both questions. They compare rates of protein evolution for genes on chromosomes that are colinear between humans and chimpanzees with genes from chromosomes that have undergone large structural rearrangements. They show that, on average, proteins from rearranged chromosomes evolved more than twice as fast as those from colinear chromosomes (that is, chromosomes with the same gene order). The most plausible interpretation of this pattern is that the chromosomal rearrangements “triggered” speciation by allowing differences under selection to accumulate in genes linked to the rearrangements, despite continued interbreeding between the two lineages for up to 3 million years after their initial divergence (see the figure).

So, why would chromosomal rearrangements affect rates of protein evolution? A simple solution to this puzzle is provided by a new model of chromosomal speciation (2–4). In traditional models, recombination between rearranged chromosomes is assumed to generate gametes carrying

chromosomal duplications or deficiencies. The unbalanced gametes or offspring produced from them may be nonviable, thereby creating a reproductive barrier. These models are unconvincing, however, because rearrangements that cause large reductions in the fitness of heterozygotes (that is, underdominant rearrangements)



Human origins. Chromosomal rearrangements appear to have triggered the separation of humans from the great apes by providing a barrier to gene flow in rearranged chromosomes. In contrast, gene flow continued for genes on colinear chromosomes.

can only be established in small inbred populations. Rearrangements that are neutral or weakly underdominant are more easily fixed in populations, but they will be ineffective as isolating barriers. Also, rearrangements that act solely to reduce hybrid fitness are unlikely to affect rates of protein evolution in loosely linked genes—the pattern reported by Navarro and Barton.

In the new model of chromosomal speciation, recombination is reduced in chromosomes heterozygous for the rearrangements (2–4), thereby minimizing the fitness effects of these rearrangements. Because recombination is required for gene flow, the rearrangements create a semipermeable reproductive barrier, where gene flow is reduced for rearranged chromosomes but continues across colinear chromosomes. As a consequence, selected differences are predicted to accumulate more readily in rearranged than in colinear chromosomes. Some of the accumulated differences are likely to cause incompatibilities in hybrids, ultimately sealing off the entire genome from gene flow and enabling completion of speciation, although this may take a very long time.

The origin of the human lineage represents a particularly appropriate test of this hypothesis for two reasons. First, biogeographic and anthropological evidence suggests that early forms of humans and chimpanzees are likely to have co-inhabited the same region of East Africa. Although it is possible that physical barriers such as rift valleys, rivers, and mountains might have provided the necessary isolation to initiate divergence among groups of apes, it seems unlikely that the different forms would have remained isolated long enough for speciation to be completed. Thus, the human-chimpanzee split may represent a kind of speciation with gene flow or “parapatric” speciation, where the effects of chromosomal barriers would be most pronounced. Second, humans and chimpanzees are known to differ in major chromosomal rearrangements involving 10 of 22 human autosomal chromosomes. The large number of chromosomes affected by rearrangements greatly increases the statis-

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tical power associated with tests of their effects on protein evolution. Moreover, 9 of the 10 rearrangements are pericentric inversions, which are known to reduce recombination in other animals (5).

To test this model of parapatric chromosomal speciation, Navarro and Barton compared rates of protein evolution for 115 autosomal genes in colinear versus rearranged chromosomes. This was accomplished by calculating the ratio of nucleotide substitutions that alter the predicted amino acid (K_A) to those that do not (K_S) for each of the 115 genes. Positive selection increases the K_A/K_S ratio, with a ratio of greater than 1 viewed as strong evidence of positive selection. Remarkably, the odds of finding significantly different proteins are about five times greater in rearranged chromosomes than in colinear chromosomes. Put another way, the average K_A/K_S ratio for genes from rearranged chromosomes of 0.84 is more than twice that of genes from colinear chromosomes ($K_A/K_S = 0.37$). Taken at face value, this suggests that interbreeding or hybridization has been going on for up to half the time of divergence between the two lineages (see the figure). Ancient hybrid zones are common in other animal and plant groups (6), so this interpretation is not as implausible as it might initially seem.

Are there other possible explanations for this result? Navarro and Barton analyzed numerous factors that sometimes correlate with rates of protein evolution—the clustering of K_A/K_S ratios along chromosomes, individual

chromosomal effects, or variation in GC content, recombination rate, or mutation rate—but came up empty-handed. Other factors that might inflate K_A/K_S ratios but could not be tested here include the possibility that rates of protein evolution increased during speciation, or that the accumulation of positively selected alleles provided the impetus for the fixation of the chromosomal rearrangements in the first place. So, even though the period of interbreeding may have been shorter than that suggested by the protein divergence estimates, the overall pattern described is robust.

Although the results presented here provide strong support for the new model of chromosomal speciation in which rearrangements act primarily as recombination modifiers, models in which rearrangements act to reduce fitness may be applicable in other situations. For example, it is well established that sterility in plants is often due to chromosomal rearrangements (7), and a recent analysis of yeast hybrids, in which genomes were engineered to reverse chromosomal differences, indicated that hybrid sterility was partly caused by chromosomal translocations (8). But how would rearrangements with large, contemporary negative effects on fitness become established? Two scenarios seem most likely. First, in highly redundant genomes, chromosomal rearrangements may have little impact on fitness, facilitating their establishment in the genome. However, as genomes become less functionally redundant over time, the fitness

effects of rearrangements would become much more severe. Second, rearrangements may be weakly underdominant individually, thereby aiding establishment, but strongly underdominant in combination, as required for reproductive isolation (9).

Although we have focused mostly on chromosomal evolution, the results of Navarro and Barton also have broad implications for our understanding of the geography of speciation. For much of the 20th century, geographic isolation was considered to be a requirement for speciation (10). However, theoretical and empirical work amassed over the past three decades has greatly enhanced the plausibility of models of speciation with gene flow (11), although specific cases are difficult to prove. The present paper not only suggests that the most famous speciation event of all represents a kind of speciation with gene flow, but also offers a means for estimating the importance of this mode of speciation in nature.

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MATERIALS SCIENCE

Muscles Made from Metal

Ray H. Baughman

Metals have long served as the “skins and bones” of machines and robots. Could they also be used as artificial muscles that directly transform electrical or chemical energy into mechanical energy? This possibility and other less challenging applications are suggested on page 312 of this issue by Weissmüller *et al.*, who show that electrochemical charge injection into porous nanostructured metals produces dimensional changes large enough to do mechanical work (1).

Shape-memory metals are already used as artificial muscles in dexterous robotic hands (2). But the actuation is indirect and requires the conversion of electrical energy to thermal energy to cause actuation, and

the dissipation of heat to reverse actuation. It has long been known that dimensional changes occur during charge injection into 13- μm -thick metal ribbons (3), but these changes were too small to be useful.

Weissmüller *et al.* (1) used a porous, electrolyte-filled assembly of pressure-compacted platinum nanoparticles as the actuator material in an electrochemical cell. A potential was applied between the platinum electrode and a counter electrode. The maximum observed actuation strain (which determines the actuator stroke) was $\sim 0.15\%$ for the platinum electrode (1)—about the same as for commercially used ferroelectric ceramics. However, the applied voltage between the actuating platinum electrode and the counter electrode was a few volts, compared with ~ 100 volts for ferroelectric actuators.

Obtaining giant capacitance per weight of actuator electrode was key for achieving

high actuator strains at low voltages (1). This is because capacitance is the derivative of stored charge with respect to potential, and actuator strains increase with increasing charge per electrode weight. The capacitance of the actuator electrode is very high, because it is proportional to the large electrochemically accessible area of the porous, nanostructured platinum and inversely proportional to the nanometer-scale separation between charge on the electrode and counter charge in the electrolyte.

The mechanisms providing actuation for the nanostructured platinum electrode are probably similar to those of carbon nanotube electrodes in similar electrochemical actuator cells (4). For low charge injection, actuator strains arise from quantum chemical effects that are linearly proportional to the injected charge. Electrostatic effects, which are quadratic in the charge on the actuator electrode, should dominate actuator strain at higher charge injection.

It is a long road from the described actuating nanostructured electrodes to practical actuators. The narrow redox stability range of the aqueous electrolytes used in

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