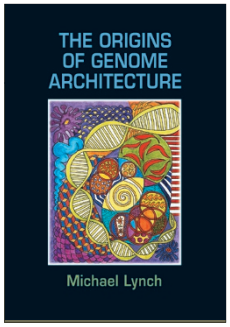
The spandrels of the genome



The Origins of Genome Architecture

By Michael Lynch

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Reviewed by Daniel Hartl

Here's a quick quiz, multiple choice: studies in molecular, cellular and developmental biology support the view that natural selection:

(A) Promotes increasing genome size and organismic complexity
(B) Favors biological organizations based on modular networks of regulatory or protein molecules
(C) Fosters robust systems that minimize the effects of mutational, developmental and environmental perturbations
(D) Results in genomic, cellular and developmental architectures that enhance the ability to evolve
(E) All of the above
(F) None of the (A)–(D)

The answer? According to Michael Lynch, the answer is (F): none of the claims is substantiated by direct evidence. The glib generalizations, he says, come from the assumption shared by many biologists that natural selection is the only mechanism of evolutionary change, and hence every observed feature of organisms must have come about because of natural selection.

But as Lynch observes, there's a lot more to modern evolutionary theory than natural selection. The trouble is that the theory is quantitative—and not only quantitative but stochastic, and hence incomprehensible to many biologists. Unlike celestial mechanics or string theory, where the foundations in complex mathematics inhibit amateurs from indulging their own celestial or dimensional fantasies, the mathematical theories of evolution as embodied in population genetics are often ignored or regarded as irrelevant. This conveniently licenses anyone to concoct any adaptive story about anything biological and put it forward as a serious contribution to the science of evolution.

Modern evolutionary theory does, of course, incorporate natural selection as a force for adaptive changes in genes and genomes. But the theory also embraces mutation, recombination and random genetic drift. Mutation in its widest sense includes insertions, deletions and transpositions; recombination also encompasses unequal crossing over and gene conversion, and genetic drift results from random changes in allele frequency that occur in all populations of finite size.

Each of these forces—mutation, recombination and random genetic drift—can explain some features of genes and genomes without recourse to adaptive scenarios. As early as 1985, when genomics began with the seemingly outlandish idea of sequencing the human genome, it was already clear that genome studies would revolutionize not only medicine but also evolutionary biology. Although the impacts of genomics on medicine have been widely recognized and reported, those on evolutionary biology have lacked a narrative of sufficient breadth and depth to capture the dramatic advances. Lynch's book not only examines the main evolutionary implications of genomics but also integrates these with modern evolutionary theory in a manner that few authors are equipped to do. If you want a good summary of what genomics has revealed about genome architecture over the last two decades, merged with a nontechnical exposition of the relevant principles of population genetics, this is the book to get.

The chapter contents include evolutionary aspects of the origin of eukaryotes, genome size, the human genome, population size, chromosome organization, genome composition, transposable elements, gene duplications, introns and RNA splicing, transcription, organelle genomes, sex chromosomes and prospects for the future. The recurring theme is that many of the major molecular features of genes and genomes in multicellular organisms can be explained without invoking natural selection. The list will raise some eyebrows: it includes the transition from the RNA world to DNA, streamlining of microbial genomes, variation in nucleotide composition within and among genomes, centromere expansion, proliferation of transposable elements, growth of untranslated regions in messenger RNAs, origin of spliceosomes and the proliferation of introns, origin of modular gene regulation, variation in the architecture of organelle genomes, mRNA editing in plant organelles and the evolution of sex chromosomes.

Lynch emphasizes the importance of limited population size in reducing the efficacy of selection, thereby inhibiting some directions of evolutionary change and encouraging others. Under these conditions, the directional effects of mutation pressure alone can often overwhelm external forces of natural selection and so drive genome evolution. As additional layers of complexity build up by nonadaptive processes, they open up new possibilities for adaptive evolution. On the flip side, the large population size of microbial organisms serves as a barrier to the emergence of gene and genomic complexity.

The problem with natural selection is that if it explains everything, it explains nothing, and instead it becomes an exercise in story telling. This impoverishes the field, as if physicists admitted only gravity or chemists only covalent bonds. The value of such skepticism is not in proving that any particular genomic feature actually arose through nonadaptive processes. The true value is in proposing nonadaptive models with specific mechanisms. The challenge for those who would invoke natural selection as causing any biological feature is therefore to propose a specific adaptive mechanism, to deduce attributes that would differ according to which hypothesis were correct and then to make the necessary observations or comparisons.

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