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The complexity ratchet: Stronger than selection!

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Abstract
Using the Aevol digital genetics platform we designed an in silico experiment to study the relationship between molecular complexity and phenotypic complexity: We evolved populations of digital organisms in an environment designed to allow survival of the simplest possible organism: one which genome encodes a single gene. By repeatedly evolving populations in this experimental framework, we observed that ≈1/3 of the lineages quickly found this simple genotype and were then stable for the rest of the experiment. At the same time, most lineages were not able to find this simple solution and showed a low gene acquisition along the 250,000 generations of the experiment. Importantly, simple organisms ended up with a very high fitness while complex genotypes ended up with a ≈10x lower fitness. This shows that, even in a simple environment, evolution leads to a complexity ratchet: each gene acquisition creates the potential for the acquisition of further genes, ultimately pushing evolution towards complex solutions even in a simple environment. Moreover, organisms engaged in this complexity funnel process were never able to outcompete the simple ones, showing that selection is not able to invert the complexity ratchet.

Methods
Aevol (www.aevol.fr) is an In Silico Experimental Evolution (ISEE – aka digital genetics) platform developed by the Beagle team to study the evolution of genome structure. Aevol is based on three principles that makes it perfectly suited to study the evolution of complexity:

A. Its genotype-to-phenotype map is simulated by a generational algorithm. Organisms’ fitness is based on a curve-fitting task: the protein triangles are summed to compute the organisms’ phenotype that is compared with a target function (red curve below).

B. At each replication the genome may undergo mutations. Aevol implements a wide range of mutational operators including switches, InDel and chromosomal rearrangements. Mutations can change complexity at both genomic and functional levels.

Experimental design and complexity measures

- Qualitative measure: “simple” organisms are those encoding only proteins with the same m and w values.
- Genomic complexity: quantity of information encoded on the genome (total amount of coding sequences).
- Functional complexity: quantity of information encoded on the proteome (number of different parameters).

Results
A. Distribution of genomic complexity for complex (top) and simple (bottom) organisms. The higher the mutation rate, the lower the genomic complexity. Simple organisms are strongly limited by mutational robustness.

B. Distribution of functional complexity for complex (top) and simple (bottom) organisms. The higher the mutation rate, the lower the functional complexity. 

Discussion
The emergence of complex organisms in a simple environment is a strong argument in favor of a complexity ratchet, i.e. an irreversible mechanism that adds components to a system but that cannot get rid of existing ones, even though this could be more favorable. Indeed, in our experiments this ratchet clicks and goes on clicking despite the selective advantage of being simple. Evolution of fitness in complex organisms shows that the ratchet is empowered by negative epistasis. Our results show that complex biological structures can flourish in conditions where complexity is not needed and that, reciprocally, the global function of complex structures could very well be simple.