The complexity ratchet: stronger than selection
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To cite this version:

HAL Id: hal-01938802
https://hal.archives-ouvertes.fr/hal-01938802
Submitted on 28 Nov 2018

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Experimental design:

- To unravel the origin of molecular complexity, we evolved populations in the simplest possible environment: the Aevol target is a triangle.
- We evolved 300 populations of 1024 individuals for 250,000 generations under 3 mutation rates and monitored the evolution of genomic and functional complexity.

Results

A. Distribution of genomic complexity for complex (top) and simple (bottom) organisms. The higher the mutation rate, the lower the genomic complexity. Genomic complexity is 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. Simple organisms approach the optimum fitness, \( f = 1 \), while fitness of complex organisms, \( f \approx 0.38 \).

A. Fitness at generation 250,000 vs functional complexity. The higher the functional complexity, the lower the fitness. Simple organisms approach the optimum fitness, \( f = 1 \), while fitness of complex organisms, \( f \approx 0.38 \).

B. Long-term evolution of functional complexity in a complex organism. Functional complexity and fitness continuously grow during the 250,000 generations but the fitness remains far below that of simple organisms.

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).

Complexity:

- Genomic complexity: amount of noncoding DNA.
- Functional complexity: quantity of functional genes.

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.

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 \( \approx 1/3 \) of the lineages quickly found this simple genome 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 an ongoing gene acquisition along the 250,000 generations of the experiment. Importantly, simple organisms ended up with a very high fitness while complex genomes ended up with a \( \approx 10x \) 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 complexification process were never able to outcompete the simple ones, showing that selection is not able to invert the complexity ratchet.

Methods

A. 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. Evolution 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).

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

B. Genome and proteome of a simple organism

C. Genome and proteome of a complex organism

References


Experimental design and complexity measures

- Genomic complexity:
  - Genomic complexity: amount of noncoding DNA.
  - Functional complexity: quantity of functional genes.

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