The complexity ratchet: stronger than selection

Vincent F Liard, Jonathan Rouzaud-Cornabas, David P. Parsons, Guillaume Beslon

The complexity ratchet: Stronger than selection!

Vincent Liard, Jonathan Rouzaud-Cornabas, David P. Parsons, Guillaume Beslon

INRIA-Beagle team (INSA-Lyon), Lyon, France

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 slow 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 complexification process were never able to outcompete the simple ones, showing that selection is not able to invert the complexity ratchet.

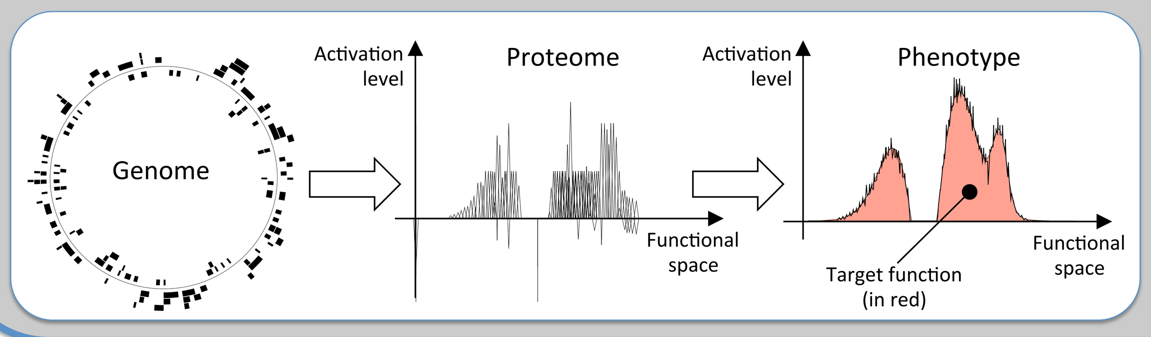
Methods

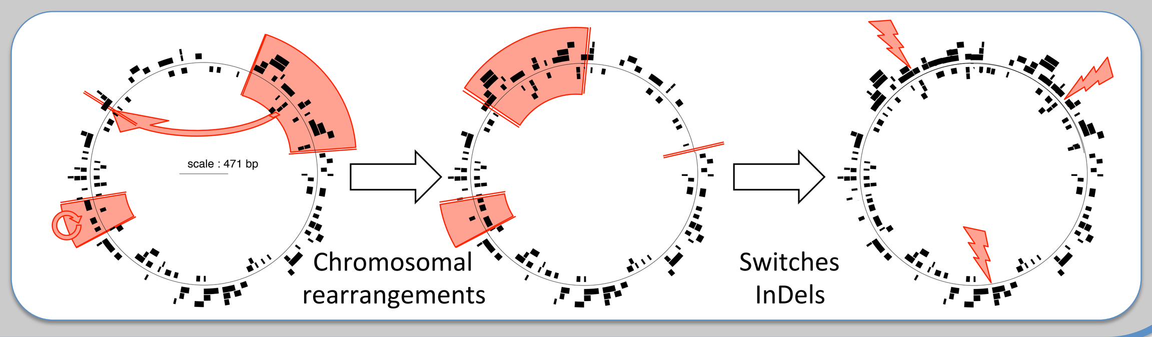
Aevol (www.aevol.fr) is an *In Silico* Experimental Evolution (ISEE – *aka* digital genetics) platform developed by the Beagle team to study the evolution of genome structure. Aevol is based on three principles that makes it perfectly suited to study the evolution of complexity:

A. Its genotype-to-phenotype map mimics biology with a realistic genomic structure and a functional structure based on a graphical formalism. Proteins are represented by triangles which parameters are computed from the gene sequence.

B. 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.

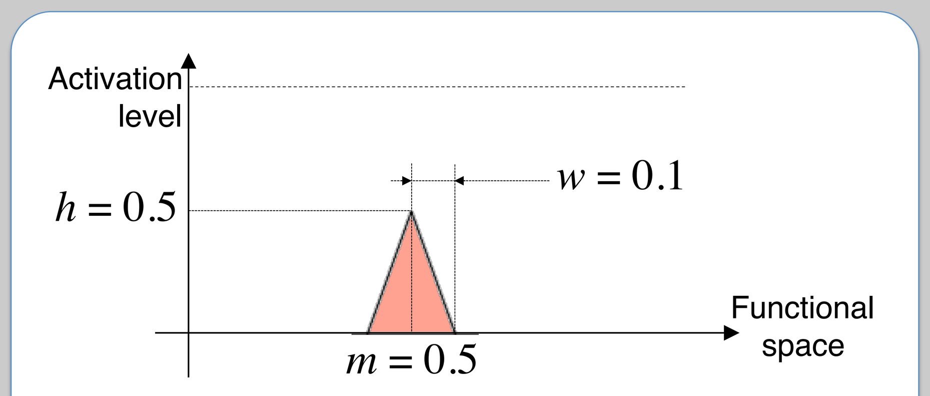




Experimental design and complexity measures

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.



Experiment-specific target function: this triangular target function can be fitted by a single gene/protein.

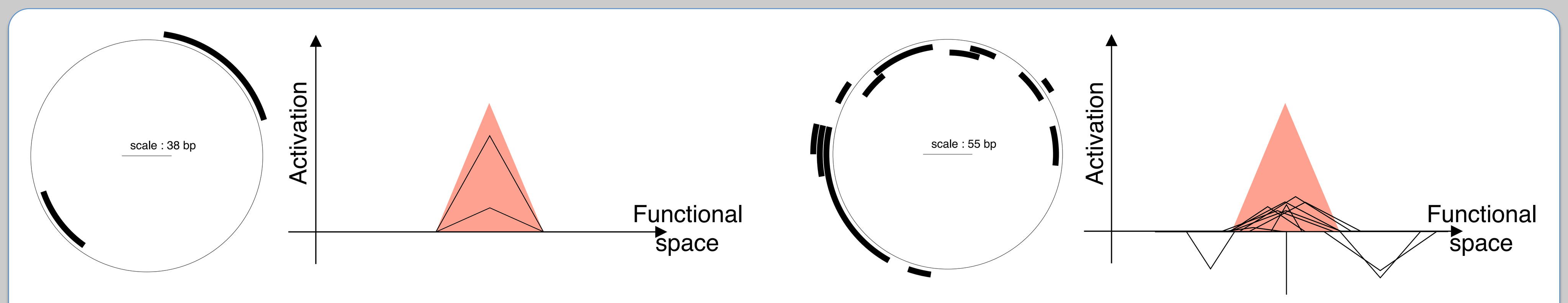
Complexity measures:

- 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

(1) Organisms evolved complex functional structures in 66% of the simulations

Whatever the mutation rate, ≈1/3 of the simulations led to "simple" organisms with few genes and a low functional complexity (A). ≈2/3 of the simulations led to "complex" organisms despite the simplicity of the target function (B).

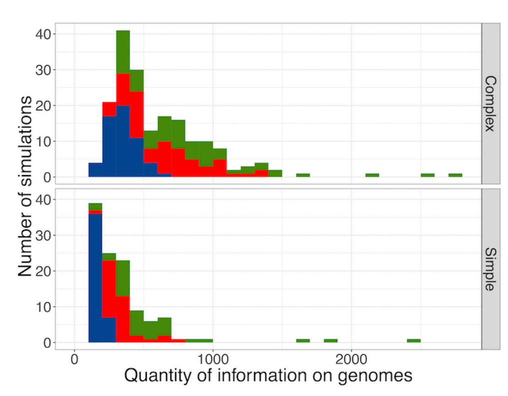


A. Genome and proteome of a simple organism B. Genome and proteome of a complex organism

(2) Complex organisms accumulate more information at the genomic and functional levels

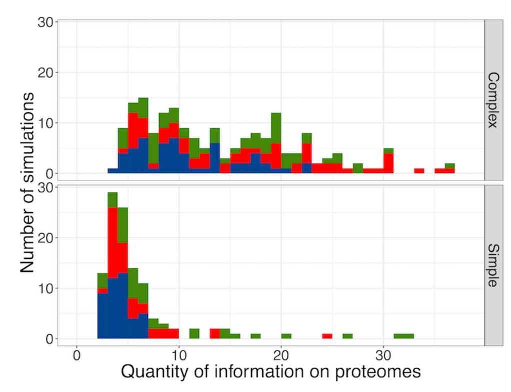
Genomic complexity is strongly bounded by mutation rates (A) due to robustness constraints on the genome (Knibbe et al., 2007; Fischer et al., 2014). Mutation rates also constrain the functional complexity (B) but this effect is less stringent at the functional level.

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.

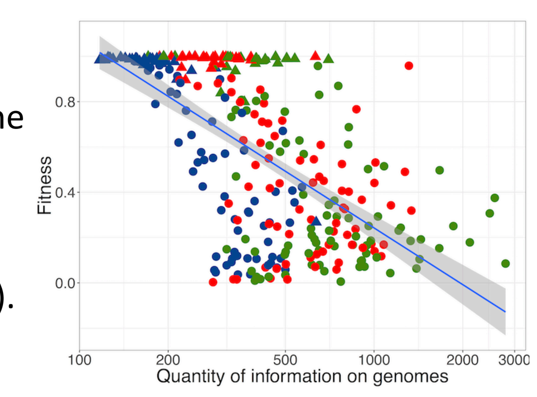
: High mutation rate
: Medium mutation rate
: Low mutation rate



(3) Simple organisms are fitter than complex ones

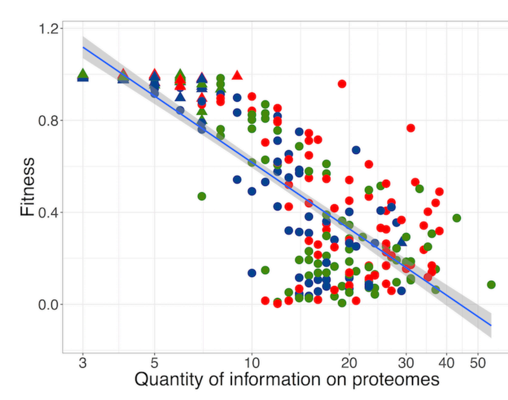
Whatever the complexity measure, we observe a clear trend for simple organisms to be fitter than complex ones after 250,000 generations. This demonstrates that in our simulations complexity is not driven by selection. On the opposite, complex functional structures have evolved in spite of selection.

A. Fitness at generation 250,000 vs genomic complexity. The higher the genomic complexity, the lower the fitness. Simple organisms approach the optimum fitness ($f_{opt} = 1$). Mean fitness of complex organisms: $f = 0.38$.



B. Fitness at generation 250,000 vs functional complexity. The higher the functional complexity, the lower the fitness.

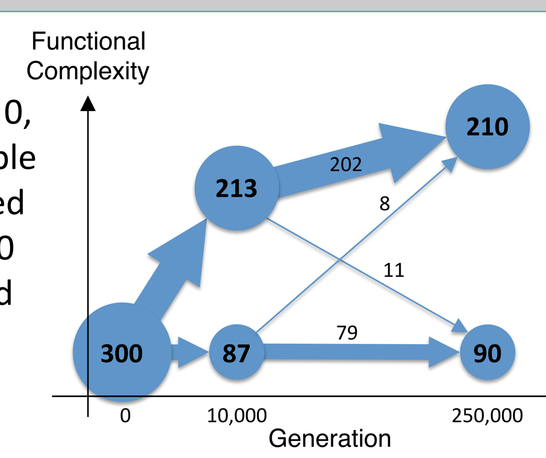
△ : Simple organisms
○ : Complex organisms



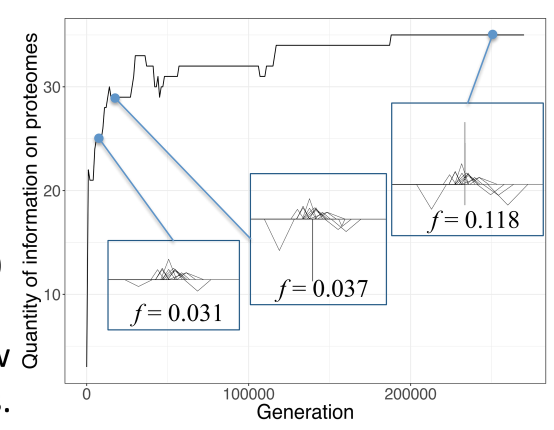
(4) Despite the advantage of being simple, complex organisms evolve greater complexity on the long term

The simple/complex identities are determined early on in the simulation and generally conserved thereafter (A). Complex organisms evolve greater complexity (B); their fitness grows but remains far below simple organisms.

A. Starting from simple organisms at generation 0, organisms' identity (simple vs complex) is determined before generation 10,000 and generally maintained for the rest of the simulation.



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.



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.

References

- Liard, V., Parsons, D. P., Rouzaud-Cornabas, J. and Beslon, G. (2018) The complexity ratchet: stronger than selection, weaker than robustness. In: *Proceedings of the 2018 Conference on Artificial Life*, Tokyo, July 2018, pp. 250-257.
- Knibbe, C., Coulon, A., Mazet, O., Fayard, J.-M. and Beslon, G. (2007) A long-term evolutionary pressure on the amount of noncoding DNA. *Molecular Biology and Evolution*, 24(10):2344-2353.
- Fischer, S., Bernard, S., Beslon, G. and Knibbe, C. (2014) A model for genome size evolution. *Bulletin of mathematical biology*, 76(9):2249-2291.

Inria informatiques mathématiques · INSA · Université de Lyon · CNRS · Seventh Framework Programme