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In silico experimental evolution shows that complexity can rise even in simple environments

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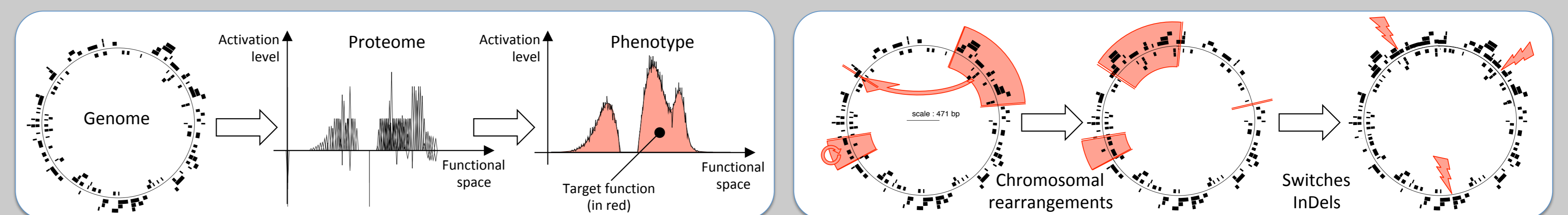
Introduction

Systems biology, often viewed as reverse engineering of biological systems, deals however with objects that have not been designed. Neither do they have a predefined purpose nor do they follow engineering rules. Indeed, we don't know what are the "design rules" that evolution imposes to biological systems while this knowledge would be a valuable interpretative framework for systems biology. One of the recurrent questions on that matter is the origin of the striking molecular complexity of biological systems. Answering this question requires deciphering the complex interactions between all the forces that drive evolution, including selective and non-selective ones. In this context, simulation is a valuable tool as it enables to observe how organisms grow in complexity (or do not) when they evolve in environments which complexity is perfectly controlled.

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:

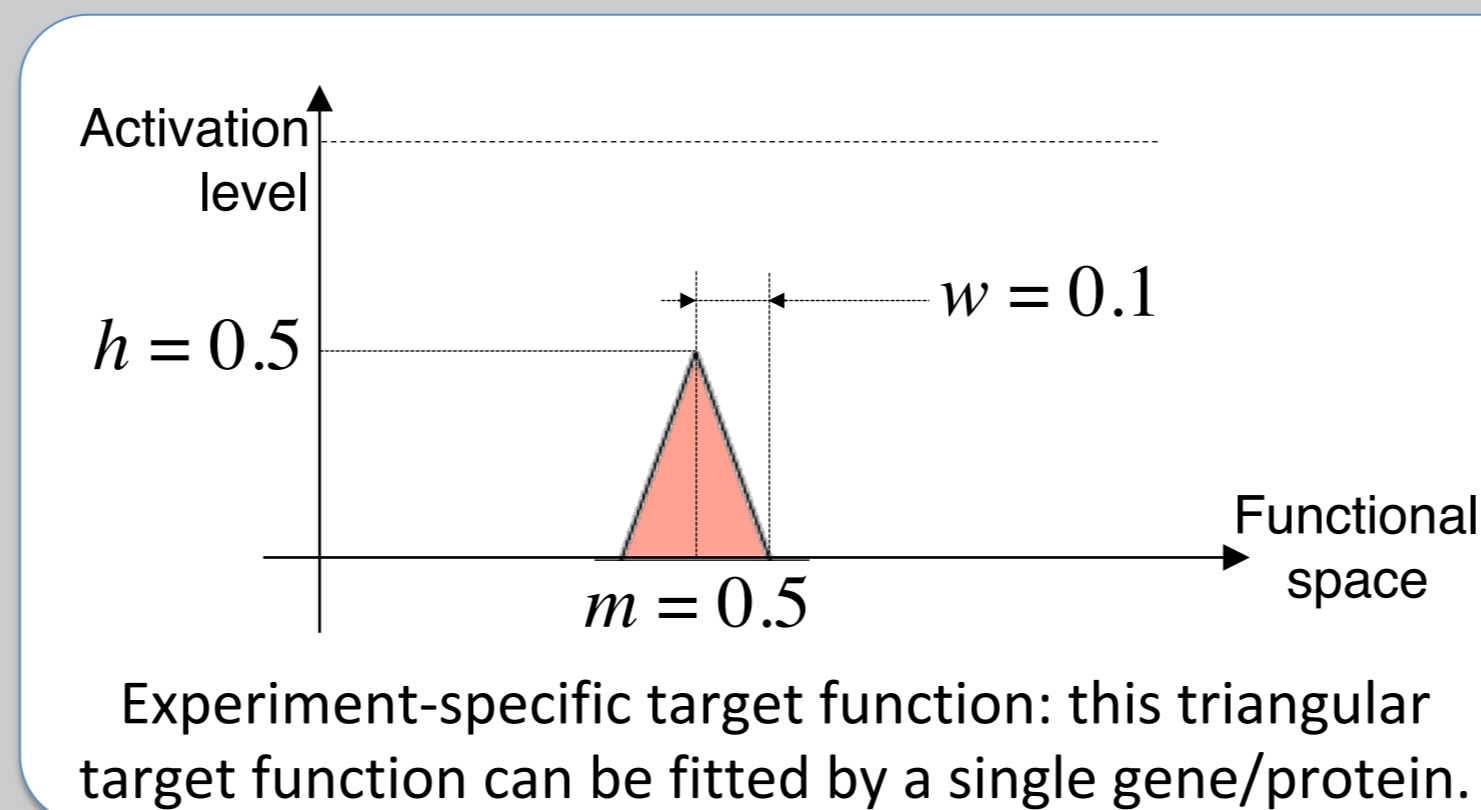
- Its genotype-to-phenotype map mimics biology with a realistic genomic structure and a functional structure based on a graphical formalism. Proteins are represented by triangles which parameters are computed from the gene sequence.
- Evolution is simulated by a generational algorithm. Organisms' fitness 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).
- 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 an environment where the simplest possible organism can thrive.
- We evolved 300 populations of 1024 individuals for 250,000 generations under 3 mutation rates and monitored the evolution complexity.



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

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

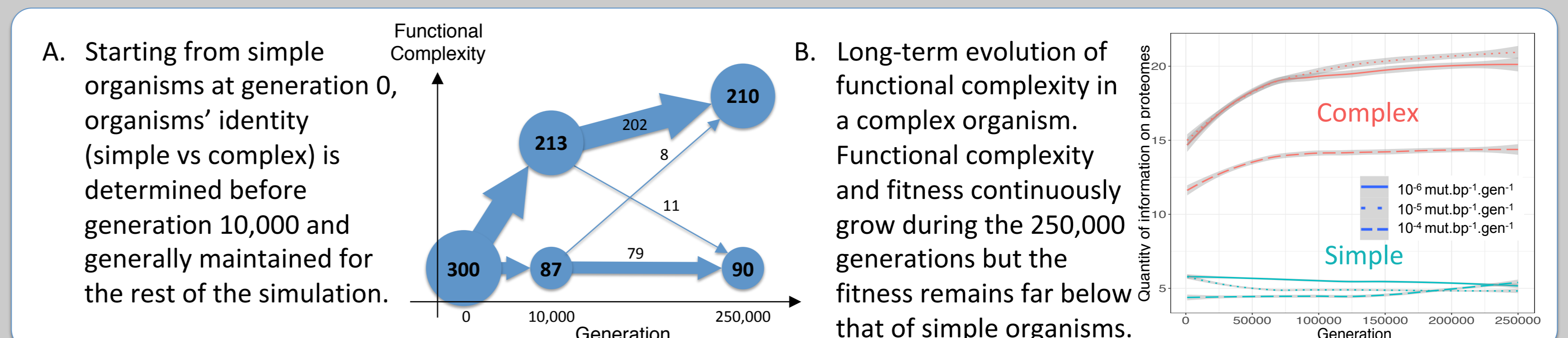
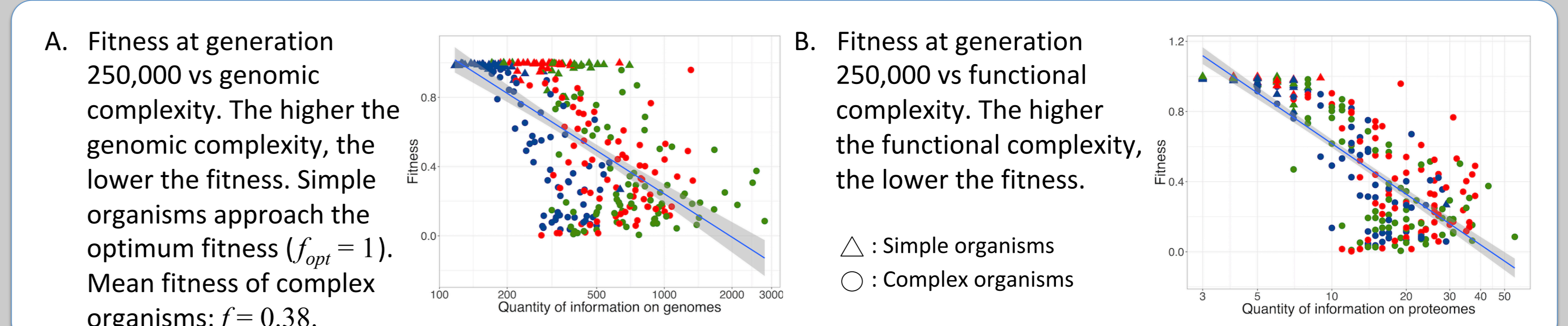
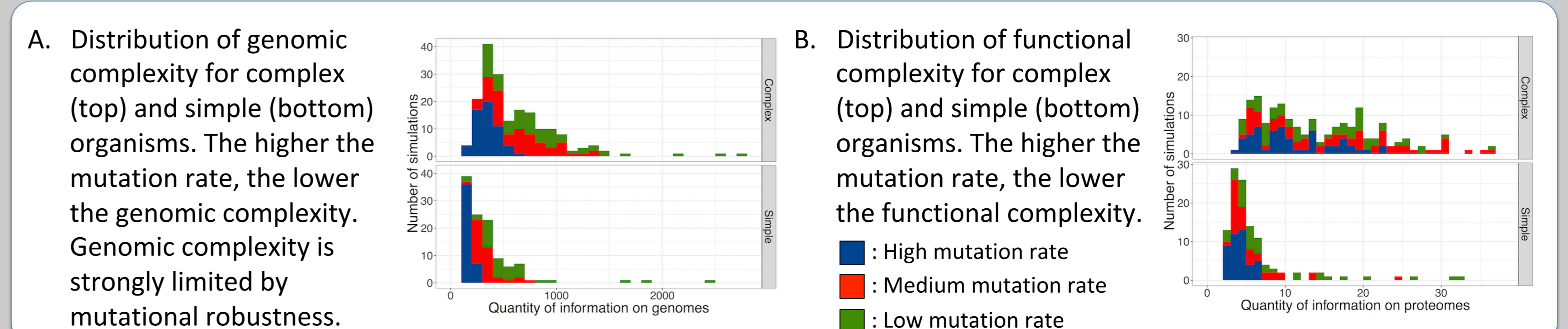
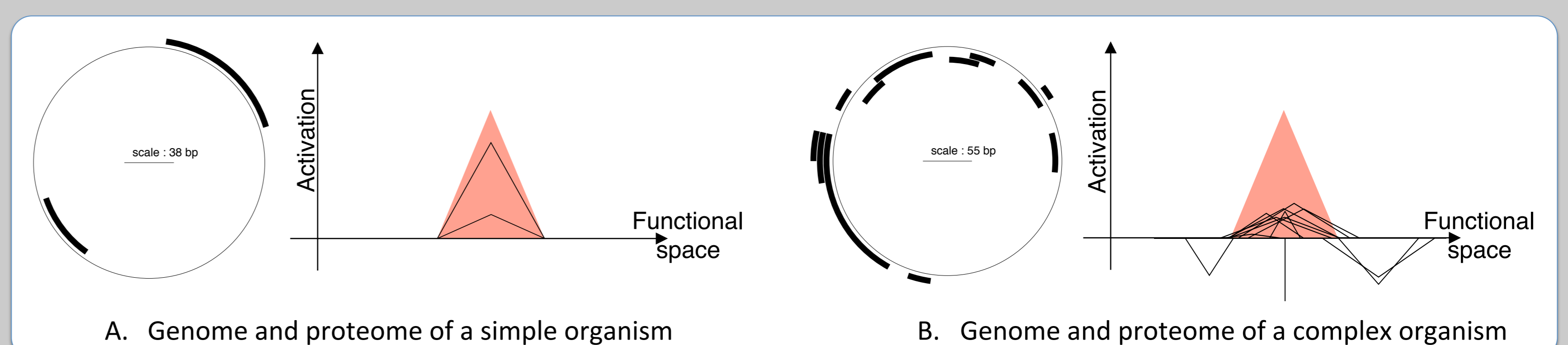
Genomic complexity is strongly bounded by mutation rates (A) due to robustness constraints on the genome (Knibbe et al., 2007; Fischer et al., 2014). Mutation rates also constrain the functional complexity (B) but this effect is less stringent at the functional level.

(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.

(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.



Discussion

Our results show that, in such a simple constant environment, there is a decoupling between the molecular complexity of the organisms and the complexity of the environment. This shows that selection for complexity is not mandatory for complexity to evolve and that complex biological structures could flourish in conditions where complexity is not needed. Reciprocally, the global function of complex biological structures could very well be simple. We think this result is greatly significant for both evolutionary biology and systems biology.

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.