Multiplicative mutations, sparseness and modularity in biological systems

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***Short Abstract* — Modularity is a common hallmark of many biological systems at all levels of organization. Despite its ubiquity, the origins of modularity are still poorly understood. Here we address this question in the context of biological networks, focusing on the mutational process. We show how mutations that are multiplicative induce network sparseness under a general range of conditions, whereas additive mutations generally do not. If the goal is modular, the network evolves modular structures, with multiplicative, but not additive mutations.**

***Keywords* — genetic algorithms, evolution, networks.**

I. Introduction

Modularity is defined as near decomposability of a system into distinct substructures with a high level of connectivity within each substructure, but much less connectivity to others. It is a repeating theme in biological systems at different levels of organization. For example, the existence of various cell types in multi-cellular organisms is a familiar manifestation of modular organization. Modularity is also found in genetic regulatory and protein interaction networks. Despite its prevalence, it remains an open question how modularity arises during evolution [1]. An abstract concept rather than a physical trait, modularity does not directly interact with the environment. Standard evolutionary models indeed generally fail to provide modular solutions. Previous works considered specific circumstances under which modularity can ensue, such as goals that vary over time and share the same set of sub-goals (modularly varying goals) [1,2].

Here we return to the basic mutation-selection process. We show that network sparseness and modularity naturally emerge under a broad range of parameters if mutations are multiplicative rather than additive. Multiplicative mutations are thought to better describe the biological mutation of binding sites, because mutations alter binding energy, which is proportional to the logarithm of affinity [3].

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II. Results

We constructed a simplified nonlinear model of a two-level network, inspired by gene regulatory networks. This system is evolved to achieve a given goal and its fitness is defined as the distance of its outcome from the goal. We used standard evolutionary algorithms [4] to evolve a population of such entities and study the effect of the mutational process on their structure. This model network is equipped with excess degrees of freedom and can attain the goal in numerous ways, most of which are non-sparse. When additive mutations are applied, sparse solutions are rarely achieved, and even if reached they are dynamically unstable. Multiplicative mutations in contrast have the property that zero terms are kept as fixed points. The combination of such mutations with selection brings about network configurations that are as sparse as possible and still satisfy the goal. If the goal is modular, the evolved network will be modular too.

As the optimal solutions span a multidimensional manifold, the model exhibits rich dynamics with occurrences of distinguishable fitness plateaus [5]. These typically happen if the network temporarily occupies a sub-optimal architecture. Escape from plateaus might take long periods because it works against the mutational trend. Lastly, we study how the time to reach modularity depends on the problem complexity.

III. Conclusions

We find that the biologically plausible mechanism of multiplicative mutations leads to sparse network configurations, and with modular goals to network modularity. This is in contrast to additive mutations, commonly applied in models, which bring about non-sparse and non-modular solutions.

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