Does Evolutionary Plasticity Evolve?

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***Abstract*—This article serves as a summary of Andreas Wagner's paper titled *Does Evolutionary Plasticity Evolved?*. Wagner firstly stated the problem domain of epigenetic stability, and illustrated what properties are required in order to facilitate an organism with epigenetic stability. According to these elicited properties, Wagner built a mathematical model, which possess similar characteristics. By studying this artificial model, Wagner concluded that epigenetic stability is heritable and summed up other inspiring opinions.**

I. Contents

Mutations that happen at lower-level genotypes may not lead to changes of phenotypes at higher levels. Epigenetic is a major cause of this phenomenon. In other words, epigenetic can be viewed as a *buffer* for changes that occur as mutations on its lowest levels, and thus stabilize the phenotype with respect to mutations. During the development of a multicellular organisms from a zygote, a large number of epigenetic interactions can take place on every level of organization.

Within a biological organism, there are thousands of genes, each of which may express a spectrum of biologically active molecules, a multitude of regulatory interactions among those molecules, which jointly build the web of cellular organization, and finally an immense number of intercellular communication processes, which create the developmental coordination that produces a finely integrated adult organism. Taken together, they constitute the intricate fabric of the epigenetic system. Due to this intricate complexity, it is intractable to assay this system directly from the traditional biological due to the lack of technologies and knowledge.

Transcriptional regulators guide how cell patterns are formed in various organisms. This transcriptional regulations are ubiquitous, such gene networks still await discovery in many other organisms. Most significantly, these gene patterns are highly conserved and show little naturally variation. In this paper, it is investigated whether this invariance reflect an evolved resilience, termed *epigenetic stability*.

This question is difficult to address experimentally. For example, the time scale for the evolution of epigenetic stability is astonishing. Consider a network with $N = 10$ genes, a density of regulatory enhancer element corresponds to 20 to 100 base pairs of DNA. If mutations affecting these elements occur at a rate of approximately 10^{-9} per base pair per generation, the experiment can take more than 10^8 generations.

Therefore, A mathematical model for gene networks was used here to provide preliminary answers. We expect this mathematical model has the following two properties:

1) A mutation on lower-level genes may not change the patterns determined by these genes.
2) Different gene regulatory networks can result in the same gene activity patterns.
3) Genes work in a collaborate fashion, and can have positive or negative effects on each other.

Furthermore, we also impose three assumptions on our model of our transcriptional regulators:

1) Gene expressions are independent from the activities on the transcriptional levels.
2) Each gene only produce one type of transcriptional activities.
3) Different enhancing effects from different genes are independent to each other.

With the assistance of this model, we can observe that epigenetic stability can be evolved. This model has two types of genes, termed *upstream* and *downstream* genes. The former refers to the genes which produce transcriptional products whereas the latter is the genes that only govern upstream genes.

For the sake of simplicity, we omit the exact mathematical model in this article, only include some points that the author thinks are of importance and subtilty.

1) Any non-zero diagonal element, $w_{ii} \neq 0$, corresponds to autoregulation of G_i by its own gene products. That is, a gene will always be regulated by its own products.
2) When recombining, rows are swapped with probability 0.5, corresponding to free recombination between genes and tight linkage among regulatory elements within a promoter. That is, changing everything among networks for a particular gene.

   As a result, patterns of epigenetic interactions acts as a *buffer* that prevents the effects of most mutations from becoming visible as altered equilibrium gene expression states.

   Another insight lies on that as decrease in the number of regulatory interactions per gene or the number of network genes, stability to mutations after evolution decreases. This may because that as the network size or connectivity density is decreased, the influence that individual mutations have on the equilibrium can increase due to a bigger proportion of connections affected.

II. Questions

- What is the *house-of-cards assumptions*?
- As c (the edge density) decreases from 1, both mean stability after evolution and variability of stability before evolution decrease. If this holds, why Espinosa-Soto utilized a ratio of 0.2?
- Recombination leaves important network properties unchanged.