Digital Investigations on the Evolution of Prokaryote Photosynthesis Regulation

Late-Breaking Abstract

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ABSTRACT

The questions about nature that we can address using digital evolution are constrained by the speed of our software and the level of abstraction used in our genetic representations. One subject that has been particularly challenging is the evolution of gene regulatory networks. We introduce a new digital evolution platform, built upon a mechanistic model of gene regulation and chemical signaling, that permits studying the evolution of an organism's adaptive decision-making and its underlying gene regulatory and signaling networks. We will discuss how this platform is being used to investigate the evolution of homeostasis and circadian rhythms in photosynthetic prokaryotes.

CCS CONCEPTS

• **Computing methodologies~Artificial life** • **Computing methodologies~Genetic algorithms**

KEYWORDS

Gene Regulatory Networks, Control Systems, Homeostasis, Autopoiesis, Circadian Rhythm, Photosynthesis

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1 INTRODUCTION

Gene regulatory networks have unique properties that make them a powerful model for evolutionary computation [2]. However, their full potential as an open-ended and biologically-relevant representation has remained largely unfulfilled. This is likely due to the complexity of such models and their high computational cost. We tackled both issues by using a new platform, ELFA, based on a mechanistic model of gene regulation and chemical signaling that is realistic and fast, to explore questions related to the evolution of homeostasis and other types of robust control systems in which biological organisms excel [7].

For our first experiment, we created the digital equivalent to a simple cyanobacterium cell and placed it in a simulated underwater environment where it is free to grow and reproduce, under the selective pressures of a limited-size population. Our environment contains a daily light cycle that follows a sinusoidal curve, providing an incentive for cells to manage energy usage by evolving gene regulation of functions such as photosynthesis, growth, and reproduction to take advantage of the light energy when it is available.

The representation ELFA uses includes both cis- and trans-regulatory elements, in the form of enhancers, transcription factors and basal promoters. It also includes mechanisms for protein-protein and protein-ligand interaction. The strength of any interaction depends on the affinity level between molecules and permits different degrees of agonism. Therefore, ELFA allows for complex networks of interaction that replicate some interesting features of natural gene-regulatory networks, such as cross-talk, differential gene activation, and combinatory logic [1].

The ancestral cell, which we use to seed our population, expresses all of its genes at low and constant rates and possesses no cis-regulatory elements. However, during evolution its descendants can suffer mutations and acquire new regulatory and functional domains that, over time, could give rise to complex regulatory circuits.

2 PHOTOSYNTHESIS REGULATION

In all oxygenic photosynthetic organisms, photosynthesis is limited by the intensity of the light field, and by the amount of CO_2 inside of the cells [3] (Fig. 1). In a natural environment just after sunrise, the rate of photosynthesis of a given organism increases proportionately to the irradiance, and then slowly plateaus at its

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maximum photosynthetic rate. At this level, the organism is said to be light saturated, and the availability of CO_2, which enters cells by diffusion, becomes the limiting factor. If the irradiance continues to increase, the rate of photosynthesis eventually declines due to photoinhibition, which is a natural mechanism to protect against light damage and CO_2 depletion.

In our model, cells can evolve to regulate their photosynthetic rate in a similar manner, taking advantage of the available light energy, while avoiding the negative effects of CO_2 depletion. The ancestral cell contains three genes expressed at different but constant rates. The first encodes for the RNA polymerase, the molecule responsible for transcribing the cell's genes. The second encodes the photosynthetic complex responsible for converting light, CO_2, and water into sugar that the cell uses for energy. The third encodes a growth factor that signals the rate at which the cell should grow, and also replicate.

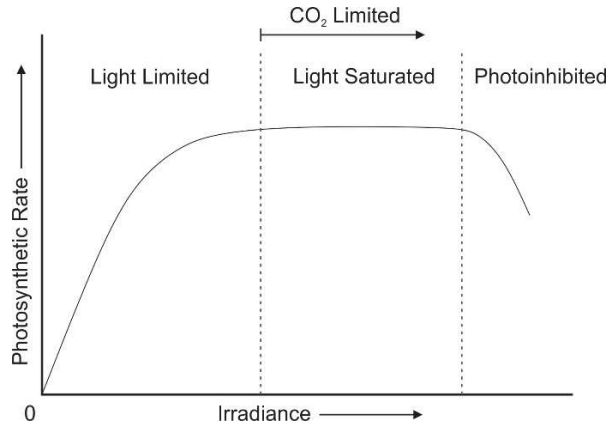

Figure 1: Schematic photosynthetic rate as a function of irradiance, illustrating maximum photosynthetic rate, and limitations due to environmental irradiance and cell CO_2 concentration.

3 SIGNALING LIGANDS AND ADDITIONAL FEATURES

We provide each cell with signals, in the form of ligands, which convey information about the conditions in the environment and within the cell. However, in order to sense them, cells must first evolve proteins that bind the specific ligands. These are the five types of ligands used in this experiment and their effects:

Reactive Oxygen Species (ROS), which is sensed as a ligand but can also be harmful at high concentrations. ROS is produced as the cell runs out of CO_2 during photosynthesis. Higher concentrations of ROS increase the probability of somatic mutations, protein damage, transcription slowdown, cell aging, and loss of energy reserves.

Cyclic AMP (cAMP), which signals starvation, and is produced as the cell runs out of energy reserves. When cAMP concentration is high, there is an increased chance of somatic mutations, protein damage, and transcription slowdown.

Irradiance level, which indicates the amount of light reaching the cell, and follows a 24-hour cycle of night and day.

Sugar reserves, which indicate the amount of energy immediately available.

Fat reserves, which indicate the amount of energy in long-term storage.

In addition, there are several costs and constraints to the cell's actions. For example, protein transcription, cell growth, and replication, all have associated energy costs [4,5]. If a cell runs out of energy it dies. All proteins are encoded with a half-life that determines the time they remain in the cell before being recycled. Cells have no age limit but can accumulate harmful protein aggregates (plaque), which are formed when proteins are damaged by excessive ROS and cannot be recycled. This, in effect, is an aging mechanism. When a cell replicates, all of its plaque is segregated into one daughter cell that 'ages' while the other is born 'rejuvenated' [6].

4 HYPOTHESES AND OBJECTIVES

By imposing the cell with costs and constraints while providing sensory feedback signals, we create the opportunity for the evolution of complex regulatory networks. We hypothesize that, over the generations, a population will evolve to increase their photosynthetic output and reproduce faster, maximizing the use of available light. Specifically, we expect the initial mutations to adjust the rate at which their genes are expressed, without adding any form of dynamic regulation. Later, the population will evolve regulatory mechanisms that allow it to time the production of proteins, as well as cell division, according to the environmental light cycle. One especially exciting outcome would be the evolution of homeostatic regulation in the form of one or more feedback control loops [1]. Another intriguing possibility is the evolution of a circadian clock.

We will demonstrate a useful tool for investigating the evolution of gene regulatory networks, including analyzing the origin and evolution of regulatory elements, and studying how genes appear, change function, or disappear. Additionally, any self-regulating cells we evolve could be used as model organisms in further experiments, such as analyzing the effects of circadian rhythm disruption or studying evolution in response to light cycles from different latitudes.

REFERENCES

[1] Alon, Uri. *An Introduction to Systems Biology: Design Principles of Biological Circuits*. CRC Press, 2006.
[2] Banzhaf, Wolfgang. "On Evolutionary Design, Embodiment, and Artificial Regulatory Networks." In *Embodied Artificial Intelligence*, 284–92. Lecture Notes in Computer Science. Springer, Berlin, Heidelberg, 2004.
[3] Kirk, John T. O. *Light and Photosynthesis in Aquatic Ecosystems*. Cambridge University Press, 2010.
[4] Lane, Nick, and William F. Martin. "Mitochondria, Complexity, and Evolutionary Deficit Spending." *Proceedings of the National Academy of Sciences* 113, no. 6 (February 9, 2016): E666–E666.
[5] Lynch, Michael, and Georgi K. Marinov. "The Bioenergetic Costs of a Gene." *Proceedings of the National Academy of Sciences* 112, no. 51 (December 22, 2015): 15690–95.
[6] Stewart, Eric J., Richard Madden, Gregory Paul, and François Taddei. "Aging and Death in an Organism That Reproduces by Morphologically Symmetric Division." *PLOS Biology 3*, no. 2 (February 1, 2005): e45.
[7] Yi, Tau-Mu, Yun Huang, Melvin I. Simon, and John Doyle. "Robust Perfect Adaptation in Bacterial Chemotaxis through Integral Feedback Control." *Proceedings of the National Academy of Sciences* 97, no. 9 (April 25, 2000): 4649–53.