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The Origin of a Genome through Spontaneous Symmetry Breaking: A Computational Modeling Study

N. Takeuchi[1*], P. Hogeweg[2], and K. Kaneko[1]

[1] Department of Basic Science, Graduate School of Arts and Sciences, the University of Tokyo, Japan, [2]Theoretical Biology and Bioinformatics Group, Utrecht University, the Netherlands,

* takeuchi@complex.c.u-tokyo.ac.jp

Understanding the origin and evolution of heredity is a fundamental challenge in biology. The heredity of the modern cell has two universal features. First, there is a functional differentiation between templates and catalysts: a genome is distinct from enzymes. Second, there is a copy-number differentiation between templates and catalysts: templates are less abundant than catalysts per cell. How did such differentiation first arise in primordial cells (protocells, for short)?

Here, we demonstrate the possibility that the first, primordial form of such differentiation arose from spontaneous symmetry breaking between complementary strands of replicating molecules. The key element of our modeling is the consideration of a conflict between evolution at the cellular level and evolution at the molecular level, which arises from a fundamental trade-off between templates and catalysts. Specifically, evolution at the molecular level tends towards the emergence of selfish replicators, whereas evolution at the cellular level tends towards the promotion of catalytic cooperation. We demonstrate that this evolutionary conflict induces the spontaneous symmetry breaking between complementary strands of replicating molecules, whereby one strand becomes catalytic and increases its copy number—like enzymes—whereas the other strand becomes non-catalytic and decreases its copy number—like a genome (Fig. 1). This is a surprising result because the model incorporates no apparent selection pressure for the symmetry breaking. Either molecular-level or cellular-level evolution alone is incapable of explaining the asymmetry. When combined, however, they bring a new dimension to their evolutionary actions, which is orthogonal to the prebuilt axis of selfishness versus cooperation. Moreover, we show that the resulting genome-like molecules reduce the aforementioned evolutionary conflict, thanks to their small copy-number. Thereby, the genome-like molecules provide long-term stability to the genetic information of protocells. Finally, we verified that our conclusions are applicable to the *in vitro* replication system envisaged in the experimental study of Sczepanski and Joyce [1], demonstrating a greater potential for the experimental testing of our work.

[1] Sczepanski JT and Joyce GF (2014) *Nature* 515:440–442.

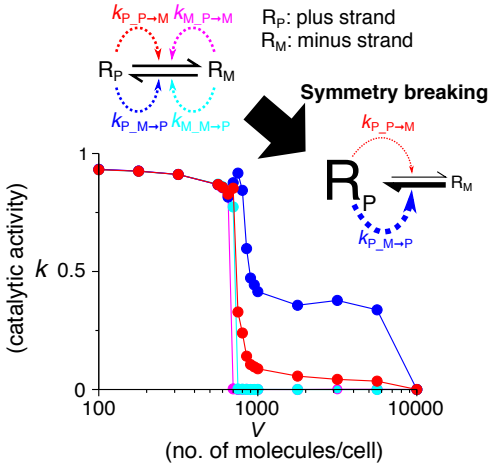

Figure 1 – Spontaneous symmetry breaking between complementary strands of replicating catalytic molecules.