The Lenski Long-Term Evolution Experiment, an example of Devolution

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Abstract

The Lenski Long-Term Evolution Experiment is an example of devolution or retrograde evolution. His bacteria are in an energy-limited environment forcing those bacteria to maintain only those biologic functions necessary to allow survival and replication. This results in the bacteria losing their cell walls because the production of these walls requires energy. This raises the question, does an environment exist where these cell walls can progressively evolve?

Keywords: hypothesis disease; rls ; propranolol

Introduction

Devolution, or retrograde evolution is discussed by Vitas and Dobovišek in reference [1]. In that paper, they "discuss the role of Darwinian selection in evolution and pose the hypothesis that Darwinian selection acts predominantly as a retrograde driving force of evolution. In this context we understand the term retrograde evolution as a degeneration of living systems from higher complexity towards living systems with lower complexity." If one looks at the Wikipedia article on "Devolution", we get the following explanation of the process: "Devolution, de-evolution, or backward evolution (not to be confused with dysgenics) is the notion that species can revert to supposedly more primitive forms over time. The concept relates to the idea that evolution has a purpose (teleology) and is progressive (orthogenesis), for example that feet might be better than hooves or lungs than gills. However, evolutionary biology makes no such assumptions, and natural selection shapes adaptations with no foreknowledge of any kind. It is possible for small changes (such as in the frequency of a single gene) to be reversed by chance or selection, but this is no different from the normal course of evolution and as such de-evolution is not compatible with a proper understanding of evolution due to natural selection." [2]

An Example of Devolution: The Lenski Long-Term Evolution Experiment (LTEE) [3] is an example of devolution or retrograde evolution. This is because the energy-limited environment that the Lenski team subjects his populations to causes his populations to evolve to a state where they use the minimum amount of energy to survive and replicate. The populations are losing the ability to produce a cell wall because the production of the cell wall puts an energy burden on those members of the population and the cell wall in this environment is not needed for survival and replication. This process is driven by the first and second laws of thermodynamics. The first law, conservation of energy, applies because population growth is limited by the energy (glucose) supplied. The second law applies to the descent with modification where replications give the possibility of random (adaptive) mutations occurring. This is an entropy-producing process. The mathematics of this entropy-producing process is described in reference [4].

The biological competition that these E. Coli bacteria are doing is causing a loss of cell wall production in the more fit members of the populations in this experimental environment. It is known that the bacterial cells in the LTEE are increasing in size. [5] At the same time, the bacteria are becoming more osmotically fragile. [6] The genes that form cell walls in the bacteria are losing their function to be more re-productively fit in this environment.

Retrograde Evolution vs Progressive Evolution: The LTEE is demonstrating retrograde evolution with the loss of the bacterial cell wall. Can these bacteria that have performed a retrograde evolutionary process be "progressively" evolved to their previous state where they have a cell wall? It is known that

the Lenski team keeps a history of this evolutionary process by freezing mixed population samples every 500 generations (75 days). [7] What would be the selection pressure to do this progressive evolutionary process? The obvious answer would be osmotic stress. Do any of his stored populations that have mutated portions of the genome that codes for cell wall production have those mutated portions mutate back to fully functional cell wall producing genes? If that is possible, is it possible for all the stored populations or does the evolutionary process become impossible after a certain number of generations of devolution? What happens to these genes in these bacteria later in the experiment? Have so many mutations accumulated in these genes that they serve no biologically functional process? Can these genes be mutated back to functional status by Darwinian evolution? Would osmotic stress be an adequate selection pressure to accomplish this progressive evolutionary process? Can bacteria that are known to be able to produce a cell wall but have evolved away that capability, re-evolve the capability of producing a cell wall?

Discussion:

This principle of retrograde evolution is important to understand because it is this principle

that allows for the evolution of drug resistance. If one considers that random mutations change the genetic sequence by disordering it (slightly), this in turn disorders the protein being produced changing the conformation of the protein slightly. This change in the conformation of a protein not only changes its shape, it changes the molecules that make up that protein. Those molecules have electrical charge and that shape and electrical charge of the protein determine the binding properties of other molecules to that protein. An antibiotic must bind to a protein for it to be effective. If mutations change the shape and electrical properties of a protein, that will affect the ability of an antibiotic to bind to the protein. This puts biochemists into competition with the process of retrograde evolution for antibiotics that still can bind to a retrograde evolved protein. This is not a hopeless competition because this selection condition being used to fight infections causes a "deformation" of a protein used by bacteria to carry out some metabolic function. This devolved protein does not function as well as the original un- mutated protein. An example of this is seen with devolved HIV viruses that are not as efficient replicators as the "wild type" viruses. The selected devolved variants are only better replicators than the "wild type" in an environment with the anti-viral agent. This effect is also seen in the LTEE were drug-resistant variants in his founder population are selected out by the energy-limited environment which favors the drug-sensitive variants.

Conclusion:

The devolutionary process demonstrated by the LTEE is important to understand. It is this

same process that causes the devolution of drug-resistant microbes. Understanding this process helps the clinician and researcher in using and developing selection pressures to combat infectious diseases.

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