Mechanisms of Adaptation to Periodic Environmental Change

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Despite Darwin's pluralistic view that "natural selection has been the main, but not the exclusive means of modification" (Darwin, 1872) (quoted in (Lewontin and Gould, 1979)), subsequent major developments in evolutionary biology such as the theories of natural selection, the modern synthesis, and molecular neo-darwinism have biased scientific explanations of evolution towards gene-centric theories (Jablonka and Lamb, 2005). There is a considerable amount of debate concerning the role of non-genetic mechanisms in evolutionary biology (e.g. (Oyama et al., 2001)) and while there is a movement towards recognition of the complexity of processes of evolution and development (e.g. theories of multiple group selection, major transitions, niche-construction, etc.) a fundamental question remains unanswered: Are non-genetic mechanisms of adaptation and inheritance ultimately compatible with gene-centered theory such as Dawkins' extended phenotype, or are these phenomena fundamentally incompatible with such reductive views? To resolve this debate, we need to better understand the relationships and interactions between concurrent mechanisms of change and adaptation that are realizable in evolving systems. To explore these relationships, we are developing a minimal computational model.

Our model has much in common with typical evolutionary robotics models but with a significant modification, the elimination of the trial-based GA. Instead of evaluating genomes and modifying the gene-pool based upon the results of the evaluation, we utilize an embedded simulation of evolution. Simply summarized, agents co-inhabit their environment with their parents. At the end of a trial in a typical GA, all non genetic elements are eliminated, but in our model other properties (e.g. the spatial distribution of the population) may continue to exist independently of the genetic system.

Methods

The model consists of a simulated 2D toroidal arena in which two-wheeled low-inertia agents are situated. In the arena are two stationary regions of food, one green and one red. Upon contact with a region of food the agents are considered to be consuming it and their health increases or decreases according to sinusoidal functions that oscillate with period $\rho * L$ where ρ is an experimental parameter representing the rate of environmental change and $L = 5000$, the maximum possible agent-lifespan in iterations. The functions are in anti-phase so that when the green food is at peak nutritional value the red food is at peak toxicity and vice versa. The changing food toxicity levels were included not as a simulation of particular biological phenomena, but to make possible the observation of different mechanisms of adaptation while experimentally varying the rate of environmental change. To encourage efficient motion, the health of all agents is steadily decreased at a rate linearly proportional to the rate of both motors. Agent health can not increase above the maximum value of 1, and a health of 0 or less means death (removal from the simulation).

The rate of the agents' motors is determined by the output of a continuous time recurrent neural network CTRNN (as described in (Beer, 1995)). Acting as inputs to the CTRNN are two forward mounted sensors capable of perceiving food regions and other agents. The sensors are distance sensitive and provide a high input for proximal sensed entities. These sensors provide a total of six inputs to the CTRNN: agent proximity (left & right), red food proximity (left & right), and green food proximity (left & right). A stomach-sensor acts as the seventh and final input to the CTRNN, indicating the relative health/toxicity value of any food currently being consumed. The CTRNN is fully interconnected and has two hidden nodes and two output nodes (left and right motor activation). All inputs are connected to all nodes.

Agents have a maximum lifespan of 5000 iterations. Agents older than 3750 iterations that have procreated less than 2 times have a chance ($p = 1.5/n^2$ where n is the number of living agents) of procreating. An agent's procreation is simulated by creating a mutated duplicate of the parent agent's genome, generating an agent from this genotype, and inserting the new child agent into the simulation in

Food Eaten	$\rho = 2$	$\rho = 10$	$\rho = \infty$	t-test	
Green	$0.74, \sigma = 0.04$	$0.60, \sigma = 0.21$	$0.11, \sigma = 0.13$	$p < 0.001$	A
Red	$0.75, \sigma = 0.04$	$0.45, \sigma = 0.25$	$0.85, \sigma = 0.15$	N/A	B
Healthy	$0.64, \sigma = 0.10$	$0.32, \sigma = 0.21$	$0.35, \sigma = 0.15$	$p < 0.001$	C
Toxic	$0.86, \sigma = 0.06$	$0.74, \sigma = 0.30$	$0.60, \sigma = 0.13$	N/A	D

Table 1: Statistics concerning populations evolved in environments with different ρ (rate of environmental change). The t-test column shows the results of a t-test performed on the highlighted figures for the given row.

proximity to its parent. The agent's genotype determines only the parameters of the CTRNN nodes.

When an extinction occurs we reset the simulation with a snapshot taken from the longest surviving populations ever found in that simulation. In using this seeding scheme, we can perform a somewhat directed search for organizations that excel at enduring in the environment that we have defined.

Periodic Environmental Change

To explore the mechanisms of change and adaptation within our model, we have experimented with the parameter ρ, the rate of environmental change. Different values of ρ produce populations that use different mechanisms to adapt to their environment.

Populations evolved in a $\rho = 2$ environment appear to utilize their ability to sense the toxicity of the food they are consuming to switch foods as the food currently being consumed becomes toxic.

The agents evolved in a $\rho = 10$ environment appear to take advantage of spatial properties of the population, namely the high chance of agents being born near the food that is healthy. The agents tend to circle, periodically feeding on whichever food they are born near. Occasionally a newly born agent will encounter the other, non-populated food. If it is toxic they will die, but if it is healthy the agent will feed and procreate, starting a population at the previously unpopulated food. As the original food becomes toxic the agents remain, eating the toxic food until they die.

The $\rho = \infty$ environment produces a population that does not move around the arena much, but instead appears to have a genetic predisposition, moving towards and then resting upon the healthy food.

Table 1 shows statistical evidence that the mechanisms described above are indeed those being employed by the evolved populations. The data were gathered by first evolving ten populations in each of four different $\rho = [0.1, 2, 10, \infty]$ environments. Individuals were then tested in a scenario in which they were made incapable of procreation and death and then placed close to toxic food and allowed to navigate through the environment for 5000 iterations (the agents maximum lifespan). This was repeated 500 times per population, alternating the colors of the food for each trial. The table indicates the frequencies of consumption of different classes of food by the agents.

The t-test result in row A in the table support the notion of a genetic predisposition present in the $\rho = \infty$ population. These populations experiment much less, consuming significantly less green food than the other populations. The t-test result in row C indicates an increased propensity in the $\rho = 2$ agents to try different food, and the $\rho = 10$ agents tend to consume the food that they start near (the toxic food). Both of these attributes support the mechanisms of adaptation hypothesized above.

Summary

We have developed a model of embedded evolution that allows for non-genetic mechanisms to exist for periods of time longer than the maximum lifespan of an agent. This model has enabled our study of concurrent mechanisms of adaptation. This exploration of the interactions between concurrent mechanisms of adaptation has demonstrated that the rate of periodic environmental change is highly relevant in the determination of the mechanism of adaptation employed by an evolving system. We expect that further study in this vein will help to develop our understanding of non-genetic mechanisms of inheritance and adaptation and their relationship to the genetic system.

References

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