Evolutionary Models for Maternal Effects in Simulated Developmental Systems

Artur Matos
amatos@create.human.nagoya-u.ac.jp

Reiji Suzuki
reiji@is.nagoya-u.ac.jp

Takaya Arita
arita@nagoya-u.jp

Graduate School of Information Science
Nagoya University
Furo-cho, Chikusa-ku, Nagoya 464-8601, Japan

ABSTRACT

Maternal influence on offspring goes beyond strict nuclear (DNA) inheritance: inherited maternal mRNA, mitochondria, caring and nurturing are all additional sources that affect offspring development, and they can be also shaped by evolution. These additional factors are called maternal effects, and their important role in evolution is well established experimentally. This paper presents two models for maternal effects, based on a genetic algorithm and simulated development of neural networks. We extended a model by Eggenberger by adding two mechanisms for maternal effects: the first mechanism attempts to replicate maternal cytoplasmic control, while the second mechanism replicates interactions between the fetus and the uterine environment. For examining the role of maternal effects in artificial evolution, we evolved networks for the odd-3-parity problem, using increasing rates of maternal influence. Experiments have shown that maternal effects increase adaptiveness in the latter model.

Categories and Subject Descriptors: I.2.6 [Artificial Intelligence]: Learning — Connectionism and neural nets

General Terms: Algorithms

Keywords: Genetic Algorithms, Neural Networks, Maternal effects, Artificial embriogeny

1. INTRODUCTION

Maternal influence on offspring goes beyond strict nuclear (DNA) inheritance: inherited maternal mRNA, mitochondria, caring and nurturing are all additional sources that affect offspring development, and they can be also shaped by evolution [3]. These additional factors are called maternal effects, and their important role in evolution is well established experimentally. There are also several theoretical models based on quantitative genetics, but in this paper we will use a different modelling approach. In this paper, we introduce two maternal effects models, based on a genetic algorithm and simulated development of neural networks. Both attempt to replicate maternal influence at the molecular level, i.e. due to chemical exchange and affected gene regulation. For examining maternal effects in artificial evolution, we evolved networks for the odd-3-parity problem, using both models, and with increasing rates of maternal influence.

GECCO'05, June 25–29, 2005, Washington, DC, USA.
ACM 1-59593-010-8/05/0006.

2. THE MODELS

We used a simulated developmental model for neural networks, based on gene regulation and cell communication. Our developmental model is based on a previous one by Eggenberger [1]. Development proceeds in a rectangular grid, each slot possibly having a neuron. Chemicals are present in the grid, generated by gene activity inside each neuron, and that diffuse through the grid. All the neurons contain the same genome, but different parts of the genome may be activated in each neuron, due to interactions through chemical diffusion. The genome is organized in an operon-like structure with structural and regulatory regions. Structural regions are responsible, if activated, for generating chemical substances. Regulatory regions activate the associated structural regions, depending on the substance's concentration in the neuron. One regulatory region controls several structural regions, with the number of structural regions per regulatory parts fixed. In contrast to Eggenberger, our model does not simulate synapse growth. Instead, the connections are established by receptors expressed in the surface of the neurons, that connect to each other whenever they have a strong affinity between them.

A genetic algorithm (GA) is used for evolving the genomes, and also for defining the maternal/offspring roles. Only reproduction with optional mutation is used, and therefore all individuals between subsequent generations are always connected by a reproduction operation. This connection is used for defining their role: for instance, an individual in generation m connected to another individual in generation $m+1$ takes the maternal role for that individual in generation $m+1$.

A diagram for both models is shown in figure 1. Development of the networks occurs in discrete time steps, for dt time steps. In both models, there is an initial period of maternal influence, from 0 to mt time steps. The models represent two distinct mechanisms for maternal influence. The first one is based on maternal cytoplasmic control (figure 1 a)): in all metazoans, the first developmental stages are under control of maternal gene products, deposited by the mother during oogenesis. Only after several cell divisions does the real zygotic genome takes control. In a similar

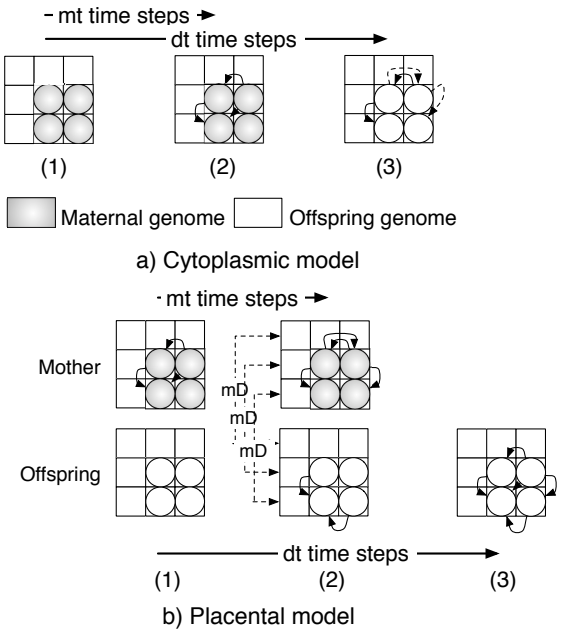

Figure 1: Diagram of both models.

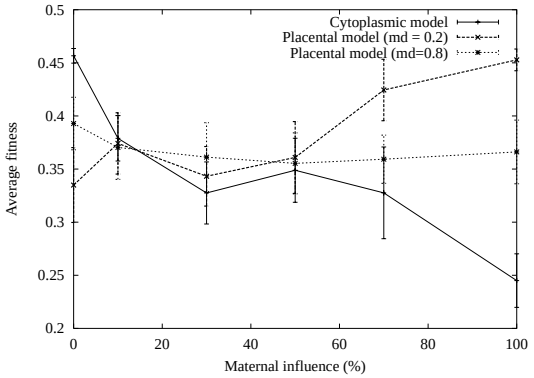

Figure 2: Average fitness value for the runs, classified by increasing maternal influence (average value + standard error).

way, in our cytoplasmic model, the maternal genome is used exclusively in all the cells of the grid until mt time steps are reached (1 - 2). Afterward, the offspring's genome replaces the previous genome in all the cells and guides the remaining development (2 - 3). The second model is based on placental interactions that occur during mammal growth (figure 1 b)): in mammals, early cytoplasmic control is reduced, but there is a significant interaction between the mother and the fetus through the placenta. This allows them to exchange chemicals that affect development in both mother and offspring. Our model attempts to reproduce this, by growing both mother and offspring in parallel: The offspring network starts from 0, while the mother's development is resumed from the previous point (dt) at which she matured. During mt time steps, their development occurs concurrently, and cells with the same position exchange chemicals with a fixed rate (mD) per time step (1 - 2). After this stage, development occurs for the offspring as usual, without any further maternal influence (2 - 3).

3. EXPERIMENTS AND RESULTS

For examining how these models affect evolution, we evolved networks for the 3-odd-parity task. The solution is defined as a neural network with at least 3 inputs, that outputs *true* whenever the number of *true* inputs is odd. We used boolean neural networks with thresholds being either 0 or 1, and the connections being either -1 or 1. All the grids were initialized with the required minimum neuron configuration for this problem, and therefore the GA only had to evolve the connections. We used a fitness function used by Gruau in [2], based on mutual information. This fitness function is defined in the range $[0, 1]$ with 1 as the best value.

We conducted three sets of experiments: one using the cytoplasmic model, and two using the placental model with two different mD values (0.2 and 0.8). For understanding the role of maternal effects in network development, in each set we conducted experiments with dt fixed at 30 time steps, and used increasing values of mt. The used mt values correspond to periods of initial maternal influence for 0%, 10%, 30%, 50%, 70% and 100% of the total developmental time (mt/dt). Each case was conducted 10 times, with different random seeds in each run. The experiments were conducted with the ECJ (Evolutionary Computation in Java) package.

For investigating any influence in adaptiveness, we computed the average fitness over all generations, in all runs sharing the same mt parameter, with the results depicted in figure 2. All the three experiment sets exhibit different effects on adaptiveness, with the two different models showing, in fact, opposite effects: the cytoplasmic model has an overall negative adaptive effect, while the placental model shows a positive one. For the $mD = 0.2$ experiments, maternal influence above 70% shows a roughly 30% increase in average fitness, a significant improvement. For $mD = 0.8$, however, this influence becomes stale, probably due to the mD value being too high.

In our opinion, increasing maternal influence may be positively affecting: 1) sensitivity to mutations, 2) development, or 3) selection response. For checking the first hypothesis, we performed mutations in the generated individuals, and computed the average distance to mutations, using the number of different links between networks. Our preliminary analysis has shown that while sensitivity to mutations changes with increasing maternal influence, this is not related to the fitness increase. As for the second hypothesis, the placental model may be positively affecting development, by increasing diversity in the connections between neurons. We are currently checking this hypothesis, and also if delay in selection response occurs in these models.

4. REFERENCES

[1] P. Eggenberger, G. Gomez, and R. Pfeifer. Evolving the morphology of a neural network for controlling a foveating retina - and its test on a real robot. In R. K. Standish, M. A. Bedau, and H. A. Abbass, editors, *Proceedings of the Eighth International Conference on Artificial Life*, pages 243–251. MIT Press, 2002.

[2] F. Gruau. *Neural Network Synthesis using Cellular Encoding and the Genetic Algorithm.* PhD thesis, Laboratoire de la Informatique du Parallelisme, Ecole Normale Superieure de Lyon, 1994.

[3] M. Rossiter. Incidence and consequences of inherited environmental effects. *Annual Review of Ecology and Systematics*, 27:451–476, November 1996.