# **Evolution of Heterogeneous Cellular Automata in Fluctuating Environments**

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#### **Abstract**

The importance of environmental fluctuations in the evolution by natural selection of living beings has been widely noted by biologists and linked to many important characteristics of life such as: modularity, plasticity, genotype size, mutation rate, learning or epigenetic adaptations. However, in artificial life simulations, environmental fluctuations are usually seen as a problem to be solved rather than an essential characteristic of evolution. We propose in this paper to use HetCAis, a heterogeneous cellular automata characterized by its ability to generate open ended long-term evolution and evolutionary progress, to measure the impact of different forms of environmental fluctuations. Our results indicate that environmental fluctuations induce mechanisms analogous to epigenetic adaptations in HetCA.

## Introduction

Environmental changes may include cyclic events such as seasonal changes or a daily cycle of light and darkness, occasional changes such as the introduction of new predator or the potential for a new source of food, or much more radical modifications such as environmental stresses induced by climate change.

Early population genetics theory assumed the environment to be constant. However, since then, work such as (Levins, 1968) or more recently (Jablonka et al., 2014), as well as the integration of the epigenetic (EIS), developmental (the "Evo-Devo" school?) and environmental (as done by poeple studying "Niche Construction" mechanisms Laland et al. (2016) dimensions to evolutionary biology, have put the emphasis on the importance of environmental fluctuations in the evolution by natural selection of living beings. Through these and other works many central questions about the mechanisms of evolution such as modularity, plasticity (West-Eberhard, 2005), size of the genotype, mutation rate and evolvability have been linked to environmental changes. Jablonka et al. (2014) stress that a changeable environments will unmask variations in the capacity of individuals to make adjustments to changed conditions and therefore promote plasticity. They advance that "For a lineage in a constantly changing environment, switching among several alternative heritable states was probably an advantage. While cells in one state survived in one set of conditions, those in other states did better in different circumstances." (*ibid. p. 318*). For them, constantly changing environment or cycling variations might explain the origin of the Epigenetic Inheritance Systems Heard and Martienssen (2014) as epigenetic mutations are more reversibles and occur more frequently than genetic mutations.

Among the work done on the topic, Lachmann and Jablonka (1996) have modelled the effects of cycling variations, such as seasonal or daily cycles, on phenotypical inheritances. Their model predicted that when the investigated cycles are longer than the reproductive cycle but relatively short otherwise, heritable variations produced by non-DNA inheritance systems are likely to be observed.

On the other hand a proportion of existing works in artificial life, especially in evolutionary robotics (Floreano and Urzelai, 2000), considers the issue of environmental variations. Some in this literature have explicitly define the environment as a driving evolutionary force (Bredèche and Montanier, 2012). However and as far as we know, a significant majority of the works in that field still consider environmental changes only as a problem to be solved and nobody has systematically studied the general properties induced by environmental fluctuation over evolutionary dynamics.

Nonetheless Lipson et al. (2002) demonstrated a correlation between the modularity and the rate of change of the environment resources. While in Yu (2007) observed that populations exploit neutrality to cope with environmental fluctuations and therefore evolve some sort of evolvability under two alternating objective functions. Both of these simulations used Genetic Programming (GP) and employed explicit fitness functions.

Taking some of the observations made in those two last studies, we propose an experimental setup that allows to quantitatively measure the influence of cyclic environmental fluctuations on the course of the evolution of a Cellular Automata (CA). We show that such fluctuations lead to the emergence of processes similar to those exhibited by epigenetic inheritance systems.

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The paper is organized as follow. Section ?? explains the general mechanisms of HetCA simulation and the implementation of environmental fluctuations in HetCA is then detailed in Section . Section describes the computer experimental setup while we report experimental results in Section ??. We discuss the implications of these results in Section ??. Finally, Section concludes the paper.

#### **HetCA**

HetCA is based on classical two-dimensional Cellular Automata (CA), but has several additional features: cells have age, decay and quiescence properties; cells utilize a heterogeneous transition function inspired by linear genetic programming (LGP); and a notion of genetic transfer of transition functions between adjacent cells has been implemented.

HetCA has been shown to exhibit long term phenotypic dynamics, high level of variance over very long run, a greater behavioral diversity than classical CA and evolves progress other three criteria (robustness size and density) given the Shanahan (2012)'s definition of Evolutionary Progress (Medernach et al., 2015).

Moreover, some emergent properties of HetCA are similar to two of the five major eukaryotic innovations which do not appear to have direct prokaryotic predecessors defined in (Smith and Szathmary, 1997) as: (1) the eukaryotic chromatin remodelling machinery; (2) the cell cycle regulation systems; (3) the nuclear envelope; (4) the cytoskeleton; and (5) the apoptosis apparatus (Koonin and Aravind, 2002). Indeed, in HetCA, the loss of the genotype of the cell as it turns to the quiescent state may seem similar to apoptosis while survival strategies such as the ones depicted in Figure 1 might akin cell cycle regulation systems

Finally, controversy over the units of selection<sup>1</sup> in evolutionary biology (Okasha, 2006) are numerous and date back to the origins of this field and there is potentially several units of selection in HetCA: genotypic selection of the transition rules but also phenotypic selection of cell group replicating patterns that one can also find in cellular automata such as game of life. This issue is important when one is interested in environmental fluctuations because, as mentioned in introduction, it is anticipated that the existence of frequent environmental fluctuation could promote phenotypic selection compared to genotypic selection.

## **Experimental Setup**

## **Environmental Fluctuations**

As in the previous version of HetCA the genotype of an individual is its transition rules encoded with CA-LGP using the function set depicted in Table 1. The mutation of genotypes is enabled using the Micro/Marco-mutation of CA-LGP as described in (Medernach et al., 2013).

op. name	<b>action</b> on inputs $(x, y)$			
abs	x			
plus	x + y			
lelta	1, if $ x-y  < 1/10000$ ; 0 o.w.			
dist	x-y			
inv	1-x			
inv2	safeDiv(1, x)			
magPlus	x+y			
max	$\max\{x,y\}$			
nin	$\min\{x,y\}$			
safeDiv	x/y if $ y  > 1/10000$ ; 1 o.w.			
afePow	$x^{y}$ , if defined; 1 o.w.			
hresh	1, if $x > y$ ; 0 o.w.			
imes	xy			
ero	1, if $ x  < 1/10000$ ; 0 o.w.			

Table 1: Function set.

To introduce environmental variations, where the new genotype of a cell was originally randomly chosen among candidate genotypes, we vary the likelihood of spread of the genotype of a cell according to the state of this cell. In this new setup, the chances P(c) of the candidate genotype of the cell c to be selected are then:  $P(c) = K(S_c) / \sum_{i=1}^n K(S_{c_i})$  with  $S_c$  state of the cell c, K(S) likelihood of spread of state S and n number of candidate genotypes. Therefore an environment E is characterized by the odds of propagation of the five living states  $E = \{K(S_1), K(S_2), K(S_3), K(S_4), K(S_5)\}$ . To mimic environmental fluctuations we initialise the simulation with  $K(S_i) = 1 \forall i \in [1, 5]$  and then we regularly change those values every f iterations from iteration 3000 of the cellular automata.

We chose to introduce four forms of environmental fluctuations described in Table 2.

Short-cycle Fluctuation: consists of alternating between two environments every 100 iterations of the cellular automaton. We chose to vary the environment every f=100 iterations to stay in the same range of frequency as described in Lispson (Lipson et al., 2002) examples 20 and 100 generations and (Yu, 2007) experiments 10, 20 and 50 generations. In fact we consider that a successful reproductive cycle involves passing a cell through the quiescent state. And this should take between two iterations (alternating between the quiescent state and a living state) and seven iterations (if a cell remains in a living state more than seven consecutive iterations it goes to the decay state and can not receive a genotype for an important period).

Light Fluctuation: consists of alternating between five environments every f=5000 iterations of the cellular automaton. The first five each prohibit a different state from the five living state, the latter gives equal chance to each of the five states.

Strong Fluctuation: consists of alternating between twelve environments every f=5000 iterations of the cellular automaton. The first eleven each prohibit a different combination of two states from the five living states, the latter gives equal chance to each of the five states.

<sup>&</sup>lt;sup>1</sup>Genotype selection, Phenotype selection, epigenetic selection, comportemental selection, multilevel selection ?...

Table 2: Environments.

Name	Short Name	Cycles	Transitions	Environment list
Stable Environment	[SE]	NA	NA	$\{1,1,1,1,1\}$
Short-cycle Fluctuations	[ScF]	100	1	$\{1, 1, 1, 0, 0\}, \{1, 1, 1, 0, 0\}$
Light Fluctuations	[LF]	5000	1	$\{1, 1, 1, 1, 0\}, \{1, 1, 1, 0, 1\}, \{1, 1, 0, 1, 1\}, \{1, 0, 1, 1, 1\}, \{0, 1, 1, 1, 1\}, \{1, 1, 1, 1, 1\}$
Strong Fluctuations	[SF]	5000	1	$ \begin{array}{l} \{0,0,1,1,1\}, \{1,1,1,0,0\}, \{0,1,0,1,1\}, \{1,0,1,1,0\}, \{0,1,1,0,1\}, \{1,1,0,1,0\}, \\ \{1,0,1,0,1\}, \{0,1,1,1,0\}, \{1,0,0,1,1\}, \{1,1,0,0,1\}, \{1,1,1,1,1\} \end{array} $

Parameter	Value
Number of living states	5
Successive living iterations before decay	7
Number of iterations for decay	375-1875
Direct transition to decay	enabled
Size of the grid	500x500
Grid boundaries	toric grid
Transition Rule (TR)	CA-LGP
Maximum (TR) size	50 program
	statements
Genotype copy neighbouring	Von Neumann
Transition rule neighbouring	Moore

Table 3: **HetCA parameters**.

Short-cycle fluctuation may be analogous to the circadian rhythms for some bacteria: very regular cycles in which these organisms have enough time to reproduce several times. While *light fluctuation* may be similar to seasonal fluctuations and *strong fluctuations* would akin ecological crisis. Although, owing to the variety of both biological temporal rhythms and reproductive cycles, the relevance of these analogies may be limited.

#### **Simulation & Results**

For each form of environmental fluctuation and the stable non fluctuating environment, we performed 50 simulations, each on 500000 iterations with the parameters listed in Table 3. We report average value with standard deviations.

### **Genotype Size**

We use the number of program statements  $(n_{prog})$  as a measure of the genotype size and compute the average size of all the current genotypes of a run every 2500 iterations. We then report the average and standard error among all the fifty runs sharing the same settings.

#### **Changes induced by environmental fluctuation**

If, as postulated in (Jablonka et al., 2014), the introduction of environmental changes leads to the selection of plasticity of individuals mechanisms or, to the emergence of phenotypic selections<sup>2</sup> using easily reversible phenotypic mutations; then phenotypes from different individuals of the same lineage observed while environmental conditions are similar should be relatively close even though, between these measures, individuals from their lineage evolved in other environmental conditions; while one may think that if the

adaptation to each environmental change is done exclusively through the selection of classical irreversible genotypic mutations, these phenotypes shall be quite different, despite the potential evolutionary convergence. That is why we want to develop a metric to measure phenotypic proximity between two iterations of the CA. To do this we simply compare the proportions of living cells in different states among the possible states. The phenotypic difference  $\sigma(t_1,t_2)$  between two iterations  $t_1$  and  $t_2$  is then calculated as:

$$\sigma(t_1, t_2) = \sum_{s=1}^{5} \left| \frac{N(s, t_1)}{\sum_{s=1}^{5} N(s, t_1)} - \frac{N(s, t_2)}{\sum_{s=1}^{5} N(s, t_2)} \right|$$

where N(s,t) is the number of cells at the state s for iteration t.

Every 5000 iterations t of the CA we perform two phenotypic comparison with an iteration t' such as  $t' \in [t-55000; t-60000]$ . The first phenotypic comparison aims to compare two similar environment and therefore we choose t' such as E(t') = E(t). The second phenotypic comparison aims to compare two disimilar environment and therefore we choose t' such as E(t') = E(t+f).

## **Density**

We also collect the *most common genotype* (most frequently occurring) in iterations 2500<sup>3</sup>, 102500, 202500, 302500, 402500 and 500000.

#### **Regular phenotype characterization**

In HetCA, to survive in the long term the genotypes must regularly release cells by transforming them into quiescent cells without genotypes. This generates patterns and cycles which are quite easy to observe in homogeneous simulations where a single genotype is tested as shown in Figure 1, but also observable, although with more difficulty, in normal, heterogeneous, HetCA simulations as seen in Figure 2. That is why, characterize phenotypes, we found it useful to measure these cycles as well as out-of regularities. At every iteration t>8 of the homogenous genotype test, we compare the states  $S_t$  and  $S_{t-1}$  of each cell to their anterior state during the eight previous iterations of the CA. We then measure whether this sequence of two states is repeated during these

<sup>&</sup>lt;sup>2</sup>Similar to the epigenetic inheritance system

<sup>&</sup>lt;sup>3</sup>(Medernach et al., 2015) has shown that the most common genotypes during the first iterations of HetCA were unlikely to have a viable survival strategy, therefore we chose to collect genotypes from iteration 2500.

eight previous iterations, and if so what is its periodicity psuch as  $p = \min p \in [2, 7], S_t = S_{t-p} \wedge S_{t-1} = S_{t-1-p}$ . We use here a sequence of two states because, if the genotype of a cell adopts a stable strategy, i.e. repeats a sequence of states, this sequence must contain a minimum of two states in order to ensure the survival of the genotype - the quiescent state and one of the living states. We have chosen to limit ourselves to a comparison of the eight previous iterations as the limit of the seven consecutive live iterations before decay involves a maximum periodicity of quiescent state of seven iterations. We have chosen to limit ourselves to a comparison of the eight previous iterations to reduce the computational cost and because the limit of the seven consecutive live iterations before decay involves, for a successful regular phenotype, a maximum periodicity of seven iterations for the quiescent state. We perform this measure only if there is at least a living state among the last two states and no state decay. We then report, for each iteration t of the CA, the sum  $\Sigma(p,t)\forall p \in [2;7]$  of cells at every possible periodicity p.

## Phenotype disturbance

To analyze the phenotypic plasticity and l or the existence of phenotypic selection it is necessary to have a measure of the stability of the phenotype. To achieve this, in each iteration l we compare the periodicity l of each cell to its previous periodicity l we then report, for each iteration l of the CA, the number of cells that changed periodicity.

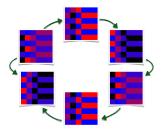


Figure 1: **Six steps survival strategy** from a genotype extracted from a HetCA simulation in a stable environment and tested here in a randomly initialized homogenous CA.

## **Qualitative Analysis**

Analysis

#### **Conclusions**

Conlusion

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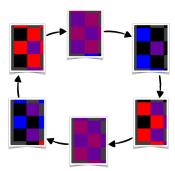


Figure 2: **Six steps survival strategy** from a HetCA simulation with *Short-cycle Fluctuation*.

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