

# Evolution of Heterogeneous Cellular Automata in Fluctuating Environments

First Author<sup>1</sup>, Second Author<sup>1,2</sup>, Third Author<sup>1,2</sup>, Fourth Author<sup>1,2</sup> and Fifth Author<sup>2</sup>

<sup>1</sup>First affiliation

<sup>2</sup>Second affiliation

corresponding@author.email

## 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, and learning or epigenetic adaptations. However, in life simulations, environmental fluctuations are usually considered as a problem to be solved rather than an essential characteristic of evolution. We propose in this paper to use 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.

add citation  
(other than  
jablonka  
maybe  
(hea)?)

## 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 stress induced by climate change.

Early population genetics theory assumed the environment to be constant. However, since then, work such as (8) or more recently (5), 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 (15), size of the genotype, mutation rate and evolvability have been linked to environmental changes. (5) stress that a changeable environment 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 considering that epigenetic mutations are more reversibles and occur more frequently than genetic mutations.

(7) 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.

Moreover, a proportion of existing work in artificial life, especially in evolutionary robotics (4), considers the issue of environmental variations and among that literature some have explicitly define the environment as a driving evolutionary power (bre) but as far as we know, nobody has systematically studied the general properties induced by such environmental fluctuation and most of the other works in that field consider environmental changes as a problem to be solved.

a semble  
un peu  
parachute  
d'enl par  
non? pas  
de lien av  
ce qu'il y  
avant ou a

This paper is organized as follows. Section , we explain the mechanisms of HetCA simulation. The implementation of environmental fluctuation in HetCA is then explained in Section . Section 5 details the computer experimental setup while we reports experimental results in Section . We discuss the implications of these results in Section . Finally, Section concludes the paper.

hypotheses  
and quest

## Background

In (9) Lipson demonstrated a correlation between the modularity and the rate of change of the environment resources. While in (16) Yu used 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.

## HetCA

HetCA is based on classical two-dimensional cellular automata (CA), but has several added features: cells have *age*, *decay* and *quiescence* properties; cells utilize a heteroge-

ion?  
er?  
sure  
at this  
ence..  
ch the in-  
ation of  
epigenetic  
, devel-  
etnmal  
ODEVO)  
envi-  
mental  
CHE  
NTRUC-  
N (lal))  
ension to  
study of  
utionary  
ogy ???

neous transition function inspired by linear genetic programming (LGP); and there exists a notion of genetic transfer of transition functions between adjacent cells.

HetCA exhibits long term phenotypic dynamics, sustains a high level of variance over very long runs and displays greater behavioural diversity than classical cellular automata(11). HetCA also exhibits Evolutionary Progress (EP) according to the three criteria : robustness, size and density of generated genotypes(10), using Shanahan’s definition of EP (13).

Moreover, some emergent properties of HECA are similar to two of the five major eukaryotic innovations which do not appear to have direct prokaryotic predecessors defined in (14) as : the eukaryotic chromatin remodelling machinery; the cell cycle regulation systems; the nuclear envelope, the cytoskeleton; and the apoptosis apparatus(6). 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 (12) are numerous and date back to the origins of this field of research 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

Originally, in HetCA, the new genotype of a cell was randomly chosen among candidate genotypes. In order to introduce environmental variations, we chose to vary the likelihood of spread of the genotype of a cell according to the state of this cell. The chances of the candidate genotype of the cell  $c$  to be selected are then:  $c = K(S_c) / \sum_{i=1}^n K(S_{c_i})$  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 is characterized by the odds of propagation of the five living states  $\{K(S_1), K(S_2), K(S_3), K(S_4), K(S_5)\}$ . 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 from iteration 3000 of the cellular automata.

We chose to introduce four forms of environmental fluctuations described in Table 3.

*Short-cycle Fluctuation*: consists of alternating between two environments every 100 iterations of the cellular automaton.

<sup>1</sup>Genotype selection, Phenotype selection, epigenetic selection, comportemental selection, multilevel selection...

We chose to vary the environment every 100 iterations to stay in the same range of frequency as described in Lispson (9) examples 20 and 100 generations and (16) 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 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 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.

*Gradual Fluctuation*: is similar to strong fluctuation except that it includes a transition phase of  $T = 60$  iterations in between two environments where likelihood of spread of state values progressively switch from the value a previous environment to the ones of the new environment. Over this phase the state spreading is defined by the following formula :  $K(S, t) = K_p(S) \times (T - t) + K_{p+1}(S) \times t$  where  $t$  is the number of iterations completed since the beginning of the transition phase;  $K_p(S)$  and  $K_{p+1}(S)$  are likelihood of state spread  $S$  for the current environment and the next environment respectively.

*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 and gradual 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.

### Common Settings

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 1. The genotypes of an individual are its transition rules encoded with CA-LGP using the function set depicted in Table 2. Mutation of genotypes is enabled and we use the Micro/Macro-mutation of CA-LGP as described in (11).

## Methodology

### 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

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 1: **HetCA parameters.**

op. name	action on inputs $(x, y)$
abs	$ x $
plus	$x + y$
delta	1, if $ x - y  < 1/10000$ ; 0 o.w.
dist	$ x - y $
inv	$1 - x$
inv2	$\text{safeDiv}(1, x)$
magPlus	$ x + y $
max	$\max\{x, y\}$
min	$\min\{x, y\}$
safeDiv	$x/y$ if $ y  > 1/10000$ ; 1 o.w.
safePow	$x^y$ , if defined; 1 o.w.
thresh	1, if $x > y$ ; 0 o.w.
times	$xy$
zero	1, if $ x  < 1/10000$ ; 0 o.w.

Table 2: **Function set.**

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 (5), 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. To do this we simply compare the proportions of living cells in each possible state. The phenotypic difference  $\sigma(t_1, t_2)$  between two iterations  $t_1$  and  $t_2$  is then calculated as:

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

<sup>2</sup>Similar to the epigenetic inheritance system

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

### 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 eight previous iterations, and if so what is its periodicity  $p$  such 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 / 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  $t$  we compare the periodicity  $p_t$  of each cell to its previous periodicity  $p_{t-1}$ . We then report, for each iteration  $t$  of the CA, the number of cells that changed periodicity.

<sup>3</sup>(10) 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.

Table 3: 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	{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}
Gradual Fluctuations	[GF]	5000	60	{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}

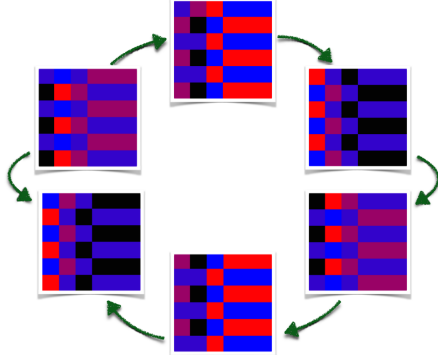


Figure 1: **Six steps survival strategy** from a genotype extracted from a HetCA simulation in a stable environment and tested here in a randomly initialized homogenous CA.

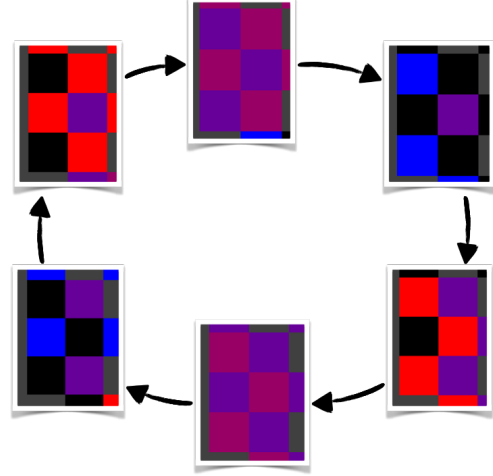


Figure 2: **Six steps survival strategy** from a HetCA simulation with *Short-cycle Fluctuation*.

## Results

### Size

Figure 3 shows genotype size under the various study conditions. The size is bounded (50 is the maximum size) and it probably limits the differences between those environments as most simulations converge towards this limit, however, one can clearly see here that *Short-cycle Fluctuation* restrict the size of the genotypes while other forms of fluctuations do not appear to have any effect on genotype size..

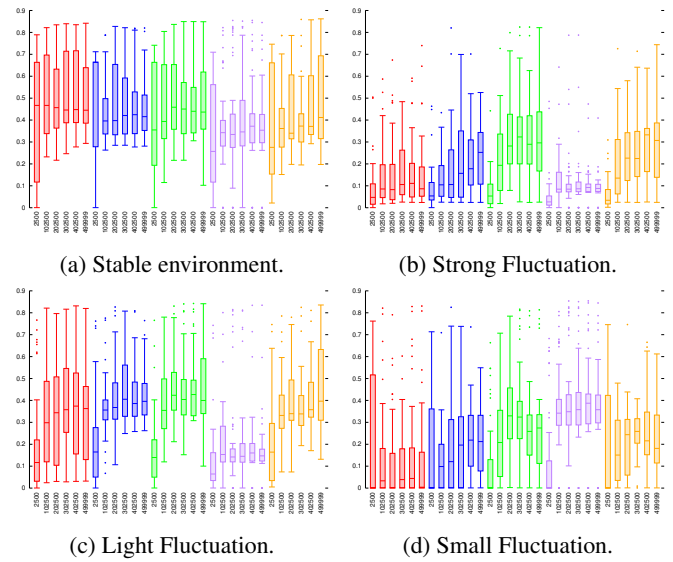


Figure 7: Density of Genotype : Each genotype density is processed in four possible different environments.

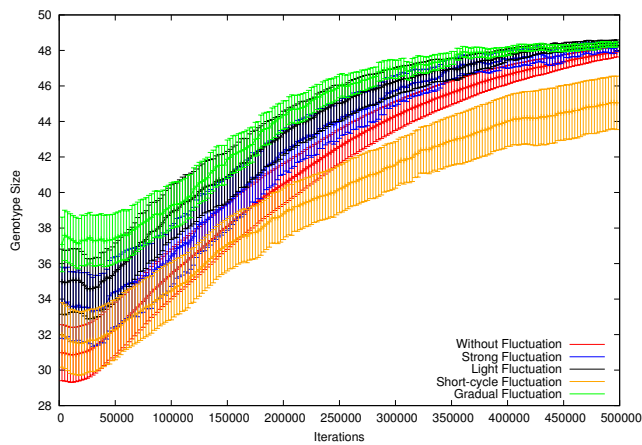


Figure 3: Size of genotypes.

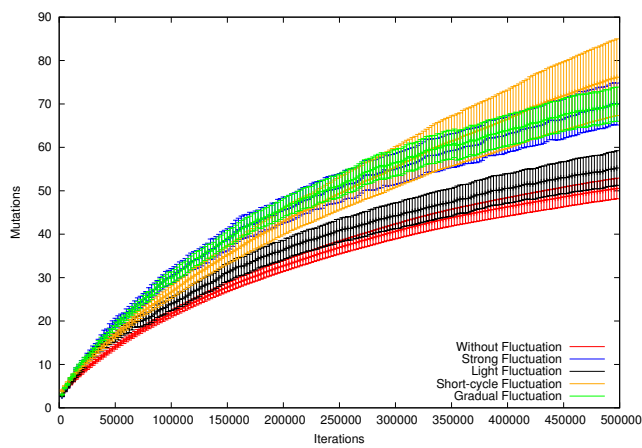


Figure 4: Mutations of genotypes.

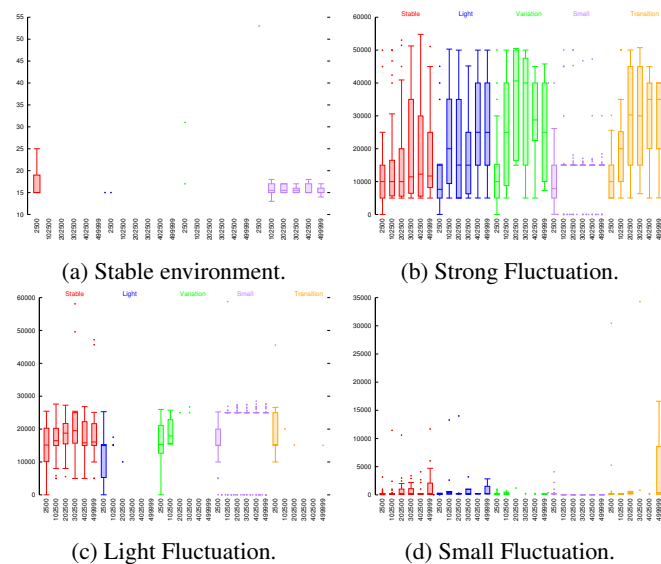


Figure 8: Last iteration with living cells of density runs that didn't reach 60000 iterations. Note that *Short-cycle Fluctuation* genotypes failures are concentrated around iteration 15000 on *Light Fluctuation* density test and around iteration 25000 on *Strong Fluctuation* density test.

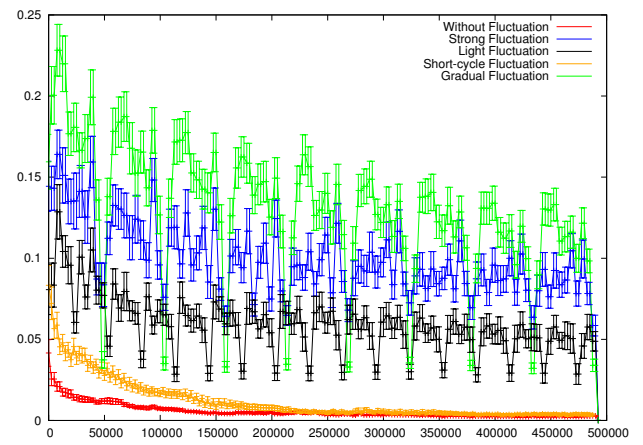


Figure 5: **Changes with following environment** : Here one can see that the impact of environmental fluctuations decreases for *Short-cycle Fluctuation* while it remains very high for other forms of environmental fluctuations.

## Discussion

## Qualitative Analysis

## Conclusions

## Acknowledgement

The authors gratefully the support of Science Foundation Ireland, grant number:

## References

- Floreano, D. and Urzelai, J. (2000). Evolutionary robots with on-line self-organization and behavioral fitness. *Neural Networks*, 13(4):431–443.
- Jablonka, E., Lamb, M. J., and Zeligowski, A. (2014). *Evolution in Four Dimensions, revised edition: Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life*. MIT press.
- Koonin, E. and Aravind, L. (2002). Origin and evolution of eukaryotic apoptosis: the bacterial connection. *Cell death and differentiation*, 9(4):394–404.
- Lachmann, M. and Jablonka, E. (1996). The inheritance of phenotypes: an adaptation to fluctuating environments. *Journal of theoretical biology*, 181(1):1–9.
- Levins, R. (1968). *Evolution in changing environments: some theoretical explorations*. Number 2. Princeton University Press.

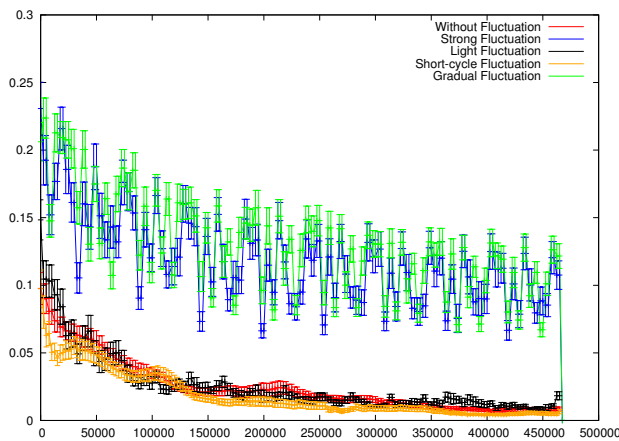


Figure 6: **Changes with similar environment** : Here one can see that the impact of environmental fluctuations decreases for *Short-cycle Fluctuation* and *Light Fluctuation* while it remains very high for other forms of environmental fluctuations.

Lipson, H., Pollack, J. B., and Suh, N. P. (2002). On the origin of modular variation. *Evolution*, 56(8):1549–1556.

Medernach, D., Fitzgerald, J., Carrignon, S., and Rya, C. (2015). Evolutionary progress in heterogeneous cellular automata (hetca). In *Proceedings of the European Conference on Artificial Life 2015 (ECAL 2015)*, volume 13, pages 512–519.

Medernach, D., Kowaliw, T., Ryan, C., and Doursat, R. (2013). Long-term evolutionary dynamics in heterogeneous cellular automata. In *Proceedings of the 15th annual conference on Genetic and evolutionary computation*, pages 231–238. ACM.

Okasha, S. (2006). *Evolution and the levels of selection*, volume 16. Clarendon Press Oxford.

Shanahan, T. (2012). Evolutionary progress: conceptual issues. *eLS*.

Smith, J. M. and Szathmary, E. (1997). *The major transitions in evolution*. Oxford University Press.

West-Eberhard, M. J. (2005). Developmental plasticity and the origin of species differences. *Proceedings of the National Academy of Sciences*, 102(suppl 1):6543–6549.

Yu, T. (2007). Program evolvability under environmental variations and neutrality. In *Proceedings of the 9th annual conference companion on Genetic and evolutionary computation*, pages 2973–2978. ACM.