EFFECT OF MUTATION AND RECOMBINATION ON THE GENOTYPE-PHENOTYPE MAP

C. R. Stephens
NNCP, Instituto de Ciencias Nucleares,
UNAM, Circuito Exterior, A.Postal 70-543
México D.F. 04510
e-mail: stephens@nuclecu.unam.mx

Abstract

The effect of genetic operators other than selection, such as mutation and recombination, on the genotype-phenotype map is considered. In particular, when the genotypic fitness landscape exhibits a "symmetry", i.e. many genotypes corresponding to the same phenotype have equal fitness values, it is shown that such operators can break this symmetry. The consequences of this "induced symmetry breaking" are investigated. Specifically, it is shown that it generically leads to an increase in order or self-organization in the system and to the phenomenon of orthogenesis. Additionally, it is shown that it potentially leads to a more robust evolution circumventing some of the problems of brittleness. The above points are supported by explicit, analytic results associated with some simple one and two-locus models and also by some much more complicated numerical simulations.

1 Introduction

Modulo the debate over the competing roles of selection and mutation the Darwinian concept of natural selection has stood alone for nearly a century and a half as the principle source of order in the natural world. More recently another paradigm has been presented [1] which draws for inspiration on the emergence of order in the physical rather than the biological world. Simply put: is order a consequence of the adaptive changes that take place in a system due to the effect of its environment, or does order appear "spontaneously", irrespective of any inherent selection? As in the selectionist/neutralist debate the correct answer is that order will appear both spontaneously and due to selection. However, for which systems one predominates over the other is a much more vexed question.

Traditionally, the tendency has been to view selection as an ordering agent and mutation and recombination as "disordering" effects. The Neutral theory [2], for instance, in its traditional guise makes no statement about any adaptive value of genetic drift, though others [3, 4, 5] have raised the issue of whether or not adaptive evolution can benefit from neutral evolution. Thus, genetic operators other than selection have generally been discounted as potential sources of order. Here, I am defining a genetic operator to be any operation H such that $P(t+1) = HP(t)$, where $P(t)$ is the population at time t.

In this short paper I will attempt to put other operators, such as mutation and recombination, onto a more democratic footing vis a vis selection. by presenting and discussing a third alternative for explaining the origin of order in biological systems that also has its origin in physics — "induced symmetry breaking". The "symmetry" here referred to is that inherent in the genotype-phenotype map when it is many-to-one, i.e. many genotypes correspond to the same phenotypic fitness value. It is of course not new to emphasize the importance of the genotype-phenotype map in Darwinian evolution, see for instance [6, 7], however it is new to show how this map may self-organize and provide a qualitative and quantitative framework within which this can be understood. In particular, we will see how and under what circumstances the phenomenon of orthogenesis may come about.

In section 2 I will introduce the concepts of order, symmetry and symmetry breaking. In section 3 I will give analytic examples of induced symmetry breaking in the context of some simple one and two-locus models. In section 4 I will briefly discuss some results found in some much more non-trivial models and in section 5 I will make some conclusions.

2 Order, Symmetry and Symmetry Breaking

I will not go into detail about a precise definition of "order". For the purposes of this paper its most salient characteristic is the following: that for a dynamical system with state space G of dimension D_G for late times the system occupies a subspace $U \subset G$ of dimension $D_U \ll D_G$. Thus, the more ordered a system is the smaller the subspace into which it dynamically evolves.

Intuitively, it is clear that selection will induce order in this sense. For example, in the presence of pure selection an entire population will eventually order itself around the optimum present in the initial population. The dynamical attractor in this case is typically of dimension zero. In the presence of mutation, such as in the Eigen model [8], the quasi-species represents the dynamical attractor. i.e. if one starts with a disordered random state then the effect of selection is to arrive at a more ordered state — the quasi-species. As is well known, however, for a large class of fitness landscapes there exists a critical mutation rate above which there is no dynamical reduction onto a smaller dimension attractor, i.e. selection has its limits.

However, we must first ask what does selection mean? Selection can be most precisely thought of in terms of fitness and the corresponding notion of a fitness landscape [9]. Fitness, $f_Q :\longrightarrow R^+$, is most naturally defined on the space of phenotypes, Q. In conjunction with the genotype-phenotype map, $\phi : G \longrightarrow Q$, where G is the space of genotypes, one may define an induced fitness function on the space of genotypes, $f_G = f_Q \circ \phi$. As the genotype-phenotype map is more often than not non-injective (many-to-one) the function f_G will be degenerate, many genotypes corresponding to the same fitness value. Thus, fitness defines an equivalence relation on G, many genotypes being equivalent selectively. A simple example of this would be the standard synonym "symmetry" of the genetic code. I will therefore refer to the equivalence of a set of genotypes under the action of selection (i.e. they're all equally fit) as a symmetry. Obviously, by definition, selection preserves this symmetry. One can see this explicitly, assuming proportional selection as a concrete example, from the evolution equation for the probability of finding a genotype C_i

$$P(C_i, t+1) = \frac{f(C_i)}{\bar{f}(t)} P(C_i, t) \qquad (1)$$

where $\bar{f}(t)$ is the average population fitness. Considering the same equation for a genotype C_j, where C_i and C_j both correspond to the same phenotype and therefore $f(C_i) = f(C_j)$, one sees that $P(C_i,t)/P(C_j,t) =$ constant, $\forall t$. We can in fact take this to be the defining characteristic of the symmetry: that for $C_g \subset G$ where $\phi(C_g) = C_q$, C_q being a given phenotype, $P(C_i,t)/P(C_j,t) =$ constant, $\forall t$, and $\forall\ C_i, C_j \in C_g$.

How may this symmetry be broken? In a finite gene pool the symmetry will be broken spontaneously by stochastic effects. This can be understood in several ways, e.g. via the theory of branching processes [10] or using Kimura's difusion approximation [2]. To lend a term from physics, such "spontaneous symmetry breaking" lies at the heart of Kauffman's ideas about the origin of order. Thus, even in the absence of selection a system can dynamically evolve to a smaller subspace, i.e. spontaneous symmetry breaking can lead to an increase in order.

I will now turn to another form of symmetry breaking by considering the effect of the other genetic operators besides selection defining

$$\begin{aligned} &P(C_i, t+1) = \\ &H(\{f(C_j)\}, \{p_k\}, \{P(C_j,t)\}, t) P(C_i, t) \end{aligned} \qquad (2)$$

where H is an operator that depends on the fitness landscape, $\{f(C_j)\}$, the probabilities, $\{p_k\}$, to implement the various genetic operators and on the population composition $\{P(C_j,t)\}$. I assume that one can write $H \equiv H_s + H_o$, where H_s is the part of the evolution operator associated with pure selection and H_o contains the effect of the other genetic operators. The landscape symmetry will thus be preserved by the action of the other genetic operators if $H_o P(C_i,t) = H_o P(C_j,t)\ \forall t$, and $\forall C_i, C_j \in C_g$. If this condition is not satisfied we will say that the symmetry has been broken by the action of the other genetic operators; instead of a spontaneous symmetry breaking there is an "induced" symmetry breaking.

As a quantitative measure of this symmetry breaking we will use the concept of "effective" fitness, defined via [11, 12]

$$P(C_i, t+1) = \frac{f_{\text{eff}}(C_i, t)}{\bar{f}(t)} P(C_i, t) \qquad (3)$$

One may think of the effective fitness as representing the effect of all genetic operators in a single "selection" factor. Hence, if only pure selection was allowed $f_{\text{eff}}(C_i,t)$ would represent the fitness value at time t required to increase or decrease $P(C_i,t)$ by the same amount as an evolution involving all the genetic operators and with selective fitness $f(C_i)$. If $f_{\text{eff}}(C_i,t) > f(C_i,t)$ then the effect of the genetic operators other than selection is to enhance the number present of genotype C_i relative to the number found in the absence of those operators. The converse is true when $f_{\text{eff}}(C_i,t) < f(C_i,t)$.

3 Analytic Examples of Induced Symmetry Breaking

We will now illustrate the phenomenon of induced symmetry breaking in some very simple examples of one and two-locus systems. Consider a single genetic locus with two alleles, 0 and 1 which have the same fitness value, f. In the absence of mutations $f_{\text{eff}}(C_i,t) = f(C_i,t) = f, \;\; \forall i = 0,1$ "Synonym" symmetry here is manifest in the fact that in the infinite population case $\Delta P(t) = P(1,t) - P(0,t)$ is constant in time. Thus, any initial deviations from homogeneity in the initial population will be preserved. For non-zero mutation rate, any initial inhomogeneity will be eliminated by the effect of mutations. i.e. if $\Delta P(t) > 0$ one will find that $f_{\text{eff}}(0,t) > f_{\text{eff}}(1,t)\ \forall t$ until the deviation is eliminated. Hence, one sees that the effect of mutations is to break the landscape symmetry between alleles 0 and 1. This mutation induced symmetry breaking brings the system into "equilibrium", i.e. into the homogeneous population state. During this approach to equilibrium the less numerous allele, 0, is "selected" more than the allele 1 in that it leaves more offspring. If the mutation rates for changing allele 1 to allele 0 and for changing allele 0 to allele 1 are not equal, but are p_1 and p_2 respectively, then the induced symmetry breaking is even more pronounced as can be seen by

$$\Delta P(t+1) = (1-2p_2)\Delta P(t) + (p_1 - p_2)P(0,t) \qquad (4)$$

In this case $\lim_{t\to\infty} \Delta P(t) \to ((p_1 - p_2)/(p_1 + p_2))$

Now consider a two-loci system, once again with two alleles, 0 and 1, evolving with respect to selection and mutation. The fitness landscape we will take to be: $f(00) = f(01) = 1$, $f(11) = 10$, $f(10) = 0.1$. The fitness landscape in this case is only partially degenerate: the states 00 and 01 having the same fitness value. However, although the fitness values are the same the effective fitness values are different: $f_{\text{eff}}(00,0) = (1 - 0.9p + 9.9p^2)$, $f_{\text{eff}}(01,0) = (1 + 9p - 9.9p^2)$, where p is the mutation rate and initial proportions of all four states are equal at $t = 0$. Here, the synonym symmetry is being broken due to the fact that the fit chromosome 11 can more easily mutate (for $p < 0.5$) to the chromosome 01. Therefore, there is a population flow away from 00 to 01 even though there is zero fitness gradient to cause it. Thus, we see a tendency for the system to evolve along a preferred direction not because of selection constraints but because the system has preferred directions of change in the face of *random* mutations. This is the phenomenon of orthogenesis and is simply a result of induced symmetry breaking and is quantitatively measured by the effective fitness function.

Naturally this phenomenon encourages one to ask just when neutral evolution is actually "neutral". In the above case it is not neutral to the presence of non-neutral adjacent mutants. The idea that neutral evolution can facilitate adaptive evolution is not new [3, 4, 5], however a clear, well defined framework within which this can be understood, induced symmetry breaking and the concept of effective fitness, is. In fact, it is clear that neutral evolution precisely leads to adaptive evolution when the effective fitness landscape is non-flat. For a flat fitness landscape where all strings have fitness f

$$f_{\text{eff}}(C_i,t) = f \sum_{j=1}^{2^N-1} \frac{P(C_j,t)}{P(C_i,t)} p^{d_{ij}} (1-p)^{N-d_{ij}} \qquad (5)$$

where d_{ij} is the Hamming distance between the strings C_i and C_j. For a homogeneous population the number of states Hamming distance d_{ij} from C_i is ${}^N C_{d_{ij}}$ thus $f_{\text{eff}}(C_j,t) = f\ \forall C_j, t$. Thus, under these circumstances the effective fitness landscape is as flat as the normal one and there is no symmetry breaking. Small deviations from homogeneity will be manifest in small corrugations of the effective fitness landscape which will gradually diminish as the population homogenizes. If the landscape only has a flat subspace then how well one can describe the population evolution as being neutral will depend on where the population is located and, if located predominantly in the flat subspace, what is the Hamming distance to states not within the subspace and what is the fitness of those states. Pictorially, if one thinks of a bowl with a flat bottom then the sides of the bowl with the largest gradient will attract the population most strongly.

Above I considered only mutation as a source of induced symmetry breaking. Similar considerations apply also to recombination. For the two-locus system mentioned above $f_{\text{eff}}(00,0) = (1 - (9.9p_c/12.1))$ and $f_{\text{eff}}(01,0) = (1 + (9.9p_c/12.1))$ where p_c is the recombination probability. Thus, once again we see the landscape symmetry broken by the effects of another genetic operator. A simple, but striking example of induced symmetry breaking can be seen with the landscape of the well known counting ones, or unitation problem. A population of 5000 8-bit strings was considered. Figure 1 is a plot of $M(l)$ versus time where $M(l) \equiv (n_{opt}(l) - n_{opt}(8))/n_{opt}(8)$. Here, $n_{opt}(l)$ is the number of optimal 2-schemata of defining length l normalized by the total number of length l 2-schemata per string, i.e. $9 - l$. By optimal 2-schemata we mean schemata containing the global optimum 11. $n_{opt}(8)$ is the number of optimal 2-schemata of defining length 8. Figure 1 is with $p_c = 1$. Averages over 30 different runs are shown. In terms of fitness there is absolutely no preference for one size of optimal two-schema versus another, however, recombination breaks this symmetry in a very dramatic fashion giving a preference for long rather than short schemata.

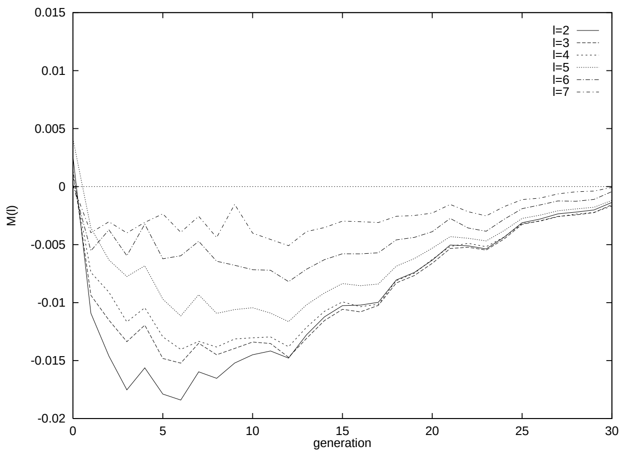

Figure 1: Graph of $M(l)$ versus t in unitation model with $p_c = 1$.

4 Numerical Examples

In the previous section I used some very simple tractable models to illustrate the phenomenon of induced symmetry breaking. In this section I will present some more non-trivial examples. For more details I refer the reader to the original articles.

i) Self-Adaptation: It is well known that mutation and recombination rates are not uniform throughout a structure such as a protein. One may well wonder why certain values are found rather than others and if or not there is any adaptive value in it. In fact, in the case of the HIV virus it can be shown that preference for non-synonymous mutations in the neutralization epitope of the virus is directly due to an induced symmetry breaking [13].

Normally one thinks of the mutation and recombination rates as exogeneous parameters. However, if one considers a system where they are coded in the chromosome, but are not directly selected for, then one has a completely autonomous system wherein one may examine whether the mutation and recombination rates across the population exhibit any degree of self-organization. More explicitly, coding the two rates into an N_c-bit extension of a chromosome of length N which represents a non-degenerate fitness landscape leads to a new one which has a degree of degeneracy of 2^{N_c}, i.e. the phenotype-genotype map is 2^{N_c} fold degenerate. In practice, starting off with a random population where the average rates are 0.5 one finds that the population in a class of interesting landscapes self-organizes until preferred mutation and recombination rates appear [14]. It is important to emphasize that such self-organization cannot come about as a consequence of selection, as by construction mutation and recombination rates are not selected for. However, the genetic operators of mutation and recombination themselves break the symmetry. The effective fitness measures the strength of this induced symmetry breaking is.

As a specific example, consider a time dependent landscape defined on 6-bit chromosomes that code the integers between 0 and 63, where the initial landscape has a global optimum situated at 10 and 11 and a local optimum at 40 and 41. However, after 60% of the population reaches the global optimum the landscape is suddenly changed to a new landscape wherein the original global optimum is now only a local optimum. The original local optimum at 40 and 41 remains the same but with a higher fitness value than the new local optimum at 10 and 11 and furthermore a new global optimum appears at 63. I will denote this landscape the "jumper" landscape. Figure 2 shows the results of an experiment where the mutation and crossover probabilities were coded in the chromosomes, either with three or eight bits to codify each probability. Tournament selection of size 5 was used and a lower bound of 0.005 for mutation imposed. The success of the self-adapting system in converging to the time dependent global optimum was compared to that of an "optimal" fixed parameter system with $p = 0.01$ and $p_c = 0.8$.

The upper curves show the relative frequencies of the optima using 8-bit and 3-bit codification and also what happens when $p_c = 0$ and only the mutation rate is coded. There are several notable features: first of all, the optimal fixed parameter system was incapable of finding the new optimum whereas the coded system had no such problem. For the case $p_c = 0$ the curve 40, 41 shows the relative frequency of the strings associated with the optimum at 40 and 41. Before the landscape "jump" this optimum is local being less fit than the global optimum at 10 and 11. After the "jump" it is fitter but less fit than the new global optimum 63 which is an isolated point.

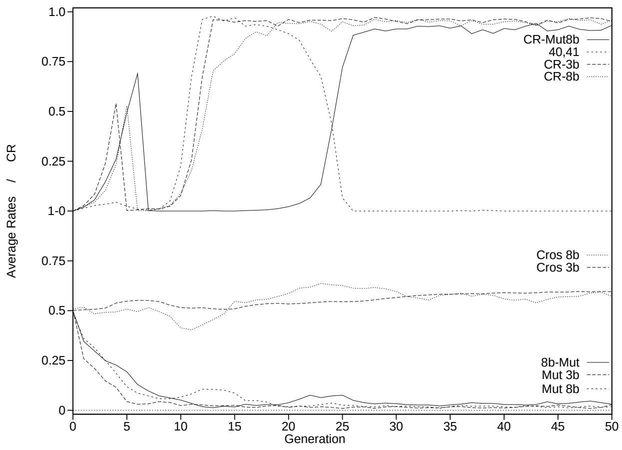

Figure 2: Graph of relative concentration of the global optimum (CR) (upper graph) and average crossover and mutation probabilities (lower graph) as a function of time for the "jumper" landscape. CR-3b and CR-8b are the results for 3-bit and 8-bit encoded algorithms. CR-Mut8b is the result for coded mutation with $p_c = 0$, with $40, 41$ being the relative concentration of strings associated with the local optimum at 40 and 41. Mut 3b, Mut 8b, Cross 3b and Cross 8b are the average mutation and crossover probabilities in 3-bit and 8-bit representations. The solid line for Mut8b is the average mutation rate in the case $p_c = 0$.

One thus sees that the global optimum was found in a two-step process after the landscape change. First the strings started finding the optima 40, 41 before moving onto the true global optimum, 63. Immediately after the jump the effective population of the new global optimum is essentially zero. The number of strings associated with 40 and 41 first starts to grow substantially at the expense of 10 and 11 strings. At its maximum the number of optimum strings is still very low, however, very soon thereafter the algorithm manages to find the optimum string which then increases very rapidly at the expense of the rest. The striking result here can be seen by comparing the changes in the relative frequencies with the changes in the average mutation rate, especially in the case $p_c = 0$. Clearly they are highly correlated. First, while the population is ordering itself around the original optimum, there is an effective selection against high mutation rates as one can see by the steady decay of the average mutation rate. After the jump there is a noticeable increase in the mutation probability as the system now has to try to find fitter strings. As the global optimum is an isolated state it is much easier to find fit strings associated with 41 and 40. The population is now concentrated on this local optimum and starts to cool down again only to find that this is not the global optimum, whereupon the system heats up again to aid the removal of the population to the true global optimum. It is clear that there is a small delay between the population changes and changes in the mutation rate. This is only to be expected given that there is no direct selective advantage in a given generation for a particular mutation rate. The selective advantage of a more mutable genotype over a less mutable one can only come about via a feedback mechanism. It is precisly this feedback process that is described and measured by the effective fitness function.

The average mutation rate also grows due to another effect which is that the new optimum is more likely to be reached by strings with high mutation rates which then grow strongly due to their selective advantage. Thus, high p strings will naturally dominate the early evolution of the global optimum. After finding the optimum however it will become disadvantageous to have a high mutation rate hence low mutation strings will begin to dominate. Induced symmetry breaking here is once again manifest in a most striking way. Although there is no direct selective benefit to differently coded strings their ability to produce offspring that can adapt to the changing landscape is very different.

ii) Neuro-genetic models: In this case an analysis was made [15] of the population dynamics of a variant of Kitano's neurogenetic model [16, 17] wherein the chromosome encodes the rules for cellular division and the phenotype is a 16-cell organism interpreted as a connectivity matrix for a feedforward neural network. Specifically, an artificial ecological environment was studied which consists of a single species composed of neural networks as individuals. Every chromosome, or genotype, is used to produce a particular architecture for a feedforward NN that consists of 12 input neurons, 4 hidden and 1 output neuron — the phenotype. A genetic algorithm is then applied to the chromosomes present in the population at each epoch which induces a search of the connectivity matrix space determined by the structure of the NN. Environmental effects are included in the fitness function that measures the learning capacity of a particular individual.

A chromosome consists of eight blocks of four genes each one of which is a three bit structure. The blocks themselves are labelled from a to h. The reproduction process always begins with block a. Thus the first four genes have a priviliged role as they label the cells that are going to be reproduced in the second step of reproduction. As an example consider the chromosome $baea.dcaa.defa.becd.aaea.aafh.haec.fgaa$. The two step reproduction process specified by this chromosome can be written

$$a \longrightarrow \begin{pmatrix} b & a \\ e & a \end{pmatrix} \longrightarrow \begin{pmatrix} d & c & b & a \\ a & a & e & a \\ a & a & b & a \\ e & a & e & a \end{pmatrix}$$

$$\leftrightarrow \begin{pmatrix} 0 & 1 & 1 & 0 & 1 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (6)$$

Thus the first block, *baea*, codes for the division of the original cell a into four cells. The first of these cells, b, then divides into four more which form the upper left quadrant, *dcaa*, of the matrix. The second cell, a, maps block a of the chromosome into the upper right quadrant etc. Finally, one constructs the connectivity matrix by reading left to right, row by row. Thus a 1 specifies a connection between an input neuron and a hidden neuron and a 0 its absence.

The genotype-phenotype map in this case is highly degnerate. For example, in the above we can change blocks c, e, f, g and h without changing the resulting phenotype. It is also a non-local function on the chromosomes since entries of block number one can target any one of the other blocks irrespective of their distance. To define a fitness function the learning speed of the NNs on a given test function was measured

$$y_c = \frac{\epsilon}{3}(x_1 + x_2 + x_3) + (1 - \epsilon)X$$

where ϵ is a noise control parameter and X is a randomly generated number. A genetic algorithm was used to search the space of network architectures for the one capable of learning this function with the smallest number of attempts. Given the highly degenerate nature of the genotype-phenotype map one might expect to see an optimum phenotype emerge corresponding to a random distribution of corresponding genotypes. However, this was not the case — certain genotypes were consistently preferred thus indicating that the genotype-phenotype symmetry was broken. The reason for this is that although degenerate genotypes were equivalent selectively the other genetic operators, mutation and recombination, broke the symmetry picking out those genotypes best able to withstand the effects of mutation and recombination, i.e. those that were most likely to lead to other "fit" neural networks. Remarkably, it was found that the induced symmetry breaking in this context could be described in terms of the emergence of an "algorithmic language" [15].

iii) Giraffe necks [18]: This model consists of a population of one thousand genotypes subject to random mutations. A genotype is a cellular automata with binary elements which gives rise to a giraffe neck size, i.e. a phenotype, given by the number of automata elements that are "switched on" at the fixed point (steady state) of the automata dynamics. As there are many different automata that can evolve to the same fixed point the genotype-phenotype mapping is highly degenerate. One "master" gene in particular plays a special role as it governs the way in which the Boolean rules used in the evolution mutate.

Each member of the population is selected for the next generation with probability $P_i = f_i/\sum_j f_j$, where f_i is the fitness of phenotype i. Initially, there are ample resources available from both small and large trees, the only selective criterion being that giraffes prefer to choose a mate among those that have similar neck size. This "social pressure" landscape is modelled by defining the fitness of the ith giraffe to be a function of its neck size n_i and the average neck size of the population, $\langle n \rangle$, with value one if $\langle n \rangle - \delta < n_i < \langle n \rangle + \delta$ and zero otherwise. Here, $\delta > 0$ is a tolerance window. Note that landscape fitness depends only on neck size, hence all genotypes that correspond to the same dynamical fixed point (phenotype) have the same fitness. Thus there is no direct selective advantage for one genotype versus another. To introduce time dependence into the landscape one imposes a short period of drought in which food begins to be available only in taller and taller trees. This period is mimicked by making $f_i = 1$ if $\langle n \rangle - \delta + \epsilon < n_i < \langle n \rangle + \delta + \epsilon$, where ϵ is a stress parameter, and zero otherwise. After this drought the landscape is restored to its original state.

The "master" gene divides the population into two genetic categories, type zero and type one, which can mutate one into the other due to the effect of purely random mutations that have a probability μ, except for the master gene which mutates at a rate ν. Type zero chromosomes, by nature of the dynamical evolution rules they are associated with, tend to give rise to giraffe offspring with shorter necks, whilst type one chromosomes, when they are expressed, tend to lead to giraffes with longer necks. Before the draught there is a period in which type one is not expressed. After a certain period of time it becomes expressed then afterwards the draught starts. The social pressure landscape implies there are two possible attractors: all type one or all type zero. The effect of the drought is to change between one and the other.

A typical experiment leads to the following results, the general behaviour can be seen in Figure 3: In the initial period of evolution, before the drought, average neck size is short. After the drought arrives the average

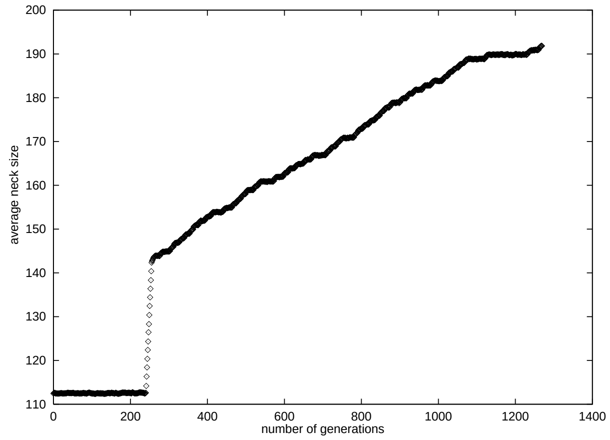

Figure 3: Graph of average giraffe neck size (in arbitrary units) as a function of time for a population of 1000 giraffes. The drought starts at generation 240 and lasts for 30 generations. Subsequent neck growth lasts for another 1000 generations. The parameter values used were: $\mu = 0.0025$, $\nu = 10^{-6}$, $\delta = 2.0$ and $\epsilon = 1.0$.

neck size grows very quickly. After it ends it continues to grow, albeit more slowly, for a substantial amount of time until a steady state is reached. These results can be explained quite simply: In the period before the draught, and before expression, type one chromosomes increase due to the effect of neutral drift. After expression they are effectively selected against due to their tendency to produce giraffes with longer necks that pass outside the tolerance threshold and therefore cannot reproduce. Thus, before the drought the effective fitness of type one chromosomes is low. However, due to the effect of mutations type one chromosomes are not eliminated totally but constitute about $1-5\%$ of the total population. After the draught starts the effective fitness of type one chromosomes increases substantially, given that they lead to giraffes of longer necks. The result is that the population becomes dominated by type one chromosomes, with a small fraction of type zero remaining due to the effects of mutation. After the end of the drought as type one chromosomes tend to produce longer necks the average neck size increases until a steady state is reached and it cannot grow anymore.

In the giraffe model there is absolutely no direct selective difference between type one and type zero chromosomes. The only advantage of one versus the other is in how they produce well adapted offspring, a quantitative measure of this being the effective fitness.

5 Conclusions

In this contribution I have tried to briefly lay out the case for induced symmetry breaking as an origin of order in biological systems. Without doubt it exists, as has been conclusively demonstrated. It is possible to see it at work in simple analytic one and two locus population models, and also numerically in several much more non-trivial examples of artificial life system as I have briefly touched upon here. The extent to which it exists in real biological systems is a question for future research. The chief difficulty in applying these ideas to the latter is that it is very difficult to assure oneself that apparent selection for a particular genotype is due to an effective selection, via a symmetry breaking effect, and not via some direct, yet unobserved, selective factor. For this reason I believe it is well worthwhile continuing with the examination of mathematical models of increasing complexity wherein one may better control the fitness landscape and the nature of the genotype-phenotype map, and also to consider artificial life systems where there is much more control over selective factors.

One might enquire as to why bother introducing the concepts of effective fitness and induced symmetry breaking. There are several reasons: first of all they allow one in a quantitative sense to understand the different mechanisms by which order may arise in biological systems. Secondly, they provide a framework within which neutral evolution and natural selection can be understood as different sides of the same coin, and in particular under what circumstances neutral mutations may lead to adaptive changes. Thirdly, induced symmetry breaking may well lead to more robust adaptive systems. It is no good having an extremely fit phenotype if when subjected to mutation at the genotypic level it typically mutates into an unfit phenotype. Rather one requires that an organism not only be fit but that it gives rise to fit offspring which in their turn give rise to fit offspring etc. Induced symmetry breaking can pick out precisely those evolutionary pathways that possess this type of robustness as is found in the neurogenetic model of section 4.

To what extent the different possible sources of order predominate will depend very much on the landscape considered and is as open to debate as the standard selectionist/neutralist argument. I believe that artificial life research can play an important role in this debate by examining the generic properties of landscapes and populations that admit as dominant one source of order versus another.

Acknowledgements

This work was partially supported through DGAPA grant number IN105197. The basic idea of induced symmetry breaking was developed in collaboration with Henri Waelbroeck to whom the author is grateful for many stimulating conversations.

References

[1] S. A. Kauffman, *The Origins of Order*, Oxford University Press, Oxford (1993).

[2] M. Kimura, *The Neutral Theory of Molecular Evolution*, Cambridge University Press, Cambridge (1983).

[3] S. Wright, Letter to M. Kimura, see W.B. Provine, *Sewall Wright and evolutionary biology*, University of Chicago Press, Chicago (1986).

[4] J. Maynard-Smith, *Natural Selection and the concept of a protein space*, *Nature* **255**, pp 563-564 (1970).

[5] M.A. Huynen, *Exploring phenotype space through neutral evolution*, *J. Mol. Evol.* **43**, pp 165-169 (1996).

[6] L.L. Gatlin, *Information Theory and the Living System*, Columbia University, New York Press, N.Y. (1972).

[7] V.A. Ratner, *Molecular Genetics, Principles and Mechanism*, (in Russian). Nauka Novosibirsk (1983).

[8] M. Eigen *Self-Organization of Matter and the Evolution of Biological Macromolecules*, *Naturwissenschaften* **58**: 465 (1971).

[9] S. Wright, *The roles of mutation, inbreeding, crossbreeding and selection in evolution*, In: Jones DF (ed) *Proceedings of the sixth international congress on genetics*, vol 1, pp356-366, Brooklyn Botanic Garden, New York (1932).

[10] Z. Taib, *Branching Processes and Neutral Evolution*, Springer Verlag, Berlin (1992).

[11] C.R. Stephens and H. Waelbroeck, *Analysis of the Effective Degrees of Freedom in Genetic Algorithms*, *Physical Review* **E57**, pp 3251-3264 (1998).

[12] C.R. Stephens and H. Waelbroeck, *Effective Degrees of Freedom of Genetic Algorithms and the Block Hypothesis*, *Proceedings of the Sixth International Conference on Genetic Algorithms*, Morgan Kaufman, San Francisco (1997).

[13] C.R. Stephens and H. Waelbroeck, *Codon Bias and Mutability in HIV Sequences* . National University of Mexico Preprint ICN-UNAM-97-09, (adap-org/9707), to be published in the *Journal of Molecular Evolution*, (1997).

[14] C.R. Stephens, I. García Olmedo, J. Mora Vargas and H. Waelbroeck, *Self-Adaptation in Evolving Systems*, *Artificial Life* **4.2**, 183-201 (1998).

[15] O. Angeles, H. Waelbroeck and C.R. Stephens, *Emergence of Algorithmic Language in Genetic Systems*, *Biosystems* **47**, 129-147 (1998).

[16] H. Kitano *Designing Neural Networks Using Genetic Algorithms with a Graph Generation System*, *Complex Syst.* **4**: 461-476 (1990).

[17] H. Kitano *Neurogenetic Learning: an Integrated Method of Designing and Training Neural Networks Using Genetic Algorithms*, *Physica* **D75**: 225-238 (1994).

[18] J. Mora, H. Waelbroeck, C.R. Stephens and F. Zertuche *Symmetry Breaking and Adaptation: Evidence From a Simple Toy Model of a Viral Neutralization Epitope*, National University of Mexico Preprint ICN-UNAM-97-10 (adap-org/0797) to be published in *Biosystems* (1997).