

Aging in Hostile Environment Modeled by Cellular Automata with Genetic Dynamics

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Abstract. We model the evolution of a population on a 2D cellular automata (CA) lattice. Every individual holds a binary “genetic code”. The code length and the number of “1”s in the chain correspond to the maximal and actual life-time of individual, respectively. The “genetic code” code is divided into three life-episodes: “youth”, “maturity” and “old age”. Only “mature” individuals can procreate. We investigate the duration of the life-episodes and their role in protecting the population from extinction in hostile environments. We observe that in the stable environment, which does not influence the life-time of individuals, the “youth” and the “maturity” periods extend extremely long during evolution, while the “old age” remains short. The situation is different for hostile plaque-like conditions. Under these circumstances, the “youth” period vanishes, while the longer “old age” period stabilizes the population growth, increases its average age and thereby increases its chance of survival. We can conclude that the idle life-episodes set up the control mechanisms, which allow for self-adaptation of the population to varying environmental conditions.

Keywords: evolution, genetic code, cellular automata, aging, Penna model

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1 Introduction

There are many approaches for modeling evolution. The simplest ones are defined by the iterative function systems, such as population equation, predator-pray paradigm and replication rule-based schemes, e.g., L-systems [18]. More complex evolutionary systems use the cellular automata (CA) [4,7,22,27], the autonomous agents [28] and the “genetic algorithm” operators [8], such as in the famous Penna model [1,11,16-17,20-23]. All of these models can be combined together, creating sophisticated artificial life-systems. On the one hand, even the trivial population equation produces complex chaotic trajectories. Advanced models are mostly uncontrollable and their behavior can be very difficult to forecast. On the other hand, simplistic approaches are limited and unrealistic due to the lack of both the spatial correlations between population members and the individual features of agents.

The cellular automata (CA) paradigm is an optimal computational vehicle for modeling population growth. It can represent both the communication layer for the agents and their living space. The entire system can be treated as a system with bounded resources due to the congested environment. Assuming the lack of individual features, which diversify the population, we can model how the colony adapts to the hostile environment by developing variety of spatial “correlation patterns” (see e.g. [2,5,7,9,14,15]). The multiplicity of forms with sophisticated shapes depends both on the micro-structural features of individuals and on the environmental conditions [7,9,19]. Emergent appearance of well-defined multi-resolutional features is the consequence of a complex exchange of information between individuals and the whole population [2,5,14].

A second kind of correlations, which appear in the feature space [13], emerges for a population, whose individual features develop with the evolution of the entire system [3,6,13]. The feature space is defined by the components of a unique “genetic code” – the vector describing an individual. The “genetic code” diversifies the individuals, dictates their adaptive capabilities and their abilities for surviving. It can evolve due to the reproduction and mutation operators. They are similar to those used in the “genetic algorithms” [8]. Reproduction accounts for dissemination of information encapsulated in the code, while mutation allows for a local search for a better solution.

One of the most interesting puzzles of evolution, which can be investigated with this model, is the role of “idle” episodes of individual’s life, such as “youth” and “old age”. It is widely known that the aging process is mainly determined by the genetic and environmental factors. The influence of many factors such as the sexual behavior and social effects on population survival was investigated, e.g., in [21,24], respectively. The organisms evolve to the state in which the life-time and reproductive ability in older age are sacrificed for the sake of early reproduction [1,16]. However, this optimal compromise can be affected, e.g., due to the lethal mutations influencing evolution in older age. The basic computational models of aging, which agree well with observations, are based on the Penna paradigm and the theory of accumulation [1,17,20]. This theory states that destructive mutations, whose consequences depend on the age of individual, can be inherited by the next generation. The mutations accumulate in their genomes influencing mainly older individuals. A general mathematical formulation for the age-structured population model with genetic mutations was given in [11]. In [16] it is shown that if the computer model of biological aging [17,23] is modified such that the late reproduction is privileged, then the Gompertz law of exponential increase of mortality can be retrieved.

Despite the great success of the Penna model allowing for understanding many processes connected to aging it has the following limitations:

1. The geographical location of the colony members remains undefined. Thus, the system evolves in the spatially uncorrelated environment with unbounded resources.
2. Only two episodes of life are considered, i.e., “youth” and “maturity”. The durations of the two are the same for each individual. Thus the “old age” is not accounted for.

In [20] a square lattice into the Penna bit-string model for biological ageing was introduced. It was used for studying the evolution of the spatial distribution of the population considering different strategies of child-care. Investigation of more substantial correlations between the age distribution in population and the type of spatial environment are possible by using a novel model proposed in this paper.

The paper is constructed as follows. First, we introduce our algorithm and list principal assumptions and definitions. Results of the simulations are shown in the following section. The model allows for investigating other important aspects of evolution, such as the role played by a congesting environment and the influence of various life-episodes: the “youth”, the “maturity” and the “old age”, on the survival ability of the whole colony. We discuss the problem of self-adaptation of population members to three types of unstable environment. Finally, our findings are summarized.

2 Model of population evolution

2.1. CA environment and rules of reproduction

Let us first assume that an ensemble of $S(t)$ individuals is spread on a 2D $N \times N$ lattice of periodic CA. In classical CA the population members evolve in time t , measured in number of evolution cycles, according to a set of pre-defined rules. We then assume additionally that each member of population holds a “genetic code” of length L . The codes are represented by chains of binary “0” and “1” components. The code length and the number of “1”s in the chain correspond to the maximal and actual life-time of individual, respectively. Only “1”s from “genetic codes” of individuals are read one by one along with the evolution time of the entire system while “0”s are skipped. Afterwards the last “1” has been read, the individual is deleted from the lattice. The code chain is divided onto three sub-chains corresponding to three episodes of life: “youth” \mathbf{y} , “maturity” \mathbf{m} and “old age” \mathbf{o} . They do not represent biological age of individuals, but rather concern their reproduction ability. Only “mature” individuals can procreate.

Let $\mathbf{A} = \{a_{ij}\}_{N \times N}$ be an array of possible locations of individuals on the 2D $N \times N$ lattice of CA. The value of $a_{ij} \in \mathfrak{R}$, $\mathfrak{R} = \{0, 1\}$ where “0” indicates that the node is unoccupied and “1” that it is occupied. An individual is defined by corresponding “genetic code” $\alpha_{ij} \in \mathfrak{R}^L$ such that:

$$\begin{aligned}
 &\text{if } (a_{ij} = 1) \text{ then} \\
 &\quad \alpha_{ij} \rightarrow [\mathbf{y}_{ij}, \mathbf{m}_{ij}, \mathbf{o}_{ij}]; \\
 &\quad \mathbf{y}_{ij} \rightarrow [y_{ij}^1, y_{ij}^2, \dots, y_{ij}^l], \\
 &\quad \mathbf{m}_{ij} \rightarrow [m_{ij}^1, m_{ij}^2, \dots, m_{ij}^m], \\
 &\quad \mathbf{o}_{ij} \rightarrow [o_{ij}^1, o_{ij}^2, \dots, o_{ij}^n], \\
 &\quad \wedge y_{ij}^k, m_{ij}^k, o_{ij}^k \in \{0, 1\}, L = n + m + l \\
 &\text{else} \\
 &\quad \alpha_{ij} \rightarrow \mathbf{0}
 \end{aligned} \tag{1}$$

Let us define the evolution rule of CA on the 2D lattice (g is generation index) given by the sequence of instructions presented in Fig. 1. In our model the individuals are treated as independent

agents, which can reproduce according to the classical recombination (*crossing-over*) operator Ω taken from genetic algorithms [8]. The recombination is allowed only for “mature” agents from the Moore neighborhood [4] (the eight closest individuals). If two “mature” individuals exist, they undergo reproduction by *crossing-over* their genomes. One of two offspring produced is selected randomly and placed in an unoccupied site. The resources of the CA system are limited locally due to both congested environment, which restricts the local space for reproduction, and finite size of the computational domain. The binary vectors $\mathbf{y}_{ij}, \mathbf{m}_{ij}, \mathbf{o}_{ij}$ represent the following epochs of individual life: “youth”, “maturity” and “old age”, respectively. The values of l, m, n represent the maximum lengths of each of the episodes while their actual durations are equal to the number of “1”s in corresponding vectors $\mathbf{y}_{ij}, \mathbf{m}_{ij}, \mathbf{o}_{ij}$, i.e., $p(\mathbf{y}_{ij}), p(\mathbf{m}_{ij}), p(\mathbf{o}_{ij})$, respectively, where $p(\boldsymbol{\alpha}_{ij})$ is the function, which returns the number of “1”s in $\boldsymbol{\alpha}_{ij}$ chain. The “counter” operator $p_k(\boldsymbol{\alpha}_{ij})$ used in Fig.1 is defined as follows:

$$\forall (a_{ij} = 1 \wedge p(\boldsymbol{\alpha}_{ij}) \geq k); p_k(\boldsymbol{\alpha}_{ij}) = k \quad (2)$$

Additionally, we assume that every individual is able to move randomly on CA lattice, provided that a free space in its closest neighborhood is available. This random motion procedure follows the reproduction process given by the sequence of instructions presented in Fig.1.

while $g < \text{MAX}$ do begin	// Initialize the following generation g .
for $i = 1$ to N do begin	// Go through every lattice site.
for $j = 1$ to N do begin	
if $a_{ij}^g = 0$ then	// If lattice site (i,j) is unoccupied
$m = \text{find_two_mature_neighbors}(i,j, \boldsymbol{\alpha}_1, \boldsymbol{\alpha}_2)$	// find two different “mature” individuals
/	// in the Moor neighborhood of a_{ij} .
if $(m=1)$ then	//If they do not exist $m=0$, otherwise $m=1$
$a_{ij}^{g+1} \rightarrow 1,$	
$(\boldsymbol{\beta}_1, \boldsymbol{\beta}_2) \rightarrow \Omega(\boldsymbol{\alpha}_1, \boldsymbol{\alpha}_2)$	//Start recombination operation.
$\boldsymbol{\alpha}_{ij} \rightarrow (\boldsymbol{\beta}_1, \boldsymbol{\beta}_2 pr),$	// pr - a probability for selection of
	// one out of two offspring $(\boldsymbol{\beta}_1, \boldsymbol{\beta}_2)$
$k_{ij} \rightarrow 1$	
else	//If a site (i,j) is occupied
if $p_{k_{ij}}(\boldsymbol{\alpha}_{ij}) = p(\boldsymbol{\alpha}_{ij})$ then	//delete it if its life-time passed
$k_{ij} \rightarrow 0, a_{ij}^{g+1} \rightarrow 0$	
else	
$k_{ij} \rightarrow p_{k_{ij}}(\boldsymbol{\alpha}_{ij}); k_{ij} \rightarrow k_{ij} + 1$	// or make it older.
end	
end	
end.	

Fig.1. The pseudo-code describing basic rules for evolution.

2.2. The models of lethal factors

Durations of both the life-episodes and the total life-time are predefined for a single individual, while they are variable due to evolution for every new generation. A shortage of space puts an upper-limit on the population growth and governs the development of some adaptation mechanisms inscribed in the “genetic code”. One can expect that after some time – due to the genetic

drift – the population will become more or less uniform. What is the main difference between this stable population and a population, which evolves in a hostile environment in which the life-time of individuals is influenced by hostile factors? To answer this question, we consider three models for portraying the hostile environment.

The Verhulst factor [26] is often used for modeling environmental features, such as competition between individuals for environmental resources food and space. In this model we assume that the probability p_V that individual can die in every evolution cycle (time) is $p_V = k_V \cdot S(t) / (N \cdot N)$, where $k_V \in (0, 1)$ – is a scaling factor, $S(t)$ – is the number of individuals in time t . The value of the Verhulst factor is independent on the age of individual and its fitness ability.

The “plaque” of a given strength ϵ_0 and a period of outbreak \mathbf{T} is the second hostile factor we modeled. We assume that, the population can be attacked by “plaque seeds”. The “seeds”, which are generated with \mathbf{T} period are initially scattered randomly on the CA lattice. The strength of the plague ϵ_0 is defined by the ratio between the number of “seeds” and the total number of individuals. If a “seed” is located at the same place as the population member, both are removed from the lattice. Otherwise, the “seed” moves randomly on the CA lattice. The “seeds” cannot reproduce.

Besides the two environmental lethal factors, we have also investigated a third genetic factor. Here we have assumed that the genome of each of the offspring undergoes \mathbf{M} lethal mutations just after replication, i.e., we select \mathbf{M} randomly chosen positions with “1”s in the “genetic code” α_{ij} and replace them with “-1”. Likewise in Penna model, this mimics some defects in DNA transcription. Both “1”s and “-1”s from “genetic codes” of individuals are read one by one along with the evolution time (“0”s are skipped). The individual dies, if the number of “-1”s exceeds a predefined threshold \mathbf{Th} . In this way we can model the accumulation of lethal mutations over an individual lifespan.

These three models of lethal factors were investigated independently. The results of modeling are demonstrated in the following section.

3 Results

The parameters assumed for a typical run are displayed in Table 1. The periodic lattice of CA 200x200 and 100x100 were considered, as being optimal since they balance well adequate representation and computational requirements. These parameters are also sufficient to obtain stable populations and partly eliminate boundary effects. At first, we discuss the results from modeling of the population evolving in a stable environment inhibiting lethal factors. Then we analyze separately the Verhulst, plaque and genetic models of hostile environments.

3.1. Stable environment

In Fig.2 the multi-resolutional structure of CA system consisting of 2 million sites is depicted. It shows clearly that the evolution is starting from a few separate and spherical clusters surrounded by a thin shell of “young” individuals. The initial ensemble is generated randomly populating P_0 sites ($P_0 \in (0, 1)$, see Table 1) on the lattice. Each individual a_{ij}^0 starts up its “counter” from the first “1” in the genetic code ($k_{ij}=1$), i.e., the whole population is initially “young”. Therefore, at the beginning of the simulation the evolution scenario depends strongly on the density P_0 of initial population (see Fig.3). For too large or too small P_0 values, after some time of evolution, the number of newborns can be marginal in contrast to massive extinction of “old age” individuals.

Table 1. Typical parameters of the simulation.

<i>Lattice size ($N \times N$ cells)</i>	100×100, 200×200	<i>Plague period</i>	50
<i>Initial density (P_0)</i>	0.2 – 0.5	<i>Dose (ϵ_0)</i>	0.4
<i>“Youth” - length</i>	32	<i>Scaling factor k_V in Verhulst model</i>	1
<i>“Mature” - length</i>	32	<i>Number of lethal mutations M</i>	1
<i>“Old age” - length</i>	32	<i>Death threshold T</i>	2-100
<i>Probability of reproduction</i>	1		
<i>Probability of mutation</i>	1		

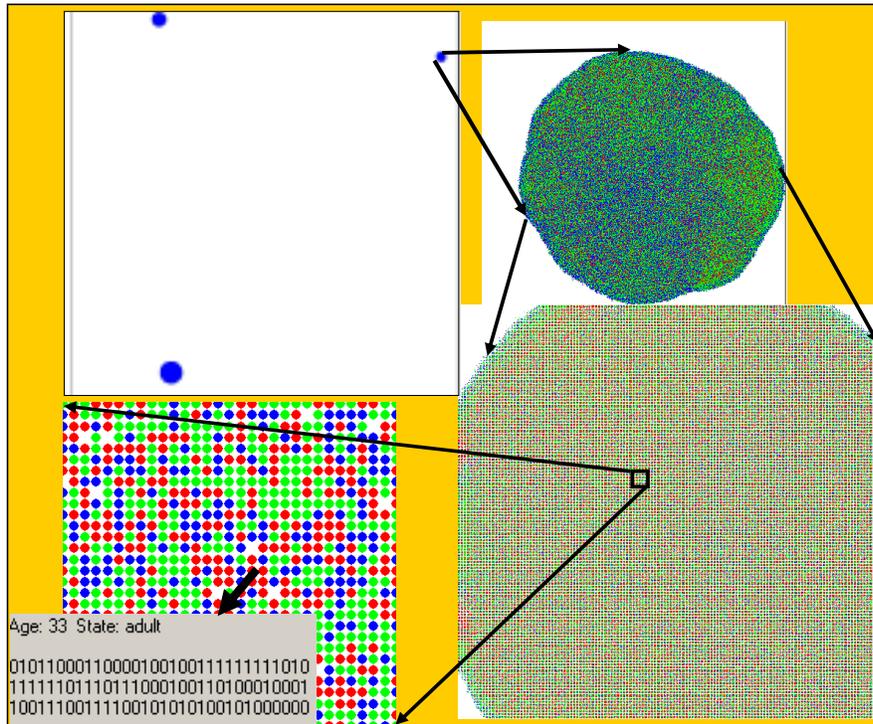


Fig.2. The population of individuals evolving on a periodic squared lattice of CA (1500x1500). Each individual has “genetic code” representing three 32 bits long episodes of an individual’s life: the “youth” – green dots, the “maturity” – blue dots and the “old age” – red dots, respectively.

This effect can be considerably reduced by increasing the mobility factor of the individuals. It also depends on the life-time and diversity of the initial population.

Let us assume that:

1. the number of “1”s in each three episodes of life has initially the Gaussian distribution,
2. $L=96$,
3. the lengths of \mathbf{y} , \mathbf{m} , \mathbf{o} vectors are identical, i.e., $l=m=n=32$ (see Eqs.(1)).

These values of L, l, m, n were selected intentionally to have a more compact representation of an individual, whose “genetic code” can be implemented then as three *float* variables. The value of L cannot be too small due to statistical validity. Of course, other configurations and vector lengths were also examined. However, the individuals, even these with the same life-time lengths, behave differently. This is due to the various lengths of the subsequent life-episodes \mathbf{y} , \mathbf{m} , \mathbf{o} .

The “genetic codes” averaged over the entire population define the global behavior of the colony. At one extreme, the population with too short “maturity” period will die quickly. On the other hand, the populations with greater reproduction potential (defined by l - the length of \mathbf{m}

vector) will tend to fill the \mathbf{m} part of vector α with “1”s. This is because the population members, which are “mature” for a longer time, have a greater chance to procreate and pass on their “genetic code” to the following generations.

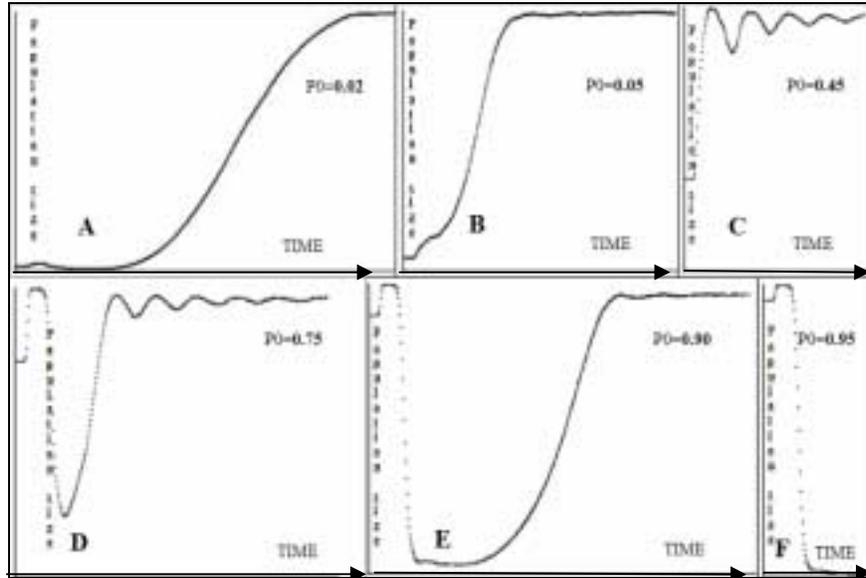


Fig.3. Various evolutionary scenarios of the growth of population size for increasing P_0 (initial population size). The simulation was started by assuming that all the individuals are “young”. A CA lattice with 100×100 grid nodes was simulated.

One can expect that the same behavior will be observed for infertile epochs of individual’s life i.e., the “youth” and the “old age”. That is, the individual’s life-time will increase due to the evolution to the maximum length L . However, the situation is completely different.

In Fig.4a we display the initial distribution of “1”s in each of \mathbf{y} , \mathbf{m} and \mathbf{o} vectors. As shown in Fig.4b, after $g=2000$ time-steps, the distributions of “1”s in each period of life undergo strong diversification.

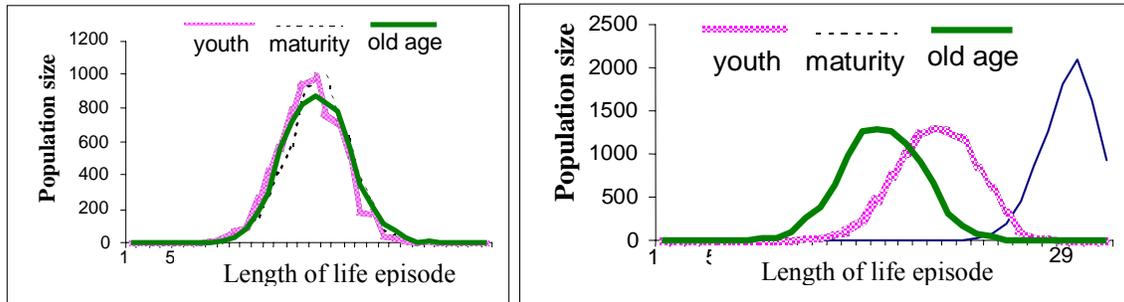


Fig.4. The plots representing the number of individuals with various lengths of \mathbf{y} , \mathbf{m} , \mathbf{o} life epochs. For initial generation of individuals the distributions are similar (the plot on the left), but after $g=2000$ steps they differ considerably (see the plot on the right).

a)

b)

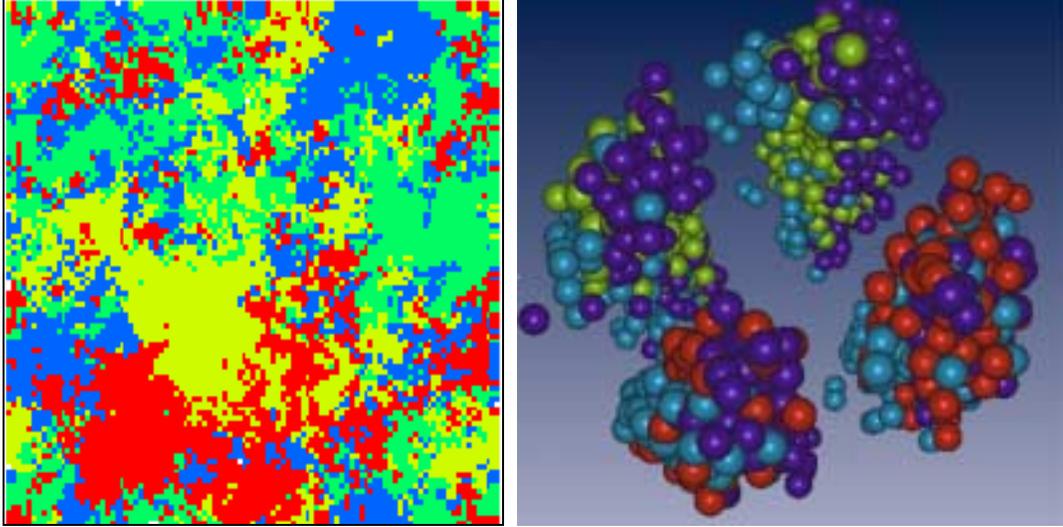


Fig.5. Appearance of clusters consisting of individuals on a 2-D lattice (a) and in the feature space (b) defined by coordinates of “genetic codes” transformed to 3-D space by using MDS procedure. Single plate in Figs.5b corresponds to a group of individuals with identical “genetic codes”. Fig.5b shows the result of *k-means* clustering in the L-dimensional feature space transformed by using MDS to a 3-D space. Various colors in b) indicate the spurious clusters obtained by using *k-means* clustering scheme. The colored 2-D clusters on the CA lattice in Fig.5a represent the four clusters recognized visually from Fig.5b.

As shown in Fig.5a, instead of initially chaotic configuration of individuals populating 2D lattice, after some time of evolution they produce distinct spatial clusters. Individuals belonging to the same spatial cluster are similar according to the Hamming distance [25]. This distance is defined in the abstract L-dimensional multi-dimensional feature space \mathfrak{R}^L [6,25] represented by the coordinates of the binary chains α_{ij} . As displayed in Fig.5b, the system consisting of agents with “genetic codes” produces clusters not only in the Cartesian two-dimensional space but also in \mathfrak{R}^L . The clusters in \mathfrak{R}^L (Fig.5b) can be extracted with clustering algorithms [12,25] and then visualized in 3-dimensional space by employing a multidimensional scaling (MDS) method [6,25].

The MDS technique plays complementary role to the clustering. As shown in Fig.5b, because of the high dimensionality of the feature space, some standard clustering schemes - such as *k-means* scheme [1] - are not able to extract the most distinct clusters of individuals. However, they can be extracted visually from the 3-D pictures produced by MDS [6,25]. As shown in Fig.5b, there exist four distinct “families” of individuals. In Fig.5a we display these families projected from the feature space (Fig.5b) onto 2-D CA lattice.

The continuation of the evolution from Fig.5 produces a stable attractor, which consists of four “families” of individuals, which have exactly the same “genetic codes”. The codes differ between clusters only on two one-bit positions. Therefore, the offspring generated due to recombination belong to one of the existing clusters. We do not obtain any global solution with only one large cluster of individuals having the same “genetic code”. This means that the fitness factor for the populations of individuals with the three life periods is not a trivial increasing function of the length of life. This situation does not hold for populations, which are only “mature” and ready for reproduction during the entire life-time ($L=m$, $l,n=0$). In this case the attractor of the evolution process would consist of individuals with “genetic codes” filled exclusively by “1”s.

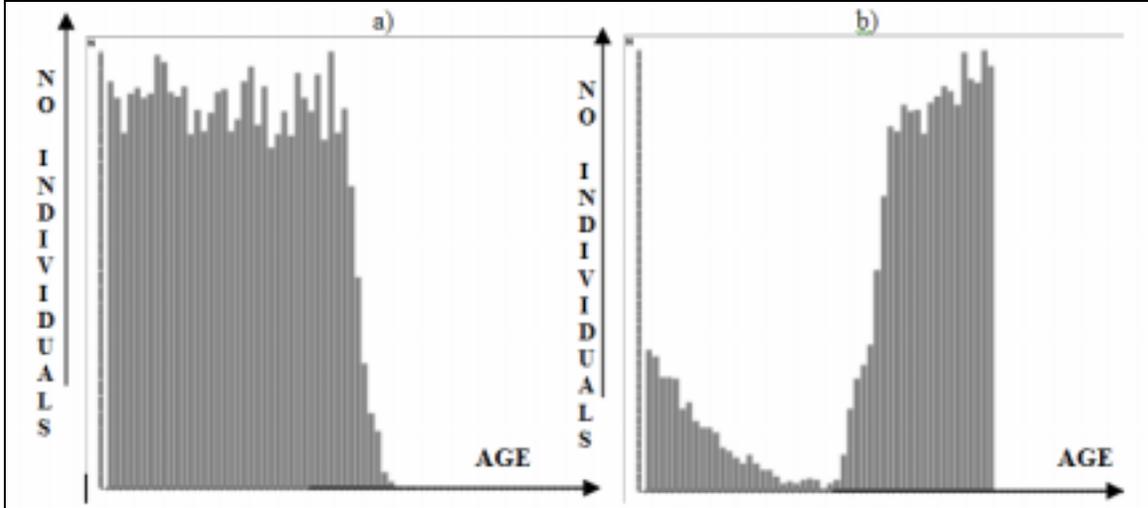


Fig.6. The histograms of number of individuals with their age for a) stable ($l=32, m=32, n=32$), and b) unstable ($l=0, m=40, n=64$) populations.

We summarize the most representative features of the attractors found in the modeling efforts in Table 2. The average durations of each life-episode $\mathbf{y}, \mathbf{m}, \mathbf{o}$ of various lengths (l, m, n), respectively, are computed after about 50.000 time-steps.

As shown in Table 2, for stable populations of individuals with limited life-time inscribed in their “genetic codes”, their “maturity” episodes fill with “1”s after a relatively short evolution time. This is obvious because a longer ability of reproduction gives a greater chance for passing the genetic code to the offspring. In extending the evolution time by about threefold, the “youth” fragments of the “genetic codes” will also be filled with “1”s. Surprisingly, even much longer simulation does not influence the “old age” lengths. The \mathbf{o}_{ij} vectors consist of a mixture of “1”s and “0”s. This observation is also valid for:

1. variable lengths of $\mathbf{y}, \mathbf{m}, \mathbf{o}$ ($l \neq m \neq n$),
2. long “old age” period ($n > m, n > l$),
3. and much shorter remaining episodes ($n \gg m, n \gg l$).

Table 2 The average number of “1”s in fragments of “genetic code” corresponding to the three life-episodes for various populations after 50,000 evolution cycles (where “var” – the length is variable, “perished” - the population dies quickly). The maximal lengths of each $\mathbf{y}, \mathbf{m}, \mathbf{o}$ episodes, l, m, n respectively, are given in brackets.

	YOUNG (y)	MATURE (m)	OLD (o)
Stable population ($l=32, m=32, n=32$)	32	32	var
($l=16, m=16, n=16$)	16	16	var
($l=8, m=8, n=8$)	8	8	var
Stable population ($l=16, m=24, n=64$)	3	24	var
($l=8, m=32, n=64$)	1	30	var
($l=0, m=40, n=64$)	0	perished	perished
Verhulst $k=1$ ($l=32, m=32, n=32$)	0	14 ± 3	18 ± 14
($l=0, m=32, n=32$)	0	12 ± 2	22 ± 10

($l=0, m=64, n=0$)	0	12 ± 2	0
Plague period < L			
($l=32, m=32, n=32$)	0	32	23 ± 7
($l=32, m=32, n=0$)	0	32	0
($l=0, m=32, n=32$)	0	32	20 ± 10
Plague period > L			
($l=32, m=32, n=32$)	0	32	0
Plague period < L			
($l=8, m=8, n=8$)	0	8	5 ± 2
Plague period > L			
($l=8, m=8, n=8$)	var	8	0

A further decrease of the “youth” episode ($l=8, m=32, n=64$) with respective extension of the “maturity” period, weakens considerably the population. For ($l=0, m=40, n=64$) it dies eventually. This behavior shows that the “youth” period accumulates the reproductive ability of the population. If this reproductive potential is released too fast, this will cause non-uniform aging (see Fig.6b), which may result in a fast extinction of the entire population.

3.2 Hostile environments

3.2.1 The Verhulst factor

In the first model the individuals die with the Verhulst probability $p_v = S(t)/(N \times N)$ in every evolution cycle. Then the chance of survival for each individual decreases roughly with time as $(1-p_v)^t$. Because, at the start of simulation all individuals are young, the population vanishes very quickly if individuals do not move. Otherwise, the number of individuals in population stabilizes. As shown in Fig.7, however, both the average age and the number of individuals in the population are considerably smaller than for the stable environment (compare Fig.5 with Fig.7 and also Fig.6a).

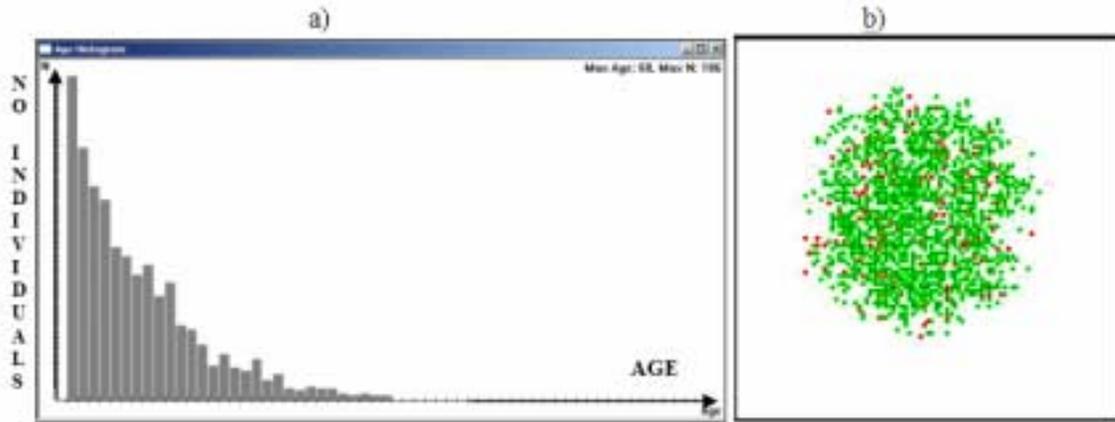


Fig.7. The population (on 200×200 lattice) with the Verhulst factor after 50.000 time-steps. The histogram (a) shows the number of individuals vs. their age for corresponding population on the CA lattice (b). The length of the “age” axis represents the maximum life-time allowed.

As shown in Table 2, for ($l=32, m=32, n=32$) the average life-time of the population is the longest. However, the length of the “youth” period shrinks to zero very fast (after 200 cycles). The “youth” period was eliminated during evolution as a main obstacle of the fast reproduction. As shown in Fig.7a, if the youngest individuals would be not able for procreation for a longer time, the number of the “mature” members of population could be too small for maintaining the

entire population. On the other hand, the “old age” period length becomes non-zero but variable. This effect is due to the fast decrease of a survival probability in time (see Fig.7a). Only few individuals are able to reach the “old age”, in spite of the high “genetic potential” they possess. The very high variability of the “old age” period is due to its marginal role on the population survival ability. Eliminating the “youth”, i.e., ($l=0, m=32, n=32$), and then the “old age” periods i.e., ($l=0, m=64, n=0$), do not change the length of the “maturity” episode. However, the average age in the first case, is slightly greater than in the second case.

3.2.2 Plaque

For a better understanding, we have assumed another sort of hostile environment – the plaque. As depicted in Fig.8, the population attacked by the periodic plague dies, if the strength (“Dose” in Table 1) of the plague ϵ_0 , defined to be the ratio of the number of “seeds” to the number of individuals, exceeds a certain threshold.

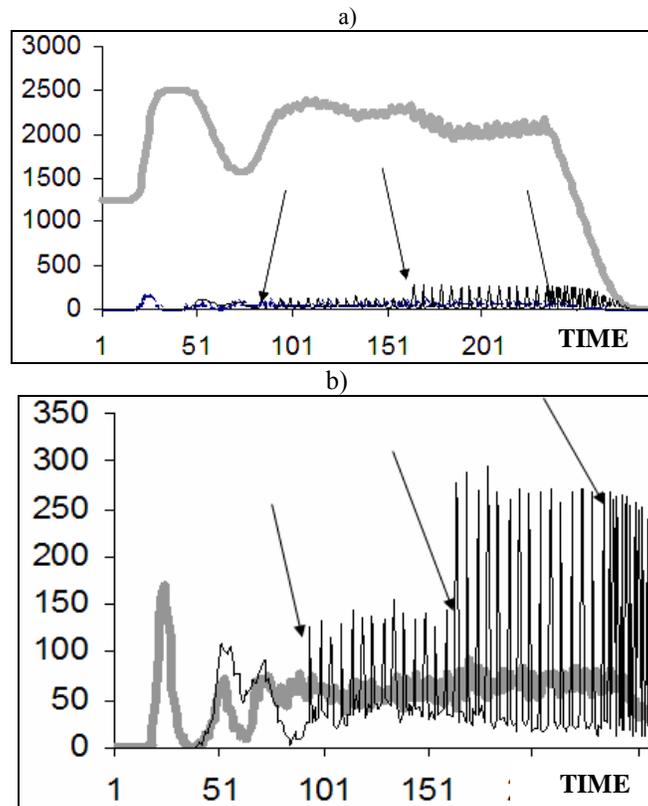


Fig.8. a) The influence of periodic plague (thin line) on the number of individuals (thick line) in time. b) The number of individuals (thick line) eliminated by the periodic plagues (thin line) in time. The arrows show the moments of new – gradually increasing – plague attacks.

The populations after long evolution in a stable environment and consisting of individuals with similar “genetic codes” and with long “youth” life-episodes die very quickly. This is due to the lack of adaptation ability, which involves diversification in the “genetic codes”. For example, a stable population with $l=m=n=32$ (e.g., Table 2), obtained after $g = 50,000$ time-steps of evolution and then assaulted by the plague, extinct during the following 100 steps. Similar population, but this time infected at the early stage of evolution (after $g = 200$ steps), survives. The “genetic codes” of individuals self-adapt to the unstable environment. As shown in Table 2, the

“genetic codes” of individuals from the attacked populations are different than those obtained for the stable environment. Moreover, they are different assuming various periods \mathbf{T} of the plague. Only the “maturity” period is set to the maximal value for the both cases.

For an outbreak with a short period \mathbf{T} , the “youth” episode is the obstacle for fast reproduction. During evolution it is eliminated completely ($\mathbf{y}=\mathbf{0}$). This observation is identical to the case of the population with the Verhulst factor. Surprisingly, the length of the “old age” period remains relatively long. Because the population can have not enough time for reproduction between subsequent plaques, it develops a sophisticated immunological system. We can explain this by assuming:

1. the “old age” is inhibited ($n=0$) and the population consists of only “mature” individuals,
2. the plaque is strong eliminating about half of population ($\epsilon_0=0.5$) from the lattice in a short time (about 10 timesteps).

At the moment when the plaque ceases, all of the survivors will produce many newborns due to the availability of free space on the lattice. Therefore, after some time, the individuals with a similar age and approximately the same life-time will be dominant in the population. Their simultaneous extinction will weaken the population (see Fig.6b). Thus, the number of “mature” individuals, which survive the following outbreak corresponding in time with extinction of “old” individuals, may be too small for starting new generations. The population can vanish eventually.

Otherwise, by assuming that the length of the “old age” episode is greater than 0 ($n>0$), we find that post-plaque demographic eruption and extinction periods can be much smaller and extended in time. The replacement of the “old age” individuals with newborns will be postponed and possible only after their deaths. Thus the population will be more stable (see Fig.6a). This can prevent the population from catastrophic correlation of the outbreak and demographic extinction. The population precipitating the “old age” episode is stronger and has a greater survival probability in unstable environment than that consisting of only “mature” individuals. We conclude that the “old” individuals collect the environmental resources (free space) for stable growth, thus eliminating dangerous post-plaque effects, such as demographic eruptions-extinction cycles.

When the plague period is greater than the average life-time of individuals and simultaneously the “strength” of the plague increases, the “old age” epoch is also eliminated due to evolution. The population has enough time for reproduction and demographic cycle does not coincide with the plague. The “old age” faction is eliminated, because by keeping the maximal length of the “mature” episode, the reproductive ability of the entire population is maximized.

3.2.3. *The genetic factor*

Apart from the Verhulst and the plaque models of the hostile environment, the lethal genetic factor from the Penna model was implemented. We have assumed that the number of lethal mutations $M=1$ and the threshold $Th=2$. The threshold Th is the number of lethal gene encounters during evolution of individual, which cause its death. Along with the evolution we observe several interesting processes. First, the “youth” period is eliminated completely in the same way as it was for the previous hostile factors. As shown in Fig.9, the lethal genes accumulate mainly at the end of the “maturity” period and in the “older age” fragments of the “genetic code”. This means that the chance of survival in younger age is the largest. Therefore, the histograms from Fig.10, showing the age distributions in the Penna populations, are not as flat as that obtained for a stable population (Fig.6a), and not as steep as for the Verhulst model (Fig.7a).

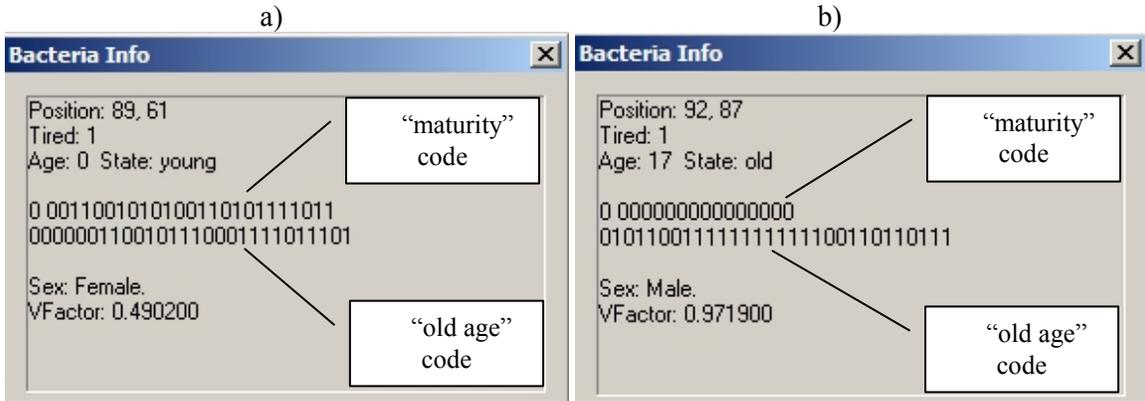


Fig.9. Typical bacteria information at the start of the simulation (a) and after 5000 evolution cycles (b). To make the results more visible, "1"s means the lethal gene occurrence, but the length of each episode of life is the number of "0" and "1"s in the corresponding chains. The "youth" period is disabled (only bacteria with age=0 are consider to be young).

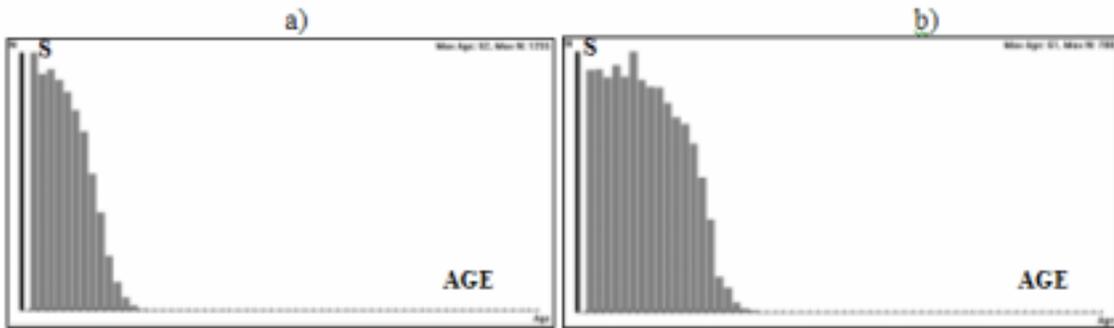


Fig.10. Age distribution for the Penna populations after 5000 evolution cycles for $T=2$: (a) ($l=0, m=64, n=0$) and (b) ($l=0, m=32, n=32$). The length of the "age" axis represents the maximum life-time allowed.

As displayed in Figs.10,11, the role of the "old age" period in increasing the average population age is evident. We investigate the evolution of two groups of populations: the first group with ($n>0$) and the second without "old age" life-episode ($n=0$). We assumed also that both of them have the "youth" period disabled and the individuals have the same maximum life-time. After 50,000 evolution cycles both the largest life-time and the largest average population age were obtained for the population with a non-zero "old age" length. For large Th our system behaves similarly to a population under a stable regime. Both the "youth" and the "maturity" periods extend to the maximum values allowed.

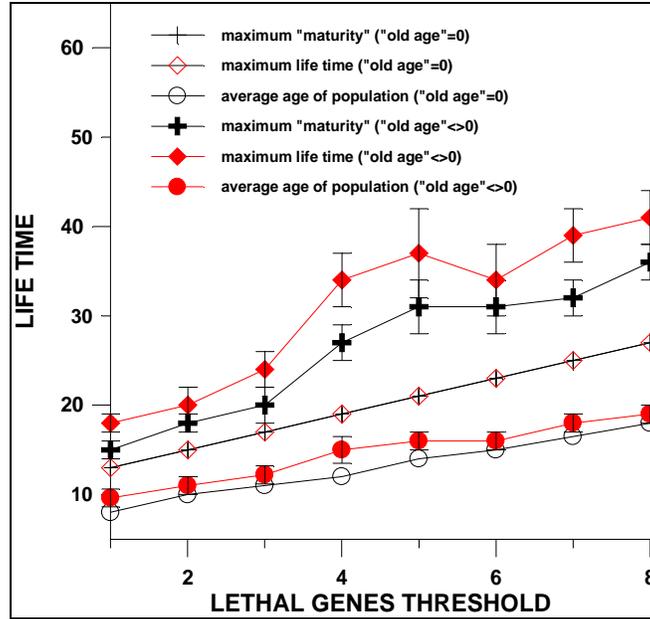


Fig.11. The length of the maximum life-time and average age of the population with the value of Th - the threshold value of the number of lethal genes sufficient for eliminating the individual.

4 Concluding remarks

In this work we have discussed the influence of the lengths of the “youth”, the “maturity” and the “old age” life episodes on population evolution in both stable and hostile environments. The duration of these periods of life depends critically on the biological, environmental and genetic features of the population. The biological organisms require some time to mature and be ready for reproduction. However, the environmental and genetic features decide about the actual length of procreation time and the total life-time. Therefore, the terms “youth”, “mature” and “old age” used in this paper have not only biological connotation. For example, an 11 years old girl from Africa can be “mature” for reproduction, while a 30 year old couple from a developed country - mature from biological point of view – but sacrificing reproduction for the sake of professional careers can be considered as “young”. The first example describes the individual who grows up in an unstable and hostile environment (i.e., the “youth” period is shortened extremely). The couple represents individuals taken from a stable population with an extended “youth” life-episode. Moreover, due to their wealth, they will be ready for procreation for a longer time than the young African girl.

We can conclude that:

1. The “maturity” period determines the reproductive power of the population and its survival ability in both stable and hostile environments. Therefore, the population increases its length to a maximum allowable value independent of the environmental conditions.
2. In the case of a stable growth the reproductive resources are accumulated in the “youth” and the “mature” episodes of life. The “old age” remains the secondary control mechanism.
3. The large value of the Verhulst factor eliminates the “youth” period sacrificing it to the sake of faster reproduction ability.
4. A population attacked by periodic plaque with a long period between outbreaks is biased only for reproduction.

5. For strong and frequent pests the “old age” period remains non-zero. The “old” individuals accumulate space, which is required for fast reproduction after the outbreaks.
6. The result of evolution in the presence of lethal mutations in the “genetic code” of individuals, depend on the value of threshold Th . Greater Th means the longer life-time. For a small value of Th , the population evolves in such a way that the lethal genes accumulate at the end of the “genetic codes” of individuals. The “old age” period stabilizes the population growth, increases its average age and thereby enhances greatly its chance of survival.

Many aspects of this model have not yet been explored. For example, an infected individual is removed from the lattice without any other consequences. The plaque therefore cannot spread out. This model can be extended by assuming that the plaque results in a long lasting infection and it causes destructive modifications in the “genetic codes” of individuals attacked. In the next model both the environmental and genome factors of evolution will be mutually dependent, i.e., the lethal mutations in genotype can be controlled by the environmental factors, such as plaque or the nutrients availability. Our model will be a valuable extension of the Penna paradigm of aging.

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References

1. de Almeida RMC, de Oliveira S Moss, Penna TJP (1997) Theoretical Approach to Biological Aging. *Physica A* **253**:366-378
2. Ben-Jacob E, Cohen I. and Levine H. (2000) Cooperative self-organization of microorganisms, *Adv. Phys.* 49(4):395-554
3. Broda A, Dzwiniel W (1996) Spatial Genetic Algorithm and its Parallel Implementation, *Lecture Notes in Computer Science*, **1184**: 97-107.
4. Chopard B, Droz M (1998) *Cellular Automata Modeling of Physical Systems*, Cambridge Univ. Press, London
5. Cohen I. Golding I. Kozlovsky Y. and Ben-Jacob E. (1999) Continuous and discrete models of cooperation in complex bacterial colonies. *Fractals* **7**:235-247
6. Dzwiniel W, Blasiak J, (1999), Method of particles in visual clustering of multi-dimensional and large data sets, *Future Generation Computers Systems*, 15:365-379.
7. Gallas JAC, Grassberger P, Herrmann HJ and Ueberholz P (1992) Noisy collective behavior in deterministic cellular automata. *Physica A* **180**:19-41
8. Goldberg D.E., (1989), *Genetic Algorithms in Search, Optimization, and Machine Learning*, Addison-Wesley Pub Co, 432 p.
9. Hermanowicz SW (2001) A Simple 2D Biofilm Model Yields a Variety of Morphological Features. *Mathematical Biosciences* **169**(1):1-14
10. Huang ZF, Stauffer D. (2001) Stochastic Penna model for biological aging, *Theory in Biosciences*, **120**(1), 21-28.
11. Ito N. (1996) Analytic approach for age-structured populations with genetic mutations, *Physica A*, **232**(1-2), 134-144.
12. Jain D, Dubes RC (1998) *Algorithms for Clustering Data*, Prentice-Hall, Advanced Reference Series.

13. Jasińska-Suwada A, Dzwiniel W (2002) Pattern recognition methods in understanding evolutionary systems, AI-METH 2002 *Artificial Intelligence Methods* November 13-15, 2002, Gliwice, Poland
14. Krawczyk K., Dzwiniel W., Yuen, D.A., (2003) Non-linear development of bacterial colony modeled with cellular automata and agent object, *Int J. Modern Phys. C* **10**:1-20.
15. Lacasta AM, Cantalapiedra IR, Auguet CE, Penaranda A and Ramirez-Piscina L (1999) Modeling of spatiotemporal patterns in bacterial colonies. *Phys. Rev. E* **59**(6):7036-7041
16. Makowiec D., Stauffer D., Zielinski M., (2001) Gompertz law in simple computer model of aging of biological population. *Int. J. Modern Phys C.*, **12**(7), 1067-1073.
17. Penna T.J.P., Stauffer, D., (1995) Efficient Monte-Carlo Simulation of Biological Aging. *Int. J. Modern. Phys.C.*, **6**(2) 233-239.
18. Prusinkiewicz, P., and Hanan, J., (1989) *Lindenmayer Systems, Fractals, and Plants*, Springer-Verlag, New York.
19. Shapiro JA (1995) The significances of bacterial colony patterns. *BioEssays*, **17**(7):597--607
20. Sousa AO, de Oliveira SM. (1999) The Penna model for biological ageing on a lattice: spatial consequences of child-care, *European Physical J.*, **9**(2), 365-369.
21. Sousa AO. (2003) Sex and recombination in the Hotzel aging model, *Theory in Biosciences*: **122**(4), 303-311.
22. Stauffer D (1991) Computer simulations of cellular automata. *J.Phys. A* **24**:909
23. Stauffer D, De Oliveira PMC, De Oliveira SM, Penna TJP, Martins JSS., (2001), Computer simulations for biological aging and sexual reproduction, *Anais da Academia Brasileira de Ciencias*, **73**(1), 15-32.
24. Stauffer D, Radomski JP. (2001) Social effects in simple computer model of aging. *Experimental Gerontology* **37**(1), 175-180.
25. Theodoris S, Koutroumbas K, (1998) *Pattern Recognition*, Academic Press, San Diego, London, Boston
26. Weisstein Eric W., Verhulst Model, (1996), From *MathWorld*--A Wolfram Web Resource. <http://mathworld.wolfram.com/VerhulstModel.html>
27. Wolfram S (2002) *A New Kind of Science*, Wolfram Media Incorporated. p 1263
28. Wooldridge M (2002) *Introduction to Multi Agent Systems*, John Wiley, p 256.