

CELLULAR AUTOMATA

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1 Introduction

In this lecture I shall present a class of mathematical tools for modeling phenomena that can be described in terms of elementary interacting objects. The goal is to make the macroscopic behavior arise from individual dynamics. I shall denote these individuals with the term automaton, in order to emphasize the main ingredients of the schematization: parallelism and locality. In my opinion, a *good* microscopic model is based on a rule that can be executed in parallel by several automata, each of which has information only on what happens in its vicinity (that can extend arbitrarily). In the following I shall use the word automaton either to refer to a single machine or to the whole set of machines sharing the same evolution rule.

These are completely discrete models: the time increases by finite steps, the space is represented by a regular lattice, and also the possible states of the automaton (the space variables) can assume one out of a finite set of values. The reason for this choice is the conceptual simplicity of the description. A single real number requires an infinity of information to be completely specified, and since we do not have any automatic tool (i.e. computer) that efficiently manipulates real numbers, we have to resort to approximations. On the other hand, these discrete models can be exactly implemented on a computer, which is a rather simple object (being made by humans). Since the vast majority of present computers are serial ones (only one instruction can be executed at a time), the parallel nature of the model has to be simulated.

The goal of a simple microscopic description does not imply that one cannot use real numbers in the actual calculations, like for instance in mean field approximations.

The class of phenomena that can be described with automata models is very large. There are real particle-like objects, such as atoms or molecules (from a classical point of view), that can be used to model the behavior of a gas. But one can build up models in which the automata represents bacteria in a culture or cells in a human body, or patches of ground in a forest.

In reality there are two classes of automata, one in which the automata can wander in space, like molecules of a gas, and another one in which the automata are stick to the cell of a lattice. I shall call the first type *molecular automata*, and the second *cellular automata*.¹

Probably the first type is the most intuitive one, since it resembles actual robots that sniff around and move. Each class has its own advantages, in term of simplicity of the description. A molecular automaton has information about its identity and its position. It can be used to model an animal, its state representing for instance the sex and the age. It is quite easy to write down a rule to make it respond to external stimuli. However, one runs into troubles when tries to associate a finite spatial dimension to this automaton. Let us suppose that the automaton occupies a cell on the lattice that represents the space. The evolution rule has to decide what happens if more than one automaton try to occupy the same cell. This is very hard to do with a true parallel, local dynamics, and can involve a negotiation which slows down the simulation. Clearly, a possible solution is to adopt a serial point of view: choose one of the automata and let it move. This is the approach of the computations based on molecular dynamics, where one tries to study the behavior of ensembles of objects following Newton's equations, or in Monte Carlo calculations. This serial approach is justified when time is continuous, and the discretization is just a computational tool, since for a finite number of objects the probability of having two moves at the same instant is vanishing, but indeed it is not a very elegant solution. Thus, molecular automata are good for point-like, non-excluding particles.

The other solution is to identify the automata with a point in space: on each cell of the lattice there is a processor that can communicate with the neighboring ones. If we say that a state represents the empty cell and another the presence of a bacterium, we associate to it a well defined portion of space. From this point of view, the bacterium is nothing but a property of space. The usual name for this kind of models is *cellular automata*.

People from physics will realize that cellular automata correspond to a field-like point of view, while molecular automata correspond to a particle point of view. In the last example above, only one particle can sit at a certain location (cell) at a certain time. Thus, we described a Fermion field. One can allow

¹See also the contribution by N. Boccara, this volume.

also an arbitrary number of particles to share the same position. This is a Boson field, in which particles lose their individuality. Following our analogy with elementary particles, we could say that molecular automata correspond to classical distinguishable particles.

The Boson field represents also the link between molecular and cellular automata. Indeed, if we relax the need of identifying each particle, and if we allow them to share their state (i.e. their identity and their position), then the two kinds of automata coincide. This suggests also an efficient way to simulate a molecular automata. Let us assume that every particle follows a probabilistic rule, i.e. it can choose among several possibilities with corresponding probabilities. Consider the case in which there are several identical particles at a certain position. Instead of computing the fate of each particle, we can calculate the number of identical particles that will follow a certain choice. If the number of identical particles at a certain position is large, this approach will speed up very much the simulation.

In the following I shall concentrate on cellular automata. They have been introduced in the forties by John von Neumann [1], a mathematician that was also working on the very first computers. Indeed, cellular automata represent a paradigm for parallel computation, but von Neumann was rather studying the logical basis of life. The idea of the genetic code was just arising at that time, so we have to take into consideration the cultural period.

From a mathematical point of view, he realized that the reproduction process implies that the organism has to include a description of itself, that we now know to be the genetic code. On the other hand, the chemistry of real world is too complex, and also the mechanics of a robot is not simply formalized. The solution was to drastically simplify the world, reducing it to a two-dimensional lattice. The result of these studies was a cellular automaton with the power of a general-purpose computer (a Turing machine), and able to read the information to reproduce itself. This solution was quite complex (each cell could assume one out of 26 states), but now we have several simplified versions. One of these is notably based on the Game of Life, a cellular automaton described in Section 4.1. For a review of these topics, see Sigmund (1993) [2].

In spite of their mathematical interests, cellular automata has been quiescent for nearly 30 years, until the introduction of the John Conway's Game of Life in the columns of Scientific American [3], around 1970. The Game of Life is a two-dimensional cellular automaton in which the cells can assume only two states: 0 for dead and 1 for live. Looking at the evolution of this cellular automaton on the display of a fast computer is quite astonishing. There are fermenting zones of space, and Life propagates itself by animal-like structures. From a prosaic point of view, there are interesting mathematical questions to be answered, as suggested in Section 4.1.

Finally, cellular automata exited the world of mathematicians around 1984, when the journal *Physica* dedicated a whole issue [4] to this topic. A good review of the first application of cellular automata can also be found in Wolfram's

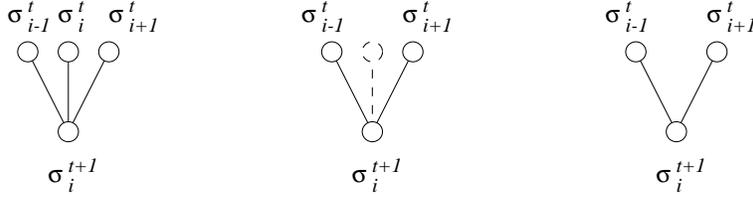


Figure 1: The $k = 1$ and $k = 1/2$ neighborhoods in $d = 1$.

collection of articles. [5]

In the following section I shall consider the mathematical framework of cellular automata, after which I shall review some applications of the concept.

2 Cellular Automata

Before going on we need some definitions. I shall denote the spatial index i and the temporal index t . Although we can have automata in a space of arbitrary dimension d , it is much simpler for the notations to consider a one-dimensional space. The state of a cell σ_i^t at position i and at time t can assume a finite number of states. Again for simplicity I consider only Boolean automata: $\sigma_i^t \in \{0, 1\}$. Assume N as the spatial dimension of the lattice. The state of the lattice can be read as a base-two number with N digits. Let me denote it with $\boldsymbol{\sigma}^t = (\sigma_1^t, \dots, \sigma_N^t)$. Clearly $\boldsymbol{\sigma} \in \{0, 2^N - 1\}$.²

The evolution rule $\boldsymbol{\sigma}^{t+1} = \mathbf{F}(\boldsymbol{\sigma}^t)$ can in general be written in terms of a local rule

$$\sigma_i^{t+1} = f(\sigma_{i-k}^t, \dots, \sigma_i^t, \dots, \sigma_{i+k}^t), \quad (1)$$

where k is the range of the interactions and the boundary conditions are usually periodic. The rule f is applied in parallel to all cells. The state of a cell at time $t + 1$ depends on the state of $2k + 1$ cells at time t which constitute its neighborhood. We consider here the simplest neighborhoods: the $k = 1$ neighborhood that in $d = 1$ consists of three cells and the $k = 1/2$ neighborhood, that in $d = 1$ consists of two cells and can be considered equivalent to the previous neighborhood without the central cell. A schematic diagram of these neighborhoods is reported in Fig. 1.

2.1 Deterministic Automata

The Game of Life and von Neumann's automaton are deterministic ones, i.e. once given the initial state the fate is in principle known, even though it can

²In this contribution I shall try to keep notation constant. I shall denote vectors, matrices and vectorial operators by bold symbols, including some Boolean functions of an argument that can take only a finite number of values (like a Boolean string), when referred as a whole.

Table 1: Example of Wolfram's code for rule 22.

σ_{i-1}^t	σ_i^t	σ_{i+1}^t	n	w_n	x_i^{t+1}
0	0	0	0	1	0
0	0	1	1	2	1
0	1	0	2	4	1
0	1	1	3	8	0
1	0	0	4	16	1
1	0	1	5	32	0
1	1	0	6	64	0
1	1	1	7	128	0
total					22

take a lot of effort.

A compact way of specifying the evolution rule for $k = 1$, $d = 1$ cellular automata has been introduced by S. Wolfram [5]. It consists in reading all possible configuration of the neighborhood $(\sigma_{i-1}^t, \sigma_i^t, \sigma_{i+1}^t)$ as a base-two number n , and summing up $w_n = 2^n$ multiplied by σ_i^{t+1} , as shown in Table 1 for the rule 22.

This notation corresponds to the specification of the look-up table for the Boolean function that constitutes the evolution rule. For an efficient implementation of a cellular automata one should exploit the fact that all the bits in a computer word are evaluated in parallel. This allows a certain degree of parallelism also on a serial computer. This approach is sometimes called *multi-spin coding* (see Section 3). In the following I shall use the symbols \oplus , \wedge and \vee for the common Boolean operations eXclusive OR (XOR), AND and OR. The negation of a Boolean variable will be indicated by a line over the variable. The AND operation will be denoted often as a multiplication (which has the same effect for Boolean variables).

Let me introduce some terms that will be used in the following. If a Boolean function f of n variables a_1, \dots, a_n is completely symmetric with respect to a permutation of the variables, than it depends only on the value of the sum $\sum_i a_i$ of these variables, and it is called *totalistic*. If the function is symmetric with respect to a permutation of the variables that correspond to the values of the cells in the neighborhood, but not to the previous value of the cell, than the function depends separately on the sum of the *outer* variables and on the previous value of the cells itself and the automaton is called *outer totalistic*.

Deterministic cellular automata are discrete dynamical systems. Given a certain state σ^t at a certain time, the state at time $t+1$ is perfectly determined. The ordered collection of states $(\sigma^0, \dots, \sigma^t, \dots)$ represents the trajectory of the system. Since for finite lattices the number of possible states is also finite, only

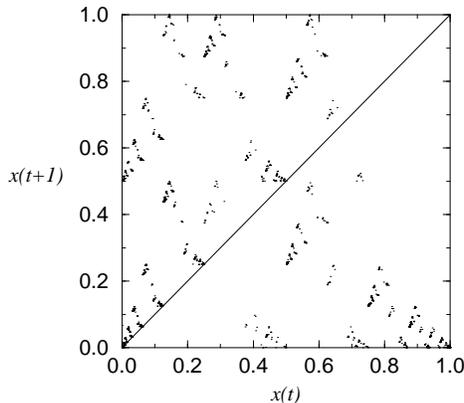


Figure 2: The return map of rule 22.

limit cycles are possible. There can be several possibilities: only one cycle, several small cycles, one big and several small, etc, where big and small refer to the length of the cycle. Moreover, one can consider the basin of attraction of a cycle, i.e. the number of states that will eventually end on it.

All these dynamical quantities will change with the size of the lattice. A physicist or a mathematician is generally interested to the asymptotic behavior of the model, but there may be a well defined size more interesting than the infinite-size limit. A table with the cycle properties of the simplest cellular automata can be found at the end of Wolfram's collection. [5]

The study of the limit cycles is clearly limited to very small lattices, especially in dimension greater than one. To extend the investigation to larger lattices, one has to resort to statistical tools, like for instance the entropy of the substrings (patches) of a certain size.

If a Boolean string \mathbf{a} , of length n appears with probability $p(\mathbf{a})$ (there are 2^n such strings), then the normalized n -entropy S_n is defined as

$$S_n = -\frac{1}{n \ln(2)} \sum_{\mathbf{a}} p(\mathbf{a}) \ln(p(\mathbf{a})). \quad (2)$$

S_n ranges from 1, if all strings appear with the same probability $p = 1/2^n$, to 0 if only one string appears.

One is generally interested in the scaling of S_n with n . For an example of the application of this method, see Grassberger's papers on rule 22. [6]

If one reads the configuration $\sigma^t = 01001001110101\dots$ as the decimal digit of the fractional part of a base-two number $x(t)$ (dyadic representation), i.e.

$$x(t) = 0.01001001110101\dots \quad (3)$$

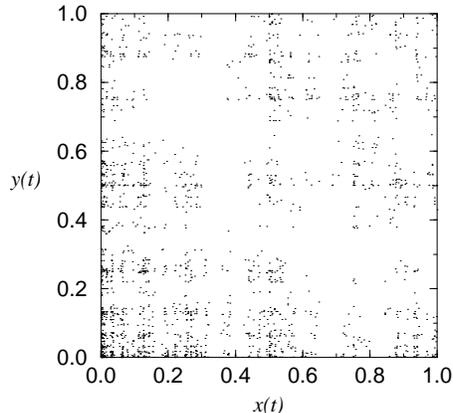


Figure 3: The attractor of rule 22.

for an infinite lattice one has a correspondence between the points in the unit interval and the configurations of the automaton. One has thus a complete correspondence between automata and maps $x(t+1) = f(x(t))$ of the unit interval. The map f is generally very structured (see Fig. 2). This correspondence helps to introduce the tools used in the study of dynamical systems.

Another possibility is the plot of the attractor and the measure of its fractal dimension. This can be performed dividing the configuration in two parts, and reading the left (right) part as a decimal number $x(t)$ ($y(t)$). The portrait of the attractor for rule 22 is given in Fig. 3.

One can try to extend to these dynamical systems the concept of chaotic trajectories. In the language of dynamical systems a good indicator of chaoticity is the positivity of the maximal Lyapunov exponent, which measures the dependence of the trajectory with respect to a small change in the initial position. This concept can be extended to cellular automata in two ways. The first solution is to exploit the correspondence between configurations and point in the unit interval. At the very end, this reduces to the study of the dynamical properties of the map f . This solution is not very elegant, since it loses the democratic point of view of the system (each cell has the same importance).

Another possibility is to look at the state of the system as a point in very high dimensional Boolean space. Here the smallest perturbation is a change in just one cell, and this damage can propagate at most linearly for locally interacting automata. However, one can measure the instantaneous spreading of the damage (i.e. the spreading in one time step), and from here it is possible to calculate a Lyapunov exponent [7]. The automata that exhibit *disordered* patterns have indeed a positive exponent for almost all starting points (trajectories), while *simple* automata can have Lyapunov exponent ranging from $-\infty$ to positive

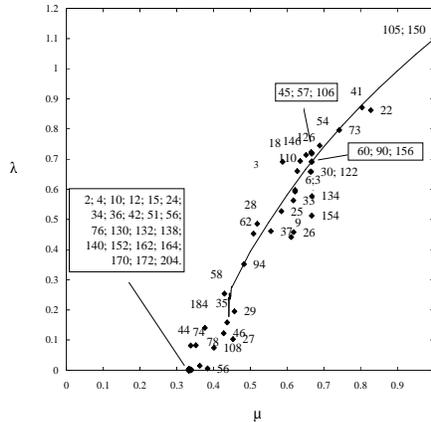


Figure 4: Lyapunov exponent for $k = 1$ cellular automata. The symbol μ denotes the instantaneous diverging rate of trajectories, and the solid line the results of a random matrix approximation. For further details, see Bagnoli *et al.* (1992). [7]

values. As for deterministic dynamical systems one interprets the trajectories with negative Lyapunov exponents as stable ones, and those with a positive exponent as unstable ones. In the simulation of an usual dynamical system, rounding effects on the state variables lead to the disappearance of the most unstable trajectories, so that the dependence of Lyapunov exponents on the trajectories looks quite smooth. We can recover this behavior with our discrete dynamical systems by adding a small amount of noise to the evolution. A plot of the maximal Lyapunov exponent for elementary cellular automata is reported in Fig. 4.

The only source of randomness for deterministic cellular automata is in the initial configuration. The counterpart of chaoticity is the dependence on the initial condition. For chaotic automata a small change in the initial configuration propagates to all the lattice. Measures of this kind are called *damage spreading* or *replica symmetry breaking*.

It is convenient to introduce the difference field (damage) h_i^t between two configurations x_i^t and y_i^t

$$h_i^t = x_i^t \oplus y_i^t \quad (4)$$

and the Hamming distance $H(t) = 1/n \sum_{i=1}^n h_i^t$. In Fig. 5 the spreading of the difference from a single site for rule 22 is reported (see also Section 3.3).

Finally, instead of studying the diverging rate of two trajectories, one can measure the strength required to make all trajectories coalesce. Once again, this is an indicator of the chaoticity of the automaton.

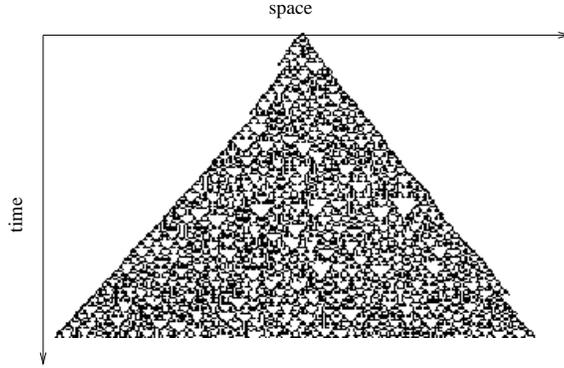


Figure 5: The temporal plot of the damage spreading for rule 22.

2.2 Probabilistic Automata

For probabilistic cellular automata the look-up table is replaced by a table of transition probabilities that express the probability of obtaining $\sigma_i^{t+1} = 1$ once given the neighborhood configuration. For $k = 1/2$ we have four transition probabilities

$$\begin{aligned}
 \tau(0, 0 \rightarrow 1) &= p_0; \\
 \tau(0, 1 \rightarrow 1) &= p_1; \\
 \tau(1, 0 \rightarrow 1) &= p_2; \\
 \tau(1, 1 \rightarrow 1) &= p_3,
 \end{aligned} \tag{5}$$

with $\tau(a, b \rightarrow 0) = 1 - \tau(a, b \rightarrow 1)$.

The evolution rule becomes a probabilistic Boolean function. This can be written as a deterministic Boolean function of some random bits, thus allowing the use of multi-site coding. In order to do that, one has to write the deterministic Boolean functions that characterize the configurations in the neighborhood with the same probability. For instance, let us suppose that the $k = 1/2$ automaton of equation (5) has to be simulated with $p_0 = 0$, $p_1 = p_2 = p$ and $p_3 = q$. The Boolean function that gives 1 only for the configurations $(\sigma_{i-1}^t, \sigma_{i+1}^t) = (0, 1)$ or $(1, 0)$ is

$$\chi_i^t(1) = \sigma_{i-1}^t \oplus \sigma_{i+1}^t \tag{6}$$

and the function that characterizes the configuration $(1, 1)$ is

$$\chi_i^t(2) = \sigma_{i-1}^t \wedge \sigma_{i+1}^t. \tag{7}$$

Let me introduce the truth function. The expression $\llbracket expression \rrbracket$ gives 1 if *expression* is true and zero otherwise (it is an extension of the delta function).

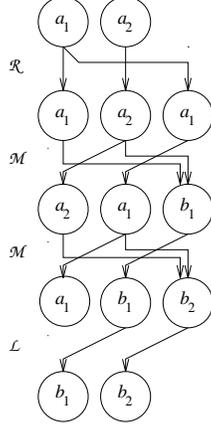


Figure 6: The application of the transfer matrix for the $k = 1/2$, $d = 1$, $N = 2$ cellular automata.

Given a neighborhood configuration $\{\sigma_{i-1}^t, \sigma_{i+1}^t\}$, σ_i^{t+1} can be 1 with probability p_i . This can be done by extracting a random number $0 \leq r < 1$ and computing

$$\sigma_i^{t+1} = \sum_i \llbracket p_i > r \rrbracket \chi_i. \quad (8)$$

Although the sum is not a bitwise operation, it can safely be used here since only one out of the χ_i can be one for a given configuration of the neighborhood. The sum can be replaced with a OR or a XOR operation.

Given a certain lattice configuration $\mathbf{a} = \boldsymbol{\sigma}^t$ at time t , the local transition probabilities allow us to compute the transition probability $\mathbf{T}_{\mathbf{b}\mathbf{a}}$ from configuration \mathbf{b} to configuration \mathbf{a} as

$$\mathbf{T}_{\mathbf{b}\mathbf{a}} = \prod_{i=1}^N \tau(a_{i-1}, a_{i+1} \rightarrow b_i). \quad (9)$$

The factorization of the matrix \mathbf{T} implies that it can be written as a product of simpler transfer matrices \mathbf{M} that add only one site to the configuration. Periodic boundary conditions require some attention: in Fig. 6 an example of decomposition $\mathbf{T} = \mathbf{L}\mathbf{M}^N\mathbf{R}$ for the simplest case $N = 2$ is shown. The lattice has been skewed for the ease of visualization.

This transition matrix defines a Markov process.

The state of the system at a certain time t is indicated by the probability $x_{\mathbf{a}}^{(t)}$ of observing the configuration \mathbf{a} . Clearly, one has $\sum_{\mathbf{a}} x_{\mathbf{a}}^{(t)} = 1$. For a deterministic automata only one component of \mathbf{x} is one, and all other are null. The time evolution of \mathbf{x} is given by the applications of the transfer matrix \mathbf{T} .

The conservation of probability implies that $\sum_{\mathbf{a}} T_{\mathbf{b}\mathbf{a}} = 1$. It is possible to prove that the maximum eigenvalue λ_1 of \mathbf{T} is one; the eigenvector corresponding to this eigenvalue is an asymptotic state of the system.

If all configurations are connected by a chain of transition probabilities (i.e. there is always the possibility of going from a state to another) than the asymptotic state is unique.

The second eigenvalue λ_2 gives the correlation length ξ :

$$\xi = -(\ln \lambda_2)^{-1}. \quad (10)$$

In our system two different correlation length can be defined: one in the space direction and one in the time direction. The temporal correlation length gives the characteristic time of convergence to the asymptotic state.

The usual equilibrium spin models, like the Ising or the Potts model, are equivalent to a subclass of probabilistic cellular automata. In this case the transition probability between two configurations \mathbf{a} and \mathbf{b} is constrained by the detailed balance principle

$$\frac{T_{\mathbf{b}\mathbf{a}}}{T_{\mathbf{a}\mathbf{b}}} = \exp(\beta\mathbf{H}(\mathbf{b}) - \beta\mathbf{H}(\mathbf{a})), \quad (11)$$

where $\mathbf{H}(\mathbf{a})$ is the energy of configuration \mathbf{a} and β is the inverse of the temperature. One can invert the relation between equilibrium spin models and probabilistic cellular automata [8] and reconstruct the Hamiltonian from the transition probabilities. In general, a cellular automata is equivalent to an equilibrium spin system if no transition probabilities are zero or one. Clearly in this case the space of configurations is connected.

These systems can undergo a phase transition, in which some observable displays a non-analytic behavior in correspondence of a well defined value of a parameter. The phase transition can also be indicated by the non-ergodicity of one phase. In other words in the phase where the ergodicity is broken the asymptotic state is no more unique. An example is the ferromagnetic transition; in the ferromagnetic phase there are two ensembles of states that are not connected in the thermodynamic limit. In the language of the transfer matrix this implies that there are two (or more) degenerate eigenvalues equal to one. The corresponding eigenvectors can be chosen such that one has positive components (i.e. probabilities) for the states corresponding to one phase, and null components for the states corresponding to the other phase, and vice versa. Indeed, the degeneration of eigenvalues correspond to the divergence of the (spatial and temporal) correlation lengths.

It is well known that equilibrium models cannot show phase transitions at a finite temperature (not zero nor infinite) in one dimension. However, this is no more true if some transition probabilities is zero or one violating the detailed balance Eq. (9). In effect, also the one dimensional Ising model exhibits a

$$\mathbf{M} = \begin{pmatrix} 1 & 1-p & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1-p & 1-q & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 1-p & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1-p & 1-q \\ 0 & p & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & p & q & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & p & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & p & q \end{pmatrix}$$

Figure 7: Transfer Matrix \mathbf{M} for $N = 2$.

phase transition at a vanishing temperature, i.e. when some transition probabilities become deterministic. In particular one can study the phase transitions of models with adsorbing states, that are configurations corresponding to attracting points in the language of dynamical systems. An automata with adsorbing states is like a mixture of deterministic and probabilistic rules. A system with adsorbing states cannot be equivalent to an equilibrium model.

Let me introduce here an explicit model in order to be more concrete: the Domany-Kinzel model [9]. This model is defined on the lattice $k = 1/2$ and the transition probabilities are

$$\begin{aligned} \tau(0, 0 \rightarrow 1) &= 0; \\ \tau(0, 1 \rightarrow 1) &= p; \\ \tau(1, 0 \rightarrow 1) &= p; \\ \tau(1, 1 \rightarrow 1) &= q. \end{aligned} \tag{12}$$

The configuration 0, in which all cells assume the value zero, is the adsorbing state. Looking at the single site transfer matrix \mathbf{M} (reported in Fig. 7 for the simple case $N = 2$), one can see that (for $p, q < 1$) every configuration has a finite probability of going into the configuration 0, while one can never exit this state. In this simple probability space a state (i.e. a vector) $\mathbf{v} = (v_0, \dots, v_7)$ that corresponds to the single configuration \mathbf{a} is given by $v_{\mathbf{a}} = \delta_{\mathbf{a}\mathbf{b}}$. The configuration 0 corresponds to the state given by the vector $\mathbf{w}^{(1)} = (1, 0, 0, \dots)$.

Let me indicate with the symbols $\lambda_i, i = 1, \dots, 8$ the eigenvalues of \mathbf{M} , with $\|\lambda_1\| > \|\lambda_2\| > \dots > \|\lambda_8\|$. The corresponding eigenvectors are $\mathbf{w}^{(1)}, \dots, \mathbf{w}^{(8)}$. Let us suppose that they form a base in this space. The Markovian character of \mathbf{M} implies that the maximum eigenvalue is 1, corresponding to the eigenvector $\mathbf{w}^{(1)}$.

A generic vector \mathbf{v} can be written as

$$\mathbf{v} = a_1 \mathbf{w}^{(1)} + a_2 \mathbf{w}^{(2)} + \dots \tag{13}$$

If we start from the vector \mathbf{v} at time $t = 0$ and we apply T times the transfer matrix \mathbf{T} , in the limit of large N this is practically equivalent to the application

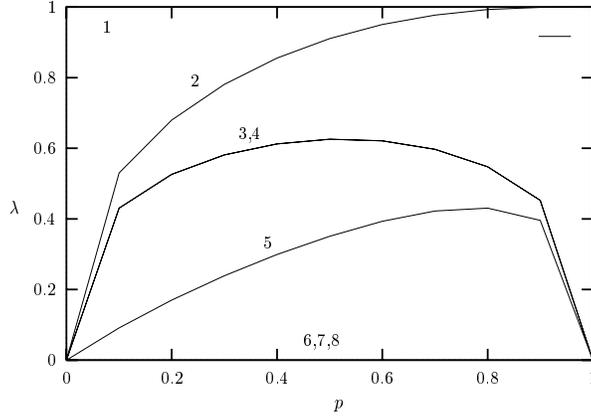


Figure 8: Modulus of the eigenvectors of the $N = 2$ transfer matrix for $p = q$.

of the matrix \mathbf{M} NT times, and we get

$$\mathbf{v}(T, N) = \mathbf{M}^{NT} \mathbf{v}(0, N) = a_1 \lambda_1^{NT} \mathbf{w}^{(1)} + a_2 \lambda_2^{NT} \mathbf{w}^{(2)} + \dots \quad (14)$$

and since all eigenvalues except the first one are in norm less than one, it follows that the asymptotic state is given by the vector $w^{(1)}$ (i.e. by the absorbing configuration 0).

This situation can change in the limit $N \rightarrow \infty$ and (after) $T \rightarrow \infty$ (the thermodynamic limit). In this limit some other eigenvalues can degenerate with λ_1 , and thus one or more configurations can survive forever. The existence of such phase transitions can be inferred from the plot of the modulus of the eigenvalues of \mathbf{M} for $N = 2$. This is reported in Fig. 8 for the case $p = q$. In this finite lattice the degeneration with the eigenvalue λ_1 (which corresponds to the configuration in which all sites take value 1) occurs for $p = 1$. As discussed in Section 4.2 in the thermodynamic limit the transition occurs at $p = 0.705\dots$. A rather different scenario exhibits for $q = 0$. In this case, as reported in Fig. 9, all eigenvalues degenerate with the first (for $p = 0.81\dots$ in the thermodynamic limit) and this implies a different dynamical behavior (see Section 4.2).

2.2.1 Critical Phenomena

The already cited phase transitions are a very interesting subject of study by itself. We have seen that the correlation length ξ diverges in the vicinity of a phase transition. The correlation between two sites of the lattice at distance r is supposed to behave as $\exp(-r/\xi)$, the divergence of ξ implies very large correlations. Let us suppose that there is a parameter p that can be varied. In

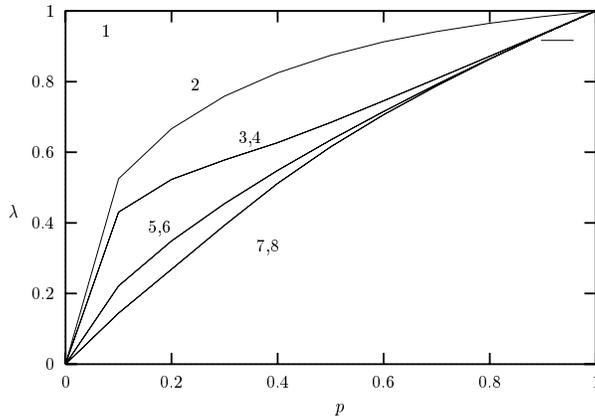


Figure 9: Modulus of the eigenvectors of the $N = 2$ transfer matrix for $q = 0$.

the vicinity of the critical value p_c of this parameter the correlation length diverges as $\xi \sim (p - p_c)^{-\nu}$, where ν is in general non integer. Also other quantities behaves algebraically near a critical point, like for instance the magnetization ρ which scales as $\rho \sim (p - p_c)^\beta$.

This power-law behavior implies that there is no characteristic scale of the phenomena. Indeed, if we change the scale in which the parameter p is measured, the proportionality factor will change but the form of the law will not. The pictorial way of explaining this phenomena is the following: suppose we have some two-dimensional system near to a phase transition where one phase is white and the other is black. There will be patches and clusters of all sizes. If we look at this specimen by means of a TV camera, the finest details will be averaged on. Now let us increase the distance from the TV camera and the specimen (of infinite extension). Larger and larger details will be averaged out. If the system has a characteristic scale ξ , the picture will change qualitatively when the area monitored will be of order of the square of this length or more. On the other hand, if we are unable to deduce the distance of the TV camera from the surface by looking at the image, the system is self-similar and this is a sign of a critical phenomena. Another sign is the slow response to a stimulation: again, the distribution of the response times follows a power-law.

The critical phenomena are very sensible to the parameter p , and a small change in it will destroy this self-similarity. In spite of their non-robustness, critical phenomena are heavily studied because their universality: since only large scale correlations are important, the details of the rule do not change the exponents of the power laws. This implies that the critical points of very different systems are very similar.

Self-similar objects are very common in nature. One example is given by

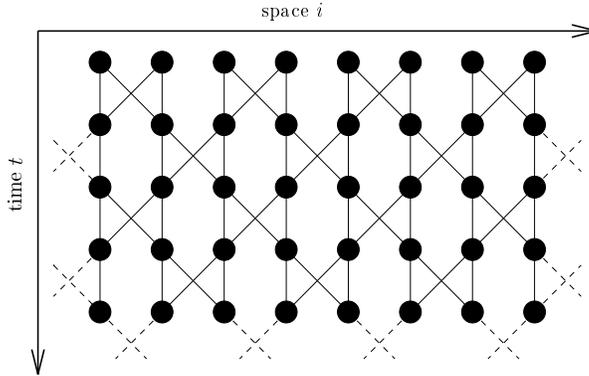


Figure 10: The lattice for the diffusion in $d = 1$.

clouds: it is almost impossible to evaluate the distance from a cloud, even when one is relatively near to it. And also power laws are found in many different fields, so are slow response times. It is impossible that we always meet usual critical phenomena, which are so delicate. It has been proposed [10] that open systems (i.e. out of equilibrium system) can auto-organize into a self-organized critical state, a state that exhibits the characteristic of a critical point still being stable against perturbations. It is indeed possible to develop simple models that exhibit this feature, but it is not clear how ubiquitous this quality is. The Game of Life of Section 4.1 is a very simple example of a Self Organized Critical (SOC) model.

2.2.2 Diffusion

The implementation of diffusion in the context of cellular automata is not very straightforward. One possibility is of course that of exchanging a certain number of pairs of cells randomly chosen in the lattice. While this approach clearly mixes the cells,³ it is a serial method that cannot be applied in parallel. A more sophisticated technique is that of dividing the lattice in pieces (a one dimensional lattice can be divided in couples of two cells, a two dimensional lattice in squares of four cells), and then rotate the cell in a patch according with a certain probability. This is equivalent to considering more complex lattices, but always with a local character, as shown in Fig. 10 for $d = 1$. The diffusion can be controlled by a parameter D that gives the probability of rotating the block. One has also to establish how many times the procedure has to be repeated

³For an application of this technique, refer to the contribution by Boccara, this volume.

(changing the blocks). A detailed analysis of the effects of this procedure can be found in Chopard and Droz (1989) [11].

3 Numerical techniques

In this section I shall review some techniques that I currently use to investigate cellular automata.

3.1 Direct simulations

The first approach is of course that of assigning each cell in the lattice to a computer word, generally of type `integer`, and to write the evolution rule using `if...` instructions. This generally produces very slow programs and waste a large amount of memory (i.e. the size of the lattices are limited).

Another possibility is to use look-up tables. In this case one adds the value of the cells in the neighborhood multiplying them by an appropriate factor (or packs them into the bits of a single word), and then uses this number as an index in an array built before the simulation according with the rule.

Alternatively, one can store the configurations in patches of 32 or 64 bits (the size of a computer word). This implies a certain degree of gymnastic to make the patches to fit together at boundaries.

Again, since often one has to repeat the simulation starting from different initial configurations, one can work in parallel on 32 or 64 replicas of the same lattice, with the natural geometry of the lattice. When using probabilistic cellular automata (i.e. random bits), one can use the same input for all the bits, thus making this method ideal for the damage spreading investigations (see Section 3.3).

Finally, one can exploit the natural parallelism of serial computers to perform in parallel simulations for the whole phase diagram, as described in Bagnoli *et al.* (1997). [12]

In order to use the last three methods, all manipulations of the bits have to be done using the bitwise operations `AND`, `OR`, `XOR`. It is possible to obtain a Boolean expression of a rule starting from the look-up table (canonical form), but this generally implies many operations. There are methods to reduce the length of the Boolean expressions. [13, 14]

3.2 Mean Field

The mean field approach is better described using probabilistic cellular automata. Given a lattice of size L , its state is defined by the probability $x_{\mathbf{a}}$ of observing the configuration \mathbf{a} . If the correlation length ξ is less than L , two cell separated by a distance greater that ξ are practically independent. The system acts like a collection of subsystems each of length ξ . Since ξ is not known a

priori, one assumes a certain correlation length l and thus a certain system size L , and computes the quantity of interest. By comparing the values of these quantities with increasing L generally a clear scaling law appears, allowing to extrapolate the results to the case $L \rightarrow \infty$.

The very first step is to assume $l = 1$. In this case the $l = 1$ cluster probabilities are $\pi_1(1)$ and $\pi_1(0) = 1 - \pi_1(1)$ for the normalization. The $l = 1$ clusters are obtained by $l = 1 + 2k$ clusters via the transition probabilities.

For the $k = 1/2$ Domany-Kinzel model we have

$$\begin{aligned}\pi'_1(1) &= (\pi_2(0,1) + \pi_2(1,0))p + \pi_2(1,1)q, \\ \pi'_1(0) &= \pi_2(0,0) + (\pi_2(0,1) + \pi_2(1,0))(1-p) + \pi_2(1,1)(1-q),\end{aligned}\quad (15)$$

where $\pi = \pi^{(t)}$ and $\pi' = \pi^{(t+1)}$.

In order to close this hierarchy of equation, one factorizes the $l = 2$ probabilities. If we call $\rho = \pi_1(1)$, we have $\pi_2(0,1) = \pi_2(1,0) = \rho(1 - \rho)$ and $\pi_2(1,1) = \rho^2$. The resulting map for the density ρ is

$$\rho' = 2p\rho + (q - 2p)\rho^2. \quad (16)$$

The fixed points of this map are $\rho = 0$ and $\rho = 2p/(2p - q)$. The stability of these points is studied by following the effect of a small perturbation. We have a change in stability (i.e. the phase transition) for $p = 1/2$ regardless of q .

The mean field approach can be considered as a bridge between cellular automata and dynamical systems since it generally reduces a spatially extended discrete system to a set of coupled maps.

There are two ways of extending the above approximation. The first is still to factorize the cluster probabilities at single site level but to consider more time steps, the second is to factorize the probabilities in larger clusters. The first approach applied for two time steps implies the factorization of $\pi_3(a,b,c) = \pi_1(a)\pi_1(b)\pi_1(c)$ and the map is obtained by applying for two time steps the transition probabilities to the $l = 3$ clusters. The map is still expressed as a polynomial of the density ρ . The advantage of this method is that we still work with a scalar (the density), but in the vicinity of a phase transition the convergence towards the thermodynamic limit is very slow.

The second approach, sometimes called *local structure approximation* [15], is a bit more complex. Let us start from the generic l cluster probabilities π_l . We generate the $l - 1$ cluster probabilities π_{l-1} from π_l by summing over one variable:

$$\pi_{l-1}(a_1, \dots, a_{l-1}) = \sum_{a_l} \pi_l(a_1, \dots, a_{l-1}, a_l). \quad (17)$$

The $l + 1$ cluster probabilities are generated by using the following formula

$$\pi_{l+1}(a_1, a_2, \dots, a_l, a_{l+1}) = \frac{\pi_l(a_1, \dots, a_l)\pi_l(a_2, \dots, a_{l+1})}{\pi_{l-1}(a_2, \dots, a_l)}. \quad (18)$$

Finally, one is back to the l cluster probabilities by applying the transition probabilities

$$\pi'(a_1, \dots, a_l) = \sum_{b_1, \dots, b_{l+1}} \prod_{i=1}^l \tau(b_i, b_{i+1} \rightarrow a_i). \quad (19)$$

This last approach has the disadvantage that the map lives in a high-dimensional (2^l) space, but the results converges much better in the whole phase diagram.

This mean field technique can be considered an application of the transfer matrix concept to the calculation of the the eigenvector corresponding to the maximum eigenvalue (fundamental or ground state).

3.3 Damage spreading and Hamming distance

In continuous dynamical systems a very powerful indicator of chaoticity is the Lyapunov exponent. The naive definition of the (maximum) Lyapunov exponent is the diverging rate of two initially close trajectories, in the limit of vanishing initial distance.

This definition cannot be simply extended to discrete systems, but we can define some quantities that have a relation with chaoticity in dynamical systems.

First of all we need a notion of distance in discrete space. The natural definition is to count the fraction of corresponding cells that have different value (I consider here only Boolean cellular automata). If we indicate with \mathbf{x} and \mathbf{y} the two configurations, the distance h between them (called the Hamming distance) is

$$h = \frac{1}{n} \sum_{i=1}^n x_i \oplus y_i. \quad (20)$$

It is possible to define an equivalent of a Lyapunov exponent [7] (see Fig. 4), but the natural application of the Hamming distance is related to the damage spreading.

For deterministic cellular automata the damage is represented by the Hamming distance between two configurations, generally starting from a small number of damaged sites, and the goal is to classify the automata according with the average speed of the damage or other dynamical quantities.

The extension to probabilistic cellular automata is the following: what will happen if we play again a rule with a different initial configuration but *the same realization of the noise*? If the asymptotic state changes, than the evolution remembers the initial configuration, otherwise it is completely determined by the noise. In the language of dynamical systems, this two scenarios correspond to a breaking of replica (the two configurations) symmetry. We can also define the difference field $h_i = x_i \oplus y_i$ with h representing its density. The breaking of

replica symmetry thus corresponds to a phase transition for h from the adsorbing state $h = 0$ to a *chaotic* state.

4 Investigation Themes

The theory described in the last section gives us the analytic tools and constitutes an interesting subject of study by itself. However, cellular automata can be studied as phenomenological models that mimic the real world from a mesoscopic point of view. The point of view of a physicist is again that of looking for the simplest model still able to reproduce the qualitative behavior of the original system. For this reason the models described in this section will be very crude; if one wants a model that mimics the system as good as possible, one can start from a simplified model and add all the features needed. An advantage of cellular automata with respect to system of differential or partial differential equations is the stability of dynamics. Adding some feature or interactions never leads to structural instabilities.

This section is of course not exhaustive.

4.1 Life

The Game of Life was introduced in the '70s by John Conway and then popularized by Martin Gardner in the columns of Scientific American [3]. It is a two dimensional, Boolean, outer totalistic, deterministic cellular automata that in some sense resembles the evolution of a bacterial population. The value 0 is associated to an empty or dead cell, while the value 1 to a live cell. The automaton is defined on a square lattice and the neighborhood is formed by the nearest and next-to-nearest neighbors. The evolution rule is symmetric in the values of the cells in the outer neighborhood, i.e. it depends on their sum. The transition rules are stated in a pictorial way as

- If a live cell is surrounded by less than two live cell, it will die by isolation.
- If a live cell is surrounded by more that three live cells, it will die by overcrowding.
- Otherwise, if a live cell is surrounded by two or three live cells, it will survive.
- An empty cell surrounded by three live cells will become alive.
- Otherwise an empty cell will stay empty.

The evolution of this rule is impressive if followed on the screen of a fast computer (or a dedicated machine). Starting from a random configuration with half cell alive, there is initially a rapid drop in the density ρ^t , followed by a long

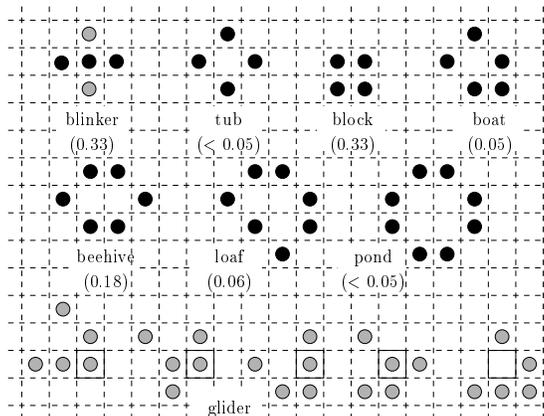


Figure 11: The most common animals in Life. The figures represent the average abundance of each animal.

phase of intense activity.⁴ After some hundreds time steps (for lattices of size order 200×200) there will emerge colonies of activity separated by nearly empty patches. In these patches there are small stable or oscillating configurations (the animals, see Fig. 4.1). Some of these configurations can propagate in the lattice (the gliders). The activity zones shrink very slowly, and sometimes a glider will inoculate the activity on a quiescent zone. Finally, after hundreds time steps, the configuration will settle in a short limit cycle, with density $\rho \simeq 0.028$. The scaling of the relaxation time with the size of the lattice suggests that in an infinite lattice the activity will last forever (see Fig. 14). The existence of a non-vanishing asymptotic density has been confirmed by recent simulations performed by Gibbs and Stauffer (1997) [17] on very large lattices (up to 4×10^{10} sites).

There are several questions that have been addressed about the Game of Life. Most of them are mathematical, like the equivalence of Life to a universal computer, the existence of *gardens of heaven*, the feasibility of a self-reproducing structure in Life, and so on (see Sigmund (1993) [2]).

From a statistical point of view, the main question concerns the dependence of the asymptotic state from the density of the initial configuration (supposed uncorrelated) and the response time of the quiescent state.

For the first question, in Fig. 12 is reported the plot of the asymptotic density versus the initial density for a lattice square lattice with $L = 256$. One can see a transition of the value asymptotic density with respect to the initial density for a value of the latter around 0.05. In my opinion this effect is due to the

⁴An exhaustive analysis of the Game of Life can be found in Bagnoli *et al* (1992). [16]

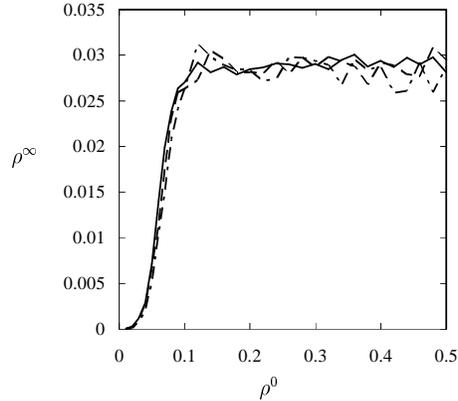


Figure 12: The plot of the asymptotic density ρ^∞ vs. the initial density ρ^0 in the Game of Life. The curves represents different lattice sizes: 96×50 (broken line), 160×100 (dashed line), 320×200 (full line).

finite size of the lattice, since there is a slight dependence on the system size. However, recent simulations performed by Stauffer [18] on very large lattices still exhibit this effect.

The response time can be measured by letting Life to relax to the asymptotic state, and then perturbing it in one cell or adding a glider. Recording the time that it takes before relaxing again to a periodic orbit and plotting its distribution, Bak *et al.* (1989) [19] found a power law, characteristic of self organized critical phenomena. One can investigate also the dynamical properties of the Game of Life, for instance the spreading of damage. Its distribution will probably follow a power law.

Another possibility is to investigate the asymptotic behavior using mean field techniques. The simplest approximations are unable to give even a rough approximation of the asymptotic density $\rho \simeq 0.028$, so it is worth to try more sophisticated approximations as the local structure one.

4.2 Epidemics, Forest Fires, Percolation

We can call this class of processes *contact processes*. They are generally representable as probabilistic cellular automata. Let me give a description in terms of an epidemic problem (non lethal)

In this models a cell of the lattice represents an individual (no empty cells), and it can stay in one of three states: healthy and susceptible (0), ill and infective (1), or immune (2).

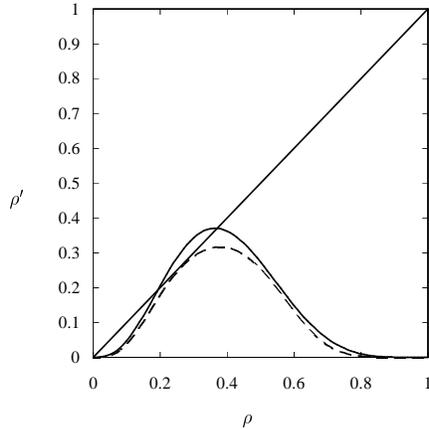


Figure 13: The mean field approximations for the Game of Life.

Let us discuss the case without immunization. This simple model can be studied also in one spatial dimension (a line of individual) + time.

Clearly the susceptible state is adsorbing. If there are no ill individual, the population always stays in the state 0. We have to define a range of interactions. Let us start with $k = 1$, i.e. the state of one individual will depend on its previous state and on that of its nearest neighbors. There is an obvious left-right symmetry, so that the automata is outer totalistic. The probability of having $\sigma_i^{t+1} = 1$ will depend on two variables: the previous state of the cell σ_i^t and the sum of the states of the neighboring cells. Let me use the totalistic characteristic functions $\chi_i^t(j)$ that take the value one if the sum of the variables in the neighborhood (here σ_{i-1}^t and σ_{i+1}^t) is j and zero otherwise (see Eq. (6, 7)). Moreover, I shall denote σ_i^t with σ' and I shall neglect to indicate the spatial and temporal indices. Then

$$\sigma' = f(\sigma, \chi(j)). \quad (21)$$

The function f gives the probability that σ' is ill (1) given its present state and the number of ill neighbors. If f does not depend on σ (i.e. the probability of contracting the infection is the same of staying ill for a given number of ill neighbors) we have again the Domany-Kinzel model Eq. (12). The parameters of this model are here the probability of contracting the infection or staying ill when an individual is surrounded by one (p) or two (q) sick individuals. The phase diagram of the model is reported in Fig. 15.

As one can see, there are two phases, one in which the epidemics last forever (*active* phase) and one in which the asymptotic state is only formed by healthy individuals (*quiescent* phase).

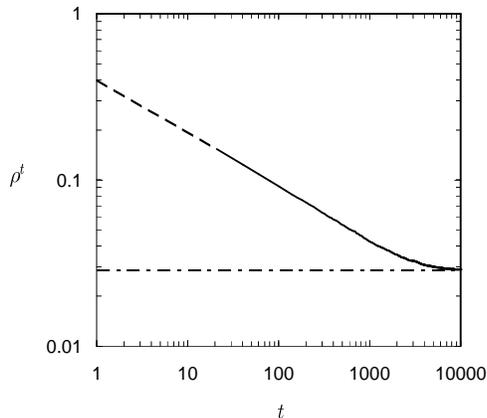


Figure 14: The temporal behavior of the density in the Game of Life.

A feature of this model is the presence of a region in the phase diagram with spreading of damage (*active phase*). This zone corresponds to $p \gg q$, i.e. it is due to an interference effect between the neighbors.⁵ This is obviously not realistic for a generic infection, but it can have some application to the spreading of a political idea.

One can also study the case in which if an individual is surrounded by a totality of ill individuals, it will certainly contract the infection ($q = 1$). This means that also the state in which all the cells have value one is adsorbing. In this case the transition is sharp (a first order transition).

The phenomenology enriches if we allow the previous value of the cell σ to enter the evolution function. Let us consider the a simplest case: a totalistic function of the three cells. Let us call p , q and w the probability of having $\sigma' = 1$ if $\chi(1)$, $\chi(2)$ or $\chi(3)$ is one, respectively. If we set $w = 1$ we have two adsorbing states and two parameters, so that the phase diagram is again two dimensional and easy to visualize (see Fig. 16). This model will be studied in detail in the contribution by Bagnoli, Boccara and Palmerini, this volume.

We see that in this case we can have both first and second order phase transitions. This observation could have some effective importance, in that a first order phase transition exhibit hysteresis, so that if the system enters one adsorbing state, it would cost a large change in the parameter to have it switch to the other state. On the other hand, a second order phase transition is a continuous one, with no hysteresis.

The model can be extended to larger neighborhoods, immune states, more dimensions and diffusion, always looking to the phase diagram and to damage

⁵See also Bagnoli (1996). [20]

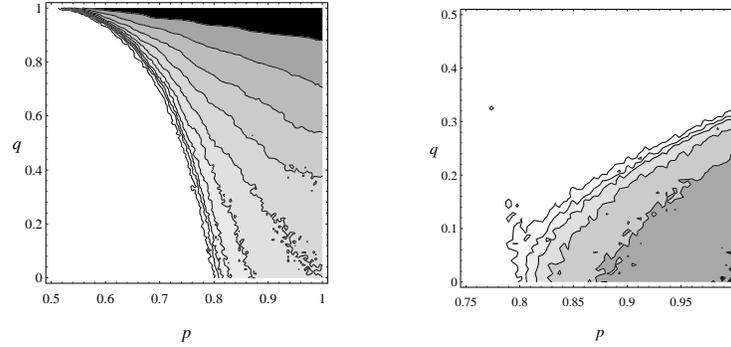


Figure 15: The phase diagram of the density (left) and damage (right) for the Domany-Kinzel model. ($0 \leq p \leq 1$, $0 \leq q \leq 1$, $N = 1000$, $T = 1000$). The gray intensity is proportional to the value of the density, ranging from white to black in equally spaced intervals.

spreading. The mathematical tools that can be used are direct simulations (with the fragment technique) and various mean field approximations (once more, the local structure technique is quite powerful).

4.3 Ecosystems

I shall use the following definition of an ecosystem: an evolving set of interacting individuals exposed to Darwinian selection. We start from the simplest model: let us consider an early ecosystem, composed by haploid individuals (like bacteria). Each individual can sit on a cell of a lattice in d space dimensions. Each individual is identified by its genetic information, represented as an integer number x . The genotype can be read as a four symbol (bases) or codon string. One could also consider the genome composed by alleles of a set of genes. I shall use here a binary coding (say, two alleles), because it is simpler to describe. All individuals have a genome of the same length l . Thus, we can interpret x as a base-two number representing the genotype or as an abstract index. The difference enters when we consider the mutations and thus we have to introduce the concept of distance in the genetic space.⁶ In the first case the genotype space is a hypercube with 2^l dimensions, in the second it is an abstract space of arbitrary dimension, that for simplicity we can consider one dimensional. Finally, we have to introduce the phenotypic distance, which is the difference in phenotypic traits between two individuals. Given a genotype x , the phenotype is represented as $g(x)$. In order to simplify the notations, I shall de-

⁶In effects, the distance in the genetic space is defined in terms of the number of mutations needed to connect (along the shortest path) two individuals (or, more loosely, two species).

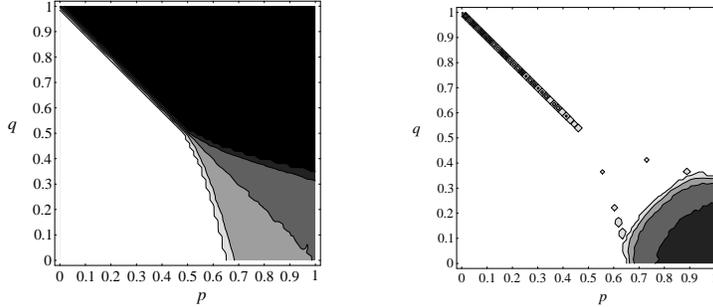


Figure 16: The phase diagram for the $k = 1$ totalistic cellular automaton with two adsorbing states.

note a function of the phenotype (say $h(g(x))$ as $h[x]$). The phenotypic distance will be denoted as $\delta(g(x), g(y)) = \delta[x, y]$. I do not consider the influence of age, i.e. a genotype univocally determines the phenotype. There could be more than one genotype that give origin to the same phenotype (polymorphism).

This automaton has a large number of states, one for each different genome plus a state (*) for representing the empty cell. Thus * represents an empty cell, 0 is the genome $(0, 0, \dots, 0)$, 1 is the genome $(0, \dots, 0, 1)$ and so on. The evolution of the automaton is given by the application of three rules: first of all the interactions among neighbors are considered, giving the probability of surviving, then we perform the diffusion step and finally the reproduction phase. I shall describe the one (spatial) dimensional system, but it can be easily generalized.

survival: An individual at site i in the state $x_i^t \neq *$ and surrounded by $2k + 1$ (including itself) neighbors in the states $\{x_{i-k}, \dots, x_{i+k}\} = \{x_i\}_k$ has a probability $\pi(x_i, \{x_i\}_k)$ of surviving per unit of time. This probability is determined by two factors: a fixed term $h[x_i]$ that represents its ability of surviving in isolation, and an interaction term $1/(2k+1) \sum_{j=i-k}^{i+k} J[x_i, x_j]$. Clearly, both the static fitness and the interaction term depend on the phenotype of the individual.

The fixed field \mathbf{h} and the interaction matrix \mathbf{J} define the chemistry of the world and are fixed (at least in the first version of the model). The idea is that the species x with $h[x] > 0$ represent autonomous individuals that can survive in isolation (say, after an inoculation into an empty substrate), while those with $h[x] < 0$ represents predators or parasites that necessitate the presence of some other individuals to survive. The distinction between autonomous and non-autonomous species depends on the level of schematization. One could consider an ecosystem with plants

and animals, so that plants are autonomous and animals are not. Or one could consider the plants as a substrate (not influenced by animals) and then herbivores are autonomous and carnivores are not. The interaction matrix specifies the necessary inputs for non autonomous species.

We can define the fitness H ⁷ of the species x_i in the environment $\{x_i\}_k$ as

$$H(x_i, \{x_i\}_k) = h[x_i] + \frac{1}{2k+1} \sum_{j=i-k}^{i+k} J[x_i, x_j]. \quad (22)$$

The survival probability $\pi(H)$ is given by some sigma-shaped function of the fitness, such as

$$\pi(H) = \frac{e^{\beta H}}{1 + e^{\beta H}} = \frac{1}{2} + \frac{1}{2} \tanh(\beta H), \quad (23)$$

where β is a parameter that can be useful to modulate the effectiveness of selection.

The survival phase is thus expressed as:

- If $x_i \neq *$ then

$$\begin{aligned} x'_i &= x_i && \text{with probability } \pi(H(x_i, \{x_i\}_j)) \\ x'_i &= * && \text{otherwise} \end{aligned} \quad (24)$$

- Else

$$x'_i = x_i = * \quad (25)$$

diffusion: The diffusion is given by the application of the procedure described in Section 2.2.2. The disadvantage of this approach is that we cannot introduce nor an intelligent diffusion (like escaping from predators or chasing for preys), nor different diffusion rates for different species. An alternative could be the introduction of diffusion on a pair basis: two cells can exchange their identity according with a given set of rules.⁸

For the moment we can use the following rule:

- Divide the lattice in neighboring pairs (in $d = 1$ and no dependence on the neighbors there are two ways of doing that)

⁷There are several definition of fitness, the one given here can be connected to the growth rate A of a genetically pure population by $A = \exp(H)$; see also Section 4.4

⁸There are several ways of dividing a lattice into couples of neighboring cells. If one want to update them in parallel, and if the updating rule depends on the state of neighboring cells, one cannot update a couple at the same time of a cell in the neighborhood. Since one has to update a sublattice after another, there is a slightly asymmetry that vanishes in the limit of a small diffusion probability iterated over several sublattices.

- Exchange the values of the cells with probability D

Clearly the probability D could depend on the value of the cells and on that of neighbors.

reproduction: The reproduction phase can be implemented as a rule for empty cells: they choose one of the neighbors at random and copy its identity with some errors, determined by a mutational probability ω .

Thus

- If the cell has value $*$ then
 - choose one of the neighbors;
 - copy its state;
 - for each bits in the genome replace the bit with its opposite with probability ω ;
- Else do nothing.

We have now to describe the structure of the interacting matrix \mathbf{J} . I shall deal with a very smooth correspondence between phenotype and genotype: two similar genotypes have also similar phenotype. With this assumptions x represents a strain rather than a species. This hypothesis implies the smoothness of \mathbf{J} .

First of all I introduce the intraspecies competition. Since the individuals with similar genomes are the ones that share the largest quantity of resources, then the competition is stronger the nearer the genotypes. This implies that $J[x, x] < 0$.

The rest of \mathbf{J} is arbitrary. For a classification in terms of usual ecological interrelations, one has to consider together $J[x, y]$ and $J[y, x]$. One can have four cases:

$J[x, y] < 0$	$J[y, x] < 0$	competition
$J[x, y] > 0$	$J[y, x] < 0$	predation or parasitism
$J[x, y] < 0$	$J[y, x] > 0$	predation or parasitism
$J[x, y] > 0$	$J[y, x] > 0$	cooperation

One has two choices for the geometry of the genetic space.

hypercubic distance: The genetic distance between x and y is given by the number of bits that are different, i.e. $d(x, y) = \|x \oplus y\|$, where the norm $\|x\|$ of a Boolean vector x is defined as $\|x\| = (1/N) \sum_{i=1}^N x_i$.

linear distance: The genetic space is arranged on a line (excluding the 0 and with periodic boundary conditions) rather than on a hypercube. This arrangement is simpler but less biological;⁹

⁹An instance of a similar (sub-)space in real organisms is given by a repeated gene (say a

4.4 Mean field approximation

Since the model is rather complex, it is better to start¹⁰ with the mean field approximation, disregarding the spatial structure. This approximation becomes exact in the limit of large diffusion or when the coupling extends on all the lattice.¹¹

Let $n(x)$ be the number of organisms with genetic code x , and n_* the number of empty sites. if N is the total number of cells, we have

$$\begin{aligned} n_* + \sum_x n(x) &= N; \\ m = \frac{1}{N} \sum_x n(x) &= 1 - \frac{n_*}{N}; \end{aligned}$$

considering the sums extended to all "not-empty" genetic codes, and indicating with m the fraction of non-empty sites (i.e. the population size).

We shall label with a tilde the quantities after the survival step, with a prime after the reproduction step. The evolution of the system will be ruled by the following equations:

$$\begin{aligned} \tilde{n}(x) &= \pi(x, n)n(x); \\ n'(x) &= \tilde{n}(x) + \frac{\tilde{n}_*}{N} \sum_y W(x, y)\tilde{n}(y). \end{aligned} \tag{26}$$

The matrix $W(x, y)$ is the probability of mutating from genotype y to x . For a given codon mutation probability μ , $W(x, y)$ is given by

hypercubic distance:

$$W(x, y) = \mu^{d(x,y)}(1 - \mu)^{l-d(x,y)}; \tag{27}$$

linear distance:

$$\begin{aligned} W(x, y) &= \mu && \text{if } |x - y| = 1; \\ W(x, x) &= 1 - 2\mu; \\ W(x, y) &= 0 && \text{otherwise.} \end{aligned} \tag{28}$$

tRNA gene): a fraction of its copies can mutate, linearly varying the fitness of the individual with the "chemical composition" of the gene. [21] This degenerate case has been widely studied (see for instance Alves and Fontanari (1996) [22]). The linear space is equivalent to the hypercubic space if the phenotype $g(x)$ does not depend on the disposition of bits in x (i.e. it depends only on the number of ones – a totalistic function): one should introduce in this case the multiplicity of a degenerate state, which can be approximated to a Gaussian, but if one works in the neighborhood of its maximum (the most common chemical composition) the multiplicity factors are nearly constants. Another example is given by the level of catalytic activity of a protein. A linear space has also been used for modeling the evolution of RNA viruses on HeLa cultures. [23]

¹⁰We also stop in this lecture with this approximation.

¹¹Since the original model is still in development, I shall not study the exact mean field version of the above description, but a simplified one.

In any case the mutations represent a diffusion process in genic space, thus the matrix W conserves the total population, and

$$\sum_x W(x, y) = \sum_y W(x, y) = 1. \quad (29)$$

Summing over the genomes x , we can rewrite Eq. (26) for m as

$$\begin{aligned} \tilde{m} &= \frac{1}{N} \sum_x \tilde{n}(x) = \frac{1}{N} \sum_x \pi(x, n)n(x) \\ m' &= \frac{1}{N} \sum_x n'(x) = \tilde{m} + \frac{\tilde{n}_*}{N^2} \sum_{xy} W(x, y)\tilde{n}(y). \end{aligned}$$

Introducing the probability distribution of occupied sites $p(x) = \frac{n(x)}{mN}$ ($\sum_x p(x) = 1$), and the average fitness $\bar{\pi}$ as

$$\bar{\pi} \equiv \frac{1}{mN} \sum_x \pi(x, n)n(x) = \sum_x \pi(x)p(x),$$

and thus

$$\tilde{m} = m\bar{\pi}; \quad \frac{\tilde{n}_*}{N} = 1 - m\bar{\pi}.$$

Using the property (29) we obtain

$$m' = m\bar{\pi}(2 - m\bar{\pi}). \quad (30)$$

The stationary condition ($m' = m$) gives

$$\begin{aligned} 1 &= \bar{\pi}(2 - m\bar{\pi}); \\ m &= \frac{2\bar{\pi} - 1}{\bar{\pi}^2}. \end{aligned}$$

The normalized evolution equation for $p(x)$ is thus

$$p'(x) = \frac{\pi(x, p, m)p(x) + (1 - m\bar{\pi}) \sum_y W(x, y)\pi(x, p, m)p(y)}{\bar{\pi}(2 - m\bar{\pi})}; \quad (31)$$

where the usual fitness function, Eq.(22), has to be written in term of $p(x)$

$$H(x, p, m) = h[x] + m \sum_y J[x, y]p(y)$$

Notice that Eq.(30) corresponds to the usual logistic equation for population dynamics if we keep the average fitness $\bar{\pi}$ constant.

4.5 Speciation due to the competition among strains

One of the feature of the model is the appearance of the species intended as a cluster of strains connected by mutations. [24]

One can consider the following analogy with a Turing mechanism for chemical pattern formation. The main ingredients are an autocatalytic reaction process (reproduction) with slow diffusion (mutations) coupled with the emission of a short-lived, fast-diffusing inhibitor (competition). In this way a local high concentration of autocatalytic reactants inhibits the growth in its neighborhood, acting as a local negative interaction.

In genetic space, the local coupling is given by the competition among genetically kin individuals. For instance, assuming a certain distribution of some resource (such as some essential metabolic component for a bacterial population), then the more genetically similar two individuals are, the wider the fraction of shared resources is. The effects of competition on strain x by strain y are modeled by a term proportional to the relative abundance of the latter, $p(y)$, modulated by a function that decreases with the phenotypic distance between x and y . Another example of this kind of competition can be found in the immune response in mammals. Since the immune response has a certain degree of specificity, a viral strain x can suffer from the response triggered by strain y if they are sufficiently near in an appropriate phenotypic subspace. Again, one can think that this effective competition can be modeled by a term, proportional to the relative abundance of the strain that originated the response, which decreases with the phenotypic distance.

Let us start with a one dimensional “chemical” model of cells that reproduce asexually and slowly diffuse (in real space), $p = p(x, t)$ being their relative abundance at position x and at time t . These cells constitutively emit a short-lived, fast-diffusing mitosis inhibitor $q = q(x, t)$. This inhibitor may be simply identified with some waste or with the consumption of a local resource (say oxygen). The diffusion of the inhibitor is modeled as

$$\frac{\partial q}{\partial t} = k_0 p + D \frac{\partial^2 q}{\partial x^2} - k_1 q, \quad (32)$$

where k_0 , k_1 and D are the production, annihilation and diffusion rates of q .

The evolution of the distribution p is given by

$$\frac{\partial p}{\partial t} = (A(x, t) - \bar{A}(t)) p + \mu \frac{\partial^2 p}{\partial x^2}, \quad (33)$$

$$\bar{A}(t) = \int A(y, t) p(y, t) dy. \quad (34)$$

The growth rate A can be expressed in terms of the fitness H as

$$A(x, t) = \exp(H(x, t)). \quad (35)$$

Due to the form of equation (33), at each time step we have

$$\sum_x p(x, t) = 1. \quad (36)$$

The diffusion rate of q , D , is assumed to be much larger than μ . The growth rate A , can be decomposed in two factors, $A(x, t) = A_0(x)A_1(q(x, t))$, where A_0 gives the reproductive rate in absence of q , so $A_1(0) = 1$. In presence of a large concentration of the inhibitor q the reproduction stops, so $A_1(\infty) = 0$. A possible choice is

$$A(x, t) = \exp(H_0(x) - q(x, t)).$$

For instance, $H_0(x)$ could model the sources of food or, for algae culture, the distribution of light.

Since we assumed a strong separation in time scales, we look for a stationary distribution $\tilde{q}(x, t)$ of the inhibitor (Eq. (32)) by keeping p fixed. This is given by a convolution of the distribution p :

$$\tilde{q}(x, t) = J \int \exp\left(-\frac{|x-y|}{R}\right) p(y, t) dy,$$

where J and R depend on the parameters k_0 , k_1 , D . In the following we shall use J and R as control parameters, disregarding their origin.

We can generalize this scenario to non-linear diffusion processes of the inhibitor by using the reaction-diffusion equation Eq. (33), with the fitness H and the kernel K given by

$$H(x, t) = H_0(x) - J \int K\left(\frac{x-y}{R}\right) p(y, t) dy \quad (37)$$

$$K(r) = \exp\left(-\frac{|r|^\alpha}{\alpha}\right), \quad (38)$$

i.e. a symmetric decreasing function of r with $K(0) = 1$. The parameters J and α control the intensity of the competition and the steepness of the interaction, respectively.

Let us consider the correspondence with the genetic space: the quantity x now identifies a genome, the diffusion rate μ is given by mutations, and the inhibitor q (which is no more a real substance) represents the competition among phenotypically related strains. The effects of competition are much faster than the genetic drift (mutations), so that the previous hypotheses are valid. While the competition interaction kernel $K(r)$ is not given by a diffusion process, its general form should be similar to that of Eq. (38): a decreasing function of the phenotypic distance between two strains. We shall refer to the p -independent contribution to the fitness, $H_0[x]$, as the static fitness landscape.

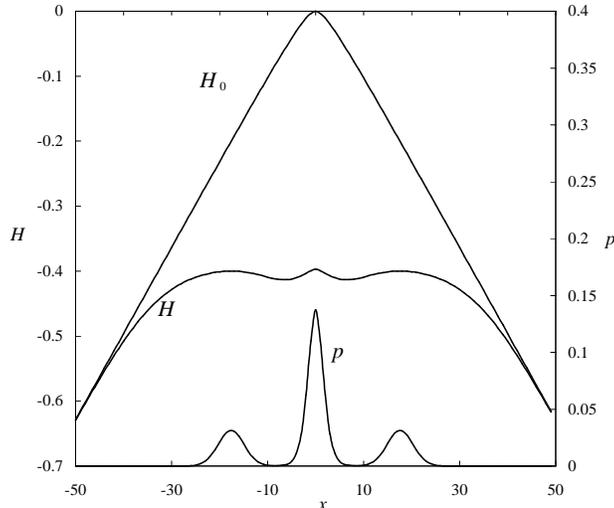


Figure 17: Static fitness H_0 , effective fitness H , and asymptotic distribution p numerically computed for the following values of parameters: $\alpha = 2$, $\mu = 0.01$, $H_0 = 1.0$, $b = 0.04$, $J = 0.6$, $R = 10$ and $r = 3$.

Our model is thus defined by Eqs. (33–38). We are interested in its asymptotic behavior in the limit $\mu \rightarrow 0$. Actually, the mutation mechanism is needed only to define the genetic distance and to allow population of an eventual niche. The results should not change qualitatively if one includes more realistic mutation mechanisms.

Let us first examine the behavior of Eq. (33) in absence of competition ($J = 0$) for a smooth static landscape and a vanishing mutation rate. This corresponds to the Eigen model in one dimension: since it does not exhibit any phase transition, the asymptotic distribution is unique. The asymptotic distribution is given by one delta function peaked around the global maximum of the static landscape, or more delta functions (coexistence) if the global maxima are degenerate. The effect of a small mutation rate is simply that of broadening the distribution from a delta peak to a bell-shaped curve [25].

While the degeneracy of maxima of the static fitness landscape is a very particular condition, we shall show in the following that in presence of competition this is a generic case. For illustration, we report in Fig. 17 the numerical computation of the asymptotic behavior of the model for a possible evolutive scenario that leads to the coexistence of three species. We have chosen a smooth static fitness H_0 (see Eq. (49)) and a Gaussian ($\alpha = 2$) competition kernel, using the genetic distance in place of the phenotypic one. The effective fitness H is almost degenerate (here $\mu > 0$ and the competition effect extends on the neighborhood

of the maxima), and this leads to the coexistence.

In the following we shall assume again that the phenotypic space is the same of the genotypic space.

4.5.1 Evolution near a maximum

We need the expression of \mathbf{p} if a given static fitness $H(x)$ or a static growth rate $A(x)$ has a smooth, isolated maximum for $x = 0$ (*smooth maximum* approximation). Let us assume that

$$A(x) \simeq A_0(1 - ax^2), \quad (39)$$

where $A_0 = A(0)$.

The generic evolution equation (master equation) for the probability distribution is

$$\alpha(t)p(x, t + 1) = \left(1 + \mu \frac{\delta^2}{\delta x^2}\right) A(x, \mathbf{p}(t))p(x, t); \quad (40)$$

where the discrete second derivative $\delta^2/\delta x^2$ is defined as

$$\frac{\delta f(x)}{\delta x^2} = f(x + 1) + f(x - 1) - 2f(x),$$

and $\alpha(t)$ maintains the normalization of $\mathbf{p}(t)$. In the following we shall mix freely the continuous and discrete formulations of the problem.

Summing over x in Eq. (40) and using the normalization condition, Eq. (36), we have:

$$\alpha = \sum_x A(x, \mathbf{p})p(x) = \bar{A}. \quad (41)$$

The normalization factor α thus corresponds to the average fitness. The quantities A and α are defined up to an arbitrary constant.

If A is sufficiently smooth (including the dependence on \mathbf{p}), one can rewrite Eq. (40) in the asymptotic limit, using a continuous approximation for x as

$$\alpha p = Ap + \mu \frac{\partial^2}{\partial x^2}(Ap), \quad (42)$$

Where we have neglected to indicate the genotype index x and the explicit dependence on \mathbf{p} . Eq. (42) has the form of a nonlinear diffusion-reaction equation. Since we want to investigate the phenomenon of species formation, we look for an asymptotic distribution \mathbf{p} formed by a superposition of several non-overlapping bell-shaped curves, where the term non-overlapping means almost uncoupled by mutations. Let us number these curves using the index i , and denote each of them as $p_i(x)$, with $p(x) = \sum_i p_i(x)$. Each $p_i(x)$ is centered around \bar{x}_i and

its weight is $\int p_i(x)dx = \gamma_i$, with $\sum_i \gamma_i = 1$. We further assume that each $p_i(x)$ obeys the same asymptotic condition, Eq. (42) (this is a sufficient but not necessary condition). Defining

$$\bar{A}_i = \frac{1}{\gamma_i} \int A(x)p_i(x)dx = \alpha, \quad (43)$$

we see that in a stable ecosystem all quasi-species have the same average fitness.

Substituting $q = Ap$ in Eq. (42) we have (neglecting to indicate the genotype index x , and using primes to denote differentiation with respect to it):

$$\frac{\alpha}{A}q = q + \mu q''.$$

Looking for $q = \exp(w)$,

$$\frac{\alpha}{A} = 1 + \mu(w'^2 + w''),$$

and approximating $A^{-1} = A_0^{-1}(1 + ax^2)$, we have

$$\frac{\alpha}{A_0}(1 + ax^2) = 1 + \mu(w'^2 + w''). \quad (44)$$

A possible solution is

$$w(x) = -\frac{x^2}{2\sigma^2}.$$

Substituting into Eq. (44) we finally get

$$\frac{\alpha}{A_0} = \frac{2 + a\mu - \sqrt{4a\mu + a^2\mu^2}}{2}. \quad (45)$$

Since $\alpha = \bar{A}$, α/A_0 is less than one we have chosen the minus sign. In the limit $a\mu \rightarrow 0$ (small mutation rate and smooth maximum), we have

$$\frac{\alpha}{A_0} \simeq 1 - \sqrt{a\mu}$$

and

$$\sigma^2 \simeq \sqrt{\frac{\mu}{a}}. \quad (46)$$

The asymptotic solution is

$$p(x) = \gamma \frac{1 + ax^2}{\sqrt{2\pi}\sigma(1 + a\sigma^2)} \exp\left(-\frac{x^2}{2\sigma^2}\right),$$

so that $\int p(x)dx = \gamma$. The solution is a bell-shaped curve, its width σ being determined by the combined effects of the curvature a of maximum and the mutation rate μ . In the next section, we shall apply these results to a quasi-species i . In this case one should substitute $p \rightarrow p_i$, $\gamma \rightarrow \gamma_i$ and $x \rightarrow x - \bar{x}_i$.

For completeness, we study also the case of a *sharp maximum*, for which $A(x)$ varies considerably with x . In this case the growth rate of less fit strains has a large contribution from the mutations of fittest strains, while the reverse flow is negligible, thus

$$p(x-1)A(x-1) \gg p(x)A(x) \gg p(x+1)A(x+1)$$

neglecting last term, and substituting $q(x) = A(x)p(x)$ in Eq. (40) we get:

$$\frac{\alpha}{A_0} = 1 - 2\mu \quad \text{for } x = 0 \quad (47)$$

$$q(x) = \frac{\mu}{(\alpha A(x) - 1 + 2\mu)} q(x-1) \quad \text{for } x > 0 \quad (48)$$

Near $x = 0$, combining Eq. (47), Eq. (48) and Eq. (39), we have

$$q(x) = \frac{\mu}{(1 - 2\mu)ax^2} q(x-1).$$

In this approximation the solution is

$$q(x) = \left(\frac{\mu}{1 - 2\mu a} \right)^x \frac{1}{(x!)^2},$$

and

$$y(x) = A(x)q(x) \simeq \frac{1}{A_0} (1 + ax^2) \left(\frac{\mu A_0}{\alpha a} \right)^x \frac{1}{x!^2}.$$

One can check the validity of these approximations by solving numerically Eq. (40); the comparisons are shown in Fig. 18. One can check that the *smooth maximum* approximation agrees with the numerics for small values of a , when $A(x)$ varies slowly with x , while the *sharp maximum* approximation agrees with the numerical results for large values of a , when small variations of x correspond to large variations of $A(x)$.

4.5.2 Speciation

I shall now derive the conditions for the coexistence of multiple species. Let us assume that the asymptotic distribution is formed by L delta peaks p_k , $k = 0, \dots, L-1$, for a vanishing mutation rate (or L non-overlapping bell shaped curves for a small mutation rate) centered at y_k . The weight of each quasi species is γ_k , i.e.

$$\int p_k(x)dx = \gamma_k, \quad \sum_{k=0}^{L-1} \gamma_k = 1.$$

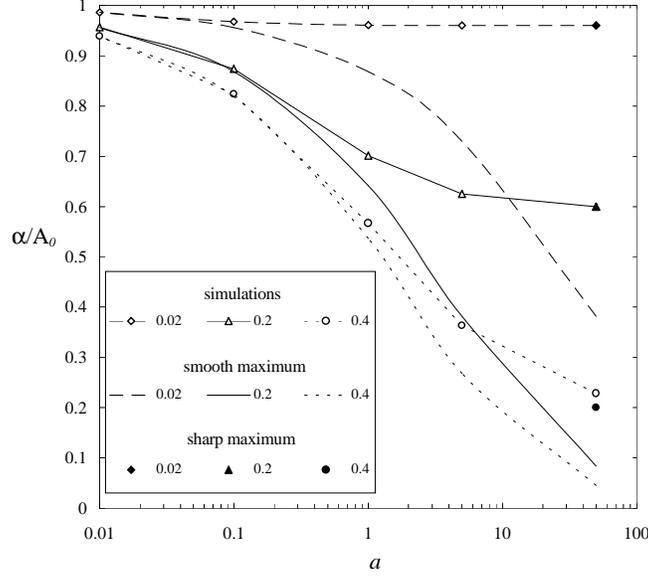


Figure 18: Average fitness α/A_0 versus the coefficient a , of the fitness function, Eq. (39), for some values of the mutation rate μ . Legend: *numerical resolution* corresponds to the numerical solution of Eq. (40), *smooth maximum* refers to Eq. (45) and *sharp maximum* to Eq. (47)

The quasi-species are ordered such as $\gamma_0 \geq \gamma_1, \dots, \geq \gamma_{L-1}$.

The evolution equations for the p_k are ($\mu \rightarrow 0$)

$$\frac{\partial p_k}{\partial t} = (A(y_k) - \bar{A})p_k,$$

where $A(x) = \exp(H(x))$ and

$$H(x) = H_0(x) - J \sum_{j=0}^{L-1} K \left(\frac{x - y_j}{R} \right) \gamma_j.$$

The stability condition of the asymptotic distribution is $(A(y_k) - \bar{A})p_k = 0$, i.e. either $A(y_k) = \bar{A} = \text{const}$ (degeneracy of maxima) or $p_k = 0$ (all other points). In other terms one can say that in a stable environment the fitness of all individuals is the same, independently on the species.

The position y_k and the weight γ_k of the quasi-species are given by $A(y_k) = \bar{A} = \text{const}$ and $\partial A(x)/\partial x|_{y_k} = 0$, or, in terms of the fitness H , by

$$H_0(y_k) - J \sum_{j=0}^{L-1} K \left(\frac{y_k - y_j}{R} \right) \gamma_j = \text{const}$$

$$H'_0(y_k) - \frac{J}{R} \sum_{j=0}^{L-1} K' \left(\frac{y_k - y_j}{R} \right) \gamma_j = 0$$

Let us compute the phase boundary for coexistence of three species for two kinds of kernels: the exponential (diffusion) one ($\alpha = 1$) and a Gaussian one ($\alpha = 2$).

I assume that the static fitness $H_0(x)$ is a symmetric linear decreasing function except in the vicinity of $x = 0$, where it has a quadratic maximum:

$$H_0(x) = b \left(1 - \frac{|x|}{r} - \frac{1}{1 + |x|/r} \right) \quad (49)$$

so that close to $x = 0$ one has $H_0(x) \simeq -bx^2/r^2$ and for $x \rightarrow \infty$, $H_0(x) \simeq b(1 - |x|/r)$. Numerical simulations show that the results are qualitatively independent on the exact form of the static fitness, providing that it is a smooth decreasing function.

Due to the symmetries of the problem, we have one quasi-species at $x = 0$ and two symmetric quasi-species at $x = \pm y$. Neglecting the mutual influence of the two marginal quasi-species, and considering that $H'_0(0) = K'(0) = 0$, $K'(y/R) = -K'(-y/r)$, $K(0) = J$ and that the three-species threshold is given by $\gamma_0 = 1$ and $\gamma_1 = 0$, we have

$$\tilde{b} \left(1 - \frac{\tilde{y}}{\tilde{r}} \right) - K(\tilde{y}) = -1,$$

$$\frac{\tilde{b}}{\tilde{r}} + K'(\tilde{y}) = 0.$$

where $\tilde{y} = y/R$, $\tilde{r} = r/R$ and $\tilde{b} = b/J$. I introduce the parameter $G = \tilde{r}/\tilde{b} = (J/R)/(b/r)$, that is the ratio of two quantities, one related to the strength of inter-species interactions (J/R) and the other to intra-species ones (b/r). In the following I shall drop the tildes for convenience. Thus

$$r - z - G \exp \left(-\frac{z^\alpha}{\alpha} \right) = -G,$$

$$Gz^{\alpha-1} \exp \left(-\frac{z^\alpha}{\alpha} \right) = 1,$$

For $\alpha = 1$ we have the coexistence condition

$$\ln(G) = r - 1 + G.$$

The only parameters that satisfy these equations are $G = 1$ and $r = 0$, i.e. a flat landscape ($b = 0$) with infinite range interaction ($R = \infty$). Since the

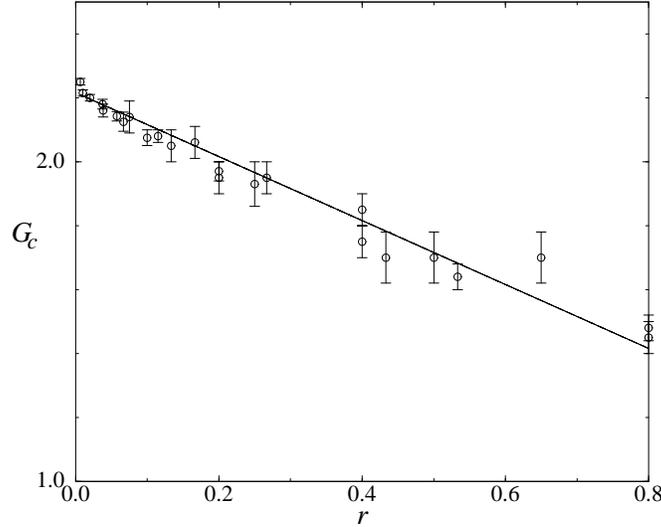


Figure 19: Three-species coexistence boundary G_c for $\alpha = 2$. The continuous line represents the analytical approximation, Eq. (50), the circles are obtained from numerical simulations. The error bars represent the maximum error.

coexistence region reduces to a single point, it is suggested that $\alpha = 1$ is a marginal case. Thus for less steep potentials, such as power law decrease, the coexistence condition is supposed not to be fulfilled.

For $\alpha = 2$ the coexistence condition is given by

$$z^2 - (G + r)z + 1 = 0,$$

$$Gz \exp\left(-\frac{z^2}{2}\right) = 1.$$

One can solve numerically this system and obtain the boundary $G_c(r)$ for the coexistence. In the limit $r \rightarrow 0$ (static fitness almost flat) one has

$$G_c(r) \simeq G_c(0) - r \tag{50}$$

with $G_c(0) = 2.216\dots$. Thus for $G > G_c(r)$ we have coexistence of three or more quasi-species, while for $G < G_c(r)$ only the fittest one survives.

I have solved numerically Eqs. (33–38) for several different values of the parameter G , considering a discrete genetic space, with N points, and a simple Euler algorithm. The results, presented in Fig. 2, are not strongly affected by the integration step. The error bars are due to the discreteness of the changing

parameter G . The boundary of the multi-species phase is well approximated by Eq. (50); in particular, I have checked that this boundary does not depend on the mutation rate μ , at least for $\mu < 0.1$, which can be considered a very high mutation rate for real organisms. The most important effect of μ is the broadening of quasi-species curves, which can eventually merge.

4.6 Final considerations

Possible measures on this abstract ecosystem model concern the origin of complexity (for instance considered equivalent to the entropy) in biological systems. One expects that steady ecosystems reach an asymptotic value of the complexity that maximizes the exploitation of energy, while perturbed ecosystems exhibit lower complexity. Another interesting topic regards the behavior of complexity in relation with the spatial extension of a system.

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