Chapter 6
Darwinian Evolution, Hypercycles and Game Theory

Adaptation and evolution are quasi synonymous in popular language and Darwinian evolution is a prime application of complex system theory. We will see that adaptation does not happen automatically and discuss the concept of "error catastrophe" as a possible root for the downfall of a species. Venturing into the mysteries surrounding the origin of life, we will investigate the possible advent of a "quasispecies" in terms of mutually supporting hypercycles. The basic theory of evolution is furthermore closely related to game theory, the mathematical theory of socially interacting agents, viz of rationally acting economic persons.

We will learn in this chapter, on one hand, that every complex dynamical system has its distinct characteristics to be considered. In the case of Darwinian evolution these are concepts like fitness, selection and mutation. These area specific notions interplay deeply with general concepts from dynamical and complex system theory, like the phenomenon of stochastic escape, which is operative in the realm of Darwinian evolution. Evolutionary processes lead, furthermore, to entire ecosystems with specific patterns of species abundances and we will discuss in this context the neutral theory of macroecology.

6.1 Introduction

Microevolution The ecosystem of the earth is a complex and adaptive system. It formed via Darwinian evolution through species differentiation and adaptation to a changing environment. A set of inheritable traits, the genome, is passed from parent to offspring and the reproduction success is determined by the outcome of random mutations and natural selection – a process denoted "microevolution"[1]

[1] Note that the term "macroevolution", coined to describe the evolution at the level of organisms, is nowadays somewhat obsolete.

Asexual Reproduction. One speaks of asexual reproduction when an individual has a single parent.

Here we consider mostly models for asexual reproduction, though most concepts can be easily generalized to the case of sexual reproduction.

Basic Terminology Let us introduce some basic variables needed to formulate the approach.

- Population M: The number of individuals.
  We assume here that M does not change with time, modeling the competition for a limited supply of resources.
- Genome N: Size of the genome.
  We encode the inheritable traits by a set of N binary variables,

$$\mathbf{s} = (s_1, s_2, \ldots, s_N), \qquad s_i = \pm 1 \ .$$

  N is considered fixed.
- Generations
  We consider time sequences of non-overlapping generations, like in a wheat field. The population present at time t is replaced by their offspring at generation $t + 1$.

In Table 6.1 some typical values for the size N of the genome are listed. Note the three orders of magnitude between simple eucaryotic life forms and the human genome.

State of the Population The state of the population at time t can be described by specifying the genomes of all the individuals,

$$\{\mathbf{s}^\alpha(t)\}, \qquad \alpha = 1 \ldots M, \qquad \mathbf{s} = (s_1, \ldots, s_N) \ .$$

We define by

$$X_{\mathbf{s}}(t), \qquad \sum_{\mathbf{s}} X_{\mathbf{s}}(t) = M \ , \tag{6.1}$$

Table 6.1 Genome size N and the spontaneous mutation rates μ, compare Eq. (6.3), per base for two RNA-based bacteria and DNA-based eucaryotes (From Jain and Krug (2006) and Drake et al. (1998))

Organism	Genome size	Rate per base	Rate per genome
Bacteriophage $Q\beta$	4.5×10^3	1.4×10^{-3}	6.5
Bacteriophage λ	4.9×10^4	7.7×10^{-8}	0.0038
E. Coli	4.6×10^6	5.4×10^{-10}	0.0025
C. Elegans	8.0×10^7	2.3×10^{-10}	0.018
Mouse	2.7×10^9	1.8×10^{-10}	0.49
Human	3.2×10^9	5.0×10^{-11}	0.16

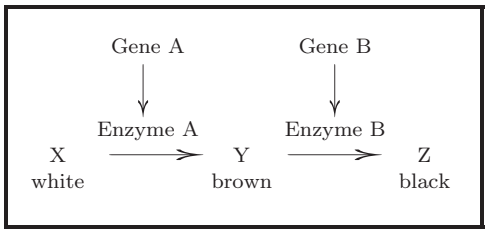

Fig. 6.1 A simple form of epistatic interaction occurs when the influence of one gene builds on the outcome of another. In this fictitious example black hair can only be realized when the gene for brown hair is also present

the number of individuals with genome $\mathbf{s}$ for each of the 2^N points $\mathbf{s}$ in the genome space. Typically, most of these occupation numbers vanish; biological populations are extremely sparse in genome space.

Combinatorial Genetics of Alleles Classical genetics focuses on the presence (or absence) of a few characteristic traits. These traits are determined by specific sites, denoted "loci", in the genome. The genetic realizations of these specific loci are called "alleles". Popular examples are alleles for blue, brown and green eyes.

Combinatorial genetics deals with the frequency change of the appearance of a given allele resulting from environmental changes during the evolutionary process. Most visible evolutionary changes are due to a remixing of alleles, as mutation induced changes in the genome are relatively rare; compare the mutation rates listed in Table 6.1.

Beanbag Genetics Without Epistatic Interactions One calls "epistasis" the fact that the effect of the presence of a given allele in a given locus may depend on which alleles are present in some other loci, as illustrated in Fig. 6.1. Classical genetics neglects epistatic interactions. The resulting picture is often called "beanbag genetics", as if the genome were nothing but a bag carrying the different alleles within itself.

Genotype and Phenotype We note that the physical appearance of an organism is not determined exclusively by gene expression. One distinguishes between the genotype and the phenotype.

- The Genotype: The genotype of an organism is the class to which that organism belongs as determined by the DNA that was passed to the organism by its parents at the organism's conception.
- The Phenotype: The phenotype of an organism is the class to which that organism belongs as determined by the physical and behavioral characteristics of the organism, for example its size and shape, its metabolic activities and its pattern of movement.

Selection acts, strictly speaking, only upon phenotypes, but only the genotype is bequeathed. The variations in phenotypes then act as a source of noise for the selection process.

Speciation One denotes by "speciation" the process leading to the differentiation of an initial species into two distinct species. Speciation occurs due to adaptation to different ecological niches, often in distinct geographical environments. We will not treat the various theories proposed for speciation here.

6.2 Mutations and Fitness in a Static Environment

Constant Environment We consider here the environment to be static; an assumption that is justified for the case of short-term evolution. This assumption clearly breaks down for long time scales, as already discussed in Chap. **??** since the evolutionary change of one species might lead to repercussions all over the ecosystem to which it appertains.

Independent Individuals An important issue in the theory of evolution is the emergence of specific kinds of social behavior. Social behavior can only arise if the individuals of the same population interact. We discuss some of these issues in Sect. 6.7 in the context of game theory. Until then we assume non-interacting individuals, which implies that the fitness of a given genetic trait is independent of the frequency of this and of other alleles, apart from the overall competition for resources.

Constant Mutation Rates We furthermore assume that the mutation rates are

- Constant over time,
- Independent of the locus in the genome, and
- Not subject to genetic control.

Any other assumption would require a detailed microbiological modeling; a subject beyond our scope.

Stochastic Evolution The evolutionary process can then be modeled as a three-stage stochastic process:

1. Reproduction: The individual α at generation t is the offspring of an individual α' living at generation $t-1$. Reproduction is thus represented as a stochastic map
$$\alpha \quad \longrightarrow \quad \alpha' = G_t(\alpha) \ , \tag{6.2}$$
where $G_t(\alpha)$ is the parent of the individual α, and is chosen at random among the M individuals living at generation $t-1$. For the reproduction process illustrated in Fig. 6.2 one has $G_t(1) = 1$, $G_t(2) = 3$, and so on.

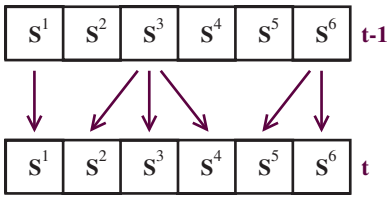

Fig. 6.2 Illustration of a basic reproduction process proceeding from generation $t-1$ to t, with individuals 1, 3, 6 having 1, 3 and 2 descendents respectively

2. Mutation: The genomes of the offspring differ from the respective genomes of their parents through random changes.
3. Selection: The number of surviving offspring of each individual depends on its genome; it is proportional to its "fitness", which is a functional of the genome.

Point Mutations and Mutation Rate Here we consider mostly independent point mutations, namely that every element of the genome is modified independently of the other elements,

$$s_i^{\alpha}(t) = -s_i^{G_t(\alpha)}(t-1) \qquad \text{with probability} \quad \mu \ , \tag{6.3}$$

where the parameter $\mu \in [0,1/2]$ is the microscopic "mutation rate". In real organisms, more complex phenomena take place, like global rearrangements of the genome, copies of some part of the genome, displacements of blocks of elements from one location to another, and so on. The values for the real-world mutation rates μ for various species listed in Table 6.1 are therefore to be considered as effective mutation rates.

Fitness and Fitness Landscape The fitness $W(\mathbf{s})$, also called "Wrightian fitness", of a genotype trait $\mathbf{s}$ is proportional to the average number of offspring an individual possessing the trait $\mathbf{s}$ has. It is strictly positive and can therefore be written as

$$W(\mathbf{s}) = e^{kF(\mathbf{s})} \quad \propto \quad \text{average number of offspring of } \mathbf{s}. \tag{6.4}$$

Selection acts in first place upon phenotypes, but we neglect here the difference, considering the variations in phenotypes as a source of noise, as discussed above. The parameters in Eq. (6.4) are denoted:

– $W(\mathbf{s})$: Wrightian fitness,
– $F(\mathbf{s})$: fitness landscape,
– k: inverse selection temperature,[2] and

[2] The probability to find a state with energy E in a thermodynamic system with temperature T is proportional to the Boltzmann factor $\exp(-\beta E)$. The inverse temperature is $\beta = 1/(k_B T)$, with k_B being the Boltzmann constant.

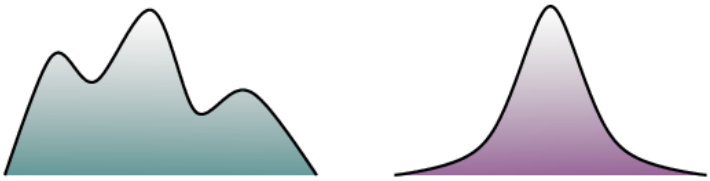

Fig. 6.3 Illustration of idealized (smooth) one-dimensional model fitness landscapes $F(\mathbf{s})$. Real-world fitness landscapes, however, contain discontinuities. *Left*: a fitness landscape with peaks and valleys, metaphorically also called a "rugged landscape". *Right*: a fitness landscape containing a single smooth peak, as described by Eq. (6.24)

– $w(\mathbf{s})$: Malthusian fitness, when rewriting Eq. (6.4) as $W(\mathbf{s}) = e^{w(\mathbf{s})\Delta t}$, where Δt is the generation time.

We will work here with discrete time, viz with non-overlapping generations, and make use only of the Wrightian fitness $W(\mathbf{s})$.

Fitness of Individuals Versus Fitness of Species We remark that this notion of fitness is a concept defined at the level of individuals in a homogeneous population. The resulting fitness of a species or of a group of species needs to be explicitly evaluated and is model-dependent.

Fitness Ratios The assumption of a constant population size makes the reproductive success a *relative* notion. Only the ratios

$$\frac{W(\mathbf{s}_1)}{W(\mathbf{s}_2)} = \frac{e^{kF(\mathbf{s}_1)}}{e^{kF(\mathbf{s}_2)}} = e^{k[F(\mathbf{s}_1)-F(\mathbf{s}_2)]} \tag{6.5}$$

are important. It follows that the quantity $W(\mathbf{s})$ is defined up to a proportionality constant and, accordingly, the fitness landscape $F(\mathbf{s})$ only up to an additive constant, much like the energy in physics.

The Fitness Landscape The graphical representation of the fitness function $F(\mathbf{s})$ is not really possible for real-world fitness functions, due to the high dimensional 2^N of the genome space. It is nevertheless customary to draw a fitness landscape, like the one shown in Fig. 6.3. However, one must bear in mind that these illustrations are not to be taken at face value, apart from model considerations.

The Fundamental Theorem of Natural Selection The so-called fundamental theorem of natural selection, first stated by Fisher in 1930, deals with adaptation in the absence of mutations and in the thermodynamic limit $M \to \infty$. An infinite population size allows one to neglect fluctuations.

The theorem states that the average fitness of the population cannot decrease in time under these circumstances, and that the average fitness becomes stationary only when all individuals in the population have the maximal reproductive fitness.

The proof is straightforward. We define by

$$\langle W\rangle_t \;\equiv\; \frac{1}{M}\sum_{\alpha=1}^{M} W\left(\mathbf{s}^\alpha(t)\right) = \frac{1}{M}\sum_{\mathbf{s}} W\left(\mathbf{s}\right) X_{\mathbf{s}}(t)\ , \tag{6.6}$$

the average fitness of the population, with $X_{\mathbf{s}}$ being the number of individual having the genome $\mathbf{s}$. Note that the $\sum_{\mathbf{s}}$ in Eq. (6.6) contains 2^N terms. The evolution equations are given in the absence of mutations by

$$X_{\mathbf{s}}(t+1) = \frac{W(\mathbf{s})}{\langle W\rangle_t}\, X_{\mathbf{s}}(t)\ , \tag{6.7}$$

where $W(\mathbf{s})/\langle W\rangle_t$ is the relative reproductive success. The overall population size remains constant,

$$\sum_{\mathbf{s}} X_{\mathbf{s}}(t+1) = \frac{1}{\langle W\rangle_t}\sum_{\mathbf{s}} X_{\mathbf{s}}(t) W(\mathbf{s}) = M\ , \tag{6.8}$$

where we have used Eq. (6.6) for $\langle W\rangle_t$. Then

$$\begin{aligned}\langle W\rangle_{t+1} &= \frac{1}{M}\sum_{\mathbf{s}} W\left(\mathbf{s}\right) X_{\mathbf{s}}(t+1) = \frac{\frac{1}{M}\sum_{\mathbf{s}} W^2(\mathbf{s}) X_{\mathbf{s}}(t)}{\frac{1}{M}\sum_{\mathbf{s}'} W(\mathbf{s}') X_{\mathbf{s}'}(t)} \\ &= \frac{\langle W^2\rangle_t}{\langle W\rangle_t} \;\geq\; \langle W\rangle_t\ ,\end{aligned} \tag{6.9}$$

since $\langle W^2\rangle_t - \langle W\rangle_t^2 = \langle \Delta W^2\rangle_t \geq 0$. The steady state

$$\langle W\rangle_{t+1} = \langle W\rangle_t, \qquad \langle W^2\rangle_t = \langle W\rangle_t^2\ ,$$

is only possible when all individuals $1 \ldots M$ in the population have the same fitness, viz the same genotype.

The Baldwin effect Variations in the phenotype may be induced not only via stochastic influences of the environment, but also through adaption of the phenotype itself to the environment, viz through learning. Learning can actually speed up evolution whenever the underlying fitness landscape is very rugged, by smoothing it out and providing a stable gradient towards to the genotype with the maximal fitness. One speaks of the "Baldwin effect".

6.3 Deterministic Evolution

Mutations are random events and the evolution process is therefore a stochastic process. But stochastic fluctuations become irrelevant in the limit of infinite population size $M \to \infty$; they average out. In this limit the equations governing evolution become deterministic and only the average transition rates are relevant. One can then study in detail the condition necessary for adaptation to occur for various mutation rates.

6.3.1 Evolution Equations

The Mutation Matrix The mutation matrix

$$Q_\mu(\mathbf{s}' \to \mathbf{s}), \qquad \sum_{\mathbf{s}} Q_\mu(\mathbf{s}' \to \mathbf{s}) = 1 \tag{6.10}$$

denotes the probabilities of obtaining a genotype $\mathbf{s}$ when attempting to reproduce an individual with genotype $\mathbf{s}'$. The mutation rates $Q_\mu(\mathbf{s}' \to \mathbf{s})$ may depend on a parameter μ determining the overall mutation rate. The mutation matrix includes the absence of any mutation, viz the transition $Q_\mu(\mathbf{s}' \to \mathbf{s}')$. It is normalized.

Deterministic Evolution with Mutations We generalize Eq. (6.7), which is valid in the absence of mutations, by including the effect of mutations via the mutation matrix $Q_\mu(\mathbf{s}' \to \mathbf{s})$:

$$X_{\mathbf{s}}(t+1)/M = \left(\sum_{\mathbf{s}'} X_{\mathbf{s}'}(t) W(\mathbf{s}') Q_\mu(\mathbf{s}' \to \mathbf{s})\right) \Big/ \left(\sum_{\mathbf{s}'} W_{\mathbf{s}'} X_{\mathbf{s}'}(t)\right) ,$$

or

$$x_{\mathbf{s}}(t+1) = \frac{\sum_{\mathbf{s}'} x_{\mathbf{s}'}(t) W(\mathbf{s}') Q_\mu(\mathbf{s}' \to \mathbf{s})}{\langle W \rangle_t}, \qquad \langle W \rangle_t = \sum_{\mathbf{s}'} W_{\mathbf{s}'} x_{\mathbf{s}'}(t) , \tag{6.11}$$

where we have introduced the normalized population variables

$$x_{\mathbf{s}}(t) = \frac{X_{\mathbf{s}}(t)}{M}, \qquad \sum_{\mathbf{s}} x_{\mathbf{s}}(t) = 1 . \tag{6.12}$$

The evolution dynamics equation (6.11) retains the overall size $\sum_{\mathbf{s}} X_{\mathbf{s}}(t)$ of the population, due to the normalization of the mutation matrix $Q_\mu(\mathbf{s}' \to \mathbf{s})$, Eq. (6.10).

The Hamming Distance The Hamming distance

$$d_{\mathrm{H}}(\mathbf{s}, \mathbf{s}') = \sum_{i=1}^{N} \frac{(s_i - s_i')^2}{4} = \frac{N}{2} - \frac{1}{2} \sum_{i=1}^{N} s_i s_i' \tag{6.13}$$

measures the number of units that are different in two genome configurations $\mathbf{s}$ and $\mathbf{s}'$, e.g. before and after the effect of a mutation event.

The Mutation Matrix for Point Mutations We consider the simplest mutation pattern, viz the case of fixed genome length N and random transcription errors afflicting only individual loci. For this case, namely point mutations, the overall mutation probability

$$Q_\mu(\mathbf{s}' \to \mathbf{s}) = \mu^{d_{\mathrm{H}}} (1-\mu)^{N - d_{\mathrm{H}}} \tag{6.14}$$

is the product of the independent mutation probabilities for all loci $i = 1, \ldots, N$, with d_H denoting the Hamming distance $d_{\rm H}(\mathbf{s}, \mathbf{s}')$ given by Eq. (6.13) and μ the mutation rate μ defined in Eq. (6.3). One has

$$\sum_{\mathbf{s}} Q_\mu(\mathbf{s}' \to \mathbf{s}) = \sum_{d_H} \binom{N}{d_H} (1-\mu)^{N-d_N} \mu^{d_N} = (1-\mu+\mu)^N \equiv 1$$

and the mutation matrix defined by Eq. (6.14) is consequently normalized. We rewrite the mutation matrix as

$$Q_\mu(\mathbf{s}' \to \mathbf{s}) \;\propto\; \exp\Big([\log(\mu) - \log(1-\mu)] d_H\Big) \;\propto\; \exp\left(\beta \sum_i s_i s_i'\right), \qquad (6.15)$$

where we denoted by β an effective inverse temperature, defined by

$$\beta = \frac{1}{2} \log\left(\frac{1-\mu}{\mu}\right). \qquad (6.16)$$

The relation of the evolution equation (6.15) to the partition function of a thermodynamical system, hinted at by the terminology "inverse temperature" will become evident below.

Evolution Equations for Point Mutations We now write the evolution equation (6.11) using the exponential representations for both the fitness $W(\mathbf{s}) = \exp[kF(\mathbf{s})]$, see Eq. (6.4) and for the mutation matrix $Q_\mu(\mathbf{s}' \to \mathbf{s})$,

$$x_{\mathbf{s}}(t+1) = \frac{1}{N_t} \sum_{\mathbf{s}'} x_{\mathbf{s}'}(t) \exp\left(\beta \sum_i s_i s_i' + kF(\mathbf{s}')\right) \qquad (6.17)$$

in a form that is suggestive of a statistical mechanics analogy. The normalization N_t in Eq. (6.17) takes care both of the average fitness $\langle W \rangle_t$ from Eq. (6.11) and of the normalization of the mutation matrix, compare Eq. (6.15).

Evolution Equations in Linear Form The evolution equation (6.17) is non-linear in the dynamical variables $x_{\mathbf{s}}(t)$, due to the average fitness $\langle W \rangle_t$ entering the normalization N_t. A suitable change of variables does, however, allow the evolution equation to be cast into a linear form.

For this purpose we introduce the un-normalized variables $y_{\mathbf{s}}(t)$ via

$$x_{\mathbf{s}}(t) = \frac{y_{\mathbf{s}}(t)}{\sum_{\mathbf{s}'} y_{\mathbf{s}'}(t)}\,. \qquad (6.18)$$

Note that the normalization $\sum_{\mathbf{s}'} y_{\mathbf{s}'}(t)$ can be chosen freely for every generation $t = 1, 2, 3, \ldots$. The evolution equation (6.17) then becomes

$$y_{\mathbf{s}}(t+1) = Z_t \sum_{\mathbf{s}'} y_{\mathbf{s}'}(t) \exp\left(\beta \sum_i s_i s_i' + kF(\mathbf{s}')\right) \ , \tag{6.19}$$

where

$$Z_t = \frac{1}{N_t} \frac{\sum_{\mathbf{s}'} y_{\mathbf{s}'}(t+1)}{\sum_{\mathbf{s}'} y_{\mathbf{s}'}(t)} \ .$$

Choosing a different normalization for $y_{\mathbf{s}}(t)$ and for $y_{\mathbf{s}}(t+1)$ we may achieve $Z_t \equiv 1$. Equation (6.19) is then linear in $y_{\mathbf{s}}(t)$.

Statistical Mechanics of the Ising Model In the following we will make use of analogies to notations commonly used in statistical mechanics. Readers who are unfamiliar with the mathematics of the one-dimensional Ising model may skip the mathematical details and concentrate on the interpretation of the results.

We write the linear evolution equation (6.19) as

$$y_{\mathbf{s}}(t+1) = \sum_{\mathbf{s}'} e^{\beta H[\mathbf{s},\mathbf{s}']}\, y_{\mathbf{s}'}(t), \qquad y_{\mathbf{s}(t+1)} = \sum_{\mathbf{s}(t)} e^{\beta H[\mathbf{s}(t+1),\mathbf{s}(t)]}\, y_{\mathbf{s}(t)} \ , \tag{6.20}$$

where we denote by $H[\mathbf{s},\mathbf{s}']$ an effective Hamiltonian[3]

$$\beta H[\mathbf{s},\mathbf{s}'] = \beta \sum_i s_i s_i' + kF(\mathbf{s}') \ , \tag{6.21}$$

and where we renamed the variables $\mathbf{s}$ by $\mathbf{s}(t+1)$ and $\mathbf{s}'$ by $\mathbf{s}(t)$. Equation (6.20) can be solved iteratively,

$$y_{\mathbf{s}(t+1)} = \sum_{\mathbf{s}(t),\ldots,\mathbf{s}(0)} e^{\beta H[\mathbf{s}(t+1),\mathbf{s}(t)]} \ \cdots \ e^{\beta H[\mathbf{s}(1),\mathbf{s}(0)]}\, y_{\mathbf{s}(0)} \ , \tag{6.22}$$

with the two-dimensional Ising-type Hamiltonian[4]

$$\beta H = \beta \sum_{i,t} s_i(t+1) s_i(t) + k \sum_t F(\mathbf{s}(t)) \ . \tag{6.23}$$

~~It has a space dimension~~ $i = 1, \ldots, N$ and a time dimension t.

[3] The energy of a state depends in classical mechanics on the values of the available degrees of freedom, like the position and the velocity of a particle. This function is denoted Hamiltonian. In Eq. (6.21) the Hamiltonian is a function of the binary variables $\mathbf{s}$ and $\mathbf{s}'$.

[4] Any system of binary variables is equivalent to a system of interacting Ising spins, which retains only the classical contribution to the energy of interacting quantum mechanical spins (the magnetic moments).

6.3.2 Beanbag Genetics: Evolutions Without Epistasis

The Fujiyama Landscape The fitness function

$$F(\mathbf{s}) = \sum_{i=1}^{N} h_i s_i, \qquad W(\mathbf{s}) = \prod_{i=1}^{N} e^{k h_i s_i} \ , \tag{6.24}$$

is denoted the "Fujiyama landscape" since it corresponds to a single smooth peak as illustrated in Fig. 6.3. To see why, we consider the case $h_i > 0$ and rewrite Eq. (6.24) as

$$F(\mathbf{s}) = \mathbf{s}_0 \cdot \mathbf{s}, \qquad \mathbf{s}_0 = (h_1, h_2, \ldots, h_N) \ .$$

The fitness of a given genome $\mathbf{s}$ is directly proportional to the scalar product with the master sequence $\mathbf{s}_0$, with a well defined gradient pointing towards the master sequence.

The Fujiyama Hamiltonian No epistatic interactions are present in the smooth peak landscape Eq. (6.24). In terms of the corresponding Hamiltonian, see Eq. (6.23), this fact expresses itself as

$$\beta H = \beta \sum_{i=1}^{N} H_i, \qquad H_i = \sum_{t} s_i(t+1) s_i(t) + \frac{k h_i}{\beta} \sum_{t} s_i(t) \ . \tag{6.25}$$

Every locus i corresponds exactly to the one-dimensional $t = 1, 2, \ldots$ Ising model βH_i in an effective uniform magnetic field $k h_i / \beta$.

The Transfer Matrix The Hamiltonian equation (6.25) does not contain interactions between different loci of the genome; we can just consider a single Hamiltonian H_i and a single gene locus i. We define with

$$\mathbf{y} = \big(y_+(t), y_-(t)\big), \qquad \mathbf{y}^T = \begin{pmatrix} y_+(t) \\ y_-(t) \end{pmatrix}$$

the un-normalized densities of individuals having $s_i = +$ and $s_i = -$ respectively. Iterative solution Eq. (6.22) then has the form

$$\mathbf{y}(t+1) = \left(\prod_{t'=0}^{t} T \right) \mathbf{y}(0) = T^{t+1}\, \mathbf{y}^T(0) \ , \tag{6.26}$$

with the 2×2 transfer matrix

$$T = \mathrm{e}^{\beta H_i [s_i(t+1), s_i(t)]}, \qquad T = \begin{pmatrix} e^{\beta + k h_i} & e^{-\beta} \\ e^{-\beta} & e^{\beta - k h_i} \end{pmatrix} \ , \tag{6.27}$$

where we have used $s, s' = \pm 1$ and the symmetrized form

$$\beta H_i = \beta \sum_t s_i(t+1)s_i(t) + \frac{kh_i}{2} \sum_t \Big[s_i(t+1) + s_i(t) \Big] \ .$$

of the one-dimensional Ising model.

Eigenvalues of the Transfer Matrix For simplicity we take

$$h_i \ \equiv \ 1, \qquad \mathbf{s}_0 = (1, 1, \ldots, 1)$$

and evaluate the eigenvalues ω of the transfer matrix,

$$\omega^2 - 2\omega\, e^{\beta}\, \cosh(k) + e^{2\beta} - e^{-2\beta} = 0 \ .$$

The solutions are

$$\omega_{1,2} = e^{\beta}\, \cosh(k) \ \pm \ \sqrt{e^{2\beta} \cosh^2(k) - e^{2\beta} + e^{-2\beta}} \ .$$

In the following we consider with $k > 0$ non-trivial fitness landscapes. In the absence of a fitness landscape, $k = 0$, the eigenvalues are $\mathrm{e}^{\beta} \pm \mathrm{e}^{-\beta}$. No adaption takes place in this case, the eigenvectors $\mathbf{y} = (1, \pm 1)$ of the transfer matrix then weight all states in genome space with equal probabilities.

Dominance of the Largest Eigenvalue The genome distribution $\mathbf{y}$ is determined by large powers of the transfer matrix, $\lim_{t\to\infty} T^t$, via Eq. (6.26). The difference between the two eigenvalues of transfer matrix is

$$\Delta\omega = \omega_1 - \omega_2 = 2\sqrt{e^{2\beta} \sinh^2(k) + e^{-2\beta}} \ . \tag{6.28}$$

and hence zero only if $k = 0$ and $\beta \to \infty$. For $\Delta\omega > 0$ the larger eigenvalue ω_1 dominates in the limit $t \to \infty$ and $\mathbf{y} = (y_+, y_-)$ is given by the eigenvalue of ω_1, with $y_+ > y_-$.

Adaption in the Absence of Epistatic Interactions The sequence $\mathbf{s}_0 = (1, \ldots, 1)$ is the state with maximal fitness. The probability to find individuals with genome $\mathbf{s}$ at a Manhatten distance d_H from $\mathbf{s}_0$ is proportional to

$$(y_+)^{N-d_H}\, (y_-)^{d_H} \ \propto \ \left(\frac{y_-}{y_+} \right)^{d_H} \ .$$

The entire population is hence within a finite distance of the optimal genome sequence $\mathbf{s}_0$ whenever $y_-/y_+ < 1$, viz for $\Delta\omega > 0$. We recall that

$$\beta = \frac{1}{2} \log\left(\frac{1-\mu}{\mu} \right), \qquad W(\mathbf{s}) = e^{kF(\mathbf{s})} \ ,$$

where μ is the mutation rate for point mutations. Thus we see that there is some degree of adaptation whenever the fitness landscape does not vanish

($k > 0$). This is the case even for a maximal mutation rate $\mu \to 1/2$, for which $\beta \to 0$.

6.3.3 Epistatic Interactions and the Error Catastrophe

The result of the previous Sect. 6.3.2, i.e. the occurrence of adaptation in a smooth fitness landscape for any non-trivial model parameter, is due to the absence of epistatic interactions in the smooth fitness landscape. Epistatic interactions introduce a phase transition to a non-adapting regime once the mutation rate becomes too high.

The Sharp Peak Landscape One possibility to study this phenomenon is the limiting case of very strong epistatic interactions; in this case, a single element of the genotype does not give any information on the value of the fitness. This fitness is defined by the equation

$$W(\mathbf{s}) = \begin{cases} 1 & \text{if } \mathbf{s} = \mathbf{s}_0 \\ 1-\sigma & \text{otherwise} \end{cases} . \tag{6.29}$$

It is also denoted a fitness landscape with a "tower". In this case, all genome sequences have the same fitness, which is lower than the one of the master sequence $\mathbf{s}_0$. The corresponding landscape $F(\mathbf{s})$, defined by $W(\mathbf{s}) = e^{kF(\mathbf{s})}$ is then equally discontinuous. This landscape has no gradient pointing towards the master sequence of maximal fitness.

Relative Notation We define by x_k the fraction of the population whose genotype has a Hamming distance k from the preferred genotype,

$$x_k(t) = \frac{1}{M} \sum_{\mathbf{s}} \delta_{d_{\mathrm{H}}(\mathbf{s},\mathbf{s}_0),k} \, X_{\mathbf{s}}(t) \ . \tag{6.30}$$

The evolution equations can be formulated entirely in terms of these x_k; they correspond to the fraction of the population being k point mutations away from the master sequence.

Infinite Genome Limit We take the $N \to \infty$ limit and scale the mutation rate, see Eq. (6.3),

$$\mu = u/N \ , \tag{6.31}$$

for point mutations such that the average number of mutations

$$u = N\mu$$

occurring at every step remains finite.

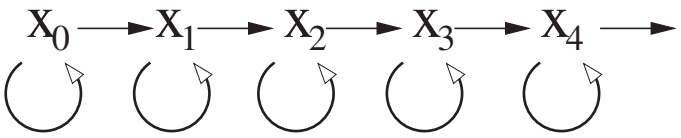

Fig. 6.4 The linear chain model for the tower landscape, Eq. (6.29), with k denoting the number of point mutations necessary to reach the optimal genome. The population fraction $x_{k+1}(t+1)$ is only influenced by the value of x_k and its own value at time t

The Absence of Back Mutations We consider starting from the optimal genome $\mathbf{s}_0$ and consider the effect of mutations. Any successful mutation increases the distance k from the optimal genome $\mathbf{s}_0$. Assuming $u \ll 1$ in Eq. (6.31) implies that

- Multiple mutations do not appear, and that
- One can neglect back mutations that reduce the value of k, since they have a relative probability proportional to

$$\frac{k}{N-k} \ll 1 \; .$$

The Linear Chain Model The model so defined consequently has the structure of a linear chain. $k = 0$ being the starting point of the chain.

We have two parameters: u, which measures the mutation rate and σ, which measures the strength of the selection. Remembering that the fitness $W(\mathbf{s})$ is proportional to the number of offspring, see Eq. (6.29), we then find

$$x_0(t+1) = \frac{1}{\langle W \rangle}\Big[x_0(t)\,(1-u)\Big] \; , \tag{6.32}$$

$$x_1(t+1) = \frac{1}{\langle W \rangle}\Big[u x_0(t) + (1-u)\,(1-\sigma)\,x_1(t)\Big] \; ; \tag{6.33}$$

$$x_k(t+1) = \frac{1}{\langle W \rangle}\Big[u x_{k-1}(t) + (1-u) x_k(t)\Big](1-\sigma) \; , \qquad k > 1, \tag{6.34}$$

where $\langle W \rangle$ is the average fitness. These equations describe a linear chain model as illustrated in Fig. 6.4. The population of individuals with the optimal genome x_0 constantly loses members due to mutations. But it also has a higher number of offspring than all other populations due to its larger fitness.

Stationary Solution The average fitness of the population is given by

$$\langle W \rangle = x_0 + (1-\sigma)(1-x_0) = 1 - \sigma(1-x_0) \; . \tag{6.35}$$

We look for the stationary distribution $\{x_k^*\}$. The equation for x_0^* does not involve the x_k^* with $k > 0$:

$$x_0^* = \frac{x_0^*(1-u)}{1-\sigma(1-x_0^*)}, \qquad 1-\sigma(1-x_0^*) = 1-u \; .$$

The solution is

$$x_0^* = \begin{cases} 1 - u/\sigma & \text{if } u < \sigma \\ 0 & \text{if } u \geq \sigma \end{cases} , \tag{6.36}$$

due to the normalization condition $x_0^* \leq 1$. For $u > \sigma$ the model becomes ill defined. The stationary solutions for the x_k^* are for $k = 1$

$$x_1^* = \frac{u}{1 - \sigma(1 - x_0^*) - (1 - u)(1 - \sigma)} \, x_0^* ,$$

which follows directly from Eqs. (6.33) and (6.35), and for $k > 1$

$$x_k^* = \frac{(1 - \sigma)u}{1 - \sigma(1 - x_0^*) - (1 - u)(1 - \sigma)} \, x_{k-1}^* , \tag{6.37}$$

which follows from Eqs. (6.34) and (6.35).

Phase Transition and the Order Parameter We can thus distinguish two regimes determined by the magnitude of the mutation rate $\mu = u/N$ relative to the fitness parameter σ, with

$$u = \sigma$$

being the transition point. In physics language the epistatic interaction corresponds to many-body interactions and the occurrence of a phase transition in the sharp peak model is due to the many-body interactions which were absent in the smooth fitness landscape model considered in Sect. 6.3.2.

The Adaptive Regime and Quasispecies In the regime of small mutation rates, $u < \sigma$, one has $x_0^* > 0$ and in fact the whole population lies a finite distance away from the preferred genotype. To see why, we note that

$$\sigma(1 - x_0^*) = \sigma(1 - 1 + u/\sigma) = u$$

and take a look at Eq. (6.37):

$$\frac{(1 - \sigma)u}{1 - u - (1 - u)(1 - \sigma)} = \left(\frac{1 - \sigma}{1 - u}\right)\left(\frac{u}{\sigma}\right) \leq 1, \qquad \text{for } u < \sigma .$$

The x_k^* therefore form a geometric series,

$$x_k^* \sim \left(\frac{1 - \sigma}{1 - u}\frac{u}{\sigma}\right)^k ,$$

which is summable when $u < \sigma$. In this adaptive regime the population forms what Manfred Eigen denoted a "quasispecies", see Fig. 6.5.

Quasispecies. A quasispecies is a population of genetically close but not identical individuals.

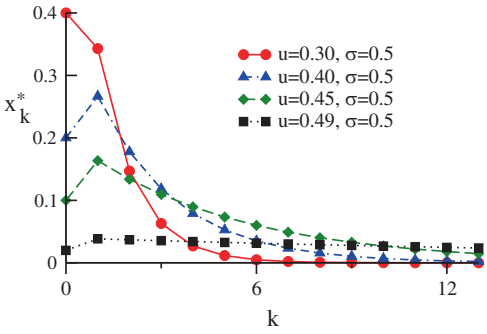

Fig. 6.5 Quasispecies formation within the sharp peak fitness landscape, Eq. (6.29). The stationary population densities x_k^*, see Eq. (6.37), are peaked around the genome with maximal fitness, $k = 0$. The population tends to spread out in genome space when the overall mutation rate u approaches the critical point $u \to \sigma$

A quasispecies does not contain a dominant genome, in contrast to a species. The distinction between a species and a quasispecies can be formulated rigorously in the limit of large populations sizes $M \to \infty$. For a quasispecies all $X_{\mathbf{s}}$ are intensive, for a species there is at least one genome $\mathbf{s}_0$ for which $X_{\mathbf{s}_0}$ is extensive, viz it scales $\propto M$.

The Wandering Regime and The Error Threshold In the regime of a large mutation rate, $u > \sigma$, we have $x_k^* = 0$, $\forall k$. In this case, a closer look at the finite genome situation shows that the population is distributed in an essentially uniform way over the whole genotype space. The infinite genome limit therefore becomes inconsistent, since the whole population lies an infinite number of mutations away from the preferred genotype. In this *wandering regime* the effects of finite population size are prominent.

Error Catastrophe. The transition from the adaptive (quasispecies) regime to the wandering regime is denoted the "error threshold" or "error catastrophe".

The notion of error catastrophe is a quite generic feature of quasispecies theory, independent of the exact nature of the fitness landscape containing epistatic interactions. A quasispecies can no longer adapt, once its mutation rate becomes too large. In the real world the error catastrophe implies extinction.

6.4 Finite Populations and Stochastic Escape

Punctuated Equilibrium Evolution is not a steady process, there are regimes of rapid increase of the fitness and phases of relative stasis. This kind of overall dynamical behavior is denoted the "punctuated equilibrium".

In this context, adaptation can result either from local optimization of the fitness of a single species or via coevolutionary avalanches, as discussed in Chap. **??**.

The Neutral Regime. The stage where evolution is essentially driven by random mutations is called the neutral (or wandering) regime.

The quasispecies model is inconsistent in the neutral regime. In fact, the population spreads out in genome space in the neutral regime and the infinite population limit is no longer reachable. In this situation, the fluctuations of the reproductive process in a finite population have to be taken into account.

Deterministic Versus Stochastic Evolution Evolution is driven by stochastic processes, since mutations are random events. Nevertheless, randomness averages out and the evolution process becomes deterministic in the thermodynamic limit, as discussed in Sect. 6.3, when the number M of individuals diverges, $M \to \infty$.

Evolutionary processes in populations with a finite number of individuals differ from deterministic evolution quantitatively and sometimes also qualitatively, the later being our focus of interest here.

Stochastic Escape. Random mutations in a finite population might lead to a decrease in the fitness and to a loss of the local maximum in the fitness landscape with a resulting dispersion of the quasispecies.

We have given a general account of the theory of stochastic escape in Chap. **??**. Here we will discuss in some detail under which circumstances this phenomenon is important in evolutionary processes of small populations.

6.4.1 Strong Selective Pressure and Adaptive Climbing

Adaptive Walks We consider a coarse-grained description of population dynamics for finite populations. We assume that

(a) The population is finite,
(b) The selective pressure is very strong, and
(c) The mutation rate is small.

It follows from (b) that one can represent the population by a single point in genome space; the genomes of all individuals are taken to be equal. The evolutionary dynamics is then the following:

(A) At each time step, only one genome element of some individual in the population mutates.
(B) If, because of this mutation, one obtains a genotype with higher fitness, the new genotype spreads rapidly throughout the entire population, which then moves altogether to the new position in genome space.

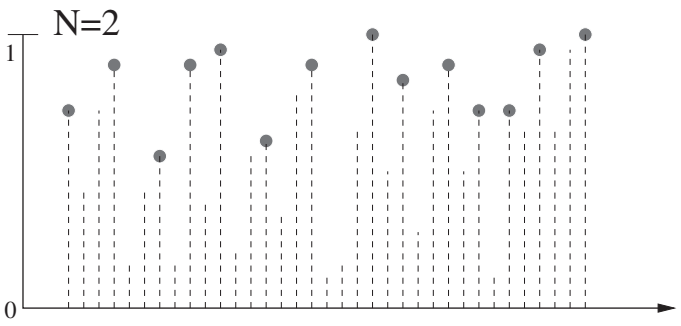

Fig. 6.6 Local fitness optima in a one-dimensional random fitness distribution; the number of neighbors is two. This simplified picture does not corresponds directly to the $N = 2$ random energy model, for which there are just $2^2 = 4$ states in genome space. It shows, however, that random distributions may exhibit an enormous number of local optima (*filled circles*), which are characterized by lower fitness values both on the *left-hand side* as well as on the *right-hand side*

(C) If the fitness of the new genotype is lower, the mutation is rejected and the population remains at the old position.

Physicists would call this type of dynamics a Monte Carlo process at zero temperature. As is well known, this algorithm does not lead to a global optimum, but to a "typical" local optimum. Step **(C)** holds only for the infinite population limit. We will relax this condition further below.

The Random Energy Model It is thus important to investigate the statistical properties of the local optima, which depend on the properties of the fitness landscape. A suitable approach is to assume a random distribution of the fitness.

The Random Energy Model. The fitness landscape $F(\mathbf{s})$ is uniformly distributed between 0 and 1.

The random energy model is illustrated in Fig. 6.6. It captures, as we will see further below two ingredients expected for real-world fitness landscapes, namely a large number of local fitness optima close to the global fitness maximum.

Local Optima in the Random Energy Model Let us denote by N the number of genome elements. The probability that a point with fitness $F(\mathbf{s})$ is a local optimum is simply given by

$$F^N = F^N(\mathbf{s}) \ ,$$

since we have to impose that the N nearest neighbors

$$(s_1, \ldots, -s_i, \ldots, s_N), \qquad (i = 1, \ldots, N), \qquad \mathbf{s} = (s_1, \ldots, s_N) \ ,$$

of the point have fitness less than F. The probability that a point in genome space is a local optimum is given by

$$\mathrm{P}\left\{\text{local optimum}\right\} = \int_0^1 F^N \mathrm{d}F = \frac{1}{N+1} \ , \tag{6.38}$$

since the fitness F is equally distributed in $[0,1]$. There are therefore many local optima, namely $2^N/(N+1)$. A schematic picture of the large number of local optima in a random distribution is given in Fig. 6.6.

Average Fitness at a Local Optimum The typical fitness of a local optimum is

$$F_{typ} = \frac{1}{1/(N+1)} \int_0^1 F\, F^N \mathrm{d}F = \frac{N+1}{N+2} = \frac{1+1/N}{1+2/N} \ \approx \ 1 - 1/N \ , \tag{6.39}$$

viz very close the global optimum of 1, when the genome length N is large. At every successful step of mutation and selection the distance from the top is divided, on average, by a factor of 2.

Successful Mutations We now consider the adaptation process. Any mutation results in a randomly distributed fitness of the offspring. A mutation is successful whenever the fitness of the offspring is bigger than the fitness of its parent. The typical fitness attained after ℓ successful steps is then of the order of

$$1 - \frac{1}{2^{\ell+1}} \ ,$$

when starting ($l = 0$) from an average initial fitness of $1/2$. It follows that the typical number of successful mutations after which an optimum is attained is

$$F_{typ} = 1 - 1/N = 1 - \frac{1}{2^{\ell_{typ}+1}}, \qquad \ell_{typ} + 1 = \frac{\log N}{\log 2} \ , \tag{6.40}$$

i.e. it is relatively small.

The Time Needed for One Successful Mutation Even though the number of successful mutations Eq. (6.40) needed to arrive at the local optimum is small, the time to climb to the local peak can be very long; see Fig. 6.7 for an illustration of the climbing process.

We define by

$$t_F = \sum_n n\, P_n, \qquad n: \ \text{number of generations}$$

the average number of generations necessary for the population with fitness F to achieve one successful mutation, with P_n being the probability that it takes exactly n generations. We obtain:

$$t_F = 1\,(1-F) + 2\,(1-F)F + 3\,(1-F)F^2 + 4\,(1-F)F^3 + \cdots$$

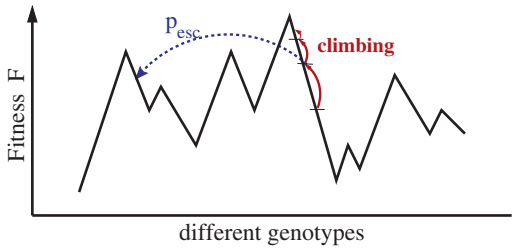

Fig. 6.7 Climbing process and stochastic escape. The higher the fitness, the more difficult it becomes to climb further. With an escape probability $p_{\rm esc}$ the population jumps somewhere else and escapes a local optimum

$$= \frac{1-F}{F}\sum_{n=0}^{\infty} n\,F^n = \frac{1-F}{F}\left(F\frac{\partial}{\partial F}\sum_{n=0}^{\infty}F^n\right) = (1-F)\frac{\partial}{\partial F}\frac{1}{1-F}$$
$$= \frac{1}{1-F}\ . \tag{6.41}$$

The average number of generations necessary to further increase the fitness by a successful mutation diverges close to the global optimum $F \to 1$.

The Total Climbing Time Every successful mutation decreases the distance $1-F$ to the top by $1/2$ and therefore increases the factor $1/(1-F)$ on the average by 2. The typical number $\ell_{\rm typ}$, see Eq. (6.40), of successful mutations needed to arrive at a local optimum determines, via Eq. (6.41), the expected total number of generations $T_{\rm opt}$ to arrive at the local optimum. It is therefore on the average

$$T_{\rm opt} = 1\,t_F + 2\,t_F + 2^2\,t_F + \cdots + 2^{\ell_{\rm typ}}t_F$$
$$= t_F\,\frac{1-2^{\ell_{\rm typ}+1}}{1-2} \;\approx\; t_F\,2^{\ell_{\rm typ}+1} = t_F\,e^{(\ell_{\rm typ}+1)\log 2}$$
$$\approx t_F\,e^{\log N} = \frac{N}{1-F} \;\approx\; 2\,N\ , \tag{6.42}$$

where we have used Eq. (6.40) and $F \approx 1/2$ for a typical starting fitness. The time needed to climb to a local maximum in the random fitness landscape is therefore proportional to the length of the genome.

6.4.2 Adaptive Climbing Versus Stochastic Escape

In Sect. 6.4.1 the average properties of adaptive climbing have been evaluated. We now take the fluctuations in the reproductive process into account and

compare the typical time scales for a stochastic escape with those for adaptive climbing.

Escape Probability When a favorable mutation appears it spreads instantaneously into the whole population, under the condition of strong selection limit, as assumed in our model.

We consider a population situated at a local optimum or very close to a local optimum, having hence a fitness close to one. Mutations occur with probability u per individuum and lead with probability F to descendents having a lower fitness. The probability p_{esc} for stochastic escape is then

$$p_{\mathrm{esc}} \;\propto\; (Fu)^M \;\approx\; u^M, \qquad F \approx 1 \;,$$

where M is the number of individuals in the population and $u \in [0,1]$ the mutation rate per genome, per individuum and per generation, compare Eq. (6.31). The escape can only happen when an adverse mutation occurs in every member of the population within the same generation (see also Fig. 6.7). If a single individual does not mutate it retains its higher fitness of the present local optimum and all other mutations are discarded within the model, assuming a strong selective pressure.

Stochastic Escape and Stasis We now consider a population climbing towards a local optimum. The probability that the fitness of a given individual increases is $(1-F)u$. It needs to mutate with a probability u and to achieve a higher fitness, when mutating, with probability $1-F$. We denote by

$$a = 1 - (1-F)u$$

the probability that the fitness of an individual does not increase with respect to the current fitness F of the population. The probability q_{bet} that at least one better genotype is found is then given by

$$q_{\mathrm{bet}} = 1 - a^M \;.$$

Considering a population close to a local optimum, a situation typical for real-world ecosystems, we can then distinguish between two evolutionary regimes:

- Adaptive Walk: The escape probability p_{esc} is much smaller than the probability to increase the fitness, $q_{\mathrm{bet}} \gg p_{\mathrm{esc}}$. The population continuously increases its fitness via small mutations.
- The Wandering Regime: Close to a local optimum the adaptive dynamics slows down and the probability of stochastic escape p_{esc} becomes comparable to that of an adaptive process, $p_{\mathrm{esc}} \approx q_{\mathrm{bet}}$. The population wanders around in genome space, starting a new adaptive walk after every successful escape.

Typical Escape Fitness During the adaptive walk regime the fitness F increases steadily, until it reaches a certain typical fitness F_{esc} for which the

probability of stochastic escape becomes substantial, i.e. when $p_{\rm esc} \approx q_{\rm bet}$ and

$$p_{\rm esc} = u^M = 1 - [1 - (1 - F_{\rm esc})u]^M = q_{\rm bet}$$

holds. As $(1 - F_{\rm esc})$ is then small we can expand the above expression in $(1 - F_{\rm esc})$,

$$u^M \;\approx\; 1 - [1 - M(1 - F_{\rm esc})u] = M(1 - F_{\rm esc})u \;,$$

obtaining

$$1 - F_{\rm esc} = u^{M-1}/M \;. \tag{6.43}$$

The fitness $F_{\rm esc}$ necessary for the stochastic escape to become relevant is exponentially close to the global optimum $F = 1$ for large populations M.

The Relevance of Stochastic Escape The stochastic escape occurs when a local optimum is reached, or when we are close to a local optimum. We may estimate the importance of the escape process relative to that of the adaptive walk by comparing the typical fitness $F_{\rm typ}$ of a local optimum achieved by a typical climbing process with the typical fitness $F_{\rm esc}$ needed for the escape process to become important:

$$F_{\rm typ} = 1 - \frac{1}{N} \;\equiv\; F_{\rm esc} = 1 - \frac{u^{M-1}}{M}, \qquad \frac{1}{N} = \frac{u^{M-1}}{M} \;,$$

where we have used Eq. (6.39) for $F_{\rm typ}$. The last expression is now independent of the details of the fitness landscape, containing only the measurable parameters N, M and u. This condition can be fulfilled only when the number of individuals M is much smaller than the genome length N, as $u < 1$. The phenomenon of stochastic escape occurs only for very small populations.

6.5 Prebiotic Evolution

Prebiotic evolution deals with the question of the origin of life. Is it possible to define chemical autocatalytic networks in the primordial soup having properties akin to those of the metabolistic reaction networks going on continuously in every living cell?

The precursor of living organisms, which are defined by a boundary, the cell membrane, between body and environment, could be, from this perspective, chemical regulation networks in a primordial soup of macromolecules evolving into the protein regulation networks of living cells once enclosed by a membrane.

6.5.1 Quasispecies Theory

We have discussed in Sect. 6.3.3 the concept of a quasispecies as a community of closely related genomes in which no single genotype is dominant. This situation is presumably also typical for prebiotic evolutionary processes. In this context Manfred Eigen formulated the quasispecies theory for a system of information carrying macromolecules through a set of equations for chemical kinetics,

$$\frac{\mathrm{d}}{\mathrm{d}t}x_i = \dot{x}_i = W_{ii}x_i + \sum_{j\neq i} W_{ij}x_j - x_i\phi(t) \ , \tag{6.44}$$

where the x_i denote the concentrations of $i = 1 \ldots N$ molecules. W_{ii} is the (autocatalytic) self-replication rate and the off-diagonal terms $W_{i,j}$ $(i \neq j)$ are the respective mutation rates.

We will use Eq. (6.44) in order to generalize the concept of a quasispecies to a collection of macromolecules. In Sect. 6.5.2 we will then consider a somewhat more realistic model for a network of chemical reactions, which might possibly be regarded as the precursor of the protein regulation network of a living cell.

Mass Conservation We can choose the flux $-x\phi(t)$ in Eigen's equations (6.44) for prebiotic evolution such that the total concentration C, viz the total mass

$$C = \sum_i x_i$$

is conserved for long times. Summing Eq. (6.44) over i we obtain

$$\dot{C} = \sum_{ij} W_{ij}x_j - C\,\phi, \qquad \phi(t) = \sum_{ij} W_{ij}x_j(t) \ , \tag{6.45}$$

for a suitable choice for the field $\phi(t)$, leading to

$$\dot{C} = \phi\,(1 - C), \qquad \frac{\mathrm{d}}{\mathrm{d}t}(C-1) = -\phi\,(C-1) \ . \tag{6.46}$$

The total concentration $C(t)$ will therefore approach 1 for $t \to \infty$ for $\phi > 0$, which we assume to be the case here, implying total mass conservation. In this case the autocatalytic rates W_{ii} dominate with respect to the transmolecular mutation rates W_{ij} $(i \neq j)$.

Quasispecies We can write the evolution equation (6.44) in matrix form

$$\frac{\mathrm{d}}{\mathrm{d}t}\boldsymbol{x}(t) = (W - 1\phi)\,\boldsymbol{x}(t), \qquad \boldsymbol{x} = \begin{pmatrix} x_1 \\ x_1 \\ \cdots \\ x_N \end{pmatrix} \ , \tag{6.47}$$

where W is the matrix $\{W_{ij}\}$. We assume here for simplicity a symmetric mutation matrix $W_{ij} = W_{ji}$. The solutions of the linear differential equa-

tion (6.47) are then given in terms of the eigenvectors $\vec{e}_\lambda$ of W:

$$W\boldsymbol{e}_\lambda = \lambda\,\boldsymbol{e}_\lambda, \qquad \boldsymbol{x}(t) = \sum_\lambda a_\lambda(t)\boldsymbol{e}_\lambda, \qquad \dot{a}_\lambda = [\lambda - \phi(t)]\,a_\lambda \ .$$

The eigenvector $\boldsymbol{e}_{\lambda_{\max}}$ with the largest eigenvalue $\lambda_{\max}$ will dominate for $t \to \infty$, due to the overall mass conservation equation (6.46). The flux will adapt to the largest eigenvalue,

$$\lim_{t\to\infty} \Big(\lambda_{\max} - \phi(t)\Big) \ \to \ 0 \ ,$$

leading to the stationary condition $\dot{x}_i = 0$ for the evolution equation (6.47) in the long time limit.

If W is diagonal (no mutations) a single macromolecule will remain in the primordial soup for $t \to \infty$. For small but finite mutation rates W_{ij} $(i \neq j)$, a quasispecies will emerge, made up of different but closely related macromolecules.

The Error Catastrophe The mass conservation equation (6.46) cannot be retained when the mutation rates become too big, viz when the eigenvectors $\vec{e}_\lambda$ become extensive. In this case the flux $\phi(t)$ diverges, see Eq. (6.45), and the quasispecies model consequently becomes inconsistent. This is the telltale sign of the error catastrophe.

The quasispecies model Eq. (6.44) is equivalent to the random energy model for microevolution studied in Sect. 6.4, with the autocatalytic rates W_{ii} corresponding to the fitness of the x_i, which corresponds to the states in genome space. The analysis carried through in Sect. 6.3.3 for the occurrence of an error threshold is therefore also valid for Eigen's prebiotic evolutionary equations.

6.5.2 Hypercycles and Autocatalytic Networks

RNA World The macromolecular evolution equations (6.44) do not contain terms describing the catalysis of molecule i by molecule j. This process is, however, important both for the prebiotic evolution, as stressed by Manfred Eigen, as well as for the protein reaction network in living cells.

> Hypercycles. Two or more molecules may form a stable catalytic (hyper) cycle when the respective intermolecular catalytic rates are large enough to mutually support their respective synthesis.

An illustration of some hypercycles is given in Figs. 6.8 and 6.9. The most likely chemical candidate for the constituent molecules is RNA, functioning both enzymatically and as a precursor of the genetic material. One speaks also of an "RNA world".

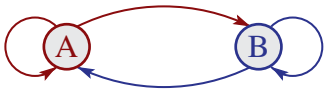

Fig. 6.8 The simplest hypercycle. A and B are self-replicating molecules. A acts as a catalyst for B, i.e. the replication rate of B increases with the concentration of A. Likewise the presence of B favors the replication of A

Reaction Networks We disregard mutations in the following and consider the catalytic reaction equations

$$\dot{x}_i = x_i \left(\lambda_i + \sum_j \kappa_{ij} x_j - \phi \right) \tag{6.48}$$

$$\phi = \sum_k x_k \left(\lambda_k + \sum_j \kappa_{kj} x_j \right) \ , \tag{6.49}$$

where x_i are the respective concentrations, λ_i the autocatalytic growth rates and κ_{ij} the transmolecular catalytic rates. The field ϕ has been chosen, Eq. (6.49), such that the total concentration $C = \sum_i x_i$ remains constant

$$\dot{C} = \sum_i \dot{x}_i = \sum_i x_i \left(\lambda_i + \sum_j \kappa_{ij} x_j \right) - C\,\phi = (1 - C)\,\phi \ \to \ 0$$

for $C \to 1$.

The Homogeneous Network We consider the case of homogeneous "interactions" $\kappa_{i \neq j}$ and uniformly distributed autocatalytic growth rates:

$$\kappa_{i \neq j} = \kappa, \qquad \kappa_{ii} = 0, \qquad \lambda_i = \alpha\, i \ , \tag{6.50}$$

compare Fig. 6.10, leading to

$$\dot{x}_i = x_i \left(\lambda_i + \kappa \sum_{j \neq i} x_j - \phi \right) = x_i \left(\lambda_i + \kappa - \kappa x_i - \phi \right) \ , \tag{6.51}$$

where we have used $\sum_i x_i = 1$. The fixed points x_i^* of Eq. (6.51) are

$$x_i^* = \begin{cases} (\lambda_i + \kappa - \phi)/\kappa \\ 0 \end{cases} \qquad \lambda_i = \alpha, 2\alpha, \ldots, N\alpha \ , \tag{6.52}$$

where the non-zero solution is valid for $\lambda_i - \kappa - \phi > 0$. The flux ϕ in Eq. (6.52) needs to obey Eq. (6.49), as the self-consistency condition.

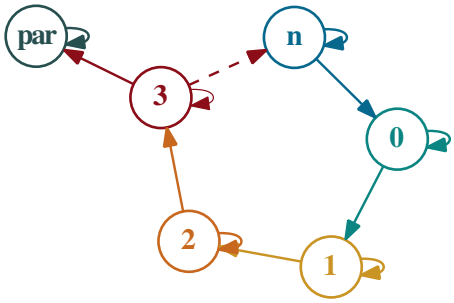

Fig. 6.9 A hypercycle of order n consists of n cyclically coupled self-replicating molecules, with each molecule providing catalytic support for the subsequent molecule in the cycle. Parasitic self-replicating molecules "par" receive catalytic support from the hypercycle without contributing to it

The Stationary Solution The case of homogeneous interactions, Eq. (6.50), can be solved analytically. Dynamically, the $x_i(t)$ with the largest growth rates λ_i will dominate and obtain a non-zero steady-state concentration x_i^*. We may therefore assume that there exists an $N^* \in [1, N]$ such that

$$x_i^* = \begin{cases} (\lambda_i + \kappa - \phi)/\kappa & N^* \le i \le N \\ 0 & 1 \le i < N^* \end{cases} , \tag{6.53}$$

compare Fig. 6.10, where N^* and ϕ are determined by the normalization condition

$$\begin{aligned} 1 = \sum_{i=N^*}^{N} x_i^* &= \sum_{i=N^*}^{N} \frac{\lambda_i + \kappa - \phi}{\kappa} = \frac{\alpha}{\kappa} \sum_{i=N^*}^{N} i + \left[\frac{\kappa - \phi}{\kappa}\right] \left(N + 1 - N^*\right) \\ &= \frac{\alpha}{2\kappa} \left[N(N+1) - N^*(N^*-1)\right] + \left[\frac{\kappa - \phi}{\kappa}\right] \left(N + 1 - N^*\right) \end{aligned} \tag{6.54}$$

and by the condition that $x_i^* = 0$ for $i = N^* - 1$:

$$0 = \frac{\lambda_{N^*-1} + \kappa - \phi}{\kappa} = \frac{\alpha(N^* - 1)}{\kappa} + \frac{\kappa - \phi}{\kappa} . \tag{6.55}$$

We eliminate $(\kappa - \phi)/\kappa$ from Eqs. (6.54) and (6.55) for large N, N^*:

$$\begin{aligned} \frac{2\kappa}{\alpha} &\simeq N^2 - (N^*)^2 - 2N^* \left(N - N^*\right) \\ &= N^2 - 2N^*N + (N^*)^2 = (N - N^*)^2 . \end{aligned}$$

The number of surviving species $N - N^*$ is therefore

$$N - N^* \simeq \sqrt{\frac{2\kappa}{\alpha}} , \tag{6.56}$$

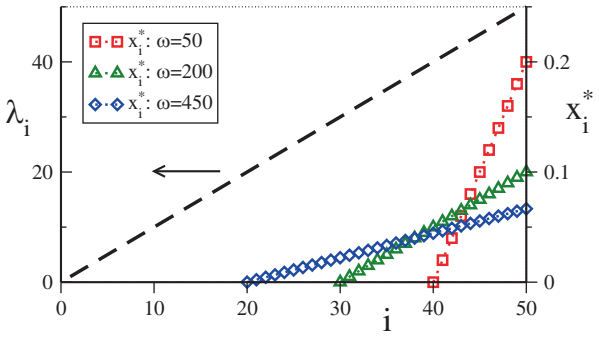

Fig. 6.10 The autocatalytic growth rates λ_i (*left axis*), as in Eq. (6.50) with $\alpha = 1$, and the stationary solution x_i^* (*right axis*) of the concentrations, Eq. (6.53), constituting a prebiotic quasispecies, for various mean intercatalytic rates $\kappa \equiv \omega$. The *horizontal axis* $i = 1, 2, \ldots, 50$ denotes the respective molecules

which is non-zero for a finite and positive inter-molecular catalytic rate κ. A hypercycle of mutually supporting species (or molecules) has formed.

The Origin of Life The scientific discussions concerning the origin of life are highly controversial to date and it is speculative whether hypercycles have anything to do with it. Hypercycles describe closed systems of chemical reactions which have to come to a stillstand eventually, as a consequence of the continuous energy dissipation. In fact, a tellpoint sign of biological activities is the buildup of local structures, resulting in a local reduction of entropy, possible only at the expense of an overall increase of the environmental entropy. Life, as we understand it today, is possible only as an open system driven by a constant flux of energy.

Nevertheless it is interesting to point out that Eq. (6.56) implies a clear division between molecules $i = N^*, \ldots, N$ which can be considered to form a primordial "life form" separated by molecules $i = 1, \ldots, N^* - 1$ belonging to the "environment", since the concentrations of the latter are reduced to zero. This clear separation between participating and non-participating substances is a result of the non-linearity of the reaction equation (6.48). The linear evolution equation (6.44) would, on the other hand, result in a continuous density distribution, as illustrated in Fig. 6.5 for the case of the sharp peak fitness landscape. One could then conclude that life is possible only via cooperation, resulting from non-linear evolution equations.

6.6 Macroecology and Species Competition

In Macroecology one disregards both the genetic basis of evolutionary processes as well as specific species interdependencies. One is interested in formulating general principles and models describing the overall properties of large communities of species.

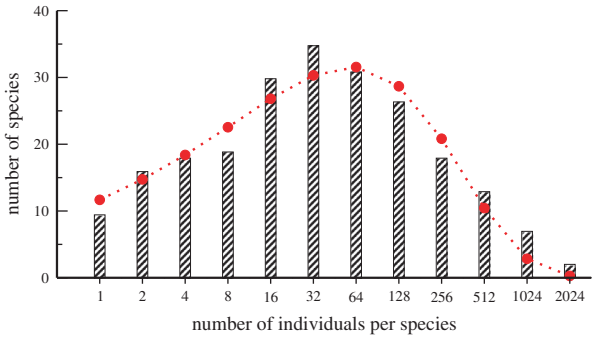

Fig. 6.11 The abundance of trees in a 50 ha patch of tropical rainforest in Panama (Bars, adapted from Volkov et al. (2003)), in comparison (*filled circles*) with results from neutral theory, Eq. (6.60)

Neutral Theory A central topic in ecology is to explain the distribution of species abundances, as illustrated in Fig. 6.11 for the count of trees in a tropical rainforest plot. Most species have around 32 individual trees in the patch examined, there are fewer species having more/less individuals. Similar species abundance distributions are found in virtually all ecosystems studied.

The neutral theory, as formulated originally by Hubbel, proposes that very simple and universal principles lead to the species abundance distribution observed in nature and that, in particular, differences between the distinct species involved are irrelevant (hence the term "neutral theory"). The two central principles of the neutral theory, which can be implemented mathematically in various fashions, involve the deterministic competition between species on one side and the influence of stochastic events, which lead to random fluctuations in populations sizes, on the other side.

Stochastic Walk Through Population Space We consider a species performing a random walk in population space. The master equation for the probability $p_x(t)$ to observe x individuals of the species at time t is given by simple birth and death events, proportional to b_x and d_x respectively:

$$\dot{p}_x(t) = b_{x-1}\, p_{x-1}(t) + d_{x+1}\, p_{x+1}(t) - [b_x + d_x]\, p_x(t)\ . \tag{6.57}$$

The birth and death processes are per capita and contain both intensive terms $\propto (x)^0$ and extensive terms $\propto (x)^1$,

$$b_x = \tilde{b}_0 + \tilde{b}_1 x, \qquad d_x = \tilde{d}_0 + \tilde{d}_1 x\ , \tag{6.58}$$

where the intensive contributions $\tilde{b}_0$ ($\tilde{d}_0$) model the cumulative effects of immigration (emigration) and speciation (extinction) respectively.

Fokker–Planck Equation of Macroecology We may treat, for large populations, x as a continuous variable and approximate the difference

$d_{x+1}p_{x+1} - d_x p_x$ occurring on the right hand side of Eq. (6.57) through a Taylor expansion,

$$d_{x+1}\,p_{x+1} - d_x\,p_x \;\simeq\; \frac{\partial}{\partial x}\big(d_x\,p_x\big) + \frac{1}{2}\frac{\partial^2}{\partial x^2}\big(d_x\,p_x\big) + \dots \;,$$

and analogously for the birth processes. We hence obtain for $p(x,t) = p_x(t)$ with

$$\frac{\partial p(x,t)}{\partial t} = \frac{\partial}{\partial x}\big(d_x - b_x\big)\,p(x,t) + \frac{1}{2}\frac{\partial^2}{\partial x^2}\big(d_x + b_x\big)\,p(x,t)$$

the "Fokker–Planck equation of macroecology" (compare Sect. **??**), which we rewrite as

$$\frac{\partial p(x,t)}{\partial t} = \frac{\partial}{\partial x}\left(\frac{x}{\tau} - b\right)\,p(x,t) + D\frac{\partial^2}{\partial x^2}\big(x\,p(x,t)\big)\;, \tag{6.59}$$

with

$$\tau = \left(\tilde{d}_1 - \tilde{b}_1\right)^{-1} > 0, \qquad b = \tilde{b}_0 - \tilde{d}_0 = 2\tilde{b}_0 > 0, \qquad D = \frac{\tilde{b}_1 + \tilde{d}_1}{2}\;,$$

when restricting to the case $b_0 = -d_0 > 0$.

Competition vs. Diffusion The parameters introduced in (6.59) have then the following interpretations:

- D induces fluctuations of the order $\sqrt{x}$ in the populations x.
- b corresponds to the net influx, caused either by immigration or by speciation.
- τ is a measure of the strength of interaction effects in the ecosystem and of the time scale the system needs to react to perturbations.

In order to understand the effect of τ in more detail we consider the case $b = 0 = D$. The Fokker–Planck equation (6.59) then reduces to

$$\tau\dot{p} = p + xp', \qquad p \;\sim\; \mathrm{e}^{-t/T}x^{\beta}, \qquad -\tau/T = 1 + \beta\;.$$

The distribution is normalizable for $\beta < -1$ and hence $T > 0$. The ecosystem would slowly die out on time scale T, as consequence of the competition between the species, when not counterbalanced by the diffusion D and the external source b. Note, that $\tau > 0$ implies that $\tilde{d}_1 > \tilde{b}_1$ and that there are, on the average, more deaths that births for all species, independent of the size of their population.

Solution of the Fokker–Planck Equation The steady-state solution $\dot{p}(x,t) = 0$ of (6.59) is the "Gamma distribution"

$$p_0(x) = A\,x^{b/D-1}\,e^{-x/(D\tau)}\;, \tag{6.60}$$

where A is a normalization constant. For a verification note that the population current J, defined via the continuity equation $\dot{p}(x,t) = -\nabla J$, vanishes in the steady state, $J = 0$, and hence

$$0 = \left(\frac{x}{\tau} - b\right) p_0(x) + D\frac{\partial}{\partial x}\big(x p_0(x)\big) = \left(\frac{x}{\tau} + D - b\right) p_0(x) + Dx p_0'(x) \ ,$$

which is satisfied by the solution (6.60). The steady state solution (6.60) fits the real-world data quite well, see Fig. 6.11.

Microscopic Models The master equation (6.57) of macroevolution can be derived from specific microscopic models. Any microscopic update rule will then lead to explicit expressions for the dependence of the birth and death rates b_x and d_x on population size (and other microscopic parameters), which will in general be similar, but not identical, to the relations postulated in Eq. (6.58).

As an example of a microscopic model consider a population of S species containing an overall number of N individuals. The relation between the species is defined pairwise. For every pair one of the species dominates with probability ρ the other species, with probability $1-\rho$ their relation is neutral.

At every time step a pair of individuals belonging to two different species S and S' is considered.

- *Stochastic process*: With probability μ the number of individuals in species S (S') is increased by one (decreased by one).
- *Competition*: With probability $1-\mu$ the number of individuals in the dominating (inferior) species is increased by one (reduced by one). No update is performed if their relation is neutral.

These update rules are conserving in the total number of individuals. The steady-state distribution obtained by this model is similar to the one obtained for the neutral model defined by the birth and death rates (6.58), shown in Fig. 6.11, with the functional form of their respective species abundancy distributions differing in details.

6.7 Coevolution and Game Theory

The average number of offsprings, viz the fitness, is the single relevant reward function within Darwinian evolution. There is hence a direct connection between evolutionary processes, ecology and game theory, which deals with interacting agents trying to maximize a single reward function denoted utility. Several types of games may be considered in this context, namely games of interacting species giving rise to coevolutionary phenomena or games of interacting members of the same species, pursuing distinct behavioral strategies.

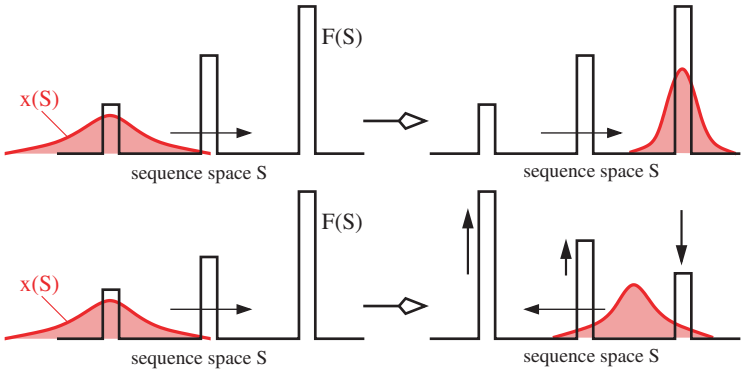

Fig. 6.12 *Top*: An evolutionary process of a single (quasi) species in a fixed fitness landscape (fixed ecosystem), here with tower-like structures, see Eq. (6.29) will in general lead to a reorganization of the density of individuals $x(\mathbf{S})$ in genome space. *Bottom*: A coevolutionary process might be regarded as changing the respective fitness landscapes $F(\mathbf{S})$

Coevolution The larger part of this chapter has been devoted to the discussion of the evolution of a single species, in Sect. 6.5.2, the stabilization of an "ecosystem" made of a hypercycle of mutually supporting species and in Sect. 6.6 general macroecological principles. We now go back to the level of a few interdependent species.

> Coevolution. When two or more species form an interdependent ecosystem the evolutionary progress of part of the ecosystem will generally induce coevolutionary changes also in the other species.

One can view the coevolutionary process also as a change in the respective fitness landscapes, see Fig. 6.12. A prominent example of phenomena arising from coevolution is the "red queen" phenomenon.

> The Red Queen Phenomenon. When two or more species are interdependent then "It takes all the running, to stay in place" (from Lewis Carroll's children's book "Through the Looking Glass").

A well-known example of the red queen phenomenon is the "arms race" between predator and prey commonly observed in real-world ecosystems.

The Green World Hypothesis Plants abound in real-world ecosystems, geology and climate permitting, they are rich and green. Naively one may expect that herbivores should proliferate when food is plenty, keeping vegetation constantly down. This doesn't seem to happen in the world, as a result of coevolutionary interdependencies. Hairston, Smith and Slobodkin proposed that coevolution gives rise to a trophic cascade, where predators keep the herbivores substantially below the support level of the bioproductivity of the plants. This "green world hypothesis" arises naturally in evolutionary models, but it has been difficult to verify it in field studies.

Avalanches and Punctuated Equilibrium In Chap. **??** we discussed the Bak and Sneppen model of coevolution. It may explain the occurrence of coevolutionary avalanches within a state of punctuated equilibrium.

> Punctuated Equilibrium. Most of the time the ecosystem is in equilibrium, in the neutral phase. Due to rare stochastic processes periods of rapid coevolutionary processes are induced.

The term punctuated equilibrium was proposed by Gould and Eldredge in 1972 to describe a characteristic feature of the evolution of simple traits observed in fossil records. In contrast to the gradualistic view of evolutionary changes, these traits typically show long periods of stasis interrupted by very rapid changes.

The random events leading to an increase in genome optimization might be a rare mutation bringing one or more individuals to a different peak in the fitness landscape (microevolution) or a coevolutionary avalanche.

Strategies and Game Theory One is often interested, in contrast to the stochastic considerations discussed so far, in the evolutionary processes giving rise to very specific survival strategies. These questions can be addressed within game theory, which deals with strategically interacting agents in economics and beyond. When an animal meets another animal it has to decide, to give an example, whether confrontation, cooperation or defection is the best strategy. The basic elements of game theory are:

- Utility: Every participant, also called an agent, plays for himself, trying to maximize its own utility.
- Strategy: Every participant follows a set of rules of what to do when encountering an opponent; the strategy.
- Adaptive Games: In adaptive games the participants change their strategy in order to maximize future return. This change can be either deterministic or stochastic.
- Zero-Sum Games: When the sum of utilities is constant, you can only win what the others lose.
- Nash Equilibrium: Any strategy change by a participant leads to a reduction of his utility.

Hawks and Doves This simple evolutionary game tries to model competition in terms of expected utilities between aggressive behavior (by the "hawk") and peaceful (by the "dove") demeanor. The rules are:

Dove meets Dove	$A_{DD} = V/2$	They divide the territory
Hawk meets Dove	$A_{HD} = V$, $A_{DH} = 0$	The Hawk gets all the territory, the Dove retreats and gets nothing
Hawk meets Hawk	$A_{HH} = (V - C)/2$	They fight, get injured, and win half the territory

The expected returns, the utilities, can be cast in matrix form,

$$A = \begin{pmatrix} A_{HH} & A_{HD} \\ A_{DH} & A_{DD} \end{pmatrix} = \begin{pmatrix} \frac{1}{2}(V-C) & V \\ 0 & \frac{V}{2} \end{pmatrix} .$$

A is denoted the "payoff" matrix. The question is then, under which conditions it pays to be peaceful or aggressive.

Adaptation by Evolution The introduction of reproductive capabilities for the participants turns the hawks-and-doves game into an evolutionary game. In this context one considers the behavioral strategies to result from the expression of distinct alleles.

The average number of offspring of a player is proportional to its fitness, which in turn is assumed to be given by its expected utility,

$$\begin{aligned} \dot{x}_H &= \Big(A_{HH}x_H + A_{HD}x_D - \phi(t)\Big)\, x_H \\ \dot{x}_D &= \Big(A_{DH}x_H + A_{DD}x_D - \phi(t)\Big)\, x_D \end{aligned} , \tag{6.61}$$

where x_D and x_H are the density of doves and hawks, respectively, and where the flux

$$\phi(t) = x_H A_{HH} x_H + x_H A_{HD} x_D + x_D A_{DH} x_H + x_D A_{DD} x_D$$

ensures an overall constant population, $x_H + x_D = 1$.

The Steady State Solution We are interested in the steady-state solution of equation (6.61), with $\dot{x}_D = 0 = \dot{x}_H$. Setting

$$x_H \ = x, \qquad\qquad x_D = 1 - x \ ,$$

we find

$$\phi(t) = \frac{x^2}{2}(V-C) + Vx(1-x) + \frac{V}{2}(1-x)^2 = \frac{V}{2} - \frac{C}{2}x^2$$

and

$$\begin{aligned} \dot{x} &= \left(\frac{V-C}{2}x + V(1-x) - \phi(t)\right) x = \left(\frac{V}{2} - \frac{V}{2}x + \frac{C}{2}\left(x^2 - x\right)\right) x \\ &= \frac{C}{2}\, x \left(x^2 - \frac{C+V}{C}x + \frac{V}{C}\right) \ = \ \frac{C}{2}\, x\, (x-1)\, (x - V/C) \\ &= -\frac{\mathrm{d}}{\mathrm{d}x} V(x) \ , \end{aligned}$$

with

$$V(x) = -\frac{x^2}{4}V + \frac{x^3}{6}(V+C) - \frac{x^4}{8}C \ .$$

The steady state solution is given by

$$V'(x) = 0, \qquad x = V/C \ ,$$

apart from the trivial solution $x = 0$ (no hawks) and $x = 1$ (only hawks). For $V > C$ there will be no doves left in the population, but for $V < C$ there will be an equilibrium with $x = V/C$ hawks and $1 - V/C$ doves. A population consisting exclusively of cooperating doves ($x = 0$) is unstable against the intrusion of hawks.

The Prisoner's Dilemma The payoff matrix of the prisoner's dilemma is given by

$$A = \begin{pmatrix} R & S \\ T & P \end{pmatrix} \qquad \begin{matrix} T > R > P > S \\ 2R > S + T \end{matrix} \qquad \begin{matrix} \text{cooperator} \mathrel{\hat{=}} \text{dove} \\ \text{defector} \mathrel{\hat{=}} \text{hawk} \end{matrix} \ . \tag{6.62}$$

Here "cooperation" between the two prisoners is implied and not cooperation between a suspect and the police. The prisoners are best off if both keep silent. The standard values are

$$T = 5, \qquad R = 3, \qquad P = 1, \qquad S = 0 \ .$$

The maximal global utility NR is obtained when everybody cooperates, but in a situation where agents interact randomly, the only stable Nash equilibrium is when everybody defects, with a global utility NP:

$$\begin{aligned} \text{reward for cooperators} = R_c &= \Big[RN_c + S(N - N_c) \Big] / N \ , \\ \text{reward for defectors} = R_d &= \Big[TN_c + P(N - N_c) \Big] / N \ , \end{aligned}$$

where N_c is the number of cooperators and N the total number of agents. The difference is

$$R_c - R_d \ \sim \ (R - T)N_c + (S - P)(N - N_c) \ < \ 0 \ ,$$

as $R - T < 0$ and $S - P < 0$. The reward for cooperation is always smaller than that for defecting.

Evolutionary Games on a Lattice The adaptive dynamics of evolutionary games can change completely when the individual agents are placed on a regular lattice and when they adapt their strategies based on past observations. A possible simple rule is the following:

- At each generation (time step) every agent evaluates its own payoff when interacting with its four neighbors, as well as the payoff of its neighbors.
- The individual agent then compares his own payoff one-by-one with the payoffs obtained by his four neighbors.

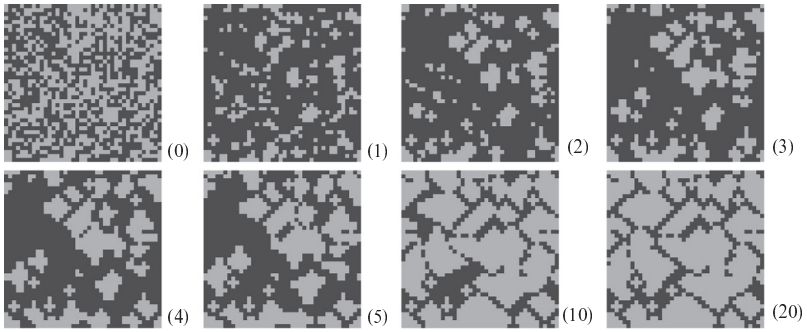

Fig. 6.13 Time series of the spatial distribution of cooperators (*gray*) and defectors (*black*) on a lattice of size $N = 40 \times 40$. The time is given by the numbers of generations in *brackets*. Initial condition: equal number of defectors and cooperators, randomly distributed. Parameters for the payoff matrix, $\{T; R; P; S\} = \{3.5; 3.0; 0.5; 0.0\}$ (From Schweitzer et al. (2002))

– The agent then switches his strategy (to cooperate or to defect) to the strategy of his neighbor if the neighbor received a higher payoff.

This simple rule can lead to complex real-space patterns of defectors intruding in a background of cooperators, see Fig. 6.13. The details depend on the value chosen for the payoff matrix.

Nash Equilibria and Coevolutionary Avalanches Coevolutionary games on a lattice eventually lead to an equilibrium state, which by definition has to be a Nash equilibrium. If such a state is perturbed from the outside, a self-critical coevolutionary avalanche may follow, in close relation to the sandpile model discussed in Chap. **??**.

Game Theory and Memory Standard game theory deals with an anonymous society of agents, with agents having no memory of previous encounters. Generalizing this standard setup it is possible to empower the agents with a memory of their own past strategies and achieved utilities. Considering additionally individualized societies, this memory may then include the names of the opponents encountered previously, and this kind of games provides the basis for studying the emergence of sophisticated survival strategies, like altruism, via evolutionary processes.

Opinion Dynamics Agents in classical game theory aim to maximize their respective utilities. Many social interactions between interacting agents however do not need explicitly the concept of rewards or utilities in order to describe interesting phenomena.

Examples of reward-free games are opinion dynamics models. In a simple model for continuous opinion dynamics $i = 1, \ldots, N$ agents have continuous opinions $x_i = x_i(t)$. When two agents interact they change their respective opinions according to

$$x_i(t+1) = \begin{cases} [x_i(t) + x_j(t)]/2 & |x_i(t) - x_j(t)| < \theta \\ x_i(t) & |x_i(t) - x_j(t)| \geq \theta \end{cases}, \tag{6.63}$$

where θ is the confidence interval. Consensus can be reached step by step only when the initial opinions are not too contrarian. For large confidence intervals θ, relative to the initial scatter of opinions, global consensus will be reached, clusters of opinions emerge on the other side for a small confidence interval.

Further Reading

A comprehensive account of the earth's biosphere can be found in Smil (2002); a review article on the statistical approach to Darwinian evolution in Peliti (1997) and Drossel (2001). Further general textbooks on evolution, game-theory and hypercycles are Nowak (2006), Kimura (1983), Eigen (1971), Eigen and Schuster (1979), and Schuster (2001). For a review article on evolution and speciation see Drossel (2001), for an assessment of punctuated equilibrium Gould and Eldredge (2000).

The relation between life and self-organization is further discussed by Kauffman (1993), a review of the prebiotic RNA world can be found in Orgel (1998) and critical discussions of alternative scenarios for the origin of life in Orgel (1998) and Pereto (2005).

The original formulation of the fundamental theorem of natural selection was given by Fisher (1930). For a review of the neutral theory of macroevolution you may consult Alonso et al. (2006), for the formulation of the neutral theory discussed in Sect. 6.6 Volkov et al. (2003).

For the reader interested in coevolutionary games we refer to Ebel and Bornholdt (2002); for an interesting application of game theory to world politics as an evolving complex system see Cederman (1997) and for a field study on the green world hypothesis Terborgh et al. (2006).

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