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D I P L O M A R B E I T

Evolution of Phenotype Determination

Stochastic simulations of adaptation in time-varying environments

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Contents

List of Figures

List of Tables

Preface

This diploma thesis is the result of my last eight month of work together with Claus Rueffler at the Faculty of Mathematics of the University of Vienna. I contributed to a project initiated by him and Peter Abrams from the University of Toronto. While the analytical work was done by the two of them, my task was to develop and run numerical simulations to investigate several questions posed on the model. As it is necessary to see my results in the context of the whole project, the analytical results will be given in the introduction and the simulation-results will always be presented in the light of the analytical findings.

The whole project including some of my results will be presented in two manuscripts, "The Evolution of Randomized Strategies" (Rueffler, Svardal, and Abrams, in prep.*b*) and "The Evolution of phenotype determination in a Lottery Model" (Rueffler, Svardal, and Abrams, in prep.*a*).

I want to thank my supervising tutor Claus Rueffler as well as my supervising Professors Joachim Hermisson from the University of Vienna and Harald Markum from the Vienna University of Technology for their support.

Abstract

In temporally or spatially varying environments species often evolve different phenotypes in response to this variety of niches. Such a phenotypic polymorphism can either be due to different genotypes present in the population or due to a mechanism that allows a single genotype to produce different phenotypes. Both possibilities were subject to many studies but rarely have they been combined in a single analysis. Here, an eco-evolutionary model for the evolution in time-varying environments is studied by means of individual based simulations. This model has been introduced by Rueffler et al. (in prep.*a*, in prep.*b*) and initially been studied by using the adaptive dynamics approximation. In this model different mechanisms of phenotype determination are allowed to evolve simultaneously. The alternatives are a canalised genotype-phenotype map, phenotypic plasticity where an environmental cue is used to produce an adapted phenotype and bet-hedging (randomisation). The simulations presented here relax the assumptions of the adaptive dynamics approximation and show that the analytical results remain valid under more general conditions. Additionally, cases are treated in which the model is not analytically tractable. Contrary to preceding work we find that genetic polymorphisms can outcompete a bet-hedging strategy if a certain mutational correlation is introduced and mutational step sizes are limited. Comparing canalised genotypes and phenotypic plasticity we find that, depending on model parameters, different outcomes – ranging from polymorphisms of canalised phenotypes to evolutionary cycles – are possible. When canalised genotypes, phenotypic plasticity and bet-hedging are all allowed for simultaneously, we find that the strategy approached by evolution is a mixture of phenotypic plasticity and bet-hedging.

Abstract

1. Introduction

The genotype of an organism is the exact configuration of the DNA in its cells that was passed to the organism by its parents at the organism's conception. The phenotype on the other hand is the totality of physical and behavioural characteristics of the organism, e.g., size of body parts, eye colour, etc. A lot of work has been done in order to determine how and to which degree the genotype maps to the phenotype (starting from Mendel's (1866) pea flower experiments down to modern genetics). For the vast majority of traits the genotype-phenotype maps remains, however, unknown. It is sure that the genotype has the greatest influence on the development of the phenotype, but other factors, mainly the environment an individual experiences and - for instance - the condition of an offspring's mother, can play an important role.

It is the phenotype that determines how successful an individual is in the environment it experiences; it is the phenotype selection acts upon. In different environments, different phenotypes will be more or less successful. Thus, the fitness of an individual clearly depends on its phenotype and the environmental conditions it experiences. In many situations it is not possible for a single phenotype to be optimally adapted to different environmental conditions simultaneously. As a result phenotypic trade-offs should be prevalent; organisms that are well adapted to one environmental condition might perform poorly in another environmental condition.

The environment is seldom constant over space and time; an individual can therefore not always be optimally adapted if its mobility is limited. In such a case evolution often leads to phenotypic polymorphisms, meaning that individuals with different phenotypes exist within a population, some better adapted to one environment, some better to others. Such polymorphisms can be generated by different mechanisms. In the following we give a short overview over possible mechanisms.

The most straightforward case is a direct or "canalised" genotype-phenotype mapping where the phenotype of an individual is exactly determined by its genotype. In this case a genetic polymorphism must be at the origin of a phenotypic polymorphism, meaning that there are different genotypes present in the population. Another possibility is bet-hedging, also called randomisation, where an individual's phenotype is randomly chosen from a set of available strategies during its development. In this case a single genotype can lead to different phenotypes. Thirdly, the phenotype could be influenced by environmental cues. Some sensory machine could use hints about future environmental conditions during early development, to equip an individual with an adapted phenotype.

In this case a phenotypic polymorphism can be produced by one single genotype as a response to different cues received by different individuals.

The first of these three mechanisms should not need any further description. There the phenotype depends exclusively on the genotype. The second and the third mechanism, though, bet-hedging and plasticity, are worth some discussion.

What is Bet-Hedging?

With "the phenotype is randomly chosen from a set of available strategies" is meant that in addition to the genetic influence there is some "rolling of dices" in the developmental process, so that different phenotypes can be observed among genetically identical clones. These "dices" can for instance be very locally changing environmental conditions or the concentration of some molecule in a cell. To distinguish this strategy from phenotypic plasticity, no information about future environmental conditions should be incorporated in this mechanism. There is evidence that such bet-hedging strategies exist in nature: Delayed seed germination would be an example. For many different plants it can be observed, that genetically identical seeds exposed to the same environmental conditions will germinate in different years. A certain percentage germinates in the first year after their creation, while the rest stays in the ground to shoot in one of the following years. This mechanism intuitively seems advantageous as it guarantees that at least some seeds encounter optimal environmental conditions after germination. A theoretical model to explain this phenomenon quantitatively is given by Cohen (1966) and Bulmer (1984). Other examples of bet-hedging strategies in nature include the insectal diapause (Hopper, 1999) and characteristics of bacteria (Dubnau and Losick, 2006; Avery, 2006) such as persister cells of *E. coli*. The underlying mechanisms for such a randomisation device are not well known up to this day. Feedback loops between genes (if gene A is expressed is inhibits the expression of gene B and vice versa, such that the gene activated first "wins") could be a potential randomisation mechanism (Smits et al., 2006). Note that in the current study a genotype can give raise to two different phenotypes.

If one considers an isolated individual, one could think that such a randomisation device is not really useful. The fact that an individuals genotype can produce two different phenotypes does not increase the individuals own performance. An individual itself always only develops one phenotype, no matter how many alternative phenotypes it could potentially have developed. The great utility of such a strategy can only be seen if one considers a line of generations on which selection acts upon. If in each year at least some individuals of a given genotype have a good chance of survival this will be beneficial to the long term growth rate of this genotype. More exactly, it can be shown (Lewontin and Cohen, 1969; Bulmer, 1980) that it is the geometric mean of the year to year growth rate (which will be called fitness) that is maximised by selection. This can be achieved by minimising the fitness variance, and that is what bet-hedging does. A parent that produces offspring with different phenotypes ensures that at least

part of the offspring has a good chance to be well adapted to the environment they experience. A complete review on the advantages of a bet-hedging strategy is given in Seger and Brockmann (1987).

What is Phenotypic Plasticity?

The third mechanism of phenotype determination we want to speak about, phenotypic plasticity, is a very general concept. In the broadest possible definition, phenotypic plasticity is the ability of an organism with a given genotype to change its phenotype in response to a changing environment. This would for instance include the production of either sun- or shade-leafs of many kinds of plants, depending on the degree of sunshine they encounter in their habitat, or the adaptation of behavioural strategies of animals. The interested reader can find more about the concept of phenotypic plasticity in DeWitt and Scheiner (2004).

In this work we use a restricted form of this concept: We focus on organisms that use environmental information during their early development in order to produce a phenotype possibly well adapted to the environment they will experience when selection takes place. To say it in other words, we only allow for adaptations of the phenotype at one point of an individuals development. One could think of the development in a seed, egg or in the mother's uterus.

In the following we will investigate a temporally changing environment. In such a case the environmental condition at the organisms conception does not necessarily need to be a good indicator for the environmental condition the organism will encounter when selection takes place. In our model the phenotypes are produced according to an environmental cue and the correlation between this cue and the actual environment when selection takes place is an external parameter depending on the predictability of the environment. Intuitively, such a mechanism will help to adapt to changing environments if the information is reliable, while it could be dangerous to "listen" to such a cue when it is not very reliable. If such a mechanism is employed, it produces a polymorphism in the sense that a single genotype can produce different phenotypes. Note, however, that if all individuals of a genetically uniform population that are born in the same year receive the same cue (which will be the case in the model presented here) they will all develop the same phenotype. But in different years different cues can be received and different phenotypes can be produced. We could thus speak of a "temporal polymorphism". Similar to the bet-hedging strategy, the plastic mechanism in our model allows for two possible phenotypes a single genotype can produce.

Now that we know that there are different possibilities how a phenotypic polymorphism can be achieved, we can ask the question: In an ecological setting that favours the development of different phenotypes, which of the three mechanisms mentioned above will be favoured by selection? The answer to this

question strongly depends on the ecological system which is regarded. There are situations, in which only one of these mechanisms is possible. In many biological settings, however, more than one outcome of the evolutionary process is possible. Then one and the same ecological situation can lead to the development of alternative ways of phenotype determination. The alternatives can be the mechanisms explained above, a genetic polymorphism, a bet-hedging strategy or phenotypic plasticity as well as other mechanisms or any combination of those.

In Rueffler, Svardal, and Abrams (in prep.*b*) we allow for the simultaneous evolution of genetic polymorphisms and bet-hedging and attempt to identify conditions that favour one mechanism over the other. This work was strongly motivated by preliminary studies by Leimar (2005). In Rueffler, Svardal, and Abrams (in prep.*a*) a model is presented that allows for the evolution of bet-hedging, plasticity and genetic polymorphisms in a common framework. For both analyses we use the mathematical framework of adaptive dynamics, which is also called evolutionary invasion analysis. Adaptive dynamics is briefly outlined in section 1.1. Section 1.2 describes the model we use and section 1.3 presents the analytical results obtained.

My task, which will be presented in the following chapters, was to develop a function, capable of performing individual based simulations to check and complement the analytical results of the investigations named above. Chapter 2 deals with the technical issues of programming, while chapter 3 presents the simulation results, decomposed into three steps:

1. bet-hedging versus genetic polymorphism (section 3.1)

2. plasticity versus genetic polymorphism (section 3.2)

3. the complete model where bet-hedging, plasticity and genetic polymorphisms are allowed for (section 3.3)

The objective of the simulations presented here is twofold:

- Adaptive dynamics makes a number of restrictive assumptions. These assumptions shall be relaxed in the simulations to test the robustness of the results based on the adaptive dynamics approximation.

- The simulations will treat situations that are not analytically tractable. More precisely, we will investigate cases with
  - more than one resident genotype simultaneously present in the population.
  - correlations between the mutational steps in the trait values of the alternative phenotypes.

1.1. Adaptive Dynamics

The mathematical framework of adaptive dynamics (A.D.), which is used for the theoretical analysis of the present problem, was developed by Dieckmann and Law (1996), Metz et al. (1996), and Geritz et al. (1997, 1998). Adaptive dynamics links population dynamics to evolutionary dynamics and incorporates the fundamental idea of frequency dependent selection from game theory.

The main assumption of the method is that the (slow) evolutionary and the (fast) population dynamic time scales can be clearly separated. For this it is necessary that mutations are sufficiently infrequent that the population has always reached a population dynamical equilibrium before a new mutant occurs. Thus, there exists not more than one mutant sub-population within the population at the same time. "Being at an population dynamic equilibrium" means for a resident population that its long term growth rate is zero. Fitness of a rare invading mutant is derived from population dynamics; the fitness of a small mutant sub-population is its long term average exponential growth rate in the environment made up by the resident population (Metz et al., 1992). To this quantity we refer as invasion fitness. The invasion fitness of a rare mutant with phenotypic trait vector x' in an environment made up by the resident with trait vector x, will be noted $w(x', x)$. As the resident is at a population dynamical equilibrium, we have $w(x, x) = 0$.

If a mutant with $w(x', x) > 0$ occurs, it has a positive probability to spread. If $w(x', x) < 0$ it will die out. Of course, even if a mutant has a positive fitness, as long as it is rare it could still accidentally die out, due to bad luck. The fundamental idea of A.D. is that once a mutant has left the region of accidental extinction, its fate can be inferred from its initial growth rate (fitness) when rare. This assumption is proven to hold under some conditions, especially that mutations have sufficiently small phenotypic effects (see Geritz et al. (2002)). In the following we will assume that these assumptions are fulfilled. If the mutant has a positive long term growth rate in the environment made up by the resident ($w(x', x) > 0$) but the resident, on the other hand, has a negative growth rate in an environment made up by the mutant ($w(x, x') < 0$), then the mutant phenotype will (except for random extinction due to bad luck) spread and eventually replace the resident which is doomed to extinction. If, however, $w(x', x) > 0$ and $w(x, x') > 0$ then the mutant can spread, but once it replaces x as resident and the former resident population becomes rare, the former resident is protected by a positive growth rate. This means that the two strategies x and x' coexist in a protected polymorphism as each of them has a positive growth rate when rare.

To sum up, the evolutionary dynamics can be seen as a succession of mutations that either

- fail to invade ($w(x', x) < 0$)
- go to fixation ($w(x', x) > 0$ and $w(x, x') < 0$)

- produce a protected polymorphism ($w(x', x) > 0$ and $w(x, x') > 0$)

It should be noted that A.D. neglects the possibility of accidental extinction of the resident population. This can be justified by the assumption that the resident population is large.

If mutations have sufficiently small phenotypic effect, then the direction of the local fitness gradient determines which mutants can invade. The components of the gradient are given by

$$S_i(x) = \left. \frac{\partial w(x', x)}{\partial x'_i} \right|_{x'=x} \tag{1.1}$$

The index i stands here for the ith component of the multi-dimensional trait vector. If $S_i(x) > 0$, mutants with $x'_i > x_i$ can invade the resident with phenotype x, whereas if $S_i(x) < 0$, this is only possible for mutants with $x'_i < x_i$ (where $x'_j = x_j$ for $j \neq i$).

If the traits evolve in small but discrete steps, the change over time is well approximated by the so-called canonical equation

$$\frac{d}{dt} x_i = m(x) \sum_j C_{ij}(x) S_j(x), \tag{1.2}$$

where the C_{ij} are the elements of the covariance matrix of the mutational increments in the different traits and $m(x)$ is a positive factor related to the mutation rate (Dieckmann and Law, 1996).

The canonical equation predicts the direction and speed of evolutionary change. If there is no covariance and if the variance in the traits is equal, this will be the direction of the local fitness gradient. The population evolves until it reaches the neighbourhood of an equilibrium point where the local fitness gradient is zero ($S_i(x) = 0$). The trait configuration corresponding to such an equilibrium point has been named "evolutionary singular strategy" by Metz and co-workers. It is important to note that such a singular strategy can be approached from very different initial conditions, depending on the shape of the actual fitness landscape. But, as we will see in the following, it need not to be approached at all. This depends on the the type of singular point.

The type of such an equilibrium point can be determined by evaluating the Jacobian matrix of the selection gradient and the Hessian matrix of invasion fitness at the singular point. A comprehensive analysis of this is given in Leimar (in press), while Geritz et al. (1997, 1998) treat in detail the case of one-dimensional traits. The current work shall be restricted to description of the possible types of equilibria. It is fundamental to clarify that there are two different questions to be posed when considering a singular point: The first is whether this point will be approached by the evolutionary process, when starting with an evolutionary strategy nearby. Leimar (in press) shows that this is always the case for any positive definite covariance matrix C in the canonical equation when the Jacobian

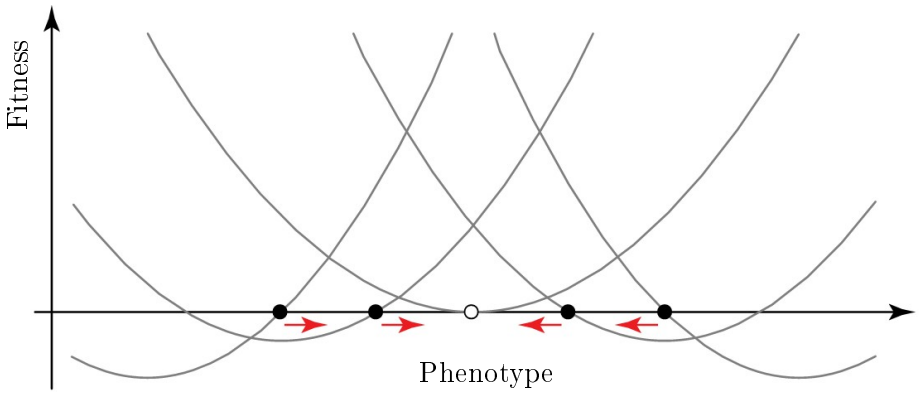


Figure 1.1.: A convergence stable fitness minimum or evolutionary branching point. Nearby phenotypes experience selection towards this singular point. But once the resident population has reached this point it turns into a fitness minimum. Source: Rueffler et al. (2006)

matrix of the selection gradient is negative definite. If this criterion is met, the singular strategy is called "strongly convergence stable". The second question is whether such an equilibrium point is a local fitness minimum or maximum. If it is a maximum, the point is locally uninvadable, meaning that, once a population at this point is established, all nearby mutants have negative fitness. This is the classical ESS-condition of evolutionary game theory. If such a point is additionally strongly convergence stable, it is called a continuously stable strategy (CSS). Leimar (in press, 2005) shows that a sufficient condition for a local maximum is the negative definiteness of the Hessian matrix of the invasion fitness at this point. On the other hand, if the Hessian is positive definite or indefinite, this point will be a minimum or a saddle point and (at least some) nearby mutants will be able to invade. In the case of a minimum the selection at such a point is called disruptive selection. If such a point is additionally convergence stable, evolutionary branching may occur. Evolutionary branching means that the population evolves towards this point and once the point is attained it splits up into two sub-population, which evolve in different directions of the phenotype space.

At first sight the separation of these two questions might seem counter-intuitive. It may seem strange that a point can be approached by evolution (convergence stability), but once the population has arrived there the point turns out to be a fitness minimum that can be invaded by nearby strategies. To understand this, one should keep in mind that the fitness landscape is not something statical, but is generated by the current resident. It is possible that a population follows the direction of the fitness gradient in trait space, while it is simultaneously "overtaken" by a fitness minimum "coming from behind". This situation is illustrated in Figure 1.1. Table 1.1 gives an overview of possible configuration of singular points.

Table 1.1.: Classification of singular points
Here we consider the one-dimensional case: If a singular point is both convergence stable and a local maximum, then it is called continuously stable strategy (CSS). A maximum, which is not convergence stable is called Garden of Eden, as a population will not converge to it from nearby and a population that has left this point by chance will evolve away from it. A minimum which is convergence stable is called branching point (BP), as all nearby populations evolve to it and then split into two distinct subpopulations. A minimum which is not convergence stable is called evolutionary repellor, because the population does not evolve towards such a point and a population initially there evolves away from it.

Extremum	Convergence stable	
	+	-
-	BP	Repellor
+	CSS	Garden of Eden

Table 1.2.: Assumptions of the A.D. approximations

Basic assumptions of Adaptive Dynamics:
- Resident is at dynamical equilibrium when mutant occurs (mutations are rare)
- Mutation in small but discrete steps (for this one needs a quantitative trait)
- Population size large

A.D. is an approximation of the evolutionary dynamcis valid in the limit of rare mutations. The assumptions made by the A.D. framework are summed up in Table 1.2. Such an approximation is necessary in order to obtain a mathematically tractable model. It is of course necessary to explore the validity of the approximation under more realistic conditions and this is one purpose of the individual-based simulations presented in the following chapters.

1.2. The Model

To investigate the questions posed in the beginning of this chapter, we use a population dynamical model first presented by Chesson and Warner (1981). They used it to address the question of coexistence of different phenotypes. We study in this so-called lottery model the evolution in temporary varying environments. We allow for the evolution of genetic polymorphisms, randomisation (bet-hedging) and phenotypic plasticity. Leimar (2005) used this model to investigate the question of the relative likelihood of the development of randomisation versus evolutionary branching (which leads to a genetic polymorphism).

The lottery model by Chesson and Warner is characterised by the following features: The population is censored in discrete time-steps. To an interval between two census-points we refer as season, year or time-step. The habitat consists of a fixed number of K patches, each of which can be occupied by one

individual. An individual that has managed to settle in a patch stays there until it dies. Now the following events occur each season:

1. **Environmental condition:** Each season is dominated by a certain environmental condition. Here we allow for two different environmental conditions. Condition 1 occurs with probability p and condition 2 with probability $1 - p$.

2. **Uniform number of offspring:** Each individual produces an equal, very large amount of clonal offspring (if no mutations occur, the offspring's genotype is an exact copy of the parental genotype).

3. **Selection among offspring:** Only a fraction of the offspring survives. The survival of an offspring depends on the current environmental condition and the offspring's phenotype, which is determined by its parent's genotype. One could thus combine the last two steps and say: The number of surviving offspring an individual has in a certain environment is a function of its genotype.

4. **Patches are emptied:** A fraction d of the adult individuals occupying patches dies. Mortality affects all individuals equally. Their genotype thus only plays a role in determining the number of surviving offspring they will produce.

5. **Empty patches filled by offspring:** The total number of surviving offspring of step 3 form an offspring pool. The patches emptied in step 4 are refilled by offspring. We assume that the number of surviving offspring is always large compared to the population size, so all empty patches will be filled each season. The offspring is highly dispersive and therefore the offspring pool is well mixed. The offspring genotypes fill the empty patches randomly with a probability equal to their frequency in the offspring pool. This is equivalent to an allocation of empty patches on a first-come first-serve basis. Offspring that does not manage to settle in a patch (the majority of it) die. After one season the population size thus always equals the patch number, K.

6. **Mutations occur:** With a certain, small, probability the freshly settled individuals carry a mutated genotype, differing from the one of their parent.

Figure 1.2 gives a schematic representation of this population dynamics. Note that selection only acts on newborn individuals. We will combine steps 2 and 3 by only considering the number of offspring surviving the selection stage per parent. More frequent genotypes among the offspring then have a higher chance to settle in the empty patches. For settled individuals death occurs randomly with equal probability each year.

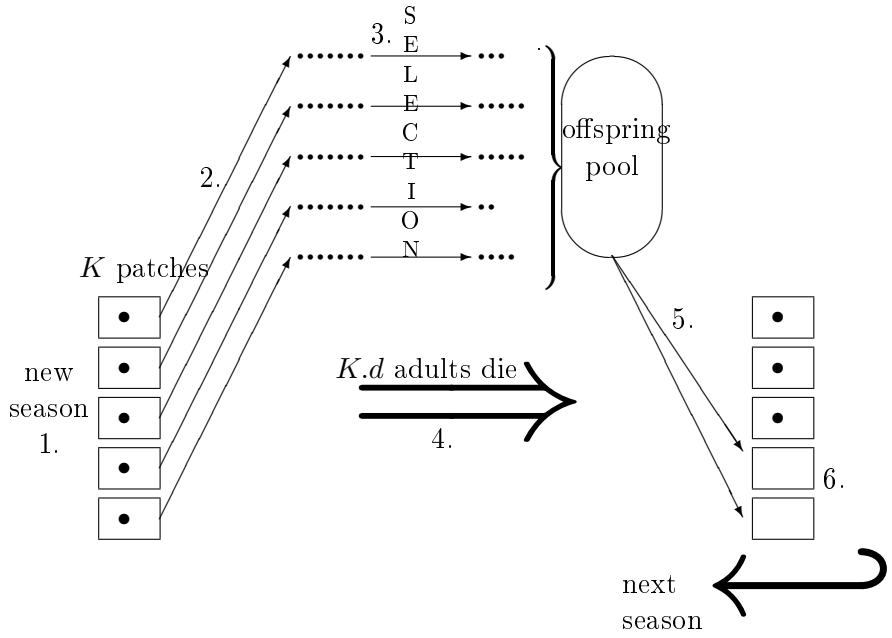


Figure 1.2.: Schematic representation of the population dynamics. The numbers correspond to the steps of the enumeration in the text.

One might criticise in our model that the offspring's phenotypes are determined by its parent's genotype rather than by its own. This has mostly technical reasons, but we think that this assumption can be justified. First, given that the organisms in our model reproduce clonally, a difference only exists in the rare case of mutation. And if a mutation occurs, this assumption just delays the impact of a mutation by one generation. Second, there are examples in nature where the phenotype on which selection acts is determined by an offspring's parent. Think for instance of seeds or eggs. Their phenotypes are determined by the mother's genotype and it is plausible that there is selection in this stage of the offspring's development (e.g. resistance of seeds or eggs against external conditions).

Biological examples of species with a life cycle similar to the one in our model include territorial tropical reef fish or sessile marine invertebrates such as corals or shells (see Butler and Keough (1981)). These species produce a large amount of larvae that are highly dispersive and once the larvae completed their development they settle in discrete patches which they occupy until they die.

Note that our model features a fixed number of patches K. For the analytical treatment, however, this patch number is assumed to be so large that all calculations can be done with frequencies instead of absolute values. The simulations will use explicit patch numbers.

In the following superscripts correspond to different genotypes in the population. The population dynamics of the ith genotype (genotypic trait value noted by z^i) living in a community of n different genotypes is described by

$$N^i(t+1) = (1-d)N^i(t) + Kd\frac{\beta(z^i)N^i(t)}{\sum_{j=1}^{n}\beta(z^j)N^j(t)}, \tag{1.3}$$

where $\beta(z^i)$ is the number of offspring produced by a single individual of genotype i that reach the settling stage during (t,t+1] and $N^i(t)$ is the number of individuals carrying genotype i. The term Kd is the number of empty patches, whereas the fraction in equation 1.3 gives the probability that an empty patch will be occupied by an individual of genotype i.

As mentioned above, each season one out of two different environmental conditions occurs. In each environmental condition the offspring will need a special adaptation in order to have a good chance to survive. Having a phenotype well adapted to one condition will mean a suboptimal adaptation to the other condition. As environmental conditions one could think of wet and dry years. Or - in the light of the example of reef fish or corals - the two environmental conditions could reflect the presence of two different kinds of predators, preying on the larvae. If one predator is dominant, one phenotype guarantees the best survival probability, while another phenotype is needed to cope best with the other predator. The phenotype of an individual is a single (scalar) quantitative trait, noted $z \in [0,1]$. The function $\alpha_i(z)$ gives the number of offspring that reach settling stage for an individual with offspring of phenotype z under environmental

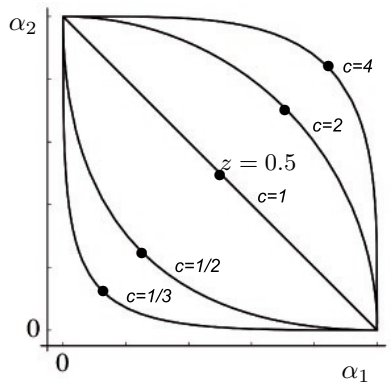


Figure 1.3.: Trade-off curve for different values of the curvature parameter c. The curvature parameter c determines the shape of the trade-off curve. The abscissa represents the number of offspring of a phenotype lying on the curve under environmental condition 1, while the ordinate gives the number of offspring under environmental condition 2. The left upper end of each curve corresponds to $z = 1$ and the right bottom end to $z = 0$.

condition i, $i \in \{1, 2\}$ .The trait value z parametrises a one-dimensional curve in the two-dimensional (α_1, α_2)-plane (see Figure 1.3). These trade-off curves for the two environmental conditions have the following form:

$$\alpha(z) = \begin{cases} \alpha_1(z) = \alpha_{1max}(1-z)^{\frac{1}{c}} & \text{in env. condition 1} \\ \alpha_2(z) = \alpha_{2max}(z)^{\frac{1}{c}} & \text{in env. condition 2,} \end{cases} \tag{1.4}$$

where the factors α_{imax} correspond to the maximum number of surviving offspring possible in environment i per individual and time-step. Specialists for environmental condition 1 are characterised by $z = 0$, whereas an individual with phenotype $z = 1$ is specialised in environment 2. The trade-off parameter determines how different phenotypes perform in the two different environments. If the trade-off curve is concave (weak trade-off, trade-off parameter $c > 1$) intermediate phenotypes have relatively good survival probabilities in both environments. In this case a generalist with $z = 0.5$ would be the only best strategy and polymorphisms do not occur. Under a strong trade-off ($c < 1$) the generalist is comparatively unfit, meaning that he performs quite poorly in both environments.

In the following we want to use this model to answer different questions. First, we investigate situations in which – starting with a population at the generalist genotype $z = 0.5$ – a genetic polymorphism evolves. At the same time we implement a mechanism that allows for bet-hedging and investigate whether bet-hedging evolves when starting from a non-bet-hedging genotype.

This task is strongly motivated by the work of Leimar (2005) who compared the relative likelihood of evolutionary branching (leading to a genetic polymorphism) and the development of a bet-hedging strategy for a similar model. For this part we try to stay as close as possible to Leimar's notations to allow a direct comparison. This notation will be described in section 1.2.1. Second, we want to generalise this approach and incorporate a mechanism that allows for bet-hedging, phenotypic plasticity, and a fixed genotype-phenotype relation. For this model we will determine which conditions favour the development of one of these possibilities and whether mixtures of the strategies appear. For the incorporation of these mechanisms, we introduce a convenient notation which is explained in section 1.2.2.

1.2.1. Framework for the comparison with Leimar's work

To incorporate bet-hedging into the model we need three trait values to characterise one genotype: (z_1, z_2, q). Here z_1 and z_2 correspond to the two different phenotypic trait values an individual with genotype (z_1, z_2, q) can have. The third dimension of the genotype space, q, corresponds to the probability that an offspring of an individual with this genotype has the phenotype z_1, whereas with probability $(1-q)$ the phenotype of an offspring is z_2. Hence, in our model a genotype is in principle capable of producing two different phenotypes. A genotype where either $z_1 = z_2$ or $q = 0$ or 1 corresponds to a non-bet-hedging strategy. In the first case the two phenotypes are simply identical, whereas in the other cases only one phenotype is produced. In such a situation a phenotypic polymorphism could only be achieved by a genetic polymorphism.

So how many offspring that reach settling stage does an individual with a certain genotype produce? The ith randomizing genotype produces larvae with phenotypes z_1^i and z_2^i in proportions q^i and $(1-q^i)$. The number of surviving offspring an individual has in an environment $j \in \{1,2\}$, $\beta_j(z^i)$, as used in equation 1.3, is given by

$$\beta_j^i = q^i \alpha_j(z_1^i) + (1-q^i)\alpha_j(z_2^i). \tag{1.5}$$

The function $\alpha_j(z_k)$, $k \in \{1,2\}$, is the number of surviving offspring if an individual would only produce offspring of phenotype z_k in environment j and is given in equation 1.4. Equation 1.5 corresponds to a simple weighting of the offspring numbers over the different phenotypes produced.

Example:

Although the analytical treatment uses infinite population size and individual frequencies, we will work with explicit numbers here for clearness. Let us consider an individual of type i with a genotype $(z_1^i, z_2^i, q^i) = (0.8, 0.2, 0.3)$. The trade-off parameter c shall be fixed at 0.6 and $\alpha_{1max} = \alpha_{2max} = 100$. In a year with environmental condition 1 the individual produces $\beta^i = 0.3\alpha_1(0.8) + 0.7\alpha_1(0.2)$

offspring. So there is 30% offspring having phenotype $z_1^i = 0.8$ and 70% offspring having phenotype $z_2^i = 0.2$. The survival functions $\alpha_1(0.8)$ and $\alpha_1(0.2)$ give 6.8 and 68.9, respectively. Finally, $\beta^i = 50.3$, which means that the individual produces 50 offspring that reach the settling stage. Whether each of them finds an empty patch to settle is of course another question. In this example with environmental condition 1, only about 2 offspring with the suboptimal phenotype $z_1^i = 0.8$ survive, whereas about 48 of the survivors have phenotype 2. In a year with environmental condition 2 it would be other way round with about 21 survivors with phenotype 1 and only 5 having phenotype 2. One can see that the bet-hedging mechanism allows an individual to have a reasonable amount of offspring under each environmental condition.

1.2.2. Framework for the complete model

Similar to the preceding section we denote individuals with different phenotypic trait values produced by the same genotype with subscripts: the ith phenotype of the jth genotype is denoted as z_i^j. Here we want to introduce a system allowing for all three strategies, bet-hedging, phenotypic plasticity and unique genotype-phenotype mapping as ways of phenotype determination. We will also be interested in mixtures of those mechanisms and possible evolutionary transitions from one mode of phenotype determination to another. A conceptual framework that allows for such transitions was presented by Leimar et al. (2006) and Leimar (2008). They introduce a switching device that processes environmental cues as well as genetic information to determine discrete phenotypes as output. We want to use a similar device capable of incorporating environmental cues and some form of randomisation in the development of two alternative phenotypes per genotype. The randomisation - bet-hedging - shall be integrated in the system via the input of some developmental noise, n, drawn from a normal distribution $N(0, 1)$. Simons and Johnston (1997) point out that a developmental instability such as this developmental noise in our model is a possible mechanism for bet-hedging to evolve. The switching device we consider for this study is shown in Figure 1.4. We assume that the environmental cue can take two values e_1 and e_2. The cue correctly predicts the future environment - in which the newborns struggle for survival - with probability r (reliability) and predicts it incorrectly with a probability $1 - r$. The switching device processes the input according to $y = a \cdot e_i + (1-a)n$, leading to the development of phenotype 1 if $y < t$ and to phenotype 2 if $y > t$. The effect of the switch can evolve though changes in the weighting factor a and the threshold value t. Hence, a genotype is characterised by (z_1, z_2, t, a). If $a = 1$ and $e_1 < t < e_2$, then the phenotype is determined according to the environmental cue, corresponding to pure phenotypic plasticity. If $a = 0$, the phenotype is determined by the effect of the developmental noise, n, with the threshold value t determining the frequency of the two possible phenotypes. In this case the system (z_1, z_2, t) is equivalent the one described in 1.2.1, where $t = \infty$ corresponds to $q = 1$ and $t = -\infty$ to

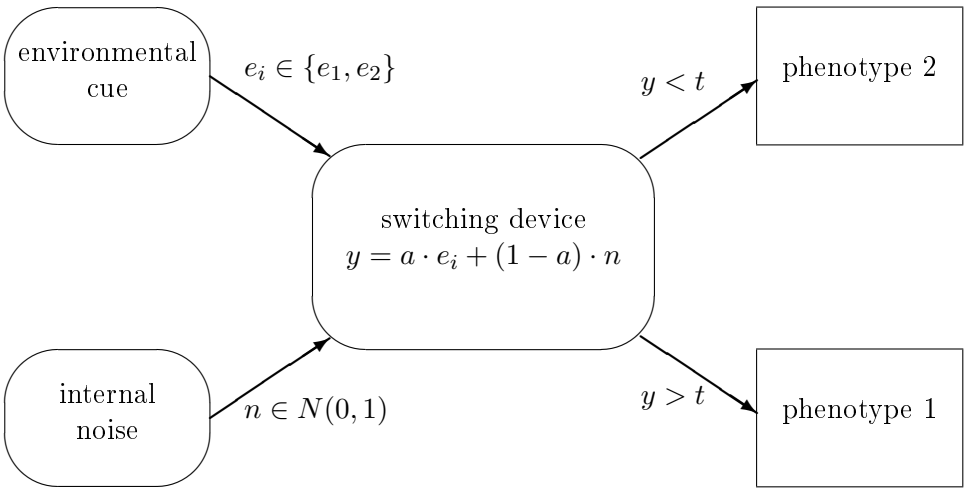


Figure 1.4.: Developmental switch with environmental cue and developmental noise as input. The switching device processes environmental and internal cues as a weighted sum to determine one of two alternative phenotypes as output. Phenotype 1 is produced when the weighted sum is larger than the threshold t and phenotype 2 if the sum is smaller.

$q = 0$. For intermediate values of a and $ae_1 < t < ae_2$ the developmental path is a mixture of bet-hedging and plasticity. One can show that

$$ae_i + (1-a)n < t \text{ with probability } \Phi\left(\frac{t - ae_i}{1-a}\right) \tag{1.6}$$

$$ae_i + (1-a)n > t \text{ with probability } \Phi\left(\frac{ae_i - t}{1-a}\right), \tag{1.7}$$

where $\Phi(x)$ is the cumulative distribution function of the normal distribution $N(0,1)$ ($\Phi(x) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{x} exp[-\frac{1}{2}v^2]dv$). The equation $\Phi\left(\frac{t-ae_i}{1-a}\right) + \Phi\left(\frac{ae_i-t}{1-a}\right) = 1$ holds of course, as every individual must either express phenotype 1 or phenotype 2.

Note that for very small t almost only phenotype 1 is expressed, whereas for very large t virtually all offspring expresses phenotype 2. In these cases we are close to a unique genotype-phenotype mapping. For $a = 0$, the system presented here is related to the one presented in the preceding section by $q = \Phi(t)$.

Table 1.3 gives the number of offspring produced by an individual with genotype (z_1, z_2, t, a) under environmental condition j for the cases where the environmental cue is correct or incorrect.

For simplicity we will mostly use a symmetric model with $-e_1 = e_2$ and $p = 0.5$. If additionally the z_1 value and the z_2 value of the singular point are of equal distance from 0.5 ($|0.5 - z_1| = |0.5 - z_2|$) - as it is the case in our model - one can prove that the singular value $t^* = 0$. Therefore we will in most of the following reduce the dimension of the model by setting $t = 0$.

Table 1.3.: Per capita number of surviving offspring as a function of the environmental condition on whether the cue, e_i, is correct or not, the value of the threshold of the switch device, t, and the weighting factor, a. The function Φ is the cumulative distribution function of the normal distribution $N(0,1)$.

Env. cond.	Cue	Per-capita number of surviving offspring	Probability
1	correct	$\beta_1^+ = \Phi\left(\frac{t-ae_1}{1-a}\right)\alpha_1(z_1) + \Phi\left(\frac{ae_1-t}{1-a}\right)\alpha_1(z_2)$	pr
1	wrong	$\beta_1^- = \Phi\left(\frac{t-ae_2}{1-a}\right)\alpha_1(z_1) + \Phi\left(\frac{ae_2-t}{1-a}\right)\alpha_1(z_2)$	$p(1-r)$
2	correct	$\beta_1^+ = \Phi\left(\frac{t-ae_2}{1-a}\right)\alpha_2(z_1) + \Phi\left(\frac{ae_2-t}{1-a}\right)\alpha_2(z_2)$	$(1-p)r$
2	wrong	$\beta_1^+ = \Phi\left(\frac{t-ae_1}{1-a}\right)\alpha_2(z_1) + \Phi\left(\frac{ae_1-t}{1-a}\right)\alpha_2(z_2)$	$(1-p)(1-r)$

1.3. Analytical Results

The results of the application of adaptive dynamics (see section 1.1) to the model described in section 1.2 are presented in detail in Rueffler et al. (in prep.*b*) and in Rueffler et al. (in prep.*a*). The following sections give a brief summary of these results. Section 1.3.1 gives the results for the model restricted to a canalized genotype-phenotype map, section 1.3.2 gives the results for the case where bet-hedging is allowed for and section 1.3.3 gives the results for the model with phenotypic plasticity. Section 1.3.4 presents the results for the complete model where all three mechanisms are possible. The section on bet-hedging mainly uses the notation described in section 1.2.1, while the framework presented in section 1.2.2 is used for the analysis of the case with plasticity and for the complete model.

1.3.1. Canalised Genotype-Phenotype Map

If z_1 and z_2 are constraint to be equal, it is sufficient to follow evolution in the one dimensional trait space (z). Searching the zeros of the selection gradient it is easy to show that $z^* = 1 - p$, where p is the probability that environment 1 occurs, is the only singular point. This trait value is convergence stable for all values of c. For $c + d > 1$ this point is a local maximum and consequently a CSS, while it is a local minimum and hence an evolutionary branching point for $c + d < 1$.

1.3.2. Bet-Hedging

By searching the zeros of the selection gradient, equation 1.1, we find $z_1^* = z_2^* = 1 - p$ as unique singular point in the 2-dimensional trait space. The third dimension, q, is selectively neutral for these trait values, which means that we have a line of equilibria $(z_1^*, z_2^*, q) = (1 - p, 1 - p, q)$ with $q \in [0, 1]$.

As mentioned in section 1.1 the definiteness of the Hessian matrix (H) determines whether a singular point is a maximum or a minimum in the fitness

landscape and the definiteness of the Jacobian matrix (J) determines whether such a point is convergence stable or not. In this model the following cases can be distinguished:

- $c < 1-d \Rightarrow$ J indefinite, H positive definite: The singular point $(1-p, 1-p)$ is a saddle point of the evolutionary dynamics. A canalised genotype ($z_1 = z_2$) can coexist with a resident at the singular point and branching in this direction is thus possible. This is the case we will be interested in.

- $1-d < c < 1 \Rightarrow$ J indefinite, H indefinite: The singular point $(1-p, 1-p)$ is still a saddle point of the evolutionary dynamics but a canalised genotype cannot coexist with the resident.

- $c > 1 \Rightarrow$ J negative definite, H negative definite: The singular point $(1-p, 1-p)$ is a continuously stable strategy (CSS). This means that it is an evolutionary attractor and once the population has settled there only the third dimension of the trait space, q, changes through neutral drift. We will not treat these cases further as the development of polymorphisms is not possible in such a setting.

In the first two cases of the listing above the population is not expected to evolve to the singular point, but instead to evolve to the boundary of the trait space (this means to extreme values of z_1 and z_2, i.e. 0 or 1). However, the question we want to treat here, also asked by Leimar (2005), is what will be the evolution of a population starting at a singular point. As the biological mechanism underlying bet-hedging is something that most likely does not exist a priori, but has to evolve, such a scenario seems realistic. In the case $c < 1 - d$ three things can happen:

1. Mutation occurs along the diagonal of the trait-space (where $z_1 = z_2$). Such a mutant can always coexist with the resident at the singular point and a protected polymorphism of two genotypes without bet-hedging develops.

2. Mutation occurs such that $z_1 \neq z_2$ and this mutant can coexist with the strategy at the singular point. Then a genetic polymorphism with two bet-hedging strategies will develop.

3. Mutation occurs such that $z_1 \neq z_2$ and this mutant cannot coexist with the resident at the singular point. Then the mutant replaces the former resident and a single bet-hedging genotype develops.

In which direction the population is most likely to evolve, depends on the slope of the fitness landscape and on possible correlations (which we will treat later). The leading eigenvector of the Hessian matrix gives the direction of the steepest slope of the fitness landscape. Without correlations this direction will be

favoured by selection. Figure 1.5(a) shows that the direction of the eigenvector (dotted line) depends on the phenotype proportion, q. At the same time it illustrates that the directions in which coexistence is possible depend as well on q. Only mutants in a direction corresponding to the grey area of Figure 1.5(a) can coexist with the resident. Others would drive the resident to extinction. The border between grey and white areas is thus the line of distinction between cases 2 and 3 of the enumeration above. Studying Figure 1.5 we can distinguish several situations (note that the specific values of q at which these situations merge depend on the particular parameter values):

- $q = 0.5$**:** If the two possible phenotypes per genotype are initially produced with equal frequency, roughly only mutants with either both increments, in z_1 and z_2, positive or both negative can coexist in a protected polymorphism with the resident (grey area in Figure 1.5(c)). The strongest selection (direction of eigenvector) is, however, in the direction where $\delta z_1 = -\delta z_2$ (dotted line in Figure 1.5(c)). This means that for $q = 0.5$ a pure bet-hedging strategy (case 3) is expected to evolve.

- $0.25 \leq q \leq 0.75$**:** In these cases the strongest selection is still in a direction where coexistence is impossible. Consequently, similar to $q = 0.5$, we expect a single bet-hedging genotype to evolve. In contrast to $q = 0.5$, mutants with z_1 and z_2 not symmetric around 0.5 are favoured now. For $q = 0.25$ and $q = 0.75$ the eigenvector points in the direction that distinguishes between angles in which coexistence is possible and impossible (see Figure 1.5(b)).

- $q < 0.25$ **or** $q > 0.75$**:** In these cases the leading eigenvector of the Hessian points in a direction where coexistence is possible (the dotted line lies in the grey area in Figure 1.5(d) for q=0.9) and we expect the evolution of a dimorphism of two bet-hedging genotypes (case 2 of the listing above).

Note that a genetic polymorphism of canalised phenotypes (mutation in the direction of the diagonal, case 1 in the enumeration above) is never favoured in our model. A more detailed discussion of the different scenarios can be found in Rueffler et al. (in prep.*b*).

In the current work we focus on the situation with equally frequent environmental conditions (p=0.5), $c < 1 - d$ and an initial population that is located at the singular point $(0.5, 0.5, q)$. Then genetic polymorphisms and bet-hedging genotypes are both possible. Leimar (2005) finds that in this model with a temporal variable environment the evolution of bet-hedging is always favoured. But Leimar only investigates the case where $q = 0.5$ and compares mutations in the direction of bet-hedgers with phenotypes that are equally well adapted for the two environments (mutational increments $\delta z_1 = -\delta z_2$) with with mutations in the direction of a pure polymorphism with $z_1 = z_2$ for each of the two sub-populations. Our results suggest, however, that if different values of q are

considered, the strongest selection can be for bet-hedging genotypes that can coexist and form a genetic polymorphism. The question "Does bet-hedging *or* a genetic polymorphism evolve?" is thus not well formulated in such cases.

The simulations presented in section 3.1 investigate the further evolution in such situations and study evolution in the case of mutational covariance between the traits.

1.3.3. Plasticity

If we fix a at 1, then the switching device presented in 1.2.2 allows for developmental plasticity but not for bet-hedging. In this case the z_1- and z_2-isocline are given (see Rueffler et al. (in prep.a)) by, respectively, the vertical and the horizontal line,

$$\left(\frac{1-p-r+pr}{1-p-r+2pr}, z_2\right) \text{ and } \left(z_1, \frac{-r+pr}{-p-r+2pr}\right). \tag{1.8}$$

When both environmental conditions are equally probable ($p = 0.5$) the expressions for the isoclines simplify to

$$(1-r, z_2) \text{ and } (z_1, r). \tag{1.9}$$

The point of intersection of the two isoclines is the only singular point of the two dimensional trait space (this point is not defined for $(r,p) \in \{(0,0),(1,0),(0,1),(1,1)\}$). This point only coincides with the singular point of canalised genotype when $r = 0.5$.

Both eigenvalues of the Jacobian matrix of invasion fitness are negative for all values of $r, p \in (0,1)$. Thus, the singular point is always convergence stable.

The eigenvalues of the Hessian matrix are positive if and only if $c < 1-d$. In that case the singular point is a branching point that can be invaded by all nearby mutants leading to a genetic polymorphism. In the opposite case, where $c > 1-d$, the eigenvalues of the Hessian matrix a negative and the singular point corresponds to a continuously stable strategy (CSS). Note that disruptive selection, leading to a genetic polymorphism occurs under exactly the same conditions as in the case of a canalized genotype-phenotype map. Hence, the evolution of phenotypic plasticity does not annihilate disruptive selection. The further evolution after a population has become polymorphic can not be predicted by an analytical treatment. It shall be investigated by simulations in the following chapters.

1.3.4. Complete Model

Here we consider the general case of the framework presented in section 1.2.2 where the weighting factor between plasticity and bet-hedging, a, can evolve freely. We focus on the case with $p = 0.5$ and assume without loss of generality that $e_1 = -e_2$. As $t^* = 0$ in the symmetrical case, we will reduce the trait space

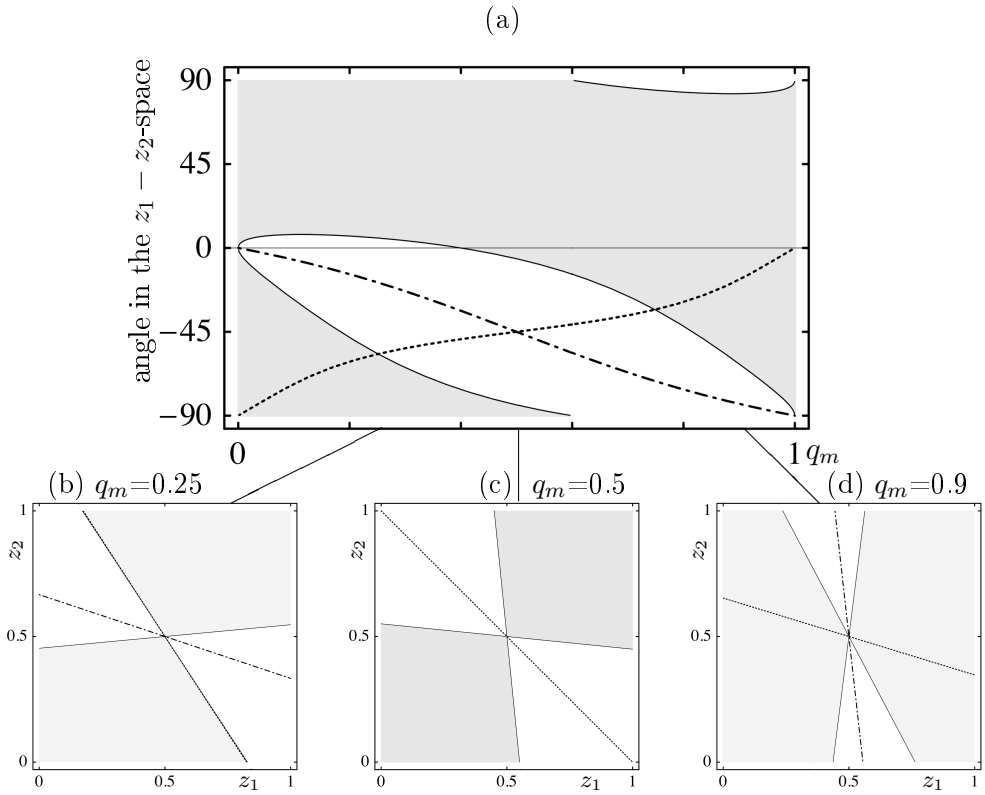


Figure 1.5.: Evolutionary dynamics at a singular point $(z_1, z_2) = (0.5, 0.5)$ for $c+d < 1$. In this case the Hessian-matrix is positive definite and the singular point is a minimum in the two-dimensional fitness landscape. The top panel (a) gives, as a function of q, the angle of the dominant eigenvector of the Hessian matrix at the singular point (dashed line). The points of the dashed line thus correspond to the direction of strongest selection. The grey area of the graph marks directions in which the coexistence of a mutant with the resident strategy at the singular point is possible. In the white area coexistence is impossible and thus evolutionary branching does not occur. The dot-dashed line gives the direction in which Leimar (2005) measures the strength of selection for bet-hedging. In this direction coexistence is not possible. However, only for $q = 0.5$ this direction follows the steepest slope of the fitness landscape marked by the dominant eigenvector. We can see that for increasing distance of q from 0.5 the direction of the dominant eigenvector of the Hessian matrix changes until it reaches a direction where coexistence is possible at $q = 0.25$ and $q = 0.75$ respectively. For $q < 0.25$ or $q > 0.75$ genetic polymorphisms can evolve. The panels (b)-(d) show the dominant eigenvector of the Hessian, the area of coexistence and the line corresponding to the direction in which Leimar measures the strength of selection for bet-hedging in the (z_1,z_2)-plane for three values of q. Line styles are the same as in panel (a). Note that in panel (b) where $q = 0.25$ the dominant eigenvector coincides with the border of the area of coexistence and that in (c) where $q = 0.5$ the direction in which Leimar measures the strength of selection for bet-hedging coincides with the dominant eigenvector. Parameter values used: $z = 0.6$, $d = 0.3$.

for the analytical treatment to (z_1, z_2, a) by setting $t = 0$. This assumption will be relaxed in the simulations. If $a < 1$ the isoclines can only be found numerically. As mentioned in the preceding section, for pure plasticity ($a = 1$) the singular point $(z_1, z_2) = (1-r, r)$ is a CSS for $c > 1-d$. However, if a is not fixed, mutants characterised by a smaller value of a can invade. A population with a slightly smaller than 1, experiences selection towards more extreme values of z. For the special case of $c = 0.6$ and $d = 0.6$ a population characterized by the z-values at the intersection of the two isoclines and $a = 0.88$ starts to experience disruptive selection in the direction of z_1 and z_2 and possibly starts to branch into a genetic dimorphism. The exact evolutionary dynamics needs to be investigated by the simulations presented in Chapter 3. If the population does not branch but becomes invaded by individuals with even smaller values of a it subsequently experiences directional selection towards $z_1 = 0$ and $z_2 = 1$. For a population with $t = 0$, $|e_i| = 0.25$, $c = 0.6$, $d = 0.6$, $z_1 = 0$ and $z_2 = 1$ in an environment with $p = 0.5$ and $r = 0.75$ selection toward smaller a proceeds until $a^* = 0.73$. The genotype $(z_1, z_2, a) = (0, 1, 0.73)$ combines elements of both bet-hedging and plasticity and cannot be invaded by any other genotype.

Given that the sign of the fitness gradient in the directions of z_1, z_2 and a is independent of the death probability d, the location of the z_1 and z_2 isoclines does not change when d is varied. The convergence and uninvadability properties of the singular point do, however, depend on d. For $c+d < 1$ the point $(z_1, z_2) = (1-r, r)$ turns into a branching point in the plane $a = 1$, while at the same time selection favours mutants with smaller values of a. The question whether this change in a occurs fast enough to prevent the populations from branching or not, will be investigated by the simulations presented in section 3.3.

2. Programming

As we have seen in the preceding chapter, the adaptive dynamics approach is based on several assumptions (see Table 1.2). In many ecological scenarios these assumptions seem unrealistic. This is the first point why individual-based simulations are important: To investigate the robustness of the results found with the adaptive dynamics method, when these assumptions are not strictly fulfilled. More precisely, we will use in the simulations finite population sizes, considerable mutational step sizes and high mutation rates. The mutation rates will be so high, that a separation of the population dynamical and the evolutionary time-scales is not realised: The population will not be at an equilibrium before a new mutant occurs, and many mutant sub-populations will exist within the population at the same time.

Another reason for the necessity of these simulations is that it is difficult to calculate invasion fitness in our stochastic environment. In some situations the deterministic treatment cannot provide any results. For example, situations with polymorphic populations that are not symmetric around the singular point are not analytically solvable. Thus, often simulations are required to know what happens after branching has occurred. Also for the case with mutational covariance between the two alternative phenotypes one has to rely on simulations.

Here we develop a general program that simulates the dynamics of the complete model and that incorporates the possibility of mutational covariance between the traits. The functions used for these purposes are written in Matlab (MathWorks, 2007). The main program is presented in section 2.1 and its graphical output in section 2.1.1, while section 2.2 describes the technical difficulties with the classification of possible evolutionary end-points.

2.1. The Main Program

The final program used for the simulations is the result of a long process of optimising and adapting earlier versions. Below, we only comment the main decisions concerning the design of the program. A very straightforward approach, keeping track of the genotype of each and every individual in the population turned out to be computationally too demanding when population sizes are larger than about 100 individuals. That is why the program finally employed uses a different approach, which does not follow every individual, but follows the different genotypes present in the population and keeps track of the number of individuals carrying these genotypes.

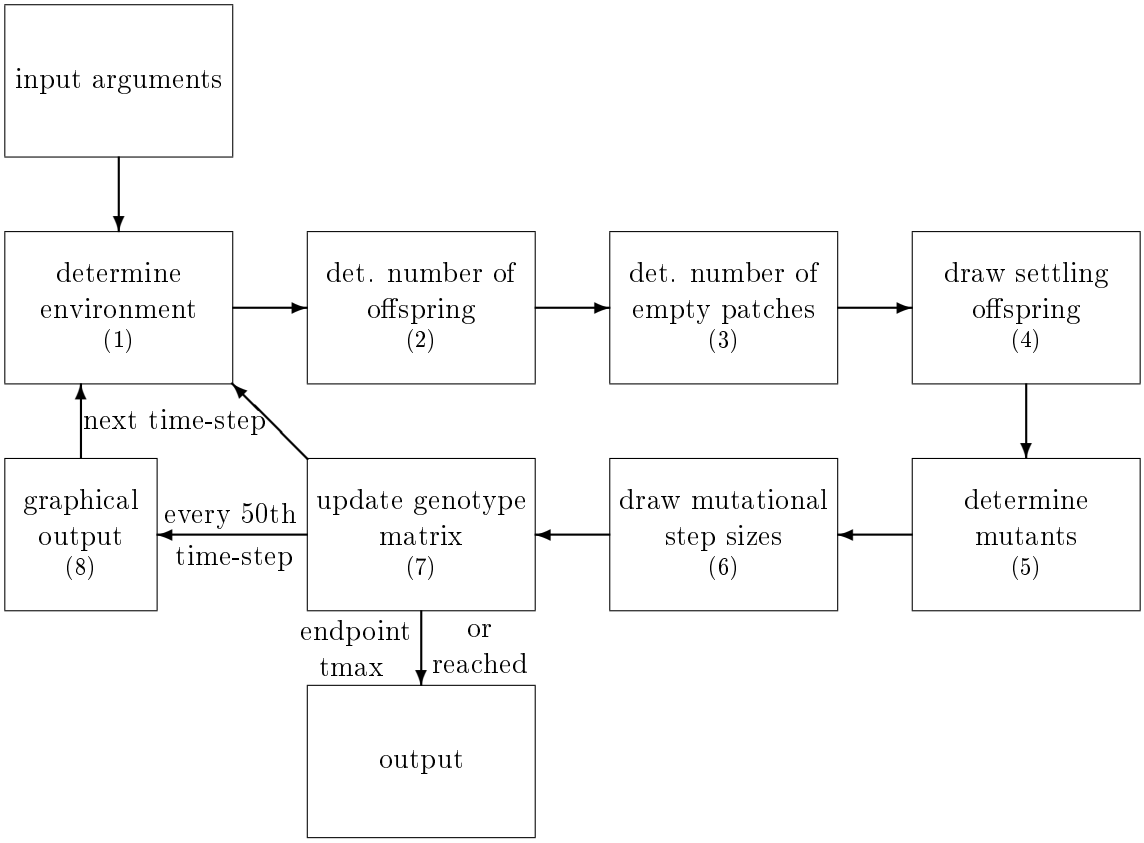


Figure 2.1.: Schematic representation of the program's structure. The numbers refer to the points of the enumeration in the main text.

The program presented here uses the notation of the complete model described in section 1.2.2. An earlier version of this program restricted to the special case where only bet-hedging is allowed for was used for the simulation presented in section 3.1. This version uses the notation described in section 1.2.2; it will not be presented here, but section 1.2.2 describes how the two notations can be transferred into each other. Figure 2.1 gives a schematic representation of the program's structure, which is detailed in the following.

Input arguments: The program takes as input arguments the initial population structure (genotypes present and their numbers) and the parameters determining the mutational process, such as mutation probability, variance-covariance matrix of the probability distribution of the mutational increments and the step sizes in trait space. Furthermore, the model parameters described in section 1.2, such as the probability to encounter environmental condition 1, p, the death probability, d, the trade off parameter, c, and the cue reliability, r, are given as input arguments. Other inputs are the maximal number of iterations, whether the program should stop when a possible "end-point" (will be discussed later) is attained and whether there should be a graphical output. The input arguments are summarized in Table A.1. The function itself consists of a loop comprising the following steps:

1. Determine the current environment.
2. Determine the number of offspring for each genotype in the population.
3. Determine the number of empty patches. Each iteration, an individual dies with a probability d.
4. Draw the number of settling offspring for each genotype.
5. Determine the total number of mutations for the different traits and allocate them to the different offspring genotypes.
6. Draw the mutational step size for each mutating individual and add the new genotypes to the populations.
7. Update the genotype matrix.
8. If not suppressed: Every 50 generations, add mean value and standard deviation of z_1, z_2, a, t to a time-series plot. Every 100 generations draw a z_1, z_2-phase diagram of the population.

Steps 1-8 are iterated until either the maximal number of generations or one of the evolutionary end-points, which will be discussed in section 2.2, is reached.

Output variables: The output parameters of the function include the mean values and standard deviations of a, z_1, z_2 and t for every time-step, the complete genotype matrix every 100 time-steps, the type of evolutionary end-point, if any

is attained, and the input parameters. These output parameters are summarized in Table A.2.

The heart of the program is the genotype matrix. Each row contains the trait values (z_1, z_2, a, t) of a different genotype and the number of settled individuals carrying this genotype. Hence, the number of rows is not fixed, but corresponds to the number of different strategies present in the population. In A.1.1 the source code of the main program, called "BetHedgingGen_2_4_f", is given. It is documented in detail and should be comprehensible for interested readers with a little bit of a Matlab background. Readers not familiar with Matlab, however, should know that Matlab source code is comparatively hard to read, as the dimensions of objects are not clearly defined and many functions can be applied to scalars, vectors, matrices and even higher dimensional objects.

As parts of our studies incorporate covariance between the two traits z_1 and z_2, the sizes of the mutational increments in z_1 and z_2 are drawn from a 2-dimensional Gaussian distribution, the shape of which is determined by a variance-covariance matrix. This means that an individual that mutates in z_1 necessarily also mutates in z_2 (only a single drawing for z_1 and z_2 in step 5 of the listing above), even if there is no covariance. This is of course unnatural, but another version of the program, where z_1 and z_2 mutate independently gives qualitatively identical results as the described program with zero covariance.

There are also other steps where the program does not exactly follow the population dynamics, but makes approximations. The comparison with the simulation results of an exact - but much slower - individual based program showed that these inaccuracies do not change the outcome. But we still want to mention these approximations here:

1. The hypergeometric distribution for the allocation of offspring to empty patches is approximated by a multinomial distribution. At one time step, the sum of offspring from all the individuals present in the population is called gamete pool. Assume that each individual has very many offspring. Then the structure of the gamete pool is nearly unchanged when one individual is removed. Thus, one can draw the genotypes settling the vacant patches from the same multinomial distribution, using the frequency distribution of the different genotypes in the gamete pool as probabilities. The approximation here is that the frequencies are not adapted after each drawing (as an individual is removed), but they stay constant for all the drawings at one time-step. The correct way would be to keep track of each individual in the offspring pool, but as we are using large numbers of offspring per individual the approximation done here can be considered as very good. Comparisons with another version where the offspring is directly allocated to the empty patches, without passing over a frequency, show identical results.

2. The hypergeometric distribution for the allocation of mutations to the settled offspring is approximated by a multinomial distribution. An ap-

proximation, very similar to the one in the first point, is done to simulate the mutational process. The function draws the total number of settled offspring mutating in z_1 and z_2 and the numbers mutating in a or t from a binomial distribution. Here again, the mutations are allocated to the different genotypes by drawing from a multinomial distribution, with the frequency distribution of the newly settled genotypes as parameter. In this case, however, the approximation is more severe than for the settling of the offspring, because the number of settled offspring is limited by the number of empty patches ((population size)$*$(death rate)) and far from being "infinite". It could be the case that - by chance - 2 individuals of one genotypes are drawn, even though only one individual with such a genotype exists among the settled offspring. In such a case the program does as if there was one more individual than empty patches. This makes that the population size may grow slightly bigger than the patch number. However, the growth is limited as in the following generation the emptied patches are only filled up to a population size equalling the total patch number. This inexactness is of course inelegant, but simulations show that, with the mutation rate used for the following analyses, the problem only occurs about once every 10000 time-steps. Other versions of the program that deal with this issue in a correct way, had, with the used parameters, an about 25% longer running time and showed qualitatively identical results. That is why this implementation was chosen.

3. The possibility of simultaneous mutations in z_1/z_2 and a or t is ignored. As mentioned above z_1 and z_2 always mutate at the same time; a and t, however, cannot mutate simultaneously or together with z_1/z_2. This means that an individual that mutated in either z_1/z_2, a or t, will not mutate in an other trait, which should biologically by chance be possible. This, being an other inelegance of the code, does not seem to have any influence on the outcome, as in the following generations a mutation in the other traits is possible anyway. Additionally, for low mutation probabilities the chance of double mutations becomes negligible.

In conclusion one can say that it was possible to create functions that are able to simulate the dynamical system described in section 1.2 with acceptable running times and in an automatized way. Several approximations were made in order to optimise the performance of the code. Most of these approximations are clearly justified by the assumptions we make in our model and the others were tested by the comparison with the outcome of exact versions of the code.

The program described in this section can be used in two different ways: Either one uses the graphical output included in the function to get direct qualitative results by observing how the population develops, or one runs simulations without any graphical output and determines the results later with the help of analyse tools. The following section presents the graphical output.

2.1.1. Graphical Output

The graphical output is shown in Figure 2.2. The shot was taken after 5900 time-steps for a simulation with a maximum of 10000 time-steps. Figure 2.2(a) gives the population wide mean values and standard deviations of a, z_1, z_2, (b) gives a $z_1 - z_2$ phase diagram and (c) gives mean value and standard deviation of the threshold t. The blue line in Figure 2.2(a) shows the mean of z_1, while the red one shows that of z_2. One can see that at time $T = 0$ the mean values of z_1 and z_2 were equal to 0.5 (panel (a)(2)), while their population wide standard deviations were 0 (panels (a)(3) and (a)(4)). The first and the last panel of graph (a) show that the weighting factor of the switching device, a, was equal to zero for the whole population at $T = 0$, while Figure 2.2(c) tells us that t was uniformly equal to 0.5 in the beginning. This example illustrates that we can often use the population wide mean values and standard deviations to infer the population structure. The values $T = 0$ show that the simulation was started at a monomorphic population $(z_1, z_2, a, t) = (0.5, 0.5, 0, 0.25)$. This corresponds to a population located at the centre of the $z_1 - z_2$-phase space, where phenotype z_1 is more common than phenotype z_2 (a t value of 0.5 corresponds to $q > 0.5$; see section 1.2.2). The phenotypes expressed will be determined solely by internal noise ($a = 0$), thus we have a bet-hedging strategy (not before z_1 and z_2 start to diverge by mutations of course). In the interval $2000 < T < 3000$ we see that the standard deviation in z_1 grows, meaning that there is a certain amount of variation in these trait values present in the population. Large standard deviations can be an indicator for genetic polymorphisms. For $T > 3000$ all standard deviations are small again. This tells us that the vast majority of the population is concentrated in a comparatively small volume of the trait space. In our example the means of z_1 and z_2 become more and more distinct while a stays small, hence we are observing the development of a bet-hedging strategy. Figure 2.2(b) shows the population in the $z_1 - z_2$-space for $T = 5900$. When the simulations are running this plot is updated every 100 time-steps. Blue circles correspond to genotypes with less than 10 individuals, green squares to 10-100 individuals and red diamonds to more than 100 individuals. We can see that the population has moved from the initial point $z_1 = z_2 = 0.5$ halfway towards the corner of the trait space where $(z_1, z_2) = (1, 0)$. Note that in our example, as in all the presented simulations, mutation rates are so high that populations are never monomorphic, but we see populations consisting of a cloud of genotypes with similar trait values. These "quasi-monomorphic" populations will not be called polymorphic. Only two distinct clouds, each composed of genotypes with similar trait values, will be referred to as dimorphisms or polymorphisms.

The graphs shown in Figure 2.2(a) and 2.2(c) are directly produced by the main program "BetHedgingGen_2_4_f", while the sub-function "plot_pip_f.m" produces the z_1, z_2-phase-diagram (Figure 2.2(b)). The documented source code of "plot_pip_f.m" is presented in appendix A.1.2.

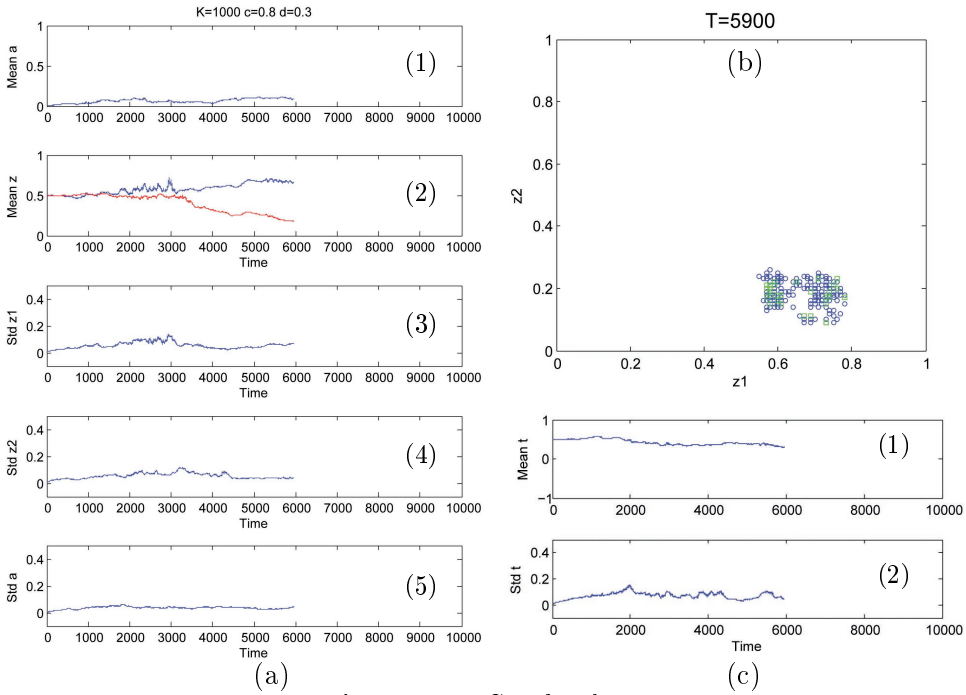


Figure 2.2.: Graphical output

Graphical output of the main program. Graph (a) gives the population wide mean of a (panel 1), z_1 (blue line) and z_2 (red line) (both panel 2) and the standard deviations of z_1 (panel 3), z_2 (panel 4) and a (panel 5) as a function of time. Graph (b) gives the z_1, z_2 phase diagram at time T. Graph (c) gives the population wide mean (panel 1) and standard deviation (panel 2) of t.

2.2. Determination of Evolutionary End-Points

The objective of this investigation is to study the evolutionary path in a situation characterised by certain parameter values. We are especially interested in the outcome of evolution, i.e., the population has reached some kind of evolutionary attractor. In the simulations this outcome will mostly be an (at least locally) optimal strategy that cannot be invaded by nearby mutants. In this case we can talk of a local fitness maximum. These evolutionary end-points will most often be situated next to the borders of the trait space (e.g. z_1 and z_2 are 0 or 1), where the local fitness gradient points in the direction of the border of the trait space. Another possible evolutionary outcome would be a cyclic succession of strategies, i.e., the occurance of evolutionary branching where one or more sub-populations evolve away from a "mother-population", die out after some time and then branching takes place again. A common definition for such evolutionary end-points would be that when such a point is reached the population stays in a restricted volume of the trait space.

In the light of the simulations presented in this work, evolutionary end-points can either be detected by looking at the data of each run of the simulation (usually via the graphical output) and interpreting it, or by developing a programmed routine capable of determining when an end-points is attained. The second approach has important advantages. First, in order to avoid stochastic effects most studies incorporate information from a large amount of simulation-runs. It would be very time consuming to examine all these runs by hand (eye). Dealing with the results in an automatised way helps to cope with the huge amount of information and to reduce the needed memory capacity. Second, a programmed routine defines clear criteria when an end-point is reached. This guarantees reproducible results. Third, how many time-steps it takes to reach an evolutionary end-point depends on the evolutionary path and can vary considerably between simulation-runs for a given set of parameters. This is due to stochastic effects. To minimize computational and storage costs it is desirable to run a simulation only until an evolutionary end-point is reached, because the further development does not provide any new information (The evolutionary dynamics has either come to a halt or it continues in predictable cycles.). An automatised stopping routine can be launched during a simulation and terminate the run if an endpoint is reached before the maximum number of time-steps was reached.

The number and kind of such evolutionary end-points depends on the examined situation, i.e., the model parameters and the initial population. Therefore the development of a detection routine for a number of simulations where a system parameter is gradually changed is not an easy task. First, one has to investigate a large number of simulations by hand to get a picture of the possible end-points. Then reliable yet not too complicated criteria how to identify such a point unambiguously in given data have to be found. Here we want to present the routines determining evolutionary end-points and define break conditions

for two situations. The comparatively simple routine presented in section 2.2.1 was employed for investigations in the bet-hedging model, while section 2.2.2 describes the more complicated routine for a situation with pure plasticity.

2.2.1. Evolutionary End-Points in a Bet-Hedging Model

Here we want to describe the development of a routine that is able to determine the evolutionary-end points in a situation where a genotype can produce alternative phenotypes by bet-hedging but not via phenotypic plasticity. This corresponds to a population where the weighting factor a of the switching device presented in 1.2.2 is fixed to 0. In a scenario where the singular point is a branching point ($c+d<1$) we want to investigate the frequency of the different evolutionary end-points as a function of the initial threshold value t (equivalent to the initial phenotype weighting factor q in the notation of section 1.2.1) and the mutational covariance between the two alternative phenotypes z_1 and z_2. The results of this investigation are presented in section 3.1.2.

A manual investigation of many runs of the program for different parameter values showed that the following end-points occur:

1. A (quasi-)monomorphic population in the neighbourhood of the bet-hedging strategy (z_1, z_2)=(1,0) and q=$1-p$.

2. A (quasi-)monomorphic population in the neighbourhood of the bet-hedging strategy (z_1, z_2)=(0,1) and q=p.

3. A (quasi-)dimorphic population with two clouds of genotypes; one situated around (z_1, z_2)=(0,0), the other one around (z_1, z_2)=(1,1), where q (respectively t) is selectively neutral and hence shows a larger variation.

To detect these end-points the function "PolyOutcome_f.m" was developed. The first input argument is the population matrix at a fixed time-step. The second input argument is a number that determines the maximum distance of a population from an end-point such that it is identified as that end-point. Remember that we will use high mutation rates that imply that resident strategies always consist of a large number (of more or less) similar mutants. Given that all the sub-populations existing at the possible evolutionary end-points are in corners of the $z_1 - z_2$-trait space, the function just counts the individuals that are contained in a quadratic neighbourhood of the corners. For end-point 1 of the listing above to be detected, more than 80% of the population has to have trait values z_1 and z_2 closer to 1 resp. 0 than the number given as second input argument. In that case the function returns the integer 1. End-point 2 is treated in an equivalent way. For end-point 3 to be attained 90% of the population has to have trait values in the quadratic neighbourhoods around (0,0) and (1,1), and none of these two neighborhoods should be empty. The function gives 3 as output. In the case where none of those criteria is fulfilled the output

is 0. The commented source code of this function is given in appendix A.1.3. It is implemented in the main program in the way that it is launched every 1000 time-steps. If it returns an outcome other than 0, the simulation is stopped.

2.2.2. Evolutionary End-Points in a Plasticity Model

In a situation that allows for the development of phenotypic plasticity but not for bet-hedging (a fixed to 1) and where the singular point is a branching point ($c+d<1$) more complicated evolutionary end-points exist. We want to investigate the type and frequency of the evolutionary end-points reached by the dynamics for different values of the trade-off parameter, c, the death rate, d, and the cue reliability, r. The threshold, t, was fixed to 0. The results of this study are presented in section 3.2. The examination of preliminary results by hand showed two different categories of end-points. On the one hand, similar to the bet-hedging model, we get stable end-points consisting of genotypes that are situated in the corners of the trait space. On the other hand, cyclic end-points occurred in a large number of cases. These cyclic end-points are of the following form:

1. branching occurs
2. sometimes a second branching event occurs to one of the two sub-populations
3. sub-populations evolve to different regions of the trait space
4. one or more of the sub-populations go extinct
5. a) if only one sub-population is left, it evolves back to the branching point and the cycle restarts at point 1
   b) if more sub populations are left, one of them branches again and the cycle is back at point 3

With cyclic end-points, it is not enough to develop a function that determines which of the corners are populated, because we could have a cycle where things change after some time. In this case a routine that determines whether an end-point is reached should not only take into account the current situation, but also the configuration at some points in the past. Technically this is realised by two functions. One, very similar to the one presented in appendix A.1.3 for the case of bet-hedging, determines which of the corners are populated and a second one compares the current result with the results of the last 15 queries (there is one query every 1000 time-steps). If all the results are identical, the corresponding end-point is detected. If the succession of results shows the characteristics of a cyclic end-point, it is detected.

In conclusion one can say that the possible evolutionary end-points depend on the investigated situation. A programmed routine that is capable of determining when an end-point is reached has to be adapted to deliver satisfying results in

different studies. Especially the detection of cyclic end-points will need new adjustments when mutation probabilities or mutational step sizes are changed.

3. Results and Discussion

This chapter contains most of the results obtained from simulations performed with the program presented in 2.1 and a discussion of the findings. Part of the simulations was performed to test the predictions of the deterministic model, while other simulations investigate situations in which an analytical treatment is impossible.

3.1. Bet-Hedging

Here we investigate the situation where the genetic architecture allows for the evolution of a bet-hedging mechanism for the determination of offspring-phenotypes but not for phenotypic plasticity. This corresponds to $a = 1$ in the formalism presented in 1.2.2 but for convenience and easier comparison with Leimar (2005) we will use the notation presented in 1.2.1. Thus genotypes are described by (z_1, z_2, q), where q is the probability that phenotype z_1 is expressed. Note that, when $a = 0$, the notations are related by $q = \Phi(t) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{t} \exp[-\frac{1}{2}v^2]dv$.

The simulations that will be presented in section 3.1.1 were performed to test the predictions of the deterministic model. As noted in Table 1.2, the adaptive dynamics approach assumes low mutation rates, such that the population dynamics is at an equilibrium before a new mutant occurs. In the simulations mutations occur with probabilities of 0.1 or 0.01 per time-step, trait and individual. With these parameters the population never reaches a population dynamical equilibrium before new mutants occur and populations always consist of extended clouds in trait space. For easier comparison with the results of the deterministic model a cloud of genotypes with similar trait values will be called monomorphic in the following, two clouds each composed of similar genotypes dimorphic or polymorphic and so on. Section 3.1.2 presents an investigation of the influence of mutational covariance between the alternative phenotypes on the outcome of evolution, while section 3.1.3 tests how the results depend on mutation rate and population size.

3.1.1. Qualitative Investigations

A large number of simulations with different parameter values was performed. The results always matched well with the deterministic predictions: In the case of a concave trade-off ($c > 1$) the population stays at the generalist genotype $(z_1, z_2, q) = (0.5, 0.5, q)$ or evolves there if initially away from this manifold. Remember that q is neutral in this case. For weakly convex trade-offs ($1 - d <$

$c < 1$) the population evolves towards a perfect bet-hedging strategy with trait values in the neighbourhood of $(z_1, z_2, q) = (1, 0, p)$ or $(0, 1, 1-p)$, where p is the probability to encounter environmental condition 1. Polymorphisms do not evolve with such parameter values. For strongly convex trade-offs ($c < 1 - d$) the singular point is a branching point in some directions of the trait space and genetic polymorphisms can occur. Again, the analytical result, that the likelihood that such polymorphisms evolve at a branching point depends on the initial proportion with which alternative phenotypes are produced, is well met. When both phenotypes are initially produced with equal frequencies ($q = 0.5$), genetic polymorphisms are extremely rare. The further the initial distribution is biased towards one of the phenotypes (q away from 0.5), the larger proportion of simulation runs with a genetic polymorphism can be observed. The interesting observation, however, is, that without any covariance these polymorphisms are only temporary and vanish after a certain time. Figure 3.1 shows the typical evolution for a case where a polymorphism forms: a population that started with a single genotype $(z_1, z_2) = (0.5, 0.5)$ (and $q = 0.9$ in this example) is composed of different mutants close to this point (Figure 3.1(a)). After some time (for a mutation rate of 0.1 typically within the first 4000 time-steps) the cloud of genotypes splits into two distinct clusters (Figure 3.1(b)). These clusters usually evolve away from the branching point in two opposite directions (c). For $q > 0.5$ the clusters move faster in the z_1-direction (horizontal), and for $q < 0.5$ they move faster in the z_2-direction (vertical). This is intuitive as in these cases the respective other trait value is less expressed. After some time one of the two clusters disappears (Figure 3.1(f)). For initial q close to 0.5 this happens before the two clusters can reach the (opposite) borders of the trait space, for q close to 0 or 1 they typically reach the borders, as can be seen in Figure 3.1(d). If q is close to 1 for instance, the two clusters usually evolve to $z_1 = 1$ and $z_1 = 0$, respectively, while z_2 is nearly neutral. At the borders the two clusters experience more or less neutral drift in z_2. Once a cluster has evolved into a corner corresponding to a perfect bet-hedger ($(0, 1)$ or $(1, 0)$), it drives the other cluster to extinction (Figure 3.1(e)) and q evolves to its optimum. This optimum equals the probability that the environmental condition individuals with trait value z_1 are specialised in occurs. In the example of Figure 3.1 this would be $q = p$, where p is the probability to encounter environment 1.

The polymorphisms in the simulations vanish after some time because at some point they stop to be protected. To understand why this is the case, one has to understand the underlying mechanism of protection. This mechanism is described in Warner and Chesson (1985) and in Ellner and Hairston (1994). Two canalised genotypes with phenotypes on the opposite sides of the singular point (one more specialised in environmental condition 1, the other one in condition 2) always benefit from a certain protection in the form of negative frequency dependant selection. This can be understood intuitively: In years where one of the two genotypes performs well (high offspring survival) the other one performs poorly (low offspring survival). Given that the number of patches is limited, a

good year leads to considerably higher relative growth rates for rare genotypes than for genotypes that are already abundant. This mechanism helps genotypes that are rare to increase in favourable years and thus protects them against extinction. The strength of this mechanism does of course depend on the model parameters such as d, c and K. A coalition of two bet-hedging genotypes, however, does not benefit from such a protection mechanism because they perform more or less equally well under both environmental conditions. That is why two genotypes are protected as long as nearly only one phenotype is expressed (q close to 0 or 1) but one of them dies out when they approach bet-hedging strategies with two equally expressed phenotypes that are each specialised in one of the two environmental conditions.

3.1.2. Mutational Covariance

The results of the preceding section show that without covariance in the mutational step size for z_1 and z_2 genetic polymorphisms are only temporal. Simulations and analytical results alike suggest that a genetic polymorphism with one cluster near $z_1 = z_2 = 0$ and another cluster near $z_1 = z_2 = 1$ (left bottom and right top corner in the phase diagram presented in figure 2.2(b)) cannot be invaded by nearby mutants. The simulations presented here investigate whether such a genetic polymorphism of the two specialists with canalised genotype-phenotype map could evolve if the development of a bet-hedging strategy was hindered to some degree. The difficulties in the development of a bet-hedging strategy shall be incorporated by two mechanisms.

- If an organism that originally produced only one phenotype starts to produce two different phentoypes, then it seems plausible that the alternative phenotype is initially produced with low frequency. This will be simulated by varying the initial frequency distribution of the two phenotypes, q. In the last section we saw, however, that extreme values of q can lead to the establishment of a temporary polymorphism but that a genetic polymorphism of the two canalised phenotypes ($(z_1, z_2) = (0, 0)$ and (1,1)) is never attained.

- If an organism that originally only produced one phenotype, starts to produce two different phentoypes, then it seems intuitive that the genetic and developmental pathways underlying these alternative phenotypes are coupled to some degree and cannot evolve totally independent. This will be modelled by a positive correlation between the mutational steps in z_1 and z_2, also called mutational covariance. Given that the advantage of a bet-hedging strategy is to simultaneously produce offspring adapted to two different environments, it is obvious that, starting at $(z_1, z_2) = (0.5, 0.5)$, the alternative phenotypes should evolve in opposite directions ($\operatorname{sign} \delta z_1 = -\operatorname{sign} \delta z_2$). The results in section 1.3.2 show that the strongest selection is indeed always in a direction where $\operatorname{sign} \delta z_1 = -\operatorname{sign} \delta z_2$. A positive

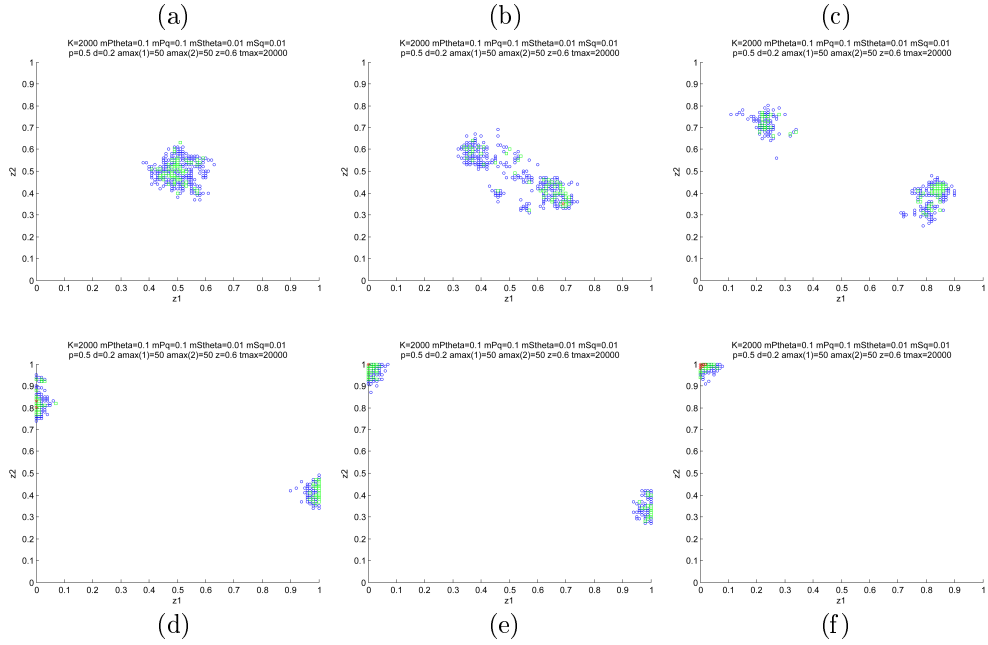


Figure 3.1.: Example of a temporary polymorphism occurring when $c < 1 - d$. The pictures show the genotypes present in the population in the $z_1 - z_2$-space. Blue circles correspond to genotypes with less than 10 individuals, green squares to 10-100 individuals and red diamonds to more than 100 individuals. After about 2000 time-steps the population separates into two distinct clusters which evolve to the borders of the trait space and eventually one cluster dies out when the other one reaches a corner corresponding to perfect bet-hedging. The proportion of phenotypes with trait value z_1, q, evolves to be equal to the probability that the environmental condition a phenotype z_1 is specialised in occurs; here this would be $q = p$. Figures (a)-(f) correspond to 1000, 2500, 3500, 5000, 6000, 7000 time-steps, respectively. The initial q-value equals 0.9. Parameter values used are $K = 2000$, $d = 0.2$, $c = 0.6$. The mutation probability is 0.1.

mutational covariance now means that mutations where z_1 and z_2 change in the same direction are more likely to occur than the opposite case.

A run of the simulations with mutational covariance between z_1 and z_2 is shown in Figure 3.2. We see that, contrary to the example without covariance in Figure 3.1, branching now occurs in the direction of the first main diagonal ($z_1 = z_2$). During the whole evolutionary process the genotypes stay close to this diagonal and in the end we get a stable genetic dimorphism of two canalised phenotypes ($(z_1, z_2) = (0, 0)$ and (1,1)).

In the following results for different combinations of initial values of q and for different amounts of correlation between z_1 and z_2 are presented. The possible evolutionary end-points are a population of perfect bet-hedgers or a genetic polymorphism of two canalised genotypes. Section 2.2.1 describes how this end-points can automatically be detected by the program. For various combinations of the initial q-value and values of the covariance between z_1 and z_2 100 runs of the simulation were performed on a computer cluster. The maximum number of time-steps was set so high that always an end-point was reached. Table 3.1 gives an overview of the parameter values used, while Figure 3.3 presents the simulation results. Covariance is measured relative to the variance of the step sizes in z_1 and z_2 (which are drawn from a normal distribution). In the absence of covariance the simulations only lead to the genetic polymorphism of canalised phenotypes as an evolutionary end-point in 4% of the cases for $q = 1$, but not for other values of q. In general one can see that the proportion of genetic polymorphisms among the end-points increases monotonically with increasing covariance and with increasing q. Only the data point $q = 0.6$ and covariance $= 0.2$ does not fit in this pattern. This phenomenon is most probably due to stochastic effects and should disappear with a higher number of simulations. Note that for intermediate values of covariance (0.3) q has a great influence on the distribution of evolutionary end-points. While for $q = 0.5$ only 19% of the runs produce a genetic polymorphism, this is the case in 96% of the runs for $q = 1$.

3.1.3. Dependence on Mutation Rate and Population Size

On the one hand we use high mutation rates and comparatively small population sizes in our simulations to relax the assumptions of the deterministic model, on the other hand this was convenient because it helped to reduce the necessary hardware resources and computation time. In situations where we could not obtain any analytical results, it is important to know how sensitive the simulation results are with respect to the chosen mutation rate and populations size. To investigate this question we take a point of the parameter space used in the previous section where both possible evolutionary end-points can be observed and study the proportion of the different outcomes in dependence on mutation rate and population size.

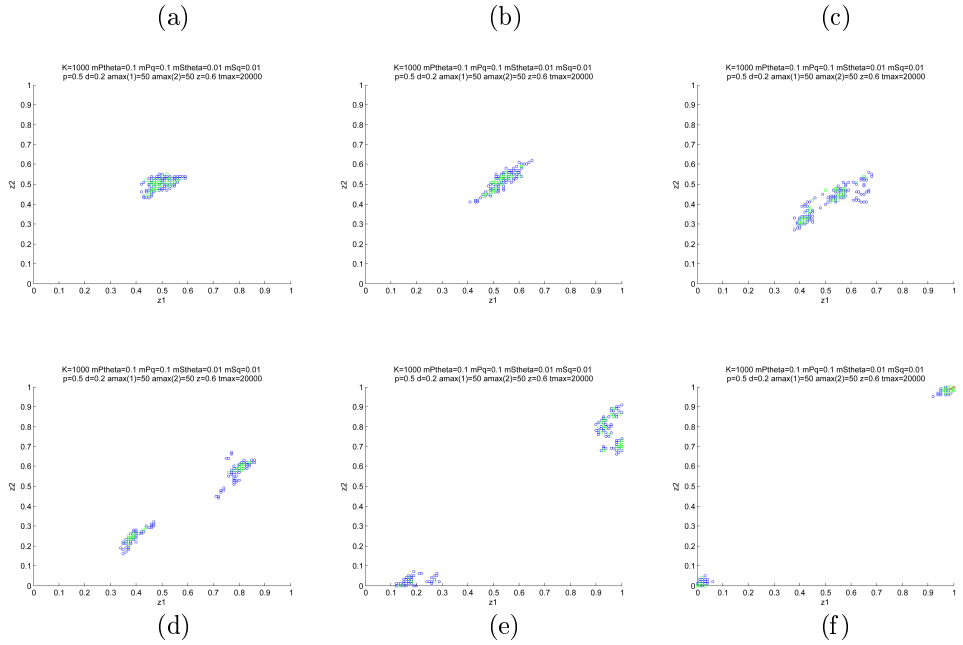


Figure 3.2.: Example of a simulation with strong correlation. The pictures show the genotypes present in the population in the z_1-z_2-space. Blue circles correspond to genotypes with less than 10 individuals, green squares to 10-100 individuals and red diamonds to more than 100 individuals. After branching the strong positive correlation holds the two clusters near the first main diagonal ($z_1 = z_2$) and the population gradually evolves to a pure genetic polymorphism of the two specialists. Panels (a)-(f) correspond to 1000, 2000, 6000, 9000, 11000, 13000 time-steps, respectively. The initial q-value is 0.8. Parameter values used are $K = 1000$, $d = 0.2$, $c = 0.6$. The mutation probability is 0.1. The covariance is 60% of the variance in the mutational step sizes of z_1 and z_2.

Table 3.1.: Input parameters used

Initial population structure	10000 individuals with (z_1, z_2)=(0.5,0.5)
	q is varied
Mutation probability	0.01 per time-step in z_1, z_2 and q
Variance	0.0001 in z_1, z_2 and q
Mutational step size	0.01 in z_1, z_2 and q
Environment distribution, p	0.5
Death probability, d	0.2
Maximal number of offspring	50 per individual
Trade-off parameter, c	0.6
Maximal simulation time	500000 time-steps

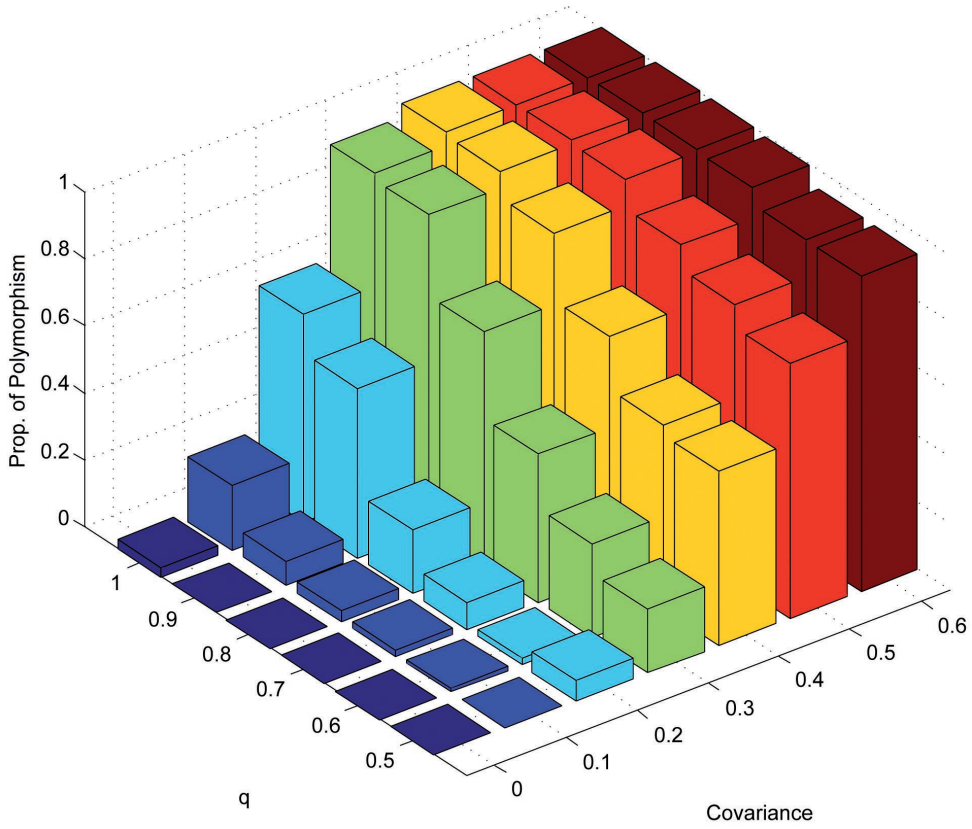


Figure 3.3.: Proportion of genetic polymorphisms of two specialists as evolutionary end-point. The other possible end-point is a perfect bet-hedging strategy with (z_1, z_2)=(0,1) or (1,0). Both a positive correlation between z_1 and z_2 and an asymmetric initial q make a genetic polymorphism as end-point more likely. With strongly asymmetric q already a small covariance shifts the outcome distribution towards the genetic polymorphism. Parameter values are summarised in Table 3.1.

In the present case we use $q = 0.8$ and a relative covariance of 0.4 and determine the proportions of the alternative end-points for different mutation rates between 0.1 and 0.001 and different population sizes between 500 and 20000. The results are presented in Figure 3.4. We see that:

- For populations smaller than 5000 individuals we have a strong dependence of the frequency of the different evolutionary end-points on the mutation rate. The proportion of polymorphisms of the two specialist increases with mutation rate. The smaller the population size, the stronger is this effect. An intuitive explanation for this dependence could not be found so far. Simultaneously, the proportion of polymorphisms decreases with population size. This is because small population sizes make a polymorphism less protected because the sub-populations are more vulnerable to stochastic effects. In the extreme case of a population size of 100 individuals polymorphisms are always only very short-lived and a polymorphic end-point is never attained.

- For populations of 10000 individuals or more the outcome does not depend on the mutation rate anymore.

Therefore we used a population size of 10000 individuals in the simulations presented in section 3.1.2. Biologically realistic mutation rates of 10^{-4} to 10^{-5} would have been too computationally demanding, but considering the fact that for population sizes ≥ 10000 the outcome is unchanged over two orders of magnitude, it seem reasonable to assume that the results presented here are valid for even smaller mutation rates.

3.2. Pure Plasticity

In this section the simulation results obtained for the model where only plasticity is allowed to evolve are presented. This means that the weighting factor of the switching device, a, is fixed to 1. The analytical results presented in section 1.3.3 reveal that for equiprobable environmental conditions ($p = 0.5$) we get the singular point $(z_1, z_2) = (1 - r, r)$, where r is the reliability of the environmental cue. This point is strongly convergence stable for all values of $r, c, d \in (0, 1)$. While the point is a continuously stable strategy for $c + d > 1$, it is a branching point in the opposite case. For $c + d < 1$ it can be invaded by mutants in all directions and coexistence is possible. The analytical treatment cannot predict how a population will evolve once it became dimorphic. To investigate this question a large amount of simulations with different values of r, c and d was performed. The cue reliability, r, was taken from $\{0.6, 0.7, 0.8, 0.9\}$, while trade-off parameter, c, and death probability, d, were varied in the interval (0,1) in steps of 0.2 such that the condition for disruptive selection ($c + d < 1$) was satisfied.

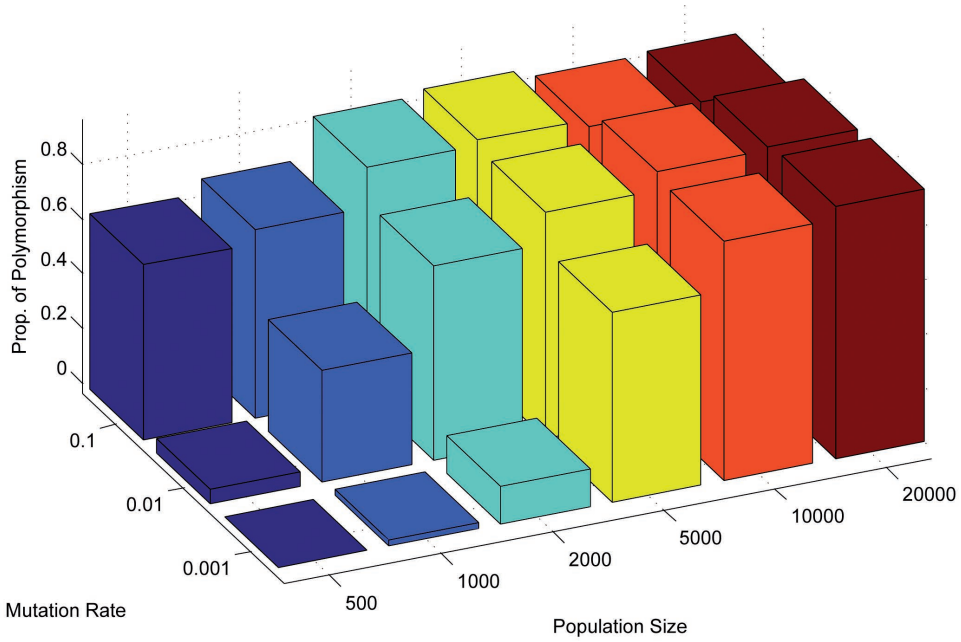


Figure 3.4.: Proportions of genetic polymorphisms among the evolutionary outcomes as a function of mutation rate and population size. The other possible outcome is a perfect bet-hedging strategy with (z_1, z_2)=(0,1) or (1,0). Simulations performed for $q = 0.8$ and covariance $= 0.4 \cdot$ variance. We see that for population sizes smaller than 5000 individuals there is a strong dependence of the outcome on the mutation rate, but for population sizes greater or equal 10000 the proportion of polymorphisms does not depend on the mutation rate. Other parameter values: Mutational step-size: 0.01; Mutational variance: 10^{-4}; $p = 0.5$, $d = 0.2$, $c = 0.6$.

3.2.1. Evolutionary Dynamics

For all parameter combinations the simulations confirmed that the population evolves to the neighbourhood of the point $(1-r, r)$ and then splits. But then a considerable variety of evolutionary trajectories and end-points is possible. Section 2.2.2 describes how these end-points can be detected by a program routine. Basically the evolutionary dynamics can be divided into two categories:

1. Branching takes place in the direction of the 45°-line. This situation is shown in Figure 3.5. Starting from the point $(1-r, r)$ (Figure 3.5(1)), the two sub-populations evolve along the line with slope 1 to the two opposite borders of the trait space ($z_2 = 1$ (2a) and $z_2 = 0$ (2b) respectively). Then again two main possibilities need to be distinguished:
   a) The sub-populations at (2a) and (2b) evolve towards the corners (1,1) and (0,0) of the trait space ((3a) and (3b)).
   b) One of the two sub-populations branches again giving raise to a third sub-population (Figure 3.6). While two sub-populations ((1b) and (2b)) evolve into the same direction as in case 1a of this enumeration, the third sub-population (2a) evolves towards the corner (0,1) (3a).
2. Branching takes place in the direction of the −45°-line. After branching at $(1-r, r)$ (Figure 3.7(1)) the two sub-populations evolve along the −45°-line in the direction of the corners (0,1) and (1,0). The corner (1,0) is always reached comparatively fast.

3.2.2. Evolutionary End-Points

Depending on the parameter values the different evolutionary trajectories lead to different evolutionary end-points. We can distinguish between three stable end-points that follow from the three types of branching described in the enumeration in section 3.2.1 and a cyclic end-point:

1. **Dimorphism of canalised specialists:** The situation described in point 1a of the enumeration above can lead to a stable end-point with two sub populations situated at (z_1, z_2)=(0,0) and (1,1). This corresponds to a genetic polymorphism of two genotypes displaying a canalised genotype-phenotype mapping. They do not use phenotypic plasticity.
2. **Trimorphism of canalised specialists and plastic genotype:** The situation described in point 1b of the enumeration above can lead to a stable genetic trimorphism of the two canalised genotypes (z_1, z_2)=(0,0) and (1,1) and the genotype $(z_1, z_2) = (0, 1)$. The latter one is using plasicity.
3. **Dimorphism of plastic and anti-plastic genotype:** The situation described in point 2 of the enumeration above can lead to a stable dimorphism

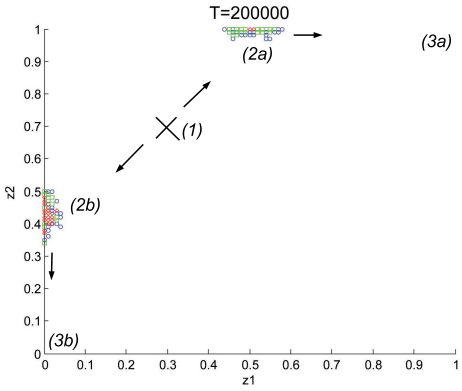


Figure 3.5.: Branching in the direction of the 45°-line in the model where only plasticity is allowed for ($a = 1$). Branching occurs at the singular point $(z_1, z_2) = (1 - r, r)$ given $c + d < 1$. At the branching point (1) the population splits into two sub populations which evolve to (2a) and (2b), respectively. Further evolution towards (3a) and (3b) may take place. Along this way one sub-population may die out or the development shown in Figure 3.6 may happen.

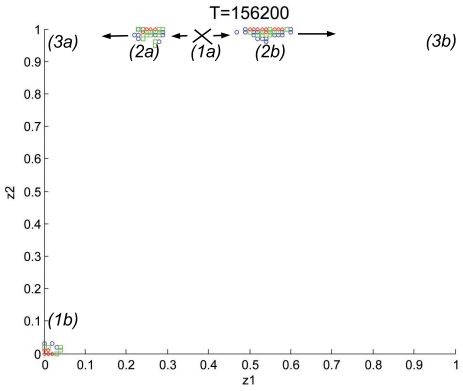


Figure 3.6.: In some cases a subsequent branching event (1a) happens to one of the two sub-populations from Figure 3.5. The newly created sub-populations at (2a) and (2b) evolve to a plastic genotype of extreme phenotypes (3a) and to a canalised phenotype (3b), respectively. The original sub-population, that did not participate in the second branching event evolves to the canalised genotype with the opposite phenotype (1b). Depending on parameter values, the sub-populations at (1b) and (2b) may die out before they reach the corners corresponding to the canalised phenotypes (0,0) and (1,1).

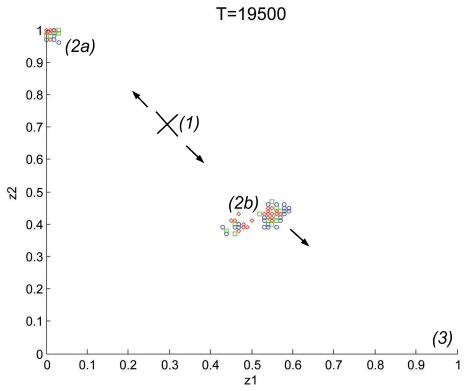


Figure 3.7.: Branching in the direction of the 45°-line. This development is equally possible as its orthogonal counterpart in Figure 3.5. The sub-populations separate at the branching point $(1-r, r)$ (1). One sub-population reaches the plastic genotype (2a) while the other one evolves in the direction of the anti-plastic genotype (3). Depending on parameter values the latter sub-population often dies out before it can reach point (3).

of sub-populations with the genotypes (z_1, z_2)=(0,1) and (z_1, z_2)=(1,0). These strategies both show phenotypic plasticity with two extreme phenotypes. The difference is that the genotype (0,1) always produces the phenotype that is perfectly adapted to the environmental condition indicated by the cue, while the genotype (1,0) always produces exactly the opposite phenotype. One could say that the second one totally mistrusts the cue.

4. **Cyclic end-point:** In many cases no stable end-point is reached. Instead cyclic end-points evolve. In this case, after branching, one of the sub-populations dies out after some time. This can either happen "somewhere along the way", before a corner of the trait space is reached, or when the sub-population under consideration is already in a corner of the trait space. Such extinction events can happen in all the scenarios listed in section 3.2.1. In the cases where two sub-populations exist (points 1a and 2 of the listing above) the surviving sub-population immediately evolves back to the branching point $(1-r, r)$ after the other one has died out. There branching occurs again and all the three possible developments are again possible. In the case of a trimorphism (point 1b of the listing in section 3.2.1) only the sub-populations evolving in the direction of (0,0) and (1,1) are vulnerable to extinction. If one of them dies out, the population at (0,1) branches again in the direction of the extinct sub-population to re-establish the situation before the extinction. If both sub-populations die out at the same time, the remaining population at (1,0) evolves back to the branching point and the various scenarios can unfold again.

3.2.3. Qualitative Results

In this section we will outline the general trend of how the parameters c, r and d influence which of the scenarios sketched above may take place.

The role of the trade-off parameter c

Low values of c correspond to a strongly convexly curved trade-off (see Figure 1.3 and equation 1.4). The simulations show that the smaller the value of c the faster the evolution towards the corners of the genotype-space proceeds after branching. The questions whether the corners can be reached before one of the sub-populations dies out and whether a stable end-point can be achieved is also influenced by c, but the other parameters play a stronger role here. In some cases, however, a smaller value of c can stabilise an end-point that was unstable for identical values of r and d and larger c.

It is intuitively clear that a strong trade-off (low values of c) speeds up evolution as it increased the strength of selection for more specialised phenotypes. Also the fact that a strong trade-off can stabilise a polymorhism of specialists can be understood intuitively because it gives the specialists advantage over intermediate phenotypes.

The role of the cue reliability r

The reliability of the cue predicting the future environment, r, directly influences how "well" a genotype displaying plasticity performs. A genotype that lies above the diagonal $z_1 = z_2$ in trait space ($z_1 < z_2$) responds to the cue and produces the phenotype that is better adapted to the predicted environmental condition, while a phenotype underneath the diagonal ($z_1 > z_2$) responds contrarily to the cue and always produces the phenotype that is better adapted to the condition not predicted. It follows that the higher the reliability of the cue the better performs the first type and the worse performs the second type. In the following the second strategy will be called anti-plasticity for convenience. For $r = 0.5$ the cue does not provide any information and the model is symmetric while in the case $r < 0.5$ the plastic and the anti-plastic strategies change roles. The simulations showed that r has a strong influence on the stability of the end-points presented in section 3.2.1:

- For $r = 0.9$ no stable evolutionary outcomes exist, independently of the values of c and d. In this case there is always a part of the population at or close to the point (0,1) and the other sub-populations detailed in section 3.2.1 die out before they reach a corner of the trait space.

- For $r = 0.8$ all runs with death rate $d = 0.1$ lead to stable evolutionary end-points. In this case the end-point with the two canalised phenotypes (0,0) and (1,1) is quite rare, while the trimorphism (0,1), (0,0) and (1,1)

is the outcome of most of the simulations. A coalition of plasticity and anti-plasticity of the extreme phenotypes (0,1) and (1,0) occurs in an intermediate number of cases.

- The trend for smaller r-values (that are still larger than one half) is as follows: The smaller the r-value the higher values of d lead to a stable end-point. For $r \leq 0.7$ the dimorphism of canalised phenotypes (0,0) and (1,1) is the most common end-point. A coalition of plasticity and anti-plasticity of the extreme phenotypes (0,1) and (1,0) is rare and a trimorphism is very rare.

If the environmental cue is highly reliable, the plastic genotype has a much higher average number of offspring than the two canalised specialists and the anti-plastic genotype. On the one hand, this explains why for high reliabilities always a sub-population of plastic genotypes evolves. On the other hand, it is intuitive that in such a case no stable end-points can be attained, as the average sizes of other sub-populations are small and they are thus vulnerable to extinction (due to a series of years with the environmental condition they are not adapted to).

For $r > 0.5$, which is always the case in our studies, the anti-plastic strategy produces an correctly adapted phenotype in on average less years than a canalised genotype and in much less years than the plastic genotype. In a dimorphism of the canalised specialists the two sub-populations have the same average size, as they have the same average number of offspring. In a dimorphism of plastic and anti-plastic genotypes, however, the sub-population following the anti-plastic strategy is always smaller, as its individuals have a lower average number of offspring. Given that the smallest sub-population is most prone to extinction, it is understandable that the "plasticity−anti-plasticity"-end-point is less frequent than the end-point including the canalised genotypes. Furthermore, it is intuitive that this effect becomes stronger for higher cue reliabilities.

The role of the death probability d

The death probability, d, plays an important role in the stability of polymorphic populations. This is intuitively clear because a small d means that settled individuals have a high chance to live for several years and therefore to produce offspring under both environmental conditions. On the other hand if d is close to 1, a genotype that only produces offspring of one phenotype has a high risk of extinction in a series of years favouring the other phenotype. The death probability determines how "protected" a polymorphism is (Warner and Chesson, 1985; Ellner and Hairston, 1994). The simulations show that:

- Values of $d \geq 0.5$ make the evolutionary development unstable, regardless of the parameters c and r. The larger the value of d, the shorter are the evolutionary cycles.

- For d=0.7 the sub-populations do not even reach the borders of the trait space before one of them goes extinct. The remaining sub-population evolves back to the branching point where it experiences disruptive selection again.

- For $d = 0.3$ the dimorphism (0,0) and (1,1) is stable for $r = 0.6$ and $r = 0.7$, while the other polymorphisms described in section 3.2.1 are not always stable.

- For $d = 0.1$ all end-points are stable given $r \leq 0.8$.

Conclusion

In conclusion one can say that regardless of the values of c, r and d, both ways of branching, in the 45°-direction (see Figure 3.5) and the $-45°$-direction (see Figure 3.7) do occur. For how long the polymorphisms persist after branching and whether they reach a stable end-point in the corners of the trait space does depend on all three parameters. Generally, branching in the 45°-direction is more frequent than in the orthogonal direction and polymorphisms produced by such a branching event exist for a longer time. Whether after branching in the 45°-direction a second branching event occurs that gives raise to a trimorphism depends on r. If the environmental cue is highly reliable, then these trimorphisms are frequent.

The death rate plays an important role in the maintenance of polymorphisms. A small value of d protects a sub-population against a succession of years their offspring is not well adapted to. To examine whether this really is the crucial feature explaining the influence of d on the outcome of the simulations, we ran simulations where the environmental conditions strictly alternate. The results of this investigation are presented in the following section.

3.2.4. Comparison With the Case of Strictly Alternating Environments

In the preceding section we saw that many cases with small d led to the establishment of stable polymorphisms, while - all other parameters being equal - no stable end-points could be found with higher values of d. Intuitively one would expect that small death rates, which correspond to a large generation overlap, protect the specialist phenotypes from extinction by giving them a chance to survive a succession of unfavourable years and to reproduce at least occasionally under optimal conditions. Small death rates protect against the stochasticity in nature. If this is the only way how d influences the dynamics then simulations with stricly alternating environmental conditions should also lead to stable end-points in cases with higher values of d. Here we investigate such simulations. Alternating environmental conditions should of course only help the dimorphic

coalition of two canalized phenotypes. Genotypes displaying plasticity still experience stochasticity in nature because the cue can be wrong. A series of years with wrong cues could still drive these genotypes to extinction if d is not sufficiently small.

To investigate the role of environmental stochasticity we performed similar simulations as in section 3.2.3 but now with strictly alternating environmental conditions 1 and 2. The results are as follows:

- For $r = 0.9$ the results are qualitatively similar to those with random environmental conditions. No stable end-point exists regardless of the values for c and d.

- For $r = 0.8$ the results are also similar to the general model. In some cases the time to extinction takes longer but stable end-points can be found for exactly the same parameter values.

- For $r = 0.7$ and 0.6 strong deviations from the results in section 3.2.3 can be observed. In most cases the simulations with alternating environmental conditions resulted in a polymorphism of the two canalised phenotypes for all values of d that satisfy $c + d < 1$. By contrast, in the case with random environmental conditions this was only possible for $d = 0.1$. As expected, the end-point of the plastic and the anti-plastic phenotype was stable under the same conditions in both investigations.

These results seem to be inconsistent as in some cases ($r = 0.6$ or 0.7) the strictly alternating environmental conditions help to establish a stable end-point for larger values of d, while in other cases ($r = 0.8$ or 0.9) they do not. One has to bear in mind that the scenario with strictly alternating environmental conditions only eliminates the stochastic variations that are due to the environment. But stochasticity that is due to the random allocation process at the settling stage can still lead to stochastic extinction. Alternatively, these results could be explained as follows: As we have seen in section 3.2.3, depending on r, evolution either favours a dimorphism with (0,0) and (1,1) or a trimorphism with a third sub-population at the plastic strategy (0,1). For low values of r the first situation is favoured, while larger values of r favour the second situation. If one examines the result presented in the current section in more detail, one can find that in cases where the trimorphism is favoured in the system with random environmental conditions, strictly alternating environments cannot help to establish stability. In situations where the original system favours the development of the dimorphism of canalised genotypes, however, the alternating environments make the outcome independent of d. This finding can to some extend be understood intuitively. The third sub-population at (0,1) makes that a sub-population at (0,0) or (1,1) even in its good years has a competitor that is equally fit (given that the cue is correct). A succession of several years in which the environmental cue is correct, can drive a sub-population at (0,0) or (1,1) to extinction, as it experiences strong

competition for the empty patches even in the years it produces a large amount of offspring. The protection mechanism from which a dimorphism of canalised phenotypes benefits does not apply in such a case.

3.3. The Complete Model

An overview of the analytical results obtained for the complete model is given in section 1.3.4. For intermediate values of the cue reliability, r, there exists a genotype that combines a bet-hedging strategy with phenotypic plasticity (intermediate a) that corresponds to a convergence stable fitness maximum that cannot be invaded by any other genotype. For example, for a population characterised by $t = 0$, $e_1 = -0.25 = -e_2$, $c = 0.6$, $d = 0.6$ in an environment with $p = 0.5$, $r = 0.75$ we find the optimal genotype to be $(z_1, z_2, a) = (0, 1, 0.73)$. The threshold value, t, was fixed for the analytical treatment but this assumption is relaxed in the simulations. The following section presents the simulation results for the parameter values listed above. Simulations are started either from a bet-hedging strategy ($a = 0$) or from pure plasticity ($a = 1$). Section 3.3.2 deals with the question whether polymorphisms can be observed in the model.

3.3.1. The Evolutionary Trajectories

We study evolution starting from two different initial conditions:

- **Initial genotype** $(z_1, z_2, a) = (0.5, 0.5, 0)$**:** Simulations were started with a genotype that is initially only sensitive to internal noise and not to the environmental cue but at the beginning produces a single generalist genotype. The simulations show that, depending on stochastics, two different evolutionary end-points can be reached. About 50% of the runs lead to the end-point predicted by the analytical results, $(z_1, z_2, a) = (0, 1, 0.73)$. In the other half of the simulations the phenotypes evolve to $(z_1, z_2) = (1, 0)$, while a stays close to 0. An example of the first case is shown in Figure 3.8. After about 1000 time steps, the initial population $(z_1, z_2, a) = (0.5, 0.5, 0)$ evolves towards smaller values of z_1 and larger values of z_2. At the same time the value of a slowly increases. As soon as the phenotpic trait values reach the opposite extremes, at $T \approx 5000$, the a-value starts to increase much faster until it reaches at $T \approx 13000$ the neigborhood of 0.73 where it stabilises. In these simulations the threshold value, t, stays close to 0. A run illustrating the second scenario is shown in Figure 3.9. There the trait values of z_1 and z_2 evolve to 1 and 0, respectively. Note the exchanged roles of z_1 and z_2 in Figure 3.8 and Figure 3.9. Remember that for pure bet-hedging the strategies $(z_1, z_2) = (0, 1)$ and $(1, 0)$ are equivalent in the sense that they correspond to the same average number of offspring and that they have the same probability of emergence. In the plasticity model, however, the point (z_1, z_2)=(1,0), corresponds to a anti-plastic genotype.

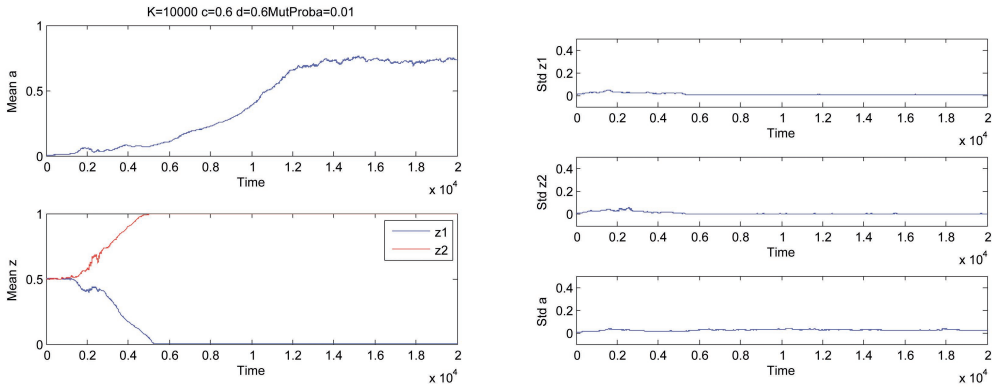
It always produces the ("wrong") phenotype that was not indicated by the cue. The population is trapped at a local fitness peak from which it could only escape if z_1 and z_2 switched roles.

- **Initial genotype** $(z_1, z_2, a) = (0.5, 0.5, 1)$**:** Simulations were started with a genotype that is initially only sensitive to the environmental cue and not to internal noise but in the beginning produces a single generalist genotype. Here only one end-point is possible. The observation of several simulation runs confirms that for the parameter values mentioned above the evolutionary end-point $(z_1, z_2, a) = (0, 1, 0.73)$ is reached by the dynamics. An exemplary run with a mutation rate of 0.01 and a population size of 10000 individuals is presented in Figure 3.10. By following the mean values of z_1, z_2 and a (which is sufficient because the small standard deviation in these parameters indicate that the populations is (quasi-) monomorphic) we see that the population evolves to the singular point $(z_1, z_2) = (1 - r, r)$=(0.25,75) in the plane corresponding to the model for phenotypic plasticity ($a = 1$). After a little more than 3000 time-steps this point is attained and then the population experiences a strong selection for smaller values of a, while at the same time z_1 and z_2 evolve to more extreme values to reach the borders of the trait space 0 and 1. After about 5500 generations the mean value of a reaches 0.73 and stays in the neighbourhood of this point for the rest of the simulation. The mean value over the last 500 time steps of the population wide mean of a is 0.7297 with a standard deviation of 0.0036. The mean value over the last 500 time steps of the population wide standard deviation in a is 0.0207. Thus, the analytical results are well met. Figure 3.10(b) shows that the population wide mean value and standard deviation of the threshold value of the switching device, t, stay very close to zero during the whole simulation. The assumption $t = 0$ used for the analytical treatment is thus justified.

3.3.2. Polymorphisms

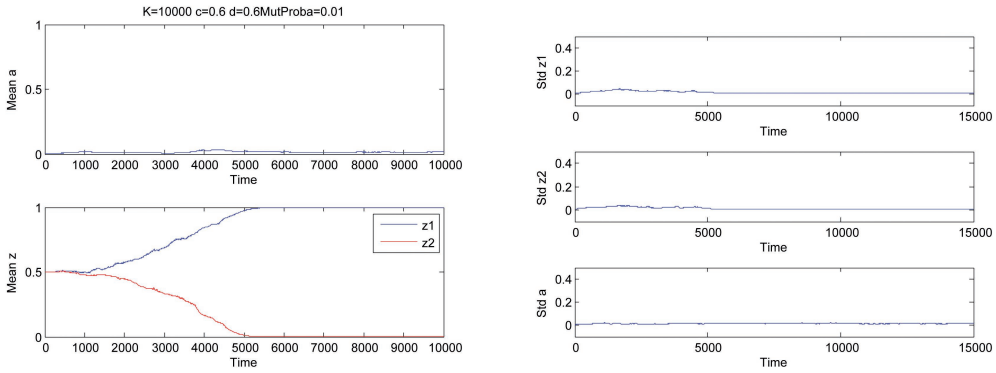
A question that came up at the presentation of the deterministic model was whether the population would experience disruptive selection in z_1 and z_2 at some point during evolution. The following list summarises analytical and simulation results.

- Using the parameter values from above, the analytical results predict that a population with z_1 and z_2 at the intersection of the two isoclines experiences disruptive selection at $a = 0.88$. A large amount of simulations was performed in order to investigate whether this disruptive selection could lead to evolutionary branching and to the establishment of a genetic dimorphism. In none of the cases a genetic dimorphism was observed. It seems that a evolves to smaller values before branching of the populations



(a) (b)

Figure 3.8.: Evolution in the complete model where starting with a genotype only sensitive to developmental noise leads to a mixture of bet-hedging and plasicity. Panel (a) shows the population wide mean values of the traits and panel (b) the standard deviations. The threshold, t (not shown), stays always close to 0.



(a) (b)

Figure 3.9.: Evolution in the complete model where the phenotypes of a bet-hedger evolve in a direction that makes the development of plasticity impossible. Panel (a) shows the population wide mean values of the traits and panel (b) the standard deviations.

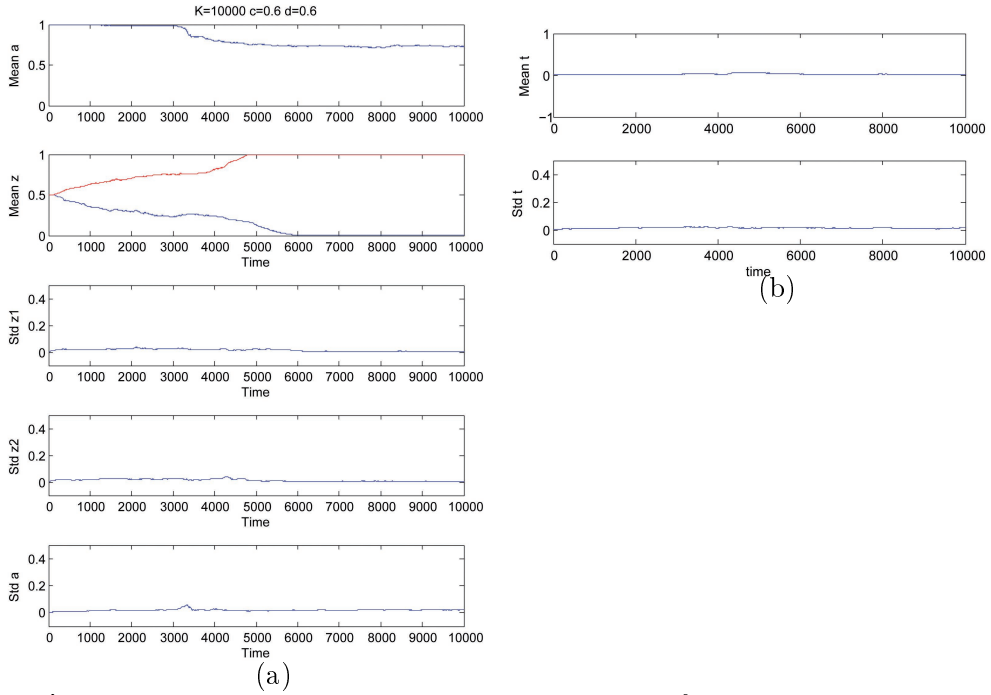


Figure 3.10.: Evolution in the complete model starting form a generalist genotype that is only sensitive to the environmental cue (plasticity) and not to internal noise (bet-hedging). Evolution starting from (z_1, z_2, a)=(0.5,0.5,1). The population wide mean values of (z_1, z_2) evolve to $(1-r, r)$ while the mean a-value equals 1. Then the value of a gradually evolves to 0.73 while z_1 and z_2 evolve to 0 and 1 respectively. Since the population wide standard deviations for the different traits stay close to zero, the mean values correspond to a (quasi-)monomorphic population of individuals with trait values close to the mean values. Graph (b) confirms that the assumption for the numerical treatment, $t = 0$, was justified as t stays always close to zero.

can take place. Even when the mutation probability in a was 5 times smaller than in z_1 and z_2 a dimorphism did not evolve. In some of the simulations, however, a slightly increased population wide standard deviation was detected in z_1 and/or z_2 at the time where $a \approx 0.88$, which could be a footprint of disruptive selection.

- Analytical results show that for a combination of a strong trade-off and strong generation overlap ($c + d < 1$) the singular point is a branching point for all values of a. We investigate the case of $d = 0.3$ with all other parameters being equal as above. Then the point $(z_1, z_2) = (1 - r, r)$ is a branching point in the plane $a = 1$. At the same time, however, selection favours smaller values of a. Numerical treatment could not reveal whether the evolutionary change in a would occur fast enough to prevent the establishment of polymorphisms. Simulations showed that sometimes temporal polymorphism occur, but they vanish after some time. In all cases the same end-point is reached as for $d = 0.6$, i.e. $(z_1, z_2, a) = (0, 1, 0.73)$. A series of six $z_1 - z_2$-phase diagrams illustrating a temporal polymorphism is shown in Figure 3.11. The population branches close to $(z_1, z_2) = (1-r, r)$ (Figure 3.11(b)). The two sub-populations slowly evolve into two opposite directions (c). After they reached the borders of the trait space (d) one of the two sub-populations dies out (e) and the population reaches the end-point $(z_1, z_2, a) = (0, 1, 0.73)$ (f). If branching does not occur, the evolution is similar to the case where $d = 0.6$, except that it takes longer until the end-point is reached (smaller d means a slower population turnover).

3.4. Discussion

Simulations as back-up for the analytical treatment

The adaptive dynamics approximation makes several assumptions. (1) Mutations occur in small, discrete steps, (2) they have to be sufficiently rare so that the population is at a population dynamical equilibrium before new mutants occur and (3) resident populations have to be sufficiently large such that stochastic effects can be neglected. In most biological scenarios, however, one or more of these assumptions are unlikely to be fulfilled. A.D. has often been criticised for these restrictions and its applicability to realistic situations has been controversially discussed. A critical discussion of the A.D. approach is given in a review by Waxman and Gavrilets (2005).

Most studies that apply the A.D. framework to a biologically motivated model back-up their analytical findings with individual-based simulations that allow for the relaxation of the A.D. assumptions. One of the main targets of the simulations presented in this work is to test the analytical results by Rueffler et al. (in prep.a, in prep.b). For the simulations we use:

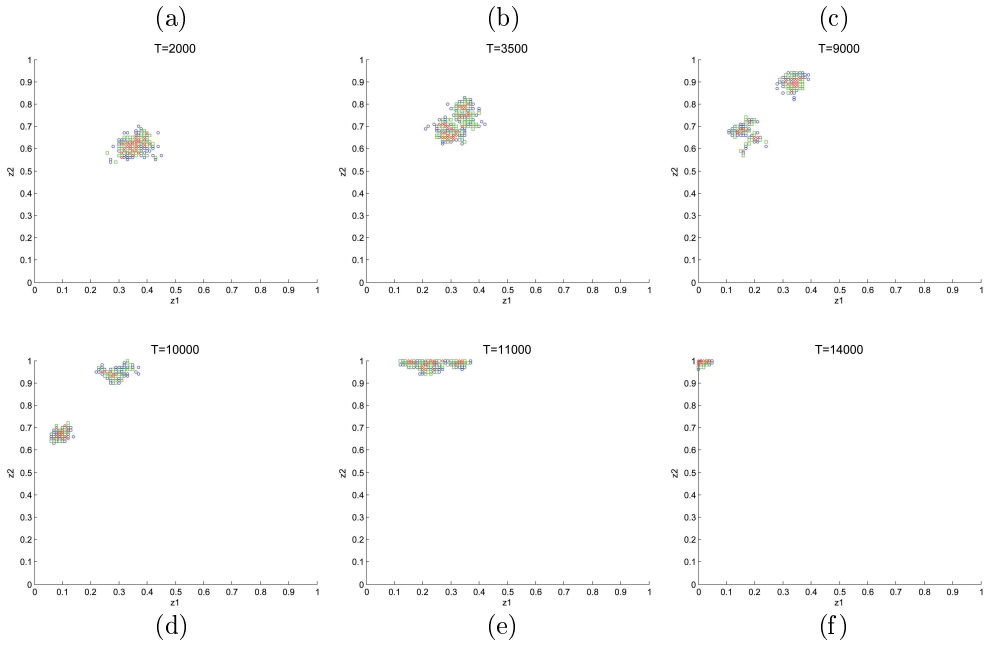


Figure 3.11.: Example for a temporal dimorphism occuring in the complete model when $c + d < 1$. Phase diagrams in the $z_1 - z_2$-space for different points in time. Note that with increasing time the a-value decreases from $a = 1$ to an intermediate equilibrium value. Parameter values: $d = 0.3$, $c = 0.6$.

- Mutational step sizes that are drawn from a normal distribution.

- High mutation probabilities, mostly 0.01 per trait, generation and individual. This leads to a situation where mutants occur frequently and no population dynamical equilibrium is reached before a new mutant occurs.

- Comparatively small population sizes of mostly 1000 or 10000 individuals.

The simulation results are always consistent with the analytical findings and suggest that the deterministic approach gives the correct predictions even tough the assumptions are violated. However, there is no formal proof for this and situations could exist in which the A.D. assumptions become crucial.

Furthermore, simulations allow to analyse situations where no analytical results could be obtained. In the present model simulations were necessary to investigate the long-term dynamics of polymorphic populations and cases with mutational covariance.

The Bet-Hedging Model

In the first part of our studies we consider genotypes that produce offspring of two different phenotypes in an environment that consists of two randomly alternating environmental conditions. The limiting case where the two alternative phenotypes are equal corresponds to a canalised genotype. We are interested in situations where coexistence is possible for canalised genotypes. For this we need a combination of sufficient generation overlap (see Warner and Chesson (1985) and Ellner and Hairston (1994) for a discussion of the role of generation overlap for coexistence) and a strong trade-off between the performances under the two environmental conditions.

One of the main motivations for this project was the extension of the work by Leimar (2005). His objective was to compare the relative likelihood for the emergence of a bet-hedging strategy and a genetic polymorphism. We extend Leimar's study in three directions:

1. Leimar only considers bet-hedging genotypes where the two alternative phenotypes are specialised to equal degree in the two environments. We also allow for bet-hedgers whose alternative phenotypes are specialised to different degrees in the two environments. In the following the first ones will be called symmetric bet-hedgers and the second ones asymmetric bet-hedgers.

2. Leimar restricts his analysis of the model to an equal initial frequency of the two alternative bet-hedging phenotypes ($q = 0.5$). We also consider cases where the two phenotypes of a bet-hedger are initially not produced in equal proportions ($q \neq 0.5$).

3. Leimar focuses on what happens at the singular point. We also consider how the evolution proceeds after a possible phenotypic diversification at the singular point.

Leimar compared the strength of selection for a monomorphic symmetric bet-hedging genotype with the strength of selection for a polymorphism of two canalised genotypes. He finds that the selection for bet-hedging is always stronger. Our results suggest that with the above extension this conclusion need not to be true. We find:

1. As soon as the two alternative bet-hedging phenotypes are initially not produced in equal proportions, the strongest selection is not anymore for symmetric bet-hedgers but for certain asymmetric bet-hedgers.

2. Coexistence (and thus the emergence of polymorphisms) is not only possible for canalised genotypes, but also for a large class of asymmetric bet-hedgers. The more the initial production of alternative phenotypes is skewed, the less asymmetric a bet-hedger genotype needs to be so that it can coexist with the resident. At some degree of initial deviation from equal proportions the strongest selection is for asymmetric bet-hedging genotypes which can coexist with the resident and the analytical results predict a dimorphism of bet-hedging strategies to evolve.

The simulations confirmed these results but showed that such dimorphisms are only temporally stable. The two bet-hedging sub-populations evolve to genotypes that produce more and more specialised phenotypes; one in the direction where the first phenotype specialises for environment 1 and the second for environment 2 and one in the opposite direction. After some time one of the two sub-populations dies out. This is due to the fact that symmetric bet-hedgers, which always evolve in the long run, do not benefit from the same protection mechanism as we described it for a polymorphism of canalised phenotypes in the results part but they are neutral if their phenotypes are equally specialised. Asymmetric bet-hedgers benefit from the protection mechanism to some degree. So the final result is a genetically monomorphic bet-hedging strategy.

In a second step we introduce a positive genetic correlation in the mutational increments of the two alternative phenotypes. This means that it is more likely that the trait values of both phenotypes mutate in the same direction, either both to higher or both to smaller values. In the case of positive correlation the dimorphism of two canalised specialists is an end-point of evolutionary dynamics in a certain fraction of the simulations. The rest of the simulations leads to the establishment of a monomorphic bet-hedging strategy with two alternative specialist phenotypes, as all of the simulations in the case without mutational correlation did. Increasing positive correlation increases the probability that the basin of attraction of the dimorphism of canalised specialists is entered.

Originally the predominant idea was that temporal fluctuations favour bet-hedging over genetic fluctuations (Seger and Brockmann, 1987). Ellner and Hairston (1994) find for our model (actually for a broader class of models) that every genetic polymorphism can be invaded by a bet-hedging genotype producing both phenotypes present in the polymorphism. Leimar (2005) shows that, starting from the singular point, selection is always strongest towards a bet-hedging strategy. In nature, however, genetic polymorphisms are commonly observed, while there are only few examples for bet-hedging strategies. This raises the question why we do not observe bet-hedging more often if it is strongly favoured over genetic polymorphisms?

We show that with some constraints on bet-hedging genetic polymorphisms are more likely to evolve and that, once they have evolved, they can be evolutionary stable. This could be an explanation for the discrepancy between the previous results and the biological observations. In our model genetic polymorphisms are favoured by genetic correlation between the traits for the alternative phenotypes and by asymmetric initial proportions of the two phenotypes. Once a polymorphism of the two canalised specialist genotypes is established, it is stable given limited mutational step sizes. Even though a bet-hedging strategy of the two phenotypes present in a genetic polymorphism could outcompete the genetic polymorphism (as Ellner and Hairston (1994) show), it cannot evolve except if one assumes extremely large mutational steps because a wide fitness valley lies in between these strategies.

Whether mutational steps are small or not is a controversely discussed issue. However, it is not unrealistic that a bet-hedging device needs a complex physiological and developmental-biological machinery. We think that genetic correlations are a good way to give the genetically canalised genotypes some "headstart" over bet-hedging genotypes without regarding the actual genetics and biological machineries. Also the assumptions of small mutational steps (actually we draw the mutations from a Gaussian distribution, so big mutational steps can occur with a small probability) seem reasonable in the light of complex adaptations necessary for a bet-hedging strategy.

Pure Plasticity

In section 3.2 we investigate the evolutionary dynamics in a situation where an environmental cue predicts (with some certainty) the environmental condition of the next season. Following this cue a genotype can produce one out of two possible phenotypes. Analytical results show that a singular point exists whose values depend on the reliability of the environmental cue. The singular point is an evolutionary stable strategy for a combination of weak trade-off and short generation overlap. The simulations investigate parameter combinations where the singular point is a branching point. The simulation results show that different evolutionary outcomes are possible, depending on the generation overlap, the strength of the trade-off and the reliability of the environmental cue. We find

cyclic end-points as well as stable polymorphisms. Stable polymorphisms include a dimorphism of the canalised genotypes producing specialised phenotypes and a dimorphism of a plastic genotype producing the two specialised phenotypes according to the cue and a plastic genotype producing always the phenotype not suggested by the cue. The latter polymorphism seems very counter-intuitive but, similar to two canalised phenotypes, the plastic genotype producing the phenotype not predicted by the cue is to some degree protected against extinction by high relative growth rates in years in which the cue is wrong (and the other genotype thus only has a very small amount of offspring). Also a stable trimorphism of the two specialised canalised genotypes and the plastic gentoype producing specialised phenotypes can be observed. The role of the different parameters we observe can be understood as follows:

- A high reliability of the environmental cue favours evolutionary end-points that include the plastic genotype, while the dimorphism of two canalised genotypes is favoured for low cue reliabilities. This is intuitive as a reliable cue means that the offspring of plastic genotypes is well adapted in most of the years.

- An increasing death rate influences the stability of the end-points negatively. A low death rate, corresponding to a high generation overlap, helps to protect the members of a polymorphisms against extinction. This can be understood considering the storage effect of generation overlap explained by Warner and Chesson (1985).

- The strength of the trade-off determines the strength (and thus the speed) of selection towards specialised phenotypes. Except for the speed of the evolutionary dynamics it has comparatively little influence on the evolutionary outcome.

For a comparison of these results with empirical data it is important to find species with life cycles similar to the one in our model and that employ a similar kind of plasticity as defined in our model.

In nature phenotypic plasticity is a widespread phenomenon. Many species display phenotypic plasticity. Basically one has to distinguish between plasticity as a response to environmental constraints (e.g. an organism stays small because it lacks nutrients to grow to normal size) and plasticity as an adaptation to the environment. There are countless examples for phenotypic plasticity as an adaptation ranging from plant's production of small thick leafs to reduce vaporisation in dry periods to the production of normal or persister eggs by Brine shrimp (*Artemia salina*) in response to salinity. Every adaptation of physical or behavioural characteristics that is not induced by a specific allele at a gene locus can be seen as phenotypic plasitcity.

The definition of phenotypic plasticity that is used in our model, however, is very restrictive. First, the adaptation only happens once in early development

(as this is the only time selection acts in our model) and, second, rather than just responding to the current environment, the plasticity uses a cue that predicts the coming environment when development is finished and selection acts. This cue can, for instance, be interpreted as the current environmental condition predicting the future environment ("if it is dry now it might be dry when I am (or my offspring is) born").

Examples where adaptation happens in early development include several species of fish. Snorrason and Skúlason (2004) study fish that colonised lakes in the northern countries that where freshly formed after the last glacial period. They bring examples of species with low genetic variance that produce morphologically very different phenotypes adapted to different foraging strategies. In this example, however, it is unlikely that there exists a specific environmental cue. The different resources are more or less stable and the purpose of adaptation is rather to avoid intraspecific competition than to adapt to changing environmental conditions.

One of the species named above, Brine shrimp, would be an example of a species that uses a cue (the current salinity) to predict future environment (will the lake dry out or not). In this case, however, the population dynamics is very different to the one described in our model. In these shrimps the plastic trait is the type of egg determining hatching-time. Hatching can either as soon as possible or, with a delay, after a dry period.

In conclusion one can say that we had difficulties to find well studied examples of species with similar population dynamics as described by our model that use an environmental cue to produce offspring adapted to changing environmental conditions. Another problem is that our results are obtained for clonally reproducing species and the generalisation to sexually reproducing organisms is non-trivial. Additionally, even if we find species that match well with the characteristics of our model, it will be very difficult to measure parameters as cue reliability and strength of trade-off which determine the outcome that our model predicts.

Nonetheless, we can draw important conclusions that should hold true in more general cases. For weak trade-offs a monomorphic population that, depending on the cue reliability, shows some degree of plasticity is expected to evolve. For strong trade-offs and sufficient generation overlap we expect a genetic variance in the population to evolve. For intermediate cue reliabilities a polymorphism of canalised specialists is an alternative evolutionary scenario to phenotypic plasticity. We expect that, similar to the bet-hedging case, already small amounts of correlation in the mutational step sizes of the alternative phenotypes strongly increase the likelihood that a dimorphism of canalized genotypes evolves.

Complete Model

For the complete model, where bet-hedging and plasticity are limiting cases produced by a switching device that processes internal noise (bet-hedging) and

environmental cues (plasticity) to determine the phenotypes of the offspring, we find that for intermediate cue reliabilities the most common evolutionary outcome is a genotype that combines elements of plasticity and bet-hedging. While this strategy will always be approached from an initially purely plastic strategy, this mixture cannot evolve if a bet-hedging strategy produces the phenotypes that correspond to the optimal adaptation for the environment not predicted by the cue in the plastic model. This, however, could be a peculiarity of our model.

Leimar et al. (2006) use a similar switching device that takes genetic and environmental information as input. For intermediate reliabilities of the environmental cue he also finds a mixture of the two forms of phenotype determination. This suggests that, in cases where cues are not perfectly reliable (which will always be the case in nature), it is advantageous to rely on more than one mechanism in order to adapt to the environment.

Limitations of our Study and Perspective

In our model a genotype can only produce two different phenotypes and there are only two possible environmental conditions. A possible extension would be to allow a genotype to produce a distribution of phenotypes and to use a continuous distribution of environmental conditions. Sasaki and Ellner (1995), however, show that stable phenotype distributions are always discrete for environmental distributions with bounded support. It would be interesting to generalise our results to a broader class of models including more general environmental and phenotype distributions as Sasaki and Ellner (1995) use them but also more general population dynamics. A start for the second point could be the model by Ellner and Hairston (1994) where seeds, diapausing eggs or adults can be the long lived state.

A very important feature that strongly influences the evolution in our model is the limited patch number. All preliminary results we refer to (Chesson and Warner, 1981; Warner and Chesson, 1985; Ellner and Hairston, 1994; Sasaki and Ellner, 1995) have a similar characteristic in their model. The selection for the limited patch or habitat number only depends on the frequency of the different types. At this stage the phenotype does not play a role for selection. The frequency dependent selection that protects polymorphisms in our model does depend on this feature of the model and does not necessarily exist in models that do not have this feature. It would be interesting to study whether our results could be generalised to different sources of frequency dependent selection as it exists in other systems (e.g. different resources).

In our model the death rate is constant over time and equal for all adults. Warner and Chesson (1985), however, stress that phenotype dependent death rates can totally change the results. The storage effect induced by the generation overlap will be reduced and then conditions for coexistence would thus be more restrictive.

For the plasticity model we present qualitative results how the frequency of dif-

ferent evolutionary outcomes depends on the parameter values. A quantitative analysis of many simulation runs would be useful here. In both the plasticity model and the complete model the incorporation of mutational correlations would be an interesting extension.

Our model does not include any spacial structure. Leimar (2005) and Leimar et al. (2006) show that spatially varying environments in combination with limited migration (or dispersal) can make an individuals genotype a cue for the environment its offspring will experience (if the offspring does not disperse too far it is likely to experience the same environment the parent was adapted to). In such a case a (spatial) genetic polymorphism is likely to evolve. This mechanism does not depend on genetic polymorphisms to be mutationally favoured as it is the case in our model and therefore is an alternative for explaining the large amount of genetic variation that is found in nature. We expect that in nature both mechanisms act together.

Finally, one should bear in mind that our model assumes clonal organisms. With sexual organisms the picture for polymorphic populations will be more difficult.

Bibliography

Avery, S. V. 2006. Microbial cell individuality and the underlying sources of heterogeneity. Nature Reviews Microbiology 4:577–587.

Bulmer, M. G., 1980. The Mathematical Theory of Quantitative Genetics. Clarendon Press.

Bulmer, M. G. 1984. Delayed germination of seeds: Cohen's model revisited. Theoretical Population Biology 26:367–377.

Butler, A. J. and Keough, M. J. 1981. Distribution of pinna bicolor gemelin (mollusca: Bivalvia) in south australia, with observations on recruitment. Transaction of the Royal Society of Southern Australia 105:29–39.

Chesson, P. and Warner, R. 1981. Environmental variability promotes coexistence in lottery competitive systems. The American Naturalist 117:923–943.

Cohen, D. 1966. Optimizing reproduction in a randomly varying environment. Journal of Theoretical Biology 12:119–129.

DeWitt, T. J. E. and Scheiner, S. M. E., 2004. Phenotpic Plasticity: Functional and Conceptual Approaches. Oxford University Press US.

Dieckmann, U. and Law, R. 1996. The dynamical theory of coevolution: A derivation from stochastic ecological processes. Journal of Mathematical Biology 34:579–612.

Dubnau, D. and Losick, R. 2006. Bistability in bacteria. Mol Microbiol 61:564–572. URL `http://dx.doi.org/10.1111/j.1365-2958.2006.05249.x`.

Ellner, S. and Hairston, N. G. Jr. 1994. Role of overlapping generations in maintaining genetic variation in a fluctuating environment. The American Naturalist 143:403–417.

Geritz, S. A. H., Gyllenberg, M., Jacobs, F. J. A., and Parvinen, K. 2002. Invasion dynamics and attractor inheritance. Journal of Mathematical Biology 44:548–560.

Geritz, S. A. H., Kisdi, É., Meszéna, G., and Metz, J. A. J. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evolutionary Ecology 12:35–57.

Geritz, S. A. H., Metz, J. A. J., Kisdi, É., and Meszéna, G. 1997. Dynamics of adaptation and evolutionary branching. Physical Review Letters 78:2024–2027.

Hopper, K. R. 1999. Risk-spreading and bet-hedging in insect population biology. Annu Rev Entomol 44:535–560. URL `http://dx.doi.org/10.1146/annurev.ento.44.1.535`.

Leimar, O. 2005. The evolution of phenotypic polymorphism: Randomized strategies versus evolutionary branching. The American Naturalist 165:669–681.

Leimar, O. 2008. Environmental and genetic cues in the evolution of phenotypic polymorphism. Evolutionary Ecology .

Leimar, O., in press. Chapter *in* Elements of Adaptive Dynamics. Cambridge University Press, Cambridge, U.K.

Leimar, O., Van Dooren, T. J. M., and Hammerstein, P. 2006. A new perspective on developmental plasticity and the principles of adaptive morph determination. The American Naturalist 167:367–376.

Lewontin, R. C. and Cohen, D. 1969. On population growth in a randomly varying environment. Proceedings of the National Academy of Sciences USA 62:1056–1060.

MathWorks, T. 2007. Matlab 2007a. Natick, MA .

Mendel, G. 1866. Versuche über Pflanzen-Hybriden. Verhandlungen des naturforschenden Vereines, Abhandlungen, Brünn 4:3–47.

Metz, J. A. J., Geritz, S. A. H., Meszéna, G., Jacobs, F. J. A., and Van Heerwaarden, J. S., 1996. Adaptive dynamics: A geometrical study of the consequences of nearly faithful reproduction. Pages 183–231 *in* S. J. van Strien and S. Verduyn Lunel, eds. Stochastic and spatial structures of dynamical systems, Proceedings of the Royal Dutch Academy of Science. North Holland, Dordrecht, Netherlands; available at http://www.iiasa.ac.at/Research/ADN/Series.html.

Metz, J. A. J., Nisbet, R. M., and Geritz, S. A. H. 1992. How should we define 'fitness' for general ecological scenarios? Trends in Ecology and Evolution 7:198–202.

Rueffler, C., Svardal, H., and Abrams, P., in prep.*a*. The evolution of phenotype determination in a lottery model. "In preperation".

Rueffler, C., Svardal, H., and Abrams, P., in prep.*b*. The evolution of randomized strategies. "In preperation".

Rueffler, C., Van Dooren, T. J. M., Leimar, O., and Abrams, P. A. 2006. Disruptive selection and then what? Trends in Ecology and Evolution 21:238–245.

Sasaki, A. and Ellner, S. 1995. The evolutionarily stable phenotype distribution in a random environment. Evolution 49:337–350.

Seger, J. and Brockmann, H. J. 1987. What is bet-hedging? Oxford Surveys in Evolutionary Biology 4:182–211.

Simons, A. and Johnston, M. 1997. Developmental instability as a bet-hedging strategy. Oikos 80:401–406.

Smits, W. K., Kuipers, O. P., and Veening, J.-W. 2006. Phenotypic variation in bacteria: the role of feedback regulation. Nature Reviews Microbiology 4:259–271.

Snorrason, S. and Skúlason, S., 2004. Chapter. 10 *in* U. Dieckmann, J. A. J. Metz, M. Doebeli, and D. Tautz, eds. Adaptive Speciation. Oxford University Press.

Warner, R. and Chesson, P. 1985. Coexistence mediated by recruitment fluctuations: A field guide to the storage effect. The American Naturalist 125:769–787.

Waxman, D. and Gavrilets, S. 2005. 20 questions on adaptive dynamics. Journal of Evolutionary Biology 18:1139–1154.

A. Appendix

A.1. Programming

A.1.1. Main Program

In the following the commented Matlab source code of the main program used for the simulations. is given Table A.1 gives a description of the input parameters while Table A.2 explains the output variables.

```
function[amean,z1mean,z2mean,tmean,astd,z1std,z2std,tstd,gen100,params,...
    outc]=BetHedgingGen_2_4_f(genstart,mP,covz1z2,var_a_t,mS,p,d,amax,...
    c,r,tmax,nobreak,noplot)

%function[amean,z1mean,z2mean,astd,z1std,z2std,tmean,tstd,gen100,params,...
%   outc]=BetHedgingGen_2_4_f(genstart,mP,covz1z2,var_a_t,mS,p,d,amax,...
%   c,r,tmax,noplot)

%Function that simulates the evolution of a population in a variable
%environment following a model proposed by
%Rueffler, Svardal, and Abrams (in prep.a).
%This function allows for the development of bet-hedging and phenotypic
%plasticiy as mechanism of phenotype determination.
%Notation used in the description follows Rueffler et al. (in prep.a). A
%description of this function can be found in my diploma thesis available
%from: hannes.svardal@univie.ac.at
%
%A modified version of the function multrnd (avalable in the internet) is
%necessary for this function to run stable. It is necessary to delete the
%query asking wheter I==1 in the function multrnd.m. Otherwise
%numerical problems make that 1==1.00000000000000001 leads to an error.
%
%The function PolyOutcome.m is needed.
%
%For further information on this function contact:
%hannes.svardal@univie.ac.at
%
%Input arguments:
%
%genstart(mx5 matrix): Matrix containing in each line the genotypic values
%[z1 z1 a n t] where n is the number of Individuals having the
%genotype defined by [z1 z2 a t], z1 and z2 correspond to the two
%alternative phenotypes and a and t are respectively weighting factor and
%threshold of the switching device presented in section 1.2.2
%and in figure 1.4

```

Table A.1.: Input Arguments

Name	Dim	Description
genstart	mx4	Genotype matrix of the initial population. Each line containing (z_1, z_2, q,Number of individuals) for one sub-population.
mP	1x3	Probability per generation of a mutation in z_1/z_2, a, t.
covz1z2	2x2	Variance-Covariance matrix between z_1 and z_2.
var_a_t	1x2	Variance in a, t
mS	1x3	Mutational step size for z_1/z_2, a, t.
p	scalar	Probability that environmental condition 1 occurs in a certain year.
d	scalar	Death probability per year.
amax	1x2	Maximal number of offspring per individual 1st entry: environment 1, 2nd entry: env. 2.
c	scalar	Trade-off parameter.
r	scalar	Reliability of the environmental cue.
tmax	scalar	Maximal number of iterations the simulation runs.
nobreak	scalar	Suppresses abort of the program when end-point was detected if 1.
noplot	scalar	Suppresses graphical output if 1.
m ... number of different genotypes present in the population		

Table A.2.: Output Parameters

Name	Dim	Description
amean	1x$tmax$	Population wide mean of a for each time-step.
z1mean	1x$tmax$	Population wide mean of z_1 for each time-step.
z2mean	1x$tmax$	Population wide mean of z_2 for each time-step.
tmean	1x$tmax$	Population wide mean of t for each time-step.
astd	1x$tmax$	Standard deviation of a within the population for each time-step.
z1std	1x$tmax$	Standard deviation of z_1 within the population.
z2std	1x$tmax$	Standard deviation of z_2 within the population.
tstd	1x$tmax$	Standard deviation of t within the population.
gen100	1x ($tmax$/100)	Cell array containing whole genotype matrix for every 100 time-steps.
params	struct	Structure containing input parameters and total simulation time.
outc	scalar	Outcome of the simulation as determined by "PolyOutcome.m"
$tmax$ ... number of time-steps the simulation ran		

```
37 %The number of lines of genstart is the number of different
38 %genotypes that are present in the initial population.
39 %The (constant) number of patches equals the sum over all n.
40
41 %mP=[mPz mPa mPt]: Array containing the mutation probabilities in z1/z2, a
42 %and t. All probabilities are taken as equal if the input is a scalar.
43 %
44 %
45 %
46 %covz1z2 (2x2 matrix): Mutational Variance—Covariance Matrix between z1
47 %and z2
48 %
49 %var_a_t=[vara vart]: Array containing the variances in a
50 %and t. Variances are taken as equal if the input is a scalar.
51 %
52 %
53 %mS=[mSz mSa mSt]: Array containing the mutational stepsizes in z1/z2, a
54 %and t. All step sizes are taken as equal if the input is a scalar.
55 %
56 %p: probability that a year brings enviroment condition 1 ((1—p) is
57 %probability for env. cond. 2)
58 %
59 %d: probability that a patch is emptied in a year (death probability of the
60 %inhabitant)
61 %
62 %amax(1x2 array): array containing [alpha1 alpha2] where alpha1,alpha2 are
63 %the maximum numbers of offspring in years with env. cond. 1,2.
64 %values of amax must both be >>1
65 %
66 %c: trade off in offspring survival funtion
67 %
68 %tmax: Number of generations the simulation runs
69 %
70 %noplot(optional):supresses graphical output if 1
71 %
72 %Output:
73 %
74 %amean(1xtmax array): array containing the mean a—value of the pop for each
75 %generation t
76 %z1mean(1xtmax array): array containing the mean z1—value of the
77 %pop for each generation t
78 %z2mean(1xtmax array):array containing the mean z2—value of the pop
79 %for each generation t
80 %astd(1xtmax array):array containing the standard deviation of a of the pop
81 %for each generation t
82 %z1std(1xtmax array): array containing the standard deviation of z1
83 %of the pop for each generation t
84 %z2std(1xtmax array): array containing the standard deviation of z2
85 %of the pop for each generation t
86 %gen100(1x(tmax/100) cell obj): cell obj. containing the geotypes of whole
87 %population (gen) of every 100 generations
88 %params (structure object): containing the input parameters and the time
89 %elapsed between start and end of this function
90 %genstart(mx4 matrix): containing the inital population genstart
```

```
%outc: result of the simulation determined by *Outcome.m, where * stands
%for different prefixes, as different functions have to be employed in
%different situations as the possible outcomes depends on the parameter
%combinations regarded.
%The scalar outc is 0 if the outcome is undefined. The other values it can
%take are described in the different functions *Outcome.m.
%The function detOutcome_* determines whether an evolutionary end-point is
%reached.

%start timer
tic

%initialise the variable defining the outcome
outc=[];

% standard deviation in a,t is the square root of the variance in a,t
stda=sqrt(var_a_t(1));
% if var_a_t is a scalar the variances for a and t are both defined by this
% scalar
if length(var_a_t)<2
    stdt=sqrt(var_a_t(1));
else
    stdt=sqrt(var_a_t(2));
end

% assignment of the mutation probabilities
%if the respective entry of mP does not exsit the first entry is taken
mPz=mP(1);
switch length(mP)
    case 3
       mPa=mP(2);
       mPt=mP(3);
    case 2
       mPa=mP(2);
       mPt=mP(1);
    case 1
       mPa=mP(1);
       mPt=mP(1);
end

% assignment of the mutational step size
%if the respective entry of mS does not exsit the first entry is taken
mSz=mS(1);
switch length(mS)
    case 3
       mSa=mS(2);
       mSt=mS(3);
    case 2
       mSa=mS(2);
       mSt=mS(1);
    case 1
       mSa=mS(1);
       mSt=mS(1);
end
```

```
145
146
147
148
149
150 %determining the (constant) number of patches from the inital number of
151 %patches
152 K=sum(genstart(:,4));
153
154 %initalising inital population:
155 gen=genstart;
156
157
158 %if input noplot is not assigned, noplot is false
159 if nargin<13, noplot=0; end;
160
161 %if input nobreak is not assigned, nobreak is false
162 if nargin<12, nobreak=0; end;
163
164 % determine the seeds of the randomization algorithm used initially by the
165 % functions rand and randn (statistics toolbox)
166 seed=rand('twister');
167 seedn=randn('state');
168
169 %save parameters for output:
170 params=struct('K',K,'mP',mP,'mS',mS,'p',p,'d',d,'amax',amax,'c',c,...
171     'tmax',tmax,'seed',seed,'seedn',seedn,'covz1z2',covz1z2,...
172     'var_a_t',var_a_t, 'genstart',genstart);
173
174 %starting the recursion from 1st to last generation (tmax)
175 %-----------------------------------------
176 %start of the iteration of the time-steps
177 %-----------------------------------------
178 for t=1:tmax
179
180
181     %calculate mean values and standard deviations of a, z1, z2, t
182     amean(t)=sum(gen(:,3).*gen(:,4))/K;
183     astd(t)=sqrt((1/sum(gen(:,4)))*sum(gen(:,4).*(gen(:,3)-amean(t)).^2));
184     z1mean(t)=sum(gen(:,1).*gen(:,4))/K;
185     z2mean(t)=sum(gen(:,2).*gen(:,4))/K;
186     z1std(t)=sqrt((1/sum(gen(:,4)))*sum(gen(:,4).*(gen(:,1)-...
187         z1mean(t)).^2));
188     z2std(t)=sqrt((1/sum(gen(:,4)))*sum(gen(:,4).*(gen(:,2)-....
189         z2mean(t)).^2));
190     tmean(t)=sum(gen(:,5).*gen(:,4))/K;
191     tstd(t)=sqrt((1/sum(gen(:,4)))*sum(gen(:,4).*(gen(:,5)-tmean(t)).^2));
192
193    % ------------------------------
194    % graphical output
195    % ------------------------------
196     %if not supressed, plot mean values, refresh plot every 50 generations:
197     if ~noplot && t/50==floor(t/50)
198         figure(9)
```

```
199         subplot(2,1,1)
200         plot(1:t,tmean);
201         ylabel('Mean t');
202         axis([0 tmax -1 1]);
203         subplot(2,1,2);
204         plot(1:t,tstd);
205         ylabel('Mean t');
206         xlabel('time');
207         axis([0 tmax -0.1 0.5]);
208         figure(7)
209         subplot(5,1,1);
210         plot(1:t,amean);
211         ylabel('Mean a');
212         title(['K=',num2str(K),' c=',num2str(c),' d=',num2str(d)]);
213         axis([0 tmax 0 1]);
214         subplot(5,1,2);
215         plot(1:t,z1mean);
216         hold on
217         plot(1:t,z2mean,'r');
218         hold off
219         xlabel('Time');
220         ylabel('Mean z');
221         axis([0 tmax 0 1]);
222         subplot(5,1,3);
223         plot(1:t,z1std);
224         xlabel('Time');
225         ylabel('Std z1');
226         axis([0 tmax -0.1 .5]);
227         subplot(5,1,4);
228         plot(1:t,z2std);
229         xlabel('Time');
230         ylabel('Std z2');
231         axis([0 tmax -0.1 .5]);
232         subplot(5,1,5);
233         plot(1:t,astd);
234         xlabel('Time');
235         ylabel('Std a');
236         axis([0 tmax -0.1 .5]);
237         drawnow;
238     end;
239     % every 100th generation, do:
240     if  t/100==floor(t/100)
241         %save the whole population in the cell array gen100
242         gen100{floor(t/100)}=gen;
243
244         %if graphical output is not suppressed, plot the current population
245         %in a phase diagram of z1,z2
246         %Plotting described in plot_pip_f.m in appendix ??
247         if ~noplot
248             figure(8);
249             clf;
250             plot_pip_f(gen,0,t)
251
252         end
```

```
253       end
254
255       %-----------------------------------------
256       %Population dynamics
257       %-----------------------------------------
258
259     % Determing whether env 1 or env 2
260       env=1+(rand([1 1])>p);
261
262     % e is -1 if env 1 and +1 if env 2
263       e=sign(env-1.5);
264     % corr determines whether the cue is correct
265       corr=binornd(1,r);
266     % the cues are symmetric around 0. ec correct if is has the same sign as
267     % e
268       ec=(e*corr+(-e*(~corr)))*0.25;
269
270     % Determining the number of offspring for each genotype (Directly using
271     % the expectation value instead of drawing from a poisson distribution)
272     % This is the implementation of formula 1.5 of chapter 1
273
274     % Number of offspring per individual if only z1 was expressed
275      offsprz1=amax(env)*((1-e)/2+sign(e)*gen(:,1)).^(1/c);
276     % Number of offspring per individual if only z2 was expressed
277      offsprz2=amax(env)*((1-e)/2+sign(e)*gen(:,2)).^(1/c);
278     % Proportion of offspring that expresses z1
279      ProbaSm_t=normcdf((gen(:,5)-gen(:,3)*ec)./(1-gen(:,3)));
280
281     % Vector containing the total number of offspring of the different
282     % genotypes present in the population
283      newgen=gen(:,4).*(ProbaSm_t.*offsprz1+(1-ProbaSm_t).*offsprz2);
284
285     % Determining number of dying adults (=emptied patches) by drawing from
286     % binomial distributions for each genotype
287      dead=binornd(gen(:,4),d);
288
289     % The number of empty patches = Number of not filled
290     % patches in the last generation + number of died adults, but cannot be
291     % smaller than zero
292      Nemptypatch=max(0,K-sum(gen(:,4))+sum(dead));
293
294     % The probability that an individal with genotype X settles is
295     % propotional to the frequency of X in the offspring pool
296     % If sum(newgen)==0 (Only possible in the beginning if there are only
297     % poorly adapted strategies), division trough 0 has to be prevented.
298     if sum(newgen)≠0
299       SettleProba=(newgen./sum(newgen))';
300     % Correction of the settling probabilities to be sure that they sum up to
301     % 1
302      [temp I]=max(SettleProba);
303      SettleProba(I)=SettleProba(I)+(1.0-sum(SettleProba));
304    % Draw how many individuals of each genotype can settle
305      newgenSettle=(mnrnd(Nemptypatch,SettleProba))';
306     else
```

```
307    % if sum(newgen)==0 no one is there who could settle
308      newgenSettle=zeros(1,length(newgen));
309    end
310
311   % If frequencies don't exactly sum up to 1 (due to numerical problems)
312   % the arrays produced by mnrnd constist only of NaN. In that case an
313   % version of the slower function multrnd is used.
314  if any(isnan(newgenSettle))
315         try
316          newgenSettle=(multrnd(Nemptypatch,SettleProba))';
317         catch
318             % display these values for problem-solving if errors occur
319             display('Problems with multrnd.m')
320             sum(SettleProba)
321             sum(SettleProba)==1
322             Nemptypatch==round(Nemptypatch)
323             Nemptypatch
324             display('Problems with multrnd.m')
325         end
326
327   end
328
329
330   % Total number of settling offspring mutating in:
331
332   % - z1/2 (Mutations in z1 and z2 are coupled. So in this model
333   % they always mutate together. The direction is determinded by the
334   % covariance matrix)
335     Mutz1=binornd(Nemptypatch,mPz);
336   % - a
337     Muta=binornd(Nemptypatch,mPa);
338   % - t
339     Mutt=binornd(Nemptypatch,mPt);
340
341
342  % Security measure that there cannot be a division trough zero.
343    if sum(newgenSettle)~=0
344  %   The probability that a mutation occurs on an initial genotype is
345  %   proportional to the frequency of this genotype among the settled
346  %   individuals
347      MutProba=(newgenSettle/sum(newgenSettle))';
348      [temp I]=max(MutProba);
349      MutProba(I)=MutProba(I)+(1.0-sum(MutProba));
350
351   %  allocating the number of mutations in z1/2 ('Mutz1') to
352   %  the different genotypes according to their frequency in the
353   %  population
354      if Mutz1>0
355      newgenMutz1=(mnrnd(Mutz1,MutProba))';
356      else
357   %  for zero mutations one has to create the mutation vector explicitly
358   %  to account for problems with input 0 to mnrnd.m occuring only on the
359   %  simulation server
360      newgenMutz1=zeros(1,length(newgen))';
```

```
361     end
362
363   %  equivalent as for z1/2
364     if Muta>0
365     newgenMuta=(mnrnd(Muta,MutProba))';
366     else
367   %  for zero mutations one has to create the mutation vector explicitly
368   %  to account for problems with input 0 to mnrnd.m occuring only on the
369   %  simulation server
370     newgenMuta=zeros(1,length(newgen))';
371     end
372
373   %  equivalent as for z1/2
374     if Mutt>0
375     newgenMutt=(mnrnd(Mutt,MutProba))';
376     else
377   %  for zero mutations one has to create the mutation vector explicitly
378   %  to account for problems with input 0 to mnrnd.m occuring only on the
379   %  simulation server
380     newgenMutt=zeros(1,length(newgen))';
381     end
382
383    else
384   %  If no individual settles noone can mutate
385     newgenMutz1=zeros(1,length(newgen))';
386     newgenMuta=zeros(1,length(newgen))';
387     newgenMutt=zeros(1,length(newgen))';
388    end
389
390
391   % If the function mnrnd.m produces NaN values as output the slower
392   % function multrnd is used instead
393     if any(isnan(newgenMutz1))
394       newgenMutz1=(multrnd(Mutz1,MutProba))';
395     end
396     if any(isnan(newgenMuta))
397       newgenMuta=(multrnd(Muta,MutProba))';
398     end
399     if any(isnan(newgenMutt))
400       newgenMutt=(multrnd(Mutt,MutProba))';
401     end
402
403
404    % the new population (here still without mutated offspring) is:
405    % old population - died individuals + offspring that can settle -
406    % part of settling offspring that mutates
407     gen(:,4)=gen(:,4)-dead+newgenSettle-(newgenMutz1+newgenMuta+newgenMutt);
408
409
410
411   %-----------------------------------------
412   %Mutational process
413   %-----------------------------------------
414
```

```
% Produce a vector with the indices of the genotypes (line number in the
% genotype matrix) for which at least one mutation occurs
  Idx_newgenMutz1=find(newgenMutz1);
  Idx_newgenMuta=find(newgenMuta);
  Idx_newgenMutt=find(newgenMutt);

  %Mutation in z1/z2
  %for all the indices of the genotypes that contain mutating offspring
  for i=1:length(Idx_newgenMutz1)

      % for each mutating individual of this initial genotype
      for j=1:newgenMutz1(Idx_newgenMutz1(i))

          % Draw the mutational step in z1/2 from a 2-dimensional
          % normal distribution, the two values beeing coupled by the
          % covariance.
          % If the new value of z1 or z2 exceeds the bounds 0,1 it
          % is set to respectivlely 0 or 1
          A=chol(covz1z2);
          zNew=max([0 0],min([1 1],[gen(Idx_newgenMutz1(i),1) ...
              gen(Idx_newgenMutz1(i),2)]+...
              round(1/mSz*randn(1,2)*A)*mSz));

          % if the new genotype already exists within the population, find
          % its index
          x=find((gen(:,1)==zNew(1)).*(gen(:,2)==zNew(2)).*...
              (gen(:,3)==gen(Idx_newgenMutz1(i),3)).*(gen(:,5)==...
          gen(Idx_newgenMutz1(i),5)),1);
          %if it doesn't exist, create it
          if isempty(x)
              gen(length(gen)+1,:)=[zNew(1) zNew(2) ...
              gen(Idx_newgenMutz1(i),3) 1 gen(Idx_newgenMutz1(i),5)];
              %if it exists, add the current individal to it
          else
              gen(x,4)=gen(x,4)+1;
          end


      end
  end



  % Mutation in a
  % Same prodecure as for z1/2 but without coupling. Mutational step
  % is drawn from normal distribution. Values exceeding the bounds are set
  % to the bounds. Detetermine whether resulting genotype is already
  % present in population. If not create it.
  for i=1:length(Idx_newgenMuta)
          for j=1:newgenMuta(Idx_newgenMuta(i))
          aNew=max(0,min(1,gen(Idx_newgenMuta(i),3)+...
              round(stda/mSa*randn)*mSa));
          x=find((gen(:,3)==aNew).*...
              (gen(:,1)==gen(Idx_newgenMuta(i),1)).*...
```

```
469                 (gen(:,2)==gen(Idx_newgenMuta(i),2)).*(gen(:,5)==...
470                  gen(Idx_newgenMuta(i),5)),1);
471                  if isempty(x)
472                      gen(length(gen)+1,:)=[gen(Idx_newgenMuta(i),1) ...
473                      gen(Idx_newgenMuta(i),2) aNew 1 ...
474                      gen(Idx_newgenMuta(i),5)];
475                 else
476                      gen(x,4)=gen(x,4)+1;
477                  end
478
479             end
480     end
481
482     % Mutation in t
483     % Same procedure as for the mutation in a
484     for i=1:length(Idx_newgenMutt)
485             for j=1:newgenMutt(Idx_newgenMutt(i))
486             tNew=max(0,min(1,gen(Idx_newgenMutt(i),5)+...
487                 round(stdt/mSt*randn)*mSt));
488
489             x=find((gen(:,5)==tNew).*...
490                 (gen(:,1)==gen(Idx_newgenMutt(i),1)).*...
491                 (gen(:,2)==gen(Idx_newgenMutt(i),2)).*(gen(:,3)==...
492             gen(Idx_newgenMutt(i),3)),1);
493                  if isempty(x)
494                      gen(length(gen)+1,:)=[gen(Idx_newgenMutt(i),1) ...
495                          gen(Idx_newgenMutt(i),2) ...
496                          gen(Idx_newgenMutt(i),3) 1 tNew];
497                 else
498                      gen(x,4)=gen(x,4)+1;
499                  end
500
501             end
502     end
503
504
505     % Clear genotypes with population sizes 0 from the populations (meaning
506     % that no individual actually has this genotype)
507     gen(gen(:,4)≤0,:)=[];
508
509     % Security measure for stability, as sometimes the value 0. is taken as
510     % beeing negative
511     if any(gen<0)
512         display('gen <0');
513     end
514     gen=abs(gen);
515
516     % Test whether any trait value is >1
517     if any(gen(:,1:3)>1)
518        display 'genvalues >1'
519     end
520
521     % Every 1000 generations determine whether the population already
522     % converged to one of the "end points" beeing the boundaries of the
```

```
523     % trait space.
524     % The "end-points" depend on the scenario one regards.
525     % The functions PlastiOutcome.m and detOutcome_Plasti.m have to be
526     % adapted for different investigations
527
528       if (t/1000)==floor(t/1000)
529           % Determine the evolutionary end points. Possible end-points
530           % depend on the scenario regarded and are described section \ref{sec:end-points}
531           % and in the documentation of PlastiOutcome.m and
532           % detOutcome_Plasti.m.
533           % PlastiOutcome.m determines the location of the population in the
534           % trait space
535           % detOutcome_Plasti.m. determies whether the location determined
536           % by PlastiOutcome.m corresponds to a stable end-point by
537           % comparing the results of the last 15 generations.
538           outc(floor(t/1000))=PlastiOutcome(gen,.12);
539           if length(outc)≥15 && ¬nobreak
540           brk=detOutcome_Plasti(outc(floor(t/1000)-14:floor(t/1000)));
541              if brk==1
542                  return;
543              end
544           end;
545       end
546
547
548
549
550
551 end
552
553 % If the population didn't converge to an endpoint bevore. Determine here
554 % the outcome of the simulation. outc=0 if non of the endpoints is reached
555 outc(end+1)=PlastiOutcome(gen,.12)
556
557 % Determine elapsed time and write it to the struct params
558 eltime=toc
559 params.eltime=eltime;
560
561 end
```

A.1.2. Phase Diagram

The commented Matlab source code of the function "plot_pip_f.m" producing the z_1, z_2-phase-diagram.

```
1 function[]=plot_pip_f(pop,params,t)
2   %function[]=plot_pip_f(pop,params,t)
3   %
4   %Function that plots a z1-z2 phase diagram for a fixed point in time.
5   %
6   %It is implemented in the main program BetHedgingGen_2_4_f.m presented
7   %in appendix A.1.1.
```

```
8   %
9   %A figure environment has to be opened in Matlab before running this
10  %function.
11  %
12  %Input arguments:
13  %
14  %pop(mx5 matrix): Matrix containing the population that should be
15  %plotted. pop(i)=[z1 z2 a n t], where n is the number of individuals of
16  %the ith genotype present in the population. i=1..m, where m is the
17  %number of different genotypes present.  Equivalent to the matrix "gen"
18  %used in BetHedgingGen_2_4_f.m. See there for a further descripiton.
19  %
20  %params(struct):System parameters as they are created in
21  %BetHedgingGen_2_4_f.m. Optional input, needed only for the diagram
22  %title.
23  %
24  %t(integer):Time-step this plot corresponds to. Optional input, needed
25  % only for the diagram title.
26  %
27  %Output arguments:
28  %
29  %none
30
31  % if no parameters are entered, the params variable is set to zero to
32  % suppress the creation of a diagram title
33    if nargin<2, params=0; end;
34  % same for t
35    if nargin<3, t=0; end;
36
37    %sumation over all genotypes with fixed z1 and z2 as we do not regard a
38    %t
39
40    %for all genotypes in the population
41    for i=1:length(pop)
42        %deterimine the genotypes that have identical z1 and z2 as the
43        %first genotype (but they differ in a and/or t)
44        log=logical((pop(:,1)==pop(1,1)).*(pop(:,2)==pop(1,2)));
45        %create a matrix containing these genotypes
46        same=pop(log,:);
47        % put the total number of individuals having this z1 and z2 in the
48        % ith line of the matrix contpop (regardless of their a and t
49        % values)
50        contpop(i,:)=[pop(1,1) pop(1,2) sum(same(:,4))];
51        % substract all the genotypes counted by this iteration of the loop
52        % from the population
53        pop=pop(¬log,:);
54        % if all genotypes have allready been contracted to contpop, stop
55        % the iteration
56        if isempty(pop)
57            break;
58        end
59    end
60
61    %few=all genotypes (z1,z2) in the population (contracted over a and t)
```

```
    %that are rarer than ported by 11 individuals
    few=find((contpop(:,3)≤10).*(contpop(:,3)>0));

    %inter= all genotypes that are possessed by 11−100 individuals
    inter=find((contpop(:,3)≤100).*(contpop(:,3)>10));

    %many=genotypes that are possessed by more than 100 individuals
    many=find((contpop(:,3)>100));

    %do not clear the diagramm after drawing once
    hold on;

    %plot the rare genotypes with blue circles
    few=plot(contpop(few,1),contpop(few,2),'o');
    set(few,'MarkerSize',3);

    %plot the intermediate genotypes with green squares
    inter=plot(contpop(inter,1),contpop(inter,2),'gs');
    set(inter,'MarkerSize',3);

    %plot the frequent genotypes with red diamonds
    many=plot(contpop(many,1),contpop(many,2),'rd');
    set(many,'MarkerSize',3);

    %clear the figure next time you plot something
    hold off;
    %axes
    xlabel('z1');
    ylabel('z2');
    axis([0 1 0 1]);

    %create a title if parameters are given as input
    if isstruct(params)
       if t
            title({['t=',num2str(t),' K=',num2str(params.K),...
                ' mP=',num2str(params.mP),' mS=',num2str(params.mS)];...
                [' p=',num2str(params.p), ' d=',num2str(params.d),...
                ' amax(1)=',num2str(params.amax(1)),...
                ' amax(2)=',num2str(params.amax(2)),...
                ' c=',num2str(params.c),' tmax=',num2str(params.tmax)]});
       else
          title({['K=',num2str(params.K),' mP=',num2str(params.mP),...
              ' mS=',num2str(params.mS)];[' p=',num2str(params.p),...
              ' d=',num2str(params.d),' amax(1)=',num2str(params.amax(1)),...
              ' amax(2)=',num2str(params.amax(2)),' c=',num2str(params.c),...
              ' tmax=',num2str(params.tmax)]});
       end
    % if no parameters are give, print only time (if available)
    elseif t
        title({['t=',num2str(t)]},'FontSize',14);
    end
end
```

A.1.3. Determining End-Points in a Bet-Hedging Model

The commented Matlab source code of the function "PolyOutcome_f.m" determining if one of the possible end-points in the situation where only bet-hedging is allowed for is reached.

```
function[outc]=PolyOutcome(gen,limit)
% function[outc]=PolyOutcome(gen,limit)
%
% Function that determines whether a population generated by the
% main program BetHedgingGen_2_4_f.m has reached one
% of the possible evolutionary end-points. The possible-end points are
% described in more detail in section 2.2.1
%
%
% The possible endpoints this function can detect:
% a) BetHedging Strategy with (z1,z2)->(1,0) (outc=1)
% b) BetHedging Strategy with (z1,z2)->(0,1) (outc=2)
% c) Genetic Polymorphism (z1,z2)->(0,0) and (1,1) (outc=3)
% if non of those can be detected outc=0
%
% Input Arguments:
%
% gen(mx4 matrix): Population matrix as created by BetHedgingGe_2_4.m
% each time step
%
% limit(double): parameter that decides how close to an endpoint the
% population has to be before the endpoint is considered as outcome
% (limit in [0.01,0.3] make sens, depending on mutation rate). Small values
% of limit mean that the population has to be close to the end-point.
%
% Output:
%
% outc(ineger): Classification of the end-point the population reached (see
% above)
%
        % Determine the total population size
        K=sum(gen(:,4));
        % Determine the number of individuals in a square being situated in
        % the left bottom (z1,z2)=(0,0) corner. Side lenght = limit
        slb=sum(gen(logical((gen(:,1)<limit).*(gen(:,2)<limit)),4));
        % Same for left top corner (z1,z2)=(0,1)
        slt=sum(gen(logical((gen(:,1)<limit).*(gen(:,2)>(1-limit))),4));
        % Same for rigth bottom corner (z1,z2)=(1,0)
        srb=sum(gen(logical((gen(:,1)>(1-limit)).*(gen(:,2)<limit)),4));
        % Same for right top corner (z1,z2)=(1,1)
        srt=sum(gen(logical((gen(:,1)>(1-limit)).*(gen(:,2)>(1-limit))),4));

        % if 90% of the population is in one of the corners
        if (slb+slt+srb+srt)>K*.9
            % if 90% of the population is in (0,0) or (1,1) corner
            % -> outcome 3
```

```
47              if ((slb≠0 && srt≠0)) && ((slb+srt)>K*.9)
48                  outc=3;
49              % outcome (1,0)?
50              elseif srb>K*.8
51                  outc=1;
52              % outcome (0,1) ?
53              elseif slt>K*.8
54                  outc=2;
55              % If individuals with about (1,0) and about (0,1) exist, take
56              % the dominating population as end-point as one of the
57              % sub-populations will die out in long term
58              elseif (slt+srb)>K*.9
59                      if slt>srb
60                          outc=2;
61                      else
62                          outc=1;
63                      end
64           % If non of these end points is reached, set outc to 0
65              else
66                  outc=0;
67              end
68          else
69              outc=0;
70          end
71
72
73  end
```