MATHEMATICAL MODELS OF FREQUENCY-DEPENDENT SELECTION WITH DOMINANCE

Dissertation

zur Erlangung des akademischen Grades
Doktor rer. nat.
an der Universität Wien

---

verfasst von
Stephan Peischl, Mat.Nr. 0100714

Studienkennzahl lt. Studienblatt:	A 091 405
Dissertationsgebiet lt. Studienblatt:	Mathematik
Betreuer:	Dr. Reinhard Bürger

Acknowledgements

First and foremost I thank my advisor Professor Reinhard Bürger. He introduced me to mathematical population genetics and altruistically helped me to develop my scientific skills. I also want to thank my collaborator Kristan Schneider, especially for his assistance during the preparation of this thesis. Furthermore, I am most grateful to Professors Nick Barton and Joachim Hermisson for serving as members of my PhD committee.

My time as a PhD student would not have been the same without my fellow office mates and colleagues. I want to thank the following people for their support and for the fun we had during the last three years: Ada Akerman, Agnes Rettelbach, Barbara Fischer, Caterina Cusulin, Christoph Krall, Claudia Bank, Claus Rueffler, Florian Schätz, Hannes Svardal, Hildegard Uecker, Jitka Polechová, Jostein Starrfelt, Laura Vossen, Mario Aigner, Michael Kopp, Peter Jungwirth, and Simon Aeschbacher.

I feel lucky to have parents who always believed in me and supported me. Their support paved the way that led to this thesis. I dedicate this thesis to them. Furthermore, I want to thank my sister Kathrin, who inspired me to study mathematics, and her two kids Julian and Frederik. Finally, I want to thank Anna Gnaiger for her inspiration and support.

Preface

In this thesis, I present part of my work in my main research area, namely mathematical population genetics. The work presented here manifests two lines of research of mathematical models of frequency-dependent selection. Dominance and its effect on the maintenance of genetic variation and speciation spans the arch between the two parts. In the first part, we study a model of intraspecific competition and assortative mating. Part I consists of two articles that are closely linked to each other. In Chapter 1, we study the evolution of dominance in an assortatively mating population under frequency-dependent intraspecific competition. In Chapter 2, we keep dominance fixed and let assortment evolve. The initial motivation of the work presented in Part I is rooted in my master thesis, in which I studied the evolution of dominance under frequency-dependent selection in a randomly mating population. In the second part, a different class of models is studied. Part II consists of a single chapter in which we study the relationship between the degree of dominance and the number of alleles that can be maintained at a stable equilibrium in two demes. This work was motivated by a recent paper of Professor Thomas Nagylaki.

Vienna, Austria — Stephan Peischl
February 2009

Contents

Introduction

Population genetics is concerned with the genetic composition of populations and how this composition changes over time. Natural selection, mutation, recombination, mating structure, spatial structure, and other genetic or ecological factors determine the change of the genetic composition of a population. These mechanisms and their interactions are studied in population genetics. Understanding the generation and maintenance of genetic variation in a population, and how this variation leads to evolutionary phenomena such as adaptation and speciation, is one of the main aims of population genetics. Since so many factors interact to determine evolutionary change, a fair amount of abstraction is necessary to understand the relevant processes. Therefore, in contrast to most other fields in biology, mathematical thinking has been part of population genetics since its very beginning. In fact, many important developments in mathematics, and especially statistics, have their origins in the classical work of Weinberg, Fisher, Haldane, Wright, and Kimura.

The probably earliest mathematical model in population genetics dates back to Gregor Mendel. He used elementary mathematics to calculate the expected frequencies of phenotypes in his experiments with peas. In the second and third decade of the twentieth century, Ronald A. Fisher, J.B.S. Haldane, and Sewall Wright reconciled Mendelian inheritance with Darwinism; this was the birth of modern population genetics. The work of Haldane, Wright, and Fisher was highly mathematical and, at that time, it was properly understood only by few biologists. Nevertheless, their work was highly influential and the modern evolutionary synthesis is now accepted by nearly all biologists. Today, the basis of hereditary mechanisms has been firmly established, mutations are known as the ultimate source of genetic variation, natural selection at the phenotypic level has been documented, and our knowledge about the molecular biology of the genes is rapidly increasing. Nevertheless, many open questions remain, for instance, questions about the origin and maintenance of sex, or the processes involved in speciation.

In this thesis, we study population-genetic models of frequency-dependent selection with dominance. Frequency-dependent selection occurs if the fitness of a genotype depends on its relative frequency in the population. Early on, the idea of frequency-dependent selection was recognized as being important (Fisher, 1930). Frequency-dependence occurs in many contexts such as mimicry (Charlesworth and Charlesworth, 1975a; Pfennig et al.,

2001), predator-prey interactions (Endler, 1988; Allen and Greenwood, 1988), intraspecific competition (Bolnick, 2004), multiple ecological niches (Levene, 1953), or non-random mating (Kirkpatrick and Nuismer, 2004), and has been invoked in the in the explanation of a number of important evolutionary phenomena. Because of the complexity of models with frequency-dependent selection, they were mainly studied under assumptions such as random mating and/or no dominance.

If two alleles in heterozygous combination produce a phenotype that resembles one of the two homozygous phenotypes, the allele that masks the effect of the other is said to be dominant. It is often convenient to assume no dominance because it greatly simplifies mathematical analysis. However, dominance is commonly observed in natural populations. For instance, it is generally accepted that mutations with major (deleterious) fitness effects are almost always recessive. Furthermore, the dominance relation between alleles may evolve (Charlesworth and Charlesworth, 1975b; Van Dooren, 1999; Otto and Bourguet, 1999; Peischl and Bürger, 2008; Bürger and Bagheri, 2008).

Throughout this thesis, we consider diploid populations that are so large that genotype frequency changes may be treated as deterministic. Furthermore, we assume discrete non-overlapping generations, we ignore mutation, and consider a finite set of possible genotypes. The evolutionary dynamics of the population in terms of the gene frequencies can be described by deterministic difference equations in finitely many dimensions. Analyzing such models is a challenging task because frequency-dependent selection generally leads to complicated nonlinear dynamics. The complexity of the considered models makes the derivation of explicit solutions of the difference equations impossible. Therefore, special emphasis is put on the qualitative analysis of the equilibrium structure and the local and global asymptotic stability properties.

In part I, the main focus is the evolution of so called 'modifier genes'. We consider a single locus that determines the ecological trait. This trait is under frequency-dependent selection. The alleles at the modifier loci affect the mating structure or the degree of dominance expressed at the ecological locus. The stability properties of the boundary equilibria that correspond to loss or fixation of a modifier allele are of special interest. For instance, fixation of a modifier allele that increases the strength of assortative mating may lead to speciation, i.e., the evolutionary splitting of a population into separate species. We are able to analytically determine conditions for asymptotic stability of these equilibria in several interesting scenarios. The analytical results are complemented by comprehensive numerical investigations. In part II we study the maintenance of genetic variation under frequency-dependent selection. Of central interest is the existence of asymptotically stable internal equilibria. At internal equilibria, all genotypes are present at positive frequency. Using a numerical approach, we determine parameter combinations that harbor potential for the maintenance of polymorphism. We prove the existence of an internal equilibrium

in a symmetric special case with three alleles. A bifurcation analysis shows that this equilibrium exchanges stability with boundary equilibria when it enters the state space. Furthermore, we can show that this equilibrium continues to exist under small perturbations of the symmetry assumptions.

In addition to elementary calculus and algebra, we use techniques such as local stability analysis and perturbation theory. In general, analytical results can only be derived in special cases or under stringent assumptions. Thus we complement the analytical results by comprehensive numerical investigations. Our numerical results, based on the exact dynamics, show that the global behavior is often much richer than suggested by analytical results. Nevertheless, analytical results, even if obtained under rather strong assumptions, are often illuminating and helpful in the interpretation of numerical data.

In the first part of this thesis, we study the evolution of dominance and assortative mating in a population genetics model with an ecological context. In this model, individuals compete for a continuum of limited resources. The ecological model is closely related to those of Bulmer (1980); Slatkin (1979); Christiansen and Loeschcke (1980); Christiansen (1982); Loeschcke and Christiansen (1984); Bürger (2002a,b, 2005) and Schneider and Bürger (2006). In particular, viability of individuals is given by a combination of frequency-independent stabilizing selection, frequency-dependent intraspecific competition, and density-dependent population regulation. Stabilizing selection is induced by the exploitation of a one-dimensional resource that is assumed to be unimodally distributed. Individuals exploit the resources according to a unimodal utilization function. The genotype of an individual determines the modal position of the utilization function. The utilization of the resource spectrum results in intraspecific competition which induces frequency-dependent selection.

Assortative mating is modeled as female preference with respect to similarities in the ecological trait (Gavrilets and Boake, 1998). Such traits have been called 'magic traits' by Gavrilets (2004). We assume that encounters between females and males of the same genotype always result in successful mating, whereas the probability for successful mating decreases with increasing distance in the phenotypic value according to a Gaussian or quadratic function. The parameter measuring the curvature of this function is a measure for the strength of assortative mating. Although we do not assume direct costs for choosiness, the disadvantage of rare males induces positive frequency-dependent sexual selection (Kirkpatrick and Nuismer, 2004).

Since we consider the interaction of two different forms of frequency-dependent selection, the net effect of selection for a given combination of parameters is not intuitively clear. In addition, one has to track the genotype frequencies instead of the gamete frequencies if mating is nonrandom. This significantly increases the dimensionality of the

system.

To illustrate the ecological setup of our model, think of birds foraging for seeds. The size of the seeds is unimodally distributed. Birds have different beak sizes and they can utilize the seeds according to a unimodal utilization function. The modal position of the utilization function is determined by the beak size of the bird. The ecological character is beak size, which is affected by stabilizing selection via the distribution of seeds and by intraspecific competition for seeds. If competition is stronger than stabilizing selection, selection can be disruptive. Under disruptive selection, a $\cup$-shaped phenotype distribution is optimal. If mating is random (with respect to the beak size), hybridization leads to a $\cap$-shaped phenotype distribution. In the simplest case, the ecological trait (the beak size) is determined by a single diallelic locus. Let homozygotes correspond to extreme types and heterozygotes correspond to intermediate types. Then, at equilibrium, heterozygotes are the least fit individuals.

In such scenarios, mechanisms that decrease the frequency or inferiority of heterozygotes at the ecological locus are evolutionary advantageous. These mechanisms include the evolution of assortative mating and the evolution of dominance. The former can lead to the formation of strong reproductive isolation, whereas the latter increases the genetic variance of the population without creating a reproductive barrier between the two extreme morphs.

Such ecological setups are commonly used in models of sympatric speciation (e.g., Dieckmann and Doebeli, 1999; Matessi et al., 2001; Pennings et al., 2008). The theoretical possibility of sympatric speciation is well demonstrated. However, there is an ongoing debate how likely sympatric speciation is in such scenarios (Dieckmann and Doebeli, 1999; Waxman and Gavrilets, 2005; Polechová and Barton, 2005). A well documented example of disruptive selection is the african finch *Pyrenestes ostrinus* (Smith, 1990, 1993). In this species, disruptive selection acts on lower mandible width and is most likely induced by intraspecific competition for seeds. In contrast to our model, the distribution of seeds is bimodal. Interestingly, in *Pyrenestes o.* assortment did not evolve. Instead, one of the two morphs is completely dominant. It is tempting to speculate about the evolution of dominance in *Pyrenestes o.*.

Since dominance and assortment are both solutions to the problem of unfit heterozygotes, they are commonly regarded as alternative responses to disruptive selection. Hence, in most previous studies they were studied separately. Here, we investigate how dominance and assortment interact if one of them is evolving. This yields interesting insights in the interaction of positive frequency-dependent selection and negative frequency-dependent selection. We consider a three locus two allele model. The first locus determines the ecological character. The second and third loci control the degree of dominance at the ecological locus and the strength of assortative mating, respectively. In Chapter 1, we investigate

the effect of a fixed degree of assortative mating on the evolution of dominance. In Chapter 2, we study the evolution of assortative mating in the presence of a fixed degree of intermediate dominance. Both chapters can in principle be read independently. The combined information from both approaches can be used to make predictions on the simultaneous evolution of dominance and assortment. We briefly treat the coevolution of dominance and assortment in the discussion of Chapter 2.

The results of Part I highlight the importance of studying global dynamics and illustrate the limitations of local stability approaches. We tease apart the effects of natural and sexual selection, and show that dominance and assortative mating are not necessarily mutually exclusive responses to disruptive selection. Under certain conditions, dominance can favor the evolution of assortment and vice versa. For instance, moderately strong assortment is most favorable for the evolution of dominance if modifiers have large effects. In addition, our results have interesting implications for models of sympatric speciation. Strong assortment evolves most easily if modifiers have large effect. Our results suggest that strong reproductive isolation can evolve only if sufficiently strong assortment is induced by a single allele substitution. Quite surprisingly, strong dominance significantly increases the parameter region in which such modifiers can go to fixation. However, if modifiers have small effects, even small degrees of dominance considerably hinder the evolution of assortment. We conclude that complete dominance is more likely to evolve than complete, or at least very strong, reproductive isolation. Dominance modifiers usually get fixed much faster than assortment modifiers. During the evolution of assortment, heterozygotes at the ecological locus become rare and selection on assortment modifiers becomes very inefficient. Furthermore, the interaction of natural and sexual selection can stop the evolution of reproductive isolation at some intermediate level.

In Part II of this thesis, we study a different source of frequency-dependent selection, namely population subdivision and environmental heterogeneity. Most natural populations are geographically structured and most environments are heterogeneous. Thus, the investigation of population structure and a heterogeneous environment has a long history in population genetics. A considerable amount of theoretical literature exists on spatially structured populations using different modeling approaches. In deterministic models, the maintenance of genetic variation is studied, whereas stochastic models are employed to study such properties as fixation time and fixation probability of mutations.

Compared to the models in Part I, the ecological setup is much simpler. Selection within demes is frequency independent and mating is random. In the absence of migration, these systems are well understood. Global convergence of trajectories was proved and the conditions for polymorphism are well understood (e.g., Nagylaki, 1992; Bürger, 2000). If either selection or migration is weak, one can reduce the analysis of migration-

selection models to the analysis of panmictic models (Karlin and McGregor, 1972; Nagylaki and Lou, 2001, 2007). This significantly simplifies the analysis. However, in general migration-selection models, many open questions exist. For instance, if there are more than two demes and more than two alleles, convergence is unresolved even for multiplicative fitnesses (hence, for selection on haploids) or no dominance. In discrete time, limit cycles have not even been excluded for two diallelic demes. This illustrates the intricacy of migration-selection models. Part II of this thesis is devoted to the effect of dominance on the potential of maintaining polymorphism in geographically structured populations.

In the absence of dominance, the number of demes is an upper bound for the number of alleles that can be maintained at a single locus (Nagylaki and Lou, 2001). Dominance can have a significant impact on the maintenance of polymorphism. One can construct examples in which an arbitrary number of alleles is maintained at a globally asymptotically stable equilibrium if there are at least two demes (Nagylaki and Lou, 2007). In these examples, locally beneficial alleles are partially dominant. This means that there is some form of genotype-environment interaction. These results can be extended to multilocus models (Bürger, 2009a,b). In Chapter 3, we study the effect of a deme-independent degree of intermediate dominance (DIDID) on the maintenance of polymorphism at a single locus in two demes. DIDID includes no dominance and the absence of genotype-environment interaction as special cases.

Among others, in part II an analytical example for the stable coexistence of three alleles in two demes under DIDID is provided. In this example, the internal equilibrium does not exist if migration is either weak or strong. This is in contrast to conventional wisdom, which states that polymorphism is usually maximized for small migration rates. Numerical computations suggest that three alleles can coexist in an open set of parameters in the full model. This continues to hold if we assume linear selection on a quantitative trait in the absence of G×E interaction. Furthermore, we identified dominance patterns and selection schemes that are particularly prone to maintenance of three alleles in two demes. Our results have interesting interpretations in terms of generalist-specialist coexistence. If generalists are fitter than specialist hybrids and have sufficiently high average fitness, two specialists can coexist with a single generalist. We also found numerical examples in which up to four alleles coexist at an asymptotically stable equilibrium. Our results complement and extend the results of Nagylaki and Lou (2001, 2007) and Nagylaki (2009).

Part I

Intraspecific Competition

Chapter 1

Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population[1]

[1]With minor changes this chapter is published as:
Peischl S. and K.A. Schneider, 2009. Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population. Evolution 64, 561 - 582.

Abstract

In models of frequency-dependent disruptive selection globally stable polymorphic equilibria with underdominance can occur. In the past, a number of possible mechanisms to reduce the inferiority of heterozygotes were studied. These evolutionary responses to negative frequency-dependent selection include the evolution of dominance and the evolution of assortative mating. Usually, different responses are treated as alternatives and hence possible interactions are not very well understood. In contrast to previous studies, we focus on the effect of assortative mating on the evolution of dominance under frequency-dependent intraspecific competition. We analyze a two-locus two-allele model, in which the primary locus has a major effect on a quantitative trait that is under a mixture of frequency-independent stabilizing selection, density-dependent selection and frequency-dependent selection caused by intraspecific competition for a continuum of resources. The second (modifier) locus determines the degree of dominance at the trait level. Additionally, the population mates assortatively with respect to similarities in the ecological trait. Our analysis shows that the parameter region in which dominance can be established decreases if small levels of assortment are introduced. In addition, the degree of dominance that can be established also decreases. On the contrast, if assortment is intermediate, sexual selection for extreme types can be established, which leads to evolution of higher levels of dominance than under random mating. For modifiers with large effects, intermediate levels of assortative mating are most favorable for the evolution of dominance. For large modifiers the speed of fixation can even be higher for intermediate levels of assortative mating than for random mating.

1.1 Introduction

Negative frequency-dependent selection and, as a special case, frequency-dependent disruptive selection resulting from intraspecific competition has long been known as a mechanism that preserves high levels of genetic variation (e.g., Cockerham et al., 1972; Clarke, 1979; Asmussen and Basnayake, 1990; Gavrilets and Hastings, 1995). This was studied, in particular, for quantitative traits (Bulmer, 1974; Slatkin, 1979; Christiansen and Loeschcke, 1980; Loeschcke and Christiansen, 1984; Bürger, 2002a,b; Bürger and Gimelfarb, 2004; Bürger, 2005; Schneider, 2006). Especially in the last decade, frequency-dependent disruptive selection became popular in the theoretical literature for modeling a number of interesting evolutionary phenomena. These include the evolution of sexual dimorphism Slatkin (1984); Bolnick and Doebeli (2003); Van Dooren et al. (2004), the evolutionary splitting of assortatively mating populations Drossel and Mckane (2000); Bolnick (2004); Kirkpatrick and Nuismer (2004); Bürger and Schneider (2006); Bürger et al. (2006); Schneider and Bürger (2006), the evolution of assortative mating and sympatric speciation Maynard Smith (1966); Udovic (1980); Doebeli (1996); Dieckmann and Doebeli (1999); Matessi et al. (2001); Kirkpatrick and Ravigné (2002); Gavrilets (2003, 2004); Dieckmann et al. (2004); Polechová and Barton (2005); Pennings et al. (2008); Kopp and Hermisson (2008), and the evolution of genetic architecture Van Doorn and Dieckmann (2006); Kopp and Hermisson (2006); Matessi and Gimelfarb (2006); Schneider (2007). The latter includes the evolution of dominance, the topic this work is devoted to.

Originally, the evolution of dominance was mainly studied in the context of reducing genetic load in the face of mutation. The common idea to models of dominance evolution is that a phenotype associated with a disadvantageous genotype should evolve to resemble a superior phenotype. For a general introduction to dominance and its evolution we refer to Bürger and Bagheri (2008). In the context of frequency-dependent selection, Wilson and Turelli (1986) found stable underdominance in a model of differential utilization of two resources. They argued that in their model evolution of dominance would be an efficient way to remove unfit heterozygotes. Although an analytical proof within their model seems infeasible, it has recently been shown that evolution of dominance can be a potent mechanism to remove unfit heterozygotes in a number of related, but different, models (Otto and Bourguet, 1999; Van Dooren, 1999; Peischl and Bürger, 2008). Another possibility for removing unfit heterozygotes is the evolution of reproductive isolation. Evolution of assortative mating and sympatric speciation have been studied intensively during the last decade and, at least theoretically, the former appears to be a feasible response to negative frequency-dependent selection.

Recently, some work has been devoted to the comparison of different responses to disruptive selection which are mainly regarded as alternatives. For instance, Leimar (2005) in-

vestigated the evolution of phenotype polymorphism, focusing on random versus genetic phenotype determination. Sexual dimorphism and evolutionary branching are treated in Van Dooren et al. (2004), whereas Bolnick and Doebeli (2003) considered sexual dimorphism and adaptive speciation. Durinx and van Dooren (2008) studied whether selection pressures are stronger for an increased level of assortment or for an increased level of dominance. For a recent review of various evolutionary responses to disruptive selection we refer to Rueffler et al. (2006). All these papers employed the perspective of adaptive dynamics (e.g., Dieckmann and Law, 1996; Geritz et al., 1998), and complemented the analytical results by numerical simulations. The adaptive-dynamics framework provides a simplified method to study the evolution of traits by investigating evolutionary singularities and their convergence and stability properties.

The main tool in this approach is the invasion fitness of initially rare mutants that occur sufficiently infrequent. Indeed, investigations of complicated frequency-dependent fitness landscapes and their singular points are often useful first steps to understand the dynamical behavior of a model. However, this approach lacks explicit investigations of the full evolutionary dynamics and, therefore, the interaction of the evolutionary phenomena is not very well understood.

In this article, we focus on the interplay between assortative mating and dominance. We perform a study of the evolution of dominance modification in an assortatively mating population that complements the results of Durinx and van Dooren (2008). We follow a population genetic rather than an adaptive dynamics approach. Our main aim is to gain a better understanding of the involved components of selection and how they interact. To tease apart these components, we assume a fixed degree of assortment and allow dominance to evolve. The evolution of assortative mating in a population expressing dominance will be treated in a follow-up paper.

We assume an explicit ecological model of frequency-dependent intraspecific competition and assortative mating. Frequency-dependent competition induces indirect selection on a modifier that alters the dominance relations. The degree of assortative mating controls the translation of direct selection on the ecological locus to indirect selection on the modifier locus. In addition, assortative mating induces positive frequency-dependent selection, i.e., selection for common types, which can promote or act against the evolution of dominance. For instance, if assortment is sufficiently strong and homozygotes are the most common types, positive frequency-dependence induces stabilizing selection around the phenotypic values of the homozygotes and thus selection for higher levels of dominance. The strength and direction of these effects depend on the genetic composition of the population and, thus, vary over time even for a fixed combination of parameters. Hence, for our purpose an invasion analysis is insufficient. Of course, a complete (nonlinear) analysis would be highly desirable, but is prohibited by the complexity of the model.

Thus, we pursue a structured and detailed numerical study considering a large part of the parameter space. We complement this by analytical results for weak selection.

Our model assumes two diallelic loci. The primary (ecological) locus has a major effect on a quantitative trait that is under a mixture of stabilizing selection and frequency-dependent selection caused by intraspecific competition for a continuum of resources. The secondary (modifier) locus determines the degree of dominance at the ecological locus. In contrast to previous studies of the evolution of dominance, we incorporate assortative mating into our model and assume that the population mates assortatively with respect to similarities in the ecological trait ('magic trait' model, cf. Gavrilets, 2004).

Previous work of Peischl and Bürger (2008) for randomly mating populations showed that, in the limit of weak selection, modifiers increasing the level of dominance can successfully invade a population at equilibrium if they increase the fitness of heterozygotes. Moreover, provided selection is disruptive, modifiers that are able to invade will also get fixed. The strength of selection on modifiers depends on the proportion of heterozygotes at equilibrium, since only their phenotypes are affected by dominance. Intuitively, one would expect that small degrees of assortative mating would decrease the strength of selection for dominance modifiers. Furthermore, for complete assortment, one expects dominance modifiers to be almost neutral near an equilibrium, because heterozygotes are very rare. Although this proves to be true, intermediate degrees of assortative mating can have a number of different and interesting effects on the evolution of dominance which depend on a subtle interplay of the different selective forces and their impact on the modifier locus.

Our study shows that the evolutionary outcome depends in a highly nonlinear way on the involved parameters. This reflects the complexity of the combination of the different selective forces. It is shown that although weak assortative mating acts against the evolution of dominance, intermediate degrees of assortment lead to selection for an increased level of dominance in a significant parameter region. The maximum degree of dominance that can evolve is higher for intermediate assortment than for random mating. In addition, for modifiers with large effects, evolution of dominance is fastest if assortment is intermediate.

1.2 The model

Our model follows closely that of Schneider and Bürger (2006). We consider a sexually reproducing population of diploid organisms with discrete generations in which both sexes have the same genotype distribution among zygotes. Its size, N, is density regulated, but sufficiently large so that random genetic drift can be ignored. Natural selection acts through differential viabilities on a polygenic trait such that individual fitness is determined by two components: by frequency-independent stabilizing selection on this trait, and by frequency-

and density-dependent competition among individuals of similar phenotype. The value of the ecological trait expressed by heterozygous individuals depends on an additional locus that modifies dominance. Furthermore, assortative mating may induce sexual selection.

1.2.1 Ecological assumptions

The first fitness component is frequency independent and reflects some sort of stabilizing selection on the ecological trait, for example, by differential supply of a resource whose utilization efficiency is phenotype dependent. As in most previous studies, we ignore environmental variation and deal directly with the fitnesses of genotypes, which we identify by their genotypic values. Therefore, we use the terms genotypic value and phenotype synonymously. We denote the ecological trait value of an individual with genotype g by Z_g.

We denote the stabilizing component of fitness acting on genotype g by $S(Z_g)$. Here, we model stabilizing selection by a Gaussian function with optimum zero,

$$S(Z_g) = \exp\{-s{Z_g}^2\}\,, \tag{1.1}$$

where $s \geq 0$ measures its strength. By $\alpha(Z_g, Z_h)$, we denote the amount of competition of genotype g with genotype h, and model it by

$$\alpha(Z_g, Z_h) = \exp\{-c(Z_g - Z_h)^2\}\,. \tag{1.2}$$

The parameter $c \geq 0$ determines the curvature of the Gaussian function (1.2).

Let P_h denote the relative frequency of individuals with genotype h. Then the intraspecific competition function $\overline{\alpha}(g)$, which measures the strength of competition experienced by genotype g in a population with genotypic distribution P, is given by

$$\overline{\alpha}(g) = \sum_h \alpha(Z_g, Z_h) P_h\,. \tag{1.3}$$

We include density-dependent population growth which, in the absence of genetic variation, follows the so-called discrete logistic equation

$$N' = \begin{cases} N(\rho - N/\kappa), & 0 \leq N < \rho\kappa\,, \\ 0\,, & N \geq \rho\kappa\,. \end{cases} \tag{1.4}$$

The carrying capacity is $K = (\rho - 1)\kappa$. Monotone convergence to K occurs for all N with $0 < N < \rho\kappa$ if $1 < \rho \leq 2$, and oscillatory convergence (at a geometric rate) if $2 < \rho < 3$. Other forms of population regulation may be used as well (cf. May and Oster, 1976;

Thieme, 2003) (see also Appendix B in Bürger, 2005, for a more detailed discussion) without significantly changing the outcome. We realize that the use of (1.4) can lead to limit cycles and even chaotic behavior. However, in the considered parameter region the dynamics are 'well-behaved' and no case of complicated behavior was detected in our numerical study.

We assume that the absolute fitness of an individual with genotype g is

$$W(g) = S(Z_g)\Big(\rho - \frac{N}{\kappa}\overline{\alpha}(g)\Big)\,, \tag{1.5}$$

where the dependence of $W(g)$ on N and P is omitted (cf. Bulmer, 1974). Here, c is a direct measure for the strength of frequency dependence. Note that frequency dependence vanishes in the limit $c \to 0$. For convenience, we will refer to weak and strong frequency dependence as weak and strong competition, respectively.

In some part of this work, we will replace (1.1) and (1.2) by the corresponding quadratic approximations, i.e., by

$$S(Z_g) = 1 - s{Z_g}^2 \tag{1.6}$$

and

$$\alpha(Z_g, Z_h) = 1 - c(Z_g - Z_h)^2\,. \tag{1.7}$$

We will refer to (1.5) with $S(Z_g)$ and $\alpha(Z_g, Z_h)$ given by (1.1) and (1.2) or by (1.6) and (1.7) as the *full model* and the *quadratic model*, respectively. Note that the quadratic model can be thought of as the weak selection approximation of the full model, i.e., as an approximation for small s and c. The quadratic model was used to study the evolution of dominance in a randomly mating population by Peischl and Bürger (2008). This weak selection approximation was also used to study closely related ecological models under different assumptions and with a different focus by Bürger and Gimelfarb (2004) and Bürger and Schneider (2006).

1.2.2 Assortative mating

In our model the probability that a female of genotype g mates with a male of genotype h depends on similarity in the ecological character.

The probability that, at a given encounter, a g-female mates an h-male is given by

$$\pi(Z_g, Z_h) = \exp\{-a(Z_g - Z_h)^2\}\,, \tag{1.8}$$

where a is a direct measure for the strength of assortative mating. Females are assumed to

mate only once, whereas males may participate in multiple matings. If an encounter was not successful, in which case a female remains unmated, she may try again unless the total number of encounters has reached a number M. This reflects the idea that choosiness has costs, for instance, because the mating period is limited (cf. Gavrilets and Boake 1998). The probability that an encounter of a female of type g with a random male results in mating is

$$\overline{\pi}(g)=\sum_h \pi(Z_g,Z_h)P_h\,, \tag{1.9}$$

and the probability that she eventually mates with a male of type h is given by $Q(g,h)P_h$, where

$$Q(g,h)=\sum_{m=0}^{M-1}(1-\overline{\pi}(g))^m\pi(Z_g,Z_h)\,. \tag{1.10}$$

Here, the first argument refers to the female. In general, Q is not symmetric in g and h.

The parameter M is a measure for the costs of choosiness females have to pay. If $M=1$, then $Q(g,h)=\pi(Z_g,Z_h)$. In this case costs are very high, which leads to strong sexual selection in both sexes. However, it also admits a number of different interpretations, for instance as a model in which both sexes are choosy (cf. Schneider, 2005) or as parental selection (cf. Gavrilets, 1998), and as a model of fertility selection (cf. Bodmer, 1965; Hadeler and Liberman, 1975). It can thus be interpreted as a model of pre- and postmating reproductive isolation, and has been used in a variety of studies (see also Matessi et al., 2001). If the encounter rate is sufficiently high ($M\gtrsim 10$), costs of choosiness can be neglected, i.e., M may be set to infinity (cf. Schneider and Bürger 2006). Then, we obtain $Q(g,h)=\pi(Z_g,Z_h)/\overline{\pi}(g)$ and $\sum_h Q(g,h)P_h=1$ for all g. Thus, assortative mating does not induce (sexual) selection among females. It does, however, induce sexual selection among males. In other words, females do not pay costs of being choosy, but males pay costs of being rare. For a more detailed discussion of this model and its relation to other work we refer to Schneider and Bürger (2006). For our study we assume $M=\infty$.

1.2.3 Genetic assumptions

We now specify our genetic assumptions. First, we assume that the ecological trait is determined by a single diallelic locus. The alleles are denoted by $\mathscr{A}_1$ and $\mathscr{A}_2$ and their effects by z_1 and z_2, respectively. For our numerical study we assume that these effects are symmetric and scaled such that the maximum and minimum ecological trait values are -1 and 1, i.e., we assume $z_1=-z_2=1/2$. In Section 1.3.3 the allelic effects are evolvable.

The dominance relations at the ecological locus are determined by a separate modifier locus. At this locus we assume that at most two alleles denoted by $\mathscr{D}_1$ and $\mathscr{D}_2$ occur. We assume that these alleles have additive allelic contributions, d_1 and d_2, to the amount of

dominance expressed at the ecological trait. Furthermore, we restrict attention to intermediate dominance, i.e., we assume $0 \leq \max(|2d_1|, |2d_2|) \leq 1$.

The ecological trait values of individuals with genotypes $\mathscr{A}_1\mathscr{A}_1/\mathscr{D}_i\mathscr{D}_j$, $\mathscr{A}_1\mathscr{A}_2/\mathscr{D}_i\mathscr{D}_j$, and $\mathscr{A}_2\mathscr{A}_2/\mathscr{D}_i\mathscr{D}_j$ $(i, j \in \{1, 2\})$ are given by

$$2z_1 \quad z_1 + z_2 + (d_i + d_j)(z_1 - z_2) \quad \text{and} \quad 2z_2\,, \tag{1.11}$$

respectively. Clearly, dominance affects only individuals that are heterozygous at the ecological locus.

Whenever we refer to modifiers increasing dominance, we call the allele at the modifier locus that codes for a higher level of dominance the mutant or the modifier allele, and the allele coding for a lower level of dominance the wild type allele. In the case of modifiers decreasing dominance, it is the other way round. We refer to the degree of dominance expressed by heterozygotes carrying only the wild type allele as the initial degree of dominance, d. Furthermore, we refer to the difference between the initial degree of dominance and the degree of dominance expressed by heterozygotes carrying exactly one modifier allele as the effect of the modifier allele, δ. Suppose $\mathscr{D}_1$ is the wild type allele, then $d = 2d_1$ and $\delta = d_2 - d_1$. Since, in the numerical study, allelic effects are symmetric at the ecological locus, we can always assume d_1 or d_2 to be nonnegative. Thus we can restrict attention to nonnegative values of d without loss of generality. For given d_1 and d_2, the model in which $\mathscr{D}_1$ is the wild type allele is formally equivalent to the model where $\mathscr{D}_2$ is the wild-type allele. Only the values for d and δ change.

1.2.4 Dynamics

The two-locus dynamics has to be described in terms of diploid genotype frequencies since zygotes (offspring) are generally not in Hardy-Weinberg proportions because of nonrandom mating. Genotypes are unordered. Let r represent an offspring genotype and g, h parental genotypes. The frequency of genotype r (among zygotes) in consecutive generations is denoted by p_r and p'_r. The frequency of r after (natural) selection is $p^*_r = p_r W_r/\overline{W}$, where $W_r = W(r)$ and $\overline{W} = \sum_r W_r p_r$ is the mean viability. After viability selection, mating and recombination occur. Let $R(gh \to r)$ designate the probability that parents with genotypes g and h produce a zygote with genotype r. $R(gh \to r)$ is determined by the pattern of recombination between loci.

The genetic dynamics is given by a system of 10 recursion relations that can be written as

$$p'_r = \frac{\overline{W}^2}{\widetilde{W}}\widetilde{W}_r\,, \tag{1.12a}$$

where

$$\widetilde{W}_r = \sum_{g,h} p^*_g p^*_h Q^*_{gh} R(gh \to r),$$

and $Q^*_{gh} = Q^*(g,h)$ (the asterisk indicates that Q is calculated from the genotypic frequencies after selection), and $\widetilde{W} = \overline{W}^2 \sum_r \widetilde{W}_r$. The demographic dynamics follows the standard recursion

$$N' = N\left(\widetilde{W}/\overline{W}\right). \tag{1.12b}$$

Thus, population growth follows equation (1.4) for a genetically monomorphic population that matches the optimum. For the full model, the complete evolutionary dynamics is given by the coupled system (1.12a) and (1.12b). We set $N' = 0$ (population extinction) if $\widetilde{W}/\overline{W} \leq 0$, and $p^*_r = 0$ if $W_r \leq 0$. For the quadratic model, population size is assumed to be constant and close to demographic equilibrium. The evolutionary dynamics are then given solely by(1.12a).

1.3 Theoretical background and analytical results

1.3.1 Components of selection

Before we start describing our results we shall first reflect upon the selection pressures involved in our model and how they interact. This will help us to guide the description and interpretation of our analytical and numerical results.

1.3.1.1 Direct selection

Direct selection acts on the ecological locus via four components. The first component is frequency-independent stabilizing selection, which favors phenotypes close to the optimum.

The second component is negative frequency-dependent selection induced by intraspecific competition, i.e., selection against common types. This favors polymorphism or, more precisely, sufficiently different phenotypes that do not suffer too much from competition. These phenotypes may be interpreted as being adapted to different ecological 'niches'. However, such a distribution cannot always be achieved, since hybrid offspring of two individuals adapted to different niches may be poorly adapted to any available ecological niche. A crucial distinction in our model is between moderate and strong competition. We call competition moderate if it induces two niches, each located at one boundary of the phenotypic range. In this case, viability selection is disruptive and the fitness function (1.5) is $\bigcup$-shaped. We refer to competition as strong if c is so large that the fitness function has two peaks not located near the boundary, or if it is even multimodal, i.e., if it has

multiple peaks. We will interpret the trait values corresponding to the fitness peaks as ecological niches. Since we allow for only two alleles at the ecological locus, we mainly focus on strengths of competition such that disruptive selection arises, i.e., $c \leq 2$.

The third component of selection is density-dependent selection resulting from population regulation. This selection component can alter the frequency-dependent effect of competition. For a given population distribution, the ratio of the fitnesses of an advantageous phenotype and a disadvantageous phenotype is larger in a population at high density than in a population at low density (cf. Bürger, 2005).

The fourth component of selection is positive frequency-dependent selection induced by assortative mating (cf. Kirkpatrick and Nuismer, 2004), which favors common phenotypes. Hence, in some sense assortative mating counteracts intraspecific competition. Even if females pay no costs of being choosy, as is assumed in our numerical investigations, one can interpret the selective disadvantage of males at low frequencies as costs of being rare. It should be mentioned that assortative mating does not counteract the modulating effect of density-dependent selection if individuals pay no costs of being choosy, since the population size is not reduced by assortative mating (i.e., $\widetilde{W} = \overline{W}^2$). This situation is different if individuals pay costs, i.e., for $M < \infty$.

1.3.1.2 Indirect selection

Direct selection on the ecological locus is translated into indirect selection on the modifier locus. In the symmetric case, i.e., if homozygotes are symmetric with respect to the optimum, stabilizing selection causes indirect selection for a reduced level of dominance. The reason is that heterozygotes are closer to the optimum at a reduced level of dominance than at an elevated level. In asymmetric cases, stabilizing selection causes indirect selection for dominance modifiers such that heterozygotes are shifted closer to the optimum.

Negative frequency-dependent selection can result in indirect selection for an increased or decreased level of dominance. It tends to modify dominance such that competition between individuals is reduced. Hence, in which direction this indirect component of selection acts depends crucially on the size of the effect of the modifier and the initial level of dominance.

Density-dependent selection has no indirect effect independent from that of frequency-dependent selection, because it acts jointly with intraspecific competition at the ecological locus, and hence also at the modifier locus.

Indirect selection pressures resulting from assortative mating can act for or against an increased level of dominance. They modify dominance to maximize the mating probabilities of males. However, there is a further important indirect effect of assortative mating. The strength of assortment determines how efficiently direct selection on the ecological

locus is translated into indirect selection for dominance modifiers. Since assortative mating reduces or (for very strong assortative mating) almost depletes the number of heterozygotes, all components of indirect selection become weaker under stronger assortative mating. Thus, it appears to be convenient to distinguish between two scenarios concerning the strength of assortative mating. If assortment is very strong, the frequency of heterozygotes will be very small at equilibrium. Thus, close to an equilibrium, dominance modifiers are almost selectively neutral. If assortment is at most moderately strong, i.e., ($a \leq 2$), heterozygotes will be present at sufficiently high frequency such that selection for modifiers can be induced.

1.3.2 Evolutionary scenarios

Depending on parameters, the various selection components can have different net impacts on the modifier locus, resulting in different evolutionary scenarios. In some of them an elevated level of dominance is selected against, in others it is favored at least in a part of the population. We want to mention here the most important situations that occurred in our numerical investigations.

Fixation scenario DS (due to disruptive selection). In this situation, the fitness of heterozygous individuals at the ecological locus that carry only the wild type allele at the modifier locus is lower than the fitness of those that carry at least one copy of the modifier. The fitness function is strongly $\bigcup$-shaped, i.e., there exist two niches at the boundaries of the phenotypic space (at -1 and +1), and the fitness at the optimum, 0, is strongly reduced (see Figure 1.1 (a)). Selection is disruptive and the heterozygotes expressing higher levels of dominance have an advantage. Therefore, the modifier allele goes to fixation. In this scenario, the driving force for fixation of the modifier is disruptive selection resulting from strong negative frequency-dependent selection.

Fixation scenario SS (due to sexual selection). In contrast to the previous case, here the driving force is sexual selection. The number of heterozygotes on the ecological locus is strongly reduced due to assortative mating and the viability fitness function is either very shallow or even $\bigcap$−shaped (see Figure 1.1 (e) and (f)). Heterozygotes carrying a modifier allele are shifted closer to one of the homozygotes. The higher chance of successful mating leads to fixation of the modifier despite a possible disadvantage in viability (see Figure 1.1 (f)).

Maintenance scenario MS. In this scenario, abbreviated in the following as **MS**, fitness is $\bigcup$-shaped and becomes asymmetric when a modifier invades, such that the minimum is close to the genotypic value of double heterozygotes (see Figure 1.1 (b)) and a broad phenotypic spectrum is favored. Therefore, the modifier will coexist with the wild type due to heterozygote disadvantage at the modifier locus. This reflects frequency-dependent disruptive selection at the modifier locus, and hence gives rise to further genotypic diversity. In this case, no single optimal value of dominance exists.

Loss scenarios L1 and L2. There exist two different scenarios for which the modifier gets lost. In the first one, abbreviated **L1**, both competition and assortative mating are weak and viability fitness is only slightly $\bigcup-$ shaped. However, positive frequency-dependence outweighs negative frequency-dependence and the modifier can not be maintained (see Figure 1.1 (c)). In the second scenario, abbreviated **L2**, the strength of assortative mating is moderate and the number of heterozygotes at the ecological locus is sufficiently reduced for the existence of a niche at the optimum of stabilizing selection (see Figure 1.1 (d)).

1.3.3 Evolution of allelic effects at the ecological locus

Before analyzing the evolution of dominance, we first investigate evolution at the ecological locus for a fixed level of dominance. We study whether polymorphic equilibria at the ecological locus can be invaded by mutant alleles (in this section, the terms mutant or mutant allele refer to a third allele at the ecological locus that is initially rare). This informs us whether a state is evolutionarily stable (see, e.g., Eshel, 1983). Moreover, the results of this section also justify the assumption of symmetric effects made in our numerical investigations in the following sections. In all cases we assume the modifier locus to be monomorphic. To derive analytical results we use the weak selection approximation for the fitness function (1.5), i.e., the quadratic model. Fitness of an individual with genotypic value g is then given by

$$W(g) = 1 - s g^2 + c(g - \bar{g})^2 V, \tag{1.13}$$

where $\bar{g}$ is the mean and V the variance of the phenotype distribution (cf. Bürger, 2005). We note that (1.13) is always $\bigcup$-shaped if $c > s$, provided $\bar{g}$ is sufficiently close to 0.

The robustness of the following results will be checked by numerical calculations using the full model described in Section 1.2. In all cases, we assume that the genotypic values of homozygotes lie on different sides of the optimum, i.e., one is positive and the other negative. In addition, we always assume that competition is stronger than stabilizing selection, i.e., $c > s$.

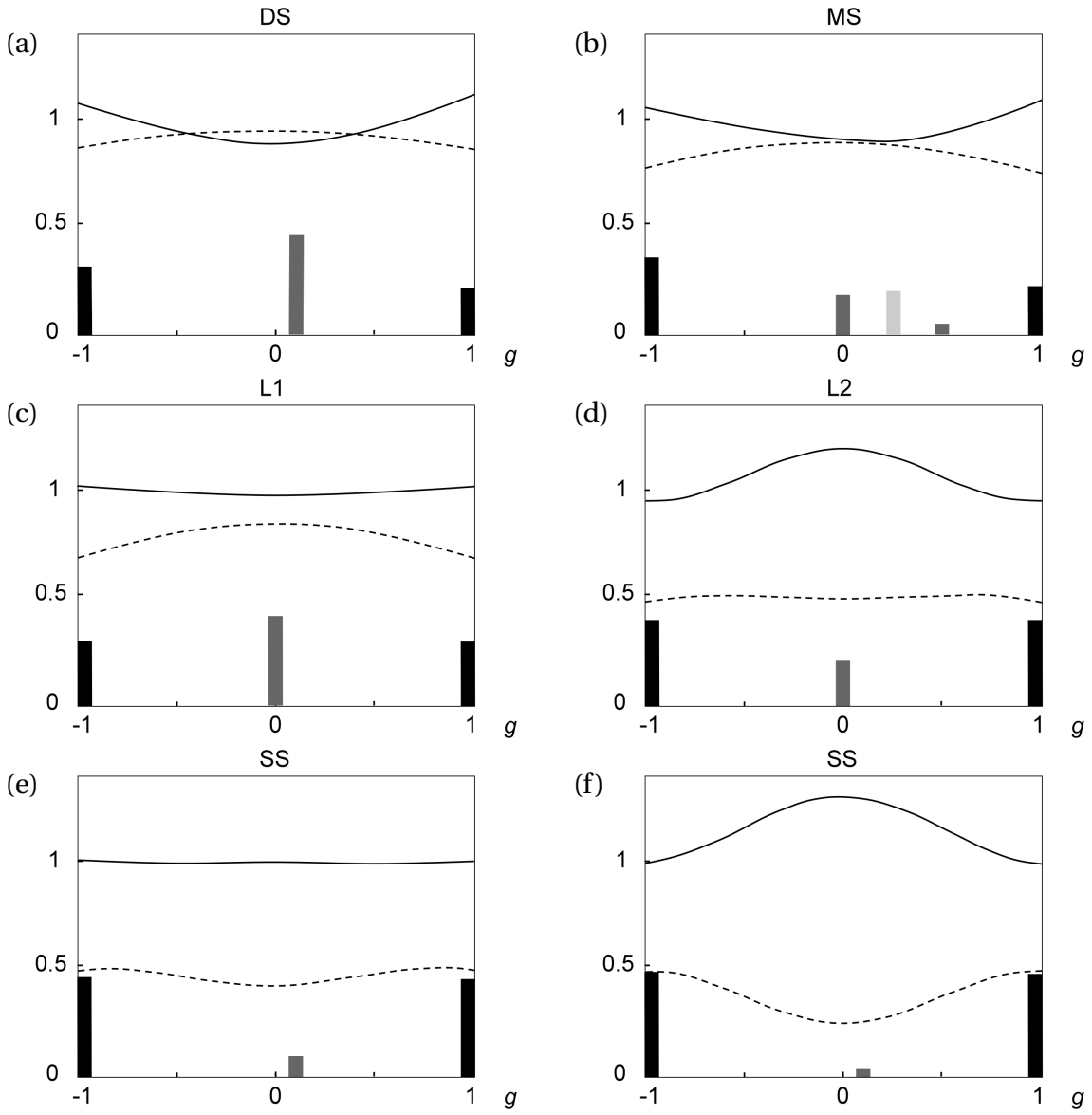

Figure 1.1: Viability $W(g)$ (solid line), mating probability $\bar{\pi}(g)$ (dashed line) and phenotype distributions (black and gray bars) at equilibrium for the different scenarios described in Section 1.3. Equilibrium frequencies of homozygotes on the ecological locus are indicated by black bars and frequencies of heterozygotes are indicated by gray bars. Frequencies of double heterozygotes are indicated in light gray and frequencies of individuals that are homozygous on the modifier locus but heterozygous on the ecological locus are indicated in a darker gray. Parameter values are $c = 0.7, a = 0.1$ in (a) (**DS**), $c = 1, a = 0.2$ in (b) (**MS**), $c = 0.2, a = 0.3$ in (c) (**L1**), $c = 1.5, a = 1$ in (d) (**L2**), $c = 0.5, a = 1$ in (e) (**SS**) and $c = 1, a = 1.5$ in (f) (**SS**). The effect of the modifier is $\delta = 0.05$ in (a), (c), (d), (e) and (f), and $\delta = 0.25$ in (b). Initial dominance is always 0.

1.3.3.1 No dominance, random mating

Here, we assume no dominance, random mating, and two alleles with allelic effects z_1 and z_2 ($z_1 < z_2$), respectively. Note that the allelic effects do not have to be symmetric in this

section. Then, the invasion fitness of a mutant allele $\mathscr{A}_3$ can be calculated straightforwardly and is given in Appendix A.1. There, it is shown that a mutant with effect z_3 can invade if and only if it widens the range of possible genotypic values, i.e., if and only if $z_3 \notin [z_1, z_2]$. Analogously, if we scale the range of possible genotypic values to [-1,1], an equilibrium with two alleles having effects $-1/2$ and $1/2$, respectively, cannot be invaded by any mutant and thus is an ESS. However, these results are derived under the assumption of weak selection and no density dependence. For the full model with density-dependent selection and fitness function (1.5) one finds via numerical investigation that these results remain valid for approximately $c \lesssim 2$ if $s = 0.1$. Thus, for the parameter c we focus on the interval $[0,2]$.

1.3.3.2 Complete dominance, random mating

Here, we assume one allele to be completely dominant, say $\mathscr{A}_1$, so that only two phenotypes are present at a polymorphic equilibrium with two alleles. For the mutant allele, $\mathscr{A}_3$, we consider two scenarios. In the first scenario, $\mathscr{A}_1$ is completely dominant over $\mathscr{A}_3$ and the genotypic value of $\mathscr{A}_2\mathscr{A}_3$ is arbitrary. The second scenario considers a mutant that is completely dominant in combination with any of the wild type alleles. Thus, $\mathscr{A}_1\mathscr{A}_3$ and $\mathscr{A}_2\mathscr{A}_3$ have the same genotypic value as $\mathscr{A}_3\mathscr{A}_3$. The trait value of $\mathscr{A}_3\mathscr{A}_3$ is arbitrary. In Appendix A.2, it is shown that, in both scenarios, mutants can only invade if they widen the range of genotypic values.

At a polymorphic equilibrium at which the full range of genotypic values is exploited, both phenotypes are present at equal frequencies. Thus, the frequency of the dominant allele is $1 - 1/\sqrt{2}$ and that of the recessive allele is $1/\sqrt{2}$. This is the only polymorphic equilibrium in this case and our invasion analysis shows that no mutant causing genotypic values within the range of possible genotypic values can invade.

1.3.3.3 No dominance, weak assortment

Here, we assume weak assortment and choose $\pi(Z_g, Z_h) = 1 - a(Z_g - Z_h)^2$. In addition, we assume no dominance. Then, neglecting second and higher-order terms in a, a diallelic polymorphism at the ecological locus can be calculated explicitly if we assume the allelic effects to be symmetric, i.e., $z_2 = -z_1$. The linearized transformation matrix for the genotype frequencies in the next generation under the assumption of rare mutants and its leading eigenvalue are given in Appendix A.3. There, it is shown that if competition is weak relative to the combined strength of stabilizing selection and assortative mating ($c < s + a/2$), mutants can only invade if their genotypic values lie within the range spanned by existing phenotypes. However, if competition is sufficiently strong, $c > s + a/2$, only mutants that widen the range spanned by existing phenotypes can invade. Thus, for strong competi-

tion and analogously to the previous cases, no mutant can invade if the boundaries of the phenotypic range are reached. Numerical calculations of the eigenvalues suggest that the results derived for random mating remain valid for asymmetric allelic effects if c is sufficiently large. Schneider and Bürger (2006) showed for a similar but haploid model that no monomorphic equilibrium can be stable under weak selection if $c > s + a/2$ (see also Schneider 2005, Bürger and Schneider 2006).

1.3.4 Invasion of dominance modifier if assortment is weak

In Appendix B, we analyze whether a modifier that increases dominance is able to invade an assortatively mating population expressing no dominance, provided assortative mating is weak. This is done under the assumption that $\pi(Z_g, Z_h) = 1 - a(Z_g - Z_h)^2$ by neglecting terms of order $O(a^2), O(s^2)$ and $O(as)$. Assuming a rare modifier, we provide the linearized matrix determining the frequency vector of genotypes that are heterozygous at the modifier locus in the next generation. We calculate the eigenvalues and show that, in the limit of weak selection and weak assortment, a modifier inducing an arbitrary degree of intermediate dominance can invade if and only if $c > s + a/2$.

1.4 Numerical methods

Because a detailed mathematical analysis of our model beyond the results derived above seems hardly feasible, we pursued an extensive numerical approach based on iterating equations (1.12a) and (1.12b). We performed two sets of calculations. In the first set the dominance modifier was assumed to initially segregate at arbitrary frequency in the population. In particular, the genotype frequencies are drawn from a uniform distribution and normalized. In the second set we assumed that the dominance modifier is initially rare, i.e., at very low frequency. We first drew random frequencies for the genotypes $\mathscr{A}_i\mathscr{A}_j/\mathscr{D}_1\mathscr{D}_1$ ($i, j \in \{1, 2\}$). Then, we added a fraction of 10^{-4} individuals, that were heterozygous at the modifier locus, to the vector of genotype frequencies proportionally to the previously drawn frequencies, i.e., assuming linkage equilibrium. To obtain the initial frequencies this vector was then normalized. Hence, initially, genotypes $\mathscr{A}_i\mathscr{A}_j/\mathscr{D}_2\mathscr{D}_2$ ($i, j \in \{1, 2\}$) were not present. For simplicity, we call the first set of iterations the *frequent-modifier* scenario, and the second situation the *rare-modifier* scenario.

Throughout our numerical investigations we assumed free recombination, i.e., the recombination rate is $1/2$, and we always chose the population growth rate to be $\rho = 2$. Moreover, because κ can be considered a scaling factor for the population size N, we did not choose it explicitly, and instead regarded N as normalized by the carrying capacity. We assumed that the initial population size matches exactly the carrying capacity, i.e., $N/\kappa = 1$.

Our model is fully determined by the parameter vector (s, c, a, d, δ). In both scenarios we used $s = 0.1$. The parameters a and c are as in the caption of Figures 1.2 (a) and 1.2 (b). Moreover, we chose various values for d and δ that are listed in the figure captions and in the description of our results. For each combination of the above parameters we chose ten different initial genotype distributions under both scenarios, subject to the constraint that the minimum Euclidean distance between any two different distributions is 0.2

For each initial distribution, we iterated the recursion relations (1.12a) and (1.12b) either until an equilibrium was reached, which was decided to be the case if the Euclidian distance between the vectors of genotype frequencies concatenated with the population size of two consecutive generations was less than 10^{-10}, or until 10^6 generations were reached. Such runs are referred to as *slow runs*. The reason was always slow convergence to equilibrium, not cyclical or chaotic behavior. For the slow runs we checked whether the criteria for numerical convergence are met when using an accuracy of 10^{-8}.

At equilibrium, we recorded the quantities of interest. These are the mating probabilities between different phenotypes, the genotype frequencies at equilibrium, the equilibrium population size, the genetic variance at equilibrium, and the speed of convergence. (Some quantities turned out to be uninformative, so we do not present results based on them.) For classifying the equilibria an allele was said to be present in the population if the frequency of at least one genotype carrying this allele was $> 10^{-4}$. Moreover, a genotype was considered to be present at equilibrium if its frequency was larger than 10^{-4}. For each parameter combination, we calculated various statistics: the average number of equilibria, the fraction of runs for which fixation, invasion/maintenance, or extinction of the modifier occurred, the fraction of slow runs, and the average speed of convergence.

For a given combination of parameters the evolutionary outcome usually depends on the initial conditions, i.e., the initial genotype distribution. To investigate this dependency we have recorded the number of different equilibria reached for different initial conditions. Moreover, disagreement between the outcomes in the frequent and the rare modifier scenario also provides information about different evolutionary outcomes. If for a given parameter combination, the same equilibrium is reached from all initial conditions in the frequent modifier scenario, and the same equilibrium is reached from all initial conditions in the rare modifier scenario, these equilibria do not necessarily coincide. If they do not coincide, this manifests the dependence on initial conditions.

To justify the assumption that the ecological locus is initially polymorphic we performed additional calculations. We numerically checked whether a polymorphic equilibrium at the ecological locus is reached for different degrees of initial dominance and assortative mating while the modifier locus was kept monomorphic. This was done for all considered parameter combinations. For each parameter combination we randomly picked ten initial genotype distributions and iterated the recursions until an equilibrium

was reached. We found only eleven cases in which convergence to a monomorphic equilibrium occurred. In each case the initial genotype distribution was already close to a monomorphic equilibrium and the strength of assortment was high. Convergence to a monomorphic equilibrium never occurred for all ten initial conditions.

1.5 Numerical results

Our main interest is to determine the fate of a modifier in populations with different degrees of assortative mating. A newly introduced modifier can either die out or be maintained. If a modifier is maintained, it can either rise to fixation or be maintained at intermediate frequency.

1.5.1 Maintenance, fixation and invasion of a modifier

The conditions under which a modifier can be maintained, can invade, or becomes fixed in the population depend in a highly nonlinear way on the involved parameters. This reflects a very subtle interplay between the various selective forces.

We start by describing the conditions under which a slightly increased level of dominance can be maintained if the wild-type allele codes for no dominance. The role of the effect of the modifier is investigated in more detail in Section 1.5.1.2, and different degrees of initial dominance are treated in Section 1.5.1.3. The case of initially rare modifiers is presented in more detail in Section 1.5.1.4

1.5.1.1 Maintenance of dominance (frequent modifier scenario)

Let us assume that the resident allele expresses no dominance and the modifier allele has effect $\delta = 0.05$. Then, the modifier can be maintained in large parameter regions (see Figure 1.2). However, it goes to fixation only in much smaller parameter regions. We will focus our attention especially on $a, c \leq 2$ (cf. Figure 1.2 (a)), which seems to be the most interesting and biologically most relevant parameter region.

For a randomly mating population ($a = 0$) and weak selection we know from Peischl and Bürger (2008) that a modifier invades and goes to fixation if selection is disruptive, i.e., if $c > s$, as is always satisfied in this study. In this model we correctly expect that this is also valid for small values of c. However, for larger values of c, this turns out not to be true in general, since sufficiently strong competition induces a more complicated viability fitness function and multiple ecological niches. More precisely, in the absence of assortment the fixation scenario **DS** (described in Section 1.3.1) applies if competition is moderate, i.e., the modifier replaces the resident allele if c is not too large (region **DS** in

Figure 1.2). If competition is strong, the maintenance scenario applies. Thus, for strong frequency dependence, the modifier will not get fixed, but is maintained at intermediate frequency due to disruptive selection at the modifier locus (region **MS** in Figure 1.2). These findings complement and extend the results of Peischl and Bürger (2008).

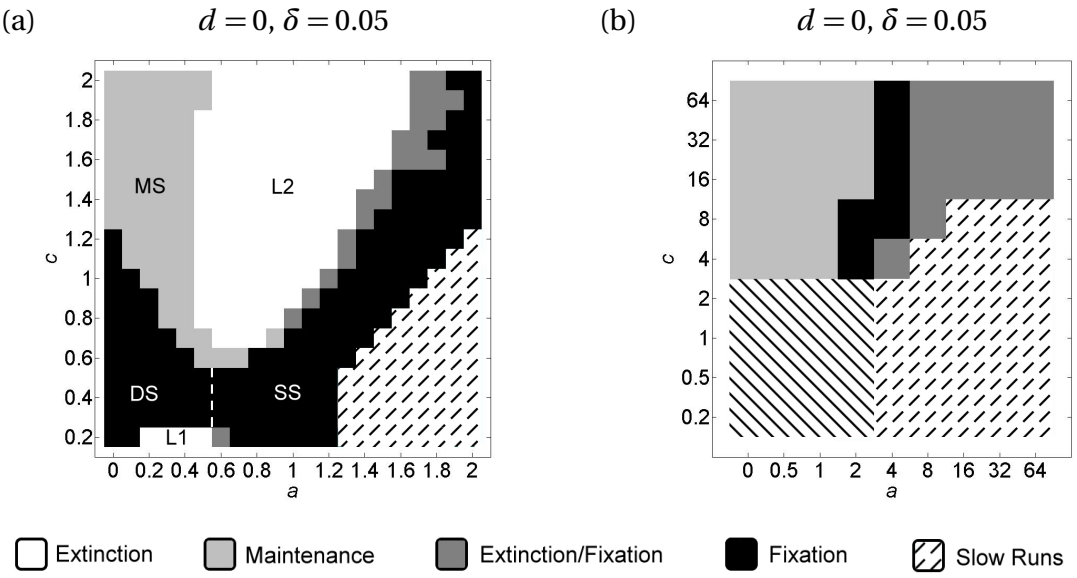

Figure 1.2: Regions of maintenance and fixation of a modifier increasing dominance slightly under the frequent modifier scenario with no initial dominance. Figure (a) uses a grid with step size 0.1 for the interval $[0,2]$ for the parameter a and a grid with step size 0.1 for the interval $[0.2,2]$ for the parameter c. Figure (b) uses a logarithmic scale for the parameters a and c, namely $a = 0$, 0.5, 1, 2 4, 8, 16, 32, 64, and $c = 0.2$, 0.5, 1, 2, 4, 8, 16, 32, 64. Moreover, the parameters $s = 0.1$, $r = 0.5$, $d = 0$ and $\delta = 0.05$ are assumed in both figures. The color code indicates the different evolutionary outcomes. In the extinction regions the modifier died out in all runs. In the maintenance regions the modifier coexisted with the wild type in all runs, whereas in the fixation region the modifier was fixed for all runs. Parameter combinations for which none of the runs equilibrated within 10^6 generations are indicated as slow run regions. The hatched area in (b) corresponds to (a).

Assortative mating induces positive frequency dependence and counteracts negative frequency dependence resulting from competition. This occurs through selection for common types, and by reducing the number of heterozygotes at the ecological locus and thus reducing the average amount of competition experienced by the individuals. Hence, assortative mating counteracts disruptive selection in two different ways. If assortment is weak ($a \lesssim 0.5$ in Figure 1.2 (a)), the maintenance scenario also applies for smaller values of c than for random mating since the number of heterozygotes is reduced. If competition is also weak, positive frequency dependence due to assortative mating 'neutralizes' disruptive selection. Therefore, stabilizing selection is the dominating force, and heterozygotes expressing no dominance (i.e., those at the optimum of stabilizing selection) are most advantageous. As a consequence, the modifier dies out in the region **L1** even if initially at

high frequency.

For $0.5 \lesssim a \lesssim 1.6$ there exists another region in which the modifier gets lost. The reason is the following: if c is neither too small nor too large (region **L2** in Figure 1.2 (a)), an ecological niche will be established at the optimum of stabilizing selection (see Figure 1.1 (d)). In addition, heterozygotes on the ecological locus are sufficiently frequent that a shift closer to one of the homozygotes does not significantly increase the chance for successful mating. Thus, stabilizing selection dominates, which leads to an advantage for heterozygotes at the optimum and consequently to extinction of the modifier.

If competition is too weak to establish a niche at the optimum of stabilizing selection, positive frequency dependence dominates. Therefore, heterozygotes at the optimum become too rare to be selectively advantageous and the fixation scenario **SS** applies (see Figure 1.1 (e)). However, for large a the fixation scenario **SS** also applies for larger values of c, such that a niche at the optimum is established (see Figure 1.1 (f)). Since heterozygotes are extremely rare, their chance for successful mating is extremely low. Shifting the phenotypic value of heterozygotes closer to one of the homozygotes leads to a substantial gain in mating success and consequently to fixation of a modifier increasing dominance. There exists a small transition region, in which assortment neutralizes competition and either extinction or fixation occurs, depending on the initial conditions. The strength of assortment in this transition region depends linearly on c (cf. Figure 1.2 (a)).

Since strong competition ($c \gtrsim 2$ in Figure 1.2 (b)) preserves large amounts of polymorphism and the location of the niches is no longer restricted to the boundary of the phenotype space, the modifier is maintained for sufficiently strong competition (**MS** region in Figure 1.2 (b)). Even if one of the niches is located at the optimum of stabilizing selection, the heterozygotes expressing dominance fill their own niche. Hence, the modifier will not go extinct.

If both competition and assortment are very strong ($a, c \geq 2$), all heterozygotes (at the ecological locus) have similar viability and positive frequency dependence dominates. Because positive frequency dependence implies selection for common types, the modifier will either be fixed or extinct depending on initial conditions. This is the reason for the extinction/fixation region in Figure 1.2 (b).

For moderately strong assortment ($a = 2,4$) a large proportion of slow runs occurs. The reason is slow convergence caused by very weak indirect selection. In addition, for very strong assortment ($a > 4$) and at most intermediate competition ($c \lesssim 4$) modifiers are almost neutral and the numerical criterion for equilibration can be met before the actual equilibrium is reached. No cycling or other complicated behavior was detected. Modifiers of sufficiently small effect are always maintained and even go to fixation for certain (large) values of c.

Summarizing, a modifier with small effect will be maintained in the population (if suf-

ficiently frequent) except in a small region of very weak competition and very weak assortment (region **L1**, $c \approx 0.2$ and $0.2 \lesssim a \lesssim 0.5$) and a region in which competition and assortment are weak to intermediate (region **L2**, $\max(0.7, 0.4+2a) \lesssim c \lesssim 2$ and $0.5 \lesssim a \lesssim 2$).

1.5.1.2 Size of the modifier effect

The above results are qualitatively robust to the size of the modifier effect. However quantitatively, the regions in the parameter space corresponding to different evolutionary outcomes change considerably (as can be seen by comparing Figures 1.2 and 1.3).

The fixation region under random mating or weak assortment (fixation scenario **DS**) becomes smaller for larger δ (cf. **DS** regions in Figures 1.2 (a), 1.3 (a) and 1.3 (c)). This can be explained by the $\bigcup$-shape of the fitness function. The minimum of the fitness function lies within the range of phenotypic values of heterozygotes (at the ecological locus). For small modifier effects it is more likely that the fitness function stays $\bigcup$-shaped in this region, and individuals carrying the modifier allele are advantageous. For larger effects, heterozygotes carrying only the modifier alleles compete stronger with one of the homozygotes. Thus, their fitness is reduced. Consequently, it is more likely that individuals that are heterozygous at both loci are at a fitness minimum. This leads to fixation of modifiers with smaller effects and to maintenance of modifiers with larger effects.

The **MS** region is almost independent of the modifier effect δ (compare Figure 1.2 (a) and Figure 1.3 (a), (c)). For an explanation, note that, in the **MS** region, the fitness function is U-shaped (as we assume $c \leq 2$), and in the absence of a modifier, the optimum is associated with a fitness minimum. In the presence of a modifier, this minimum is shifted towards the double heterozygote, leading to disruptive selection at the modifier locus (see Section 1.3.2 and Figure 1.1 (b)). In contrast, in the **L2** region (for sufficiently large a), the optimum is associated with a fitness maximum (see Figure 1.1 (d)), and no disruptive selection is induced at the modifier locus. Thus, whether the **MS** or the **L2** scenario applies (for small a) depends only on whether there is a fitness minimum or maximum at the optimum in the absence of a modifier. This is, of course, independent of δ. In addition, if $c \geq 4$, there are several ecological niches, and each phenotype fills its own niche, independent of the modifier size. Thus, the maintenance regions are also independent of δ for large c.

The region **L2**, in which extinction of a modifier occurs if competition and assortment are intermediate, increases with increasing δ (cf. Figures 1.2 (a) and 1.3 (a)). The explanation is simple. As described in Section 1.5.1.1, assortment reduces the number of heterozygotes such that they have a selective advantage. This is even amplified by larger δ since competition becomes weaker at the optimum and stronger at the boundary of the phenotypic range.

If δ increases, the **SS** region loses ground to the region **L2**. Apparently, the reason for this is the trade-off between viability and mating chance (see Figure 1.1 (f)). On the other

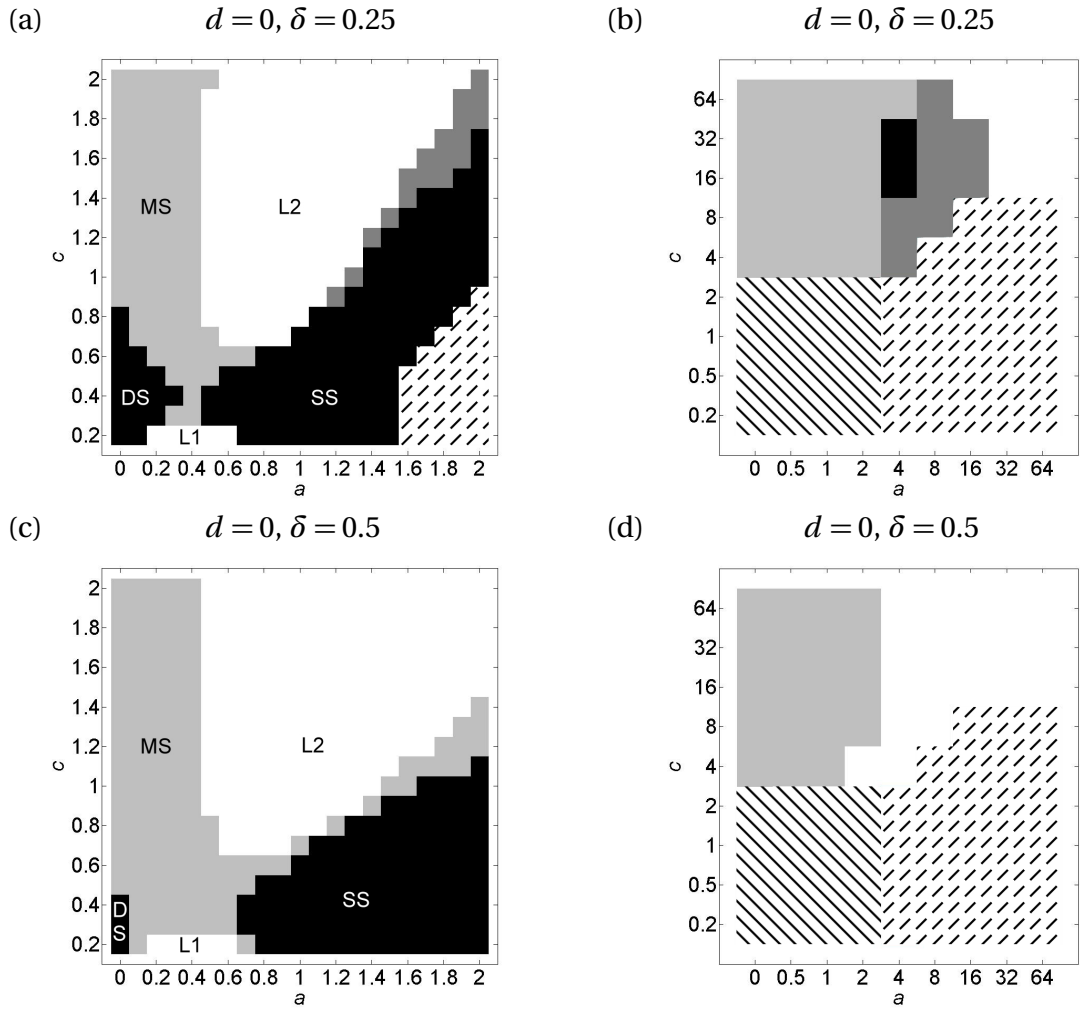

Figure 1.3: Regions of maintenance and fixation of dominance modifiers of large effects under the frequent modifier scenario. Initially there is no dominance, i.e., $d = 0$. The values for the parameters a and c were chosen as in Figure 1.2 and we used $s = 0.1$ and $r = 0.5$. Moreover, $\delta = 0.25$ in figures (a) and (b), and $\delta = 0.5$ in figures (c) and (d). The color code for the different regions is as in Figure 1.2. The hatched areas in (b) and (d) correspond to (a) and (b), respectively.

hand, the region **SS** fills up some part of the slow-runs region if δ increases. Modifiers with larger effect are under stronger selection, and hence, slow runs are less likely. (Note that slow runs cannot be regarded as pertaining to the fixation regions, because modifiers are not fixed within a biologically meaningful time.) Thus, in total the **SS** region increases with increasing δ.

For strong competition and strong assortment, the conditions for fixation of a modifier become more restrictive with increasing δ. The modifier must be sufficiently frequent to become fixed. Otherwise it will be lost. The larger the modifier effect, the more likely the modifier will go extinct, even if it is initially very frequent (cf. Figures 1.2 (b) and 1.3 (b) and

(d)). The reason is that, because of strong assortment, the proportion of heterozygotes (at the ecological locus) is low. If all heterozygotes have similar phenotypic values, the costs of being rare for males are reduced. For larger δ, these costs rise. Hence, the number of heterozygotes is reduced by removing either the wild type or the modifier, depending on which one is initially more frequent. Additionally, competition for one of the homozygotes is reduced by eliminating a modifier of large effect. This becomes especially pronounced for large modifiers (region of extinction/fixation in Figures 1.3 (b) and (d)).

1.5.1.3 Dependence on initial level of dominance

The initial level of dominance has a strong effect on the evolutionary outcome. To understand this, it is helpful to compare two situations. In the first situation, assume a given initial level of dominance and a modifier with small effect. In the second situation, assume no initial dominance and a modifier with large effect that leads to the initial level of dominance of the first situation if the modifier becomes fixed.

As seen from comparison of Figures 1.3(a) and 1.4(a), the regions of fixation of the two situations are similar. (Note that fixation of a modifier with effect $\delta = 0.25$ leads to $d = 0.5$.) From the second situation we know that a high level of dominance is favorable. In the **DS** regions the fitness function is $\bigcup$-shaped, and the modifier becomes fixed because viability of heterozygotes on the ecological locus is increased if they experience a higher level of dominance. Hence, in the first situation, fixation of the modifier will occur as long as the fitness function remains $\bigcup$-shaped. Because of increased competition with one of the homozygotes, the modifier decreases the slope of the fitness function. Hence, there is an 'optimum' degree of dominance, i.e., the largest degree of dominance that does not change the sign of the slope of the viability fitness function. The modifier will go to fixation if it establishes a level of dominance that is closer to the optimum level. Otherwise it will go extinct. The latter occurs especially for large degrees of initial dominance (see Figure 1.4 (c) and (d)). Therefore, this is also a reason why the regions of extinction become larger. In the **SS** regions, modifiers become fixed because a higher degree of dominance increases the chance of successful mating. Therefore, selection for modifiers with large effects in a background with no initial dominance will be more efficient than for modifiers with small effects in a comparable situation with large initial dominance (note that in the latter situations all heterozygotes are close to each other) (cf. **SS** regions in Figures 1.3 (a) and 1.4 (a), (c)). This suggests that, if assortment is intermediate, modifiers with larger effects can establish higher levels of dominance than a series of modifiers with small effects

The largest effect of the initial level of dominance is on the **MS** region (cf. **MS** regions in Figures 1.2 (a), 1.3 (a), (c) with Figures 1.4 (a), (c)). As described in section 1.3.2, the polymorphism at the dominance locus is maintained by disruptive selection. As long as the fitness is $\bigcup-$shaped ($c \leq 2$) this selective force will be readily established without initial

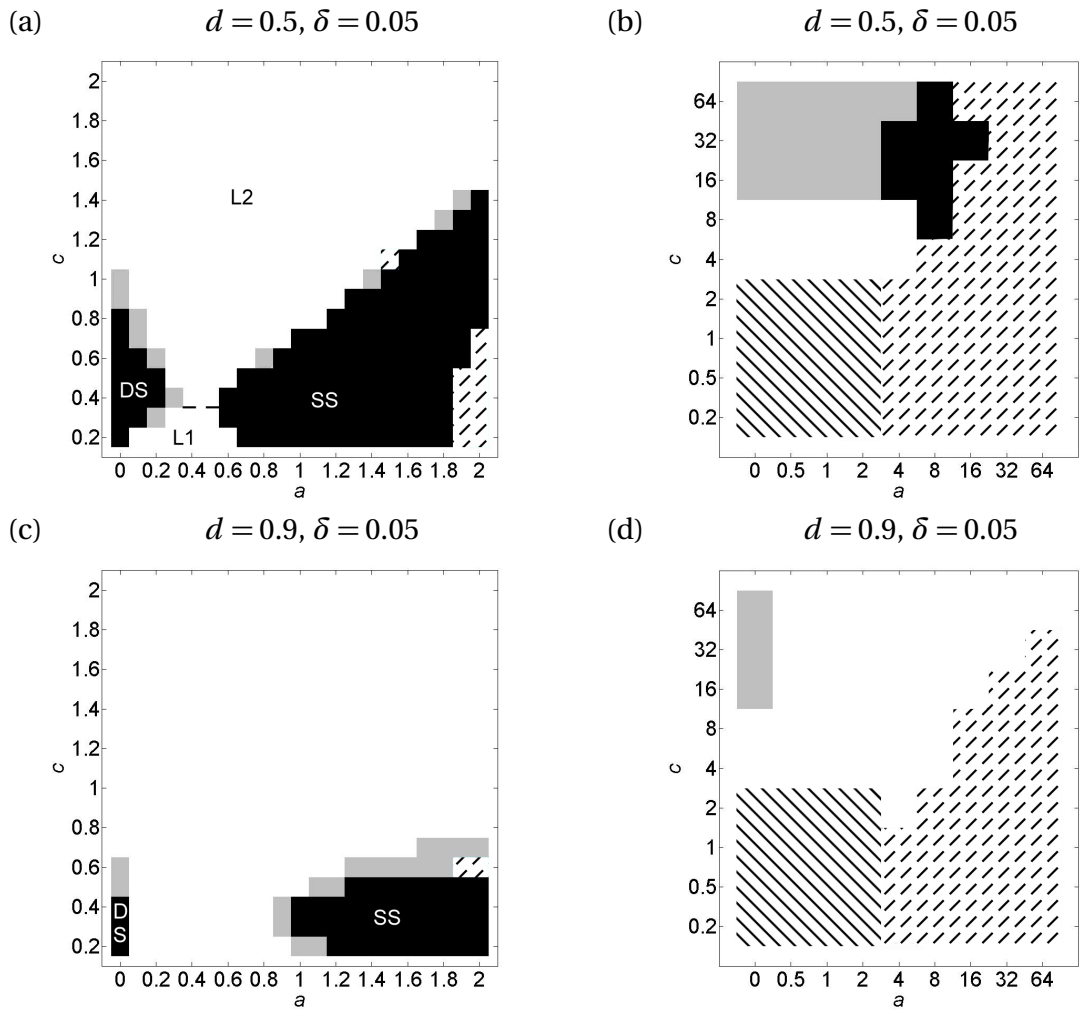

Figure 1.4: Regions of maintenance and fixation of a dominance modifier of small effect ($\delta = 0.05$) under the frequent modifier scenario with different degrees of initial dominance. The parameters a, c, s and r are as in Figure 1.2. We have $d = 0.5$ in figures (a) and (b), and $d = 0.9$ in figures (c) and (d). The color code for the different regions is as in Figure 1.2. The hatched areas in (b) and (d) correspond to (a) and (b), respectively.

dominance but hardly with it. The reason is the following: In the absence of dominance the fitness minimum matches the optimum, i.e., the heterozygotes are exactly at the fitness minimum. In the presence of a modifier, the fitness minimum is shifted close to phenotypic value of the double heterozygotes (Figure 1.1(b)), resulting in disruptive selection at the modifier locus. In contrast, for a positive initial degree of dominance, the fitness minimum lies between the (ecological) heterozygote and one of the homozygotes. If a modifier increasing dominance is introduced, the fitness minimum is closer to the heterozygotes carrying two copies of the modifier than to the double heterozygotes (as long as the modifier effect is not too large; data not shown). Hence, no disruptive selection is induced at

the modifier locus if modifier effects are small.

A similar explanation holds for $2 \lesssim c \lesssim 8$. If c is sufficiently large the modifier will be maintained since numerous niches exist that will be filled. However, the larger the initial degree of dominance, the less likely it becomes that heterozygotes fall into different ecological niches (because a modifier increasing dominance must be small) (cf. light grey region in Figures 1.2 (b), 1.3 (b), (d) with Figures 1.4 (b), (d)). Hence the changes in the maintenance regions are less pronounced for large c.

Note further, that, especially for large a, modifiers of small effect are easier maintained because heterozygotes at the ecological locus pay lower costs of being rare if their phenotypic value is closer to one of the homozygotes. This is the reason why the regions of fixation for large a and c are larger in the situation in which an initial level of dominance is already established, than in comparable situations with no initial dominance.

1.5.1.4 Frequent vs. rare modifier

In the rare modifier scenario, modifiers can get lost, invade or become fixed. As expected, the region of extinction becomes larger if the modifier is initially rare. This is most pronounced for moderate or strong assortment (see Figure 1.5). The reason is that the modifier is selected against because of costs of being rare caused by assortative mating. In other words, for stronger assortment, there exist multiple stable equilibria (especially at the boundaries) that may have a small region of attraction. Under the rare modifier scenario, initial frequencies are more likely to lie within the small regions of attraction of the boundary equilibria.

If assortment is at most moderately strong ($a \lesssim 1.2$), modifiers that will be maintained if already present at sufficiently high frequency are also able to invade (compare Figures 1.2 (a) and 1.3 (a) with Figures 1.5 (a) and (c), respectively). In other words, the evolutionary outcomes under the frequent and rare modifier scenarios coincide if assortment is not too strong (cf. Figures 1.2 and 1.5). The reason is that for weak or moderate assortment often a globally stable equilibrium exists, and hence the initial frequency of the modifier does not matter for the evolutionary outcome. Mainly, the regions in which a modifier either goes to fixation or dies out in the frequent modifier scenario are replaced by extinction regions in the rare modifier scenario.

The size of the modifier effect and the initial degree of dominance have a strong influence on the above results. Although the results are qualitatively robust for all considered modifier effects and initial degrees of dominance, differences in the evolutionary outcome under the frequent and rare modifier scenarios become less pronounced with increasing modifier effect or initial degree of dominance (cf. Figures 1.3, 1.4 and 1.5). The reason is that, if the phenotypic value of heterozygotes carrying a modifier is shifted closer to the phenotypic value of one of the homozygotes, the probability of mating with a homozygote

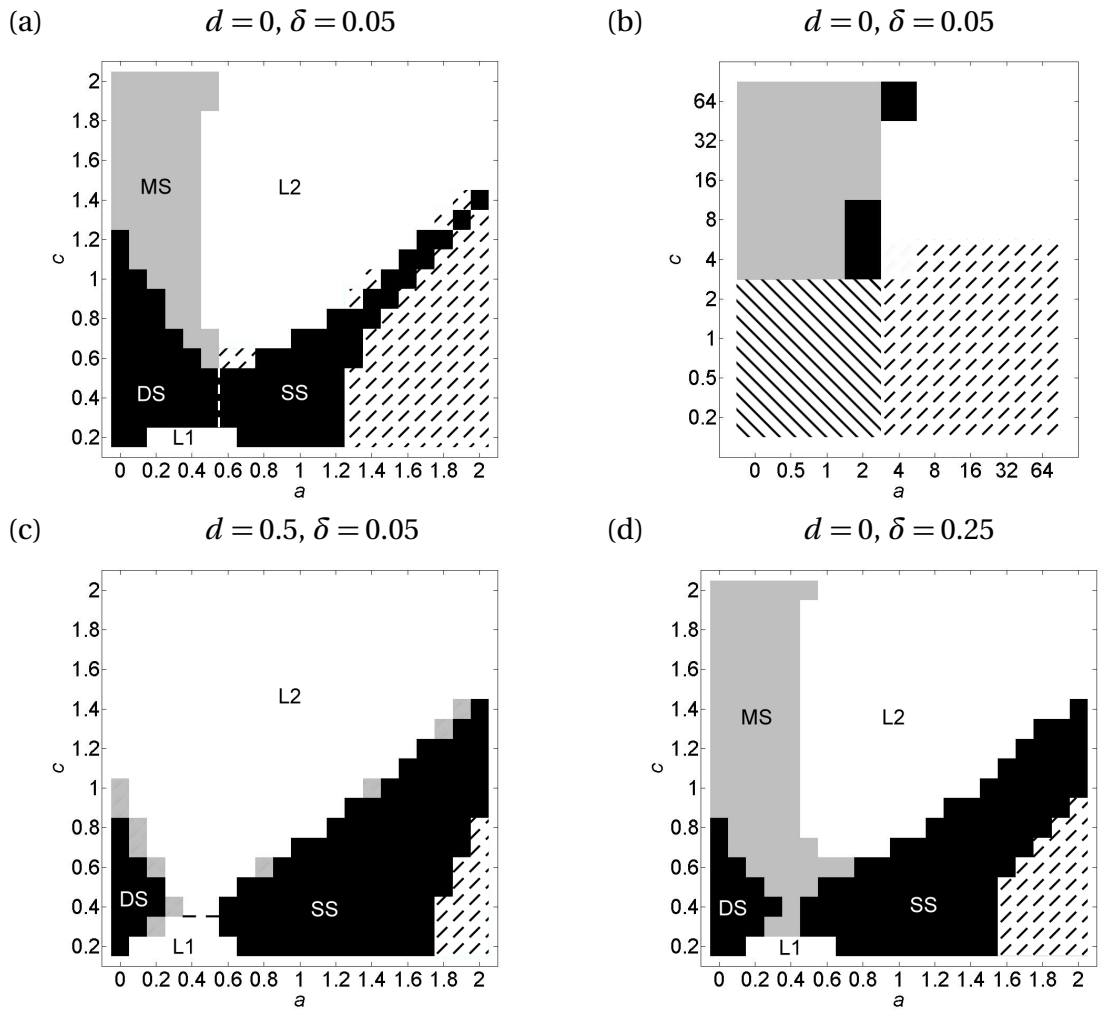

Figure 1.5: Regions of invasion and fixation of modifiers under the rare modifier scenario. The color code for the different regions and the parameters c, a, s and r are as in Figure 1.2. In Figures (a) and (b) we chose $\delta = 0.05$ and no initial dominance. In figure (c) we chose $\delta = 0.05$ and $d = 0.5$. In figure (d) we chose $\delta = 0.25$ and $d = 0$. The hatched area in (b) corresponds to (a).

increases. Consequently, the costs of being rare decrease for individuals that carry a modifier.

In addition, we can use the fixation time of an initially rare modifier as an estimate for the strength of selection for a modifier. In Figures 1.6 (a) and (b) the mean fixation time of a rare modifier is shown for small and large modifier effects, respectively. If the modifier effect is small, $\delta = 0.05$, the sweep is always fastest under random mating (see Figure 1.6 (a)). This is not surprising since the number of heterozygotes goes down with increasing assortment and thus the translation of selection to the modifier locus becomes less efficient. In addition, sexual selection is very weak for modifiers with small effects. If modifier effects are large, $\delta = 0.5$, especially sexual selection becomes stronger and evolu-

tion of dominance can be faster in the fixation region **SS** than under random mating (see black outlined region in Figure 1.6 (b)). However, if assortment is too strong, heterozygotes are too rare to establish strong selection for dominance and evolution becomes very slow. Noteworthy, if $\delta = 0.5$, the fastest as well as the slowest sweep always occurred for some $a > 0$. Apparently, in the fixation region **SS**, there is a trade-off between the intensity of sexual selection, which increases with increasing a, and the translation to the modifier locus, which is most efficient for random mating.

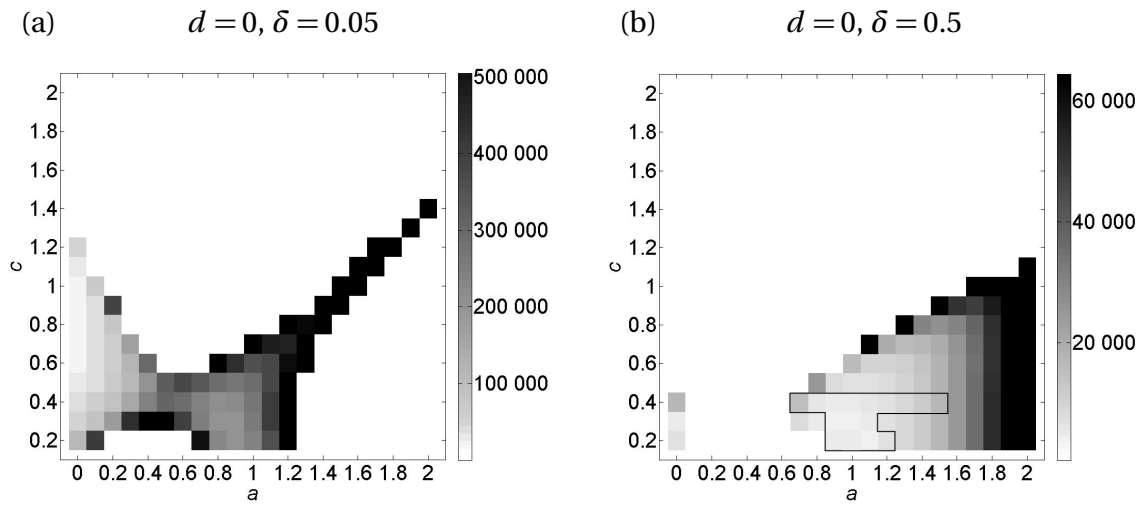

Figure 1.6: Time until fixation of an initially rare modifier. The modifier has effect $\delta = 0.05$ in (a) and $\delta = 0.5$ in (b). The black outlined region in Figure (b) indicates the parameter region in which a modifier gets fixed faster than in the corresponding region under random mating.

1.5.2 Evolutionary outcomes

In the numerical study, the same equilibrium was reached for all initial conditions in a large parameter region. However, in the regions in which a modifier either invades or goes extinct obviously at least two evolutionary outcomes are possible. In fact, exactly two were observed. For strong assortment ($a \geq 8$), almost always more than two equilibria were observed. This is because positive frequency dependence causes boundary equilibria to be stable. For 'extreme' parameters it also seems to be a numerical artefact. Because of reduced heterozygosity, indirect selection at the modifier is almost absent, such that the modifier becomes selectively nearly neutral. Hence, the direct selection process acting on the ecological locus is much faster and, after an 'equilibrium' is reached at the ecological locus, the selection process is so weak that the numerical criteria for equilibration are met, although an equilibrium is not reached yet. Indeed, if assortment is strong, for a large parameter region, most trajectories 'converge' to different equilibria.

1.5.3 Evolution of dominance

An interesting question is whether it is possible that complete dominance evolves by repeated invasion and fixation of modifiers in a population that initially expresses no dominance. Our numerical iterations for the rare modifier scenario provide a partial answer to this question.

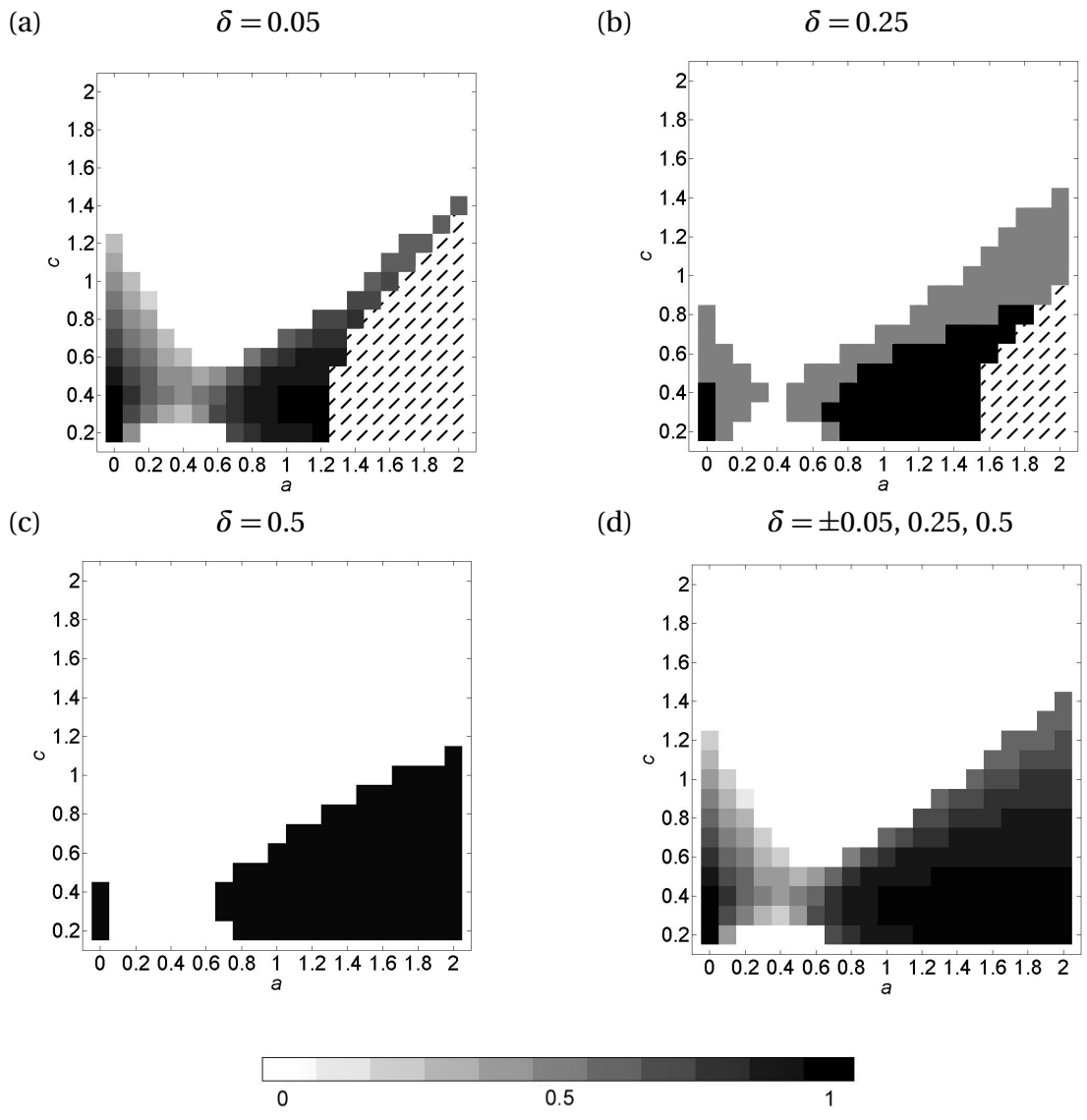

Figure 1.7: Maximal degree of dominance that can be achieved through recurrent invasion and fixation events of modifiers. The parameters a, c, s and r are as in Figures 1.2. The size of the modifier effect is fixed in figures (a), (b), and (c). Figure (a) uses $\delta = 0.05$ (10 steps to complete dominance), in (b) $\delta = 0.25$ (2 steps to complete dominance), and in (c) $\delta = 0.5$ (complete dominance in a single step). Figure (d) shows the degree of dominance that is evolutionary stable if modifiers of different positive and negative effects are considered. Black corresponds to complete dominance and white to no dominance, different shades of grey correspond to different degrees of intermediate dominance.

We obtain a parameter region in which dominance is able to evolve if we take the intersection of the parameter regions in which the modifiers become fixed for the various initial levels of dominance. For instance, if we build the intersections of the regions in which a modifier of effect 0.05 becomes fixed for the initial dominance levels 0 to 0.9 in steps of size 0.1, we obtain a region in which complete dominance can evolve via a series of mutations of small effect. (Note that if a modifier of effect 0.05 is fixed, the amount of dominance expressed by the heterozygotes is increased by 0.1 and complete dominance requires ten substitutions). Similarly, a 'maximum' degree of dominance can be obtained (see Figure 1.7 (a)).

Complete dominance can evolve mainly for intermediate assortment. The region in which complete dominance is able to evolve grows with larger modifier effects (see Figures 1.7 (a), (b), (c)). The reason is that complete dominance is often not the 'optimum' level of dominance. It is more likely that the optimal degree of dominance is reached by invasion and fixation of modifiers with small effect. Then, no further increase in dominance is possible. This fine tuning is not possible with modifiers having large effects, e.g., $\delta = 0.25$ or $\delta = 0.5$ (see Figures 1.7 (b) and (c)).

In Figure 1.7 (a) - (c) we considered only evolution by invasion and fixation of modifiers that have the same effect, i.e., $\delta = 0.05$, $\delta = 0.25$ or $\delta = 0.5$ in Figures 1.7 (a), (b) or (c), respectively. One could also study the evolution of dominance by invasion and fixation of a sequence of modifiers that have different effects. If we restrict attention to modifiers that increase the level of dominance, our simulations yield exactly the same picture as Figure 1.7 (c). This suggests that the black region in Figure 1.7 (c) is an estimate for the maximum parameter region in which complete dominance can evolve via allele substitutions. If, in addition, we allow modifiers decreasing dominance and construct the level of dominance for which no further evolution is possible, our data yields Figure 1.7 (d). The main difference between Figures 1.7 (a) and (d) is that dominance can evolve in the region of slow runs of Figure 1.7 (a) because modifiers with large effect experience stronger selection.

The above approach is not exact and it is conservative. It is not exact, because although we assume that the modifier is initially at very low frequency (which is a necessary assumption if one considers a mutational process) the population is not necessarily near an equilibrium. It is conservative because it yields only the parameter region in which complete dominance can evolve through a series of substitutions at the modifier locus.

1.6 Discussion

In the last decade, the study of divergence within populations and eventual sympatric speciation has become very popular, e.g., Doebeli (1996); Dieckmann and Doebeli (1999); Kon-

drashov and Kondrashov (1999); Matessi et al. (2001); Turelli et al. (2001). In these models, a quantitative character is maintained polymorphic by frequency-dependent disruptive selection. The African finch *Pyrenestes ostrinus* (e.g., Smith, 1990; Slabbekoorn and Smith, 2000) was often cited to justify the ecological setup of these model. However, in *Pyrenestes ostrinus*, evolution of assortative mating did not occur. Instead, the problem of unfit heterozygotes is solved by one morph being completely dominant.

Evolution of higher levels of dominance and of assortative mating are two mechanisms that can remove unfit heterozygotes. Since, in the literature, traditionally these mechanisms are studied separately, it is natural to regard them as alternatives (cf. Durinx and van Dooren, 2008). However, in this work we emphasize the importance of the interplay between the two mechanisms.

We studied the evolution of dominance modifiers on a quantitative trait in an assortatively mating population. In our model this trait is assumed to be determined by a single diallelic locus. The trait is under a mixture of stabilizing and negative frequency-dependent selection. The latter results from intraspecific competition with respect to similarities in the trait under consideration. We further assumed the effects of the alleles to be symmetric with respect to the optimum of stabilizing selection. The mating behavior follows the model of Matessi et al. (2001), which was originally formulated by Gavrilets and Boake (1998). More precisely, choosiness is expressed in females only, who pick their mating partners based on similarities in their trait values. Hence, we studied a so called 'magic trait' model (cf. Gavrilets, 2004). The dominance modifiers occur at a second unlinked locus and alter the dominance relation between the resident alleles at the first locus.

In our model, a mixture of four different components of selection is acting on the ecological locus. Selection for modifier alleles that induce different degrees of dominance is indirect and transmitted by heterozygotes at the ecological locus. Since we assumed weak stabilizing selection, the two most important forms of selection are negative and positive frequency-dependent selection, induced by competition and assortative mating, respectively. Negative frequency dependence favors sufficiently different and rare types over common ones, whereas positive frequency dependence induces costs of being rare. However, assortative mating also changes the number of heterozygotes on the ecological locus and thus the amount of competition experienced by the individuals as well as the efficiency of the transmission of selection from the ecological locus to the modifier locus. Due to these interactions, it is not intuitively clear which components of selection are responsible for the final outcome. We teased apart these components and identified the selective forces that lead to the different outcomes in the various parameter regions.

Under the assumptions of no dominance, weak selection and weak assortative mating, we were able to derive simple conditions on the strengths of assortment, competition, and stabilizing selection under which a modifier inducing an arbitrary degree of intermediate

dominance is able to invade. From this condition, $c > s + a/2$, one can see that small degrees of assortment counteract disruptive selection. This condition is also valid in the full model with population regulation, provided assortment is weak.

Because of the complexity of the model, the establishment of analytical results beyond the ones provided seems hardly feasible. Hence, we complemented our analytical results by extensive numerical investigations. Our main focus was on the small-parameter scale, where $0 \leq a \leq 2$ and $0.2 \leq c \leq 2$. Additionally, we investigated more extreme parameters on a logarithmic scale. Depending on parameters, a modifier allele can be either lost, be maintained at a intermediate frequency or go to fixation. More precisely, we described five different scenarios (section 1.3.2). If a frequent modifier becomes fixed, it is not necessarily able to invade a population at low frequency. For weak assortment ($a \lesssim 0.5$) and for intermediate assortment ($0.5 \lesssim a \lesssim 4$), there exist parameter regions in which dominance modifiers are able to invade and substitute the resident allele. These regions depend strongly on the size of the modifier effect and the initial level of dominance.

If assortment is absent or weak ($a \lesssim 0.5$) and competition is moderately strong, the fixation scenario **DS** applies. Weak assortative mating induces stabilizing selection around the phenotypic value of heterozygotes on the ecological locus that carry only the wild type allele at the modifier locus, i.e., selection against modifier alleles. However, the effects of assortative mating are outweighed by the $\bigcup$-shape of the viability fitness function. The fixation region **DS** decreases with increasing modifier effect since disruptive selection gets weaker with an increasing number of individuals expressing dominance and an 'optimal' level of dominance in the population might be reached before the modifier becomes fixed. Similarly, the region decreases with increasing initial dominance. Since viability is the driving force here, it is not surprising that the initial frequency of the modifier has no significant effect. This region vanishes for $a > 0.5$ and disruptive viability selection can no longer be established at the ecological locus because heterozygotes are so rare that they experience very little competition. This illustrates, as intuitively expected and predicted by analytical results for weak selection, that small degrees of assortative mating counteract disruptive selection.

Another component of selection, namely positive frequency dependence, can still induce selection for higher levels of dominance if assortment is intermediate ($0.5 \lesssim a \lesssim 4$). In this fixation scenario (**SS**), modifiers can invade and go to fixation if viability fitness is very shallow or even $\bigcap$-shaped. Individuals that are heterozygous at the ecological locus are rare, but not too rare to translate selection efficiently to the modifier locus. Because of the reduced number of heterozygotes, they experience very little competition and thus may have an advantage in terms of viability. However, they pay severe costs of being rare. Even a small shift closer to one of the homozygotes can lead to an increased mating success that outweighs the viability disadvantage and thus leads to fixation of the modifier. Of

course, modifiers with larger effects get fixed more easily in this scenario.

Modifiers coexist with the wild-type if disruptive selection on the modifier locus is established (**MS** region). On the logarithmic scale this occurs in a much larger parameter region than that in which it becomes fixed. On the small scale the fixation region is usually larger or at least as large as the maintenance region.

Furthermore, we investigated the regions in which the modifier is getting lost because no further increase in dominance can be established. In fact no dominance at all is favored in these parameter regions. Two different scenarios corresponding to different parameter regions, abbreviated as **L1** and **L2**, are responsible for the loss of modifiers increasing dominance.

In the first and by far smaller region, **L1**, positive frequency-dependent selection resulting from assortative mating neutralizes negative frequency-dependent selection induced by intraspecific competition. Thus, stabilizing selection is the driving force. Clearly this induces selection for no dominance.

In the second region, **L2**, an ecological niche is established at the optimum because of weak to intermediate assortment and intermediate competition. The fraction of heterozygotes is reduced compared with the **L1** scenario. However, heterozygotes at the ecological locus are sufficiently frequent that a shift closer to one of the homozygotes does not significantly increase their chance to mate successfully. Thus, stabilizing selection dominates which leads to extinction of the modifier.

In our model, we assumed that the allelic effects at the ecological locus are symmetric with respect to the optimum. This is assumption is justified not only by the results from section 1.3.3, but also by the studies of Schneider and Bürger (2006) and Matessi and Gimelfarb (2006). Nevertheless, we will now discuss possible consequences of relaxing the symmetry assumption. First, in an asymmetric model, the heterozygote phenotype might evolve more easily towards either one of the homozygotes (i.e., the model is no longer symmetric with respect to positive or negative d). We expect that it will evolve towards the optimum in the loss scenarios (**L1** and **L2**), but away from it in the maintenance and fixation scenarios. The reasons are the following: The phenotypic distribution at equilibrium is asymmetric such that the homozygous genotype closer to the optimum is more abundant. In the regions **L1** and **L2** stabilizing selection dominates and we expect dominance to evolve such that the phenotype of the heterozygotes matches the optimum. In the fixation regions, dominance will evolve more easily in the other direction, i.e., away from the optimum, because competition is weaker in this part of the phenotypic spectrum (cf. Figure 2 in Peischl and Bürger, 2008). However, in the **SS** region, especially modifiers with large effect could sweep faster through the population if they cause heterozygotes to resemble the more abundant homozygote, i.e., the one closer to the optimum. Thus, further investigation of the asymmetric case is necessary to gain better understanding. In general, we

expect that modifiers sweep more easily through the population in the regions in which slow runs occur in the symmetric case. Furthermore, we expect asymmetry to decrease the likelihood of a stable polymorphism of modifier alleles (i.e., the **MS** regime).

The fixation time of an initially rare modifier can differ significantly within and between the two fixation scenarios. The sweep of modifiers with small effects is much quicker in randomly mating populations than in assortatively mating populations. This is not surprising since the number of heterozygotes at the ecological locus determines how efficient selection is translated to the modifier locus. Modifier with large effects experience stronger selection and thus the time spent for invasion and fixation is in general much shorter. Notably, compared to random mating, evolution of dominance can be faster for intermediate assortment if modifier effects are large. In particular, a modifier inducing complete dominance in a single step gets fixed most quickly if $a \approx 1$. Then, sexual selection for higher levels of dominance can be most efficient (cf. Figure 1.1 (e)).

We also identified regions in which complete dominance can evolve by successive invasion and fixation of dominance modifiers (see Figure 1.7). If disruptive selection is the driving force for higher levels of dominance, increasing assortative mating decreases the parameter region in which dominance can be established. In addition, the degree of dominance that can be established also decreases. If assortment is intermediate and the fixation scenario **SS** applies, the degree of dominance as well as the parameter region increase with increasing assortment. For modifiers with large effects, intermediate levels of assortative mating can be most favorable for the evolution of dominance.

Our study differs from previous studies on the evolution of dominance modification in several respects. Here, evolution of dominance is investigated in an ecological context that is related to recent studies of divergence within a population or sympatric speciation, (e.g., Dieckmann and Doebeli, 1999; Matessi et al., 2001; Bürger and Schneider, 2006; Bürger et al., 2006; Pennings et al., 2008). This focus is different from previous studies which usually involved a spatial structure. For instance, Otto and Bourguet (1999); Van Dooren (1999) studied the evolution of dominance in two-niche models, and Dickinson and J. (1973) studied the evolution of dominance and assortative mating in two-niche models.

For an ecological model with random mating, which can be regarded as a weak selection approximation of ours, Matessi and Gimelfarb (2006) studied the evolution of an ecological trait under disruptive selection. They did not consider dominance modifiers, but assumed that mutant alleles at the ecological locus express 'arbitrary' dominance relations. They showed that a symmetric ESS with two alleles, one being completely dominant over the other, exists. However, the focus of their study was on the evolution of the genetic architecture (genotype-phenotype map) and thus was completely different from ours.

Recently, two other studies focused on the evolution of dominance modifiers in a ran-

domly mating population under frequency-dependent selection. One by Peischl and Bürger (2008) and another by Durinx and van Dooren (2008). The work of Peischl and Bürger (2008) showed that modifiers are able to invade whenever fitness is $\cup$-shaped and the mean genotypic (phenotypic) value is sufficiently close to the minimum of the fitness function. It is also shown there that modifiers that are able to invade a population go to fixation, again provided the fitness is $\cup$-shaped. Notably, the converse is not true, i.e., modifiers that go to fixation if sufficiently frequent are not necessarily maintained if they occur at very low frequency. These results are based on the assumptions of weak selection and quadratic fitness functions. As one intuitively expects, they generalize to small degrees of assortment. However, the present study shows that the evolutionary outcome depends in a complex and nonlinear way on the involved parameters, especially if selection is strong. Thus, our study complements and extends the results of Peischl and Bürger (2008).

Durinx and van Dooren (2008) studied the evolution of dominance modifiers and of assortative mating modifiers based on an adaptive dynamics approach. For a population near an evolutionary branching point that mates randomly and expresses no dominance, they explored under which conditions the evolution of assortative mating or dominance is the more likely evolutionary response. In their work, they do not assume a specific ecological model. They only assume that disruptive selection maintains genetic variation at an ecological locus. However, their results are restricted by the assumptions implicitly imposed by invasion dynamics. In addition, in the context of assortative mating the assumption of a population being close to a branching point (as made for some of the results of Durinx and van Dooren, 2008) is restrictive. Near such a point all phenotypes will be almost indistinguishable, especially in the presence of environmental variance. Therefore, it seems hardly plausible that individuals mate completely assortatively and are able to determine the genotype of their possible mating partners.

To the best of our knowledge, all ecological models in the speciation literature that employ disruptive selection behave very similarly for weak selection. They can be approximated by the models of Matessi et al. (2001) or Bürger (2005) (cf. also Schneider, 2006; Matessi and Schneider, 2009). This concerns especially the models of Roughgarden (1972); Bulmer (1974); Slatkin (1979); Christiansen and Loeschcke (1980) and Bürger (2002a,b). The model of Matessi et al. (2001) is the simplest model that satisfies the assumptions of Durinx and van Dooren (2008). All models that satisfy their assumptions can be approximated by the quadratic model of Matessi et al. (2001), since the assumption of a phenotypic distribution close to a branching point implicitly assumes weak selection (by eventual re-scaling of the parameters). Hence, in the context of disruptive selection, our model is almost as general as theirs. Additionally our approach allows us to study strong selection.

One of the main conclusions of Durinx and van Dooren (2008) was that assortative mating and dominance are not just alternative evolutionary responses, but exclusive alter-

natives, where one already established mechanism decreases the probability for the other to appear. Our results suggest that assortative mating counteracts the evolution of dominance as long as the degree of assortment is weak, but also that sexual selection induced by moderately strong assortative mating can support dominance evolution. This does not necessarily contradict the conclusions made by Durinx and van Dooren (2008) since we did not consider modifiers increasing the degree of assortative mating. However, our results suggest that dominance can evolve along with assortative mating. The reason for the different conclusions is that their approach differs from ours in several respects. In the following we explain why different results are established.

We study the possibility of dominance evolution in populations under assortative mating whereas Durinx and van Dooren (2008) consider evolution of dominance and assortative mating as alternative evolutionary responses. Thus, we include various aspects that are not, or only partially, taken into consideration in the study of Durinx and van Dooren (2008), e.g., the impact of different sizes of the modifier effect, the impact of the strength of assortative mating, the initial level of dominance on the modifiers, or strong selection. We did not only find that moderate assortment is favorable for the evolution of dominance, but also that complete dominance is more likely to evolve through a short sequence of modifiers with large effects or at once.

In addition, the underlying models of assortative mating are different. We use the model originally introduced by Gavrilets and Boake (1998), which implies costs of being rare. Durinx and van Dooren (2008) use a model similar to the one of O'Donald (1960), which does not lead to sexual selection. The occurrence of sexual selection and the possibility of larger modifier effects appear to be crucial for our results. In fact, Durinx and van Dooren (2008) found that in populations with partial assortative mating, modifiers that induce complete dominance could sometimes invade whereas modifiers with small effect could never invade. Figure 1.1 (e) shows a similar behavior in our model. Mating probabilities are nearly equal for all phenotypic values, and modifiers inducing complete dominance have a tiny viability-fitness advantage whereas modifiers with small effect have a viability disadvantage. However, dominance can evolve in our model even if viability fitness is $\bigcap$-shaped. In the situation shown in Figure 1.1 (f), modifiers of small and large effect can invade and become fixed.

Summarizing, we provided a systematic study of the evolution of dominance modifiers under frequency-dependent selection and assortative mating. Our approach provides deeper results for a broader parameter spectrum than previous work on related topics. This study shows that assortative mating is a catalyzer for the evolution of dominance in a quite large parameter region. Therefore, our results suggest that the evolution of dominance can be a companion to the evolution of assortative mating. Hence, it may be a significant factor in the context of speciation, whose importance was not appreciated enough yet. The

simultaneous evolution of assortative mating and dominance is a topic that deserves to be studied in more detail.

Acknowledgements: This work was funded by a grant of the Vienna Science and Technology Fund (WWTF) to Reinhard Bürger. The authors are grateful to Reinhard Bürger for useful suggestions and assistance during the preparation of the manuscript. We gratefully acknowledge the helpful comments and suggestions on an earlier draft made Tom Van Dooren and Joachim Hermisson. We especially want to thank Michael Kopp for his fruitful comments.

Appendix

A ESS at the ecological locus

We label the phenotypes and genotype frequencies according to Table 1.1.

Genotype	$\mathscr{A}_1\mathscr{A}_1$	$\mathscr{A}_1\mathscr{A}_2$	$\mathscr{A}_2\mathscr{A}_2$	$\mathscr{A}_1\mathscr{A}_3$	$\mathscr{A}_2\mathscr{A}_3$
Effect	Z_1	Z_2	Z_3	Z_4	Z_5
Frequency	p_1	p_2	p_3	p_4	p_5

Table 1.1: Notation for genotypes in Appendix A.

A.1 No dominance, random mating

We assume additive genetics and assign the alleles $\mathscr{A}_1$ and $\mathscr{A}_2$ the allelic effects z_1 and z_2, respectively. The invasion fitness of a mutant allele with allelic effect z_3 is then given by $\lambda = W_3/\overline{W}$, where W_3 is the marginal fitness of the mutant allele and $\overline{W}$ the mean fitness of the population. Allele frequencies at equilibrium are

$$\hat{p}_{\mathscr{A}_1} = 1/2 - \frac{s(z_1+z_2)}{(c+s)(z_1-z_2)}, \quad \hat{p}_{\mathscr{A}_2} = 1/2 + \frac{s(z_1+z_2)}{(c+s)(z_1-z_2)}, \tag{1.14}$$

where $\hat{p}_{\mathscr{A}_i}$ denotes the frequency of allele $\mathscr{A}_i$ at equilibrium. Then, the invasion fitness of a mutant allele $\mathscr{A}_3$ is given by

$$\lambda = 1 + (z_1 - z_3)(z_2 - z_3)(c - s) + O(s^2). \tag{1.15}$$

A mutant can invade if and only if $\lambda > 1$. Thus, given $c > s$ and neglecting $O(s^2)$ terms, it is obvious that only mutants can invade that widen the range spanned by the existing genotypes.

A.2 Complete dominance, random mating

We assume that one of the alleles, say $\mathscr{A}_1$, is completely dominant. To the genotypes $\mathscr{A}_1\mathscr{A}_1$ and $\mathscr{A}_2\mathscr{A}_2$ we assign the trait values Z_1 and Z_3, respectively. Since we assume complete dominance, $\mathscr{A}_1\mathscr{A}_2$ individuals have the genotypic value $Z_2 = Z_1$. Thus, allele frequencies at equilibrium can be calculated straightforwardly and are given by

$$\hat{p}_{\mathscr{A}_1} = 1 - \sqrt{\frac{1}{2} + \frac{s(Z_1+Z_3)}{2c(Z_1-Z_3)}}, \quad \hat{p}_{\mathscr{A}_2} = \sqrt{\frac{1}{2} + \frac{s(Z_1+Z_3)}{2c(Z_1-Z_3)}}. \tag{1.16}$$

Note that this equilibrium always exists, i.e., it is real and between zero and one if $c > s$ and $\operatorname{sign} Z_1 = \operatorname{sign}(-Z_3)$. Mutants with genotype $\mathscr{A}_1\mathscr{A}_3$ and $\mathscr{A}_2\mathscr{A}_3$ have genotypic values Z_4 and Z_5, respectively. Considering equilibria that do not span the full range of genotypic values we consider two scenarios, either $\mathscr{A}_1$ or $\mathscr{A}_3$ is completely dominant. First, assume $\mathscr{A}_1$ is completely dominant and hence $Z_1 = Z_4$. This leads to the invasion fitness

$$\lambda = 1 + \frac{(c-s)\sqrt{2c(Z_1-Z_3)+s(Z_1+Z_3)}(Z_1-Z_5)(Z_3-Z_5)}{\sqrt{c}\sqrt{Z_1-Z_3}(2+c(Z_1-Z_3)^2)-s(Z_1^2+Z_3^2)}. \tag{1.17}$$

If $\mathscr{A}_3$ is completely dominant, we have $Z_4 = Z_5$ and invasion fitness is given by

$$\lambda = 1 + \frac{2(c-s)(Z_1-Z_5)(Z_3-Z_5)}{2+c(Z_1-Z_3)^2-s(Z_1^2+Z_3^2)}. \tag{1.18}$$

In both cases it is straightforward to see that only mutants can invade that widen the range of genotypic values.

If we set $Z_1 = Z_2 = -1$, $Z_3 = 1$, i.e., the full range of genotypic values is spanned and one allele is completely dominant, invasion fitness is given by

$$\lambda = 1 - \frac{(c-s)\left[2(1-Z_4^2)-\sqrt{2}(Z_4^2-Z_5^2)\right]}{2+4c-2s}. \tag{1.19}$$

To show that no mutant can invade it is sufficient to show that the second factor in the numerator is positive. A simple rearrangement yields that this is equivalent to

$$Z_4^2(1-\frac{1}{\sqrt{2}})+\frac{1}{\sqrt{2}}Z_5^2 < 1. \tag{1.20}$$

Assuming $Z_4, Z_5 \in [-1,1]$ and excluding the case $Z_4^2 = Z_5^2 = 1$ in which a mutant is selectively neutral, it is easily seen that no mutant can invade.

A.3 No dominance, weak assortment

We assume $\pi(Z_g, Z_h) = 1 - a(Z_g - Z_h)^2$, i.e., we neglect second and higher-order terms in a. In addition, we assume additive effects and $z_1 = -z_2$, i.e., equally sized allelic effects in opposite directions. Then, the following equilibrium can be calculated

$$\hat{p}_1 = \hat{p}_3 = \frac{1}{4} + a\frac{z_1^2\left(1-(6c-4s)z_1^2\right)^2}{2\left(1+2z_1^2(2c-s)\right)^2}, \quad \hat{p}_2 = \frac{1}{2} - a\frac{z_1^2\left(1-(6c-4s)z_1^2\right)^2}{\left(1+2z_1^2(2c-s)\right)^2}. \tag{1.21}$$

Genotype	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{D}_1\mathscr{D}_1}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{D}_1\mathscr{D}_1}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{D}_1\mathscr{D}_1}$	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{D}_1\mathscr{D}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{D}_1\mathscr{D}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{D}_2\mathscr{D}_1}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{D}_1\mathscr{D}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{D}_2\mathscr{D}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{D}_2\mathscr{D}_2}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{D}_2\mathscr{D}_2}$
Effect	Z_1	Z_2	Z_3	Z_4	Z_5	Z_6	Z_7	Z_8	Z_9	Z_{10}
Frequency	p_1	p_2	p_3	p_4	p_5	p_6	p_7	p_8	p_9	p_{10}

Table 1.2: Notation for genotypes in Appendix B.

Initially, the modifier allele, with allelic effect z_3, is rare and thus only appears in heterozygotes. Then, given the modifier is rare, the linearized transformation matrix for the vector $(p_4, p_5)^T$ is given by

$$T = \frac{1}{\overline{W}} \begin{pmatrix} W(Z_4)\alpha & W(Z_5)\beta \\ W(Z_4)\gamma & W(Z_5)\delta \end{pmatrix}, \tag{1.22}$$

where

$$\alpha = \frac{1}{2}p_1^*(Q_{14}^* + Q_{41}^*) + \frac{1}{4}p_2^*(Q_{24}^* + Q_{42}^*), \tag{1.23}$$

$$\beta = \frac{1}{2}p_1^*(Q_{15}^* + Q_{51}^*) + \frac{1}{4}p_2^*(Q_{25}^* + Q_{52}^*), \tag{1.24}$$

$$\gamma = \frac{1}{2}p_3^*(Q_{34}^* + Q_{43}^*) + \frac{1}{4}p_2^*(Q_{24}^* + Q_{42}^*), \tag{1.25}$$

$$\delta = \frac{1}{2}p_3^*(Q_{35}^* + Q_{53}^*) + \frac{1}{4}p_2^*(Q_{25}^* + Q_{52}^*). \tag{1.26}$$

It can be seen easily that the leading eigenvalue of T is

$$\lambda = 1 + (c - s - \frac{a}{2})(z_3^2 - z_1^2) + O(a^2) + O(s^2) + O(as) \tag{1.27}$$

as $a \to 0$ and $s \to 0$. Thus, if $c > s + a/2$, mutants with effect z_3 can invade if and only if $z_3^2 > z_1^2$.

B Invasion of a dominance modifier

Now, we label the genotypic values and the genotype frequencies according to Table 1.2.

Again, we choose $\pi(Z_g, Z_h) = 1 - a(Z_g - Z_h)^2$ and assume weak assortment, i.e., we neglect second and higher order terms in a. Then, in the absence of a modifier the following equilibrium can be calculated

$$\hat{p}_1 = \hat{p}_3 = \frac{1}{4} + \frac{a(2 - 2s + 3c)^2}{8(2 - s + 2c)^2},\ \hat{p}_2 = \frac{1}{2} - \frac{a(2 - 2s + 3c)^2}{4(2 - s + 2c)^2}. \tag{1.28}$$

We are interested in a condition for invasion of a rare modifier and thus neglect $O(p_i p_j)$

terms for $i,j=4,...,10$. For a rare modifier the genetic composition of the population is adequately described by the vector $(p_1,p_2,p_3,p_4,p_5,p_6,p_7)^T$ and exploiting the symmetry at equilibrium, it is easily verified that the linearized recursion matrix of the vector $(p_4,p_5,p_6,p_7)^T$ is

$$U=\frac{1}{\overline{W}}\begin{pmatrix} W(Z_1)\alpha & W(Z_5)\beta_1 r & W(Z_5)\beta_1(1-r) & 0 \\ 0 & W(Z_5)\beta_1(1-r) & W(Z_5)\beta_1 r & W(Z_3)\gamma \\ W(Z_1)\gamma & W(Z_5)\beta_2 r & W(Z_5)\beta_2(1-r) & 0 \\ 0 & W(Z_5)\beta_2(1-r) & W(Z_5)\beta_2 r & W(Z_3)\alpha \end{pmatrix}, \tag{1.29}$$

where

$$\alpha = \frac{1}{2}p_1^*(Q_{14}^*+Q_{41}^*)+\frac{1}{4}p_2^*(Q_{24}^*+Q_{42}^*), \tag{1.30}$$

$$\beta_1 = \frac{1}{2}p_1^*(Q_{15}^*+Q_{51}^*)+\frac{1}{4}p_2^*(Q_{25}^*+Q_{52}^*), \tag{1.31}$$

$$\beta_2 = \frac{1}{2}p_1^*(Q_{35}^*+Q_{53}^*)+\frac{1}{4}p_2^*(Q_{25}^*+Q_{52}^*), \tag{1.32}$$

$$\gamma = \frac{1}{2}p_1^*(Q_{17}^*+Q_{71}^*)+\frac{1}{4}p_2^*(Q_{27}^*+Q_{72}^*). \tag{1.33}$$

A perturbation analysis yields the following approximations for the eigenvalues of U:

$$\lambda_1 = 1+\frac{\delta^2}{2}(c-s-\frac{a}{2})+O(a^2)+O(s^2)+O(as), \tag{1.34}$$

$$\lambda_2 = 1+\frac{\delta^2}{2}(c-s-\frac{a}{2})+O(a^2)+O(s^2)+O(as)+O(r), \tag{1.35}$$

$$\lambda_3 = O(a^2), \tag{1.36}$$

$$\lambda_4 = O(a^2). \tag{1.37}$$

Neglecting $O(a^2),O(s^2)$ and $O(as)$ terms, a simple condition for invasion of a dominance modifier can be obtained. Straightforward calculations yield that $\lambda_1,\lambda_2>-1$ in the whole parameter range and that $\lambda_2<\lambda_1$ if and only if

$$\left(\frac{a}{4}+\frac{c}{2}+\frac{a\delta^2}{2}+\delta^2 s-c\delta^2-\frac{s}{2}-1\right)r-\frac{a}{2}\frac{1-2r}{r(1-r)}<0. \tag{1.38}$$

Since $a,c,s\in[0,1/4]$ and $\delta\in[-1,1]$ it follows that $\lambda_2<\lambda_1$ in the considered parameter region. Thus, since $\lambda_1>1$ if and only if

$$c>s+a/2, \tag{1.39}$$

(1.39) is a necessary and sufficient condition for invasion. In particular, the invasion con-

dition is independent of r.

Chapter 2

The evolution of assortative mating under frequency-dependent intraspecific competition with dominance [1]

[1] This chapter is based on the unpublished manuscript Peischl S. and K.A. Schneider, 2010. The evolution of assortative mating under frequency-dependent intraspecific competition with dominance.

Abstract

In this article we study the influence of dominance on the evolution of higher levels of assortative mating. We perform a population-genetic analysis of a two-locus two-allele model, in which the primary locus has a major effect on a quantitative trait that is under a mixture of frequency-independent stabilizing selection, and density- and frequency-dependent selection caused by intraspecific competition for a continuum of resources. The trait is determined by a single locus (ecological locus) and expresses intermediate dominance. The second (modifier) locus determines the degree of assortative mating and is expressed by females only. Assortative mating is based on similarities in the quantitative trait ('magic trait' model). Conditions for the invasion of assortment modifiers are derived analytically in the limit of weak selection and weak assortment. For the full model, extensive numerical iterations are performed to study the global dynamics. This allows us to gain a better understanding of the interaction of the different selective forces. Remarkably, depending on the size of modifier effects, dominance can have different effects on the evolution of assortment. We show that dominance hinders the evolution of assortment if modifier effects are small, but promotes it if modifier effects are large. The latter was not detected in previous work on the evolution of dominance and assortment based on an adaptive dynamics approach.

2.1 Introduction

In sexually reproducing populations, mating occurs generally not at random but shows positive or negative correlations with respect to certain characteristics. If pairing of similar males and females is more or less likely than expected by chance positive or negative assortative mating occurs, respectively. For instance, in humans positive assortative mating has been reported for characteristics such as age, IQ, height, weight, educational and occupational level, and physical and personality characters (Spuhler, 1968; Garrison et al., 1968; Epstein and Guttman, 1984; Ho, 1986).

Although assortative mating was studied over the last forty years in the theoretical literature, it received attention of a much broader audience during the last decade, as a possible mechanism leading to sympatric speciation, i.e., speciation without geographical isolation. While classical work focusing on assortative mating studied the mating mechanism itself and kept the strength of assortative mating constant, e.g., O'Donald (1960), Crosby (1970), Moore (1979), Felsenstein (1981), in the last ten years the evolution of the mating mechanism under a given ecological scenario was a topic of interest, e.g., Doebeli (1996), Dieckmann and Doebeli (1999), Kondrashov and Kondrashov (1999), Matessi et al. (2001), Pennings et al. (2008).

Recent studies involving assortative mating were strongly connected to divergence of a quantitative trait within a population or even to sympatric speciation, e.g., Doebeli (1996), Dieckmann and Doebeli (1999), Kondrashov and Kondrashov (1999), Matessi et al. (2001), Bürger et al. (2006), Pennings et al. (2008). In these models a quantitative character was maintained polymorphic by frequency-dependent disruptive selection. Disruptive selection in these models was caused by negative frequency-dependent selection, which was motivated by intraspecific competition for common resources. Assortative mating occurred either with respect to similarities in this 'ecological' character (magic-trait model, cf. Gavrilets, 2004), or with respect to an additional mating character. The above mentioned studies used the classical models of resource utilization by Roughgarden (1972), Bulmer (1974, 1980), Slatkin (1979), or Christiansen and Loeschcke (1980), which all behave similar as long as selection is weak (cf. Bürger, 2005; Schneider, 2006).

The African finch *Pyrenestes Ostrinus* was often cited to justify the above described ecological setup (e.g., Smith, 1990, 1993; Slabbekoorn and Smith, 2000; Bürger and Gimelfarb, 2004; Bürger and Schneider, 2006; Matessi and Gimelfarb, 2006). However, assortative mating did not evolve in the African finch. Instead the finches express dominance, a mechanism that has been neglected in the above mentioned studies, because they assumed no dominance, i.e., additive genetics.

Recently, a few studies focused on finding general conditions for the evolution of assortment (Durinx and van Dooren, 2008; Otto et al., 2008; de Cara et al., 2008; Barton and

de Cara, 2009). However, only two attempts have been made that explicitly study dominance and assortative mating. The first, Durinx and van Dooren (2008), studied the evolution of assortative mating vs. the evolution of dominance using an adaptive-dynamics approach. The second, Peischl and Schneider (2009), studied the evolution of dominance in an assortative mating population using a comprehensive numerical approach based on the exact dynamics. Durinx and van Dooren (2008) showed that, in the limit of infinitesimally small modifier effects, selection for assortment modifiers is initially stronger than selection for dominance modifiers. Furthermore, they claimed that assortative mating and dominance are alternative and mutually exclusive responses to disruptive selection. In contrast, Peischl and Schneider (2009) suggest that the evolution of dominance can be promoted by intermediately strong assortative mating. Moreover, Peischl and Schneider (2009) emphasize the importance of the interplay between these evolutionary mechanisms. A necessary step towards understanding the interplay between dominance and assortment is to clarify the influence of dominance on the evolution of assortative mating.

In this article we study the evolution of assortative mating with respect to an ecological character that expresses dominance. We pursue a population-genetic approach that complements and extends the results of Durinx and van Dooren (2008). We assume an explicit ecological model of frequency-dependent intraspecific competition and assortative mating. Frequency-dependent competition induces indirect selection on a modifier that determines the strength of assortative mating. Dominance relations and the degree of assortative mating control the translation of direct selection at the ecological locus to indirect selection at the modifier locus. In the limit of weak selection, we are able to derive simple invasion conditions for assortment modifiers in a number of interesting scenarios. However, for a fixed combination of parameters, the strength and direction of these effects depend on the genetic distribution of the population and thus vary over time. Hence, for our purpose an invasion analysis is insufficient. Of course, a complete (nonlinear) analysis would be highly desirable, but the complexity of the model prohibits such an analysis. Thus, we pursue a structured and detailed numerical study examining a large part of the parameter space.

We perform a numerical analysis of a two-locus two-allele model, in which the primary (ecological) locus has a major effect on a quantitative trait that is under a mixture of stabilizing selection and frequency-dependent selection caused by intraspecific competition for a continuum of resources. The ecological model follows the one formulated by Bulmer (1974, 1980). Moreover, we assume mating to be assortative. More precisely, females choose mating partners based on similarities in the ecological character. The model of assortative mating used here follows that of Matessi et al. (2001), which was originally formulated by Gavrilets and Boake (1998). The secondary locus determines the degree of assortment. In contrast to previous studies of the evolution of assortative mating we as-

sume that the ecological locus expresses dominance. Our approach is closely related, but complementary, to the one of Peischl and Schneider (2009).

Our results show that dominance does not counteract an initial increase of assortative mating. However, the level of assortment that can evolve in small steps is strongly reduced if there is some degree of dominance. By contrast, if modifiers have large effect, dominance can act as a catalyzer for the evolution of assortative mating. The region in which strong assortment can evolve is maximized for a certain degree of dominance. Furthermore, this 'optimal' degree of dominance increases with increasing modifier effect. We will also discuss the implications of the evolution of assortative mating. If assortative mating is sufficiently strong, divergence within the population occurs. This will eventually lead to sympatric speciation. Dominance can be a mechanism that enforces divergence. Together with the results of the preceding study of Peischl and Schneider (2009) our results enable us to draw conclusions to which level assortment and dominance is likely to evolve.

2.2 The model

We consider a model that is closely related to that of Peischl and Schneider (2009). It assumes a sexually reproducing, diploid, density-regulated population with discrete generations in which both sexes have the same genotype distribution among zygotes. Random genetic drift is neglected by assuming that the population size, N, is sufficiently large. Selection acts through differential viabilities on a quantitative character. Because selection is assumed to act on this character we refer to it as the 'ecological character'. The viability of an individual is determined by frequency-independent stabilizing selection and by frequency- and density-dependent competition. The trait value of an individual expresses an intermediate degree of dominance. We refer to this trait as the ecological trait. Furthermore, the population mates assortatively with respect to the ecological trait ('magic trait'). This induces sexual selection. The degree to which an individual mates assortatively depends on its expression at an additional locus that modifies the degree of assortment.

2.2.1 Ecological assumptions

These assumptions follow closely those in Schneider and Bürger (2006), Bürger and Schneider (2006), and Bürger et al. (2006), where they are motivated. As in most previous studies, we ignore environmental variation and deal directly with the fitnesses of genotypic values. Therefore, we use the terms genotypic value and phenotype synonymously. We denote the ecological trait value of an individual having genotype g by Z_g.

The frequency-independent fitness component reflects stabilizing selection on the ecological trait, for instance, by differential supply of a resource whose utilization efficiency is

phenotype dependent. The stabilizing component acting on genotype g is denoted by $S(Z_g)$. Here, $S(Z_g)$ is modeled by the Gaussian function with optimum zero

$$S(Z_g) = \exp[-s{Z_g}^2]\,, \tag{2.1}$$

where $s \geq 0$ measures its strength. We refer to the trait value zero as the position of the optimum or just as the 'optimum'.

The amount of competition of genotype g with genotype h is denoted by $\alpha(Z_g, Z_h)$. We model it by the Gaussian function

$$\alpha(Z_g, Z_h) = \exp[-c(Z_g - Z_h)^2]\,. \tag{2.2}$$

The parameter $c \geq 0$ determining the curvature of $\alpha(Z_g, Z_h)$ implies that competition between individuals of similar trait value is stronger than between individuals of very different trait value, as it will be the case if different phenotypes preferentially utilize different food resources. Let P_h denote the relative frequency of individuals with genotype h. Then the intraspecific competition function $\overline{\alpha}(g)$, which measures the strength of competition experienced by genotype g in a population with distribution P, is given by

$$\overline{\alpha}(g) = \sum_h \alpha(Z_g, Z_h) P_h\,. \tag{2.3}$$

We include density-dependent population growth, which, in the absence of genetic variation, follows the logistic equation

$$N' = \begin{cases} N(\rho - N/\kappa), & 0 \leq N < \rho\kappa\,, \\ 0\,, & N \geq \rho\kappa\,. \end{cases} \tag{2.4}$$

The carrying capacity is $K = (\rho - 1)\kappa$. Monotone convergence to K occurs for all N with $0 < N < \rho\kappa$ if $1 < \rho \leq 2$, and oscillatory convergence (at a geometric rate) if $2 < \rho < 3$. Other forms of population regulation may be used as well (cf. Appendix B, Bürger 2005). Following Bulmer (1974, 1980), we assume that the absolute fitness of an individual with genotype g is

$$W(g) = S(Z_g)\Big(\rho - \frac{N}{\kappa}\overline{\alpha}(g)\Big)\,, \tag{2.5}$$

where the dependence of $W(g)$ on N and P is omitted. Although c is a direct measure for the strength of the frequency-dependent effect of competition, rather than of competition itself, for convenience, we shall refer to c as the strength of competition.

In some part of this work we will replace (2.1) and (2.2) by the corresponding quadratic

approximations, i.e., by

$$S(Z_g) = 1 - s{Z_g}^2 \tag{2.6}$$

and

$$\alpha(Z_g, Z_h) = 1 - c(Z_g - Z_h)^2 \,. \tag{2.7}$$

In addition, we will assume constant population size close to the demographic equilibrium. Then fitness of an individual with genotype g is given by

$$W(g) = 1 - sZ_g^2 + c(Z_g - \overline{g})^2 V, \tag{2.8}$$

where $\overline{g}$ is the mean and V the variance of the phenotype distribution (cf. Bürger 2005). As long as the mean genotypic value is sufficiently close to zero, W is $\cup$-shaped if and only if $c > s$ and $\cap$-shaped if and only if $c < s$. We will refer to (2.5) with $S(Z_g)$ and $\alpha(Z_g, Z_h)$ given by (2.1) and (2.2), or by (2.6) and (2.7) as the *full model* or the *quadratic model*, respectively. Note that the quadratic model can be regarded as the weak-selection approximation of the full model, i.e., as an approximation for small s and c.

The quadratic model was used to study the evolution of dominance in a randomly mating population by Peischl and Bürger (2008). This weak-selection approximation was also used to study closely related ecological models under different assumptions and with another focus by Bürger and Gimelfarb (2004) and Bürger and Schneider (2006). The Gaussian choice has the advantage that weak and strong selection can be modeled, but it is prohibitive to a general mathematical analysis.

2.2.2 Assortative mating

We assume that mating is assortative according to the model of Matessi et al. (2001), which is a particular case of the Gavrilets and Boake (1998) model. The probability that a random encounter between a female and a male results in mating depends on similarities in the ecological character ('magic trait'). More precisely, the probability that at a given encounter, a g-female mates an h-male is given by $\pi(g,h)$, and modeled by

$$\pi(g,h) = \exp[-a_g(Z_g - Z_h)^2] \,, \tag{2.9}$$

where a_g is the strength of assortment expressed by a female with genotype g. In fact, a_g depends only on the modifier locus and is a direct measure for the strength of assortative mating. Note that $a_g = 0$ means that a female mates randomly, whereas $a_g = +\infty$ means

that she mates only males that show an identical value of the ecological trait. In this article we always assume $a_g \geq 0$, i.e., we consider only positive assortative mating.

Females are assumed to mate only once, whereas males may participate in multiple matings. If an encounter was not successful, in which case she remains unmated, she may try again unless the total number of encounters has reached a maximum number M. This reflects the idea that choosiness has costs, for instance, because the mating period is limited. The probability that an encounter of a g-female with a random male results in mating is

$$\overline{\pi}(g) = \sum_h \pi(g,h) P_h \,, \tag{2.10}$$

and the probability that she eventually mates with an h-male is denoted by $Q(g,h)P_h$, which is calculated to be

$$Q(g,h) = \sum_{m=0}^{M-1} (1-\overline{\pi}(g))^m \pi(g,h). \tag{2.11}$$

Here, the first argument refers to the female. Note, that in general Q is not symmetric in g and h.

The maximum number of possible encounters, M, is a measure for the costs of choosiness payed by females. If $M = 1$, then $Q(g,h) = \pi(g,h)$. This leads to strong sexual selection in both sexes (provided females express assortative mating). If the encounter rate is high enough ($M \gtrsim 10$, cf. Schneider and Bürger, 2006), M may be chosen to be infinity, and we obtain $Q(g,h) = \pi(g,h)/\overline{\pi}(g)$. Then, $\sum_h Q(g,h)P_h = 1$ for all g, and assortative mating does not induce (sexual) selection among females. It does, however, induce sexual selection among males. For a more detailed discussion of this model we refer to Schneider and Bürger (2006).

2.2.3 Genetic assumptions

Regarding the underlying genetics, we assume that the ecological trait is determined by a single diallelic locus. We denote the alleles segregating at this locus by $\mathscr{A}_1$ and $\mathscr{A}_2$, and their effects by z_1 and z_2, respectively, which we assume to be symmetric, i.e., $z_1 = -z_2$. By rescaling the parameters a, c, and s we can assume without loss of generality that

$$z_1 = \frac{1}{2} \quad \text{and} \quad z_2 = -\frac{1}{2}.$$

Moreover, d is the degree of dominance. Hence, individuals with the allele configurations $\mathscr{A}_1\mathscr{A}_1$, $\mathscr{A}_1\mathscr{A}_2$, and $\mathscr{A}_2\mathscr{A}_2$ at the ecological locus, have trait values 1, d, and -1, respectively.

Here, we consider only intermediate dominance, i.e., $-1 \leq d \leq 1$. Clearly, $d = 0$, $d = 1$, or $d = -1$ means no dominance, complete dominance of $\mathscr{A}_1$, or complete dominance of $\mathscr{A}_2$, respectively. The symmetry assumption implies that we can assume $d \geq 0$ without loss of generality.

The strength of assortment expressed by females is determined by a separate diallelic, autosomal locus. The two alleles at this locus are denoted by $\mathscr{M}_1$ and $\mathscr{M}_2$. The alleles have effects a_1 and a_2, respectively, which additively determine the strength of assortment expressed by females. Hence, a female carrying the allele combination $\mathscr{M}_1\mathscr{M}_1$, $\mathscr{M}_1\mathscr{M}_2$, or $\mathscr{M}_2\mathscr{M}_2$ expresses assortment at strength

$$2a_1, \quad a_1 + a_2, \quad \text{or} \quad 2a_2, \tag{2.12}$$

respectively.

Whenever we refer to modifiers increasing assortment, we call the allele at the modifier locus that codes for a higher level of assortment the mutant or the modifier allele, and the allele coding for a lower level of assortment the wild-type allele. In the case of modifiers decreasing assortment, it is the other way round. We refer to the degree of assortment expressed by heterozygotes carrying only the wild-type allele as the initial degree of assortment, a. Furthermore, we refer to the difference between the initial degree of assortment and the degree of assortment expressed by heterozygotes carrying exactly one modifier allele as the effect of the modifier allele, $\tilde{a}$. If $\mathscr{M}_1$ is the wild-type allele, then

$$a = 2a_1 \quad \text{and} \quad \tilde{a} = a_2 - a_1. \tag{2.13}$$

2.2.4 Dynamics

The two-locus dynamics has to be described in terms of diploid genotype frequencies since zygotes (offspring) are generally not in Hardy-Weinberg proportions because of assortative mating. Genotypes are unordered. Let r represent an offsprings' genotype and u, v parental genotypes. The frequency of genotype r (among zygotes) in consecutive generations is denoted by p_r and p'_r. The frequency of r after (natural) selection is $p^*_r = p_r W_r / \overline{W}$, where $W_r = W(r)$ and $\overline{W} = \sum_r W_r p_r$ is the mean viability. After viability selection, mating and recombination occur. Let $R(uv \to r)$ designate the probability that parents with genotypes u and v produce a zygote with genotype r. $R(uv \to r)$ is determined by the pattern of recombination between the two loci.

The genetic dynamics is given by a system of 10 recursion equations that can be written as

$$p'_r = \frac{\overline{W}^2}{\widetilde{W}} \widetilde{W}_r, \tag{2.14}$$

where

$$\widetilde{W}_r = \sum_{u,v} p^*_u p^*_v Q^*_{uv} R(uv \to r), \tag{2.15}$$

$Q^*_{uv} = Q^*(u,v)$ (the asterisk indicates that Q is calculated from the genotypic frequencies after selection) and $\widetilde{W} = \overline{W}^2 \sum_{r,u,v} p^*_u p^*_v Q^*_{uv} R(uv \to r)$. The demographic dynamics follows the standard recursion

$$N' = N \frac{\widetilde{W}}{\overline{W}}. \tag{2.16}$$

Thus, for a genetically monomorphic population that matches the optimum, population growth follows (2.4). The complete evolutionary dynamics is given by the coupled system (2.14) and (2.16). We set $N' = 0$ (population extinction) if $\widetilde{W}/\overline{W} \leq 0$, and $p^*_r = 0$ if $W_r \leq 0$.

In the quadratic model, population size is assumed constant and the dynamics is given by (2.14).

2.3 Components of selection and selection regimes

Before we start describing our results, we discuss the different selection pressures and their effect on selection at the modifier locus.

2.3.1 Components of selection

Modifier alleles affect the strength of assortative mating but not the phenotypic value of an individual that carries the modifier. In addition, we assume that modifiers do not have a direct fitness effect. Hence, selection at the modifier locus is indirect. This means that direct selection at the ecological locus is translated to indirect selection at the modifier locus. An increase in the strength of assortment leads to a decrease in the frequency of heterozygotes at the ecological locus. Therefore, higher levels of assortment are favored if heterozygotes are, on average, less fit than homozygotes (Matessi et al., 2001; Durinx and van Dooren, 2008; Pennings et al., 2008; Otto et al., 2008). We call the net effect of selection disruptive if heterozygotes (at the ecological locus) are less fit than homozygotes, and stabilizing if homozygotes are fitter than heterozygotes. The strength of selection at the modifier locus depends on the frequency of heterozygotes at the ecological locus. Selection is transmitted more efficiently if the frequency of heterozygotes is high. If the frequency of heterozygotes goes to zero, selection at the modifier locus vanishes.

Selection acts directly at the ecological locus via four components. The first component of selection in our model is frequency-independent stabilizing selection. We assume symmetric allelic effects with respect to the optimum of stabilizing selection (for a discussion of this assumption see Peischl and Schneider, 2009). Thus, phenotypes close to the

middle of the phenotypic range are favored by stabilizing selection. This leads to heterozygote advantage and selection against modifiers that increase assortment. Since we assume symmetric allelic effects, heterozygote advantage is strongest in the absence of dominance. In the numerical part of this work, we only consider stabilizing selection that is weak compared to negative frequency-dependent selection ($s = 0.1 < c$).

The second component is negative frequency-dependent selection induced by intraspecific competition. It favors sufficiently different phenotypes such that competition between individuals is minimized. We interpret these phenotypes as being adapted to different ecological niches, where we interpret the location of the maxima of (2.5) (or (2.8)) as ecological niches. We focus on competition that is at most moderately strong. Then (2.5) (or (2.8)) is $\cup-$shaped in the absence of dominance and assortative mating, i.e., $0.2 \leq c \leq 2$. If (2.5) (or (2.8)) is $\cup-$shaped, two ecological niches exist, coinciding with the phenotypic values of the homozygotes, i.e., -1 and +1. In this situation intraspecific competition favors an increase in genetic variance and therefore higher levels of assortment. However, assortment may change the shape of (2.5) (or (2.8)). If heterozygotes are rare because of assortative mating, a niche in the middle of the phenotypic range can be established, which can lead to selection for lower levels of assortment. Dominance generally decreases the difference in viability between homozygotes and heterozygotes. This weakens indirect selection at the modifier locus.

The third component, density-dependent selection, acts jointly with intraspecific competition. For a given population distribution, the fitness ratio of advantageous to disadvantageous phenotypes is larger in high-density than in low-density populations.

The forth component is positive frequency-dependent selection induced by assortative mating. Positive frequency-dependence favors common types over rare types. Hence, it counteracts intraspecific competition in this sense. Although we assume no costs of choosiness, the disadvantage of low-frequency males can be interpreted as costs of being rare. Hence, positive frequency-dependence is stabilizing if heterozygotes are common, and disruptive if heterozygotes are rare. The difference in the mating success of heterozygotes and homozygotes determines whether higher or lower levels of assortment are favored by positive frequency-dependent selection. Thus, weak initial assortment favors a decrease in the strength of assortment, and strong initial assortment favors an increase in the strength of assortment. However, the strength of assortment also determines the efficiency of indirect selection. If sexual selection is strong because of high levels of assortment, indirect selection at the modifier locus may nevertheless be very weak because of a reduced frequency of heterozygotes at the ecological locus. In addition, dominance decreases the difference in mating success between homozygotes and heterozygotes, and thus the strength of selection at the modifier locus.

2.3.2 Selection regimes

The net impact of the different selection components on the modifier locus depends crucially on the combination of parameters. In general, competition and sexual selection act in opposite directions and its not straightforward to determine the net effect of selection. For instance, the net effect of selection can be disruptive although either sexual or natural selection is stabilizing. In addition, dominance can have a strong effect on direction and strength of selection at the modifier locus.

Here, we present the most important selection regimes we encountered in our analysis. The different regimes highlight the subtle interplay of competition and assortative mating. We label the regimes according to the dominating selection pressure: 'C' stands for competition, and 'S' for sexual selection due to assortative mating. The superscripts '+' and '−' indicate selection for higher or lower levels of assortment, respectively. The direction of selection at the modifier locus was determined by calculating the rate of change of modifier alleles. Figure 2.1 illustrates viability and mating success in the different regimes.

C^+: Disruptive competition.
This regime applies if viability is ∪−shaped and positive frequency-dependence is absent or weak ($a \leq 0.5$). Two niches exist at the boundary of the phenotypic range, and stabilizing sexual selection is too weak to counteract disruptive selection resulting from competition (Figure 2.1 (a)). Therefore, higher levels of assortment are favored in this scenario. Dominance weakens disruptive selection at the ecological locus. Thus, this scenario is not very robust to changes in the degree of dominance. Furthermore, the region in which this scenario applies decreases with increasing assortment. In our model, this scenario is necessary to start the evolution of assortment in an initially randomly mating population.

S^+: Disruptive sexual selection.
This regime applies if assortment is sufficiently strong to establish strong disruptive sexual selection. Additionally, competition needs to be weak ($0.2 \leq c \leq 0.6$, see Figure 2.1 (b)) or moderate ($0.6 \leq c \leq 1$, Figure 2.1 (c)). Then, heterozygotes are rare compared to homozygotes, and heterozygous males pay higher costs of being rare. Consequently, an increase in assortative mating is favored. However, selection at the modifier locus is weak because of the low frequency of heterozygotes at the ecological locus. In addition, dominance decreases the difference between phenotypic values of heterozygotes and homozygotes. Therefore, selection for assortment can be very weak in this scenario.

C^-: Strong competition.
Here, competition is strong enough to establish a niche in the middle of the phenotypic

range. Since we restrict attention to at most moderate competition, i.e., $c < 2$, a sufficiently low frequency of heterozygotes at the ecological locus, i.e., sufficiently strong assortative mating, is necessary for the establishment of a niche in the middle of the phenotypic range. Assortative mating may induce disruptive sexual selection in this scenario (Figure 2.1 (d)). However, higher levels of assortment are not favored because heterozygotes have a significantly higher viability than homozygotes. The strength of competition necessary to establish a niche in the middle of the phenotypic range depends crucially on the frequency of heterozygotes at the ecological locus, i.e., the strength of assortment. If the degree of dominance increases, heterozygote advantage decreases.

S^-: Stabilizing sexual selection.
In this regime, assortment is moderately strong. Heterozygotes are still common and sexual selection leads to sufficiently strong stabilizing selection that outweighs disruptive selection resulting from competition. Then, a $\cap-$shaped phenotype distribution is optimal and higher levels of assortment are disadvantageous. Competition can be weak ($0.2 \leq c \leq 0.6$, Figure 2.1 (e)) or moderate ($0.6 \leq c \leq 1$, Figure 2.1 (f)) in this scenario. Dominance increases the parameter region in which this scenario applies. In particular, dominance makes it harder for heterozygotes to exploit a niche in the middle of the phenotype range (Figure 2.1 (f)).

Noteworthy, in the quadratic model, the scenario C^- is impossible if $c > s$. In the quadratic model, the frequency of heterozygotes at the ecological locus only changes the intensity of disruptive competition, but not the $\cup-$shape of viability. This reflects a very important difference between the quadratic and the full model.

2.4 Analytical results

To derive analytical results we use the quadratic model and assume a population of constant size close to demographic equilibrium. In addition, whenever we speak of weak assortment, we choose the probability that a g-female mates an h-male at a given encounter as

$$\pi(g,h) = 1 - a_g(Z_g - Z_h)^2, \tag{2.17}$$

i.e., the first-order Taylor approximation in a_g of (2.9) around 0. This imposes the restriction $a_g \in [0, 1/4]$.

Throughout this section we assume that the population is at an equilibrium, where the modifier locus is monomorphic and the ecological locus is polymorphic (cf. Peischl and Schneider, 2009). The state of the population is then perturbed by the occurrence of a

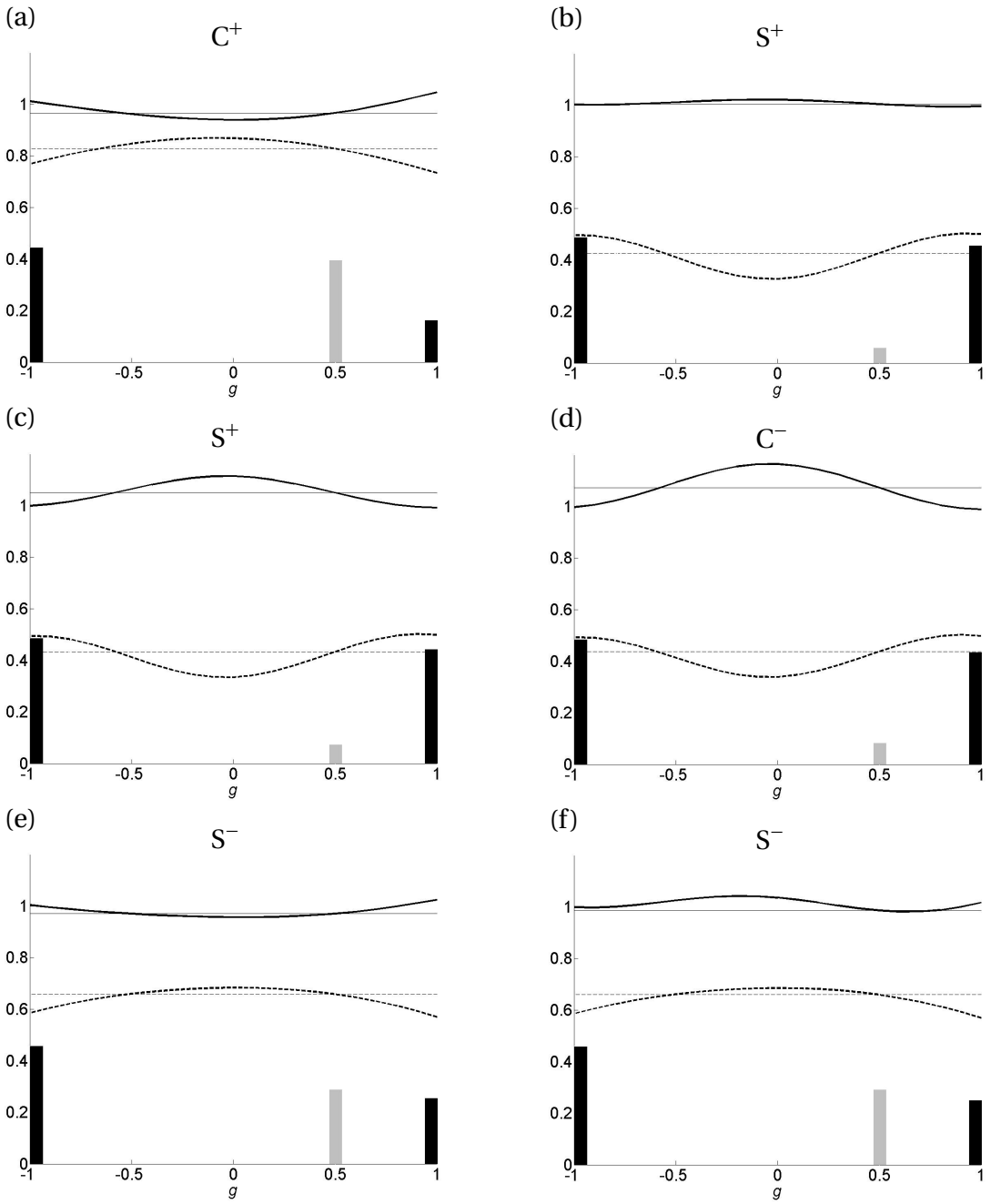

Figure 2.1: Viability $W(g)$ (thick solid line), mating probability $\overline{\pi}(g)$ (thick dashed line) and phenotype distributions (black and gray bars) at equilibrium in the different scenarios described in Section 2.3.2. Thin straight lines show the viability (solid) and the mating probability (dashed) of heterozygotes. Equilibrium frequencies of homozygotes on the ecological locus are indicated by black bars and frequencies of heterozygotes are indicated by gray bars. Parameter values are (a) $c = 0.5, a = 0.3$, (b) $c = 0.5, a = 1.2$, (c) $c = 0.7, a = 1.2$, (d) $c = 0.8, a = 1.2$, (e) $c = 0.5, a = 0.5$, and (f) $c = 0.8, a = 0.5$. The other parameters are $d = 0.5$, $s = 0.1$, and $r = 0.5$ in all figures.

modifier allele at low frequency. We present invasion criteria for such modifiers in various

scenarios. These conditions are derived by calculating (approximations for) the leading eigenvalue of the linearized transition matrix of the gene frequency vector at equilibrium, i.e., we perform a local stability analysis. Equilibria can be calculated explicitly only if dominance is complete or absent, and if the population mates either randomly or completely assortatively. However, by using standard perturbation techniques, approximations for the equilibria and their eigenvalues can be derived in a number of interesting cases such as weak or strong initial assortment, and weak or strong dominance. The equilibria and the derivations of the following results are given in Appendix A.

2.4.1 No dominance

The case of no dominance is the simplest and has previously been treated in the literature in a number of similar but different models (Matessi et al., 2001; Durinx and van Dooren, 2008; Pennings et al., 2008).

2.4.1.1 Modifiers with small effects

By small effect we mean that $\tilde{a} \ll 1$, so that we can neglect second and higher order terms in $\tilde{a}$. The assumption of no dominance and small modifier effects allows us to use an invasion criterion derived in Matessi et al. (2001). Useful application of this criterion requires the explicit knowledge of genotype frequencies at equilibrium. In addition, we also derive approximations for the leading eigenvalues. This gives us an estimate of the strength of selection on a rare modifier allele.

Weak initial assortment

We address three questions. First, when will a modifier inducing a small degree of assortment invade a randomly mating population? Second, when will it go to fixation provided it is sufficiently frequent? And third, when can a modifier invade a population that already expresses a small degree of initial assortative mating?

Let $a \ll 1$ so that we can use (2.17). We show in Appendix A that a modifier increasing assortment invades the population at equilibrium if and only if

$$c > s + \frac{a}{2}. \tag{2.18}$$

Hence, in a randomly mating population ($a = 0$) a modifier invades if and only if $c > s$. Furthermore, a modifier decreasing assortment can invade if and only if the inequality in (2.18) is reversed.

The above implies that a sufficiently frequent modifier that increases assortment be-

comes fixed if and only if

$$c > s + \tilde{a}. \tag{2.19}$$

Strong initial assortment

If the population expresses strong assortment, i.e., if $\varepsilon := \exp[-a]$ is sufficiently small to neglect terms of order $\mathcal{O}(\varepsilon^5)$ and higher, it is possible to derive conditions for the spread of modifiers slightly increasing the strength of assortment. In contrast to the case of weak initial assortment, modifiers can always invade a strongly assortatively mating population. This also means that if a sufficiently high level of assortment is established, modifiers that decrease the strength of assortment cannot invade. Furthermore, consider a modifier with (infinitesimally) small effect $\tilde{a}$ and an initial degree of assortment, a, such that $\exp[-(a+2\tilde{a})] \ll 1$. Provided such a modifier is sufficiently frequent, it will also go to fixation.

Concluding, a modifier inducing a small degree of assortment invades a randomly mating population if and only if selection is disruptive, i.e., $c > s$ (C^+ regime). The modifier may however not be able to go to fixation. This is the case if $c < s + \tilde{a}$ (S^- regime). Hence, the individuals in the population will express different degrees of assortment. However, if the modifier goes to fixation, disruptive selection is sufficiently strong and a new modifier increasing assortment can invade. If assortment is sufficiently strong, modifiers increasing assortment will always invade if rare, and go to fixation if sufficiently frequent (S^+ regime).

2.4.1.2 Modifiers with large effects

Initial random mating

As shown in Appendix A.2, a modifier that increases assortment can invade a randomly mating population if and only if $c > s$, independently of the size of the modifier effect. In fact, the invasion condition does not change even for arbitrary mate-choice functions that induce positive assortment (see Appendix A.2). This includes the case of a modifier that causes individuals that carry at least one copy of the modifier to mate completely assortative, i.e., if $g = \mathscr{A}_i\mathscr{A}_j/\mathscr{M}_1\mathscr{M}_1$, $i,j \in \{1,2\}$, we set $\pi(g,h) = 1$ and otherwise

$$\pi(g,h) = \begin{cases} 1 & \text{if} \quad Z_g = Z_h \\ 0 & \text{if} \quad Z_g \neq Z_h. \end{cases} \tag{2.20}$$

Furthermore, modifiers with sufficiently large effect always go to fixation if they are sufficiently frequent (regime S^+).

Complete initial assortment

Now, assume a population that initially mates completely assortatively and an initially rare modifier that decreases the strength of assortment by a small amount. We show in Appendix A that such a modifier can never invade. If such a modifier could invade, complete assortment cannot be achieved by small steps.

To summarize, in the absence of dominance, modifiers with small effects can invade a randomly mating population, but may not be able to get fixed. In contrast, modifiers with large effect can invade whenever selection is disruptive, and, in addition, they also go to fixation if they are sufficiently frequent. Thus, we conclude that complete reproductive isolation is most likely to evolve in large steps if there is no dominance.

2.4.2 Weak or strong dominance

How does dominance affect the evolution of assortative mating? Analytical results in models with dominance are difficult to obtain and hence rare in the literature. In our model, two cases are analytically tractable to some extent, namely weak and strong dominance. The invasion criterion for modifiers of small effect cannot be used in the case of dominance. Instead, we have to calculate approximations for the leading eigenvalues.

Let us start with the case of weak dominance. Let dominance be sufficiently weak to neglect terms of order $\mathcal{O}(d^3)$ and higher. In this case, the leading eigenvalue of the linearized transition matrix is

$$\lambda = 1 + \tilde{a}\left[\frac{1}{8}(1-2d^2)(c-s) + \mathcal{O}(s^2) + O(d^3)\right] + \mathcal{O}(\tilde{a}^2). \tag{2.21}$$

Hence, a modifier can invade if and only if $c > s$. Although the strength of selection for a modifier is a decreasing function in d, the invasion criterion is not affected by weak dominance.

Next, we treat the case of strong dominance. We set $\delta := 1-d$ and assume that terms of order $\mathcal{O}(\delta^2)$ and higher can be neglected. The leading eigenvalue is

$$\lambda = 1 + \tilde{a}\left[(3\sqrt{2}-4)\delta(c-s) + \mathcal{O}(\delta^2) + \mathcal{O}(s^2)\right] + \mathcal{O}(\tilde{a}^2), \tag{2.22}$$

and a modifier increasing assortment can invade if $c > s$. In the case of complete assortment, $\delta = 0$, modifiers for assortative mating are selectively neutral and the leading eigenvalues equals 1. This can easily be generalized to modifiers with arbitrary effect.

These results suggest that dominance decreases the strength of selection for assortment modifiers, but has no effect on the condition for invasion (cf. Durinx and van Dooren, 2008).

2.4.3 Assortment vs. dominance

Here, we compare the (initial) strength of selection for an increased level of assortment with the selection pressure for an increased level of dominance. Peischl and Schneider (2009) calculated the strength of selection for a rare dominance modifier in a randomly mating population for the same ecological model. Hence, we can compare the strength of selection for the different modifiers. If the modifier effects go to zero, the selection coefficients for a dominance modifier and an assortment modifier behave differently (see Appendix A.5). The strength of selection for a dominance modifier decreases faster than the strength of selection for an assortment modifier. This is consistent with results of Durinx and van Dooren (2008), who showed that in symmetric cases selection for an increased level of assortment is stronger than selection for an increased level of dominance if both modifiers have infinitesimally small effects.

2.5 Numerical methods

A detailed mathematical analysis of our model beyond the results derived above seems infeasible. Thus, we additionally pursue a comprehensive numerical analysis. Our approach consists of two parts.

First, we numerically calculated the invasion fitness of an initially rare modifier of effect $\tilde{a} = 0.05$ in a population close to equilibrium for several values of c, d, and a (see Figure 2.2). By invasion fitness we mean the leading eigenvalue of the linearized transition matrix described in Appendix A. Invasion fitness helps us to identify regions in which higher levels of assortment are favorable if the modifier locus is fixed for the wild-type allele. However, our main focus is to obtain a complete picture of the global dynamics by performing numerical iterations of the coupled system (2.14) and (2.16).

For the iterations, we performed three sets of calculations. In the first set the assortment modifier was assumed to initially segregate at random frequency in the population. In particular, the genotype frequencies are drawn from an uniform distribution and then normalized. In the second set we assumed that the assortment modifier is initially rare, i.e., at frequency 10^{-4}. Furthermore, we assumed that initially the genotypes $\mathscr{A}_i\mathscr{A}_j/\mathscr{M}_2\mathscr{M}_2$ ($i, j \in \{1, 2\}$) were not present. In the third set, the assortment modifier was assumed to initially segregate at high frequency. We proceeded analogously to second scenario, but the initial frequency of the modifer allele was $1 - 10^{-4}$. For simplicity, we call the first set of iterations the *standard* scenario, the second situation the *rare-modifier* scenario, and the third situation the *frequent-modifier* scenario.

Throughout our numerical investigations we assumed free recombination, i.e., the recombination rate is $1/2$, and we always chose the population growth rate to be $\rho = 2$. More-

over, because κ can be considered a scaling factor for the population size N, we did not choose it explicitly, and instead regarded N as normalized by the carrying capacity. We assumed that the initial population size matches exactly the carrying capacity, i.e., $N/\kappa = 1$.

Our model is fully determined by the parameter vector $(s, c, d, a, \tilde{a})$. In all scenarios we used $s = 0.1$. The other parameters were varied as described below. Moreover, we chose various values for a and $\tilde{a}$ that are listed in the figure captions and in the description of our results. For each combination of the above parameters we chose ten different initial genotype distributions under all three scenarios, subject to the constraint that the minimum Euclidean distance between any two different distributions is 0.2.

For each initial distribution, we iterated the recursion relations (2.14) and (2.16) either until an equilibrium was reached, which was decided to be the case if the Euclidian distance between the vectors of genotype frequencies concatenated with the population size of two consecutive generations was less than 10^{-10}, or until 10^6 generations were reached. Such runs are referred to as *slow runs*. The reason was always slow convergence to equilibrium, not cyclical or chaotic behavior.

2.6 Numerical results

2.6.1 Invasion fitness

We numerically calculate the invasion fitness of an initially rare modifier. Figure 2.2 shows the invasion fitness of a modifier with small effect ($\tilde{a} = 0.05$) as a function of the initial level of assortment in the absence of dominance (a), and for $d = 0.5$ (b). We note that all results are qualitatively robust with respect to the size of the modifier effect.

First, we consider no dominance, i.e., $d = 0$ (Figure 2.2 (a)). For weak assortment ($a \approx 0$), a modifier that increases the degree of assortment can always invade. The reason is that (2.5) is $\cup$−shaped in the considered parameter region and the C^+ regime applies. If initial assortment increases, positive frequency-dependence increases and disruptive selection at the ecological locus decreases. Therefore, there exists a region in which the S^- regime applies provided competition is weak ($c \lesssim 0.5$) and assortment is intermediate ($0.1 \lesssim a \lesssim 0.8$). In this region, positive frequency-dependence outweighs negative frequency-dependence and selection is 'overall' stabilizing. If competition is moderately strong ($0.5 \lesssim c \lesssim 1.5$) and assortment intermediate ($0.5 \lesssim a \lesssim 1.5$), the C^- regime applies and a modifier increasing assortment cannot invade. Apparently, $c \approx 0.5$ is optimal for the evolution of assortment in small steps. Then, the C^+ regime applies if $0 < a \lesssim 1.5$. If initial assortment is high ($a \gtrsim 1.5$), modifiers are almost neutral and the S^+ regime applies if $c \lesssim 1.5$. If $c \gtrsim 1.5$, disruptive sexual selection cannot outweigh the viability advantage of heterozygotes at the ecological locus. Thus, the C^- regime applies. In general, selection for

modifiers is very weak if $a \gtrsim 2$. This is because the selective strength at the modifier locus depends on the frequency of heterozygotes at the ecological locus, which is very low for high levels of initial assortment.

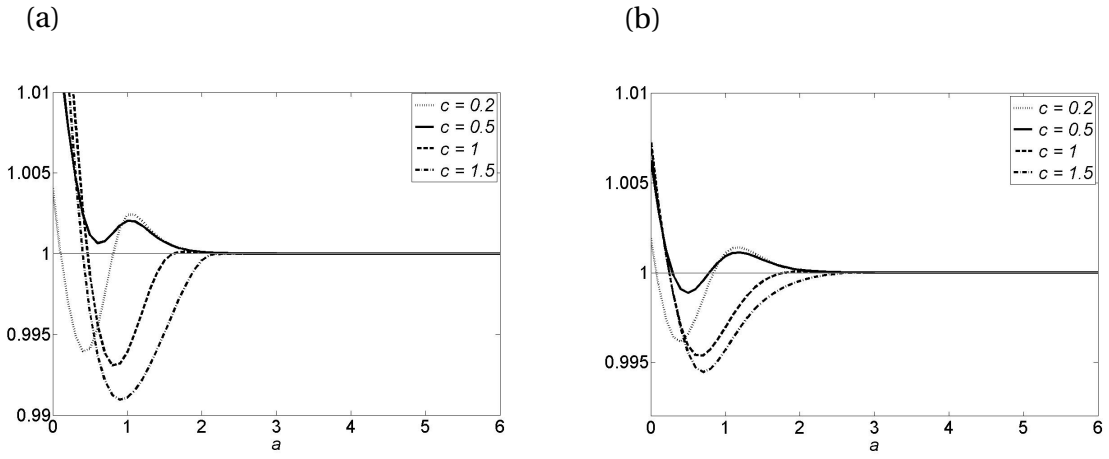

Figure 2.2: Invasion fitness as a function of the initial degree of assortment for various values of c and d. In (a) $d = 0$ and in (b) $d = 0.5$. The modifier effect is $\tilde{a} = 0.05$ in both figures. Furthermore, we used $s = 0.1$ and $r = 0.5$.

Next, we consider intermediate dominance, $d = 0.5$ (Figure 2.2 (b)). Selection at the modifier locus is in general weaker. The reason is that heterozygotes are more similar to one of the homozygotes. Hence, the fitness differences are smaller, and heterozygotes have a higher chance to mate. However, the narrow region in which assortment can evolve in (infinitesimally) small steps ($c \approx 0.5$) vanishes in the presence of dominance. If $0.3 \lesssim a \lesssim 0.8$, dominance decreases disruptive competition at the ecological locus more strongly than the differences in mating success between homozygotes and heterozygotes. Therefore, the S^- regime applies (Figure 2.1 (e)) and assortment cannot further evolve. Dominance has no significant effect on invasion fitness if assortment is sufficiently strong ($a \gtrsim 2$). Then, the S^+ ($c \lesssim 1.5$) or C^- ($c \gtrsim 1.5$) regime applies and selection at the modifier locus is very weak. These findings suggest that dominance hinders the build-up of reproductive isolation in small steps.

Although the concept of invasion fitness is a useful first step in understanding the evolutionary dynamics, no information about the global dynamics can be obtained. Together with the other parameters, the degree of assortment determines, which of the regimes described in Section 2.3.2 applies. Since assortment evolves in our model, different regimes can apply at different points in time for a fixed set of parameters. Our analytical results on the evolution of assortment show that the build-up of reproductive isolation is most likely if modifier alleles have large effects (see also Matessi et al., 2001; Pennings et al., 2008; Durinx and van Dooren, 2008; Otto et al., 2008). However, predictions based on invasion

fitness are most accurate for small modifier effects. Thus, it is necessary to consider the global dynamics of the model to gain complete understanding of the effect of dominance on the evolution of assortative mating.

2.6.2 Global dynamics

Here, we consider the whole evolutionary trajectory of the gene-frequency vector and the population size. A newly introduced modifier can either rise to fixation, die out, or can be maintained at intermediate frequency. Furthermore, the existence of multiple stable equilibria is possible. Consequently, the fate of a modifier may depend on its initial frequency.

2.6.2.1 Invasion, maintenance and fixation of a modifier with small effect

First, we consider modifiers of small effect ($\tilde{a} = 0.05$) in an initially randomly mating population. The impact of the modifier effect's size is discussed in Section 2.6.2.2, and the effect of the initial degree of assortment in Section 2.6.2.3.

Figure 2.3 illustrates the evolutionary outcome for a modifier with effect $\tilde{a} = 0.05$. No multiple stable equilibria were detected. Thus, all results apply for the standard, rare-modifier, and frequent-modifier scenario. Higher levels of assortment are favored in almost the whole parameter space. Only if dominance is almost complete ($d \approx 0.9$) and competition is moderately strong ($c \gtrsim 0.8$), assortment cannot evolve at all. Then, (2.5) is $\cap-$shaped and the C^- regime applies. In the remaining parameter range (2.5) is $\cup-$shaped if the modifier is rare, and the C^+ regime applies. However, whether a modifier can also go to fixation depends crucially on competition and dominance. Remember that in the quadratic model without dominance, a modifier with small effect $\tilde{a}$ goes to fixation if competition is sufficiently strong, i.e., $c > s + \tilde{a}$. Apparently, this result can be extended to the full model with dominance. Since dominance decreases the effect of competition, we expect that the value of c that is necessary for fixation increases with increasing d. In fact, a modifier cannot go to fixation if c is small and $d > 0$ (see $c = 0.2$ in Figure 2.3). If the modifier is close to fixation, the S^- regime applies and thus the modifier is maintained at intermediate frequency. Similarly, small assortment modifiers cannot go to fixation if the degree of dominance exceeds a certain critical value ($d \gtrsim 0.7$). The reason is that disruptive selection is very weak if dominance is sufficiently strong. If the strength of assortment increases, selection becomes stabilizing. If $c \lesssim 0.8$, the S^- regime applies if the modifier is sufficiently frequent, and if $c \gtrsim 0.8$, the C^- regime applies if the modifier is sufficiently frequent. In both cases, a modifier will spread while rare, but cannot go to fixation.

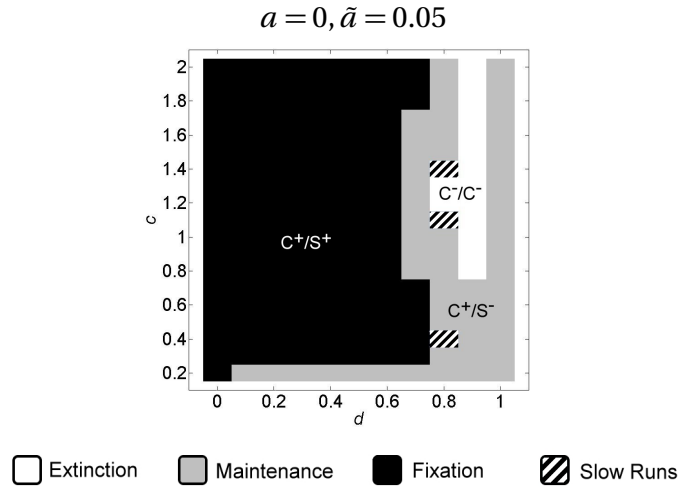

Figure 2.3: Regions of maintenance and fixation of a modifier increasing assortment slightly ($\tilde{a} = 0.05$) in an initially randomly mating population. We used a grid with stepsize 0.1 for the parameters $d \in [0,1]$ and $c \in [0.2,2]$. The other parameters are $s = 0.1$ and $r = 0.5$. In addition to the color code, different regions are labeled R_r/R_f, where R_r and R_f are the selection regimes that apply if the modifier is rare or frequent, respectively. The color code indicates the different evolutionary outcomes. In the extinction regions, the modifier died out in all runs. In the maintenance regions, the modifier coexisted with the wild-type in all runs, whereas in the fixation region the modifier was fixed for all runs. Parameter combinations for which none of the runs equilibrated within 10^6 generations are indicated as slow run regions.

2.6.2.2 Size of the modifier effect

As discussed in Section 2.4, the size of the modifier effect plays a crucial role in the evolution of assortment. Assortment reduces the frequency of heterozygotes at the ecological locus. Hence, it increases the viability of individuals in the middle of the phenotypic range. In addition, assortative mating induces sexual selection, which can be stabilizing or disruptive, depending on the strength of assortment. Finally, if assortment is very strong, selection at the modifier locus will be very inefficient because the frequency of heterozygotes at the ecological locus is strongly reduced. For a fixed set of parameters, different regimes can apply at different points in time, especially if modifier effects are large. This may result in multiple stable equilibria. An initially rare modifier with large effect can become fixed only if sufficiently strong disruptive sexual selection is established during its sweep. Figure 2.4 illustrates the evolutionary outcome of modifiers with different effect sizes. We first observe that the effect size does not affect the region in which an initially rare modifier is lost. The reasons for loss of modifiers are the same as in the case of modifiers with small effect. In contrast, the fixation region depends in a nonlinear and complicated way on the modifier effect and the initial frequency of the modifier.

First, we consider a modifier with effect $\tilde{a} = 0.5$ (Figure 2.4 (a)). Again, no multiple

stable equilibria were detected. The fixation region collapses to a narrow region in the parameter space ($0.3 \lesssim c \lesssim 0.6$ and $0 \lesssim d \lesssim 0.6$). In this region, the C^+ regime applies if the modifier is rare, and the scenario S^+ applies if the modifier is frequent. If $d \gtrsim 0.6$ and $c \lesssim 0.6$, heterozygotes at the ecological locus are less fit than homozygotes if the modifier is sufficiently rare (regime C^+). If the modifier increases in frequency, competition in the middle of the phenotpye range is reduced because of dominance and assortment, and the S^- regime applies. Consequently, the modifier cannot become fixed. If $c \gtrsim 0.7$, competition is strong enough to establish a niche in the middle of the phenotypic range during the spread of a modifier, i.e., the C^- regime applies for a sufficiently frequent modifier. As a rule of thumb, modifiers with intermediate effect can only go to fixation if they manage to jump the "gap" in which one of the S^- or C^- regimes applies (cf. Figure 2.2).

If modifiers have large effect ($\tilde{a} \gtrsim 2$), disruptive sexual selection is strong for frequent modifiers. Therfore, initially frequent modifiers go to fixation in a wide parameter range. These parameter ranges are hatched in Figures 2.4 (b) - (d). However, fixation was only observed if the modifier is initially at very high frequency, i.e., in the frequent-modifier scenario. Since we are primarily interested in the build up of reproductive isolation, we restrict attention to the standard and the rare-modifier scenario for the rest of the section.

If the modifier effect is moderately strong ($\tilde{a} = 2$; Figure 2.4 (b)), the fixation region increases compared to the case $\tilde{a} = 0.5$. In particular, a broader range of values for c allows fixation of the modifier. The reason is that the S^- (for small c) and C^- (for intermediate c) regimes are less likely to occur during the spread of modifiers with sufficiently large effect. The range for d in which modifiers become fixed also increases compared to the case $\tilde{a} = 0.5$. Weak disruptive selection is sufficient for invasion. This occurs if d is large. If a modifier increases in frequency, strong disruptive sexual selection will be established and the modifier will go to fixation. Interestingly, intermediate dominance is most favorable for fixation of a modifier. If the level of assortment increases in (a part) of the population, a niche in the middle of the phenotypic range may appear. Dominance impedes heterozygotes to exploit such a niche (cf. Figure 2.1 (c)). This means that the S^+ regime can be easier established if dominance is moderately strong. If dominance is strong, there is almost no difference in mating success between homozygotes and heterozygotes at the ecological locus. If competition is sufficiently strong, the regime C^- applies if the modifier rises in frequency. Consequently, an initially rare modifier does not become fixed if the degree of dominance is high and competition is at least moderately strong. This explains why intermediate dominance maximizes the size of the fixation region.

Next we consider modifiers that lead to (almost) complete reproductive isolation if fixed. Figure 2.4 (c) and (d) illustrate the fate of a modifier with effect $\tilde{a} = 8$ and $\tilde{a} = 16$, respectively. Quite surprisingly, the positive effect of dominance on the fixation of modifiers is most pronounced if modifiers have large effects. Strong assortment, which is quickly

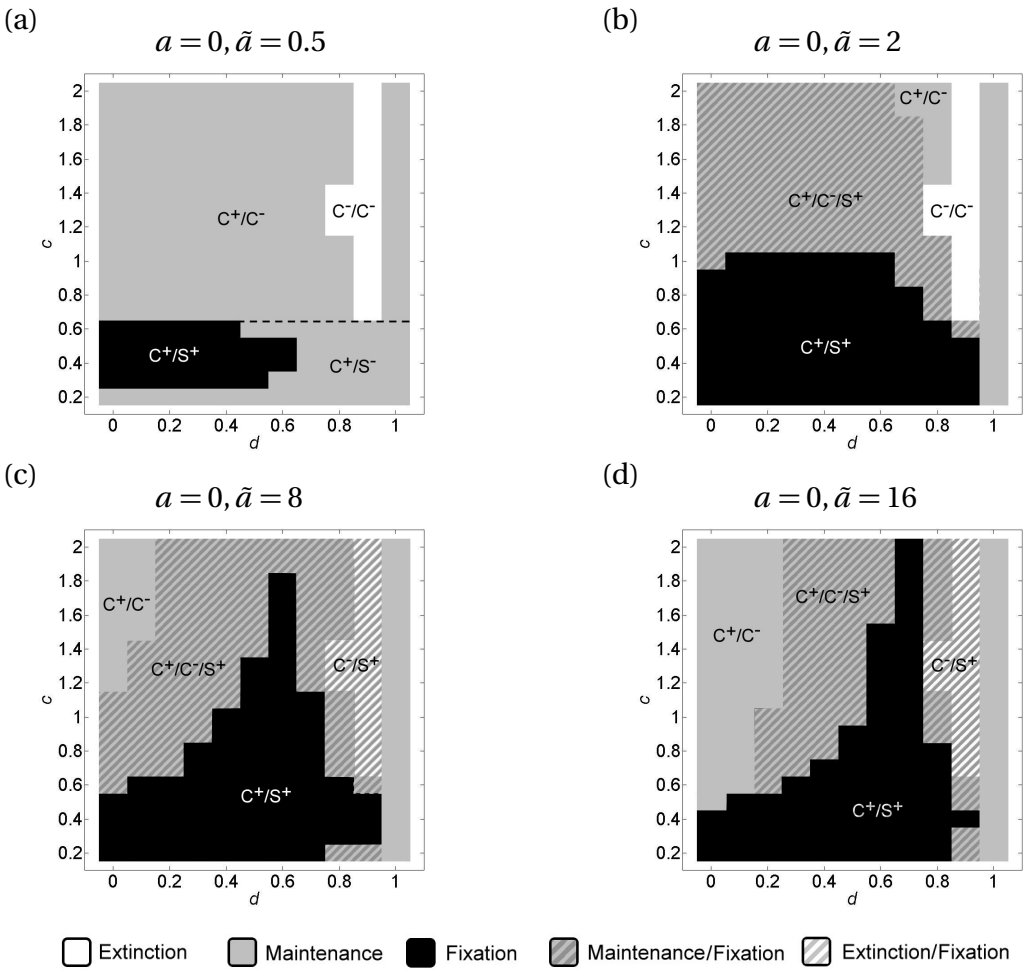

Figure 2.4: Regions of maintenance and fixation of a modifier increasing assortment with different effects in an initially randomly mating population. We used a grid with stepsize 0.1 for the parameters $d \in [0,1]$ and $c \in [0.2,2]$. In all figures we used $s = 0.1$ and $r = 0.5$. The modifier effects are (a) $\tilde{a} = 0.5$, (b) $\tilde{a} = 2$, (c) $\tilde{a} = 8$, and (d) $\tilde{a} = 16$. In addition to the color code, different regions are labeled R_r/R_f or $R_r/R_i/R_f$, where R_r, R_i, and R_f are the selection regimes that apply if the modifier is rare, at intermediate frequency, or frequent, respectively.

established if modifiers have large effect, leads to extremely strong disruptive sexual selection. If $c \gtrsim 0.5$, dominance is necessary for fixation of the modifier. In the absence of dominance and if $c \gtrsim 0.5$, the reduced mating success of heterozygotes is compensated by the existence of a niche in the middle of the phenotype range before the modifier goes to fixation (Figure 2.5 (a)). Consequently, an initially rare modifier will not spread to fixation. The presence of dominance does not change the strength of sexual selection unless it is sufficiently strong (Figure 2.5 (b)). The “valley" of low mating probabilities in the middle of the phenotypic range becomes deeper and flatter with increasing assortment. Dominance has almost no effect on the strength of disruptive sexual selection as long as the phenotypic

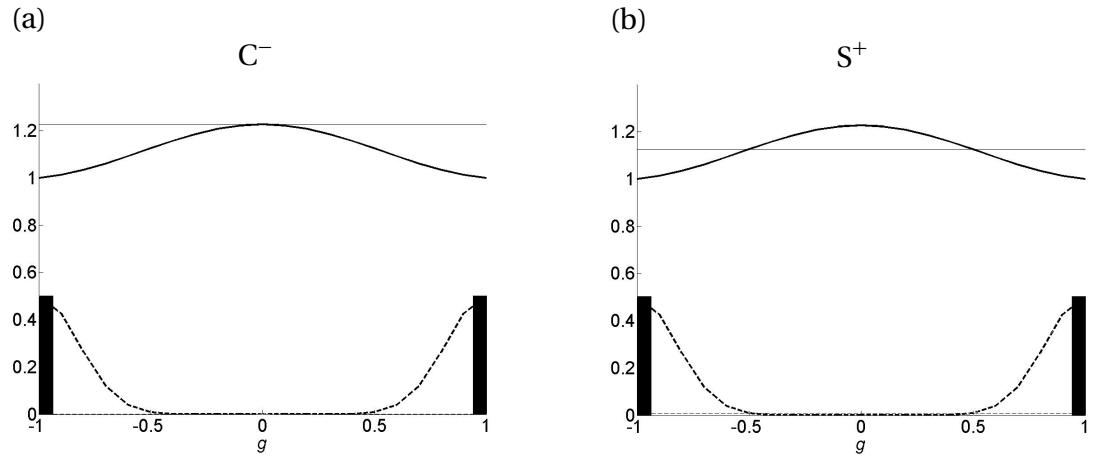

Figure 2.5: Viability $W(g)$ (thick solid line), mating probability $\overline{\pi}(g)$ (thick dashed line) and phenotype distributions (black and gray bars) at the fixation equilibrium if the modifier has large effect ($\tilde{a} = 8$). In (a) there is no dominance and the modifier cannot go to fixation. In (b), dominance is intermediate ($d = 0.5$) and the modifier goes to fixation if sufficiently frequent. The strength of competition is $c = 0.8$ in both figures. Furthermore, $s = 0.1$ and $r = 0.5$. Thin straight lines show the viability (solid) and the mating probability (dashed) of heterozygotes. Equilibrium frequencies of homozygotes at the ecological locus are indicated by black bars and frequencies of heterozygotes are indicated by gray bars.

value of heterozygotes at the ecological locus stays in this valley. In contrast, if dominance increases, the viability of heterozygotes decreases strongly (Figure 2.5 (b)). This explains why the optimal degree of dominance increases with increasing modifier effect.

2.6.2.3 Dependence on the initial level of assortment

As argued in Section 2.6.1, the initial degree of dominance is an important factor for the evolution of assortment. Here, we investigate the effect of the initial level of assortment. Figure 2.6 illustrates the evolutionary outcome of modifiers with small effect for various initial degrees of assortment. No multiple stable equilibria were detected. Even a small amount of initial dominance leads to a substantial change of the region in which modifiers are maintained. The maintenance region shrinks with increasing initial assortment and approaches its minimum at $a \approx 0.5$ (see Figure 2.6 (a)). If assortment is weak ($a \lesssim 0.5$), sexual selection is stabilizing. Thus, the C^+ region decreases with increasing assortment. If competition is weak ($c \lesssim 0.5$), stabilizing sexual selection outweighs disruptive selection at the ecological locus and the S^- regime applies. Furthermore, dominance decreases the effect of competition. Therefore, if competition is weak the S^- region is established for weaker assortment. If competition is strong ($c \gtrsim 0.5$), a niche in the middle of the phenotype spectrum can be established if the frequency of heterozygotes is reduced. Thus, the C^+ region is replaced by the C^- region if initial assortment increases.

The fixation and maintenance regions increase again if the initial degree of assortment is intermediate ($0.5 \lesssim a \lesssim 2$). Then, disruptive sexual selection can be established if $c \lesssim 1$. Dominance slightly decreases the region in which a modifier is maintained or goes to fixation. However, the effect of dominance is less pronounced compared with the case of weak initial assortment. If initial assortment is intermediate, evolution can be very slow and slow runs are observed. If assortment is strong ($a \gtrsim 2$) only slow runs are observed (data not shown). This is consistent with our results about invasion fitness. We conclude that establishment of high levels of assortment via a series of invasion and fixation of modifiers with small effect seems unlikely.

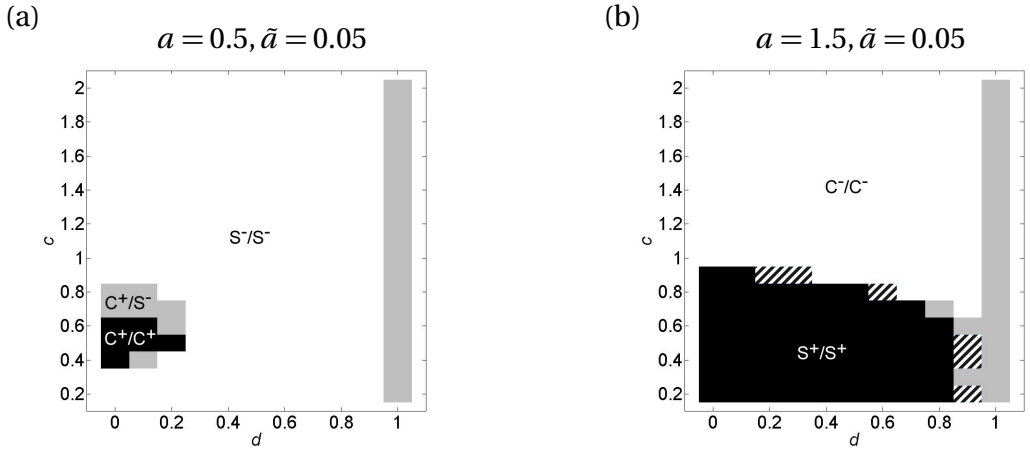

Figure 2.6: Regions of maintenance and fixation of a modifier increasing assortment slightly ($\tilde{a} = 0.05$). We used a grid with stepsize 0.1 for the parameters $d \in [0,1]$ and $c \in [0.2,2]$. In all figures we used $s = 0.1$, $r = 0.5$, and $\tilde{a} = 0.05$. The degree of initial assortment is (a) $a = 0.5$ and (b) $a = 1.5$.

2.6.3 Evolution of assortative mating

The build-up of reproductive isolation via allele substitutions of initially rare modifiers with small effects faces several problems. Positive frequency-dependence due to an intermediate level of assortment can lead to overall stabilizing selection because it outweighs disruptive selection resulting from competition. On the other hand, if assortment is weak or moderate, and competition sufficiently strong, a niche in the middle of the phenotype range appears if heterozygotes become sufficiently rare. Finally, for high levels of assortment, a severely reduced frequency of heterozygotes can neutralize selection at the modifier locus.

Our approach allows us to construct sequences of invasion and fixation modifiers with different effects. In this way, several estimates can be constructed. If we consider only initially rare modifiers with small effect, we get an estimate for the degree of assortment that

can evolve by small steps (see Figure 2.7 (a)). Figure 2.7 (a) shows that only small degrees of assortment can evolve in this way. Except for a small region of intermediate competition and very small dominance, only small levels of assortment can evolve. Furthermore, assortment does not evolve above a moderate level ($a = 2$).

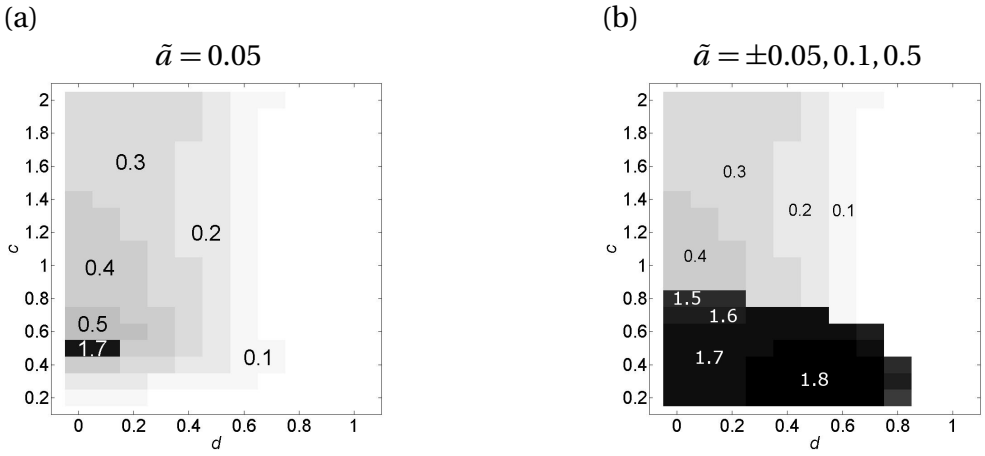

Figure 2.7: Evolutionary stable degrees of assortment that can evolve via allele substitutions of initially rare modifiers if modifiers have small positive effect (a), or various positive or negative effects (b). The other parameters are $s = 0.1$ and $r = 0.5$.

In Figure 2.7 (b) we consider modifiers of slightly larger effect and also allow modifiers with negative effect ($\tilde{a} = \pm 0.05, 0.1, 0.5$). This gives us an estimate for the evolutionary stable degree of assortment if modifier effects are small, but sufficiently large to jump the gap described in Section 2.6.2.3. Then, the region in which moderate assortment evolves increases substantially. However, strong levels of assortment, which are necessary for speciation, cannot evolve.

Thus, we conclude that evolution of assortment is most likely if modifier effects are large, so that complete reproductive isolation can be established in a single step. Then however, a moderately strong degree of dominance is favorable for the evolution of strong reproductive isolation and hence also for sympatric speciation.

2.6.4 Rate of evolution

It is not only relevant whether an initially modifiers becomes fixed, but also whether this happens within a biologically meaningful time. Therefore, for a fixed parameter combination, we recorded the mean fixation time of a modifier (over all initial conditions). Figure 2.8 shows the mean fixation time of initially rare modifiers in an initially randomly mating population. If the modifier effect is small ($\tilde{a} = 0.05$, Figure 2.8 (a)), the C^+ regime applies during the spread of a modifier, and dominance mainly weakens disruptive selection at

the ecological locus. If the modifier effect is large ($\tilde{a} = 8$, Figure 2.8 (b)), the time until fixation is much longer compared to modifiers with small effect. Initially, while the C^+ regime applies, selection for modifiers with large effect is stronger than for modifiers with small effect. However, the frequency of heterozygotes is reduced very quickly and then the scenario S^+ applies until fixation. As we have discussed above, selection is very weak in the S^+ regime. Therefore, the time until fixation increases if modifier effects are large. Similarly, the time until fixation increases with increasing initial assortment (data not shown).

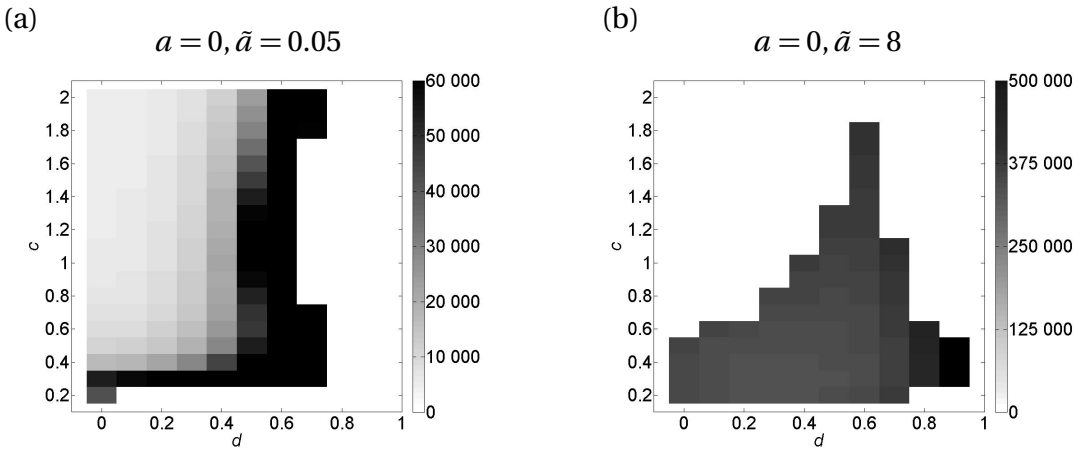

Figure 2.8: Mean fixation time of an initially rare modifier with small ($\tilde{a} = 0.05$, (a)), or large ($\tilde{a} = 8$, (b)) effect. The other parameters are $s = 0.1$ and $r = 0.5$. Note that we used different scales in the figures.

2.6.5 Speciation

The evolution of sufficiently high levels of reproductive isolation can lead to speciation. By speciation we mean that the population is split into two different phenotypic clusters with hardly any gene flow between the clusters.

We shall say there occurs speciation if the following two conditions hold:

1. The probability that two individuals with different genotype at the ecological locus mate is less than 10^{-4}.

2. The probability that two individuals with the same genotype at the ecological locus produce offspring with a genotype other than their own is less than 10^{-4}.

The critical threshold for the strength of assortment that is necessary for speciation depends on the strength of competition and dominance. One should mention that indirect selection is already very weak for $a \gtrsim 2$. Thus, the occurrence of speciation may depend

critically on the threshold values used in the definition of species clusters. Smaller thresholds may increase the values for a that are necessary for speciation. In our case, the critical degree of assortment necessary for speciation is $a \approx 8$.

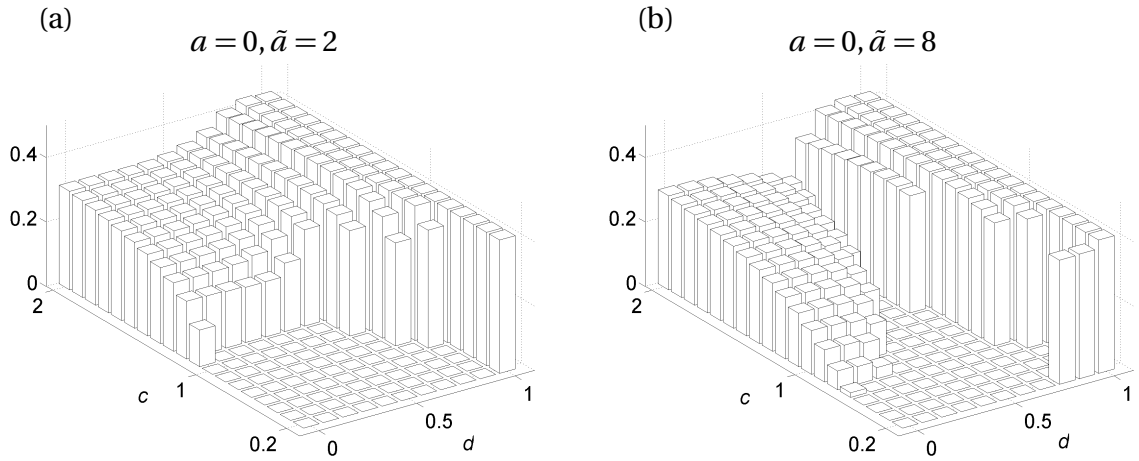

Figure 2.9: Frequeny of heterozygotes at the ecological locus at equilibrium. Parameter values are $s = 0.1$, $r = 0.5$, $\tilde{a} = 2$ in (a), $\tilde{a} = 8$ in (b), and $a = 0$ in both figures.

Our results show that establishment of sufficiently high degrees of assortment for the occurrence of speciation is unlikely if modifiers have small effect. If the population mates initially randomly and modifiers have a sufficiently large effect ($\tilde{a} \gtrsim 4$), speciation occurs in the parameter range in which modifiers become fixed. In the regions, in which modifiers are maintained at intermediate frequency, speciation could, at least theoretically, occur as well. Our analysis shows that the region in which speciation occurs coincides exactly with the fixation regions of modifiers with sufficiently large effect. This suggests that our results are robust with respect to changes of the threshold value in our definition of speciation. In fact, the equilibrium frequency of heterozygotes at the ecological locus is quite high in the maintenance regions. Figure 2.9 shows the frequency of heterozygotes at equilibrium for a modifier with effect $\tilde{a} = 2$ (a) and $\tilde{a} = 8$ (b) in an initially randomly mating population. We conclude that fixation of modifiers with sufficiently large effect is necessary for speciation.

2.7 Discussion

Intraspecific competition, or, more generally, negative frequency-dependent selection, is a commonly used ecological setup to model the evolution of assortment and symptaric speciation (e.g., Udovic, 1980; Wilson and Turelli, 1986; Dieckmann and Doebeli, 1999; Matessi et al., 2001; Pennings et al., 2008). The african finch *Pyrenestes ostrinus*, an often cited justification for this ecological setup, however, did not evolve assortment (Smith, 1990, 1993),

but avoids unfit heterozygotes because one morph is completely dominant. Assortative mating and dominance are commonly considered as alternative evolutionary responses to avoid heterozygous disadvantage (e.g., Durinx and van Dooren, 2008). Peischl and Schneider (2009), however, emphasized the importance of their interactions.

Here, we studied the evolution of assortative mating under intraspecific competition in the presence of dominance. In our model, a single diallelic (ecological) locus has a major effect on a quantitative trait under a mixture of stabilizing selection, intraspecific competition, and density regulation. The trait expresses an arbitrary degree of intermediate dominance. An additional diallelic (modifier) locus determines the strength of assortative mating with respect to the ecological trait ('magic trait', cf. Gavrilets, 2004). Assortative mating follows Matessi et al. (2001), based on the original formulation by Gavrilets and Boake (1998): choosiness is expressed only in females, who pick their mates based on similarities in their trait values. Although our model ignores direct costs for choosy females, assortative mating induces sexual selection, which may be stabilizing or disruptive, depending on the strength of assortment.

In our model, negative frequency dependence (caused by intraspecific competition) favors sufficiently different and rare types. This is opposed by positive frequency dependence (caused by assortative mating) selecting for similar and common types. The amount of competition and sexual selection experienced by the individuals changes if assortment evolves because the frequency of heterozygotes (at the ecological locus) change. Hence, as assortment increases, selection is less efficiently transmitted from the ecological to the modifier locus. Since, for given parameters, it is not straightforward which selective components are responsible for the final evolutionary outcome, we identified four different selection regimes (see Section 2.3.2) that are helpful to interpret our results:

Heterozygotes are common for weak assortment and competition leads to disruptive selection, i.e., selection for higher levels of assortment (regime C^+). If competition is too weak, stabilizing (sexual) selection dominates and assortment cannot evolve (regime S^-). Strong assortment induces disruptive selection because rare heterozygotes are selected against. This heterozygote disadvantage can be compensated by strong competition and assortment cannot evolve (regime C^-). However, if assortment is sufficiently strong relative to competition, even stronger assortment can evolve (regime S^+).

We derived simple invasion and fixation conditions under the assumptions of weak selection and/or weak assortative mating. If assortment is initially weak, higher levels of assortment can evolve whenever competition is sufficiently strong ($c > s + a/2$, regime C^+) in the absence of dominance. Modifiers with small effect do not necessarily go to fixation if they can invade (regime S^-). In contrast, modifiers with large effect become fixed if sufficiently frequent (regime S^+). Thus, strong assortment evolves easier if modifiers have large effect. If dominance is either weak or almost complete, and the population initially mates

randomly, assortment can evolve if $c > s$. Hence, dominance has no significant effect on the initial evolution of assortment starting from randomly mating.

The complexity of the model prohibits further analytical investigations. Thus, we pursued a thorough numerical approach to study arbitrarily strong assortment and competition, and different modifier effect sizes. We focused on parameter combinations that lead to disruptive selection under random mating. Hence, an initial increase of assortment occurred almost in the whole parameter space (cf. Figure 2.3). However, the modifier's fixation region depends strongly on its initial frequency, its effect size, and the degree of dominance.

For small modifiers (Figures 2.2, 2.3, 2.6, 2.7 (a)) complete assortment can evolve only if competition is moderately strong and dominance is weak (see Figure 2.2 and 2.7 (a)). If competition is weak, only partial reproductive isolation can evolve because stabilizing sexual selection neutralizes disruptive selection due to competition (regime S^-) (cf. Matessi et al., 2001; Pennings et al., 2008; Otto et al., 2008). For sufficiently strong competition intermediate phenotypes become advantageous as assortment increases (regime C^-) (cf. Pennings et al., 2008). Noteworthy, the regime C^- does not exist in the quadratic model and was therefore not detect by Matessi et al. (2001). In general, dominance decreases the parameter range in which assortment can further evolve because the regimes S^- or C^- are easier established. Even small degrees of dominance can cause a significantly lower level of evolutionary stable assortment (see Figure 2.7 (a)). This complements the findings of Durinx and van Dooren (2008), who claimed that dominance hinders the evolution of assortment.

Disruptive sexual selection can be established very quickly during the spread of large modifiers. An initially rare, sufficiently large modifier can “jump over the gap" in which either the S^- or C^- regime applies. Thus, in certain parameter regions only sufficiently large modifiers can become fixed (cf. Figure 2.3 and 2.4). In particular, dominance supports the evolution of reproductive isolation if modifiers have sufficiently large effect.

The reason is that small degrees of dominance have little effect on the strength of disruptive sexual selection if assortment is sufficiently strong, but the viability disadvantage of heterozygotes vanishes as dominance increases (see Figure 2.5). This effect is reversed for very strong dominance. Hence, intermediate dominance is optimal for the evolution of assortment in large steps. Moreover, as assortment increases, higher levels of dominance become necessary to compensate heterozygote disadvantage resulting from sexual selection. Therefore, the optimal degree of dominance increases with increasing modifier effect. It should be mentioned that assortment cannot decrease by sufficiently large, rare modifiers in a wide parameter range (hatched area in Figure 2.4).

We also studied fixation times of initially rare modifiers. The evolution of assortment is very slow if sexual selection is the driving force for fixation (see Figure 2.8 (b)). By no means

can invasion fitness be used as a proxy for fixation time. Although the initial strength of selection increases with increasing modifier effect, fixation of large modifiers usually takes longer than fixation of small modifiers (cf. Figures 2.8 (a) and (b)). Furthermore, the fixation time of large modifiers is minimized for intermediate dominance.

Finally, we briefly studied the occurrence of speciation in our model. Modifiers with large effect are much more likely to establish strong reproductive isolation, a prerequisite for speciation. For such modifiers, our results suggest that intermediate dominance is most supportive for sympatric speciation. In general, the build-up of strong reproductive isolation is rather slow. The reason is that selection at the modifier locus is very weak if heterozygotes at the ecological locus become rare. In a natural population, evolution of assortment might stop at some intermediate level. Only if sufficiently strong assortment evolves by a single allele substitution, the occurrence of speciation seems likely.

Our present results combined with those of Peischl and Schneider (2009) allow us to draw conclusions about the simultaneous evolution of dominance and assortment. Peischl and Schneider (2009) assume the same ecological model, but the level of assortment is a fixed parameter and the degree of dominance evolves. As shown there, the evolution of dominance is impeded by small degrees of assortment but supported by intermediate degrees. In particular, time to fixation is minimized for modifiers inducing complete dominance and intermediate assortment. Peischl and Bürger (2008), Peischl and Schneider (2009), and our results show that fixation times of dominance modifiers are usually shorter than those of assortment modifiers. Hence, we conclude that complete dominance is often the more likely evolutionary outcome. However, mutation rates and mutational step sizes play decisive roles in the simultaneous evolution of dominance and assortment. We expect that neither complete dominance, nor complete assortment will evolve unless one of them evolves very quickly. This coincides with the fact that dominance can support the evolution of reproductive isolation by large modifiers (which are initially rare), but hinders the evolution of intermediate levels of assortment in small steps (see Figure 2.7(a)).

Note that our model does not incorporate (direct) costs for choosiness. Although weak costs for choosiness do not necessarily prohibit the evolution of strong assortative mating (cf. Kopp and Hermisson, 2008; Pennings et al., 2008; Otto et al., 2008), evolution of assortative mating will become less likely. This is in accordance with our conclusion that complete dominance is more likely to evolve than complete assortative mating.

Our study differs from previous work on the evolution of assortment because we explicitly studied the effect of dominance and considered the global dynamics. Furthermore, we studied a large part of the parameter space, including intermediate levels of assortment and large modifier effects. Hence we detected previously unobserved phenomena. Furthermore, we can draw conclusions on the simultaneous evolution of assortative mating and dominance.

Durinx and van Dooren (2008) studied the evolution of dominance or assortative mating using an adaptive-dynamics approach. They compared the invasion fitness of dominance and assortment modifiers of small effect, and concluded that dominance and assortment are mutually exclusive alternatives where the occurrence of one decreases the likelihood of the other. Our results yield a more complete picture. Dominance hinders the evolution of assortment if modifier effects are small, but promotes it if modifier effects are large. For a detailed discussion of the differences between our approach and the approach of Durinx and van Dooren (2008) we refer to Peischl and Schneider (2009).

The importance of modifiers of large effect, which may overcome the gap in which either the S^- or C^- regime applies, was also pointed out in a similar study by Pennings et al. (2008). They explored the evolution of assortative mating in a two-locus two-allele version of the Dieckmann and Doebeli (1999) model. Notably, they used a different ecological model (Roughgarden, 1972), assumed no dominance, and considered several forms of competition. In the absence of dominance, their results are similar to ours. For large modifier effects their results rely on individual-based simulations and suggest that complete assortment evolves within reasonable time if mutations at the modifier locus are sufficiently large and frequent. Our model, however, suggests that the evolution of strong assortative mating takes very long. Apparently, small population sizes and high mutation rates strongly facilitate the evolution of complete reproductive isolation (see also Dieckmann and Doebeli, 1999; Doebeli and Dieckmann, 2005; Waxman and Gavrilets, 2005).

Otto et al. (2008) investigated the evolution of assortment in a more general two-locus two-allele model, based on a local stability analysis and a quasi-linkage equilibrium (QLE) approach. They studied different forms of assortment, and assumed no concrete ecological model. They found simple conditions for the evolution of assortative mating. In the absence of costs, higher levels of assortative mating are favored when homozygotes are, on average, fitter than heterozygotes. However, their derivations often required absence of dominance or weak selection, and the QLE assumption might be problematic for strong assortment. Interestingly, they found that dominance can promote the evolution of assortment under directional selection, i.e., assortment can evolve during during a selective sweep of a partially recessive, beneficial mutation. Moreover, assortative mating evolves easier without sexual selection, provided viability selection is disruptive ($\cup$-shaped). However, in models of intraspecific competition, rare heterozygotes can be at a fitness maximum, which would stop the evolution of assortment in the absence of sexual selection. In our model, dominance supports the evolution of assortment only under disruptive sexual selection.

As Matessi et al. (2001), Durinx and van Dooren (2008), Pennings et al. (2008), and Otto et al. (2008) we assumed that a single diallelic locus determines the trait value. Although the equilibrium structures are largely consistent with those in multi-locus mod-

els (Doebeli, 1996; Dieckmann and Doebeli, 1999; Bolnick, 2006; Doebeli et al., 2007), in the latter more than two reproductively isolated species can evolve (Bolnick, 2006; Bürger et al., 2006). A recent study of a multilocus version of the model of Pennings et al. (2008) performed by Rettelbach et al. (unpublished) shows that the genetic architecture of the ecological trait hardly influences the parameter range in which two reproductively isolated species can evolve. Since, in multilocus competition models, disruptive selection often concentrates all genetic variation at a single locus (Van Doorn and Dieckmann, 2006; Kopp and Hermisson, 2006), our result should extend to such cases. (Note, however, that the maintenance of multilocus polymorphism depends highly on genetic constraints, (cf. Schneider, 2007).)

Recently, de Cara et al. (2008) found that the evolution of assortment requires underdominance or epistasis at the fitness level in their study using a multilocus framework. Hence, intermediate dominance at the trait level might have important consequences in multilocus models for the evolution of assortment and deserves further attention. Noteworthy, in de Cara et al. (2008), intermediate degrees of assortment were not evolutionary stability, which disagrees with our results and those of Matessi et al. (2001), Pennings et al. (2008), and Otto et al. (2008). However, the results of Doebeli (1996); Dieckmann and Doebeli (1999); Bolnick (2006); Doebeli et al. (2007) and Rettelbach et al. (unpublished) on multilocus models suggest that the disagreement is due to the different assumptions about selection and not a consequence of the genetic architecture.

All this suggests that our results are robust with respect to variations in the specific intraspecific-competition model, but highly dependent on the assumptions about assortative mating. Our results should continue to hold as long as assortative mating induces positive frequency-dependent selection. Predicting the robustness of our results to changes in the genetic architecture seems more difficult. We expect our results to hold in multilocus models as long as intraspecific competition causes negative frequency-dependent selection.

We showed that dominance and assortment are not necessarily exclusive alternative responses to disruptive selection. However, unless modifiers have large effects, already quite low degrees of dominance severely limit the potential for the evolution of female choosiness. Our results suggest that dominance is the more likely evolutionary response to intraspecific competition. Furthermore, we emphasized the importance of studying global dynamics and the limitations of invasion fitness approaches. However, the evolution of assortment or dominance are not the only possible responses to disruptive selection (Rueffler et al., 2006). Other responses include the evolution of sexual dimorphism (Van Dooren et al., 2004), niche width (Ackermann and Doebeli, 2004), and bet hedging (Leimar, 2005). The co-evolution of genetic architecture, individual specialization, and assortative mating

is a fascinating area of research that still harbors many challenges for future studies.

Acknowledgements: This work was supported by grants of the Vienna Science and Technology Fund (WWTF) and of the Austrian Science Foundation (FWF) (P21305-N13) to R. Bürger. Reinhard Bürger's helpful comments and corrections on an earlier draft of the manuscript are gratefully acknowledged. We also want to thank Agnes Rettelbach and Joachim Hermisson for sharing an unpublished manuscript and preliminary results.

Appendix

A Invasion and fixation of assortment modifiers

Here, we derive invasion and fixation conditions for modifiers inducing stronger or weaker assortment. We label the genotypic values and the genotype frequencies according to Table 2.1.

Genotype	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{M}_1\mathscr{M}_1}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{M}_1\mathscr{M}_1}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{M}_1\mathscr{M}_1}$	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{M}_1\mathscr{M}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{M}_1\mathscr{M}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{M}_2\mathscr{M}_1}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{M}_1\mathscr{M}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_1}{\mathscr{M}_2\mathscr{M}_2}$	$\frac{\mathscr{A}_1\mathscr{A}_2}{\mathscr{M}_2\mathscr{M}_2}$	$\frac{\mathscr{A}_2\mathscr{A}_2}{\mathscr{M}_2\mathscr{M}_2}$
Trait value	Z_1	Z_2	Z_3	Z_4	Z_5	Z_6	Z_7	Z_8	Z_9	Z_{10}
Fitness	W_1	W_2	W_3	W_4	W_5	W_6	W_7	W_8	W_9	W_{10}
Frequency	p_1	p_2	p_3	p_4	p_5	p_6	p_7	p_8	p_9	p_{10}

Table 2.1: Notation for genotypes in Appendix A.

In all cases we assume that fitness is given by (2.8) and that population size is constant and close to demographic equilibrium. To derive invasion conditions we can neglect matings between individuals carrying a modifier allele, i.e., $\mathscr{O}(p_i p_j)$ terms ($i,j \in \{4,\ldots,10\}$). Moreover, the genetic composition of the population is adequately described by the vector $(p_1,p_2,p_3,p_4,p_5,p_6,p_7)^T$. It is easily verified that the linearized recursion matrix of the vector $(p_4,p_5,p_6,p_7)^T$ is

$$U = \frac{1}{\overline{W}} \begin{pmatrix} W_1\alpha_1 & W_5\beta_1 r & W_5\beta_2(1-r) & 0 \\ 0 & W_5\beta_1(1-r) & W_5\beta_2 r & W_3\gamma_1 \\ W_1\alpha_2 & W_5\beta_3 r & W_5\beta_4(1-r) & 0 \\ 0 & W_5\beta_3(1-r) & W_5\beta_4 r & W_3\gamma_2 \end{pmatrix}, \tag{2.23}$$

where

$$\alpha_1 = \frac{1}{2}p_1^*(Q_{14}^*+Q_{41}^*)+\frac{1}{4}p_2^*(Q_{24}^*+Q_{42}^*), \tag{2.24a}$$

$$\alpha_2 = \frac{1}{2}p_3^*(Q_{34}^*+Q_{43}^*)+\frac{1}{4}p_2^*(Q_{24}^*+Q_{42}^*), \tag{2.24b}$$

$$\beta_1 = \frac{1}{2}p_1^*(Q_{15}^*+Q_{51}^*)+\frac{1}{4}p_2^*(Q_{25}^*+Q_{52}^*), \tag{2.24c}$$

$$\beta_2 = \frac{1}{2}p_1^*(Q_{16}^*+Q_{61}^*)+\frac{1}{4}p_2^*(Q_{26}^*+Q_{62}^*), \tag{2.24d}$$

$$\beta_3 = \frac{1}{2}p_3^*(Q_{35}^*+Q_{53}^*)+\frac{1}{4}p_2^*(Q_{25}^*+Q_{52}^*), \tag{2.24e}$$

$$\beta_4 = \frac{1}{2}p_3^*(Q_{36}^*+Q_{63}^*)+\frac{1}{4}p_2^*(Q_{26}^*+Q_{62}^*), \tag{2.24f}$$

$$\gamma_1 = \frac{1}{2}p_1^*(Q_{17}^*+Q_{71}^*)+\frac{1}{4}p_2^*(Q_{27}^*+Q_{72}^*), \tag{2.24g}$$

$$\gamma_2 = \frac{1}{2}p_3^*(Q_{37}^*+Q_{73}^*)+\frac{1}{4}p_2^*(Q_{27}^*+Q_{72}^*). \tag{2.24h}$$

An asterisk indicates that genotype frequencies after selection are used. The genotype frequencies of individuals carrying exactly one copy of the modifier in the next generation are then given by $U\cdot(p_4,p_5,p_6,p_7)^T$ and the leading eigenvalue of T determines whether a rare modifier will spread or die out. If the leading eigenvalues is larger than 1 in modulus, the modifier will spread, otherwise it goes extinct. Similarly, one can determine whether a sufficiently frequent modifier will rise to fixation or not. Clearly, for $\tilde{a}=0$ the modifier allele is selectively neutral and the leading Eigenvalue λ of U is 1.

For a modifier with small effect, i.e., $\tilde{a}-a$ will be small and the leading eigenvalue of U can be written as

$$\lambda = 1+\tilde{a}\phi(a,s,c,d)+O(\tilde{a}^2). \tag{2.25}$$

The sign of ϕ determines whether a modifier can invade or not. A rare modifier will spread if and only if $\phi>0$.

Note that it follows from (2.25) that a modifier decreasing assortment will invade if and only if $\phi<0$.

A.1 No dominance, small modifier effect

We start by observing that the case of no dominance allows a few simplifications of the matrix U. Restricting attention to symmetric equilibria, i.e., $\hat{p}_1=\hat{p}_3$, is convenient since all

the equilibria we were able to calculate are symmetric. The matrix U simplifies to

$$\frac{1}{\overline{W}}\begin{pmatrix} W_1\alpha_1 & W_5\beta_1 r & W_5\beta_1(1-r) & 0 \\ 0 & W_5\beta_1(1-r) & W_5\beta_1 r & W_3\gamma_1 \\ W_1\gamma_1 & W_5\beta_1 r & W_5\beta_1(1-r) & 0 \\ 0 & W_5\beta_1(1-r) & W_5\beta_1 r & W_3\alpha_1 \end{pmatrix}, \tag{2.26}$$

since

$$\alpha_1=\gamma_2, \quad \alpha_2=\gamma_1, \quad \beta_1=\beta_2=\beta_3=\beta_4 \tag{2.27}$$

holds at a symmetric equilibrium with no dominance. Furthermore, $W_1 = W_3$. Then, the characteristic polynomial of $\overline{W}U$ is

$$\begin{aligned} P(x)=&\Big(x^2-x(\alpha_1 W_1+\beta_1(1-2r)W_5)+\beta_1(\alpha_1-\gamma_1)W_1W_5\Big) \\ &\times\Big(x^2-x(\alpha_1 W_1+\beta_1 W_5)+\beta_1(\alpha_1-\gamma_1)W_1W_5\Big). \end{aligned} \tag{2.28}$$

As shown in Matessi et al. (2001), the leading eigenvalue of U is the larger of the two solutions of

$$x^2-x(\alpha_1 W_1+\beta_1 W_5)+\beta_1(\alpha_1-\gamma_1)W_1W_5=0 \tag{2.29}$$

divided by $\overline{W}$. Since U is irreducible, the leading eigenvalue is positive and simple. For symmetric equilibria, i.e., $\hat{p}_1=\hat{p}_3$, it is also shown in Matessi et al. (2001) that the leading eigenvalue is larger than one if and only if

$$\Delta=\frac{W_1}{\overline{W}}\Big(1-\frac{1}{2}(Q^*_{12}-Q^*_{21})p_2\Big)>1. \tag{2.30}$$

To derive useful invasion conditions from (2.30) one needs to know the gene frequencies at equilibrium.

A.1.1 Weak initial assortment

Assume that the wild-type allele is fixed at the modifier locus. If the wild-type allele codes for random mating, we obtain the following equilibrium

$$\hat{p}_1=\hat{p}_3=\frac{1}{4},\ \hat{p}_2=\frac{1}{2}. \tag{2.31}$$

Now, consider a small degree of initial assortment a. If $a \ll 1$, we obtain the following

equilibrium

$$\begin{aligned}\hat{p}_1 &= \frac{1}{4}+\frac{a(2-2s+3c)^2}{8(2-s+2c)^2}+\mathcal{O}(a^2),\\ \hat{p}_2 &= \frac{1}{2}-\frac{a(2-2s+3c)^2}{4(2-s+2c)^2}+\mathcal{O}(a^2),\\ \hat{p}_3 &= \frac{1}{4}+\frac{a(2-2s+3c)^2}{8(2-s+2c)^2}+\mathcal{O}(a^2).\end{aligned} \tag{2.32}$$

Substitution of (2.32) into (2.30) yields

$$\Delta = 1+\frac{a-2(c-s)}{4}+\mathcal{O}(a^2)+\mathcal{O}(s^2)+\mathcal{O}(as). \tag{2.33}$$

Thus, assuming weak selection and weak assortment, we get the following condition for invasion

$$c > s+\frac{a}{2}. \tag{2.34}$$

This result includes the case of random mating for $a=0$. An approximation for the leading eigenvalue can also be calculated directly in this case,

$$\lambda = 1+(\tilde{a}-a)\frac{2(c-s)-a}{16}+\mathcal{O}\big((\tilde{a}-a)^2\big). \tag{2.35}$$

In addition, one can infer from (2.35) that a sufficiently frequent modifier with small effect $\tilde{a}$ goes to fixation in a population in which the wild-type codes for random mating if and only if $c < s\tilde{a}$.

A.1.2 Strong initial assortment

We set $\varepsilon = \exp(-a)$ and assume $\varepsilon \approx 0$, so that terms of order $\mathcal{O}(\varepsilon^5)$ can be neglected. Up to fourth order in ε, the equilibrium with the modifier locus fixed for the wild-type allele is

$$\begin{aligned}\hat{p}_1 &= \frac{1}{2}-\varepsilon^4\frac{1+2c-s}{1+3c-2s}+\mathcal{O}(\varepsilon^5),\\ \hat{p}_2 &= 2\varepsilon^4\frac{1+2c-s}{1+3c-2s}+\mathcal{O}(\varepsilon^5),\\ \hat{p}_3 &= \frac{1}{2}-\varepsilon^4\frac{1+2c-s}{1+3c-2s}+\mathcal{O}(\varepsilon^5).\end{aligned} \tag{2.36}$$

Then, the criterion (2.30) for invasion is

$$\varepsilon^4\frac{1+4c-3s}{1+3c-2s}+\mathcal{O}(\varepsilon^5) > 0, \tag{2.37}$$

which is always satisfied since $a, c \in [0, 1/4]$.

A.2 No dominance, large modifier effects

A.2.1 Invasion

Assume no dominance and an population fixed for the wild-type allele at the modifier locus. The wild-type allele codes for random mating. If genotype g carries exactly one copy of the modifier, we set

$$\pi(g,h) = \begin{cases} 1 & if Z_g = Z_h \\ k & if |Z_g - Z_h| = 1 \\ K & if |Z_g - Z_h| = 2, \end{cases} \tag{2.38}$$

where $k = \exp(-\tilde{a})$ and $K = \exp(-4\tilde{a})$. The leading eigenvalue of U, i.e, the larger of the two solutions of (2.29), is

$$\lambda = 1 + \frac{(1-K)(c-s)}{2(3+5K+8k)} + \mathcal{O}(s^2). \tag{2.39}$$

Thus, a modifier increasing assortment can invade a randomly mating population if and only if $c > s$. This result holds for any choice of k and K such that $0 \leq k, K < 1$.

Similarly, in an initially weakly assortatively mating population, the leading eigenvalue of U is

$$\lambda = 1 + \frac{(1-K)[2(c-s)-a]}{4(3+5K+8k)} + \mathcal{O}(a^2) + \mathcal{O}(s^2). \tag{2.40}$$

Consequently, a modifier increasing assortment can invade a population that mates weakly assortatively if and only if $c > s + a/2$. Note, that for the limit, we assumed that $a \ll k, K$, i.e., that the degree of assortative mating caused by the modifier is large compared to the initial degree of assortment.

A.2.2 Fixation

We consider a modifier with large effect $\tilde{a}$, i.e., such that $\varepsilon := \exp[-2\tilde{a}] \ll 1$. The wild type allele at the modifier locus codes for random mating. If the modifier is fixed, the equilibrium gene frequencies at equilibrium are given by (2.36). The leading eigenvalue of U at (2.36) is

$$\lambda = 1 - \exp(-4\tilde{a}) \frac{1-2s+3c}{3-4s+7c} + \mathcal{O}\left(\exp(-6\tilde{a})\right). \tag{2.41}$$

Because $a, s \in [0, 1/4]$, $\lambda < 1$ and the modifier goes to fixation if it is sufficiently frequent.

A.2.3 Complete assortment

Assume that the wild-type allele codes for complete assortment. If genotype g carries exactly one copy of the modifier, we set

$$\pi(g,h) = \begin{cases} 1 & \text{if } Z_g = Z_h\,, \\ \varepsilon & \text{if } |Z_g - Z_h| = 1\,, \\ \varepsilon^4 & \text{if } |Z_g - Z_h| = 2\,, \end{cases} \tag{2.42}$$

where $0 < \varepsilon \ll 1$, i.e., the modifier decreases the strength of assortment by a small amount. Then, the leading eigenvalue of U is given by

$$\lambda = 1 - \varepsilon \frac{1 - 2s + 3c}{3 + 3s - 7c} + \mathcal{O}(\varepsilon^8). \tag{2.43}$$

The fraction in the above expression is always positive because $s, c \in [0, 1/4]$. Thus, a modifier that slightly decreases assortment can not invade a completely assortatively mating population.

A.3 Weak dominance, random mating

The above described invasion criterion is only valid in the absence of dominance. The reason is that the symmetry of the model without dominance is crucial in the derivation of (2.32). Thus, we have to derive the leading eigenvalue directly as a Taylor Series in $(\tilde{a} - a)$. Assume that the population is fixed for the wild-type allele at the modifier locus and that the wild-txpe codes for random mating.

To calculate the equilibrium with monomorphic modifier locus coding for random mating, we assume dominance to be sufficiently weak to terms of order $\mathcal{O}(d^3)$. We obtain

$$\begin{aligned} \hat{p}_1 &= \frac{(c + c\,d + s)^2}{4(c+s)^2} + \mathcal{O}(d^3), \\ \hat{p}_2 &= \frac{1}{2} - \frac{(c^2 d^2)}{2(c+s)^2} + \mathcal{O}(d^3), \\ \hat{p}_3 &= \frac{(c - c\,d + s)^2}{4(c+s)^2} + \mathcal{O}(d^3). \end{aligned} \tag{2.44}$$

Consider the characteristic polynomial P of U at the equilibrium (2.44). For $\tilde{a} = 0$, we know that $P(1) = 0$. More precisely, 1 is the leading eigenvalue if $\tilde{a} = 0$. Thus, if $\tilde{a} \neq 0$, the

leading eigenvalue of U is of the form

$$\lambda = 1 + \tilde{a}\phi + \mathcal{O}(\tilde{a}^2), \tag{2.45}$$

for some ϕ that does not depend on $\tilde{a}$, as $\tilde{a} \to 0$. By neglecting terms of order $\mathcal{O}(\tilde{a}^2)$, the Taylor expansion of P at $\hat{p}$ leads to

$$\phi = \frac{(1-2d^2)(c-s)}{8} + \mathcal{O}(d^3) + \mathcal{O}(s^2). \tag{2.46}$$

It follows that the strength of selection for assortment modifier decreases under weak dominance. However, the condition for invasion is not affected by a small degree of dominance.

A.4 Strong dominance, random mating

We set $\delta = (1-d)$. By strong dominance we mean that $d \approx 1$ such that we can neglect terms of order (δ^2). We derive the equilibrium

$$\begin{aligned}
\hat{p}_1 &= \frac{1}{2} + \frac{\big((4-3\sqrt{2})c + (-2+\sqrt{2})s\big)}{4c}\delta + \mathcal{O}(\delta^2), \\
\hat{p}_2 &= \sqrt{2} - 1 + \frac{\big((-7+5\sqrt{2})c + (3-2\sqrt{2})s\big)}{2c}\delta) + \mathcal{O}(\delta^2), \\
\hat{p}_3 &= \frac{3}{2} - \sqrt{2} + \frac{(3-2\sqrt{2}) + c\big(-3+2\sqrt{2}+(-7+5\sqrt{2})\big)}{2c}\delta + \mathcal{O}(\delta^2).
\end{aligned} \tag{2.47}$$

and

$$\begin{aligned}
\phi &= \frac{(-161564+114243\sqrt{2})(c-s)\delta}{19601-13860\sqrt{2}+(-66922+47321\sqrt{2})(c-s)\delta} + \mathcal{O}(\delta^2) && (2.48)\\
&= (3\sqrt{2}-4)(c-s)\,\delta + \mathcal{O}(\delta^2) + \mathcal{O}(s^2). && (2.49)
\end{aligned}$$

Since $3\sqrt{2} > 4$, the result follows immediately.

A.5 Assortment vs. dominance

Peischl and Schneider (2009) calculated the invasion fitness of a modifier that induces an arbitrary degree of dominance in a randomly mating population. The leading eigenvalue of the linearized transition matrix for a rare dominance modifier with effect d is given by

$$\lambda_d = 1 + d^2\frac{(c-s)}{2+(2c-s)}. \tag{2.50}$$

For an assortment modifier with effect $\tilde{a} \ll 1$ in an initially randomly mating population we get from (2.35)

$$\lambda_{\tilde{a}} = 1 + \tilde{a}\frac{c-s}{8} + \mathcal{O}\left(\tilde{a}^2\right). \tag{2.51}$$

If the modifier effects d and $\tilde{a}$ go to 0, λ_d and $\lambda_{\tilde{a}}$ behave qualitatively differently. Because $\lambda_d = 1 + \mathcal{O}(d^2)$ and $\lambda_{\tilde{a}} = 1 + \mathcal{O}(\tilde{a})$, the strength of selection for a dominance modifier decreases faster than the strength of selection for an assortment modifier.

Part II

Migration and Selection

Chapter 3

Dominance and the maintenance of polymorphism in multiallelic migration-selection models with two demes[1]

[1]With minor changes this chapter is submitted to Theoretical Population Biology as:
Peischl, S., 2010. Dominance and the maintenance of polymorphism in multiallelic migration-selection models with two demes.

Abstract

The maintenance of genetic variation in a spatially heterogeneous environment has been one of the main research themes in theoretical population genetics. Despite considerable progress in understanding the consequences of spatially structured environments on genetic variation, many problems remain unsolved. One of them concerns the relationship between the number of demes, the degree of dominance, and the maximum number of alleles that can be maintained by selection in a subdivided population. In this work, we study the potential of maintaining genetic variation in a two-deme model with deme-independent degree of intermediate dominance, which includes absence of G×E interaction as a special case. We present a thorough numerical analysis of a two-deme three-allele model, which allows us to identify dominance and selection patterns that harbor the potential for stable triallelic equilibria. The information gained by this approach is then used to construct an example in which existence and asymptotic stability of a fully polymorphic equilibrium can be proved analytically. Noteworthy, in this example the parameter range in which three alleles can coexist is maximized for intermediate migration rates. Our results can be interpreted in a specialist-generalist context and (among others) show when two specialists can coexist with a generalist in two demes if the degree of dominance is deme independent and intermediate. The dominance relation between the generalist allele and the specialist alleles play a decisive role. We also discuss linear selection on a quantitative trait and show that G×E interaction is not necessary for the maintenance of more than two alleles in two demes.

3.1 Introduction

The role of environmental heterogeneity and population subdivision as a factor maintaining genetic variation has been the topic of numerous studies in the past (e.g., Levene, 1953; Felsenstein, 1976; Karlin, 1982). Spatially varying selection has been considered as a likely explanation of high levels of genetic variation. In fact, classical population genetics theory shows that a spatially heterogeneous environment can maintain more variation than a homogeneous environment (Levene, 1953; Felsenstein, 1976; Karlin, 1982; Nagylaki and Lou, 2001). In particular, protected polymorphism is possible in the absence of overdominance, which is not the case in panmictic models. Therefore, and because intermediate dominance is the most commonly observed form of dominance, we restrict attention to intermediate dominance.

Analytical results in discrete space migration-selection models mainly concern conditions for protected polymorphism (Prout, 1968; Christiansen and Feldman, 1975; Karlin, 1982; Nagylaki, 1992), and the equilibrium and stability structure of models with no or intermediate dominance (Nagylaki and Lou, 2001; Karlin and Campbell, 1980; Eyland, 1971; Nagylaki and Lou, 2007; Nagylaki, 2009). Considerable progress has been made in understanding the interaction of migration and selection in a number of scenarios. Especially the cases of strong and weak migration are well understood (Karlin and McGregor, 1972; Nagylaki and Lou, 2001, 2007). For a review of single-locus migration-selection models we refer to Nagylaki and Lou (2008). Recently, some of these results have been extended to multilocus models (Bürger, 2009a,b). However, even in simple cases many important open questions and unsolved problems remain. For instance, the relation between the number of demes, the degree of dominance, and the number of alleles that can be maintained at a single locus is not well understood.

In discrete-space migration-selection models with no dominance, the number of demes is a generic upper bound for the number of alleles present at equilibrium (Nagylaki and Lou, 2001). Nagylaki (2009) focused on the maintenance of polymorphism in migration-selection models with intermediate dominance. In particular, he investigated to which extent the results for no dominance can be generalized to intermediate dominance. He showed that in the Levene model, in which individuals disperse independently of their deme of origin, the dynamics under deme-independent degree of intermediate dominance (DIDID) is, generically, qualitatively equivalent to that under no dominance. This generalization however fails if other modes of migration are assumed.

If migration is weak relative to selection, local fitnesses determine which alleles will be maintained. Then, only alleles that appear in the fittest genotype in at least one deme are maintained. Thus, if dominance is intermediate and migration is weak, the number of demes is an upper limit for the number of alleles that can be maintained, provided

in each deme a single genotype has highest fitness. For strong migration one can prove the existence of globally asymptotically stable equilibria with an arbitrary number of alleles segregating if there are at least two demes (Nagylaki and Lou, 2007). If migration is strong relative to selection, the average fitnesses (weighted properly according to the migration pattern) determine which alleles are maintained. For instance, if locally beneficial alleles are dominant, polymorphism can be maintained because of average heterozygous advantage. However, construction of such examples (Nagylaki, 2009, Sect. 4.3) requires deme-dependent dominance, i.e., some form of genotype-environment (G×E) interaction. A deme-independent degree of intermediate dominance includes the case of no G×E interaction as an important special case.

With DIDID, the weak- and strong-migration limits fail to capture the full spectrum of possible evolutionary outcomes. In particular, the strong-migration limit always yields fixation of a single allele. Nagylaki (2009) suggested to explore analytically the maintenance of three alleles under DIDID in two demes. Numerical work by Danninger and Bürger (personal communication) shows that unstable and locally or globally asymptotically stable triallelic polymorphisms can occur in such models.

In this work, we focus on the potential of maintaining multiallelic polymorphism in a two-deme model with arbitrary migration if there is DIDID. However desirable a complete analytical study of this model would be, its complexity prohibits such an analysis. Thus, we pursue a combined approach and perform a thorough numerical analysis as well as analytical investigations. The numerical approach is used to identify the parameter regions in which stable triallelic equilibria exist. It is a challenging task to study the effects of selection, dominance, and migration parameters because of the rather large number of parameters in our model. However, in a symmetric special case we are able to calculate the internal equilibrium and derive necessary and sufficient conditions for its existence. In addition, a bifurcation analysis is performed and it is shown that the internal equilibrium exchanges stability with a monomorphic or dimorphic equilibrium when it enters the state space. It is shown that these results continue to hold under small perturbations of the symmetry assumptions.

In Section 3.2 we present the model. Section 3.3 briefly reviews some results on the maintenance of polymorphism in migration-selection models that will guide our intuitive explanations. The number of alleles that can be maintained under DIDID is investigated numerically in Section 3.4. In Section 3.5 we restrict attention to three alleles and divergent selection such that the fittest allele in one deme is the least fit in the other. This selection scheme includes linear divergent selection without allele-environment interaction as an important special case. There, we identify dominance patterns that enable maintenance of all three alleles and investigate the effect of the various parameters on the existence of internal equilibria. In Section 3.6, we present an analytical bifurcation analysis of a special

case in which three alleles are maintained at an asymptotically stable internal equilibrium. Finally, in Section 3.7 we briefly study linear divergent selection in the absence of G×E interaction. There, we show that that G×E interaction is not necessary for the maintenance of more than two alleles in two demes.

3.2 The model

We consider a randomly mating population with discrete, nonoverlapping generations, in which the two sexes need not be distinguished. The population is subdivided into two panmictic colonies (demes) that exchange adult migrants independently of genotype. In each of the demes, selection acts through differential viabilities, which are time and frequency independent. Mutation and random genetic drift are ignored.

We consider J alleles at a single locus. Let $p_{i,k} = p_{i,k}(t)$ denote the frequency of allele i in deme k at time t. Fitness of an individual in deme k with genotype A_iA_j is given by $w_{ij,k}$. Following the standard recursion relations, allele frequencies after selection are given by

$$p^*_{i,k} = p_{i,k}\frac{W_{i,k}}{\bar{W}_k}, \tag{3.1}$$

where $W_{i,k} = \sum_j w_{ij,k}p_{j,k}$ is the marginal fitness of allele i in deme k and $\bar{W}_k = \sum_i W_{i,k}p_{i,k}$ is the mean fitness in deme k. The state space of this model is $\mathscr{S}_J \times \mathscr{S}_J$, where $\mathscr{S}_J = \{x \in \mathbb{R}^J : \sum_{i=1}^J x_i = 1\}$ denotes the J-dimensional simplex.

After selection, migration occurs. The backward migration matrix is given by

$$M = \begin{pmatrix} m_{11} & m_{12} \\ m_{21} & m_{22} \end{pmatrix}, \tag{3.2}$$

where m_{kl} is the probability that an adult individual in deme k migrated from deme l. We assume soft selection, i.e., population size is regulated within each deme. Thus, M is constant (Nagylaki, 1992). After migration, allele frequencies in the next generation are given by

$$p'_{i,k} = \sum_l m_{kl}p^*_{i,l}. \tag{3.3}$$

Since $m_{k1} + m_{k2} = 1$, we set

$$M = \begin{pmatrix} 1-m_1 & m_1 \\ m_2 & 1-m_2 \end{pmatrix}. \tag{3.4}$$

Unless otherwise stated, we exclude the case $m_1 = m_2 = \frac{1}{2}$, which corresponds to a spe-

cial case of the Levene model, and assume $0 < m_1, m_2 < \frac{1}{2}$. In the Levene model with deme-independent intermediate dominance, the dynamics are qualitatively equivalent to the case of no dominance, i.e., no stable three-allele polymorphism is possible (Nagylaki, 2009).

From Section 3.4 on we assume DIDID (Nagylaki, 2009), i.e., dominance relations are the same in every deme. Thus, fitnesses can be written as

$$w_{ij,k} = w_{ii,k} d_{ij} + w_{jj,k}(1 - d_{ij}), \tag{3.5}$$

where $d_{ij} \in [0,1]$, $d_{ii} = \frac{1}{2}$, $1 \le i, j \le J$, and $k \in \{1,2\}$. We abbreviate this assumption by DIDID (deme-independent degree of intermediate dominance).

For a more detailed presentation of the general migration-selection model we refer to Nagylaki (1992) and references therein.

3.3 Maintenance of polymorphism

3.3.1 Local stability of monomorphic equilibria

Since we ignore mutation, monomorphic equilibria exist always in this model. Local stability of at least one of the monomorphic states means that all genetic variation can be lost. Instability of all monomorphic states on the other hand implies that a least two alleles persist in the population (unless there are heteroclinic cycles, a possibility that has not yet been excluded).

We denote the equilibrium at which only allele A_i is present by $\hat{p}^{(i)}$. At fixation of allele i, only fitnesses of heterozygous individuals carrying exactly one A_i allele are required to determine local stability. Because we assume soft selection, we can rescale fitnesses in each deme without loss of generality. To obtain a simple condition for local stability of $\hat{p}^i$ (i arbitrary but fixed), we rescale fitnesses such that

$$\begin{array}{lcc} & A_iA_i & A_iA_j \\ \text{deme 1} & 1-\alpha_j^{(i)} & 1 \\ \text{deme 2} & 1-\beta_j^{(i)} & 1 \end{array} \tag{3.6}$$

where $1 \le j \le J$, $j \ne i$, and $\alpha_j^{(i)}, \beta_j^{(i)} \le 1$.

The following result can be derived straightforwardly and is well known for two alleles (e.g., Nagylaki 1992 and references therein). The equilibrium $\hat{p}^{(i)}$ is unstable if and only if

$$\alpha_j^{(i)} > 0 \text{ and } \beta_j^{(i)} > 0 \qquad \text{or} \qquad \operatorname{sign}(\alpha_j^{(i)}) \ne \operatorname{sign}(\beta_j^{(i)}) \quad \text{and} \quad \frac{m_1}{\alpha_j^{(i)}} + \frac{m_2}{\beta_j^{(i)}} < 1 \tag{3.7}$$

holds for at least one $j \neq i$.

If $\text{sign}(\alpha_j^{(i)}) \neq \text{sign}(\beta_j^{(i)})$, it follows immediately from (3.7) that $\hat{p}^{(i)}$ is unstable if $\alpha_j^{(i)} > m_1$ or $\beta_j^{(i)} > m_2$. For sufficiently weak migration, all monomorphic equilibria are unstable if $\text{sign}(\alpha_j^{(i)}) \neq \text{sign}(\beta_j^{(i)})$. Obviously, $\text{sign}(\alpha_j^{(i)}) \neq \text{sign}(\beta_j^{(i)})$ is not a sufficient condition for instability of all monomorphic equilibria if migration is strong. The example in Section 3.6 illustrates this.

Since in general boundary equilibria cannot be derived analytically, we are not able to derive useful protection conditions for more than two alleles. Additional assumptions are necessary to investigate the maintenance of polymorphism explicitly.

3.3.2 Polymorphic equilibria

Theorem 2.4 in Nagylaki and Lou (2001) states that if there is no dominance, the number of demes is a generic upper bound for the number of alleles present at equilibrium. By generic we mean that the statement holds for a dense open set of full measure. By a simple modification of the proof this result can be extended to multiplicative fitnesses (Nagylaki and Lou, 2007). By Remark 4.4 in Nagylaki and Lou (2007) it extends to the case of intermediate dominance if migration is sufficiently weak. However, Remark 4.15 in Nagylaki and Lou (2007) demonstrates that the theorem does not extend to sufficiently strong migration. In conclusion, we cannot expect polymorphism involving more alleles than there are demes if there is either no dominance, multiplicative fitnesses, or intermediate dominance and weak migration.

In Nagylaki and Lou (2007) the weak-migration limit and strong-migration limit were derived and applied to different examples. Their results can be used to find out how much genetic variation can be maintained under such conditions.

3.3.2.1 Weak migration

As already mentioned, under weak selection and with intermediate dominance, the number of demes is an upper limit for the number of alleles present at equilibrium. In the following, we discuss this result in more detail. We assume intermediate dominance and no migration, and that in each deme one allele is the fittest, i.e., it has highest homozygous fitness. In the absence of migration, in each deme the fittest allele is fixed and the corresponding equilibrium is globally asymptotically stable (e.g., Nagylaki, 1992; Bürger, 2000). If in the absence of migration all equilibria are hyperbolic, the weak-migration limit can be applied and yields the following result: A globally asymptotically stable equilibrium with exactly the same alleles present exists if migration is sufficiently weak. This equilibrium is also globally asymptotically stable and close to the equilibrium of the model without migration (Karlin and McGregor, 1972; Nagylaki and Lou, 2007). Since in each deme a single

allele is fixed in the absence of migration, the number of demes is an upper bound for the number of alleles present at equilibrium if migration is sufficiently weak.

3.3.2.2 Strong migration

To apply the strong-migration limit, selection has to be sufficiently weak relative to migration. Then, we expect rapid reductions to small quantities of the gene-frequency differences among demes. Evolution will be almost panmictic for suitably averaged gene frequencies. For details we refer to Nagylaki and Lou (2007). In the limiting panmictic system, fitnesses are obtained by averaging over demes. In Remark 4.15 of Nagylaki and Lou (2007) an example of a two-deme system is constructed in which all trajectories converge to an internal equilibrium with arbitrarily many alleles present at positive frequency. However, this construction is not possible if DIDID is assumed. It can easily be seen that dominance is intermediate in the strong-migration limit if dominance is intermediate and deme independent in the original system. In fact, in the limiting system, there exists a globally asymptotically stable monomorphic equilibrium.

If we assume intermediate dominance and weak migration, the degree of dominance has no influence on the maintenance of genetic variation. For a fixed set of fitnesses, the same alleles are maintained under every dominance pattern. If migration is strong, no dominance and DIDID yield global fixation of the same allele. In contrast, a deme-dependent degree of intermediate dominance can yield maintenance of an arbitrary number of alleles. Although these results seem to suggest that the number of demes is an upper bound for the number of alleles that can be maintained under DIDID, recent numerical work by Danninger and Bürger (personal communication) shows that asymptotically stable triallelic equilibria exist in a two-deme model with a deme-independent degree of complete dominance. In the next section we numerically investigate the existence of asymptotically stable equilibria with up to four alleles under DIDID.

3.4 Numerical results for multiple alleles

Arbitrarily many alleles can be maintained in two demes if there is dominance. However, the known examples require that dominance relations are different in the two demes, which means that there is some form of G×E interaction. In contrast, in the absence of dominance at most two alleles can be maintained in two demes. Nagylaki (2009) suggested to investigate the maintenance of more than two alleles in two demes under DIDID. Here, we extend the results by Danninger and Bürger (personal communication) and explore when maintenance of more than two alleles in two demes is possible under DIDID.

We set $J = 4$ and numerically iterated the system (3.1) and (3.3) with randomly chosen fitness values until equilibration. In addition, we performed a set of iterations with $J = 5$. However, no internal equilibria were detected if $J = 5$. Therefore, we only show results for $J = 4$. We assumed symmetric migration and chose $m = m_1 = m_2 = 0.05, 0.1, ..., 0.5$, and for each value of m we randomly picked 10^5 fitness sets satisfying DIDID. More precisely, fitness values of homozygotes and dominance coefficients were drawn independently from a uniform distribution on $[0,1]$. These fitness sets include sets in which one allele is the fittest in both demes. In such a scenario, the fittest allele always becomes fixed. Thus, we performed a second set of iterations in which such fitness combinations were excluded. We call the first set the 'random fitness set' and the second set the 'divergent selection set'. For each parameter set we randomly picked 10 initial conditions such that the minimum Euclidean distance between any two initial conditions is 0.25, and that each allele is initially present at positive frequency. We stopped the iteration if the distance between the allele frequency vectors of two consecutive generations was less than 10^{-10}. We call this equilibration. If equilibration did not occur after 10^6 generations, we terminated the run and called it a slow run. At equilibrium we recorded the allele frequencies and the number of alleles present at equilibrium. We decided that an allele is present in the population if its frequency is larger than 10^{-4} in each deme. Noteworthy, no slow runs or complicated behavior such as limit cycles or chaotic behavior were detected. We call an equilibrium asymptotically stable if at least one trajectory converged to it, and globally asymptotically stable if all 10 trajectories converged to it.

In Figure 3.1 the results of these iterations are summarized. Surprisingly, asymptotically stable internal equilibria were found. At such equilibria all four alleles are maintained at positive frequency.

From the obtained data we can identify the selection scheme that harbors the highest potential for polymorphism. Let n denote the number of alleles present at equilibrium. If $n = 2$, in each deme one of the homozygotes was the fittest genotype. If $n = 3$, in 92% of all cases the fittest allele in one deme was the least fit in the other deme. If $n = 4$, in about 85% the fittest allele in one deme was the least fit in the other.

3.5 Three alleles and DIDID

Consider three alleles, denoted by A_1, A_2, and A_3. We assume opposite directional selection in each deme such that the fittest allele in one deme is the least fit in the other. Thus we

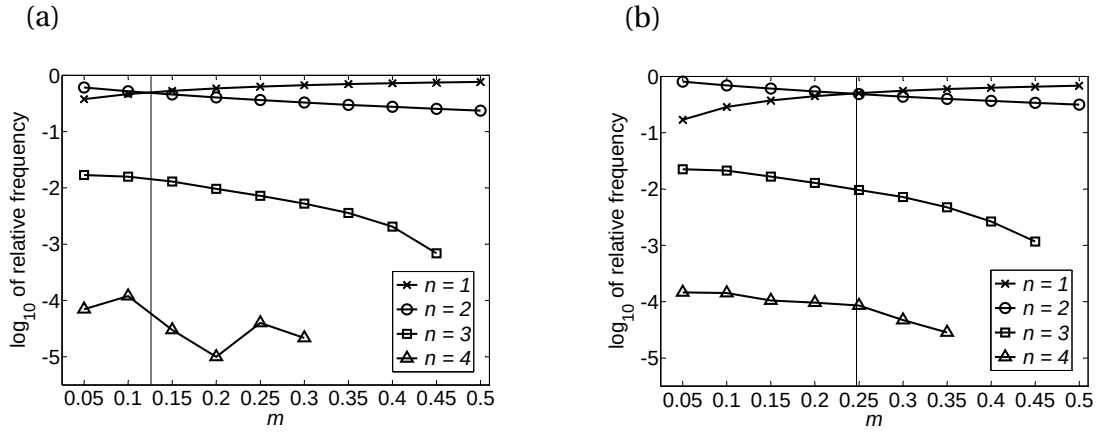

Figure 3.1: Proportions of runs that lead to maintenance of n alleles, where $n = 1, ..., 4$. In (a) the results for the random fitness set is shown and in (b) for the divergent selection set. The vertical line shows the maximum migration rate for which a polymorphic equilibrium is more likely than a monomorphic one. It is at $m \approx 0.125$ in (a) and at $m \approx 0.25$ in (b).

assign homozygous fitnesses according to the following table

$$\begin{array}{lccc} & A_1A_1 & A_2A_2 & A_3A_3 \\ \text{deme 1} & 1+b_1 & 1 & 1-a_1 \\ \text{deme 2} & 1-a_2 & 1 & 1+b_2. \end{array} \tag{3.8}$$

We posit $1 \geq a_1, a_2 > 0$, and $b_1, b_2 > 0$. Since A_2A_2 homozygotes are nowhere the fittest or the least fit individuals, we call A_2 the intermediate allele. Furthermore, for the same reasons, we sometimes refer to A_2A_2 individuals as generalists, and to A_1A_1 and A_3A_3 individuals as specialists. Heterozygotes carrying exactly one copy of each specialist allele, i.e., A_1A_3 individuals, are sometimes referred to as specialist hybrids. For a given set of dominance parameters (d_{12}, d_{13}, d_{23}), the fitnesses of heterozygotes are given by (3.5).

3.5.1 Numerical approach

Using the selection pattern (3.8) for homozygotes and assuming DIDID, we performed numerical iterations of the system (3.1) and (3.3) with randomly chosen coefficients $a_1, a_2 \in [0,1]$, $b_1, b_2 \in [0,\infty)$, randomly chosen dominance parameters $d_{12}, d_{13}, d_{23} \in [0,1]$, and randomly chosen migration rates $m_1, m_2 \in [0, \frac{1}{2}]$. In particular, for the selection coefficients we picked two uniformly distributed random vectors $x, y \in [0,1] \times [0,1] \times [0,1]$ and normalized them by dividing each entry by the entry with the intermediate value. We denote the normalized versions of x and y by $\tilde{x}$ and $\tilde{y}$. Then we set $a_1 = 1 - \min(\tilde{x})$, $b_1 = \max(\tilde{x}) - 1$ and $a_2 = 1 - \min(\tilde{y})$, $b_2 = \max(\tilde{y}) - 1$. For each set of parameters $(d_{12}, d_{13}, d_{23}, a_1, a_2, b_1, b_2, m_1, m_2)$,

we randomly picked ten initial conditions as described above. Each of the ten initial conditions was iterated until equilibration and the equilibrium allele frequencies were recorded. In total, we performed 10 runs for each of the 10^5 randomly chosen parameter sets. Figure 3.2 (a) shows the relative frequency of the different types of equilibria. Neither cycling, other complex behavior, nor slow runs were detected in our numerical analysis.

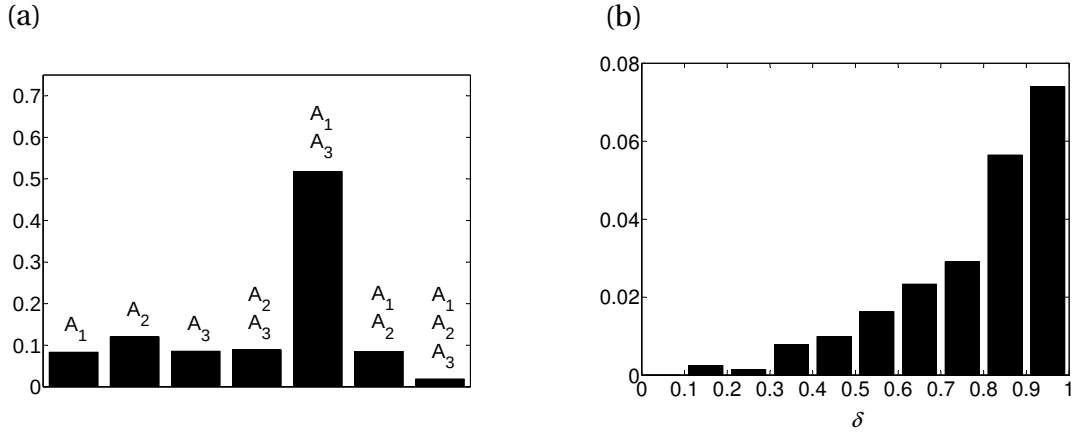

Figure 3.2: (a) Relative frequency of the different types of equilibria under the fitness scheme (3.8). The bars are labeled according to the alleles present at equilibrium. (b) Conditional frequency of parameter combinations that yield an asymptotically stable internal equilibrium as a function of δ, the average amount of dominance (3.9). Frequencies are conditioned on δ being in the indicated decimal interval to avoid bias resulting from the nonuniform distribution of δ.

3.5.2 Dominance patterns

First, we consider the role of dominance in maintaining polymorphism and introduce

$$\delta = \sqrt{\frac{(d_{12}-0.5)^2+(d_{13}-0.5)^2+(d_{23}-0.5)^2}{0.75}} \tag{3.9}$$

as a measure for its average amount. In the absence of dominance, $\delta = 0$ and if dominance is always complete, $\delta = 1$. We note that δ is not uniformly distributed if the d_{ij} are uniformly distributed. Figure 3.2 (b) shows the fraction of runs that converged to an internal equilibrium as a function of δ. Apparently, dominance patterns with large δ are much more likely to yield an internal equilibrium than dominance patterns with lower δ. Thus, dominance patterns with high degrees of dominance seem to be most prone to stable coexistence of all three alleles.

Figure 3.3 (a) shows the relative frequencies of dominance parameters among parameter combinations that yield an asymptotically stable internal equilibrium. Recall that A_i is dominant over A_j if $d_{ij} > 0.5$. If $d_{ij} = 0.5$, A_i and A_j interact additively. Apparently,

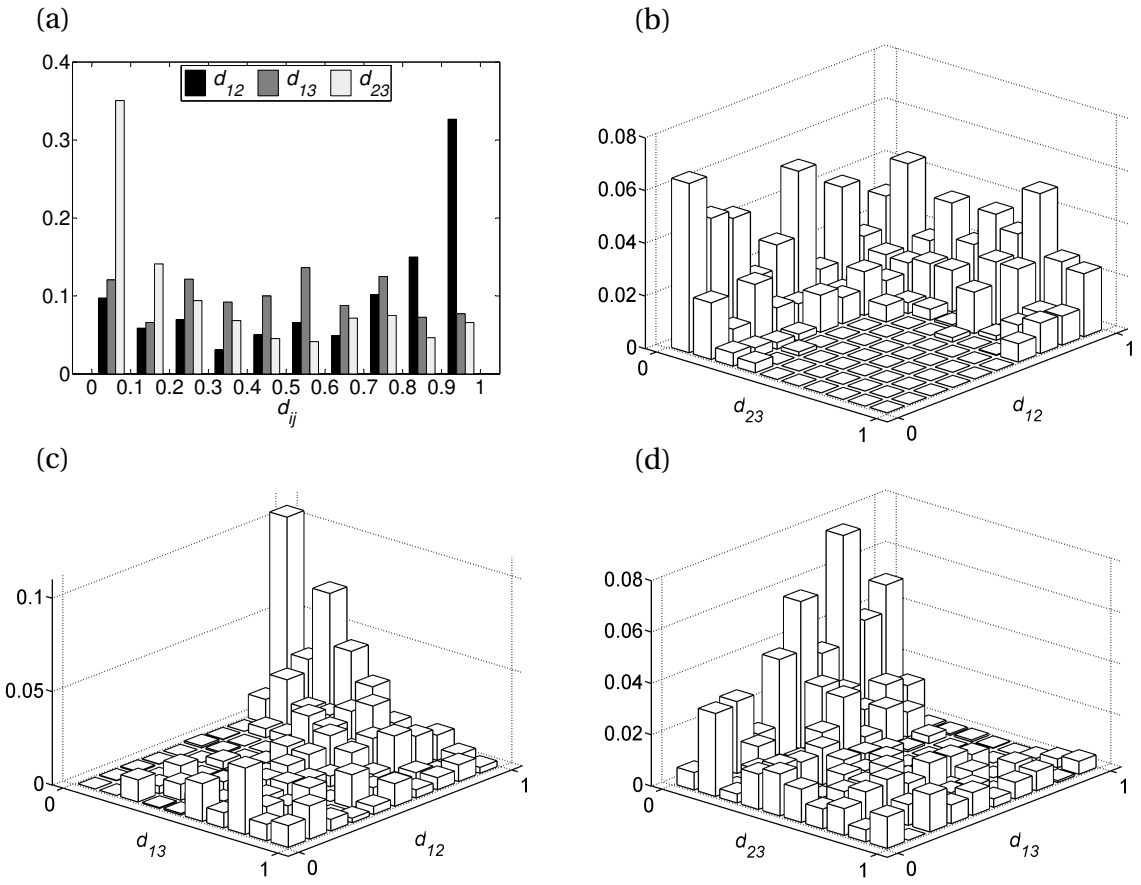

Figure 3.3: Distribution of dominance parameters. Figure (a) shows the relative frequency of single dominance parameters d_{ij} among all parameter combinations that yield an asymptotically stable internal equilibrium. Figures (b) - (d) show the relative frequency of pairwise combinations of dominance parameters among all parameter combinations that yield an asymptotically stable internal equilibrium. In all cases, histograms are shown with class width 0.1 starting at 0.

partial or complete recessivity of A_2 is favorable for the existence of stable triallelic polymorphism. In contrast, the degree of dominance between A_1 and A_3 seems to have very little effect on the likelihood of an asymptotically stable internal equilibrium. However, to identify dominance patterns prone to polymorphism, we have to consider the relative frequencies of combinations of dominance parameters. Figure 3.3 (b), (c) and (d) show the relative frequencies of pairwise combinations of dominance parameters among all parameter combinations that yield an asymptotically stable internal equilibrium. One can infer from Figure 3.3 (b) that partial recessivity of A_2 ($d_{12} > \frac{1}{2}$ or $d_{23} < \frac{1}{2}$) appears to be necessary for the existence of an asymptotically stable internal equilibrium. If A_2 is (partial) dominant over both A_1 and A_3, no stable internal equilibrium was detected. Figure 3.3 (c) and (d) are less informative.

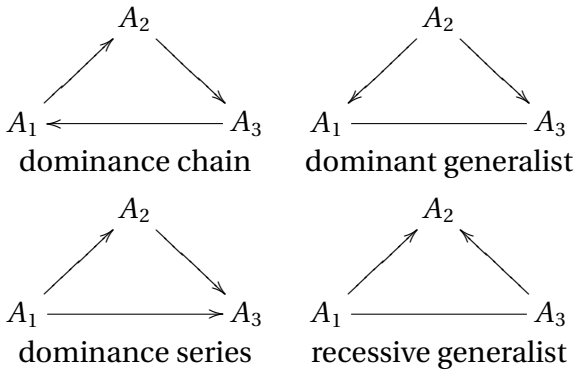

Figure 3.4: Dominance patterns. The lines connecting the alleles represent the dominance relations. A line without arrows indicates an arbitrary degree of dominance, an arrow indicates (partial) dominance of the allele at the base. The dominance series and the dominance chain also include the cases in which the direction of all arrows is reversed. Each scenario comprises one fourth of the parameter space.

To distinguish between different dominance patterns, we use the classification provided in Table 3.1 (cf. Nagylaki, 2009). In Figure 3.4, graphical representations of the dominance patterns are given for illustration. The relative frequencies of the different dominance patterns among all parameter combinations that yield an asymptotically stable internal equilibrium are also given in Table 3.1. Three of the four dominance pattern allow maintenance of all three alleles, namely recessive generalist, dominance chain, and dominance series. In addition, the fraction of globally asymptotically stable equilibria is given for the different scenarios. Noteworthy, asymptotically stable internal equilibria almost always are globally asymptotically stable.

We note that the given classification of dominance patterns is (almost) a partitioning of the set of all dominance parameter vectors (d_{12}, d_{13}, d_{23}). By almost we mean that we did not include the set for which at least one dominance parameter equals $\frac{1}{2}$, except if the intermediate allele is always recessive or dominant. However, the excluded set has measure zero and the classification is a partitioning of all observed dominance parameter vectors. Furthermore, each pattern in Table 3.1 represents one quarter of the space of all dominance patterns. We note that the scenarios recessive generalist and dominant generalist could also be considered as a dominance series. However, it appears to be convenient to study them separately in this context.

The dominance chain, in which every allele is (partially) dominant over exactly one other allele, applies in 48% of all observed cases of stable internal equilibria. This suggests that this scenario harbors the highest potential for coexistence of three alleles. The second scenario, with the intermediate allele (partially) recessive, applies in about 40% of all detected stable internal equilibria. In contrast, if the intermediate allele is always (partially) dominant, no stable triallelic equilibria were detected. The third scenario, the dominance

Pattern	Parameters	Relative frequency among dominance patterns that yield a stable internal equilibrium	Fraction of globally asymptotically stable equilibria among stable internal equilibria
dominance chain	$d_{12}, d_{23} > 0.5$ and $d_{13} < 0.5$, $d_{12}, d_{23} < 0.5$ and $d_{13} > 0.5$	48%	97%
recessive generalist	$d_{12} > 0.5$ and $d_{23} < 0.5$	40%	95%
dominance series	$d_{12}, d_{13}, d_{23} > 0.5$, $d_{12}, d_{13}, d_{23} < 0.5$	12%	79%
dominant generalist	$d_{12} < 0.5$ and $d_{23} > 0.5$	0%	-

Table 3.1: Classification of dominance patterns, the relative frequencies of the different dominance patterns among those patterns that yield an asymptotically stable internal equilibrium, and the percentage of globally asymptotically stable equilibria among all stable internal equilibria. The relative frequency of each dominance pattern among all parameter combinations is $\frac{1}{4}$.

series, applies in the remaining 12% of parameter combinations for which all three alleles were maintained at an asymptotically stable equilibrium.

3.5.3 Selection coefficients

Here, we study the distribution of selection coefficients among parameter combinations that yield an asymptotically stable internal equilibrium and try to identify necessary conditions for the existence of an asymptotically stable internal equilibrium. Figure 3.5 shows the relative frequencies of the selection coefficients a_1 and b_1 among parameter combinations that yield an asymptotically stable internal equilibrium. The distributions of the selection coefficients that yield an asymptotically stable internal equilibrium are biased towards high values of a_1 and a_2, and towards low values of b_1 and b_2. This suggests that, in order to get an asymptotically stable fully polymorphic equilibrium, it is favorable that the fitness of A_2A_2 homozygotes is closer to the least fit genotype than to the most fit in each deme, i.e., $a_1 > b_1$ and $a_2 > b_2$. However, $a_1 > b_1$ and $a_2 > b_2$ is not a necessary condition for the existence of an asymptotically stable internal equilibrium. In fact, in a few cases an asymptotically stable internal equilibrium exists if $a_1 < b_1$ and $a_2 < b_2$ (data not shown). Another interpretation of Figure 3.5 is that $a_1 > b_2$ and $a_2 > b_1$ is favorable for the existence of an asymptotically stable internal equilibrium. Again, $a_1 > b_2$ and $a_2 > b_1$ is not necessarily satisfied at an asymptotically stable internal equilibrium. In the following we elaborate both interpretations in more detail.

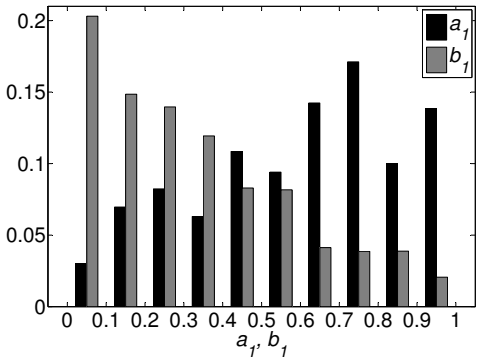

Figure 3.5: Relative frequency of selection coefficients among parameter combinations that yield an asymptotically stable internal equilibrium. The corresponding plot for a_2 and b_2 is very similar and not shown.

In the case of no dominance between A_1 and A_3, $a_i > b_i$ is equivalent to $w_{13,i} < w_{22,i}$, i.e., generalists perform better than specialist hybrids. For arbitrary d_{13}, generalists have a

higher fitness than specialist hybrids in deme 1 if and only if

$$\frac{b_2}{a_2+b_2} < d_{13}, \tag{3.10}$$

and in deme 2 if and only if

$$\frac{a_1}{a_1+b_1} > d_{13}. \tag{3.11}$$

It turns out that at least one of (3.10) or (3.11) holds in all observed cases of an asymptotically stable internal equilibrium (data not shown). Apparently, a necessary condition for the existence of an asymptotically stable triallelic equilibrium is that generalists have to be fitter than specialist hybrids in at least one deme. We note that if A_1 is completely dominant over A_3 (or vice versa), (3.10) ((3.11), respectively) holds in every selection scheme.

Next, we introduce the average fitness of genotypes over demes,

$$\bar{w}_{ij} := \sum_k c_k w_{ij,k}, \tag{3.12}$$

where

$$c_1 = \frac{m_2}{m_1+m_2} \quad \text{and} \quad c_2 = 1-c_1 \tag{3.13}$$

are the entries of the left eigenvector of M associated to the eigenvalue 1. If migration is symmetric, $c_1 = c_2 = \frac{1}{2}$ and $\bar{w}_{22} > \max(\bar{w}_{11}, \bar{w}_{33})$ is equivalent to $a_1 > b_2$ and $a_2 > b_1$. We know from Section 3.3 that for sufficiently weak migration A_2 will always get lost if selection is given by (3.8). If migration is strong relative to selection, A_2 will get fixed if and only if $\bar{w}_{22} > \max(\bar{w}_{11}, \bar{w}_{33})$. Intuitively, one would expect that A_2 can not be maintained if $\bar{w}_{22} < \min(\bar{w}_{11}, \bar{w}_{33})$. This turns out to be wrong. In fact, an asymptotically stable internal equilibrium can exist even if the generalist genotype is on average the least fit (data not shown). In Section 3.5.5 we will investigate this phenomenon in more detail. However, the majority of observed stable internal equilibria satisfied $\bar{w}_{22} > \min(\bar{w}_{11}, \bar{w}_{33})$. In particular, if the intermediate allele is recessive, $\bar{w}_{22} > \min(\bar{w}_{11}, \bar{w}_{33})$ was always satisfied at an asymptotically stable internal equilibrium. Thus, $\bar{w}_{22} > \min(\bar{w}_{11}, \bar{w}_{33})$ appears to be a necessary condition for the existence of an asymptotically stable internal equilibrium if the intermediate allele is recessive.

3.5.4 Migration rates

The distribution of migration rates among parameter combinations that yield an asymptotically stable internal equilibrium does not provide much information on the effect of

gene flow on the maintenance of polymorphism. If we restrict attention to symmetric migration, i.e., $m_1 = m_2$, and consider different dominance patterns separately, useful information can be extracted. If migration is symmetric, a small migration rate seems to increase the likelihood of an asymptotically stable internal equilibrium in the dominance chain (gray bars in Figure 3.6). We will investigate this in more detail in Section 3.5.5. In contrast, if the intermediate allele is recessive, migration rates yielding full polymorphism tend to be intermediate rather than small (black bars in Figure 3.6). We will discuss the existence of stable internal equilibria if the intermediate allele is recessive in more detail in Sections 3.5.6 and 3.6. Noteworthy, under the condition $\bar{w}_{22} < \min(\bar{w}_{11}, \bar{w}_{33})$ cases of stable triallelic equilibria exist. In all of these observed cases the dominance chain applies. In every other scenario, the average fitness of A_2A_2 homozygotes was higher than the average fitness of one (or both) of the specialists. This suggests that the dominance chain can lead to maintenance of polymorphism for a wide range of selection parameters.

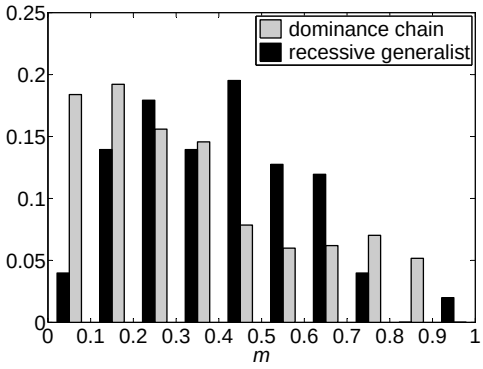

Figure 3.6: Relative frequency of the migration rate $m = m_1 = m_2$ among parameter combinations that yield an asymptotically stable internal equilibrium.

3.5.5 The symmetric dominance chain

Here we consider a special case of the dominance chain. We introduce the single (scalar) dominance parameter d and set $d_{12} = d_{23} = 1 - d_{13} = d$. Then, $d = 0.5$ yields no dominance, whereas $d = 1$ and $d = 0$ yield a complete dominance chain. In addition, we assume $m_1 = m_2 = m$. We performed numerical iterations of this system for various values of d (see Figure 3.7 (a)) and all other parameters chosen randomly as described in Section 3.5.1. Because of the symmetry of this particular dominance chain we restrict attention to $d \in [0, \frac{1}{2}]$.

Figure 3.7 (a) shows the fraction of the parameter space for which an asymptotically stable internal equilibrium exists as a function of m for various values of d. It gives inter-

esting insights into the interaction of dominance and gene flow. In general, the fraction of the runs that converge to a fully polymorphic equilibrium decreases with increasing d or increasing m. In addition, for $d \approx 0$ and m small, a surprisingly large fraction of runs converged to a completely polymorphic equilibrium. This fraction decreases very quickly as d increases. Apparently, the dominance chain harbors a very high potential for polymorphism if dominance is complete or nearly complete. The reason seems to be that each allele appears in a genotype that has high fitness in at least one deme if dominance is very high. For instance, if $d = 0$ only three different phenotypes are present and each allele appears in a genotype that is the fittest in one deme. If migration is sufficiently weak, all alleles will be maintained because each allele will spread in at least one deme if it is rare. This explains the large fraction of runs that converged to a fully polymorphic equilibrium in the case of a complete dominance chain. If migration rates are small and each allele is completely dominant over exactly one other allele, apparently a wide range of selection coefficients can lead to stable coexistence of all three alleles. In fact, if $d = 0$ and migration rates are small, in about 75% of all parameter combinations an asymptotically stable internal equilibrium exists (see Figure 3.7 (a)).

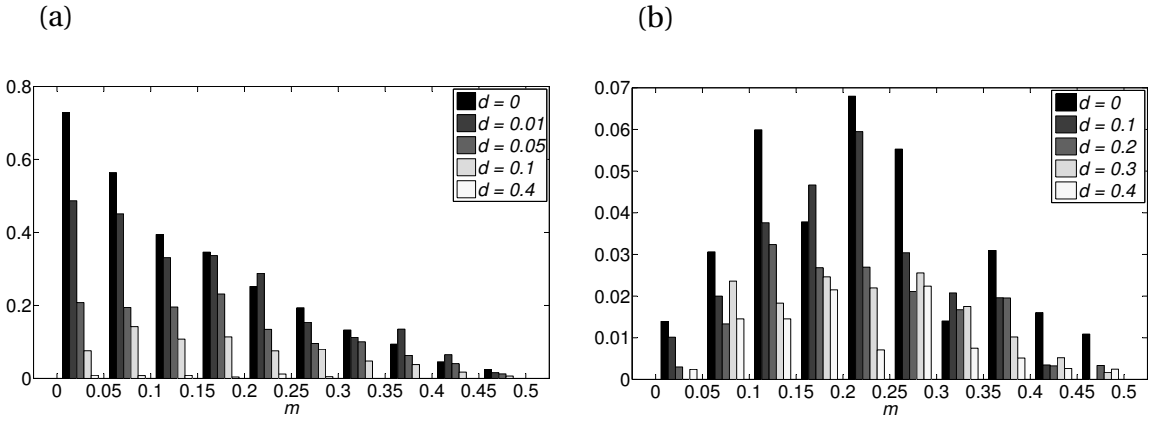

Figure 3.7: Fraction of parameter space in which an asymptotically stable internal equilibrium exists for various values of d and m. In (a) the dominance chain is shown and in (b) the recessive generalist scenario.

Among the runs that converged to an asymptotically stable internal equilibrium, $\bar{w}_{22} < \min(\bar{w}_{11}, \bar{w}_{33})$ holds in a surprisingly large fraction of runs. However, with increasing d this fraction gets smaller and if $d > 0.05$, the average fitness of A_2A_2 homozygotes was always higher then than the average fitness of at least one of the specialist homozygotes (data not shown).

3.5.6 A recessive generalist

Analogously to the dominance chain, we assume symmetric migration and introduce the single dominance parameter d. The following choice for d appears to be convenient while capturing all essential properties of the scenario: $1-d_{12}=d_{23}=d$. In addition, we assume $d_{13}=\frac{1}{2}$. Using this dominance pattern, we iterated (3.1) and (3.3) for randomly chosen parameters. The parameters were drawn as described in Section 3.5.1. First, we note that no stable triallelic polymorphism was found if A_2 is (partially) recessive, i.e., if $d\geq\frac{1}{2}$. Thus, we restrict attention to $d\in[0,\frac{1}{2}]$. Not surprisingly, the fraction of parameter combinations which lead to an asymptotically stable internal equilibrium increases with decreasing d, i.e., if A_2 becomes more recessive (see Figure 3.7 (b)).

Figure 3.7 (b) also shows that intermediate migration rates are always more favorable for the coexistence of three alleles then small or large migration rates. If gene flow is low, we expect the maintenance of two specialist alleles. For strong gene flow, the strong migration limit suggests the fixation of a single allele. If the intermediate allele is recessive, intermediate migration rates seem to be favorable for the existence of stable internal equilibria because generalist homozygotes are sufficiently well adapted to both niches, i.e., $\bar{w}_{22}>\min(\bar{w}_{11},\bar{w}_{33})$, and specialist hybrids are less fit than generalist homozygotes in at least one deme, i.e., $w_{13,i}<w_{22,i}$ for at least one $i\in\{1,2\}$.

To investigate this in more detail, we study a model of a recessive generalist that is amenable to explicit mathematical analysis.

3.6 An analytically tractable model

We assume DIDID and that selection is given by (3.8). We posit absence of dominance between A_1 and A_3, i.e., $d_{13}=\frac{1}{2}$. In this model we can show that there exists an open set of parameter values such that a asymptotically stable internal equilibrium exists. Aˆdenotes an equilibrium value. We call an equilibrium symmetric if $\hat{p}_{1,1}=\hat{p}_{3,2}$ and $\hat{p}_{3,1}=\hat{p}_{1,2}$.

3.6.1 A symmetric special case

Since the complexity of the model prohibits the direct derivation of analytical results, we first restrict attention to a special case with a large degree of symmetry. We then show that the results about the existence and stability of the polymorphic equilibrium are also valid if the symmetry assumptions are relaxed.

We consider the selection scheme

$$\begin{array}{lccc} & A_1A_1 & A_2A_2 & A_3A_3 \\ \text{deme 1} & 1+b & 1 & 1-a \\ \text{deme 2} & 1-a & 1 & 1+b, \end{array} \tag{3.14}$$

where $1 \geq a > 0$, $b > 0$, and assume that migration rates are equal, i.e., $m = m_1 = m_2$. We set $d_{12} = 1 - d_{23} = d$. Note, that $a > b$ is equivalent to $\bar{w}_{22} > \max(\bar{w}_{11}, \bar{w}_{33})$. Thus, averaged over demes, A_2A_2 is either the fittest ($a > b$) or the least fit ($a < b$) genotype. In addition, $a > b$ implies that in each deme A_2A_2 homozygotes are fitter than specialist hybrids.

Under these assumptions we are able to calculate a completely polymorphic equilibrium and the conditions for its existence. In addition it is shown that the polymorphic equilibrium exchanges its stability with a monomorphic or a dimorphic equilibrium when it enters the state space.

Before we start with a description of the equilibria, let us define two quantities that will be essential for the bifurcation analysis:

$$\mu_1 := \frac{ab}{2(a-b+ab)}, \tag{3.15}$$

$$\mu_2 := \frac{ab(1-d)}{2ab(1-d)+a-b}. \tag{3.16}$$

The following Lemma can be derived straightforwardly.

Lemma 3.6.1. *The bifurcation points μ_1 and μ_2 have the following properties:*

(i) $\mu_1, \mu_2 \in \left(0, \frac{1}{2}\right)$ *if and only if* $a > b$.

(ii) $\mu_1 > \frac{1}{2}$ *if and only if* $\frac{b}{1+b} < a < b$ *and*
$\mu_2 > \frac{1}{2}$ *if and only if* $\frac{b}{1+2b(1-d)} < a < b$.

(iii) $\mu_1 < 0$ *if and only if* $a < \frac{b}{1+b}$ *and*
$\mu_2 < 0$ *if and only if* $a < \frac{b}{1+2b(1-d)}$.

(iv) If $a > b$, $\mu_1 < \mu_2$ *holds if and only if* $d < \frac{1}{2}$.

The proofs of the theorems are given in the appendix.

3.6.1.1 Monomorphic equilibria

Theorem 3.6.2. *The monomorphic equilibria $\hat{p}^{(1)}$ and $\hat{p}^{(3)}$ are always unstable. If $d \in (0,1)$, the equilibrium $\hat{p}^{(2)}$ is asymptotically stable if $a > b$ and $m > \mu_2$. It is unstable if $a < b$ or $m < \mu_2$. If $a = b$, then $\hat{p}^{(2)}$ is unstable if $d < 1$.*

Therefore, if $0 < d < 1$ and m is sufficiently small ($m < \mu_2$), all monomorphic equilibria are unstable and at least some genetic variation will be maintained if there are no heteroclinic cycles. In particular, even if $a > b$, so that the average fitness of A_2A_2 individuals is highest, A_2 can become fixed only if the migration rate is sufficiently high ($m > \mu_2$).

3.6.1.2 Dimorphic equilibria

Now we study the boundary faces, where two alleles are present, i.e., $p_{i,1} + p_{j,1} = 1, p_{i,2} + p_{j,2} = 1, (i,j) \in \{(1,2),(2,3),(1,3)\}$. We denote the boundary face at which only alleles A_i and A_j are present by B_{ij}. Since we do not permit mutation, each of these faces is invariant. On each B_{ij} one or more equilibria may exist. However, an internal equilibrium can be calculated explicitly only if $(i,j) = (1,3)$.

We consider B_{13}. Under the assumptions of our model, a single symmetric equilibrium exists on B_{13}. We denote this equilibrium by $\hat{p}^{(13)}$. It is given in the appendix. Numerical investigations suggest that this is the only equilibrium on this manifold. Conditions for the stability of this equilibrium can not be derived analytically for the whole parameter range. However, we can determine parameter values when $\hat{p}^{(13)}$ changes its stability.

Theorem 3.6.3. *Assume $a \geq b$. The equilibrium $\hat{p}^{(13)}$ changes its stability at $m = \mu_1$. In a small neighborhood of $m = \mu_1$ it is asymptotically stable if $m < \mu_1$ and unstable if $m > \mu_1$.*

If $a < b$, $\mu_1 \notin [0, \frac{1}{2}]$. Thus, a local stability analysis in a small neighborhood of μ_1 does not yield useful information. However, if $0 < d < 1$, the weak-migration limit can be applied and yields global stability of $\hat{p}^{(13)}$. The strong-migration limit cannot be applied because A_1A_1 and A_3A_3 homozygotes have equal average fitness (3.12), which leads to non-hyperbolic equilibria.

3.6.1.3 Trimorphic equilibria

The next two results treat existence and stability of a triallelic polymorphism.

Theorem 3.6.4. *Let $a > b$ and either $0 \leq d < \frac{1}{2}$ and $I = (\mu_1, \mu_2)$ or $\frac{1}{2} < d \leq 1$ and $I = (\mu_2, \mu_1)$. If $m \in I$, there exists an internal equilibrium, denoted $\hat{p}^{(123)}$. It is given by*

$$\hat{p}_{1,1} = \hat{p}_{3,2} = \frac{a}{b\psi}, \quad \hat{p}_{2,1} = \hat{p}_{2,2} = \frac{b\psi - a - b}{b\psi} \quad \text{and} \quad \hat{p}_{3,1} = \hat{p}_{1,2} = \frac{1}{\psi}, \tag{3.17}$$

where

$$\psi = \frac{a(a+b)(2d-1)(2m-1)}{[ab(1-2m)(1-d) - m(a-b)]}. \tag{3.18}$$

This equilibrium is the only symmetric equilibrium. It does not exists if $a < b$.

Theorem 3.6.5. *Let $0 < d < 1$. If m increases from 0 to $\frac{1}{2}$, the equilibrium $\hat{p}^{(123)}$ exchanges its stability with $\hat{p}^{(13)}$ at $m = \mu_1$ and with $\hat{p}^{(2)}$ at $m = \mu_2$. If $d < \frac{1}{2}$ ($d > \frac{1}{2}$, respectively) the stability of $\hat{p}^{(123)}$ changes from unstable to asymptotically stable (asymptotically stable to unstable, respectively) when it enters the state space.*

Note that the existence of $\hat{p}^{(123)}$ requires $a > b$. This means that, averaged over demes, the fitness of A_2A_2 is highest and in each deme generalists are fitter than specialist hybrids. This nicely reflects the conditions we identified in Section 3.5. For low migration rates, specialists are advantageous because it is likely that their offspring will experience selection in the same deme. On the other hand, high migration rates favor a single generalist. However, if A_2 is recessive and $a > b$, there always exists a range of intermediate migration rates that leads to coexistence of all three alleles. This is in contrast to conventional wisdom, which states that polymorphism is usually maximized for low migration rates.

To determine the volume of the parameter space in which $\hat{p}^{(123)}$ exists, we integrate the length of the interval I over a and b. Since we are interested in an asymptotically stable polymorphism, we only consider $d \in [0, \frac{1}{2}]$ and $a > b$. Then,

$$V(d) = 4\int_0^1 \int_0^a (\mu_2 - \mu_1)\, db\, da, \tag{3.19}$$

where $V(d)$ is the volume of the parameter space in which $\hat{p}^{(123)}$ exists. The factor 4 enters because we assume $0 < b < a < 1$ and $0 < m < \frac{1}{2}$, which is one quarter of the volume of the cube $[0,1] \times [0,1] \times [0,1]$. If $d = 0$, this integral can be calculated explicitly:

$$\begin{aligned} V(0) &= 4\int_0^1 \frac{a^2[(3-8a+6a^2)\log(a) - (a-1)(2a-1+2(a-1)\log(2a^2))]}{2(1-3a+2a^2)^2}\, da \\ &= \frac{5\pi^2}{12} - 4 \approx 0.1123. \end{aligned} \tag{3.20}$$

Figure 3.8 (a) displays $V(d)$ if $0 \le d \le \frac{1}{2}$.

3.6.1.4 Structural stability

Here, we relax some of the symmetry assumptions and show that Results 3.6.4 and 3.6.5 about the existence and stability of $\hat{p}^{(123)}$ are structurally stable. We assume that selection is given by (3.8) and there is no dominance between A_1 and A_3. For convenience, we set $1 - d_{12} = d_1$ and $d_{23} = d_2$. Then, A_2 is completely recessive if $d_1 = d_2 = 0$ and completely dominant if $d_1 = d_2 = 1$.

The next results establishes the existence and local stability of an internal equilibrium in this case.

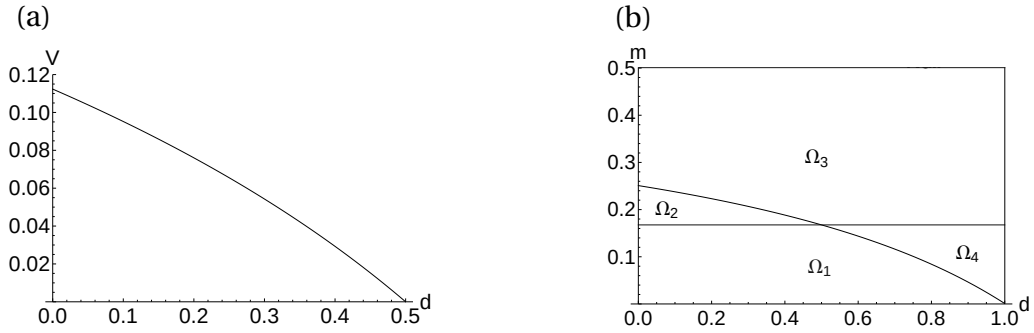

Figure 3.8: (a) The volume of the parameter space in which $\hat{p}^{(123)}$ exists as a function of d. (b) Regions of existence and stability of equilibria. In region Ω_1, $\hat{p}^{(13)}$ is globally asymptotically stable; in region Ω_3, $\hat{p}^{(2)}$ is globally asymptotically stable. The internal equilibrium $\hat{p}^{(123)}$ exists and is globally asymptotically stable in region Ω_2. In region Ω_4, $\hat{p}^{(13)}$ and $\hat{p}^{(2)}$ are both asymptotically stable, and $\hat{p}^{(123)}$ exists but is unstable. The monomorphic equilibria and $\hat{p}^{(13)}$ exist in the whole parameter space. The completely polymorphic equilibrium $\hat{p}^{(123)}$ exists only in regions Ω_2 and Ω_4. The other parameter values are $a = 0.5$ and $b = 0.25$.

Theorem 3.6.6. *Let a_2, b_2, m_2 and d_2 be fixed such that $\hat{p}^{(123)}$ exists and is asymptotically stable if $a_1 = a_2$, $b_1 = b_2$, $d_1 = d_2$, $m_1 = m_2$. Then, for (a_1, b_1, m_1, d_1) sufficiently close to (a_2, b_2, m_2, d_2), there exists an internal equilibrium close to $\hat{p}^{(123)}$. It is isolated and asymptotically stable.*

3.6.1.5 Numerical results about global stability

Let selection be given by (3.14), and assume that migration is symmetric and $d_1 = d_2$. We performed numerical iterations of the recursion relations (3.1) and (3.3) to determine global stability properties of the equilibria. Additionally, we checked whether stable equilibria other than the ones described in the above analysis exist.

First, let $a < b$. Then, in all runs all trajectories converged to $\hat{p}^{(13)}$. Therefore, we conclude:

Result 3.6.7. *Assume $0 < a < b$. Then, $\hat{p}^{(13)}$ is globally asymptotically stable.*

Now let $a > b$. No further stable equilibria than those stated above were found. If $d < \frac{1}{2}$, asymptotically stable equilibria were always globally asymptotically stable. If $m \notin I$, the same holds for $d > \frac{1}{2}$ as well. If $d > \frac{1}{2}$ and $m \in I$, every trajectory converged to either $\hat{p}^{(13)}$ or $\hat{p}^{(2)}$. Thus, for $a > b$, we can state the following results.

Result 3.6.8. *Suppose $a > b$ and $0 \leq d < \frac{1}{2}$, i.e., A_2 is (partially) recessive.*

(i) If $m < \mu_1$, then $\hat{p}^{(13)}$ is globally asymptotically stable.

(ii) If $m \in I$, $\hat{p}^{(123)}$ exists and is globally asymptotically stable.

(iii) If $m > \mu_2$, $\hat{p}^{(2)}$ is globally asymptotically stable.

Result 3.6.9. *Suppose $a > b$ and $\frac{1}{2} < d \leq 1$, i.e., A_2 is (partially) dominant.*

(i) If $m < \mu_2$, then $\hat{p}^{(13)}$ is globally asymptotically stable.

(ii) If $m \in I$, $\hat{p}^{(123)}$ exists but is unstable. Both $\hat{p}^{(13)}$ and $\hat{p}^{(2)}$ are asymptotically stable and the final outcome depends on the initial allele distribution.

(iii) If $m > \mu_1$, $\hat{p}^{(2)}$ is globally asymptotically stable.

Apparently, our local stability analysis covers all possible outcomes (see Figure 3.8 (b)). If $a > b$ and A_2 is partially or completely recessive, the migration rate determines the stability of the three possible attractors. If $d > \frac{1}{2}$ however, multiple asymptotically stable equilibria can occur. The internal equilibrium $\hat{p}^{(123)}$ exists but is unstable. If migration is intermediate, i.e., $m \in I$, the initial distribution of alleles determines whether A_2 dies out or gets fixed.

3.7 Absence of G×E interaction

We study linear selection on a quantitative trait in the absence of G×E-interaction at the trait level. Let u_i be the contribution of allele A_i to the genotypic value of an individual. Then, the genotypic value u_{ij} of an individual with genotype A_iA_j can be written as $u_{ij} = u_i d_{ij} + u_j(1-d_{ij})$. Linear selection means that there exist parameters α_k and e_k such that fitness can be written as

$$w_{ij,k} = \alpha_k u_{ij} + e_k, \tag{3.21}$$

$k \in \{1,2\}$. We assume three alleles and divergent selection. For definiteness we set $\alpha_1 < 0 < \alpha_2$. In terms of the selection scheme (3.8), it is an easy exercise to show that selection is linear if and only if

$$\frac{a_1}{a_1+b_1} = \frac{b_2}{a_2+b_2}. \tag{3.22}$$

Thus, the selection scheme (3.8) includes linear selection on a quantitative trait without G×E-interaction as a special case.

We performed numerical iterations of the system (3.1) and (3.3) with randomly drawn parameters that satisfy (3.22). More precisely, we set $x = \frac{a_1}{a_1+b_1}$ and drew it from a uniform distribution on $[0,1]$. Next, we set $y = a_1 + b_1$ and $z = a_2 + b_2$ and drew y and z independently from a uniform distribution on $[0, \frac{1}{x}]$ and $[0, \frac{1}{1-x}]$, respectively. Then,

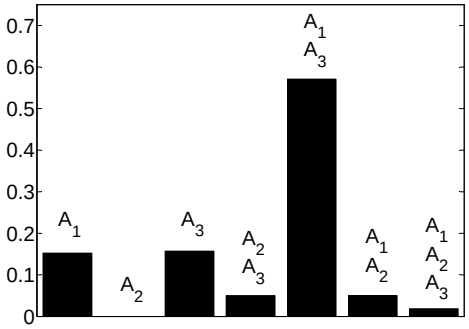

Figure 3.9: Relative frequency of the different types of equilibria if we assume linear divergent selection in the absence of G×E interaction. The bars are labeled according to the alleles present at equilibrium.

$a_1 = xy, b_1 = (1-x)y, a_2 = (1-x)z, b_2 = xz$. All other parameters were drawn as described in Section 3.5.1.

In general, the influence of the parameters on the maintenance of all three alleles is very similar to the more general case in which selection is given by (3.8). Therefore, we only discuss situations in which the behavior is significantly different and highlight interesting phenomena. Figure 3.9 shows the relative frequency of the different types of equilibria. Interestingly, the intermediate allele A_2 never got fixed. Since fixation of A_2 seems most likely under strong migration, we use the strong-migration limit to give an explanation. The average fitnesses (3.12) of homozygotes are:

$$\bar{w}_{11} = 1 + \frac{m_2 b_1 - m_1 a_2}{m_1 + m_2}, \tag{3.23}$$

$$\bar{w}_{22} = 1, \tag{3.24}$$

$$\bar{w}_{33} = 1 + \frac{m_1 a_2 - m_2 b_1}{m_1 + m_2}. \tag{3.25}$$

In the strong-migration limit, the allele with highest homozygous fitness becomes fixed. Thus, the monomorphic equilibrium $\hat{p}^{(2)}$ is globally asymptotically stable if and only if $\bar{w}_{22} > \max(\bar{w}_{11}, \bar{w}_{33})$. Using (3.22), it follows that $\bar{w}_{11} < \bar{w}_{22}$ if and only if $\frac{b_1}{a_2} < \frac{m_1}{m_2}$, and $\bar{w}_{33} < \bar{w}_{22}$ if and only if $\frac{b_1}{a_2} > \frac{m_1}{m_2}$. Thus, generically, either $\bar{w}_{11} < \bar{w}_{22} < \bar{w}_{33}$ or $\bar{w}_{11} > \bar{w}_{22} > \bar{w}_{33}$ holds. Consequently, A_2 cannot become fixed under strong migration.

In the symmetric recessive generalist scenario, described in Section 3.6.1, (3.22) reduces to $a = b$. In this case, the strong-migration limit cannot be applied. However, Theorem 3.6.2 confirms that $\hat{p}^{(2)}$ is always unstable. Furthermore, the internal equilibrium $\hat{p}^{(123)}$ does not exist if $a = b$.

This suggests that the recessive generalist scenario harbors less potential for the maintenance of triallelic polymorphism if selection is linear and G×E interaction is absent. In fact, the relative frequency of internal equilibria in which the generalist is recessive decreases compared to the selection scheme (3.8). If selection is linear and G×E interaction is absent, the dominance chain applies in about 63% of all stable internal equilibria, the recessive generalist scenario applies in about 19% of all stable internal equilibria, and the dominance series in the remaining 18%.

3.8 Discussion

We studied the potential of maintaining multiallelic polymorphism at a single locus in a discrete-time two-deme model with arbitrary migration and deme-independent degree of intermediate dominance (DIDID). The biological important assumption on which this study is based is the assumption of DIDID (cf. Nagylaki, 2009). DIDID includes the case of no G×E interaction as an important special case. Our main focus was on the existence of stable three-allele polymorphism.

Multiple alleles in two demes are briefly studied in Section 3.4. We showed numerically that up to four alleles can be maintained at an asymptotically stable internal equilibrium. No cases were found in which more than four alleles coexisted. This does, however, not necessarily mean that four is the maximum number of alleles that can be maintained. Equilibria with four alleles segregating were found only very rarely, in a proportion of about 10^{-5} of all runs. Thus, if five (or more) alleles are able to coexist, it seems likely that one has to perform a huge number of runs to detect them. The parameter range in which polymorphic equilibria exist seems to shrink dramatically with increasing number of alleles present at equilibrium (see Figure 3.1).

From Section 3.5 on, we restrict attention to three alleles and opposite directional selection in the two demes, such that the fittest allele in one deme is the least fit in the other deme. We investigated how the dominance pattern, the selection intensities, and the migration rates determine the maintenance of triallelic polymorphism. As a rule of thumb we can say that larger degrees of dominance are more favorable for three-allele coexistence than dominance patterns with lower degrees of dominance (see Figure 3.2). In addition, we observed that partial recessivity of the intermediate allele is crucial for the existence of an asymptotically stable internal equilibrium (see Figure 3.3 (b)). If the intermediate allele is partially dominant, stable coexistence of all three alleles was never found. In all other considered dominance patterns (see Table 3.1), stable internal equilibria were found. The two most frequent patterns among all parameter combinations that yield an asymptotically stable internal equilibrium, namely a recessive generalist and the dominance chain, were studied in more detail. Noteworthy, the complete dominance chain, in which every

allele is completely dominant over one other allele, harbors a surprisingly high potential of maintaining all three alleles (see Figure 3.7 (a)).

In general, small migration rates lead to a higher likelihood of maintaining polymorphism (see Figure 3.1). However, the fraction of the parameter space in which all three alleles coexist is not for every dominance pattern maximized at small migration rates (see Figure 3.6 (b) and 3.7). Actually, only in the dominance chain with almost complete dominance, small migration rates are optimal for maintaining all three alleles. Then, three alleles can be maintained in up to 75% of the remaining parameter space. Under the other dominance scenarios, intermediate migration rates are more favorable for coexistence of three alleles.

Furthermore, we investigated the fitness patterns that lead to a stable internal equilibrium. Apparently, two conditions seem to play an important role. First, generalists have to be fitter than specialist hybrids in at least one deme. This condition was satisfied in all detected parameter combinations with an asymptotically stable internal equilibrium. It is independent of the migration rate. The second condition, $\bar{w}_{22} > \min(\bar{w}_{11}, \bar{w}_{33})$, compares the average fitness of homozygous individuals. Although, this condition is not necessary for the existence of an asymptotically stable internal equilibrium, it was satisfied if dominance is not close to complete or if the intermediate allele is recessive. Especially in the case of a complete dominance chain, stable internal equilibria were detected although $\bar{w}_{22} < \min(\bar{w}_{11}, \bar{w}_{33})$.

These results suggest that two different selection regimes permit coexistence of three alleles. The first regime, the dominance chain with (almost) complete dominance, harbors the highest potential for stable triallelic equilibria. For instance, in a complete dominance chain, only three different phenotypes exist and every allele appears in a genotype that is the fittest in one of the two demes. If gene flow between the two demes is sufficiently low, three alleles can coexist at an asymptotically stable internal equilibrium. In the second regime, it is helpful to interpret our results in a specialist-generalist context. The second regime applies if generalists are fitter than specialist hybrids in at least one deme and $\bar{w}_{22} > \min(\bar{w}_{11}, \bar{w}_{33})$. For low migration rates, specialists are advantageous and A_2 is lost. For strong gene flow, a single allele gets fixed. However, intermediate migration rates lead to maintenance of all three alleles, i.e., coexistence of two specialists and a single generalist.

The latter scenario is nicely illustrated by the analytical example in Section 3.6. We proved the existence of an internal equilibrium and performed a bifurcation analysis. The migration rate serves as a bifurcation parameter. If the two above-mentioned conditions on selection parameters hold and m increases, the internal equilibrium (one generalist and two specialists present) bifurcates into existence and exchanges stability with the diallelic boundary equilibrium at which two specialists are present. If m is further increased, the internal equilibrium leaves the state space and exchanges stability with the monomorphic

equilibrium where one generalist is present. The degree of dominance is crucial in this example, it determines stability of the internal equilibrium (see Figure 3.8). If the intermediate allele is recessive, the internal equilibrium is asymptotically stable. It is unstable if the intermediate allele is dominant. This seems to be intuitive. If A_2 is recessive, it is (partially) locally adapted when heterozygous, thus increasing the chance of coexistence.

In general, global asymptotic stability of the equilibria could only be determined via numerical iterations. An analytical proof of the global coexistence of all three alleles would be desirable. For instance, a permanence argument could validate our intuitive explanations. In addition, an analytical example for an internal equilibrium in the dominance chain is missing in our analysis. Finding such an example would be desirable for a more complete understanding of stable three-allele coexistence in two demes.

Finally, we briefly studied linear selection on a quantitative trait without G×E interaction. If we assume divergent selection, this is special case of the selection scheme (3.8). We numerically showed that stable internal equilibria exist in the absence of G×E interaction. Thus, we can conclude that G×E interaction is not necessary for the maintenance of three alleles in two demes. Furthermore, we never observed fixation of the generalist allele A_2. Under divergent selection, fixation of A_2 is impossible if migration is weak and seems most likely if migration is strong. In the absence of G×E-interaction, the strong-migration limit always yields fixation of either A_1 or A_3. This explains why we never observed fixation of A_2. However, there exist situations in which a generalist can coexist with one or both of the specialists.

In the following, we relate our work to previous results. Nagylaki and Lou (2007) considered a model that is similar to ours. They assume multiple alleles at a single locus and intermediate dominance. Their work treats several important limiting cases (weak migration, strong migration, weak migration and weak selection). Theorem 3.5 in Nagylaki and Lou (2007) concerns the loss of a specified allele if both selection and migration are weak. In particular, they showed that in the slow evolution approximation specialists always outcompete generalists in the absence of dominance. This also holds if migration is weak relative to selection and dominance is intermediate but otherwise general (cf. Section 3.3.2.1 in this work). In contrast, in the limit of strong migration an arbitrary number of alleles can be maintained. However, deme-dependent dominance is necessary for the construction of such examples. If DIDID is assumed, the strong-migration limit yields fixation of a single allele. Our results show that the weak- and strong-migration limits do not cover all possible equilibrium structures. For instance, intermediate migration rates may yield an internal equilibrium when neither weak nor strong migration does.

Nagylaki (2009) focuses on multiallelic equilibria in two demes with intermediate dominance. In particular, he investigates whether (and if so, to which extent) the results for no

dominance can be generalized to intermediate dominance or DIDID. He shows that in the Levene model, DIDID and no dominance generically lead to qualitatively equivalent dynamics. If dominance is intermediate but deme dependent, this generalization fails in the Levene model and also if there is arbitrary migration. Our results confirm this. In particular, our work complements that of Nagylaki and Lou (2007) and Nagylaki (2009), and can be seen as an attempt to fill the gap between the different limiting cases.

Nagylaki and Lou (2007) and Nagylaki (2009) demonstrate that deme-dependent dominance, which means that there is some form of G×E interaction, can be a mechanism that leads to the maintenance of high levels of genetic variation. Our work shows that, even in the absence of G×E interaction, certain dominance patterns can lead to stable coexistence of more alleles than there are demes. A question that naturally arises is how much variation can be maintained if the underlying genetic architecture is able to evolve.

Otto and Bourguet (1999) studied the evolution of dominance in two diallelic demes. Their results show that selection favors higher levels of dominance if this reduces the migration load. In particular, locally beneficial alleles tend to become more dominant. To minimize migration load, the locally advantageous alleles should evolve to be completely dominant in each deme. In fact, deme-dependent dominance due to G×E interaction can be found in nature. For instance, Bourguet et al. (1996) showed that in plant species that are exposed to pesticides, dominance values can depend on genetic background and environmental conditions (see also Bourguet, 1999). Thus, in principle, a flexible genetic architecture could act as a catalyzer for the maintenance of high levels of genetic variation in a spatially structured environment.

Star et al. (2007b, 2008) considered evolvable fitnesses in a diploid two-deme model with arbitrary (symmetric) migration and showed that high levels of polymorphism can be maintained. In fact, more alleles were maintained than with a fixed genetic architecture (Star et al., 2007a). Mutational steps had arbitrary effects in their study, and over- and underdominance was allowed as well. Thus, comparison of their results with ours is difficult.

Summarizing, we provided a systematic study of the effect of deme-independent dominance on the maintenance of genetic variation in a simple two-deme model. Our study reveals interesting phenomena and raises several new questions. Since we analyzed a rather simple model, further investigations are necessary to gain a better understanding of the role of a spatially heterogeneous environment as a factor maintaining genetic variation. This will require the development of new analytical tools (e.g., the generalization of the concept of protected polymorphism to more than two alleles), and further numerical investigations (e.g., about the consequences of the evolution of the genetic architecture on the maintenance of genetic variation).

Acknowledgements: This work was funded by a grant from the Vienna Science and Tech-

nology Fund (WWTF) to R. Bürger and by grant P21305-N13 of the Austrian Science Foundation (FWF) to R. Bürger. The author is grateful to Reinhard Bürger for many discussions, fruitful comments, and assistance during the preparation of the manuscript. I gratefully acknowledge the helpful comments and suggestions on an earlier draft made by Ada Akerman and Claudia Bank.

Appendix

A Proofs

We do not give complete algebraic proofs here, which would be very lengthy. However, all the formulas given can be easily checked with *Mathematica* or similar software.

Proof of Theorem 3.6.2. First we consider the equilibrium $\hat{p}^{(1)}$ defined by $p_{1,1} = p_{1,2} = 1$. Due to our symmetry assumptions, the treatment of the equilibrium $\hat{p}^{(3)}$ is analogous. The eigenvalues are readily determined and of rather simple form. However, for our purpose it is sufficient to consider just one of them,

$$\lambda = \frac{(1-2m)(2-a+b)}{(1-m)(2-a+b) - \sqrt{(a+b)^2(1-2m)+(2-a+b)^2 m^2}}. \tag{3.26}$$

As can be checked easily, $\lambda > 1$ if and only if $0 < (a+b)^2(1-2m)$, because $a, b > 0$. Because $m < \frac{1}{2}$, the monomorphic equilibria $\hat{p}^{(1)}$ and $\hat{p}^{(3)}$ are always unstable.

Now, we consider local stability of $\hat{p}^{(2)}$. Due to our symmetry assumptions the characteristic polynomial of the Jacobian has the form

$$P(x) \;=\; Q(x)^2 \tag{3.27}$$

where

$$\begin{aligned} Q(x) \;&=\; x^2 - (1-m)\big[2+(b-a)(1-d)\big]x \\ &\quad +(1-2m)\big[1-a(1-d)\big]\big[1+b(1-d)\big]. \end{aligned} \tag{3.28}$$

Hence we have two distinct eigenvalues, each with multiplicity two. They are given by

$$\begin{aligned} \lambda_1 \;=\; \lambda_2 = \frac{1}{2}\Big[&[2-(a-b)(1-d)](1-m) \\ &\pm\sqrt{(a+b)^2(1-d)^2(1-2m)+[2-(a-b)(1-d)]^2 m^2}\Big]. \end{aligned} \tag{3.29}$$

Because $m < \frac{1}{2}$, the term under the square root is always positive and both eigenvalues are real. Obviously, Q is convex. In addition,

$$Q(0) = (1-2m)\big[1-a(1-d)\big]\big[1+b(1-d)\big] > 0 \tag{3.30}$$

and

$$Q'(0) = -(1-m)\big[2+(b-a)(1-d)\big] < 0. \tag{3.31}$$

Thus, both eigenvalues are positive and we only have to consider the larger of the two eigenvalues,

$$\begin{aligned}\lambda_1 &= \frac{1}{2}\Big[[2-(a-b)(1-d)](1-m) \\ &\quad +\sqrt{(a+b)^2(1-d)^2(1-2m)+[2-(a-b)(1-d)]^2m^2}\Big].\end{aligned} \tag{3.32}$$

Straightforward calculations yield that $\hat{p}^{(2)}$ is asymptotically stable if $a > b$ and $m > \mu_2$, and $\hat{p}^{(2)}$ is unstable if $a < b$ or $m < \mu_2$. If $a = b$,

$$\lambda_1 = 1 - m + \sqrt{m^2 + a^2(1-d)^2(1-2m)} \tag{3.33}$$

and $\hat{p}^{(2)}$ is unstable if $d < 1$. □

Proof of Theorem 3.6.3. The equilibrium $\hat{p}^{(13)}$ is given by

$$\hat{p}^{(13)}_{1,1} = \frac{a+b-4\,m(1-a)+\sqrt{(a+b)^2+16\,(1-a)\,(1+b)\,m^2}}{2\,(a+b)\,(1+2\,m)}, \tag{3.34}$$

$$\hat{p}^{(13)}_{2,1} = 0, \quad \hat{p}^{(13)}_{3,1} = 1-\hat{p}^{(13)}_{1,1}, \quad \hat{p}^{(13)}_{1,2} = \hat{p}^{(13)}_{3,1}, \quad \hat{p}^{(13)}_{2,2} = 0, \quad \hat{p}^{(13)}_{3,2} = \hat{p}^{(13)}_{1,1}. \tag{3.35}$$

We set $m = \mu_1 + \epsilon$ and neglect second- and higher-order terms in ϵ. If $\epsilon = 0$, the eigenvalues are

$$\lambda_1 = 1, \tag{3.36}$$

$$\lambda_2 = \frac{1}{2}(2+b-a-ab), \tag{3.37}$$

$$\lambda_3 = \frac{a-b}{a+ab-b}, \tag{3.38}$$

$$\lambda_4 = \frac{(a-b)(2+b-a-ab)}{2(a+ab-b)}. \tag{3.39}$$

Since we assume $a \geq b$, all eigenvalues are non-negative and λ_1 is the leading one. If $|\epsilon| > 0$, we get the following expression for the leading eigenvalue

$$\lambda_1 = 1 + \epsilon\frac{2(a-b+ab)^2 d}{a^2+b^2+ab(a-b)} + O(\epsilon^2). \tag{3.40}$$

Since the fraction in the above expression is always positive, the result follows immediately.

□

Proof of Theorem 3.6.4. It can be straightforwardly checked that $p'_{i,k} = p_{i,k}$ holds at $\hat{p}^{(123)}$ and that $\hat{p}^{(123)}$ is the only symmetric internal equilibrium. In the following, we determine the parameter range for which $\hat{p}^{(123)}$ is feasible. It follows immediately from (3.17) that the allele frequencies are positive and smaller than 1 if and only if $\psi > \frac{a+b}{b}$.

Let us consider the migration rate as a function of ψ:

$$m(\psi) = \frac{a(a+b)(2d-1) - 2ab(d-1)\psi}{2a(a+b)(2d-1) + 2\big[2ab(1-d) + a - b\big]\psi}. \tag{3.41}$$

We note that m has a discontinuity at

$$\psi_c = \frac{a(a+b)(1-2d)}{2ab(1-d) + a - b}. \tag{3.42}$$

It can easily be checked that $\psi_c > \frac{a+b}{b}$ if and only if

$$\min\left(\frac{b}{1+b}, \frac{b}{1+2b(1-d)}\right) < a < \max\left(\frac{b}{1+b}, \frac{b}{1+2b(1-d)}\right). \tag{3.43}$$

In particular, m is continuous in ψ on $(\frac{a+b}{b}, \infty)$ if $a > b$.

The derivative of m with respect to ψ is given by

$$\frac{dm}{d\psi} = \frac{a(a^2-b^2)(1-2d)}{2\big[a(a+b)(2d-1) + (2ab(1-d) + a - b)\psi\big]^2}. \tag{3.44}$$

If $a > b$, m is monotonically increasing in ψ if $d < \frac{1}{2}$ and monotonically decreasing in ψ if $d > \frac{1}{2}$, and vice versa if $a < b$.

Let $a > b$ and $d < \frac{1}{2}$. We derive the range of migration rates for which $\psi > \frac{a+b}{b}$. Because m is monotonically increasing in ψ, the minimal migration rate for which $\hat{p}^{(123)}$ exists is

$$\mu_1 = m\left(\frac{a+b}{b}\right) = \frac{ab}{2(a-b+ab)}. \tag{3.45}$$

The maximum migration rate for which the equilibrium exists is

$$\mu_2 = \lim_{\psi\to\infty} m(\psi) = \frac{ab(1-d)}{2ab(1-d) + a - b}. \tag{3.46}$$

It follows immediately that $\mu_1 < \mu_2$. We know from Lemma 3.6.1 that $0 < \mu_1$ and $\mu_2 < \frac{1}{2}$. This proves the existence of the equilibrium if $a > b$ and $m \in I = [\mu_1, \mu_2] \subset \left(0, \frac{1}{2}\right)$. For the case $d > \frac{1}{2}$, analogous arguments lead to the corresponding result.

Next, we show that $\hat{p}^{(123)}$ does not exist if $a \leq b$. If $a = b$,

$$\psi = \frac{1-2d}{1-d} < \frac{a+b}{b} = 2. \tag{3.47}$$

Thus, the equilibrium does not exist if $a = b$.

Now, let $a < b$. We show that $\hat{p}^{(123)}$ does not exist if $d < \frac{1}{2}$. The case $d > \frac{1}{2}$ can be treated analogously. If $d < \frac{1}{2}$, m is monotonically decreasing in ψ.

We have to consider three cases. First, let $a > \frac{b}{1+b}$. Then, m is continuous in ψ on $\left(\frac{a+b}{b}, \infty\right)$ and μ_2 is the minimal migration rate such that $\hat{p}^{(123)}$ exists. Because $\frac{b}{1+2b(1-d)} < \frac{b}{1+b}$, Lemma 3.6.1 shows that $\mu_2 > \frac{1}{2}$. Thus, $\hat{p}^{(123)}$ does not exist because $m < \frac{1}{2}$.

Next, let $\frac{b}{1+2b(1-d)} < a < \frac{b}{1+b}$. Then, m has a discontinuity at $\psi_c > \frac{a+b}{b}$ and

$$\lim_{\psi \to \psi_c^-} m(\psi) = -\infty, \tag{3.48}$$

$$\lim_{\psi \to \psi_c^+} m(\psi) = +\infty. \tag{3.49}$$

Thus, $\hat{p}^{(123)}$ exists if and only if $m < \mu_1$ or $m > \mu_2$. Lemma 3.6.1 tells us that $\mu_1 < 0$ and $\mu_2 > \frac{1}{2}$. Consequently, $\hat{p}^{(123)}$ does not exist because $0 < m < \frac{1}{2}$.

Finally, let $a < \frac{b}{1+2b(1-d)}$. Then, m is continuous in the considered parameter range and $\hat{p}^{(123)}$ exists if and only if $\mu_2 < m < \mu_1$. According to Lemma 3.6.1, μ_1 and μ_2 are negative if $a < \frac{b}{1+2b(1-d)}$. Thus, $\hat{p}^{(123)}$ does not exist and the proof is complete. □

Proof of Theorem 3.6.5. We know already that $\hat{p}^{(2)}$ and $\hat{p}^{(13)}$ change stability at μ_2 and μ_1, respectively. Furthermore, $\hat{p}^{(13)} = \hat{p}^{(123)}$ if $m = \mu_1$, and $\hat{p}^{(2)} = \hat{p}^{(123)}$ if $m = \mu_2$. It is left to show that $\hat{p}^{(123)}$ changes stability when it passes through $\hat{p}^{(2)}$ or $\hat{p}^{(13)}$. To this aim we consider the leading eigenvalues of the Jacobian evaluated at $\hat{p}^{(123)}$ and set $m = \mu_1 + \epsilon$ or $m = \mu_2 + \epsilon$. First, we let $m = \mu_1 + \epsilon$. If $\epsilon = 0$, the eigenvalues are

$$\lambda_1 = 1, \tag{3.50}$$

$$\lambda_2 = 1 - \frac{1}{2}(a + ab - b), \tag{3.51}$$

$$\lambda_3 = 1 - \frac{ab}{a - b + ab}, \tag{3.52}$$

$$\lambda_4 = 1 - \frac{a^2(1+b) + b^2(1-a)}{2(a - b + ab)}, \tag{3.53}$$

and λ_1 is the leading one. For $\epsilon \neq 0$, $|\epsilon|$ small, we get

$$\lambda = 1 - \epsilon \frac{2[a - (1-a)b]^2}{b^2(1-a) + a^2(1+b)} + O(\epsilon^2). \tag{3.54}$$

The fraction in (3.54) is always positive. If $d < \frac{1}{2}$, $\hat{p}^{(123)}$ exists if $\epsilon > 0$. If $d > \frac{1}{2}$, $\hat{p}^{(123)}$ exists if

$\epsilon < 0$. Thus, the first part of the theorem is proved.

Now we let $m = \mu_2 + \epsilon$. If $d < \frac{1}{2}$, $\hat{p}^{(123)}$ exists if $\epsilon < 0$. If $d > \frac{1}{2}$, $\hat{p}^{(123)}$ exists if $\epsilon > 0$. For $\epsilon = 0$ the characteristic polynomial is given by

$$\begin{aligned} P(x) &= (x-1)^2\{(b-a)\big[a(d-1)+1\big]\big[b(d-1)-1\big] \\ &\quad -\big[2ab(1-d)+a-b\big]x\}^2. \end{aligned} \tag{3.55}$$

The eigenvalues are

$$\lambda_1 = \lambda_2 = \quad 1 \quad \text{and} \tag{3.56}$$

$$\lambda_3 = \lambda_4 = \quad 1-(1-d)\frac{a^2+ab(1-d)(a-b)+b^2}{2ab(1-d)+a-b}. \tag{3.57}$$

Because $a > b$, $\lambda_3, \lambda_4 < 1$. For $\epsilon \neq 0$, $|\epsilon|$ small, we get

$$\lambda_1 = 1+\epsilon\frac{a(a^2-b^2)(2d-1)}{2\big[b^2[1-a(1-d)]+a^2[1+b(1-d)]\big]}+O(\epsilon^2), \tag{3.58}$$

$$\lambda_2 = 1+\epsilon\frac{(a^3-ab^2)(7-10d)+8a^2b(a+b)(1-d)^2}{2\big[b^2[1-a(1-d)]+a^2[1+b(1-d)]\big]}+O\epsilon^2. \tag{3.59}$$

Since the denominator in λ_1 is positive, $\lambda_1 < 1$ if $d < \frac{1}{2}$ and $\epsilon < 0$, and $\lambda_1 > 1$ if $d > \frac{1}{2}$ and $\epsilon > 0$. Thus, we know that $\hat{p}^{(123)}$ is unstable if $d > \frac{1}{2}$. It is left to show that $\lambda_2 < 1$ if $d < \frac{1}{2}$. Simple calculations yield that $\lambda_2 < 1$ if and only if

$$(7-10d)(a-b)+8ab(1-d)^2 > 0. \tag{3.60}$$

Consequently, because $d < \frac{1}{2}$ and $a > b$, $\lambda_2 < 1$. □

Proof of Theorem 3.6.6. Let a_2, b_2, m_2, and d_2 be fixed such that $\hat{p}^{(123)}$ exists. We set $\Delta p_{i,k} = p'_{i,k} - p_{i,k}$, $i,k \in \{1,2\}$ and define the map

$$F : \mathbb{R}^8 \to \mathbb{R}^4 \tag{3.61}$$

by setting its components as follows:

$$F_1(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a_1, b_1, m_1, d_1) = \Delta p_{1,1}, \tag{3.62}$$

$$F_2(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a_1, b_1, m_1, d_1) = \Delta p_{1,2}, \tag{3.63}$$

$$F_3(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a_1, b_1, m_1, d_1) = \Delta p_{2,1}, \tag{3.64}$$

$$F_4(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a_1, b_1, m_1, d_1) = \Delta p_{2,2}. \tag{3.65}$$

We denote the derivative of F with respect to (a_1, b_1, m_1, d_1) evaluated at $(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a, b, m, d)$

by $DF(p_{1,1}, p_{2,1}, p_{1,2}, p_{2,2}, a, b, m, d)$. Theorem 3.6.4 tells us that

$$F(\hat{p}_{11}^{(123)}, \hat{p}_{21}^{(123)}, \hat{p}_{12}^{(123)}, \hat{p}_{22}^{(123)}, a_2, b_2, m_2, d_2) = 0. \tag{3.66}$$

We seek a map

$$f : (a_1, b_1, m_1, d_1) \mapsto (p_{11}, p_{21}, p_{12}, p_{22}), \tag{3.67}$$

such that

$$F(f(a_1, b_1, m_1, d_1), a_1, b_1, m_1, d_1) = 0. \tag{3.68}$$

By the Implicit Function Theorem such a map exists on a small neighborhood of $(a_1, b_1, m_1, d_1) = (a_2, b_2, m_2, d_2)$ if and only if

$$DF(\hat{p}_{11}^{(123)}, \hat{p}_{21}^{(123)}, \hat{p}_{12}^{(123)}, \hat{p}_{22}^{(123)}, a_2, b_2, m_2, d_2) \tag{3.69}$$

is invertible. This is the case if and only if

$$\det(DF(\hat{p}_{11}^{(123)}, \hat{p}_{21}^{(123)}, \hat{p}_{12}^{(123)}, \hat{p}_{22}^{(123)}, a_2, b_2, m_2, d_2)) \neq 0. \tag{3.70}$$

Using *Mathematica*, this determinant calculates to

$$\frac{16(a-b)^2 m^2 \phi_1^2 \phi_2^4 \phi_3 \phi_4}{a^7 b^7 (a+b)^4 (2d-1)^7 (2m-1)^9}, \tag{3.71}$$

where

$$\begin{aligned}
\phi_1 &= ab(1-2m) + 2m(a-b), && (3.72)\\
\phi_2 &= ab(1-d)(1-2m) - m(a-b), && (3.73)\\
\phi_3 &= ab(1-d)(1-2m) - 2m(a-b), && (3.74)\\
\phi_4 &= ab(3-2d) - 4m(a-b) - abm(6-4d). && (3.75)
\end{aligned}$$

The zeros of ϕ_1 and ϕ_2 as a function of m coincide with the boundaries of the region where $\hat{p}^{(123)}$ is feasible. Simple calculations yield that $\hat{p}^{(123)}$ is not feasible at the zeros of ϕ_3 and ϕ_4. In particular, if $a > b$, $m > \frac{1}{2}$ at a zero of ϕ_3 or ϕ_4. Thus, the desired map f exists on a small neighborhood of $(a_1, b_1, m_1, d_1) = (a_2, b_2, m_2, d_2)$. □

Curriculum Vitae

Date of birth: May 2, 1981
Place of birth: Guessing, Austria
Citizenship: Austrian

EDUCATION

Jan. 2007 - Feb. 2010	Ph.D. studies at the Department of Mathematics (Univ. of Vienna), advised by Prof. Reinhard Bürger.
Dec. 2006	Master exam with distinction in mathematics at the University of Vienna.
Oct. 2001 - Dec. 2006	Studies of mathematics at the University of Vienna.

ACADEMIC CAREER

Since Jan. 2009	Research Assistant at the Department of Mathematics (University of Vienna), fully supported by the Austrian Science Foundation (FWF project P21305-N13) to R. Bürger.
Jan. 2007 - Dec. 2008	Research assistant at the Department of Mathematics (University of Vienna), fully supported by a grant of the Vienna Science and Technology Fund (WWTF) to R. Bürger.

List of Publications

Articles

i. Stephan Peischl (2010). Dominance and the maintenance of polymorphism in multiallelic migration-selection models with two demes. Submitted to Theoretical Population Biology.

ii. Stephan Peischl and Kristan A. Schneider (2009). The Evolution of Dominance in Assortatively Mating Populations. Evolution 64: 561 - 582.

iii. Stephan Peischl and Reinhard Bürger (2008). Evolution of dominance under frequency-dependent intraspecific competition. J. Theor. Biol. 251: 210 – 226.

MA thesis

Evolution of dominance under frequency-dependent selection (2006). Advised by Prof. Reinhard Bürger.

Conference proceedings

i. Stephan Peischl and Kristan A. Schneider (2009). Evolution of Dominance and Assortative Mating under Frequency-Dependent Intraspecific Competition. Abstract Guide of the 12th ESEB Congress.

Selected Talks and Presentations

i. Evolution of dominance and assortative mating under frequency-dependent disruptive selection.
   Poster at the 12th ESEB Congress 2009 in Turin, Italy.

ii. Polymorphism in migration-selection models with dominance.
   Talk at 'Evolution in Metapopulations' workshop 2009 in La Fouly, Switzerland.

iii. Evolution of dominance and assortative mating under frequency-dependent disruptive selection.
   Talk at 'Mathematical Models in Ecology and Evolution 2009' meeting in Bristol, UK.

Zusammenfassung

In dieser Dissertation präsentiere ich Teile meiner wissenschaftlichen Arbeit über mathematische Modelle in der Populationsgenetik. Die Dissertation besteht aus zwei Teilen welche sich beide mit frequenzabhängiger Selektion und Dominanz beschäftigen. Im ersten Teil betrachte ich ein Modell mit innerspezifischer Kompetition und assortativer Paarung. Dieser Teil besteht aus zwei eng miteinander verbundenen Kapiteln. Im ersten Kapitel wird die Evolution von Dominanz unter assortativer Paarung analysiert. Kapitel Zwei behandelt die Evolution von assortativer Paarung mit einem konstanten Grad von Dominanz. Diese Arbeit baut auf meiner Diplomarbeit auf in welcher ich die Evolution von Dominanz unter frequenzabhängiger Selektion in einer sich zufällig paarenden Population studiert habe. Teil zwei der Dissertation besteht aus einem einzelnen Kapitel und behandelt eine andere Form von frequenzabhängiger Selektion. In diesem Kapitel analysiere ich den Zusammenhang zwischen dem Grad von Dominanz und der Anzahl der Allele die durch Selektion und Migration in zwei verschiedenen Lebensräumen erhalten werden können. Dieses Kapitel wurde durch einen kürzlich erschienenen Artikel von Professor Thomas Nagylaki motiviert.

Bibliography

Ackermann, M., Doebeli, M., 2004. Evolution of niche width and adaptive diversification. Evolution 58, 2599–2612.

Allen, J. A., Greenwood, J. J. D., 1988. Frequency-Dependent Selection by Predators. Phil. Trans. R. Soc. Lond. 319, 485 – 503.

Asmussen, M. A., Basnayake, E., 1990. Frequency-dependent selection: The high potential for permanent genetic variation in the diallelic, pairwise interaction model. Genetics 125, 215–230.

Barton, N. H., de Cara, M. A., 2009. The evolution of strong reproductive isolation. Evolution 63, 1171–1190.

Bodmer, W. F., 1965. Differential Fertility in Population Genetics Models. Genetics 51, 411–424.

Bolnick, D. I., 2004. Can intraspecific competition drive disruptive selection? An experimental test in natural populations of sticklebacks. Evolution 58, 608–618.

Bolnick, D. I., 2006. Multi-species outcomes in a common model of sympatric speciation. J. Theor. Biol. 241, 734–744.

Bolnick, D. I., Doebeli, M., 2003. Sexual dimorphism and adaptive speciation: two sides of the same ecological coin. Evolution 57, 2433–2449.

Bourguet, D., 1999. The evolution of dominance. Heredity 83, 1–4.

Bourguet, D., Prout, M., Raymond, M., 1996. Dominance of insecticide resistance presents a plastic response. Genetics 143, 407–416.

Bulmer, M. G., 1974. Density-dependent selection and character displacement. Am. Nat. 108, 45–58.

Bulmer, M. G., 1980. The Mathematical Theory of Quantitative Genetics. Oxford University Press, New York.

Bürger, R., 2000. The Mathematical Theory of Selection, Recombination, and Mutation. Wiley, Chichester.

Bürger, R., 2002a. Additive genetic variation under intraspecific competition and stabilizing selection: A two-locus study. Theor. Pop. Biol. 61, 197–213.

Bürger, R., 2002b. On a genetic model of intraspecific competition and stabilizing selection. Am. Nat. 160, 661–682.

Bürger, R., 2005. A multilocus analysis of intraspecific competition and stabilizing selection on a quantitative trait. J. Math. Biol. 50, 355–396.

Bürger, R., 2009a. Multilocus selection in subdivided populations i. Convergence properties for weak or strong migration. J. Math. Biol. 58, 939–978.

Bürger, R., 2009b. Multilocus selection in subdivided populations ii. Maintenance of polymorphism under weak or strong migration. J. Math. Biol. 58, 979–997.

Bürger, R., Bagheri, H. C., 2008. Dominance and its evolution. Encyclopedia of ecology Elsevier B.V, Oxford, 945 – 952.

Bürger, R., Gimelfarb, A., 2004. The effects of intraspecific competition and stabilizing selection on a polygenic trait. Genetics 167, 1425 – 1443.

Bürger, R., Schneider, K. A., 2006. Intraspecific competitive divergence and convergence under assortative mating. Am. Nat. 167, 190–205.

Bürger, R., Schneider, K. A., Willensdorfer, M., 2006. The conditions for speciation through intraspecic competition. Evolution 60, 2185–2206.

Charlesworth, D., Charlesworth, B., 1975a. Theoretical genetics of Batesian mimicry I. single-locus models. J. Theor. Biol. 55, 283–303.

Charlesworth, D., Charlesworth, B., 1975b. Theoretical genetics of Batesian mimicry III. Evolution of dominance. J. Theor. Biol. 55, 325–337.

Christiansen, F. B., 1982. Natural selection related to the biotic environment. Evolution and the Genetics of Populations 29, 85–102.

Christiansen, F. B., Feldman, M. W., 1975. Subdivided populations: a review of the one- and two-locus deterministic theory. Theor. Pop. Biol. 7, 13–38.

Christiansen, F. B., Loeschcke, V., 1980. Evolution and intraspecific exploitative competition. I. One-locus theory for small additive gene effects. Theor. Pop. Biol. 18, 297–313.

Clarke, B., 1979. The evolution of genetic diversity. Proc. R. Soc. Lond. 205, 453–474.

Cockerham, C. C., Burrows, P. M., Young, S. S., Prout, T., 1972. Frequency-dependent selection in randomly mating populations. Am. Nat. 106, 493–515.

Crosby, J. L., 1970. The evolution of genetic discontinuity: Computer models of the selection of barriers to interbreeding between subspecies. Heredity 25, 253–297.

de Cara, M. A., Barton, N. H., Kirkpatrick, M., May 2008. A model for the evolution of assortative mating. Am. Nat. 171, 580–596.

Dickinson, H., J., A., 1973. Theoretical considerations of sympatric divergence. Am. Nat. 107, 256–274.

Dieckmann, U., Doebeli, M., 1999. On the origin of species by sympatric speciation. Nature 400, 354–357.

Dieckmann, U., Doebeli, M., Metz, J. A. J., Tautz, D., 2004. Adaptive Speciation. Cambridge Univ. Press, Cambridge, U.K.

Dieckmann, U., Law, R., 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. J. Math. Biol. 34, 579–612.

Doebeli, M., 1996. A quantitative genetic competition model for sympatric speciation. J. Evol. Biol. 9, 893–909.

Doebeli, M., Blok, H. J., Leimar, O., Dieckmann, U., 2007. Multimodal pattern formation in phenotype distributions of sexual populations. Proc. Biol. Sci. 274, 347–357.

Doebeli, M., Dieckmann, U., 2005. Adaptive dynamics as a mathematical tool for studying the ecology of speciation processes. J. Evol. Biol. 18, 1194–1200.

Drossel, B., Mckane, A., 2000. Competitive speciation in quantitative genetic models. J. Theor. Biol. 204, 467–478.

Durinx, M., van Dooren, T. J. M., 2008. Assortative mate choice and dominance modification: Alternative ways of removing heterozygote disadvantage. Evolution 63, 334–352.

Endler, J. A., 1988. Frequency-dependent predation, crypsis and aposematic coloration. Philos. Trans. R. Soc. Lond., B, Biol. Sci. 319, 505–523.

Epstein, E., Guttman, R., 1984. Mate selection in man: evidence, theory, and outcome. Soc. Biol. 31, 243–78.

Eyland, E. A., 1971. Moran's island migration model. Genetics 69, 399–403.

Felsenstein, J., 1976. The theoretical population genetics of variable selection and migration. Annu. Rev. Genet. 10, 253–280.

Felsenstein, J., 1981. Skepticism towards santa rosalia, or why are there so few kinds of animals. Evolution 35, 124–138.

Fisher, R. A., 1930. The genetical theory of natural selection. Oxford: Clarendon Press.

Garrison, R., Anderson, E., Reeds, S., 1968. Assortative marriage. Soc. Biol. 15, 113–127.

Gavrilets, S., 1998. One-locus two-allele models with maternal (parental) selection. Genetics 149, 1147–1152.

Gavrilets, S., 2003. Models of speciation: what have we learned in 40 years? Evolution 57, 2197–2215.

Gavrilets, S., 2004. Fitness Landscapes and the Origin of Species. Vol. 41 of Monographs in Population Biology. Princeton Uiversity Press.

Gavrilets, S., Boake, C. R. B., 1998. On the evolution of premating isolation after a founder event. Am. Nat. 152, 706–716.

Gavrilets, S., Hastings, A., 1995. Intermittency and transient chaos from simple frequency-dependent selection. Proc. R. Soc. Lond. Ser. B 261, 233–8.

Geritz, S. A. H., Kisdi, E., Meszéna, G., Metz, J. A. J., 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evol. Ecol. 12, 35–57.

Hadeler, K. P., Liberman, U., 1975. Selection models with fertility differences. J. Theor. Biol. 2, 19–32.

Ho, H., 1986. Assortative mating in unwed-birth parents? Adoptive and nonadoptive parents. Soc. Biol. 33, 77–86.

Karlin, S., 1982. Classifications of selection-migration structures and conditions for a protected polymorphism. Evolutionary Biology 14, 61–204.

Karlin, S., Campbell, R. B., 1980. Selection-Migration Regimes Characterized by a Globally Stable Equilibrium. Genetics 94, 1065–1084.

Karlin, S., McGregor, J., 1972. Application of method of small parameters to multi-niche population genetics models. Theor. Pop. Biol. 3, 210–238.

Kirkpatrick, M., Nuismer, S. L., 2004. Sexual selection can constrain sympatric speciation. Proc. Biol. Sci. 271, 687–693.

Kirkpatrick, M., Ravigné, V., 2002. Speciation by natural and sexual selection: models and experiments. Am. Nat. 159, 22–35.

Kondrashov, A. S., Kondrashov, F. A., 1999. Interactions among quantitative traits in the course of sympatric speciation. Nature 400, 351–354.

Kopp, M., Hermisson, J., 2006. Evolution of genetic architecture under frequency-dependent disruptive selection. Evolution 60, 1537–1550.

Kopp, M., Hermisson, J., 2008. Competitive speciation and costs of choosiness. J. Evol. Biol. 21, 1005–1023.

Leimar, O., 2005. The evolution of phenotypic polymorphism: randomized strategies versus evolutionary branching. Am. Nat. 165, 669–681.

Levene, H., 1953. Genetic equilibrium when more than one ecological niche is available. Am. Nat. 87, 331–333.

Loeschcke, V., Christiansen, F. B., 1984. Evolution and intraspecific exploitative competition. II. A two-locus model for additive gene effects. Theor. Pop. Biol. 26, 228–264.

Matessi, C., Gimelfarb, A., 2006. Discrete polymorphisms due to disruptive selection on a continuous trait. I: The one-locus case. Theor. Pop. Biol. 69, 283–295.

Matessi, C., Gimelfarb, A., Gavrilets, S., 2001. Long-term buildup of reproductive isolation promoted by disruptive selection: How far does it go? Selection 2, 41–64.

Matessi, C., Schneider, K. A., 2009. Optimization under frequency-dependent selection. Theor. Pop. Biol. 76, 1 – 12.

May, R. M., Oster, G. F., 1976. Bifurcations and dynamic complexity in simple ecological models. Am. Nat. 110, 573–599.

Maynard Smith, J., 1966. Sympatric speciation. Am. Nat. 100, 637–650.

Moore, W. S., 1979. A single locus mass-action model of assortative mating, with comments on the process of speciation. Heredity 42, 173–186.

Nagylaki, T., 1992. Introduction to Theoretical Population Genetics. Springer, Berlin.

Nagylaki, T., 2009. Polymorphism in multiallelic migration-selection models with dominance. Theor. Pop. Biol. 75, 239–259.

Nagylaki, T., Lou, Y., 2001. Patterns of multiallelic polymorphism maintained by migration and selection. Theor. Pop. Biol. 59, 297–313.

Nagylaki, T., Lou, Y., 2007. Evolution under multiallelic migration-selection models. Theor. Pop. Biol. 72, 21–40.

Nagylaki, T., Lou, Y., 2008. The dynamics of migration-selection models. Tutorials in Mathematical Biosciences IV, 117–170.

O'Donald, P., 1960. Assortive mating in a population in which two alleles are segregating. Heredity 15, 389–396.

Otto, S. P., Bourguet, D., 1999. Balanced polymorphisms and the evolution of dominance. Am. Nat. 153, 561–574.

Otto, S. P., Servedio, M. R., Nuismer, S. L., 2008. Frequency-dependent selection and the evolution of assortative mating. Genetics 179, 2091–2112.

Peischl, S., Bürger, R., 2008. Evolution of dominance under frequency-dependent intraspecific competition. J. Theor. Biol. 251, 210–226.

Peischl, S., Schneider, K. A., 2009. Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population. Evolution 64, 561–582.

Pennings, P. S., Kopp, M., Meszéna, G., Dieckmann, U., Hermisson, J., 2008. An analytically tractable model for competitive speciation. Am. Nat. 171, E44–E71.

Pfennig, D. W., Harcombe, W. R., Pfennig, K. S., 2001. Frequency-dependent batesian mimicry. Nature 410, 323–323.

Polechová, J., Barton, N. H., 2005. Speciation through competition: a critical review. Evolution 59, 1194–1210.

Prout, T., 1968. Sufficient conditions for multiple niche polymorphism. Am. Nat. 102, 493 – 496.

Roughgarden, J., 1972. Evolution of niche width. Am. Nat. 106, 683–718.

Rueffler, C., Van Dooren, T. J., Leimar, O., Abrams, P. A., 2006. Disruptive selection and then what? Trends Ecol. Evol. 21, 238–245.

Schneider, K. A., 2005. Competitive divergence in non-random mating populations. Theor. Pop. Biol. 68, 105–118.

Schneider, K. A., 2006. A multilocus-multiallele analysis of frequency-dependent selection induced by intraspecific competition. J. Math. Biol. 52, 483–523.

Schneider, K. A., 2007. Long-term evolution of polygenic traits under frequency-dependent intraspecific competition. Theor. Pop. Biol. 71, 342–366.

Schneider, K. A., Bürger, R., 2006. Does competitive divergence occur if assortative mating is costly? J. Evol. Biol. 19, 570 – 588.

Slabbekoorn, H., Smith, T. B., 2000. Does bill size polymorphism affect courtship song characteristics in the african finch pyrenestes ostrinus? Biol. J. Linn. Soc. 71, 737–753.

Slatkin, M., 1979. Frequency- and density-dependent selection on a quantitative character. Genetics 93, 755–771.

Slatkin, M., 1984. Ecological Causes of Sexual Dimorphism. Evolution 38, 622 – 630.

Smith, T. B., 1990. Resource use by bill morphs of an african finch: Evidence for intraspecific competition. Ecology 71, 1246–1257.

Smith, T. B., 1993. Disruptive selection and the genetic basis of bill size polymorphism in the african finch pyrenestes. Nature 363, 618–620.

Spuhler, J. N., 1968. Assortative mating with respect to physical characteristics. Eugen. Q. 15, 128–139.

Star, B., Stoffels, R. J., Spencer, H. G., 2007a. Evolution of fitnesses and allele frequencies in a population with spatially heterogeneous selection pressures. Genetics 176, 1743–1751.

Star, B., Stoffels, R. J., Spencer, H. G., 2007b. Single-locus polymorphism in a heterogeneous two-deme model. Genetics 177, 1625–1633.

Star, B., Trotter, M. V., Spencer, H. G., 2008. Evolution of fitnesses in structured populations with correlated environments. Genetics 179, 1469–1478.

Thieme, H. R., 2003. Mathematics in population biology. Princeton Series in Theoretical and Computational Biology. Princeton University Press, Princeton, NJ.

Turelli, M., Barton, N., Coyne, J., 2001. Theory and speciation. Trends Ecol. Evol. 16, 330–343.

Udovic, D., 1980. Frequency-dependent selection, disruptive selection, and the evolution of reproductive isolation. Am. Nat. 116, 621–641.

Van Dooren, T., Durinx, M., Demon, I., 2004. Sexual dimorphism or evolutionary branching? Evol. Ecol. Res. 6, 857–871.

Van Dooren, T. J. M., 1999. The evolutionary ecology of dominance-recessivity. J. Theor. Biol. 198, 519–532.

Van Doorn, G. S., Dieckmann, U., 2006. The long-term evolution of multilocus traits under frequency-dependent disruptive selection. Evolution 60, 2226–2238.

Waxman, D., Gavrilets, S., 2005. Issues of terminology, gradient dynamics and the ease of sympatric speciation in Adaptive Dynamics. J. Evol. Biol. 18, 1214–1219.

Wilson, D. S., Turelli, M., 1986. Stable underdominance and the evolutionary invasion of empty niches. Am. Nat. 127, 835.