International Institute for
Applied Systems Analysis
Schlossplatz 1
A-2361 Laxenburg, Austria

Tel: +43 2236 807 342
Fax: +43 2236 71313
E-mail: publications@iiasa.ac.at
Web: www.iiasa.ac.at

Interim Report **IR-02-062**

On a Genetic Model of Intraspecific Competition and Stabilizing Selection

Reinhard Bürger (reinhard.buerger@univie.ac.at)

Approved by

Ulf Dieckmann (dieckman@iiasa.ac.at)
Project Leader, Adaptive Dynamics Network

August 2002

IIASA STUDIES IN ADAPTIVE DYNAMICS NO. 68

The Adaptive Dynamics Network at IIASA fosters the development of new mathematical and conceptual techniques for understanding the evolution of complex adaptive systems.

Focusing on these long-term implications of adaptive processes in systems of limited growth, the Adaptive Dynamics Network brings together scientists and institutions from around the world with IIASA acting as the central node.

Scientific progress within the network is collected in the IIASA Studies in Adaptive Dynamics series.

No. 1 Metz JAJ, Geritz SAH, Meszéna G, Jacobs FJA, van Heerwaarden JS: *Adaptive Dynamics: A Geometrical Study of the Consequences of Nearly Faithful Reproduction.* IIASA Working Paper WP-95-099 (1995). van Strien SJ, Verduyn Lunel SM (eds): Stochastic and Spatial Structures of Dynamical Systems, Proceedings of the Royal Dutch Academy of Science (KNAW Verhandelingen), North Holland, Amsterdam, pp. 183-231 (1996).

No. 2 Dieckmann U, Law R: *The Dynamical Theory of Coevolution: A Derivation from Stochastic Ecological Processes.* IIASA Working Paper WP-96-001 (1996). Journal of Mathematical Biology 34:579-612 (1996).

No. 3 Dieckmann U, Marrow P, Law R: *Evolutionary Cycling of Predator-Prey Interactions: Population Dynamics and the Red Queen.* IIASA Preprint (1995). Journal of Theoretical Biology 176:91-102 (1995).

No. 4 Marrow P, Dieckmann U, Law R: *Evolutionary Dynamics of Predator-Prey Systems: An Ecological Perspective.* IIASA Working Paper WP-96-002 (1996). Journal of Mathematical Biology 34:556-578 (1996).

No. 5 Law R, Marrow P, Dieckmann U: *On Evolution under Asymmetric Competition.* IIASA Working Paper WP-96-003 (1996). Evolutionary Ecology 11:485-501 (1997).

No. 6 Metz JAJ, Mylius SD, Diekmann O: *When Does Evolution Optimize? On the Relation Between Types of Density Dependence and Evolutionarily Stable Life History Parameters.* IIASA Working Paper WP-96-004 (1996).

No. 7 Ferrière R, Gatto M: *Lyapunov Exponents and the Mathematics of Invasion in Oscillatory or Chaotic Populations.* Theoretical Population Biology 48:126-171 (1995).

No. 8 Ferrière R, Fox GA: *Chaos and Evolution.* IIASA Preprint (1996). Trends in Ecology and Evolution 10:480-485 (1995).

No. 9 Ferrière R, Michod RE: *The Evolution of Cooperation in Spatially Heterogeneous Populations.* IIASA Working Paper WP-96-029 (1996). The American Naturalist 147:692-717 (1996).

No. 10 van Dooren TJM, Metz JAJ: *Delayed Maturation in Temporally Structured Populations with Non-Equilibrium Dynamics.* IIASA Working Paper WP-96-070 (1996). Journal of Evolutionary Biology 11:41-62 (1998).

No. 11 Geritz SAH, Metz JAJ, Kisdi É, Meszéna G: *The Dynamics of Adaptation and Evolutionary Branching.* IIASA Working Paper WP-96-077 (1996). Physical Review Letters 78:2024-2027 (1997).

No. 12 Geritz SAH, Kisdi É, Meszéna G, Metz JAJ: *Evolutionary Singular Strategies and the Adaptive Growth and Branching of the Evolutionary Tree.* IIASA Working Paper WP-96-114 (1996). Evolutionary Ecology 12:35-57 (1998).

No. 13 Heino M, Metz JAJ, Kaitala V: *Evolution of Mixed Maturation Strategies in Semelparous Life-Histories: The Crucial Role of Dimensionality of Feedback Environment.* IIASA Working Paper WP-96-126 (1996). Philosophical Transactions of the Royal Society of London Series B 352:1647-1655 (1997).

No. 14 Dieckmann U: *Can Adaptive Dynamics Invade?* IIASA Working Paper WP-96-152 (1996). Trends in Ecology and Evolution 12:128-131 (1997).

No. 15 Meszéna G, Czibula I, Geritz SAH: *Adaptive Dynamics in a 2-Patch Environment: A Simple Model for Allopatric and Parapatric Speciation.* IIASA Interim Report IR-97-001 (1997). Journal of Biological Systems 5:265-284 (1997).

No. 16 Heino M, Metz JAJ, Kaitala V: *The Enigma of Frequency-Dependent Selection.* IIASA Interim Report IR-97-061 (1997). Trends in Ecology and Evolution 13:367-370 (1998).

No. 17 Heino M: *Management of Evolving Fish Stocks.* IIASA Interim Report IR-97-062 (1997). Canadian Journal of Fisheries and Aquatic Sciences 55:1971-1982 (1998).

No. 18 Heino M: *Evolution of Mixed Reproductive Strategies in Simple Life-History Models.* IIASA Interim Report IR-97-063 (1997).

No. 19 Geritz SAH, van der Meijden E, Metz JAJ: *Evolutionary Dynamics of Seed Size and Seedling Competitive Ability.* IIASA Interim Report IR-97-071 (1997). Theoretical Population Biology 55:324-343 (1999).

No. 20 Galis F, Metz JAJ: *Why Are There So Many Cichlid Species? On the Interplay of Speciation and Adaptive Radiation.* IIASA Interim Report IR-97-072 (1997). Trends in Ecology and Evolution 13:1-2 (1998).

No. 21 Boerlijst MC, Nowak MA, Sigmund K: *Equal Pay for all Prisoners/ The Logic of Contrition.* IIASA Interim Report IR-97-073 (1997). American Mathematical Society Monthly 104:303-307 (1997). Journal of Theoretical Biology 185:281-293 (1997).

No. 22 Law R, Dieckmann U: *Symbiosis Without Mutualism and the Merger of Lineages in Evolution.* IIASA Interim Report IR-97-074 (1997). Proceedings of the Royal Society of London Series B 265:1245-1253 (1998).

No. 23 Klinkhamer PGL, de Jong TJ, Metz JAJ: *Sex and Size in Cosexual Plants.* IIASA Interim Report IR-97-078 (1997). Trends in Ecology and Evolution 12:260-265 (1997).

No. 24 Fontana W, Schuster P: *Shaping Space: The Possible and the Attainable in RNA Genotype-Phenotype Mapping.* IIASA Interim Report IR-98-004 (1998). Journal of Theoretical Biology 194:491-515 (1998).

No. 25 Kisdi É, Geritz SAH: *Adaptive Dynamics in Allele Space: Evolution of Genetic Polymorphism by Small Mutations in a Heterogeneous Environment.* IIASA Interim Report IR-98-038 (1998). Evolution 53:993-1008 (1999).

No. 26 Fontana W, Schuster P: *Continuity in Evolution: On the Nature of Transitions.* IIASA Interim Report IR-98-039 (1998). Science 280:1451-1455 (1998).

No. 27 Nowak MA, Sigmund K: *Evolution of Indirect Reciprocity by Image Scoring/ The Dynamics of Indirect Reciprocity.* IIASA Interim Report IR-98-040 (1998). Nature 393:573-577 (1998). Journal of Theoretical Biology 194:561-574 (1998).

No. 28 Kisdi É: *Evolutionary Branching Under Asymmetric Competition.* IIASA Interim Report IR-98-045 (1998). Journal of Theoretical Biology 197:149-162 (1999).

No. 29 Berger U: *Best Response Adaptation for Role Games.* IIASA Interim Report IR-98-086 (1998).

No. 30 van Dooren TJM: *The Evolutionary Ecology of Dominance-Recessivity.* IIASA Interim Report IR-98-096 (1998). Journal of Theoretical Biology 198:519-532 (1999).

No. 31 Dieckmann U, O'Hara B, Weisser W: *The Evolutionary Ecology of Dispersal.* IIASA Interim Report IR-98-108 (1998). Trends in Ecology and Evolution 14:88-90 (1999).

No. 32 Sigmund K: *Complex Adaptive Systems and the Evolution of Reciprocation.* IIASA Interim Report IR-98-100 (1998). Ecosystems 1:444-448 (1998).

No. 33 Posch M, Pichler A, Sigmund K: *The Efficiency of Adapting Aspiration Levels.* IIASA Interim Report IR-98-103 (1998). Proceedings of the Royal Society London Series B 266:1427-1435 (1999).

No. 34 Mathias A, Kisdi É: *Evolutionary Branching and Coexistence of Germination Strategies.* IIASA Interim Report IR-99-014 (1999).

No. 35 Dieckmann U, Doebeli M: *On the Origin of Species by Sympatric Speciation.* IIASA Interim Report IR-99-013 (1999). Nature 400:354-357 (1999).

No. 36 Metz JAJ, Gyllenberg M: *How Should We Define Fitness in Structured Metapopulation Models? Including an Application to the Calculation of Evolutionarily Stable Dispersal Strategies.* IIASA Interim Report IR-99-019 (1999). Proceedings of the Royal Society of London Series B 268:499-508 (2001).

No. 37 Gyllenberg M, Metz JAJ: *On Fitness in Structured Metapopulations.* IIASA Interim Report IR-99-037 (1999). Journal of Mathematical Biology 43:545-560 (2001).

No. 38 Meszéna G, Metz JAJ: *Species Diversity and Population Regulation: The Importance of Environmental Feedback Dimensionality.* IIASA Interim Report IR-99-045 (1999).

No. 39 Kisdi É, Geritz SAH: *Evolutionary Branching and Sympatric Speciation in Diploid Populations.* IIASA Interim Report IR-99-048 (1999).

No. 40 Ylikarjula J, Heino M, Dieckmann U: *Ecology and Adaptation of Stunted Growth in Fish.* IIASA Interim Report IR-99-050 (1999). Evolutionary Ecology 13:433-453 (1999).

No. 41 Nowak MA, Sigmund K: *Games on Grids.* IIASA Interim Report IR-99-038 (1999). Dieckmann U, Law R, Metz JAJ (eds): The Geometry of Ecological Interactions: Simplifying Spatial Complexity, Cambridge University Press, Cambridge, UK, pp. 135-150 (2000).

No. 42 Ferrière R, Michod RE: *Wave Patterns in Spatial Games and the Evolution of Cooperation.* IIASA Interim Report IR-99-041 (1999). Dieckmann U, Law R, Metz JAJ (eds): The Geometry of Ecological Interactions: Simplifying Spatial Complexity, Cambridge University Press, Cambridge, UK, pp. 318-332 (2000).

No. 43 Kisdi É, Jacobs FJA, Geritz SAH: *Red Queen Evolution by Cycles of Evolutionary Branching and Extinction.* IIASA Interim Report IR-00-030 (2000).

No. 44 Meszéna G, Kisdi É, Dieckmann U, Geritz SAH, Metz JAJ: *Evolutionary Optimisation Models and Matrix Games in the Unified Perspective of Adaptive Dynamics.* IIASA Interim Report IR-00-039 (2000).

No. 45 Parvinen K, Dieckmann U, Gyllenberg M, Metz JAJ: *Evolution of Dispersal in Metapopulations with Local Density Dependence and Demographic Stochasticity.* IIASA Interim Report IR-00-035 (2000).

No. 46 Doebeli M, Dieckmann U: *Evolutionary Branching and Sympatric Speciation Caused by Different Types of Ecological Interactions.* IIASA Interim Report IR-00-040 (2000). The American Naturalist 156:S77-S101 (2000).

No. 47 Heino M, Hanski I: *Evolution of Migration Rate in a Spatially Realistic Metapopulation Model.* IIASA Interim Report IR-00-044 (2000). The American Naturalist 157:495-511 (2001).

No. 48 Gyllenberg M, Parvinen K, Dieckmann U: *Evolutionary Suicide and Evolution of Dispersal in Structured Metapopulations.* IIASA Interim Report IR-00-056 (2000). Journal of Mathematical Biology 45:79-105 (2002).

No. 49 van Dooren TJM: *The Evolutionary Dynamics of Direct Phenotypic Overdominance: Emergence Possible, Loss Probable.* IIASA Interim Report IR-00-048 (2000). Evolution 54: 1899-1914 (2000).

No. 50 Nowak MA, Page KM, Sigmund K: *Fairness Versus Reason in the Ultimatum Game.* IIASA Interim Report IR-00-57 (2000). Science 289:1773-1775 (2000).

No. 51 de Feo O, Ferrière R: *Bifurcation Analysis of Population Invasion: On-Off Intermittency and Basin Riddling.* IIASA Interim Report IR-00-074 (2000). International Journal of Bifurcation and Chaos 10:443-452 (2000).

No. 52 Heino M, Laaka-Lindberg S: *Clonal Dynamics and Evolution of Dormancy in the Leafy Hepatic Lophozia Silvicola.* IIASA Interim Report IR-01-018 (2001). Oikos 94:525-532 (2001).

No. 53 Sigmund K, Hauert C, Nowak MA: *Reward and Punishment in Minigames.* IIASA Interim Report IR-01-031 (2001). Proceedings of the National Academy of Sciences of the USA 98:10757-10762 (2001).

No. 54 Hauert C, De Monte S, Sigmund K, Hofbauer J: *Oscillations in Optional Public Good Games.* IIASA Interim Report IR-01-036 (2001).

No. 55 Ferrière R, Le Galliard J: *Invasion Fitness and Adaptive Dynamics in Spatial Population Models.* IIASA Interim Report IR-01-043 (2001). Clobert J, Dhondt A, Danchin E, Nichols J (eds): Dispersal, Oxford University Press, pp. 57-79 (2001).

No. 56 de Mazancourt C, Loreau M, Dieckmann U: *Can the Evolution of Plant Defense Lead to Plant-Herbivore Mutualism.* IIASA Interim Report IR-01-053 (2001). The American Naturalist 158: 109-123 (2001).

No. 57 Claessen D, Dieckmann U: *Ontogenetic Niche Shifts and Evolutionary Branching in Size-Structured Populations.* IIASA Interim Report IR-01-056 (2001). Evolutionary Ecology Research 4:189-217 (2002).

No. 58 Brandt H: *Correlation Analysis of Fitness Landscapes.* IIASA Interim Report IR-01-058 (2001).

No. 59 Dieckmann U: *Adaptive Dynamics of Pathogen-Host Interacations.* IIASA Interim Report IR-02-007 (2002). Dieckmann U, Metz JAJ, Sabelis MW, Sigmund K (eds): Adaptive Dynamics of Infectious Diseases: In Pursuit of Virulence Management, Cambridge University Press, Cambridge, UK, pp. 39-59 (2002).

No. 60 Nowak MA, Sigmund K: *Super- and Coinfection: The Two Extremes.* IIASA Interim Report IR-02-008 (2002). Dieckmann U, Metz JAJ, Sabelis MW, Sigmund K (eds): Adaptive Dynamics of Infectious Diseases: In Pursuit of Virulence Management, Cambridge University Press, Cambridge, UK, pp. 124-137 (2002).

No. 61 Sabelis MW, Metz JAJ: *Perspectives for Virulence Management: Relating Theory to Experiment.* IIASA Interim Report IR-02-009 (2002). Dieckmann U, Metz JAJ, Sabelis MW, Sigmund K (eds): Adaptive Dynamics of Infectious Diseases: In Pursuit of Virulence Management, Cambridge University Press, Cambridge, UK, pp. 379-398 (2002).

No. 62 Cheptou P, Dieckmann U: *The Evolution of Self-Fertilization in Density-Regulated Populations* . IIASA Interim Report IR-02-024 (2002). Proceedings of the Royal Society of London Series B 269:1177-1186 (2002).

No. 63 Bürger R: *Additive Genetic Variation Under Intraspecific Competition and Stabilizing Selection: A Two-Locus Study.* IIASA Interim Report IR-02-013 (2002). Journal of Theoretical Population Biology 61:197-213 (2002).

No. 64 Hauert C, De Monte S, Hofbauer J, Sigmund K: *Volunteering as Red Queen Mechanism for Co-operation in Public Goods Games.* IIASA Interim Report IR-02-041 (2002). Science 296:1129-1132 (2002).

No. 65 Dercole F, Ferrière R, Rinaldi S: *Ecological Bistability and Evolutionary Reversals under Asymmetrical Competition.* IIASA Interim Report IR-02-053 (2002). Evolution 56:1081-1090 (2002).

No. 66 Dercole F, Rinaldi S: *Evolution of Cannibalistic Traits: Scenarios Derived from Adaptive Dynamics.* IIASA Interim Report IR-02-054 (2002).

No. 67 Bürger R, Gimelfarb A: *Fluctuating Environments and the Role of Mutation in Maintaining Quantitative Genetic Variation.* IIASA Interim Report IR-02-058 (2002). Genetical Research 80:31-46 (2002).

No. 68 Bürger R: *On a Genetic Model of Intraspecific Competition and Stabilizing Selection.* IIASA Interim Report IR-02-062 (2002).

Issues of the IIASA Studies in Adaptive Dynamics series can be obtained at www.iiasa.ac.at/Research/ADN/Series.html or by writing to adn@iiasa.ac.at.

Contents

Abstract

A genetic model is investigated in which two recombining loci determine the genotypic value of a quantitative trait additively. Two opposing evolutionary forces are assumed to act: (i) stabilizing selection on the trait, favoring genotypes with an intermediate phenotype, and (ii) intraspecific competition mediated by that trait, favoring genotypes whose effect on the trait deviates most from that of the prevailing genotypes. Accordingly, fitnesses of genotypes have a frequency-independent component describing stabilizing selection, and a frequency- and density-dependent component modeling competition. We study how the underlying genetics, in particular recombination rate and relative magnitude of allelic effects, interacts with the conflicting selective forces and derive the resulting, surprisingly complex, equilibrium patterns. It is investigated under which conditions disruptive selection on the phenotypes can be observed, and how much genetic variation can be maintained in such a model. A number of unexpected phenomena are discovered, for instance that with little recombination the degree of stably maintained polymorphism and the equilibrium genetic variance can decrease as the strength of competition increases relative to the strength of stabilizing selection. In addition, mean fitness at the stable equilibria is usually much lower than the maximum possible mean fitness, and often even lower than the fitness at other, unstable, equilibria. Thus, the evolutionary dynamics in this system is almost always nonadaptive.

About the Authors

Reinhard Bürger
Institute for Mathematics
University of Vienna
Strudlhofgasse 4
A-1090 Vienna, Austria
and
Adaptive Dynamics Network
International Institute for Applied Systems Analysis
A-2361 Laxenburg, Austria

Acknowledgements

I thank J. Travis and J. Hermisson for several useful suggestions that helped to clarify and improve the presentation. Part of this work was performed when the author was visiting the Adaptive Dynamics Network at the International Institute of Applied Systems Analysis (IIASA) in Laxenburg, Austria. Its hospitality is gratefully acknowledged. This visit was funded by a grant from the Austrian Ministery of Science. Financial support was also provided by the Austrian Fonds zur Förderung der wissenschaftlichen Forschung, Project P14682-MAT.

On a Genetic Model of Intraspecific Competition and Stabilizing Selection

Reinhard Bürger

Biological evolution results from the interplay of the selection caused by the ecological system a population is embedded in and the genetic mechanisms occuring along with reproduction. Evolutionary ecology and genetics each are flourishing fields, but only a minority of studies have contributed to their integration. In population-genetics modeling, the ecology is typically packed into the fitness function and, in the vast majority of investigations, this fitness function is assumed to be constant, as if populations lived in a static environment. Although the consequences of physical environmental change, such as periodic or random changes, have been explored to some extent, the interactive effects between a population and its own environment have mostly been ignored. Such feedback, induced for instance through the exploitation of the available resources or predation on or from other populations, leads to frequency- and density-dependent selection.

By contrast, the interactions within a population and between a population and its biotic and abiotic environment are the central topics of ecological research. The evolutionary consequences of frequency-dependent selection have been investigated mainly within the framework of evolutionary game theory (e.g. Maynard Smith 1982, Hofbauer and Sigmund 1998) and, more recently, also within what is sometimes called adaptive dynamics theory (cf. Dieckmann 1997). Common to approaches within these frameworks is, with very few exceptions, a lack of genetics, i.e., reproduction is usually assumed to be asexual and populations monomorphic.

Frequency-dependent selection has been included in population-genetics theory since its conception (Fisher 1930), but concrete studies remained sporadic for a long time (e.g. Wright 1948). Only much later has the theory of one-locus models under frequency- (and density-) dependent selection been developed more systematically (e.g., Clark 1972, Cockerham et al. 1972, Matessi and Jayakar 1976, Asmussen 1983). In these investigations, fitnesses, or growth rates, are assigned directly to genotypes and assumed to depend in a linear logistic, hyperbolic, or, more generally, in a monotone decreasing way on the strength of competition perceived by that genotype. The strength of competition experienced by, say, genotype i is expressed in the form $\sum_j \alpha_{ij} P_j N$, where the α_{ij} are coefficients measuring competition between genotypes i and j, P_j is the frequency of genotype j, and N is the population size (cf. Asmussen 1983). The focus of these studies is on the exploration of the basic properties of the resulting models, such as equilibrium structure, conditions for a protected polymorphism, examination of the possibility of multiple polymorphic equilibria, or search for optimization principles. A general conclusion that can be drawn from these investigations is that the conditions for maintaining a stable polymorphism

are much relaxed compared with constant fitnesses because heterozygote advantage is no longer required. Another feature of frequency- and density-dependent selection, most generally explored by Nagylaki (1979), is that neither mean fitness nor population size are maximized, though for weak selection approximate optimization results with error estimates can be derived.

Whereas in the above-mentioned approaches frequency dependence enters in a relatively abstract way, in another line of research the relevant parameters have been derived from explicit models of differential resource utiliziation by genotypes (Christiansen and Loeschcke 1980, Matessi and Jayakar 1981, Loeschcke and Christiansen 1984). These authors examined the effects of intraspecific exploitative competition for a linear resource spectrum using models based on the niche concept of MacArthur and Levins (1967) and some of its generalizations (Roughgarden 1972, Christiansen and Fenchel 1977). Since this work is closely related to ours, we shall return to its discussion further below. Similar types of models are employed in the treatments of competition for one resource by Matessi and Gatto (1984) and of differential utilization of two resources by Wilson and Turelli (1989).

Various aspects of frequency-dependent selection have also been investigated within the framework of quantitative genetics and phenotypic evolution (e.g. Bulmer 1974, 1980; Lande 1976; Slatkin 1979; Taper and Case 1992; Charlesworth 1993; Day and Taylor 1996). In contrast to the ESS-related approaches in which the fate of a rare mutant in an otherwise monomorphic, usually asexual, population is studied, the quantitative-genetic models realistically assume variable continuous traits, but with a Gaussian frequency distribution of fixed genetic and phenotypic variance. Under this assumption, the dynamics of the mean value of the trait under selection can be described by relatively simple difference or differential equations, but application to long-term evolution is problematic because it requires the genetic variance to change on a much slower time scale than the mean, an assumption that is questionable (e.g., Turelli 1988, Bürger 2000). Interestingly, despite their fundamental differences, the ESS-related and these quantitative-genetic models share substantial mathematical similarities; under some conditions even similar results are obtained (e.g. Iwasa et al. 1991, Abrams et al. 1993, Taylor and Day 1997).

From a population-genetics point of view, the ESS-related models, the phenotypic quantitative-genetic models, and the one-locus models all are based on rather restrictive, though very different, assumptions. Quantitative traits – and many traits of ecological importance belong to this category – are determined by several or many gene loci that may be linked. Such traits usually exhibit substantial genetic variability and its amount can neither be ignored nor be expected to be constant accross many generations. Indeed, different assumptions about the underlying genetics, may yield qualitatively different conclusions about the maintenance of genetic variation under frequency-dependent selection (Slatkin 1979).

It is the purpose of this paper to further diminish the gap between ecological and population-genetic modeling by using an explicit genetic model of a quantitative trait to explore the consequences of a balance between two opposing evolutionary forces: stabilizing selection on the trait and intraspecific competition for a one-dimensional rescource continuum. To this end, we proceed from the standpoint of population genetics and incorporate both frequency-independent stabilizing selection as well as

the ecological interactions between genotypes into the, then frequency- and density-dependent, fitness function of the trait. As a first step in approximating reality, in which a quantitative trait may be determined by a small number of major genes assisted by a larger number of minor genes (Lynch and Walsh 1998, Chap. 13), the trait is assumed to be determined by two recombining loci of arbitrary effect. The advantage of such an approach is that population-genetic modeling has a firm foundation, namely the laws of Mendelian genetics, and 'mechanistic' models are available, whereas in some ecologically oriented approaches approximations are used to include basic genetics that are difficult to justify or verify.

Part of the motivation for the present investigation originated from the desire to study and understand the mechanisms by which heritable variation in quantitative traits is maintained. Although, this has been a major research program in evolutionary genetics for the past thirty years (receiving much momentum through the work of Lande 1975) and substantial progress has been achieved, many open questions remain (for a comprehensive review see Bürger 2000, Chaps. VI and VII). However, the examination of some mechanisms has been neglected, in particular those related to ecology. One reason may be that the early work of Bulmer (1974, 1980) and Slatkin (1979) showed that frequency-dependent selection can increase the genetic variance of a trait under stabilizing selection, but only if a number of prerequisites are fulfilled, in particular, competition must be strong enough. Therefore, and because many quantitative traits of interest to geneticists and breeders are unlikely to be under frequency-dependent selection, frequency-dependence has not been considered as being a factor of general relevance in maintaining heritable variation. It may, however, be of importance for traits of ecological relevance, some of which show very high heritabilities (cf. Mousseau and Roff 1987), but this has not yet been properly investigated. Because substantial heritabilities are a common feature among quantitative traits, much of the work on the maintenance of genetic variation has focused on mechanisms that have the potential of being general agents in promoting genetic variation, such as mutation.

This study is related to the work of Bulmer (1974), Slatkin (1979), Christiansen and Loeschcke (1980), and Loeschcke and Christiansen (1984), who investigated the role of intraspecific competition in maintaining heritable variation on the basis of various quantitative-genetic models. Bulmer considered variation at a diallelic locus of infinitesimally small effect on a normally distributed trait with given variance. Slatkin explored, among others, a continuous genotype model of Lande's (1975) kind, also assuming a Gaussian distribution of phenotypes. Although slightly different assumptions about the fitnesses are employed, the two models yield qualitatively similar results: the genetic variance maintained at a stable equilibrium depends in a threshold-like manner on the strength of competition relative to the strength of stabilizing selection, i.e., with weak competition no genetic variance is maintained, with strong competition disruptive selection balances stabilizing selection and much genetic variance can be maintained. A similar result was proved by the present author for a two-locus model with equivalent loci (Bürger 2002). Slatkin (1979) also investigated a one-locus model, but it produced different equilibria because of constraints on the relationship between the mean and the variance imposed by the genetic assumptions.

Christiansen and Loeschcke (1980) examined the equilibrium structure of a one-locus model with multiple alleles and determined conditions under which a polymorphism is stable. Roughly, their main conclusion is that if competition is strong relative to stabilizing selection, then for a broad range of parameters two alleles are maintained in the population, but almost never more than two. For weak competition, the conditions under which more than one allele is maintained are rather restrictive. Loeschcke and Christiansen (1984) studied a two-locus model, mainly for tightly linked loci, and assumed strong competition. Then, in general, two-locus polymorphisms are maintained. Since their work is the one which is most closely related to the present one, this relation will be examined in a separate section and in the Discussion.

This paper is also concerned with a two-locus model of a quantitative trait, but from a quite different perspective than in Loeschcke and Christiansen (1984). In a certain sense, the present analysis considers stabilizing selection as given and investigates the consequences of increasingly strong competition and frequency dependence. Two special cases of the presently used model, namely loci of equal effects and free recombination, were treated in a previous paper (Bürger 2002). Here, a more general model with arbitrary linkage and arbitrary locus effects is analyzed. We determine all possible equilibrium structures, investigate how the equilibrium genetic variance depends on the genetic and ecological parameters, explore under which conditions empirically detectable disruptive selection on the phenotypes occurs, and examine the extent to which the evolutionary dynamics is adaptive and mean fitness is maximized. It turns out that the interaction of the genetic system with the selective forces induced by the ecological model leads to a number of surprising, previously unobserved, phenomena.

The Model

We consider a randomly mating diploid population with discrete generations and equivalent sexes that is sufficiently large to ignore random genetic drift. Selection acts only through differential viabilities. Individual fitness is assumed to be determined by two components: (i) by stabilizing selection on a quantitative character, and (ii) by competition among individuals.

The first component is frequency independent and may reflect some sort of direct selection on the trait, for example through differential supply of a resource whose utilization efficiency is phenotype dependent. However, frequency-independent stabilizing selection could as well be caused by indirect selection through pleiotropic side effects of alleles that primarily contribute to a fitness-related trait (e.g. Robertson 1967, Hill and Keightley 1988, Bürger 2000, Chap. VII). We ignore environmental variation and deal directly with the fitnesses of genotypic values (see also below). For simplicity, we will sometimes use the words genotypic value and phenotype synonymously. Following the population-genetic tradition starting with Wright (1935), stabilizing selection is modeled by the quadratic function

$$S(g) = 1 - g^2/(2V_\mathrm{s}) \,, \tag{1}$$

where V_s is an inverse measure for the strength of stabilizing selection. Of course,

$S(g)$ is assumed to be positive on the range of possible phenotypes, thus restricting the admissible values of V_s.

The second component of fitness is frequency dependent. We assume that competition between phenotypes g and h can be described by

$$\alpha(g,h) = 1 - \frac{1}{2\sigma_\alpha^2}(g-h)^2 \;, \tag{2}$$

with the obvious constraint that the maximum difference between genotypic values must be less than $\sqrt{2\sigma_\alpha^2}$. Thus competition between individuals of similar phenotype will be much stronger than between individuals of very different phenotype, as it will be the case if different phenotypes preferentially utilize different food resources. In this context, σ_α^2 can be interpreted as the variance of the genotypes' utilization functions which differ only in their mean but not in their variance (cf. Christiansen and Loeschcke 1980). Small σ_α^2 means that phenotypes are specialized and implies a strong frequency-dependent effect of competition. In the limit $\sigma_\alpha^2 \to \infty$, frequency dependence vanishes because every phenotype can utilize nearly the full resource spectrum.

Let $P(h)$ denote the relative frequency of individuals with phenotype h. Then the intraspecific competition function $\bar{\alpha}_P(g)$, which measures the strength of competition perceived by phenotype g if the population distribution is P, is given by

$$\bar{\alpha}_P(g) = \sum_h \alpha(g,h)P(h)$$

and calculated to be

$$\bar{\alpha}_P(g) = 1 - \frac{1}{2\sigma_\alpha^2}[(g-\bar{g})^2 + \sigma_g^2] \;. \tag{3}$$

Here, $\bar{g}$ and σ_g^2 denote the mean and variance, respectively, of the distribution P of genotypic values.

Similar to Bulmer's model (1974, 1980), we assume that the absolute fitness of an individual with genotypic value (phenotype) g is given by

$$W(g) = \left(\rho - \frac{N}{\kappa}\,\bar{\alpha}_P(g)\right) S(g) \;, \tag{4}$$

where ρ and κ are positive parameters and N denotes the total population size. For notational simplicity, the dependence of $W(g)$ on N and P is omitted. We note that the fitness function $W(g)$ belongs to the class of fitness functions used by Asmussen (1983) in her study of a single diallelic locus (cf. Introduction).

In the context of density-dependent growth models, the parameter ρ in (4) is related to the growth rate of the population and κ is proportional to the carrying capacity. The precise relation of the present model to those of Slatkin (1979), Christiansen and Loeschcke (1980), and Loeschcke and Christiansen (1984), who assumed a Lotka-Volterra-type functional form for fitness, is worked out in the following section. In these studies the functions S and α specified in (1) and (2) are assumed Gaussian, but the quadratic functions used here will be adequate approximations, unless selection or competition are very strong. In particular, the present choice

enables us to deduce some results analytically and, as explained in the Discussion, does not lead to certain special effects that a Gaussian fitness function causes under strong selection. In addition, environmental noise has a smoothing (and weakening) effect on the fitnesses assigned to genotypic values, hence the fitness function modeling selection on genotypic values can usually be assumed to be smooth (cf. Nagylaki 1989). Therefore, a quadratic fitness function $S(g)$ will be the natural one as a model of stabilizing selection as long as selection is sufficiently weak or locus effects sufficiently small that the fitness function can be approximated by its Taylor polynomial of degree two.

The genetic assumptions are as follows. The trait values g are determined additively by two loci (no dominance or epistasis), each with two alleles, A_1 and A_2, B_1 and B_2. After reproduction, the four gametes A_1B_1, A_1B_2, A_2B_1, A_2B_2 have relative frequencies p_1, p_2, p_3, p_4, respectively. We label these gametes by $i = 1, 2, 3, 4$. Frequencies in the subsequent generation are denoted by p_i'. Let the contributions of the alleles A_1, A_2, B_1, and B_2 to the genotypic value g of the trait be $\beta - \frac{1}{2}\gamma_1$, $\beta + \frac{1}{2}\gamma_1$, $-\beta - \frac{1}{2}\gamma_2$, and $-\beta + \frac{1}{2}\gamma_2$, respectively, where β is an arbitrary constant. Because of additivity, the effects of the gametes A_1B_1, A_1B_2, A_2B_1, and A_2B_2 are $-\frac{1}{2}(\gamma_1+\gamma_2)$, $-\frac{1}{2}(\gamma_1-\gamma_2)$, $\frac{1}{2}(\gamma_1-\gamma_2)$, and $\frac{1}{2}(\gamma_1+\gamma_2)$. The resulting genotypic values are shown in table 1. For definiteness, we assume $\gamma_1 \geq \gamma_2 > 0$ and refer to these loci as major and minor, respectively. The parameters γ_1 and γ_2 are the effects of allelic substitution at locus one and two, respectively. For brevity, we call them the effects of the loci.

For the recursion relations that describe the demographic and genetic dynamics, we need to derive the fitnesses of the genotypes, the (marginal) fitnesses of the gametes, and the mean fitness of the population. To this end it is useful to introduce the following parameters:

$$\gamma = \tfrac{1}{2}(\gamma_1 + \gamma_2)\,, \quad e = \frac{\gamma_1 - \gamma_2}{2\gamma}\,, \quad s = \frac{\gamma^2}{2V_\mathrm{s}}\,. \tag{5}$$

Table 1. The genotypic values in the additive model.

	B_1B_1	B_1B_2	B_2B_2
A_1A_1	$-\gamma_1-\gamma_2$	$-\gamma_1$	$-\gamma_1+\gamma_2$
A_1A_2	$-\gamma_2$	0	γ_2
A_2A_2	$\gamma_1-\gamma_2$	γ_1	$\gamma_1+\gamma_2$

Here, γ^2 may be called the average (substitional) effect of the loci on the trait, e measures the disparity of effects ($0 \leq e < 1$, and $e \geq \frac{1}{3}$ if the effects differ by a factor of two or more), and s is a measure for the strength of stabilizing selection on genotypes ($0 < s < \frac{1}{4}$ because $S(g)$ must be positive). Following from (1) and (5), the fitness of the most extreme genotypes under stabilizing selection alone is $1-4s$. In the absence of competition, this yields a special case of the so-called symmetric viability model (Karlin and Feldman 1970) with the genotypic fitness values given in table 2.

Table 2. The fitnesses of genotypes, $S(g)$, caused by stabilizing selection.

	B_1B_1	B_1B_2	B_2B_2
A_1A_1	$1-4s$	$1-(1+e)^2s$	$1-4e^2s$
A_1A_2	$1-(1-e)^2s$	1	$1-(1-e)^2s$
A_2A_2	$1-4e^2s$	$1-(1+e)^2s$	$1-4s$

To exploit the symmetries of the model, the following coordinates are introduced:

$$x = p_1 + p_4\,, \quad y = p_1 - p_4\,, \quad z = p_2 - p_3\,. \tag{6}$$

A straightforward calculation shows that the mean genotypic value, or mean phenotype, is

$$\bar{g} = -2\gamma(y + ez)\,, \tag{7}$$

and the genetic variance is

$$\sigma_g^2 = 2\gamma^2[x - y^2 - 2eyz + e^2(1 - x - z^2)]\,. \tag{8}$$

Therefore, the competition function $\bar{\alpha}_P(g)$ can now be calculated by substituting (7) and (8) into (3). We shall write it in the form

$$\bar{\alpha}_P(g) = 1 - \frac{\gamma^2}{2\sigma_\alpha^2}\,\varphi_P(g)\,, \tag{9}$$

where explicit expressions for $\varphi_P(g) = [(g-\bar{g})^2 + \sigma_g^2]/\gamma^2$ $(g \geq 0)$ are given in table 3. The value $\varphi_P(-g)$ is obtained from $\varphi_P(g)$ by the simultaneous substitutions $y \to -y$ and $z \to -z$.

Table 3. The values of $\varphi_P(g)$ for $g \geq 0$.

$\varphi_P(\gamma_1 + \gamma_2)$	$= 2[2 + x + 4y + y^2 + 2ez(2 + y) + e^2(1 - x + z^2)]$
$\varphi_P(\gamma_1)$	$= 1 + 2x + 4y + 2y^2 + 2e(1 + 2y + 2z + 2yz) + e^2(3 - 2x + 4z + 2z^2)$
$\varphi_P(\gamma_1 - \gamma_2)$	$= 2[x + y^2 + 2ey(2 + z) + e^2(3 - x + 4z + z^2)]$
$\varphi_P(\gamma_2)$	$= 1 + 2x + 4y + 2y^2 - 2e(1 + 2y - 2z - 2yz) + e^2(3 - 2x - 4z + 2z^2)$
$\varphi_P(0)$	$= 2[x + y^2 + 2eyz + e^2(1 - x + z^2)]$

As a convenient measure for the strength of competition, or rather the amount of frequency dependence induced by competition, we introduce the dimensionless 'coefficient of competition'

$$c = \frac{\gamma^2/(2\sigma_\alpha^2)}{(\rho\kappa/N) - 1}\,. \tag{10}$$

Then we can write (4) as $W(g) = (\rho - N/\kappa)w(g)$, where

$$w(g) = [1 + c\varphi_P(g)]S(g)\,, \tag{11}$$

and large c means a strong frequency-dependent fitness effect of competition. The genotype- and, hence, frequency-independent effect of competition is subsumed in $\rho - N/\kappa$ and irrelevant for the genetic dynamics. We will often view stabilizing selection as given and consider frequency dependence as a perturbation of strength c.

Because in the recursion relations (12) for the gamete frequencies multiplicative constants cancel, we can use the (scaled) fitnesses $w(g)$. We write w_{ij} for the fitness $w(g)$ of genotype $g = ij$ consisting of the gametes i and j. The (marginal) fitness of gamete i is given by $w_i = \sum_{j=1}^{4} w_{ij} p_j$, and the mean fitness is $\bar{w} = \sum_{i,j=1}^{4} w_{ij} p_i p_j = \sum_{i=1}^{4} w_i p_i$. The w_{ij} and, consequently, the w_i and $\bar{w}$ can be calculated straightforwardly from (11) by resorting to tables 2 and 3. The explicit expressions, however, are formidable and not given. With a formula manipulation program such as *Mathematica* (Wolfram 1996) these calculations are easily automated.

Since random mating is assumed and gamete frequencies are measured after reproduction and before selection, Hardy-Weinberg proportions obtain and the genetic dynamics can be described in terms of gamete frequencies by the well-known system of recursion relations

$$\bar{w} p_i' = p_i w_i - \eta_i r w_{14} D \;, \quad i = 1, 2, 3, 4 \tag{12}$$

(e.g. Bürger 2000, Chap. II.1). Here $\eta_1 = \eta_4 = 1$, $\eta_2 = \eta_3 = -1$, r is the recombination fraction, and $D = p_1 p_4 - p_2 p_3$ measures linkage disequilibrium. The ecological dynamics follows the standard recursion

$$N' = N \overline{W} \;, \tag{13}$$

where $\overline{W} = (\rho - N/\kappa)\bar{w}$ is the mean absolute fitness. We assume that the demographic equilibrium is locally stable (which may require a sufficiently small growth rate) and the population size is given by the resulting equilibrium value. Then the genetic dynamics becomes density-independent. This indeed is an admissible approximation if selection is sufficiently weak (Nagylaki 1979).

Relations Between the Models

Here, we examine the relation between the ecological model used in the present investigation, basically due to Bulmer (1974, 1980), and models that are based on the Lotka-Volterra competition equations.

Slatkin (1979) considered a phenotypic character in a population of size N (we omit the time dependence) and, following Roughgarden (1972), assumed that fitness of an individual with phenotypic value z is given by the Lotka-Volterra functional form

$$W_{\mathrm{S}}(z) = 1 + R - \frac{RN}{k(z)} \bar{\alpha}_P(z) \;, \tag{14}$$

where

$$\bar{\alpha}_P(z) = \int \alpha(z - y) P(y)\, dy \;. \tag{15}$$

Here $1 + R$ is the maximum fitness in the absence of competition, $k(z)$ represents resources that can be utilized by an individual of type z, $\alpha(z - y)$ represents the competition between individuals of type z and y for the limiting resource, and P denotes the Gaussian density of the trait which has variance σ_z^2. As a model for $k(z)$, Slatkin used a function proportional to a Gaussian density, i.e.,

$$k(z) = K \exp\left[-\frac{z^2}{2\sigma_k^2}\right] , \tag{16}$$

where K may be interpreted as the carrying capacity and the variance σ_k^2 measures the range of available resources. Thus, small σ_k^2 means a small such range, hence strong stabilizing selection. Similarly, as an example of α he took

$$\alpha(z - y) = \exp\left[-\frac{(z-y)^2}{2\sigma_\alpha^2}\right] , \tag{17}$$

where σ_α^2 measures the extent of competition between individuals. Slatkin showed that with these choices, the fitness function (14) leads to a stable equilibrium with nonzero genetic variance σ_g^2 only if $\sigma_k^2 - \sigma_\alpha^2 > \sigma_e^2$, where σ_e^2 is the environmental variance, i.e., $\sigma_z^2 = \sigma_g^2 + \sigma_e^2$. If environmental variance is ignored ($\sigma_e^2 = 0$), then genotypic and phenotypic value can be identified and the condition for maintaining variation becomes $\sigma_k^2 > \sigma_\alpha^2$, i.e., the spectrum of available resources must be broader than any single consumer's niche.

Two models, closely related ecologically but on a more explicit genetic basis, were investigated by Christiansen and Loeschcke (1980) and Loeschcke and Christiansen (1984). Respectively, they considered a trait that is determined either by one locus with several possible alleles, and by two loci with two alleles each. Environmental effects are ignored. Following Christiansen and Fenchel (1977, Chap. 3), they assumed that fitness of individuals with genotype g is given by an expression analogous to (14), but with $R = R(g)$ depending on g and a constant proportion of $R(g)$ and $k(g)$, i.e., $R(g)/k(g) \equiv V$. Then their fitness function can be written as

$$W_{\mathrm{CL}}(g) = 1 + V[k(g) - N\bar{\alpha}_P(g)]$$

with $\bar{\alpha}_P(g)$ as in (15), but with a sum over all possible genotypes instead of an integral. Loeschcke and Christiansen (1984) employed (16) and (17) for k and α, whereas Christiansen and Loeschcke (1980) used quadratic approximations.

Developing W_{CL} into a Taylor series and omitting terms of order two or higher in $1/\sigma_\alpha^2$ and $1/\sigma_k^2$, as well as mixed terms, a polynomial of degree four in g is obtained. Equating the terms up to order two in g with those of our model [(1), (2), (4)], we obtain the following relations between our parameters and those of Christiansen and Loeschcke:

$$\kappa = \frac{1}{V} , \quad \rho = 1 + VK , \tag{18a}$$

and

$$V_s = \sigma_k^2 \frac{1 + V(K - N) + VN(\sigma_g^2 + \bar{g}^2)/(2\sigma_\alpha^2)}{VK} \tag{18b}$$

$$\approx \sigma_k^2 \frac{1 + V(K - N)}{VK} = \sigma_k^2 \frac{\rho\kappa - N}{\kappa(\rho - 1)} , \tag{18c}$$

where the approximation is valid if σ_α^2 is large, i.e., the frequency-dependent effect of competition is weak. Actually, Christiansen and Loeschcke used a slightly different notation: the σ_α^2 used here corresponds to their $2W^2$, this σ_k^2 to their $\sigma^2 + W^2$, and they set $K = 1$ or, equivalently, scaled N to N/K.

Importantly, for sufficiently weak stabilizing selection and competition, the two models become formally equivalent. In the analysis of their two-locus model, Loeschcke and Christiansen (1984) assumed that competition is stronger than stabilizing selection in the sense that (in their notation) $\sigma > W$. In the present notation, this means $\sigma_k^2 > \sigma_\alpha^2$. In our model, this assumption translates to $\frac{c}{s} > \frac{N}{K} = \frac{N}{\kappa(\rho-1)}$ (using [5], [10], [18]), which is typically close to 1 at equilibrium. We shall return to this observation in the Discussion.

Finally, we note that relations analogous to (18) can be derived for the model of Slatkin (1979). They read

$$\kappa = \frac{K}{R}\,, \quad \rho = 1 + R\,, \tag{19a}$$

and

$$V_\mathrm{s} = \sigma_k^2 \, \frac{1 + R - \frac{RN}{K}\left(1 - \frac{\sigma_g^2 + \bar{g}^2}{2\sigma_\alpha^2}\right)}{\frac{RN}{K}(1 - \frac{\sigma_g^2 + \bar{g}^2}{2\sigma_\alpha^2})}\,, \tag{19b}$$

$$\approx \sigma_k^2 \, \frac{1 + R(1 - N/K)}{RN/K} = \sigma_k^2 \, \frac{\rho\kappa - N}{N}\,, \tag{19c}$$

which differs from (18c) by the factor N/K. Thus, the condition $\sigma_k^2 > \sigma_\alpha^2$ in Slatkin's model reads $c > s$ in our model.

Equilibria and Their Stability Properties

For loci of equal effects ($e = 0$) fairly complete global stability results are proved in Bürger (2002). For unequal effects, even in the absence of competition such results are not available. Nevertheless, with stabilizing selection alone ($c = 0$) the model is well understood and the possible equilibria and their local stability properties have been derived (see Gavrilets and Hastings 1993, and Bürger 2000, Chap. VI.2). In this case, at most one locus can be maintained polymorphic if linkage is loose. This occurs if the effects of the loci differ by more than a factor of two, i.e., if $e > \frac{1}{3}$. For tightly linked loci, two types of stable polymorphic equilibria exist. Their stability conditions are complementary and linkage dependent.

The general case with $c > 0$ is much more complex, and often equilibria and their stability properties can be determined only numerically. Three different numerical methods have been used: numerical solution of the equilibrium conditions gives all possible equilibria; numerical evaluation of the eigenvalues yields asymptotic stability results; iteration of the recursion relations yields global stability results. Yet, some analytic results can be derived. We restrict our attention to positive recombination rates ($r > 0$). Therefore, equilibria involving three gametes cannot exist, nor equilibria at which either only the gametes with large effects, A_1B_1 and A_2B_2, or only the gametes with small effects, A_1B_2 and A_2B_1, are present.

The interaction of stabilizing selection, intraspecific competition, and recombination leads to a complex equilibrium structure, which is illustrated in figures 1 and 2. The precise conditions for existence and stability may be found in the Appendix. It may be noted that if $(\hat{p}_1, \hat{p}_2, \hat{p}_3, \hat{p}_4)$ is an equilibrium, then $(\hat{p}_4, \hat{p}_3, \hat{p}_2, \hat{p}_1)$ is also an equilibrium, and both have the same stability properties. Thus, all except symmetric equilibria, which by definition satisfy $\hat{p}_1 = \hat{p}_4$ and $\hat{p}_2 = \hat{p}_3$, coexist in pairs.

The six types of equilibria that may be stable are listed in table 4. Our analysis indicates that stability of one type excludes stability of any other type (see Appendix). Thus, there are never more than two stable equilibria. Types (d), (e), and (f) are asymptotically stable whenever they exist. Further equilibria can exist at the boundary, but they are always unstable (see Appendix).

Table 4. The six types of stable equilibria.

(a)	A pair of monomorphic equilibria ($p_2 = 1$ or $p_3 = 1$)
(b)	A pair of equilibria for which the locus with the larger effect (the major locus) is polymorphic, and the minor locus is monomorphic for one or the other allele
(c)	A symmetric equilibrium with $D < 0$
(d)	A symmetric equilibrium with $D \geq 0$
(e)	A pair of (polymorphic) asymmetric equilibria with $D < 0$
(f)	A pair of polymorphic equilibria with $D = 0$

Since the stable symmetric equilibrium with $D < 0$ and that with $D \geq 0$ are maintained in different regions of the parameter space, they are classified as different types. The symmetric equilibrium exhibits positive linkage disequilibrium if and only if

$$c > c_2 = \frac{s}{1 - 5(1 + e^2)s} \, . \tag{20}$$

According to our numerical results it is globally stable in this case. Therefore, positive linkage disequilibrium occurs if and only if (20) is satisfied, i.e., if the frequency dependence induced by competition is sufficiently strong relative to stabilizing selection. At the symmetric equilibrium, $D = p_1 - \frac{1}{4}$ holds, and high (positive) linkage disequilibrium is maintained only if the loci are tightly linked (results not shown).

For given $s = 0.05$ and four different values of the disparity e of locus effects, figure 1 displays the regions of stability of the different types of equilibria as a function of the coefficient of competition c and of the recombination rate r. Although r could assume any value between 0 and 0.5, and c could be any nonnegative number, a restricted parameter range is shown, because a further increase of either r or c does not alter the equilibrium structure.

Equilibria not existing under pure stabilizing selection ($c = 0$) occur only if $c > c_1$ (A.5), the value at which the interior equilibria with $D = 0$ bifurcate from the single-locus polymorphisms. It is at this value, which is not much less than c_2 (20), that frequency dependence becomes strong enough to dominate frequency-independent stabilizing selection and induce a markedly different equilibrium structure.

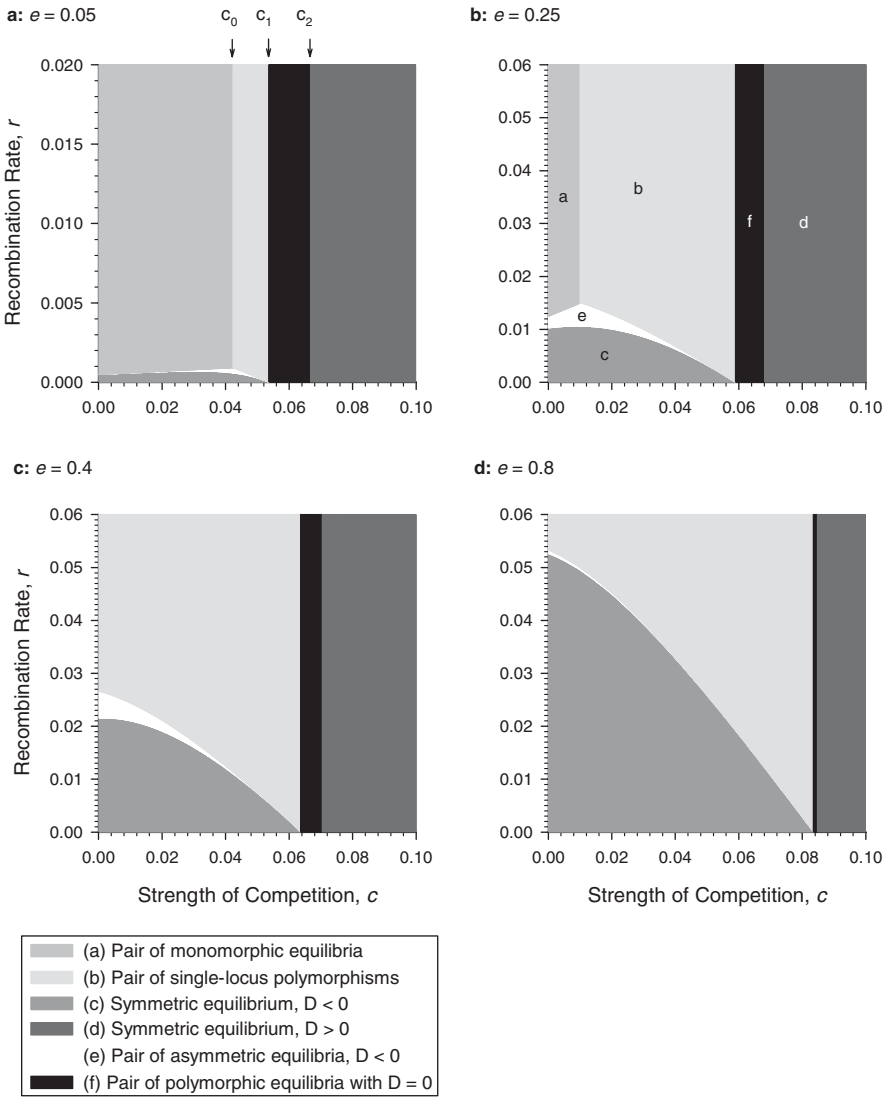

Figure 1: Regions of stability of the six possible types of stable equilibria for the four inidicated values of the disparity of locus effects e. In all cases, the strength of stabilizing selection is $s = 0.05$, so the fitness of the extreme genotypes under stabilizing selection alone is 80% of the maximum possible fitness. Note also that for better visibility, in figure 1A the range of displayed values r is smaller. The white region (e) extends to $r = 0$ and $c = c_1$ in all cases but becomes very thin, thus invisible. The values c_0, c_1, and c_2 are defined in (A.1b), (A.5), and (20), respectively.

By way of example we discuss how r and c affect the position of the stable equilibria. We do this for the case that the effects of the loci are different, but differ by less than a factor of two. Figure 2 displays the position of the stable equilibria as a function of the strength of competition as c increases from 0 to 0.1. As indicated, each panel is for another recombination rate. Since $s = 0.05$ and $e = 0.25$, each of the curves in figure 2 represents the location of the equilibrium along the respective horizontal line with ordinate r in figure 1B. The arrows in figure 2A indicate the

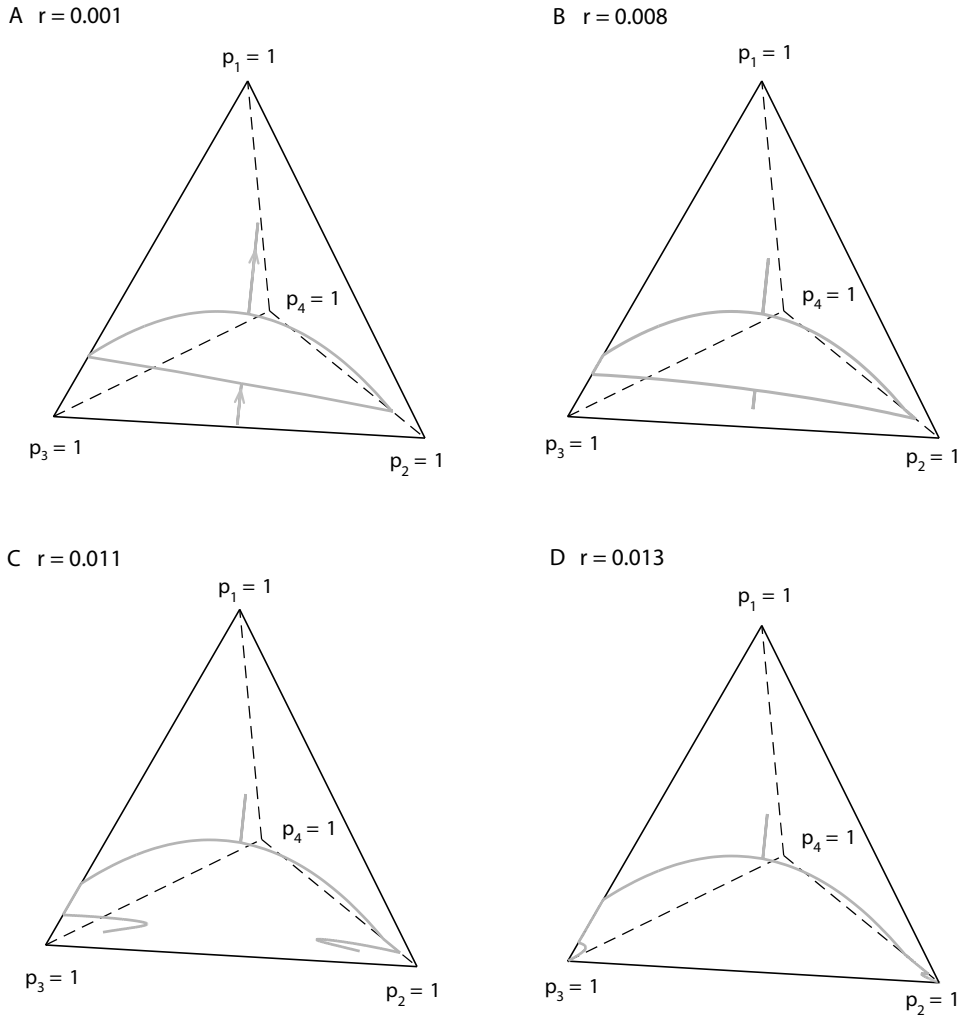

Figure 2: Positions of stable equilibria as c increases from 0 to 0.1. In all four cases, we have $s = 0.05$ and $e = 0.25$. For a detailed description see the main text.

direction of increasing c, which is the same in all figures.

In the following we describe the four cases.

r = 0.001 (figure 2A): For $c = 0$, the symmetric equilibrium with $D < 0$ is stable. It is close to the boundary edge $p_2 + p_3 = 1$, hence exhibits strong negative linkage disequilibrium. As c increases it moves toward the center along the symmetric line $\{p_1 = p_4, p_2 = p_3\}$, in direction of the arrow. If $c = 0.0562$, which is the value that solves $r_2(0.05, c, 0.25) = 0.001$ (cf. [A.8] and [A.9]), the asymmetric equilibria bifurcate from the symmetric equilibrium, which becomes unstable but continues to exist for larger c. The asymmetric equilibria are stable whenever they exist and move rapidly to the boundary, which they hit if $c = 0.0563$, the value that solves $r_1(0.05, c, 0.25) = 0.001$ (cf. [A.6] and [A.9]). They leave the simplex after exchanging stability with the single-locus polymorphisms, which are unstable for smaller values of c. For $c > 0.0563$ the single-locus polymorphisms are stable and move in direction of increasing $p_1(p_4)$, but become unstable at $c = c_1 = 0.0588$ (A.5) when the pair of interior equilibria with $D = 0$ bifurcates (A.10). As c increases

further, this pair of stable equilibria moves towards the center of the simplex and, at the center, merges with the (then still unstable) symmetric equilibrium if $c = c_2 = 0.0681$ (cf. [20] and [A.7]) and ceases to exist (a pitchfork bifurcation occurs). For all larger values of c, the symmetric equilibrium (now with $D > 0$) is globally stable and moves in direction of increasing $p_1 + p_4 (= 2p_1)$. In the limit $c \to \infty$ (not displayed), the coordinate p_1 of the symmetric equilibrium converges to a value $< \frac{1}{2}$, unless $r = 0$, when $p_1 + p_4 = 1$ can be reached.

$\mathbf{r = 0.008}$ (figure 2B): Similar as above, but at $c = 0$ the symmetric equilibrium lies further in the interior of the simplex than for $r = 0.001$, hence shows less linkage disequilibrium. The asymmetric equilibria bifurcate from the symmetric equilibrium if $c = 0.033$ (calculated from [A.8]) and hit the boundary if $c = 0.0365$ (A.9). Because c_1 and c_2 are independent of r, the other bifurcation values are the same as above.

$\mathbf{r = 0.011}$ (figure 2C): For $0 \leq c \leq 0.0267$, the asymmetric equilibria are stable and first move toward the interior, but then curve back to the boundary, which they hit if $c = 0.0267$. Then the single-locus polymorphisms become stable and the description from case $r = 0.001$ applies.

$\mathbf{r = 0.013}$ (figure 2D): For $0 \leq 0.0103$, the value that solves $r_0(0.05, c, 0.25) = 0.013$ (A.1a), the monomorphic equilibria are stable. Then the pair of asymmetric equilibria bifurcates, moves a little into the interior, and back to the boundary again, which they hit if $c = 0.0193$, the value that solves $r_1(0.05, c, 0.25) = 0.013$ (A.6). Then the single-locus polymorphisms become stable and the description from the case $r = 0.001$ applies.

If $r \geq r_{0,\max}$ (A.2), which for the parameters in figure 2 means $r \geq 0.0150$, the bifurcation structure is simpler because for $0 \leq c \leq c_0 = 0.0103$ (A.1b) the monomorphic equilibria are stable, then the single-locus polymorphisms bifurcate and are stable if $0.0103 < c \leq 0.0588$. For larger c the description from case $r = 0.001$ applies, i.e., if $c_1 = 0.0588 < c < 0.0681 = c_2$, the pair of interior equilibria with $D = 0$ is stable, and if $c > c_2 = 0.0681$, the symmetric equilibrium $D > 0$ is globally stable. The qualitatively identical case of free recombination was investigated in Bürger (2002). Figure 4 in that article displays the dependence of the regions of stability of the four possible equilibrium patterns (a,b,d,f) as a function of c and e.

Maintenance of Genetic Variation

Because the joint transformation $y \to -y$ and $z \to -z$ preserves the property of being an equilibrium, equations (7) and (8) inform us that if a pair of equilibria is stable, then each of them has the same variance but the mean phenotypes have opposite signs.

For loosely linked loci of similar effects ($e < \frac{1}{3}$, i.e., $\gamma_1 < 2\gamma_2$), stabilizing selection alone maintains no genetic variation. Weak frequency dependence ($c \leq c_0$; [A.1b]) does not change this; cf. figures 1A,B. If both loci have equal effects, then the genetic variance shows an almost threshold-like behavior (see figure 3 in Bürger 2002). As figure 3A shows, this extends to loci of very similar effects ($e = 0.05$), but already much less so to loci whose effects differ by a factor of $\frac{5}{3}$ ($e = 0.25$). For very different

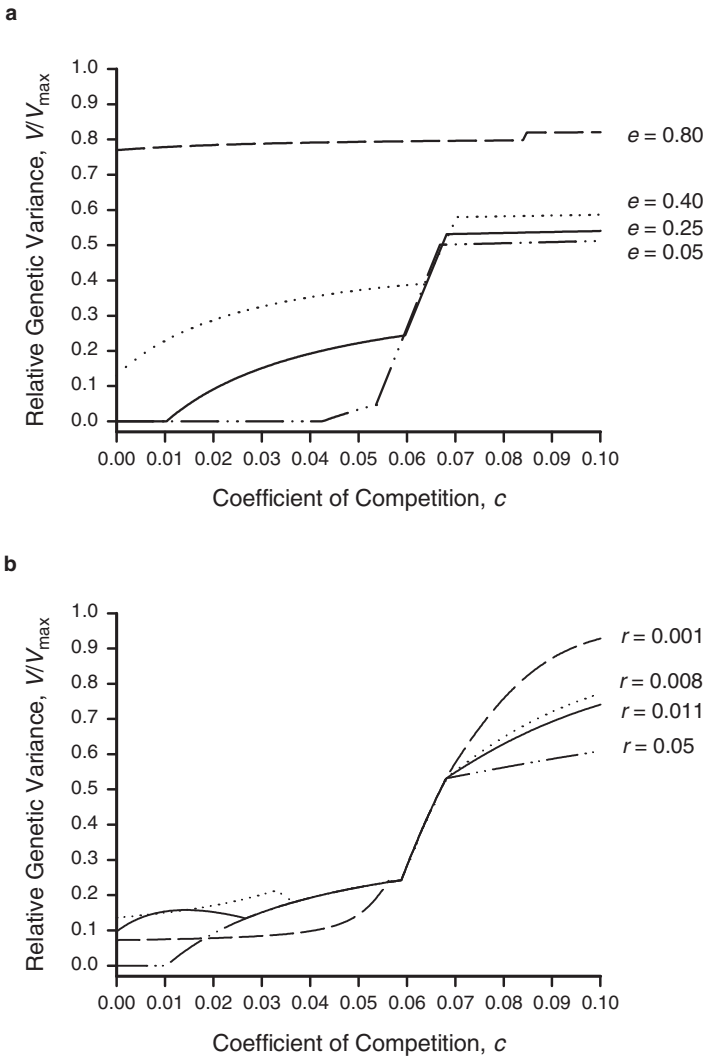

Figure 3: Genetic equilibrium variance (V) relative to the maximum possible variance ($V_{\max} = 2\gamma^2$) as a function of the strength of competition, c. In both figures we have $s = 0.05$. Figure 3A displays $V/V_{\max}$ for free recombination and four different values of e, as indicated. Figure 3B displays $V/V_{\max}$ for fixed $e = 0.25$, but for four different values of the recombination rate r, as indicated.

effects ($e > \frac{1}{3}$), stabilizing selection alone can maintain much genetic variance in this two-locus system, because then the major locus displays overdominance and is responsible for most of the genetic variance that can be maintained in a two-locus system. In this case competition adds little.

Figure 3A displays the genetic variance (V) relative to the maximum possible variance ($V_{\max} = 2\gamma^2$) for free recombination and different values of e. The rapid, almost linear, increase in variance occurs when the pair of interior equilibria with $D = 0$ is stable and moves to the center of the simplex. For strong frequency dependence, the symmetric equilibrium with $D > 0$ is stable, and the variance quickly asymptotes.

For tightly linked loci and weak frequency dependence, the situation is much

more complex because stabilizing selection alone can maintain stable polymorphisms. Surprisingly, an increase of competition may lead even to a slight decrease in genetic variance. This is clearly visible from the curves for the values $r = 0.008$ and $r = 0.011$ in figure 3B, and occurs in the range $0.01 \leq c \leq 0.04$ (cf. figure 1B). For $0.0588 \leq c \leq 0.0681$, the pair of interior equilibria with $D = 0$ exists and is stable. As they move to the center of the simplex, a marked, almost linear, increase in variance occurs. For strong competition, i.e., if the symmetric equilibrium with $D > 0$ is stable, there is always a high amount of genetic variation maintained; the tighter the linkage, the higher the variance.

Under stabilizing selection alone the equilibrium mean phenotype does not necessarily coincide with the optimum phenotype, which, in the present model, is at the midpoint of the range of possible phenotypes. As shown by figure 4, which is based on the parameters of figure 3B, strong competition is required for the mean phenotype to coincide with this midpoint, whereas for intermediate values of c the deviation may be decreased or increased.

Disruptive or Stabilizing Selection?

In the absence of other selective forces, intraspecific competition of the kind considered in the present model induces disruptive selection by favoring genotypes whose effect on the trait deviates most from that of the prevailing genotypes. Given the putative importance of frequency-dependent selection on the one hand and of stabilizing selection on the other hand, it is of interest to identify the conditions for which frequency dependence leads to detectable disruptive selection on a trait subject to frequency-independent stabilizing selection. From a theoretical point of view, it is reasonable to speak of disruptive selection if the fitness function has at least two distinct maxima. We concentrate on populations in equilibrium.

For loci of equal effects, it was shown analytically that the equilibrium fitnesses of the phenotypic values exhibit disruptive selection if and only if frequency dependence is strong enough to maintain both loci polymorphic (Bürger 2002). For loci of unequal effects, the situation is more complex and we can derive only sufficient conditions under which disruptive selection occurs. The fitnesses of all genotypes can be calculated from (11) and tables 2 and 3. At the symmetric equilibrium these expressions greatly simplify because $y = z = 0$. If competition is sufficiently strong that the symmetric equilibrium exhibits $D > 0$, i.e., if (20) holds, the following relations between the equilibrium fitnesses are easily derived:
If $e < \frac{1}{3}$, so that $\gamma_1 - \gamma_2 < \gamma_2$, then

$$w(0) < w(\gamma_1 - \gamma_2) < w(\gamma_2) < w(\gamma_1) \; . \tag{21a}$$

If $e > \frac{1}{3}$, so that $\gamma_2 < \gamma_1 - \gamma_2$, then

$$w(0) < w(\gamma_2) < w(\gamma_1 - \gamma_2) < w(\gamma_1) \; . \tag{21b}$$

These relations are valid for all admissible parameter values of s and r, provided (20) holds. Because of the symmetry properties of the equilibrium, the fitness function is symmetric, i.e., $w(-g) = w(g)$. Therefore, (21) shows that disruptive selection

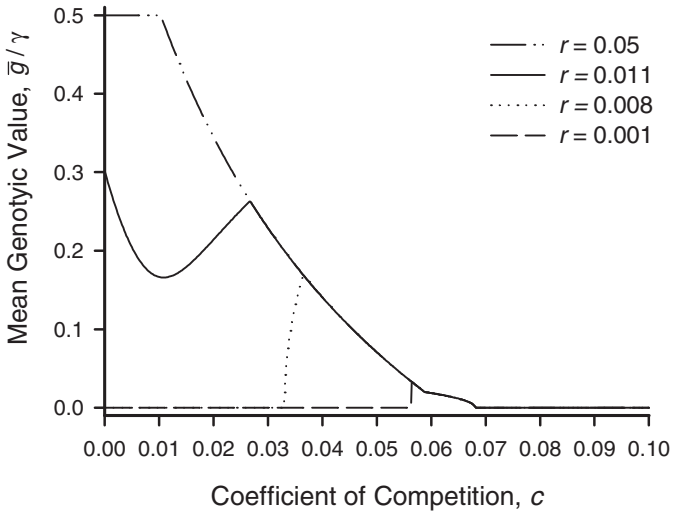

Figure 4: Equilibrium mean phenotype relative to the average locus effect γ as a function of the strength of competition, c. The parameters are the same as in figure 3B.

always occurs if (20) holds. However, the genotypes with the most extreme effects do not necessarily have the maximum fitness. They do so (i.e., in addition to (21), $w(\gamma_1) < w(\gamma_1 + \gamma_2)$ holds) for all values of r if and only if

$$c > \frac{s}{1 - (7 + 2e + e^2)s} \,. \tag{22}$$

Hence, a sufficient condition for the fitness function to be U-shaped for any choice of the genetic parameters e and r is

$$c > \frac{s}{1 - 10s} \,. \tag{23}$$

Disruptive selection may actually occur for weaker frequency dependence than suggested by the above analysis. Numerical results indicate that, as in the case of loci of equal effects, disruptive selection always occurs if the interior equilibria with $D = 0$ are stable; actually, with linked loci it may occur for even smaller values of c, namely when the single-locus polymorphisms are still stable. An example is presented in figure 5. It displays the equilibrium fitnesses of the phenotypes (at one of the at most two stable equilibria) for a sequence of different values of c. The figure legend contains the information which equilibrium is stable at each of these values.

Closer examination shows that for the parameter values on which figure 5 is based, disruptive selection occurs if $c > 0.058$ and the extreme genotypes have highest fitness if $c > 0.076$. As expected, both of these critical values are smaller than the analytically derived sufficient conditions. Indeed, (20) and (22) give the respective values 0.068 and 0.080.

The conditions (20) or (22) giving rise to disruptive selection are quantitatively similar to the condition $\sigma_k^2 > \sigma_\alpha^2$ resulting from Slatkin's (1979) model (cf. the

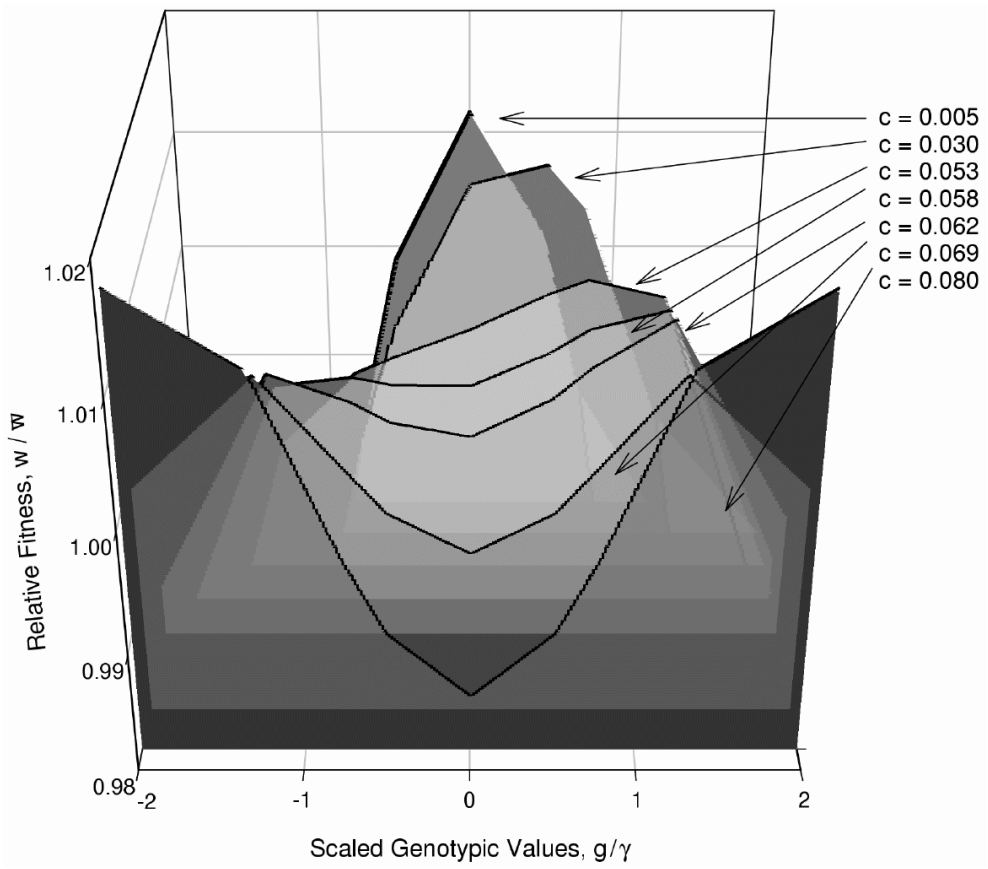

Figure 5: Equilibrium fitnesses (at one of the at most two stable equilibria) of the phenotypes relative to mean fitness for a sequence of different values of c. The fixed parameters are $s = 0.05$, $e = 0.025$, and $r = 0.011$. The following indicates which equilibrium is stable for a given c: $c = 0.005$: the pair of asymmetric equilibria ($D < 0$); $c = 0.030$: the pair of single-locus polymorphisms; $c = 0.053$: the pair of single-locus polymorphisms; $c = 0.058$: the pair of single-locus polymorphisms; $c = 0.062$: the pair of interior equilibria with $D = 0$; $c = 0.069$: the symmetric equilibrium ($D > 0$); $c = 0.080$: the symmetric equilibrium ($D > 0$).

section Relations Between the Models) or models of character displacement (e.g., Roughgarden 1976, Slatkin 1980, Brown and Vincent 1987). Like the inclusion of concrete genetics may lead to deviations from this simple condition, so does the inclusion of spatial resource heterogeneity (Day 2000).

In practice, fitnesses of quantitative traits are often determined by performing a least-squares approximation to the measured fitnesses by a polynomial of low degree, typically linear or quadratic. (More precisely, linear or quadratic selection gradients or differentials are determined; cf. Lande and Arnold 1983, Kingsolver et al. 2001.) Therefore, we investigated under which conditions disruptive selection can actually be detected by such a method. This will be the case, if the approximating quadratic polynomial has a positive leading coefficient. By numerical examples we found positive leading coefficients whenever the symmetric equilibrium satisfies $D > 0$, i.e., if (20) holds. In particular, it is not necessary that the stronger conditions (22)

or (23) are fulfilled in order to be able to detect disruptive selection. If c is in the range where the polymorphic equilibria with $D = 0$ are stable, then the resulting fitness function is very flat and the approximating polynomial may have positive or negative leading coefficient. Since the range of c-values for which this occurs is rather small, we can conclude that disruptive selection should be almost always detectable if the frequency-dependent fitness effect of competition is strong enough to maintain both loci polymorphic and in nonnegative linkage disequilibrium. Thus, it should be almost always detectable if it leads to an equilibrium configuration that cannot be explained by stabilizing selection alone. A similar conclusion is likely to hold for the cubic spline technique of Schluter (1988) which should be more sensitve in detecting disruptive selection.

Non-Maximization of Fitness

It has long been known that mean fitness can decrease in multilocus systems, and mean fitness is usually not maximized at equilibrium (Kojima and Kelleher 1961, Ewens 1979). It is also an old hat that under frequency-dependent selection there may be initial conditions leading to a steady decrease of mean fitness (Wright 1948, Ewens 1979). Also other optimization criteria may easily fail under frequency- and density-dependent selection (cf. Nagylaki 1979, Asmussen 1983, Day and Taylor 1996). Often, these facts are more or less ignored. Particularly in ecological modelling and life-history theory, optimization-of-fitness arguments are frequently used and the evolutionary dynamics is considered as being adaptive. The present model shows that such an approach may be highly misleading in an ecologically and genetically reasonable context.

Table 5 presents numerical data on the mean fitness (relative to the maximum possible one) at the single-locus polymorphisms ($\bar{\omega}_{\mathrm{bd}}$) and at the symmetric equilibrium ($\bar{\omega}_{\mathrm{sym}}$), as well as the proportion of the state space in which mean fitness is higher than at the stable equilibrium. In none of the numerical examples does a stable equilibrium have maximum possible fitness (though sometimes it is very close to). Actually, this seems to be the case for all parameter combinations, unless special relations are satisfied. Indeed, for fixed but arbitrary coordinates y and z, mean fitness $\bar{w}$ is a monotone increasing function in x if (and only if)

$$c > \frac{s}{2 - 11s - e^2 s} \; . \tag{24}$$

Therefore, $\bar{w}$ attains its maximum at the boundary of the simplex and, as shown by a simple calculation, always at the point $p_1 = p_4 = \frac{1}{2}$. With recombination, however, this can never be an equilibrium. If

$$c < \frac{s}{2 - s - 3e^2 s} \; , \tag{25}$$

then mean fitness is monotone decreasing in x and the maximum is again attained at the boundary, namely at $p_2 = p_3 = \frac{1}{2}$. Also this point can never be an equilibrium.

Table 5. Equilibrium mean fitnesses.[1]

		$\bar{\omega}_{\rm bd}$	$\bar{\omega}_{\rm sym}$	higher $\bar{\omega}$
$c = 0.02$	$r = 0.001$	0.993	**1.000**	0.003
	$r = 0.008$	0.993	**0.997**	0.02
	$r = 0.5$	**0.993**	0.987	0.10
$c = 0.03$	$r = 0.001$	0.995	**0.997**	0.22
	$r = 0.008$	0.995	**0.998**	0.14
	$r = 0.5$	**0.995**	1.000	0.31
$c = 0.04$	$r = 0.001$	0.974	**0.971**	0.48
	$r = 0.0037$	0.974	**0.974**	0.39
	$r = 0.0063$	0.974	**0.977**	0.34
	$r = 0.0069$	**0.974**	0.977	0.33
	$r = 0.008$	**0.974**	0.978	0.30
	$r = 0.5$	**0.974**	0.988	0.10
$c = 0.07$	$r = 0.001$	0.917	**0.956**	0.13
	$r = 0.008$	0.917	**0.953**	0.14
	$r = 0.5$	0.917	**0.951**	0.15

[1]The column entitled $\bar{\omega}_{\rm bd}$ contains the mean fitnesses at the boundary equilibrium (with the major locus polymorphic) relative to the maximum possible fitness in the simplex (i.e., $\bar{\omega}_{\rm bd} = \bar{w}_{\rm bd}/\bar{w}_{\rm max}$). The column $\bar{\omega}_{\rm sym}$ contains the (relative) mean fitness at the symmetric equilibrium, and the column entitled higher $\bar{\omega}$ contains the proportion of the simplex that has higher mean fitness than the stable equilibrium. Stable equilibria are indicated by bold numbers. The strength of stabilizing selection is $s = 0.05$ and the disparity of effects is $e = 0.25$.

It is only for intermediate values of c that the maximum mean fitness is attained in the interior of the simplex; then typically on the symmetric line $\{p_1 = p_4,\ p_2 = p_3\}$. For the parameters of table 5 ($s = 0.05$, $e = 0.25$), this range is $0.0256 < c < 0.0346$. However, the parameters c, e, and r have to fulfill a specific relation in order that mean fitness is maximized at the symmetric equilibrium. In the case $c = 0.03$ of the table, the symmetric equilibrium is very close to, but not at, the position of maximum fitness. Particularly remarkable is the case $c = 0.04$ and $r = 0.001$, in which mean fitness is maximized if $p_1 = p_4 = \frac{1}{2}$, but the (globally) stable symmetric equilibrium has coordinates $p_1 = p_4 = 0.019$. Hence, less than 4% instead of all gametes, as suggested by optimality considerations, have large effects and the stable equilibrium is not located near the position of maximum mean fitness but at the opposite end of the state space. As the table also shows, the proportion of the simplex in which mean fitness is higher than at the stable equilibria may be rather large.

Even more interestingly, the table shows that for $c = 0.02$, 0.03, 0.04, equilibria may be stable at which mean fitness is lower than at other, unstable, equilibria. The most striking example is that for $c = 0.04$, when it is only in the range $0.0037 < r < 0.0063$ that the stable equilibrium has higher mean fitness than all other equilibria.

The boundary equilibrium has lower mean fitness than the symmetric equilibrium whenever it is stable (which is the case if $c \geq 0.0069$). In the range $0.0063 < r < 0.0069$, the asymmetric equilibria are stable and have mean fitnesses between those of the symmetric and the boundary equilibria. Thus, changing a parameter in this model, for instance r, may lead to a switch in the stability properties of equilibria in the opposite direction of what would be expected, namely an equilibrium with relatively high mean fitness may loose its stability and an equilibrium with lower mean fitness may gain it.

Discussion

The interaction of stabilizing selection, intraspecific competition, and recombination leads to a surprisingly complex equilibrium structure which is illustrated in figures 1 and 2. In the absence of frequency dependence ($c = 0$), stabilizing selection alone can maintain one or a pair of stable two-locus polymorphisms if linkage is sufficiently tight (Gavrilets and Hastings 1993). Such polymorphisms always exhibit negative linkage disequilibrium. Interestingly, whenever such polymorphisms are maintained in the absence of, or for weak, frequency dependence and the frequency-dependent effect of competition (c) increases, then these polymorphisms are driven to the boundary and for a range of values of c a pair of single-locus polymorphisms is stable, but no two-locus polymorphism. Thus, for tightly (but not necessarily very tightly) linked loci, always a loss in the degree of polymorphism occurs. If c is increased further, then eventually a pair of stable equilibria bifurcates from the boundary equilibria, which become unstable, moves into the interior and at the center of the simplex merges with the symmetric equilibrium if $c = c_2$ (20). For all larger values of c, the symmetric equilibrium is globally stable and exhibits positive linkage disequilibrium. If linkage is tight and the frequency-dependent effect of competition strong, then high positive linkage disequilibrium is maintained.

For sufficiently high recombination rates, pure stabilizing selection maintains both loci monomorphic if their effects are similar, and it maintains the major locus polymorphic if the effects differ by more than a factor of two. Weak frequency dependence does not lead to a qualitative change. For loci of similar effects, moderate frequency dependence makes the major locus polymorphic. As in the case of tight linkage, increasingly strong competition then drives these boundary equilibria into the interior, and eventually a stable symmetric equilibrium with positive linkage disequilibrium is maintained. This holds for loci of any effects.

The striking phenomenon that with linked loci, interior equilibria are driven to the boundary as c increases, and then, at a larger value of c that is independent of r (at $c = c_1$, [A.5]), another type of equilibria moves inward and merges with the symmetric equilibrium, which becomes stable thereafter, requires explanation. Why is there no 'path' of stable *interior* equilibria connecting the stable equilibria with $D < 0$ and the stable symmetric equilibria with $D > 0$? If stabilizing selection maintains a two-locus polymorphism, which requires low recombination, then negative linkage disequilibrium is maintained because the genotypes closest to the optimum, in particular the double heterozygotes, are selectively favored. This leads to an overrepresentation of gametes of small effects. By contrast, competition

favors the genotypes with the most extreme effects, hence also the gametes with large effects, and recombination produces the gametes with small effects, which are selected against. Therefore, competition promotes positive linkage disequilibrium, and its degree is inversely related to the recombination rate. The genotypes that are strongest selected against by competition are the double heterozygotes whose phenotype coincides with the optimum 0. Thus, they are sensitive even to weak competition, when overdominance may still be present but is no longer strong enough to maintain both loci polymorphic because recombination produces too many genotypes of low fitness. By contrast, if the single-locus polymorphisms are stable, then the double heterozygotes are not present in the population. Most interestingly, there is a large range of parameters for which this latter equilibrium configuration is stable, but selectively unfavorable, i.e., mean fitness at the (stable) boundary equilibria is lower than at the (unstable) symmetric equilibrium. Thus, although a symmetric interior equilibrium would be selectively favored, recombination breaks up too many genotypes of high fitness, so that under the combined action of selection and recombination only the single-locus polymorphisms can be stable. Only for a strong frequency-dependent effect of competition ($c > c_1$, cf. [A.5], [A.10]) are the genotypes with very large or extreme effects sufficiently much favored that they are maintained in the population despite stabilizing selection. Under the slightly stronger, but much simpler, condition $c > c_2$ (20), there is a unique stable two-locus polymorphism with positive linkage disequilibrium.

The present analysis shows that competition inducing strong frequency dependence ($c > c_2$) is a potent force to maintain high levels of additive genetic variation. It only partially confirms the results of Bulmer (1974, 1980) and Slatkin (1979) of a threshold-like dependence of the genetic variance on the strength of competition. Their analyses were based on various much simpler genetic models assuming a Gaussian phenotype distribution. In the present model a threshold-like dependence occurs only if the loci have similar effects and are loosely linked. However, for moderately or tightly linked loci a new phenomenon is observed. There is always an intermediate range of values c, for which stronger frequency dependence leads to a loss in the degree of polymorphism, i.e., stable interior equilibria are driven to the boundary. During this process also additive genetic variance is lost but the effect may be too weak to be of practical importance. In contrast to the models of Bulmer and Slatkin, in our model the mean equilibrium phenotype does in general not coincide with the fitness optimum of stabilizing selection, but may deviate substantially from it (figure 4). Also the distribution of genotypic values is bimodal in our model if the frequency dependence is strong enough to induce positive linkage disequilibrium ($c > c_2$).

It is yet unknown to what extent these phenomenona occur in multilocus systems. A study of pure stabilizing selection has shown that with four or more additive loci the probability of a stable polymorphism involving two or more loci is very low if the effects of the loci and the recombination rates are drawn randomly from uniform distributions (Bürger and Gimelfarb 1999). If one, two, or perhaps three linked loci of major effect contribute to a trait, and if these loci are assisted by a number of loci of minor effect, then phenomena such as those discovered here might well be common. For weak frequency dependence the equilibrium structure will be

determined mainly by stabilizing selection. But as the frequency-dependent effect of competition increases, genotypes with extremer values will be selectively favored, i.e., genotypes consisting of an increasing number of either plus or minus alleles. Eventually, the two most extreme genotypes will have the highest fitness, and they are composed of either only plus or minus alleles. Recombination will produce all other genotypes. Thus, it seems likely that a cascade of bifurcations will occur as the frequency-dependent effect of competition increases, during which more and more loci become polymorphic and linkage disequilibrium increases. The extension of the present model to multiple loci, and its analysis, will be the subject of future study.

Let us now briefly discuss the relation to the work of Loeschcke and Christiansen (1984). Their model is rather similar to the present one, except that they use a Lotka-Volterra-like functional form for fitness, that stabilizing selection is modeled by a Gaussian function not a quadratic, and that they restrict their attention to strong competition relative to stabilizing selection (i.e., $\sigma > W$ in their notation; cf. the section Relations Between the Models). Although this assumption of strong competition is quantitatively slightly different from our assumption $c > c_2$, in their model it restricts the parameter range to the one for which in the present model the symmetric equilibrium with positive linkage disequilibrium is (globally) stable. Thus, they do not study the parameter range corresponding to our $c < c_2$ on which most of the present focus has been. Because Loeschcke and Christiansen assume a Gaussian fitness function, they observe a richer equilibrium structure in that case. The reason is that, even in the absence of frequency dependence, i.e., for pure stabilizing selection, the model with a quadratic fitness function may have different equilibrium patterns than the model with a Gaussian fitness function, because in the first model the range of admissible phenotypes is restricted by the requirement of positive fitness. Thus, for quadratic selection the fitness function is concave on the range of phenotypic values, but not for Gaussian selection. Indeed, for a Gaussian fitness function and with strong selection (so that there are phenotypes in the 'tails' of the Gaussian function), up to three symmetric equilibria can exist, two of which may be simultaneously stable: one with negative, the other with positive linkage disequilibrium (Gavrilets and Hastings 1994, Gimelfarb 1996). This is markedly different from quadratic selection, where an equilibrium with positive linkage disequilibrium never exists. Even if recombination is strong relative to selection, the Gaussian model has a more complex equilibrium structure than the quadratic (Nagylaki 1989). A comprehensive analysis of the two-locus model with Gaussian stabilizing selection has been performed only recently (Willensdorfer 2002, Willensdorfer and Bürger, unpublished). This complex equilibrium structure under strong Gaussian selection persists if intraspecific competition is added, and most of the article of Loeschcke and Christiansen (1984) is devoted to study this interaction. Their use of the Lotka-Volterra approach instead of the present one apparently makes little difference.

In one aspect the work of Loeschcke and Christiansen (1984) is more general than ours: they also explore the case where the fitness optimum is displaced from the middle of the phenotypic range. Depending on the magnitude of this displacement, this may lead to directional selection. In this case they show that intraspecific competition has little influence on the equilibrium structure. It is very likely that

this general conclusion will be also valid for the present model.

It has long been known that in multilocus models mean fitness is in general not maximized at a stable equilibrium and evolution is not necessarily adaptive (e.g., Kojima and Kelleher 1961, Moran 1964, Ewens 1979). However, often this phenomenon has been ignored or considered as being irrelevant. Here we have shown that in an ecologically important context maximization of mean fitness at equilibrium almost always fails. For a broad range of parameters stable equilibria are far away from the location in the state space at which mean fitness is maximized, and their fitness may be substantially lower. For instance, whenever $c > s/(2-11s-e^2s)$ (24), mean fitness is maximized on the boundary of the simplex at $p_1 = p_4 = \frac{1}{2}$. Thus, it would be selectively most favorable to have only the extreme gametes in the population, both at the same frequency. However, if $c < c_2$ (20), then the stable equilibria satisfy $p_1 + p_4 < \frac{1}{2}$, hence the actual proportion of extreme gametes is less than 50%. If in addition linkage is tight, $p_1 + p_4$ may become extremely small at the (unique) stable equilibrium, which then is located near the boundary of the state space opposite to where mean fitness is maximized. In such a case, the mean fitness may be higher than at the stable equilibrium on nearly 50% of the state space. As expected, the stable equilibrium is in general not a critical point of the fitness function. There is also a remarkably large range of parameters in which equilibria are stable that have lower mean fitness than other, unstable, equilibria.

Therefore, this simple model shows that the evolutionary dynamics in ecological systems with frequency-dependent selection acting on a genetically determined trait may be highly nonadaptive, and critical points of the fitness surface bear little relevant information about the dynamics or equilibrium properties of the model. However, even if a stable polymorphism coincides with a critical point of the fitness surface, methods relying on the invasion analysis of a rare mutant in a monomorphic population may be insufficient for deriving the correct evolutionary properties of this equilibrium (Christiansen 1991). It should be also kept in mind that the genetics in this model is very simple, because the trait is assumed to be determined additively, i.e., without dominance or epistasis in gene effects. The inclusion of such (biologically likely) genetic complications, as well as the consideration of more loci, could easily lead to more complex behavior.

Disruptive selection is generally considered as being an important agent in promoting genetic variation and a necessary prerequisite for evolutionary diversification such as character divergence or speciation. Although disruptive selection on quantitative traits is observed in nature, its frequency in relation to stabilizing or directional selection has not yet been firmly established (Endler 1986, Kingsolver et al. 2001). A well documented example is that of disruptive selection on bill characters in the African finch *Pyrenestes* (Smith 1990, 1993), where two morphs differ substantially in lower mandible width and, to a lesser extent, in some related characters. Apparently, these morphs are randomly breeding with respect to these traits. Disruptive selection is probably related to seed quality, because large morphs feed more efficiently on a hard-seeded species of sedge and small morphs on a soft-seeded species. The trait(s) under putative disruptive selection (as determined by the cubic spline technique of Schluter, 1988) shows a distinct bimodal distribution. However, it is unlikely that this case of disruptive selection can be explained in terms

of a model like the present one, because there is no continuous resource spectrum, but there simply are two very different types of seed available. Hence, there is no competition between similar phenotypes that would induce frequency-dependent fitnesses. Therefore, and because the environment in which these finches live appears to be rather constant over many years, it seems likely that a model with a fixed, frequency-independent bimodal fitness function is appropriate for describing selection. There is also evidence that the bill-size polymorphism is caused by a single autosomal diallelic locus with complete dominance for large-bill. This could not be explained by the present model in which no or only very weak disruptive selection occurs if a single locus is polymorphic and the distribution of the trait is unimodal. Extension of the model to include dominance at the major locus could lead to a different behavior.

For the present model, which involves a conflict of selective forces – stabilizing versus disruptive selection caused by frequency dependence – we have shown that empirically detectable disruptive selection on the phenotypic level occurs if the frequency-dependent effect of competition is strong enough to maintain a stable polymorphism, in particular, whenever positive linkage disequilibrium is maintained. As disruptive selection may occur for a wider parameter range, the conditions to observe disruptive selection, even if counterbalanced by stabilizing selection, are not restrictive as long as competition has an appreciable frequency-dependent effect on fitness. This suggests that intraspecific competition is an important factor in maintaining genetic variation of a quantitative trait only if disruptive selection is actually observed.

It has been shown for purely ecological models that different assumptions about competition or the inclusion of spatial heterogeneity may influence the conditions giving rise to disruptive selection (Brown and Pavlovic 1992, Day 2000). The extent to which multilocus genetics would affect our conclusion remains to be explored. However, as the above example suggests, the sole observation of disruptive selection should not be taken as evidence for the action of frequency-dependent selection. Of course, our findings and conclusions do not question the possibility that frequency-dependent selection may be responsible for the high genetic variability of some ecologically important traits. More studies, both empirical, to determine the frequency and the causes of disruptive selection in nature, and theoretical, to explore the generality of the present results, would be needed.

References

Abrams, P.A., Harada, Y., and H. Matsuda. 1993. On the relationship between quantitative genetic and ESS models. Evolution 47:982-985.

Asmussen, M.A. 1983. Density-dependent selection incorporating intraspecific competition. II. A diploid model. Genetics 103:335-350.

Brown, J.S., and N.B. Pavlovic. 1992. Evolution in heterogeneous environments: effects of migration on habitat specialization. Evolutionary Ecology 6:360-382.

Brown, J.S., and T. Vincent. 1987. Coevolution as an evolutionary game. Evolution 41:66-79.

Bulmer, M.G. 1974. Density-dependent selection and character displacement. American Naturalist 108:45-58

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

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

Bürger, R. 2002. Additive genetic variation under intraspecific competition and stabilizing selection: A two-locus study. Theoretical Population Biology, in press.

Bürger, R., and Gimelfarb, A. 1999. Genetic variation maintained in multilocus models of additive quantitative traits under stabilizing selection. Genetics 152:807-820.

Charlesworth, B. 1993. Natural selection on multivariate traits in age-structured populations. Proceedings of the Royal Society of London B 251:47-52.

Christiansen, F.B. 1991. On conditions for evolutionary stability for a continuously varying character. American Naturalist 138:37-50.

Christiansen, F.B., and T.M. Fenchel. 1977. Theories of Populations in Biological Communities. Springer Verlag, Berlin Heidelberg New York.

Christiansen, F.B., and V. Loeschcke. 1980. Evolution and intraspecific exploitative competition. II. One-locus theory for small additive gene effects. Theoretical Population Biology 18:297-313.

Clark, B. 1972. Frequency-dependent selection. American Naturalist 106:1-13.

Cockerham, C.C., Burrows, P.M., Young, S.S., and T. Prout. 1972. Frequency-dependent selection in randomly mating populations. American Naturalist 106:493-515.

Day, T. 2000. Competition and the effect of spatial resource heterogeneity on evolutiionary diversification. American Naturalist 155:790-803.

Day, T., and P.D. Taylor. 1996. Evolutionary stable versus fitness maximizing life histories under frequency-dependent selection. Proceedings of the Royal Society of London B 263: 333-338.

Dieckmann, U. 1997. Can adaptive dynamics invade? Trends in Ecology and Evolution 12:128-131.

Endler, J.A. 1986. Natural Selection in the Wild. Princeton University Press.

Ewens, W.J. 1979. Mathematical Population Genetics. Springer Verlag, Berlin Heidelberg New York.

Fisher, R.A. 1930. The Genetical Theory of Natural Selection. Oxford: Clarendon Press.

Gavrilets, S., and A. Hastings. 1993. Maintenance of genetic variability under strong stabilizing selection: a two-locus model. Genetics 134: 377-386.

Gavrilets, S., and A. Hastings. 1994. Maintenance of multilocus variability under strong stabilizing selection. Journal of Mathematical Biology 32:287-302.

Gimelfarb, A. 1996. Some additional results about polymorphisms in models of an additive quantitative trait under stabilizing selection. Journal of Mathematical Biology 35:88-96.

Hill, W.G., and P. Keightley. 1988. Interrelations of mutation, population size, artificial and natural selection. Pages 57-70 *in* Weir, B.S., Eisen, E.J., Goodman, M.M., and Namkoong, G. (eds.) Proceedings of the Second International Conference on Quantitative Genetics. Sinauer, Sunderland, MA.

Hofbauer, J., and K. Sigmund. 1998. Evolutionary Games and Population Dynamics. Cambridge University Press.

Iwasa, Y., Pomiankowsi, A., and S. Nee. 1991. The evolution of costly mate preferences. II. The "handicap" principle. Evolution 45:1431-1442.

Karlin, S., and M.W. Feldman. 1970. Linkage and selection: two locus symmetric viability model. Theoretical Population Biology 1:39-71.

Kingsolver, J.G., H.E. Hoekstra, J.M. Hoekstra, D. Berrigan, S.N. Vignieri, C.E. Hill, A. Hoang, P. Gibert, and P. Beerli. 2001. The strength of phenotypic selection in natural populations. American Naturalist 157:245-261.

Kojima, K., and T.M. Kelleher. 1961. Changes of mean fitness in random-mating populations when epistasis and linkage are present. Genetics 36:527-540.

Lande, R. 1975. The maintenance of genetic variability by mutation in a polygenic character with linked loci. Genetical Research 26:221-235.

Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. Evolution 30:314-334.

Lande, R., S.J. Arnold. 1983. The measurement of selection on correlated characters. Evolution 37:1210-1226.

Loeschcke, V., and F.B. Christiansen. 1984. Evolution and intraspecific exploitative competition. II. A two-locus model for additive gene effects. Theoretical Population Biology 26:228-264.

Lynch, M., and B. Walsh. 1998. Genetics and Analysis of Quantitative Traits. Sinauer, Sunderland, Mass.

MacArthur, R., and R. Levins. 1967. The limiting similarity, convergence, and divergence of coexisting species. American Naturalist 101: 377-385.

Matessi, C., and M. Gatto. 1984. Does K-selection imply prudent predation? Theoretical Population Biology 25:347-363.

Matessi, C., and S.D. Jayakar. 1976. Models of density-frequency dependent selection for exploitation of resources. Pages 707-712 *in* S. Karlin and E. Nevo, eds. Population Genetics and Ecology. Academic Press, New York.

Matessi, C., and S.D. Jayakar. 1981. Coevolution of species in competition: a theoretical study. Proceedings of the National Academy of Sciences of the USA 78:1081-1084.

Maynard Smith, J. 1982. Evolution and the Theory of Games. Cambridge University Press.

Moran, P.A.P. 1964. On the nonexistence of adaptive topographies. Annals of Human Genetics 27:383-393.

Mousseau, T.A., and D.A. Roff. 1987. Natural selection and the heritability of fitness components. Heredity 58:181-197.

Nagylaki, T. 1979. Dynamics of density- and frequency-dependent selection. Proceedings of the National Academy of Sciences of the USA 76:438-441.

Nagylaki, T. 1989. The maintenance of genetic variability in two-locus models of stabilizing selection. Genetics 122:235-248.

Robertson, A. 1967. The nature of quantitative genetic variation. Pages 265-280 *in* Brink, R.A. (ed.) Heritage from Mendel. Madison: University of Wisconsin Press.

Roughgarden, J. 1976. Resource partitioning among competing species–a coevolutionary approach. Theoretical Population Biology 9:388-424.

Roughgarden, J. 1972. Evolution of niche width. American Naturalist 106: 683-718.

Schluter, D. 1988. Estimating the form of natural selection on a quantitative trait. Evolution 42:849-861.

Slatkin, M. 1979. Frequency- and density-dependent selection on a quantitative character. Genetics 93:755-771.

Slatkin, M. 1980. Ecological character displacement. Ecology 61:163-177.

Taper, M.L., and T.J. Case. 1992. Models of character displacement and the theoretical robustness of taxon cycles. Evolution 46:317-333.

Taylor, P.D., and T. Day. 1997. Evolutionary stability under the replicator and the gradient dynamics. Evolutionary Ecology 11:579-590.

Turelli, M. 1988. Population genetic models for polygenic variation and evolution. Pages 601-618 *in* Weir, B.S., Eisen, E.J., Goodman, M.M., and Namkoong, G. (eds.) Proceedings of the Second International Conference on Quantitative Genetics. Sinauer, Sunderland, MA.

Willensdorfer, M. 2002. A Two-Locus Model of Gaussian Stabilizing Selection. Master thesis, University of Vienna.

Wilson, D.S., and M. Turelli. 1989. Stable underdominance and the evolutionary invasion of empty niches. American Naturalist 127:835-850.

Wolfram, S. 1996. Mathematica, 3rd ed. Cambridge University Press.

Wright, S. 1935. Evolution in populations in approximate equilibrium. Journal of Genetics 30:243-256.

Wright, S. 1948. On the role of directed and random changes in gene frequency in the genetics of populations. Evolution 2:279-294.

A Appendix: Conditions for the Existence and Stability of Equilibria

It is straightforward to show that if $(\hat{p}_1, \hat{p}_2, \hat{p}_3, \hat{p}_4)$ is an equilibrium, then $(\hat{p}_4, \hat{p}_3, \hat{p}_2, \hat{p}_1)$ is also an equilibrium, and both have the same stability properties. In terms of the coordinates (x, y, z) this means that the simultaneous transformation $y \to -y$ and $z \to -z$ preserves the property of being an equilibrium, as well as the stability properties of this equilibrium. Thus, all, except symmetric ($y = z = 0$), equilibria coexist in pairs.

Monomorphic Equilibria

There always exist the four corner equilibria at which both loci are monomorphic. Of these, the equilibria $\hat{p}_1 = 1$ and $\hat{p}_4 = 1$, i.e., fixation of one of the gametes with large genotypic effect (A_1B_1, A_2B_2), are always unstable. Analytical computation of the eigenvalues shows that the equilibria $\hat{p}_2 = 1$ (fixation of A_1B_2) and $\hat{p}_3 = 1$ (fixation of A_2B_1) are locally asymptotically stable if and only if the following conditions are satisfied:

$$r \geq r_0 = \frac{4e^2(c+s)}{4e^2c+1} \tag{A.1a}$$

and

$$c \leq c_0 = \frac{s(1-3e)}{(1+e)[1-s(1-e)^2]} \; . \tag{A.1b}$$

It may be noted that $r_0 = r_0(s, c, e)$ is an increasing function in each of the variables s, c, and e (in c, because $4e^2s \leq 1$ holds by assumption). For $0 \leq e \leq \frac{1}{3}$, $c_0 = c_0(s, c, e)$ is increasing in s and decreasing in e.

Condition (A.1b) shows that the monomorphic equilibria can never be stable if $e > \frac{1}{3}$ nor if $c > s/(1-s)$, where the latter inequality is obtained by setting $e = 0$ (recall that $e > \frac{1}{3}$ if the effects of the loci differ by more than a factor of two). In figures 1A and 1B, condition (A.1a) determines the lower boundary of the region of stability of the monomorphic equilibria, and (A.1b) determines the right boundary. Because r_0 is increasing in c, it attains its maximum at $c = c_0$. Hence validity of (A.1b) implies (A.1a) if

$$r \geq r_0(s, c_0, e) = r_{0,\max} = \frac{4e^2s(1-e)[2-s(1-e^2)]}{1-s+e+es(1+5e-13e^2)} \; . \tag{A.2}$$

Evaluation of r_0 at $c = 0$ informs us that the monomorphic equilibria are never stable if $r < 4e^2s$.

Numerical iteration of the recursion relations suggest that each of the two monomorphic equilibria is globally attractive for half of the state space whenever it is asymptotically stable. For equal effects ($e = 0$) this was proved in Bürger (2002).

Single-Locus Polymorphisms

There may exist up to four equilibria with one locus polymorphic and one locus monomorphic. Only the equilibria with the major locus (the A locus) polymorphic can be asymptotically stable. They are located on the edges $p_1+p_3 = 1$ or $p_2+p_4 = 1$ of the simplex, and exist if and only if

$$c > c_0 = \frac{s(1-3e)}{(1+e)[1-s(1-e)^2]} , \tag{A.3}$$

cf. (A.1b). Thus, they exist only if the monomorphic equilibria are unstable. If (A.3) is fulfilled, which is always the case if $e > \frac{1}{3}$, then there is a uniquely determined equilibrium on $p_1 + p_3 = 1$ with $0 < p_1 < 1$. It is the unique solution in $(0, 1)$ of the third-order equation

$$\begin{aligned} 4cs(1+e)^3p_1^3 - 6cs(3-e)(1+e)^2p_1^2 + 2(1+e)[s+c+2cs(3+2e-3e^2)]p_1 \\ + s(1-3e) - c(1+e)[1-(1-e)^2s] = 0 . \end{aligned} \tag{A.4}$$

The equilibrium coordinate $\hat{p}_1$ is an increasing function of c and always satisfies $\hat{p}_1 < \frac{1}{2}$.

Numerical computations show that this equilibrium is asymptotically stable if

$$c \le c_1 , \tag{A.5a}$$

where $c_1 = c_1(s, e)$ is the unique positive solution of

$$\begin{aligned} &c^3[(1-e^2)^2s^3(1-12e+e^2) + 7(1-e)^2s^2(9+14e+9e^2) - 16s(3-16e+3e^2) - 16] \\ &\quad + c^2s[(1-e)^2s^2(61+94e+61e^2) + 4s(13+94e+13e^2) - 32] \\ &\quad + 12cs^2[2s(4+7e+4e^2)+1] + 36s^3 = 0 \end{aligned} \tag{A.5b}$$

(this condition being explained below [A.10]), and if

$$r \ge r_1 = r_1(s, c, e) , \tag{A.6}$$

where r_1 can be determined by numerical evaluation of the eigenvalues. Apparently, r_1 decreases as c increases and determines the lower boundary of the region of stability of this pair of single-locus polymorphisms (see figure 1). At the left boundary, i.e., at $c = c_0$, we have $r_1(s, c_0, e) = r_{0,\max}$ (A.2) if $e \le \frac{1}{3}$, and $r_1(s, 0, e) = \frac{4}{3}es$ if $e > \frac{1}{3}$ (cf. Bürger 2000, p. 205). It may be noted that if $e \ge 0$, then $c_1 \ge c_0$ and equality holds if and only if $e = 0$.

By symmetry, analogous results are valid for the equilibrium at the edge $p_2+p_4 = 1$ which is obtained from (A.4) by substituting p_4 for p_1.

The single-locus polymorphic equilibria at the edges $p_1 + p_2 = 1$ and $p_3 + p_4 = 1$ exist if and only if

$$c > \frac{1+3e}{(1-e)[(1-s(1+e)^2)]} .$$

The right-hand side coincides with c_0 (A.1b) if $e = 0$. For $e > 0$, these equilibria exist only for larger values than those at the other two edges and are always unstable.

Two-Locus Polymorphisms

Analytically explicit determination of all interior equilibria seems to be impossible. Analytical calculations combined with numerical searches revealed that three classes of interior equilibria may exist and be stable: a symmetric equilibrium, a pair of asymmetric equilibria satisfying $D < 0$, and a pair of equilibria with $D = 0$.

Symmetric equilibria

There always exists one symmetric equilibrium, $\hat{p}_1 = \hat{p}_4$ and $\hat{p}_2 = \hat{p}_3$. It is the uniquely determined solution of the equation

$$32cs(1-e^2)^2p_1^3 + 2(1-e^2)[s - c(1+2r+7s-17e^2s)]p_1^2$$
$$-[r+s-e^2s-c(1+r-s+e^2(1+3r+8s)-9e^4s)]p_1 + \tfrac{1}{4}r(1+2ce^2) = 0$$

such that $0 \le p_1 \le \frac{1}{2}$.

If

$$c < c_2 = \frac{s}{1-5s(1+e^2)} , \tag{A.7}$$

then the coordinate $\hat{p}_1$ of the equilibrium satisfies $0 < \hat{p}_1 < \frac{1}{4}$, hence $D < 0$. In this case, the symmetric equilibrium is asymptotically stable if and only if

$$r \le r_2 = r_2(s,c,e) , \tag{A.8}$$

where r_2 can be determined by numerical evaluation of the eigenvalues (see figure 1). Iteration of the recursion relations suggests that then the symmetric equilibrium is globally stable. Because of the constraint $r \ge 0$, (A.8) can be satisfied only if $0 \le c \le c_1$; cf. (A.5b).

If $c = 0$, then r_2 can be determined explicitly, i.e.,

$$r_2(s,0,e) = \tfrac{2}{3}s(-1-e^2+\sqrt{1+14e^2+e^4}) ,$$

and the symmetric equilibrium can be proved to be asymptotically stable (see Bürger 2000, pp. 205–207, where a different notation is used). By continuity, this extends to small (positive) values of c. Apparently, r_2 is decreasing as a function of c, whence the symmetric equilibrium with $D < 0$ can be stable only if $r \le r_2(s,0,e)$.

If $c \ge c_2$, cf. (20), then $\hat{p}_1 \ge \frac{1}{4}$, hence $D \ge 0$, and apparently the symmetric equilibrium is globally asymptotically stable. It is important to note that $c_1 \le c_2$ always holds, and equality is obtained if and only if $e = 1$.

It can be shown that the position $\hat{p}_1$ of the symmetric equilibrium, and therefore the amount of linkage disequilibrium $\hat{D} = \hat{p}_1 - \frac{1}{4}$, is an increasing function of c. The absolute value of $\hat{D}$ increases with decreasing r. If $c > c_2$, then for every $s \ge 0$ and $r > 0$, $\hat{p}_1$ approaches an upper limit $< \frac{1}{2}$ as $c \to \infty$.

Asymmetric equilibria

In the absence of competition ($c = 0$), asymmetric interior equilibria exist and are asymptotically stable if $e > 0$ and

$$r_2(s,0,e) < r < \begin{cases} r_0(s,0,e) = 4e^2s & \text{if } e \le \frac{1}{3} , \\ r_1(s,0,e) = \frac{4}{3}es & \text{if } e > \frac{1}{3} . \end{cases}$$

They can be calculated explicitly (e.g., Bürger 2000, p. 205). Because of continuity, they also exist for sufficiently small $c > 0$. Unfortunately, for $c > 0$ their explicit calculation seems to be impossible. Numerical iteration of the recursion relations and numerical solution of the equilibrium conditions suggest that they are asymptotically stable whenever they exist, which is the case if and only if

$$r_2(s,c,e) < r < \begin{cases} r_0(s,c,e) & \text{if } c < c_0 \text{ and } e \leq \frac{1}{3}\,, \\ r_1(s,c,e) & \text{otherwise}\,, \end{cases} \tag{A.9}$$

is satisfied. Here, r_0, r_1, and r_2 are as in (A.1a), (A.6) and (A.8), respectively.

Equilibria with D = 0

If

$$c_1 < c < c_2\ , \tag{A.10}$$

then numerical calculations show that for arbitrary r a pair of interior equilibria exists that satisfy $D = 0$ (recall that c_1 and c_2 depend only on s and e). They are asymptotically stable whenever they exist. Since $D = 0$, their position is independent of r. For $e = 0$ the exact location of these equilibria can be computed and their asymptotic stability was proved in Bürger (2002). Because $c_1 = c_1(s,e)$ is an increasing function in e with $c_1(s,0) = c_0(s,0)$ and $c_1(s,1) = c_2(s,1)$, the range of values c for which these equilibria exist decreases to 0 as e increases to 1.

Using c as a bifurcation parameter, they enter the simplex if $c = c_1$ through the single-locus polymorphisms on the edges $p_1 + p_3 = 1$ and $p_2 + p_4 = 1$, apparently by an exchange of stability bifurcation (cf. the description of the bifurcations in figure 2A in the main text). This lower bound c_1 for their range of existence can be computed as follows: Because $D = 0$, the conditions for their existence can be reduced to two polynomial equations in y and z, one of degree three, the other of degree four. The equilibria are located on the boundary if and only if $z = y - 1$ (for one of them). Comparing the resulting equation with the defining equation of that boundary equilibrium, the coordinate y at which the bifurcation from the single-locus polymorphisms occurs can be calculated by solving a quadratic equation. Then simple algebra yields the condition (A.5b) for the corresponding c.

Apparently, none of the above described types of equilibria can be simultaenously stable, hence there are never more than two stable equilibria.