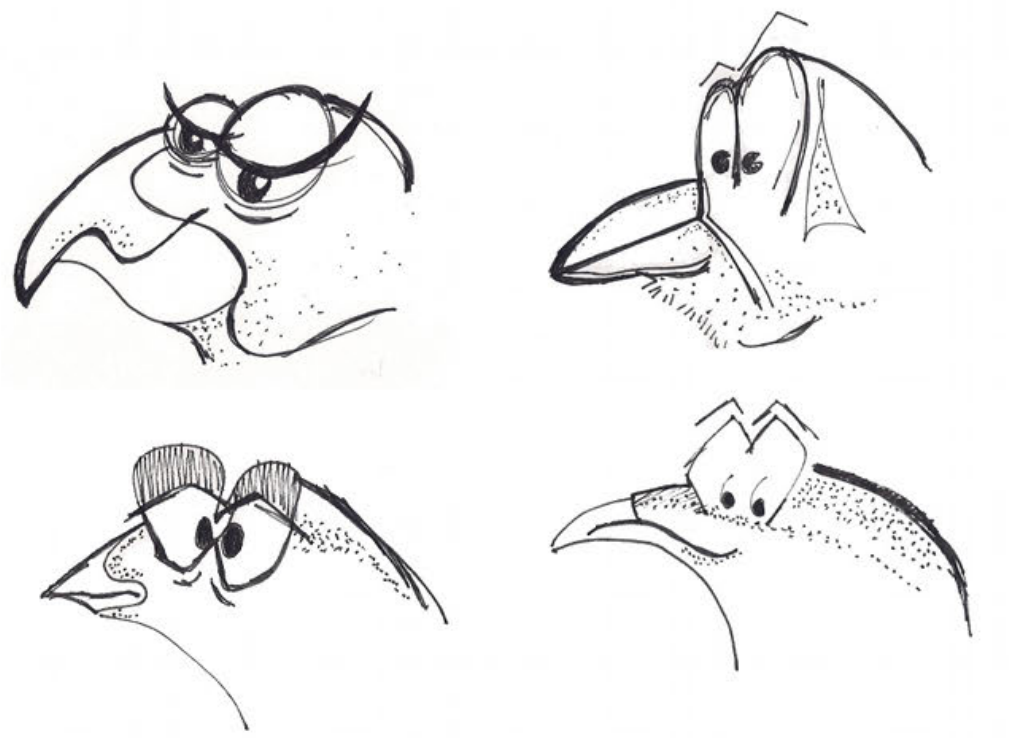
MATE CHOICE AND SPECIATION

PERSPECTIVES FROM ADAPTIVE DYNAMICS AND POPULATION GENETICS



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Mate choice and speciation:

perspectives from adaptive dynamics and population genetics

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List of Publications

This thesis is based on the following articles, which are referred in the text by their Roman numerals:

I Éva Kisdi and Tadeáš Přiklopil. 2011. **Evolutionary branching of a magic trait.** *Journal of Mathematical Biology 63: 361–397.*

II Tadeáš Přiklopil. 2012. **On invasion boundaries and the unprotected coexistence of two strategies.** *Journal of Mathematical Biology 64: 1137–1156.*

III Tadeáš Přiklopil. 2012. **Chaotic dynamics of allele frequencies in condition-dependent mating systems.** *Theoretical Population Biology 82: 109–116.*

IV Tadeáš Přiklopil, Éva Kisdi and Mats Gyllenberg. **The perfect female's sequential search for males and reproductive isolation by assortative mating.** *Manuscript.*

Author's contribution

I had the principal role in the development of the methods and analysis of the models as well as the writing of all the included articles.

Abstract

Speciation theory is undergoing a renaissance period, largely due to the new methods developed in molecular biology as well as advances in the mathematical theory of evolution. In this thesis, I explore mathematical techniques applicable to the evolution of traits relevant to speciation processes. Some of the theory is further developed and is part of a general framework in the research of evolution.

In nature, sister species may coexist in close geographical proximity. However, the question as to whether a speciation event has been a local event driven by the interactions (perhaps complex ones) of individuals that affect their survival and reproduction, has not yet been satisfactorily answered. This is the key issue I address in my thesis. The emphasis is given to the role of non-random mating in an environment where individuals experience diversifying ecological selection. Firstly, I investigate the role of assortative mating, and find that assortative mating works against the speciation process in the initial stages of diversification. However, once the population has diversified, ecological and sexual selection drives the population to a state of reproductive isolation. Secondly, I explore a scheme where individuals choose mates according to the level of adaptation of the mate. I find, that when the level of adaptation to the environment depends on the structure of the population in a frequency-dependent manner, the dynamics of the population may be highly complex and even chaotic. Furthermore, this setting does not facilitate reproductive isolation when mating happens across the habitats. However, if mate choice takes into account the survival and reproduction of the progeny, reproductive isolation can be maintained.

Finally, some advances are made in the theory of adaptive dynamics, which along with the theory of population genetics, has been a focal tool in this thesis. My contribution to adaptive dynamics is to resolve an open question on the coexistence of similar strategies near so-called singular points. Singular points play a central role in the theory of adaptive dynamics and their existence is essential to a continuous diversification process.

Contents

1 Motivation

In 1833, a British entomologist and archaeologist John Obadiah Westwood made the earliest known evaluation of global biodiversity. In his work, published in the Magazine of Natural History, he estimated how many insect species live on each plant species in England and extrapolated the figure across the globe (Westwood 1833, Zimmer 2011). Westwood wrote, "If we say $400,000$, we shall, perhaps, not be very wide of the truth". Indeed, he was not very wide of the truth, as up to date scientists have found a bit more than a million insect species. If we add the rest of the known eukaryotes (including few vertebrates such as $8,000$ reptiles, $10,000$ birds, $24,000$ fish and $5,000$ mammals) and prokaryotes, we get a total number of 1.4 million species (Bisby et al. 2012). However, the estimate of the total number of species across the world ranges between 3 and 100 million (Erwin 1983, May 1988, May 2010, Mora et al. 2011). Moreover, estimates of the total progeny of evolution range from 5 to 50 billion species (Raup 1991).

There are two points I want to make. First, the figures above show how tremendous a journey of continuous diversification, adaptation, speciation and extinction life has undergone to account for the great number of species there exist today and the even greater number of species having ever existed. In addition to the grand variation between species, individuals also within species may vary greatly (think of humans, for example, and the variety we come in) thus showing a remarkable biodiversity on our planet. This leads me to the second point, the variation in the observed life forms, and, if I may generalize, our compulsion to categorize every object around us. As Mayr noted (Mayr 1982), are species just simply theoretical constructs of the human mind or are they realities of nature? Or in the words of Coyne and Orr (2004), can assemblages of individuals be objectively partitioned into discrete units, or are species just subjective divisions of nature for human convenience? In *The Origin of Species*, Charles Darwin seemed to believe that species are not real:

> In short, we shall have to treat species in the same manner as those naturalists treat genera, who admit that genera are merely artificial combinations made for convenience. This may not be a cheering prospect; but we shall at least be freed from the vain search for the undiscovered and undiscoverable essence of the term species (Darwin 1859, p. 485, cited by Coyne and Orr 2004).

It must be noted, however, that Darwin took a different position on the subject in his later work (Darwin 1871), where he accepted the idea of discontinuity and even proposed that it might result from reproductive barriers (unpublished work, see Barret et al. 1987). Indeed, if we accept the concept of species, the definition for sexually reproducing species must include some restriction in reproduction between populations (we come back to the proposed species definitions later). Nonetheless, we must acknowledge the difficulty of the task to decide whether "species" are distinct or not

(and hence appreciate the earlier stance of Darwin and other more recent evolutionists such as J. B. S. Haldane) when considering, for example, the peculiar case of ring species. A well known example, especially to people living up North, is the circumpolar species ring of *Larus* gulls (Mayr 1942, Haffer 1982; but see Liebers et al. 2004). The Herring Gull *L. argentatus*, which lives primarily in Great Britain and Ireland, can hybridize with the American Herring Gull (living in North America), which can also hybridize with the East Siberian Herring Gull, the subspecies of which, *L. vegae birulai* can hybridize with *L. heuglini*, which in turn can hybridize with the Siberian Lesser Black-backed Gull. All four of these live across the north of Siberia. The last is the eastern representative of the Lesser Black-backed Gulls back in north-western Europe, including Great Britain. Now, the Lesser Black-backed Gulls (the species we ended up with) and Herring Gulls (the species we started with) are sufficiently different that they do not normally interbreed; thus the group of gulls forms a continuum except where the two ends meet in Europe. To put it simply, if population A interbreeds with population B which in turn interbreeds with population C but C doesn't interbreed with A, how many species are there?

Despite of such examples, biologists do now recognize that species are discrete entities and have proposed several species concepts, of which perhaps the following is often the most suitable:

> Species are groups of interbreeding natural populations that are reproductively isolated from other such groups (Mayr 1995).

In this work I will use a slightly looser definition, by adding that reproductive isolation needs to be substantial but not necessarily complete (Coyne and Orr 2004).

There are various reproductive barriers that can impede the exchange of genes, which can be divided into three categories: premating, postmating and prezygotic, and postzygotic. Examples include the differences in cross-attraction between members of different groups hence preventing them from initiating copulation (premating barriers), intrinsic problems with storage of gametes that cannot effect fertilization (postmating and prezygotic barriers), and the production of hybrids that suffer lower viability because they cannot find an appropriate ecological niche (postzygotic barrier).

Thus, why and how these barriers arise, and which ones have the leading role in speciation process? To answer "why", I will quote again Mayr (Mayr 1969, p.316), "The segregation of the total genetic variability of nature into discrete packages, so called species, which are separated from each other by reproductive barriers, prevents the production of too great a number of disharmonious incompatible gene combinations." Hence, the question why are there species is just reduced to applying the fundamental laws of natural selection. I am thus concerned only in the "which ones and how" part of the question, and attempt to answer it in two parts. In the first part, I will simply state that given enough time, and excluding the possibility of extinction, any pair of *geographically* isolated (i.e. allopatric) populations is likely to

evolve almost any reproductive barriers. This is true, simply, because there is no gene exchange between geographically isolated populations. Successive mutations of the genetic material will lead to the accumulation of genetic differences and affect the morphological, behavioral (and all other genetically based) properties of individuals. Time and the randomness of mutation events will take care of the rest (although, there are species that diverged 5 million years ago and can still produce viable and fertile hybrids, Wen 1999). The second part of the answer is concerned with the case where populations are not geographically isolated so that there is gene exchange, at least to some degree, between diverging populations. This is the motif of my Thesis.

2 Mate choice and reproductive isolation

Consider sexually reproducing populations that live in sympatry, that is, populations that live in close enough contact such that genes could be potentially exchanged freely by breeding. This scenario, in terms of geography, is the exact opposite to the case discussed above where no gene flow is possible due to complete geographic separation. Even if full sympatry (as well as full allopatry) is an oversimplification of real biological systems, it is a useful setting to study the evolution of reproductively isolating barriers. It is useful, because many closely related taxa do live in close proximity and can be approximated by assuming sympatry and hence has its value on its own; but also because by default it isolates essential mechanisms involved in the speciation process and hence clarifies their true roles.

In sympatric setting, a mechanism that reduces gene flow between populations is discriminative mate choice. In the past decade there is a strong consensus that mate choice has evolved as a response to disruptive ecological selection (to avoid producing unfit individuals) rather than as an arbitrary sexual preference. The main focus of this work is therefore on (female) mate choice and its consequences on the level of reproduction between incipient populations that undergo disruptive selection in various sympatric ecological settings. In earlier models of sympatric speciation it was assumed that ecological selection acts on a trait defined by one locus, and that another locus controls mate choice. However, recombination between the ecological and the mating locus hinders the non-random association of alleles at the two loci (linkage disequilibrium), and unless ecological selection is strong, speciation seems improbable (Felsenstein 1981). This problem is avoided when a trait under ecological selection and a trait affecting mate choice are pleiotropic expressions of the same gene(s). Such traits are called magic (Gavrilets 2004, Servedio et al. 2011), but despite their conspicuous name, an increasing amount of evidence indicates that they might be common (Servedio et al. 2011). A closely related concept to magic traits is a condition-dependent expression of sexual ornaments (Zahavi 1975, Grafen 1990, Iwasa et al. 1991, Servedio et al. 2011). Here, well adapted males are in a good condition which enables them to carry an ornament that reveals the quality of their genes, allowing females to evolve a preference associated to such traits.

As magic traits and traits with condition-dependent expression solve the issue of recombination vs. linkage disequilibrium, they may play an important role in the speciation process. Therefore, in this work I will study whether magic and condition-dependent traits enable reproductive isolation, and if they do, under which ecological conditions it is most likely.

3 The mathematics of speciation

In this work, the strength of reproductive isolation is investigated by studying the level of hybridization between incipient species. To gain a clearer insight into the relative strengths of various selective forces, I consider populations with only one (autosomal) locus that is under ecological selection. Supposing there are at most two coexisting alleles, say a and A, the populations contain only three genotypes: homozygotes aa, AA and heterozygotes aA. Because a limited interbreeding between the two homozygotes decreases the production of heterozygotes (recall Mendel), studying the level of hybridization (i.e. strength of reproductive isolation) is reduced to examining how many heterozygotes aA there are in the population. In more mathematical terms, we search for attractors where the frequency of heterozygotes is close to zero. Therefore, an essential framework I will use is *population genetics* (Ewans 2004, Hartl 2007). Population genetics, which utilizes the mathematical theory of dynamical systems, is concerned with allele frequency distribution and its change under the influence of the four main evolutionary processes: natural selection, mutation, genetic drift and gene flow. The mutation events I will link to another mathematical framework called *adaptive dynamics* (Metz et al. 1996, Geritz et al. 1998). Because I assume populations to be large, I will ignore the effect of drift.

I will first give a description of the life-cycle of the population to motivate the build-up of the dynamical system used throughout my work. The life-cycle of an individual is divided into an ecological selection phase and a mating phase. During the first phase, frequency-dependent selection acts on a continuous trait ϕ_g that is determined by genotype g. Frequency-dependence means that the survival of the individual depends not only on its own genotype but also on its interactions with other individuals and hence on the frequency distribution of the population. Denoting with v_g the frequency-dependent survival probability of genotype g and with $\bar{v}$ the average survival probability in the population, the frequency of g after ecological selection is $\tilde{P}_g = P_g w_g$, where $w_g = \frac{v_g}{\bar{v}}$.

Individuals that survive ecological selection enter the second phase of the life-cycle, the mating season. At this moment, I only assume that mate choice is correlated with the female and male genotype (which is above described to be the target of ecological selection, hence think for example of magic traits or condition-dependent traits from the previous section), and I denote with $Q_{g,h}\tilde{P}_h$ the probability that a female of genotype g and a male with genotype h mate and produce offspring. All the factors that deal with mate choice, for example sampling and criteria for accepting/declining

males for mating, are then contained in Q. Now, we can write the dynamical system that describes the change of genotype frequencies from generation t to generation $t+1$ as

$$P_r^{(t+1)} = \frac{1}{\bar{Q}} \sum_{g,h} \tilde{P}_g^{(t)} \tilde{P}_h^{(t)} Q_{g,h} R_{g,h\to r}, \tag{1}$$

where $\bar{Q} = \sum_{g,h} \tilde{P}_g^{(t)} \tilde{P}_h^{(t)} Q_{g,h}$ is the mean mating success and $R_{g,h\to r}$ denotes the probability that parents with genotypes g and h produce an offspring with genotype r according to the Mendelian rules.

The dynamical system (1) gives the dynamics of genotype frequencies on the so-called *ecological time-scale*. To include mutation events, which are considerably rarer events compared to events that happen on generation to generation basis, I need to couple (1) with a framework that considers longer time-scales. The current leading framework that deals with the *evolutionary time-scale* is adaptive dynamics (AD) theory (Metz et al. 1996, Geritz et al. 1998).

Adaptive dynamics provides results that describe the continuous change of a trait (also called a strategy; e.g. size of a bird's beak), which is subject to mutation and selection. The fate of a new mutation is determined by an invasion exponent (or invasion fitness function) which is defined as the expected growth rate of an initially rare mutant in the environment set by the currently existing traits (called residents). Before each mutation, the residents are assumed to reach their population dynamical/genetical attractor (given for example by system (1)). I will write the invasion fitness function of a mutation y in a resident population with strategy x, as

$$s_x(y). \tag{2}$$

If function (2) is positive, a mutant might invade the resident population x, and if it is negative it will not (it must be noted that the above notation can cause confusion if a resident strategy x defines several resident attractors, however, in this work all the resident attractors are unique.)

Using invasion fitness functions $s_x(y)$, AD finds and classifies special points in the trait space that could be described as decisive points in the evolution of the trait. These points are called singular strategies, and depending simply on the second order derivatives of $s_x(y)$, singular strategies might be, for example, evolutionary endpoints where no new mutations are able to invade the residents, or, evolutionary branching points (for the complete classification see Geritz et al. 1998). At the evolutionary branching point selection turns disruptive due to frequency-dependent fitness, enabling two distinct trait values to coexist and coevolve further away from each other.

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In Article **I** the framework of AD is applied to the evolution of alleles (as in Kisdi and Geritz 1999), where alleles are assumed to act additively on a magic trait ϕ. Supposing that the female mating preference (that acts on the magic trait) is fixed, we study the evolution of the magic trait itself, focusing on evolutionary paths that lead to limited production of heterozygotes and thus reproductive isolation. This contrasts to the previous work where the magic trait was fixed and the evolution of mating preference was investigated (Matessi et al. 2001, Pennings et al. 2008). Supposing that mutations have only a small effect on the magic trait, we investigate conditions for two things that need to happen for reproductive isolation to evolve: evolution to an evolutionary branching point (diversification process that guarantees polymorphism, see above), and further evolution to a polymorphic singularity where the homozygotes stop interbreeding.

Mating preference is based on the similarity of the female ϕ_g and male trait ϕ_h, such that at an encounter with a male the probability to accept him for mating is given by

$$\pi_{g,h} = \pi(\phi_g - \phi_h), \tag{3}$$

where π is assumed to be a twice continuously differentiable function and it attains its maximum at 0. Locally near 0, it is hence a concave function and it describes the fact that with increasing difference between their phenotypes the probability of accepting the male decreases. The width of the function indicates the strength of preference and is controlled by another loci which is expressed only in females. The narrower is the function, the stronger is the preference for males of her own type. Function (3) thus describes mating that is assortative with respect to the magic trait.

First we show that for arbitrary ecological selection (for arbitrary functions w that are twice continuously differentiable), functions of type (3) hinder evolutionary branching. The stronger is the mating preference, that is, the more concave function (3) is, the more it interferes with disruptive ecological selection that tries to diversify the magic trait. This is because according to (3) each mutation is being disfavored in males as initially the allele is rare and hence it deviates from the common resident allele. Therefore, common females discriminate against rare males and assortative mating has a stabilizing effect. Moreover, stronger assortative mating turns the polymorphism of alleles unprotected (i.e. rare alleles go extinct) or polymoprhism may even be lost. Unprotected coexistence is the topic of Article **II**.

Suppose now that the assortative mating and disruptive ecological selection are balanced such that evolutionary branching can happen (either assuming assortative mating to be weak or disruptive selection strong). From now on we need to specify functions π and w, because analyzing evolution further can not be done locally as near monomorphic singularities. We take π to be a Gaussian function and w will follow a version of the Levene model (Levene 1953) as adopted by Kisdi and Geritz (1999). It turns out that the evolution of the magic trait may reach a polymorphic evolutionary stable singularity where the homozygotes are reproductively isolated

only if disruptive selection is considerably strong. This is because assortative mating needs to be strong for the homozygotes to stop interbreeding, and therefore disruptive selection needs to be strong as well to allow the polymorphism to arise in the first place (via evolutionary branching). However, a polymorphic singularity with sufficient reproduction isolation does exist even if evolutionary branching is not possible. This motivates me to conclude with the following conjecture: if mating is described with functions of type (3), speciation seems the most plausible if assortative mating evolves only in the later stage of the diversification process, or if the effect of a mutation on the trait value is big so that the formation of polymorphism does not require evolutionary branching.

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In the previous section, strong assortative mating was said to cause unprotected coexistence of two alleles. Unprotected coexistence means that while the attractor in the interior of the population state space is stable, thus enabling the coexistence of two alleles, the boundary equilibria (or at least one of them) are stable as well and hence alleles near the boundary (fixation) equilibria are not protected from extinction. Article **II** gives, in terms of invasion fitness functions (2), a sufficient condition for the unprotected coexistence of two arbitrary one-dimensional strategies. In addition, it connects the condition to the categorization of singular strategies (Geritz et al. 1998; see above) and the Classification Theorem (Geritz 2005; see below) by showing that a particular degeneracy unfolds as unprotected coexistence.

Suppose the dynamics of strategies is given either by a discrete-time system

$$\begin{aligned} N^{t+1} &= G_\mu(x, E^t)N^t \\ M^{t+1} &= G_\mu(y, E^t)M^t \end{aligned} \tag{4}$$

or a continuous-time system

$$\begin{aligned} \dot{N}(t) &= F_\mu(x, E(t))N(t) \\ \dot{M}(t) &= F_\mu(y, E(t))M(t). \end{aligned} \tag{5}$$

where N, M are population densities of strategies x and y, respectively, F_μ, G_μ are some continuous and sufficiently smooth functions and $\mu \in \mathbb{R}^k$ is an auxiliary parameter. E denotes the environment that contains all factors that influence the population growth, including the effect what population itself has on the environment, and hence it may be a function of strategies x, y and population densities N, M (Metz et al. 1992, Gyllenberg and Metz 2001, Geritz 2005). Note that a wide class of dynamical systems can be written in the form (4) or (5), for example equation (1), when rewritten in

terms of allele frequencies instead of genotype frequencies (to get invariant boundaries of the population state space).

Article **II** rests on two theorems. The first theorem says that when two groups of individuals, one with strategy x and the other with strategy y, both loose or gain the ability to invade each other, then in the neighborhood of (x, y) they coexist in an unprotected way. In mathematical terms, when the boundary equilibria $(\hat{N}, 0)$ and $(0, \hat{M})$ change stability at the same values of x and y, then in the neighborhood of (x, y) there exists an interior stable equilibrium while at least one of the equilibria $(\hat{N}, 0)$ or $(0, \hat{M})$ are stable (see Article **II** for additional but minor technical conditions). The sufficient condition (with minor technical conditions) for unprotected coexistence is then

$$s_x(y) = 0 = s_y(x), \qquad \text{for } x \neq y. \tag{6}$$

Even though analytical conditions that satisfy (6) can sometimes be found (Article **II**; Levene model), we often need to rely on numerical methods. Moreover, the condition is only sufficient, and, doesn't tell us for instance the size of the parameter region where unprotected coexistence occurs. Nevertheless, solving (6) is substantially easier than seeking for interior equilibria in the whole parameter space. The true power of this theorem lies, however, in the fact that condition (6) comes for free when constructing so-called pairwise and mutual invasibility plots frequently used in adaptive dynamics (introduced in population genetics literature Christiansen and Loescke 1980, Motro 1982 and used e.g by Kisdi and Meszena 1993, 1995, Metz et al. 1996, Dieckmann 1997, Claessen and Dieckmann 2002, Doebeli et al. 2007). Mutual invasibility plots help in analyzing the mutation-selection process by indicating for a range of (x, y) values the sign of the invasion fitness function. Hence, near an intersection of the contour-lines $s_x(y) = 0$ and $s_y(x) = 0$, strategies x and y are in unprotected coexistence.

The second theorem is based on an observation that if contour-lines $s_x(y) = 0$ and $s_y(x) = 0$ intersect in some neighborhood of $x = y$, then the intersection (and unprotected coexistence) approaches a singular strategy x^* when the cross-derivative $D_{12}s_{x^*}(x^*) = \left[\frac{\partial s_x(y)}{\partial x \partial y}\right]_{x=y=x^*} \to 0$. This complements the Classification Theorem of Geritz (2005) which proves that unprotected coexistence does not exist near $x = y$ whenever $D_{12}s_{x^*}(x^*) \neq 0$.

I would like to note that the detection of unprotected (co)existence is important for at least two reasons. Firstly, detecting protected existence of populations has been dominating studies in finding parameter regions where the population is viable, thus neglecting possibly many ecological scenarios where the population can exist "stably and well" but in an unprotected way. With this I mean, that if the basin of attraction of the stable (unprotected) existing population is large, unprotected existence of the population and its extinction due to fluctuations that move it outside of the basin can't in fact be distinguished from protected existence of the population and its extinction due to environmental stochasticity. However (and secondly) when the basin of attraction is small, it can have disastrous consequences. The problem of

course is that the population appears to exist stably but when conditions change little past the critical point, extinction is inevitable.

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The assumption of assortative mating made in Article **I**, where female preference for males is based on the similarity of their ecological traits, has the difficulty that it allows maladapted females to prefer maladapted males. This motivates the assumption that female preference in fact acts on male traits that are honest indicators of his condition (Zahavi 1975, Grafen 1990, Møller and Jennions 2001, Kokko et al. 2003, Cotton et al. 2004, Andersson and Simmons 2006), thus enabling females to mate with males that are well adapted to the environment and potentially sire offspring that will be well adapted as well. Under disruptive selection, however, this setting may lead to matings across the adaptive valley, when one of the extreme phenotypes is at greater advantage. Consequently, this favors the production of unfit phenotypes unless some form of assortative mating mechanism is in place (see for example van Doorn et al. 2009). In Article **III** I show that if the male indicator trait is correlated with his condition and condition is frequency dependent, then mate choice leads to wild dynamics of allele/genotype frequencies with periodic or even chaotic orbits.

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In Article **IV** we introduce a mating model that corrects for the unrealistic biological assumptions discussed above, some of them adopted from Gavrilets and Boake 1998 (also used e.g. by Matessi et al. 2001, Kirkpatrick and Nuismer 2004, Schneider 2005, Schneider and Bürger 2006, Pennings et al. 2008, Kopp and Hermisson 2008, Peischl and Schneider 2009, Ripa 2009). Mating model of Gavrilets and Boake (1998) assumes the following. During a mating season, females have a constant maximum number of encounters with males, and at each encounter female choosiness (the male acceptance criteria) remains the same. The maximum number of encountered males is hence independent of the size of the population, which can be justified only if population size (or male availability) has remained constant over a long period of time. The more critical assumption is however that female choosiness is fixed throughout the season. Certainly, if the mating season is at its end and a female is still unmated, the male acceptance criterion should be loosened (this is what in fact happens in many natural systems, see e.g. Backwell and Passmore 1996, Thomas et al. 1998, Gray 1999, Kodric-Brown and Nicoletto 2001, Moore and Moore 2001).

To correct for these factors, we build a mating model which allows the choosiness to be relaxed, the number of males to encounter to be density dependent and where the criteria for accepting a male at an encounter is based on benefits males are offering

(Andersson and Simmons 2006). We find, that the strategy which maximizes the female benefits belongs to time-threshold strategies: accept a male at an encounter after a certain time-threshold connected to his genotype; but if encountered before, reject him and continue with the search. The optimal time-thresholds are given in terms of the benefits. Because the benefits may depend on the genotypic frequencies of the resident population, the time-thresholds that are optimal in the given equilibrium population represent an evolutionarily stable strategy (Maynard Smith 1982).

We present an analytical condition when random mating is an ESS, or, conversely, a condition which shows when females are under selection pressure to evolve a mating preference and thus mate non-randomly. Also, we derive conditions for the various mating strategies under which the fixation equilibria are stable/unstable. Finally, and most importantly, we search for specific ecological scenarios that induce homozygote females to mate assortatively and we provide conditions for reproductive isolation. Here, the benefits were assumed to be the reproductive value of the offspring. As expected, a criterion for reproductive isolation is that females are expected to sample enough males, so that declining bad choices (males that provide little benefits) is not that costly and encountering good males is probable. Surprisingly, however, the outcome of reproductive isolation seems more likely when disruptive selection is *weaker*, as stronger disruptive selection enables the existence of alternative mating strategies that do not facilitate reproductive isolation.

4 Concluding remarks and future perspectives

In this Thesis I considered unresolved questions in speciation research by applying and combining theories of population genetics and adaptive dynamics. I used well established models describing essential phenomena of nature and extended them to reveal aspects of mate choice on reproductive isolation in both ecological (Articles **III** and **IV**) and evolutionary time-scales (Article **I**), as well as created new models that provide a relevant framework for studying the role and interplay between ecology and mate choice in the process of speciation. Furthermore, my work on the theory of adaptive dynamics itself (Article **II**) cleared some unsolved issues and addressed some aspects of the maintenance of polymorphisms.

There is clearly still work to be done. A necessary extension to my work is the evolution of mate choice itself (but see the application of results of Pennings et al. 2008 in Article **I**). For example, it would be valuable to find out whether the mating strategy described in Article **IV**, where a particular solution of a time-threshold tactic was found to be an ESS, is also a convergence stable strategy, i.e. stable on the evolutionary time-scale. If the ESS mating strategy where reproductive isolation is possible is convergence stable, further questions arise about the ecological conditions under which it is so. However, if it is not, that would force us to take a different turn, as the model in Article **IV** describes the best possible scenario; females are making the perfect choice when selecting males under the prevailing ecological setting. Or

maybe females are not perfect after all?

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References

[1] Andersson, M., and L.W. Simmons. (2006) *Sexual selection and mate choice* Trends Ecol. Evol. 21: 296–302.

[2] Backwell, P. R. Y., and N.I. Passmore. 1996. *Time constraints and multiple choice criteria in the sampling behaviour and mate choice of the fiddler crab,* Uca annulipes. Behav. Ecol. Soc. 38: 407–416.

[3] Barret, P.H., Gautrey P. J.,Hebert, S., Kohn, D. and Smith, S. (1987) *Charles Darwin's notebooks, 1834-1844: Geology, transmutation of species, metaphysical enguires.* Cornell University Press, Ithaca, New York.

[4] Bisby F., Roskov Y., Culham A., Orrell T., Nicolson D., Paglinawan L., Bailly N., Appeltans W., Kirk P., Bourgoin T., Baillargeon G., Ouvrard D., eds (2012) Species 2000 & ITIS Catalogue of Life, 30th May 2012. Digital resource at www.catalogueoflife.org/col/. Species 2000: Reading, UK.

[5] Christiansen F. B. , Loeschke V. (1980) *Evolution and intraspecific exploitation competition I. One-locus theory for small additve gene effects.* Theor. Popul. Biol. 18: 297–313.

[6] Claessen D. & Dieckmann, U. (2002) *Ontogenetic niche shift and evolutionary branching in structured populations.* Evol. Ecol. Res. 4: 189–217.

[7] Cotton, S., Fowler, K., Pomiankowski, A., 2004. *Do sexual ornaments demonstrate heightened condition-dependent expression as predicted by the handicap hypothesis?* Proc. R. Soc. Lond. B 271: 771–783.

[8] Coyne, J. A. & Orr, H. A. (2004). *Speciation.* Sinauer associates.

[9] Darwin, C. (1859). *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life.* (1st ed.). London: John Murray.

[10] Darwin, C.(1871). *The Descent of Man, and Selection in Relation to Sex.* (1st ed.). London: John Murray.

[11] Dieckmann, U. (1997) *Can adaptive dynamics invade?* Trends in Ecology and Evolution 12: 128–131.

[12] Doebeli, M., Block, H.J., Leimar, O., Dieckmann, U. (2007) *Multimodal pattern formation in phenotype distributions of sexual populations.* Proc. Biol. Sci. 1608: 347–57.

[13] Erwin, T. L. (1983) *Tropical forest canopies: the last biotic frontier.* Bul. Ent. Soc. Am. 29: 14–19.

[14] Ewens, W.J. (2004) *Mathematical Population Genetics* 2nd Edition. Springer-Verlag, New York.

[15] Felsenstein, J. (1981) *Scepticism towards Santa Rosalia, or why are there so few kinds of animals?* Evolution 35: 124–138.

[16] Gavrilets, S., and C. R. B. Boake. (1998) *On the evolution of premating isolation after a founder event.* Am. Nat. 152: 706–716.

[17] Gavrilets S. (2004) Fitness landscapes and the origin of species. Princeton University Press.

[18] Geritz, S.A.H. (2005). *Resident-invader dynamics and the coexistence of similar strategies.* J. Math. Biol. 50: 67–82.

[19] Geritz, S. A. H. & Kisdi, É. & Meszéna, G. & Metz, J. A. J. (1998) *Evolutionarily singular strategies and the adaptive growth and branching of the evolutioary tree.* Evol. Ecol. 12: 35–57.

[20] Grafen, A. (1990) *Biological signals as handicaps.* J. Theor. Biol. 144: 517-546.

[21] Gray, D. (1999) *Intrinsic factors affecting female choice in house crickets: time of first reproduction, female age, nutritional condition, body size, and size-relative reproductive investment.* J. Insect Behav. 12: 691–700.

[22] Gyllenberg, M., Metz, J.A.J. (2001) *On fitness in structured metapopulations.* J. Math. Biol. 43: 545–560.

[23] Haffer, J. (1982) Systematik und Taxonomie der *Larus argentatus*?Artengruppe. In Handbuch der Vögel Mitteleuropas, vol. 8 (ed. U. N. Glutz von Blotzheim & K. M. Bauer), pp. 502–514. Wiesbaden: Akad. Verlagsges.

[24] Hartl, D. (2007) *Principles of Population Genetics.* Sinauer Associates.

[25] Iwasa, Y., Pomiankowski, A., Nee, S., (1991) *The evolution of costly mate preferences. II. The "handicap" principle* Evolution 45: 1431–1442.

[26] Kirkpatrick M. & S. L. Nuismer. (2004) *Sexual selection can constrain sympatric speciation.* Proc. R. Soc. Lond. B 271: 687–693.

[27] Kisdi É, Meszéna G (1993) *Density dependent life history evolution in fluctuating environments.* In Adaptation in a Stochastic Environment (C.W. Clark and J. Yoshimura, eds). Lecture Notes in Biomathematics 98: 26–62.

[28] Kisdi É, Meszéna G (1995) *Life history with lottery competition in a stochastic environment: ESSs which do not prevail.* Theor. Pop. Biol. 47: 191 – 211.

[29] Kisdi É. & S. A. H. Geritz. (1999) *Adaptive dynamics in allele space: Evolution of genetic polymorphism by small mutations in a heterogeneous environment.* Evolution 53: 993–1008.

[30] Kodric-Brown, A., and P.F Nicoletto. 2001. *Age and experience affect female choice in the guppy (*Poecilia reticulata*)* Am. Nat. 157: 316–323.

[31] Kokko, H., Brooks, R., Jennions, M.D., Morley, J., 2003. *The evolution of mate choice and mating biases.* Proc. R. Soc. Lond. B 270: 653-664.

[32] Kopp, M., and J. Hermisson. (2008) *Competitive speciation and costs of choosiness.* J. Evol. Biol. 21: 1005–1023.

[33] Liebers, D., de Knijff P., Helbig A. J. (2004) *The herring gull complex is not a ring species.* Proc. Roy. Soc. B 271 1542: 893–901.

[34] Levene, H. (1953) *Genetic equilibrium when more than one ecological niche is available.* Am. Nat. 87: 331–333.

[35] Matessi C., A. Gimelfarb & S. Gavrilets. (2001) *Long-term buildup of reproductive isolation promoted by disruptive selection: How far does it go?* Selection 2: 41–64.

[36] May, R. M. (1988) *How many species are there on Earth?* Science 241: 1441–1449.

[37] May, R. (2010) *Tropical arthropod species, more or less?* Science 329: 41–42.

[38] Maynard Smith, J. (1982) *Evolution and the theory of games.* Cambridge University Press, Cambridge.

[39] Mayr, E. (1942) *Systematics and the origin of species.* New York: Columbia University Press.

[40] Mayr, E. (1969) *Principles of Systematic Zoology.* New York: Mcgraw-Hill, p. 316.

[41] Mayr, E. (1982) *The Growth of Biological Thought. Cambridge* (Mass.): Belknap P. of Harvard U.P

[42] Mayr, E. (1995). *Species, classification, and evolution.*Pp. 3–12 in R. Arai, M. Kato, and Y.Doi (eds.) Biodiversity and Evolution. Natonial Science Museum Foundation, Tokyo.

[43] Metz, J.A.J., Nisbet R.M., Geritz, S.A.H. (1992) *How should we define "fitness" for general ecological scenarios?* Trends Ecol. Evol. 7: 198–202.

[44] Metz, J.A.J., Geritz, S.A.H., Meszéna, G., Jacobs, F.J.A., Van Heerwaarden, J.S. (1996) *Adaptive dynamics: A geometrical study of the consequences of nearly faithful reproduction.* In Stochastic and Spatial Structures of Dynamical Systems (S.J. van Strien and S.M. Verduyn Lunel, eds) 183–231 North-Holland, Elsevier.

[45] Moore, P.J., and A.J. Moore. (2001) *Reproductive aging and mating: The ticking of the biological clock in female cockroaches.* Evolution 98: 9171–9176.

[46] Mora, C., Tittensor, D.P., Adl, S., Simpson, A. G. B., Worm, B. (2011) *How Many Species Are There on Earth and in the Ocean?* PLoS Biol. doi:10.1371/journal.pbio.1001127

[47] Motro, U. (1982) *Optimal rates of dispersal I. Haploid populations.* Theor. Pop. Biol. 21: 394-411.

[48] Møller, A. P., and M.D. Jennions. (2001) *How important are direct fitness benefits of sexual selection?* Naturwissenschaften 88: 401–415.

[49] Peischl S. & K. Schneider. (2009) *Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population.* Evolution 64-2: 561–582.

[50] Pennings P. S., M. Kopp, G. Meszéna, U. Dieckmann & J. Hermisson. (2008) *An analytically tractable model for competitive speciation.* Am. Nat. 171: E44–E71.

[51] Raup, D. M. (1991) *Extinction: Bad Genes or Bad Luck?* W.W. Norton and Company. New York.

[52] Ripa, J. (2009) *When is sympatric speciation truly adaptive? An analysis of the joint evolution of resource utilization and assortative mating.* Evol. Ecol. 23: 31–52.

[53] Schneider K. A. (2005) *Competitive divergence in non-random mating populations.* Theor. Pop. Biol. 68: 105–118.

[54] Schneider K. A. & R. Bürger. (2006) *Does competitive divergence occur if assortative mating is costly?* J. Evol. Biol. 19: 570–588.

[55] Servedio M. R., G. S. van Doorn, M. Kopp, A. Frame & P. Nosil. (2011) *Magic traits in speciation: 'magic' but not rare.* TREE 26: 389–397.

[56] Thomas, F., C. Liautard, F. Cézilly, and F. Renaud. (1998) *A finite time horizon influences sequential mate choice in male* Gammarus aequicauda *(Amphipoda).* Canadian J. Zool. 76: 401–405.

[57] van Doorn, S., Edelaar, P., Weissing, F. (2009) *On the origin of species by natural and sexual selection.* Science 326, 1704–1707.

[58] Wen, J. (1999) *Evolution of Eastern Asian and Eastern North American disjunct distributions in flowering plants.* Annu. Rev. Ecol. Syst. 30: 421–455.

[59] Westwood, J.O. (1833) *On the probable number of species of insects in the creation; together with the descriptions of several minute Hymenoptera.* Mag. Nat. Hist. J. Zool Bot. Min. Geol Met. 6: 116–123.

[60] Zahavi, A. (1975) *Mate selection - a selection for a handicap.* J. Theor. Biol. 53: 205–214.

[61] Zimmer, C. (2011) *How Many Species? A Study Says 8.7 Million, but It's Tricky.* New York TImes. August 30, D3.

Article I

Evolutionary branching of a magic trait

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Evolutionary branching of a magic trait

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Abstract We study the adaptive dynamics of a so-called magic trait, which is under natural selection and which also serves as a cue for assortative mating. We derive general results on the monomorphic evolutionary singularities. Next, we study the long-term evolution of single-locus genetic polymorphisms under various strengths of assortativity in a version of Levene's soft-selection model, where natural selection favours different values of a continuous trait within two habitats. If adaptive dynamics leads to a polymorphism with sufficiently different alleles, then the corresponding homozygotes cease to interbreed so that sympatric speciation occurs.

Mathematics Subject Classification (2000) 92D15, Problems related to evolution

1 Introduction

The origin of species is a cardinal question of biology. For speciation to occur in sexually reproducing organisms, two essential processes need to take place (Coyne and Orr 2004): First, genetic polymorphism must arise and be maintained in the population; and second, reproductive isolation must evolve such that genetically different lineages cease to interbreed. Most traditional models of sympatric speciation (e.g. Maynard Smith 1966; Dickinson and Antonovics 1973; Caisse and Antonovics 1978; Udovic 1980; see Gavrilets 2004 for review) assumed that ecological selection maintains polymorphism in one locus, whereas a second locus controls reproductive isolation via mate choice (for example it may control flowering time in plants, where early and late flowering individuals are reproductively isolated from one another). In such models,

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ecologically different lineages become reproductively isolated if linkage disequilibrium arises between the two loci such that the ecological trait and mate choice become genetically correlated. Recombination between the ecological locus and the mating locus, however, efficiently destroys any linkage disequilibrium, rendering speciation impossible unless ecological selection is strong (Felsenstein 1981).

Reproductive isolation might arise easier if mate choice is based on the ecological trait itself, with like individuals mating preferentially with each other. For example, different habitats or different pollinators may exert ecological selection for early vs late flowering in plants (as it happens between edaphic plants and their normal varieties, cf. Macnair and Gardner 1998). In this case, mate choice itself is under ecological selection; or in other words, reproductive isolation evolves as a natural byproduct of ecological divergence. After Gavrilets (2004), traits which are under ecological selection and also influence mate choice are called *magic traits*. Magic traits are free of the problem of recombination because the mating cue and the ecological trait are one and the same.

Empirical evidence shows that magic traits are common. Body size, a common target of ecological selection, is also a common cue for mating. Body size is a magic trait in sticklebacks (Nagel and Schluter 1998; Hatfield and Schluter 1999; Rundle 2002), in sea horses (Jones et al. 2003), intertidal snails (Cruz et al. 2004), in amphipods (Wellborn 1994; McPeek and Wellborn 1998), and in Drosophila (Hegde and Krishna 1997). Colour patterns serve as mating cues but are also under disruptive ecological selection in butterflies (Jiggins et al. 2001, 2004) and in coral reef fishes (Puebla et al. 2007). The shape and colour of animal-pollinated flowers are obvious candidates for magic traits, although the empirical evidence is less clear in this case (Waser and Campbell 2004; Gegear and Burns 2007). The call frequency of bats determines both the prey and the mates they can locate (Kingston and Rossiter 2004). The flowering time of some plants (Macnair and Gardner 1998) and the time of eclosion and mating in the apple maggot fly (Felsenstein 1981) are also magic traits. Beak morphology and song are determined partly by the same genes in Darwin's finches (Podos 2001; Huber et al. 2007), such that there is a common magic trait on the genetic level that correlates with both phenotypic traits, one influencing resource use and the other used for mate choice. Many of the examples mentioned above are putative cases of incipient sympatric speciation. Conversely, in most cases where ongoing sympatric speciation is suspected and the mechanisms of ecological divergence and reproductive isolation are known, reproductive isolation emerges as a byproduct via ecological selection on a magic trait (Bolnick and Fitzpatrick 2007).

In this paper, we analyze the adaptive dynamics of a magic trait. Adaptive dynamics (Geritz et al. 1997, 1998) is a leading mathematical framework to investigate how continuous traits evolve under ecological selection and small mutational steps, and, in particular, how diversity evolves via evolutionary branching. Since evolutionary branching of a magic trait can lead to reproductive isolation as a byproduct, the adaptive dynamics of magic traits offer an analytically tractable model of speciation.

Whether speciation occurs via the evolution of a magic trait depends on whether reproductive isolation becomes sufficiently strong. Reproductive isolation, in turn, depends on how big a difference the diverging lineages evolve in their magic traits, and how strongly mate choice is discriminative (the latter referred to as mating assortativity

or "choosiness"). Several models have investigated the evolution of choosiness while they kept magic trait values unchanged (Matessi et al. 2001; Pennings et al. 2008; Kopp and Hermisson 2008; Otto et al. 2008). Here we make the complementary assumption that the magic trait evolves and the level of choosiness remains fixed. Our results however also enable conclusions to be drawn on the joint evolution of the magic trait and of choosiness, provided that the latter evolves sufficiently slowly (see Sect.4).

In the first part of the paper, we analyze the evolution of magic traits in monomorphic populations and address in particular the question whether evolutionary branching occurs in a diploid sexual population under the most popular mating model (introduced by Doebeli 1996, Gavrilets and Boake 1998 and Matessi et al. 2001, and used e.g. by Kirkpatrick and Nuismer 2004; Schneider 2005; Schneider and Bürger 2006; Pennings et al. 2008; Kopp and Hermisson 2008; Ripa 2009). In this part, we accommodate arbitrary ecological selection and thereby provide general results for the stability properties of monomorphic evolutionary singularities under sexual reproduction and assortative mating. Stability properties of a monomorphic singularity were also analyzed by Schneider (2005) in a special case of the mating model considered here, but with haploid genetics and in only one specific ecological model.

The second part of this paper investigates the evolution of the magic trait after evolutionary branching has taken place, and in particular asks whether the evolutionary divergence of the magic trait continues far enough to provide reproductive isolation of the strength seen inbetween biological species. Whereas evolutionary branching depends only on the local properties of the fitness function and therefore can be analyzed without making particular assumptions about the ecological system, evolution after branching is determined by global properties that depend on the concrete ecological model at hand. In this second part, we use the so-called Levene's soft-selection model, a simple ecological model with selection in two contrasting habitats. The population genetics of this model is extremely well known (Levene 1953; see e.g. Roughgarden 1979; Hartl and Clark 1989, Nagylaki and Lou 2001, 2006; Nagylaki 2009; Bürger 2010), and it served as a classic framework of speciation models (e.g. Maynard Smith 1966; Felsenstein 1981). Furthermore, it was used to explore how adaptive dynamics can be applied to evolving alleles in diploid sexual populations under random mating (Kisdi and Geritz 1999; Van Dooren 1999, Geritz and Kisdi 2000). Here we add assortative mating to the Levene model to study speciation after evolutionary branching of a magic trait.

2 General model: monomorphic singularities and evolutionary branching

We consider a population of sexually reproducing diploid individuals with discrete generations. The population is assumed to be sufficiently large to ignore random genetic drift. A continuous trait $\phi \in X \subseteq \mathbb{R}$ is determined by a single autosomal locus which evolves by mutation and natural selection. We assume that the map between homozygote genotypes and phenotypes is a bijection, and denote the alleles of the locus by the phenotype of the corresponding homozygote individual such that $\phi_{xx} = x$ (when appropriate, we shall also use single-letter designations such as g for a diploid genotype). The genotype-phenotype map ϕ_{xy} is assumed to be at least twice

continuously differentiable with respect to the allelic values x and y and to be strictly monotonic such that $\frac{\partial \phi_{xy}}{\partial x} \neq 0$ [see Van Dooren (2000) for the consequences of violating this assumption]. We assume no difference between maternally and paternally derived alleles so that $\phi_{xy} = \phi_{yx}$. With these assumptions, the allelic effects are locally additive (i.e., $\phi_{xy} \to \frac{1}{2}(\phi_{xx} + \phi_{yy})$ as $|y - x| \to 0$) and $\frac{\partial \phi_{xy}}{\partial y}\big|_{y=x} = \frac{1}{2}$.

Let $P_g^{(t)}$ denote the frequency of diploid genotype g among the newborn offspring in generation t. An offspring with genotype g survives to adulthood with probability $v_{E(t)}(\phi_g)$, where the selective environment $E(t)$ is determined by which phenotypes are present and what is their population density in generation t. In this section, we do not have to specify the concrete form of ecological selection encapsulated in v; later we shall investigate an example based on Levene (1953) multiple habitat model. For convenience, let $w_E(\phi_g) = Bv_E(\phi_g)$ denote the absolute genotypic fitness in ecological selection, where B is the average number of offspring produced by a mated female. We shall assume that $w_E(\phi_g)$ is positive for all admissible values of its arguments, twice differentiable with respect to ϕ_g and E, and E depends sufficiently smoothly on the phenotypes and their population densities. After ecological selection, the frequency of genotype g among the adults is

$$\tilde{P}_g^{(t)} = \frac{w_{E(t)}(\phi_g)}{\bar{w}_{E(t)}} P_g^{(t)}, \tag{1}$$

where $\bar{w}_{E(t)} = \sum_g P_g^{(t)} w_{E(t)}(\phi_g)$.

Adult females choose mates nonrandomly such that a female of genotype g mates (and produces offspring) with a male of genotype h with probability $Q_{g,h}\tilde{P}_h^{(t)}$. The quantity $Q_{g,h}$ measures the affinity of g females towards h males and depends on their phenotypic resemblance as described below by Eq. (7). Note that in general $Q_{g,h} \neq Q_{h,g}$. $\sum_h Q_{g,h}\tilde{P}_h^{(t)}$ may be less than 1, in which case the female remains unmated with a positive probability.

The genotypic frequencies at the beginning of the new generation are

$$P_r^{(t+1)} = \frac{1}{\bar{Q}} \sum_{g,h} \tilde{P}_g^{(t)} \tilde{P}_h^{(t)} Q_{g,h} R_{g,h\to r}, \tag{2}$$

where $\bar{Q} = \sum_{g,h} \tilde{P}_g^{(t)} \tilde{P}_h^{(t)} Q_{g,h}$ is the mean mating success and $R_{g,h\to r}$ denotes the probability that parents with genotypes g and h produce an offspring with genotype r according to the Mendelian rules. Total population size changes according to

$$N^{(t+1)} = \bar{Q}\bar{w}_{E(t)} N^{(t)}. \tag{3}$$

2.1 Assortative mating

Mate choice is based on phenotypic similarity of the ecological trait ϕ between the mating partners. The assumption that the same trait ϕ determines fitness in ecological selection and controls mate choice makes ϕ a "magic" trait as called by Gavrilets (2004). To formulate the probability of mating, we follow the assumptions Doebeli

1996, Gavrilets and Boake (1998) and Matessi et al. (2001). These assumptions are widely used (see e.g. Kirkpatrick and Nuismer 2004; Schneider 2005; Schneider and Bürger 2006; Pennings et al. 2008; Kopp and Hermisson 2008; Ripa 2009).

Assume that females encounter males at random. If a female with phenotype ϕ_g encounters a male with phenotype ϕ_h, she accepts the male for mating with probability

$$\mu(\phi_g, \phi_h) = \mu_{max}\pi(\phi_h - \phi_g), \tag{4}$$

where $0 < \mu_{max} \leq 1$ is the maximum mating probability and π is a twice continuously differentiable function that attains its maximum at 0 with $\pi(0) = 1$ and is bounded away from zero for all admissible values of $\phi_h - \phi_g$. If the female does not accept the male, she may try again until the total number of encounters has reached a maximum number M. Females mate at most once but males can participate in several matings. The probability that an encounter between a female of type ϕ_g and a random male results in mating is

$$\bar{\mu}(\phi_g) = \sum_h \mu(\phi_g, \phi_h)\tilde{P}_h \tag{5}$$

and the probability that she eventually mates with a male of type ϕ_h is

$$\sum_{i=0}^{M-1} [1 - \bar{\mu}(\phi_g)]^i \mu(\phi_g, \phi_h)\tilde{P}_h \tag{6}$$

such that we have

$$Q_{g,h} = \mu(\phi_g, \phi_h)\frac{1 - (1 - \bar{\mu}(\phi_g))^M}{\bar{\mu}(\phi_g)} \tag{7}$$

to be inserted into Eq. (2). Matessi et al. (2001) observed that with $M = 1$, the model can be seen as a model of fertility selection (Bodmer 1965; Hadeler and Liberman 1975) or as a model of parental selection (Gavrilets 1998); Schneider (2005) studied the evolution of a magic trait in a haploid model with $M = 1$, $\mu_{max} = 1$.

With $M < \infty$, females may remain unmated. This results in sexual selection favoring common females: Females whose phenotype is rare prefer to mate with rare male phenotypes and therefore run a higher risk of remaining unmated. With $M \to \infty$, females experience no sexual selection. Males, however, may remain unmated also in this case, and the average number they mate depends on their phenotype and on the phenotypic distribution of females. Males therefore always experience frequency-dependent sexual selection next to natural selection on the ecological trait.

In a population monomorphic for allele x, females are eventually mated with probability

$$Q_{xx,xx} \equiv Q = 1 - (1 - \mu_{max})^M. \tag{8}$$

This probability is independent of the resident phenotype but is less than 1, unless $\mu_{max} = 1$ so that females accept the first male for mating or $M \to \infty$ so that females can keep trying until they mate. The mating process therefore affects the dynamics of the population [see Eq. (3)] even if every male and female is equally likely to mate.

2.2 Invasion fitness

We assume that alleles undergo mutations with small phenotypic effect, mutant alleles have initially low frequency, and mutations occur infrequently such that the resident population has reached its population genetic attractor by the time a new mutant comes along. Under these assumptions, we can use adaptive dynamics (Geritz et al. 1998) to study long-term evolution in the space of alleles (Kisdi and Geritz 1999). For simplicity, we also assume that the resident population dynamics attains a unique point attractor such that the environment E is constant and uniquely determined by the resident allele(s) (see Geritz et al. 2002 for extension to multiple attractors). We shall write $w_{\hat{E}(\phi_{xx})}(\phi_g)$ to denote ecological fitness of genotype g in the environment set by a monomorphic resident population with phenotype ϕ_{xx}.

In Appendix 1, we derive invasion fitness (the marginal fitness of a rare allele) as shown in Eq. (11) below; here we shall arrive at the same result in a more heuristic way. Consider a mutant allele y in a population otherwise monomorphic for the resident allele x. If the mutant allele is sufficiently rare, then the probability of forming a mutant homozygote offspring is negligible. This is obvious in the case of random mating, but remains true also in our assortative mating model given that $w_{\hat{E}(\phi_{xx})}(\phi_{xy}) > 0$ and $\pi(\phi_{xx} - \phi_{xy}) > 0$ (see Appendix 1). The dynamics of the mutant allele are then governed by the dynamics of heterozygotes,

$$P_{xy}^{(t+1)} = \frac{1}{2}\frac{Q_{xy,xx} + Q_{xx,xy}}{Q}\tilde{P}_{xx}^{(t)}\tilde{P}_{xy}^{(t)} + \mathcal{O}((\tilde{P}_{xy}^{(t)})^2) \tag{9}$$

(cf. Eqs. (2), (8)). In the first term of this equation, $Q_{xy,xx}\tilde{P}_{xx}^{(t)}$ is the probability for a heterozygote female to eventually mate with a resident homozygote male; in the second term, $Q_{xx,xy}\tilde{P}_{xy}^{(t)}$ is the probability for a resident homozygote female to eventually mate with a heterozygote male; both types of mating produce heterozygote offspring with probability $1/2$. With the resident population in equilibrium, $Q w_{\hat{E}(\phi_{xx})}(\phi_{xx}) = 1$ [cf. Eq. (3)] and hence from Eq. (1), we obtain $\tilde{P}_{xy}^{(t)} = Q w_{\hat{E}(\phi_{xx})}(\phi_{xy}) P_{xy}^{(t)}$. Substituting this and noting that $\tilde{P}_{xx}^{(t)} = 1 + \mathcal{O}(\tilde{P}_{xy}^{(t)})$ in Eq. (9), we arrive at

$$P_{xy}^{(t+1)} = \frac{1}{2}\left(Q_{xy,xx} + Q_{xx,xy}\right) w_{\hat{E}(\phi_{xx})}(\phi_{xy}) P_{xy}^{(t)} + \mathcal{O}((P_{xy}^{(t)})^2) \tag{10}$$

from which the marginal fitness of the rare allele, i.e., the invasion fitness of allele y in the resident population of x is

$$W_x(y) = \frac{1}{2}\left(Q_{xy,xx} + Q_{xx,xy}\right) w_{\hat{E}(\phi_{xx})}(\phi_{xy}). \tag{11}$$

Simplifying from Eq. (7),

$$Q_{xy,xx} = 1 - \left[1 - \mu_{max}\pi(\phi_{xx} - \phi_{xy})\right]^M \tag{12}$$

describes sexual selection on heterozygote females in Eq. (11) and

$$Q_{xx,xy} = \left[1 - (1 - \mu_{max})^M\right]\pi(\phi_{xy} - \phi_{xx}) \tag{13}$$

gives sexual selection on males.

If mating is random, i.e., $\pi \equiv 1$ and a female accepts any male with probability μ_{max}, then Eq. (11) simplifies to

$$W_x(y) = Q w_{\hat{E}(\phi_{xx})}(\phi_{xy}). \tag{14}$$

We thus recover the marginal fitness of the rare allele $W_x(y)$ as the fitness of heterozygotes in ecological selection. Recall, however, that the mating process affects the dynamics of the population and therefore affects the resident environment $\hat{E}(\phi_{xx})$ even with random mating. When we compare results obtained for assortative mating with those under random mating, we always assume that the mating process is as described above (with $\pi \equiv 1$ for random mating), and therefore only a fraction Q of the resident females is mated, regardless of whether mating is assortative or random. Simply removing the mating process from the model, and assuming instead that each female mates with the first male she encounters, would introduce a change in the ecological environment unless $Q = 1$, i.e., unless $\mu_{max} = 1$ or $M \to \infty$.

To obtain invasion fitness in a polymorphic resident population, note that a rare mutant allele y in a resident population with alleles x_1 and x_2 is almost exclusively in heterozygotes and therefore the initial invasion dynamics can be written as

$$\mathbf{P}_{het}^{(t+1)} = \mathbf{M}\mathbf{P}_{het}^{(t)}, \tag{15}$$

where $\mathbf{P}_{het} = (P_{x_1y}, P_{x_2y})^T$ and $\mathbf{M}$ is a 2×2 matrix that depends on the allelic values y and x_1, x_2 (see Appendix 1 for details). The invasion fitness of the mutant, $W_{x_1,x_2}(y)$, is the dominant eigenvalue of $\mathbf{M}$. For small mutations (i.e., if either $|y - x_1|$ or $|y - x_2|$ is sufficiently small), this is equivalent to

$$\tilde{W}_{x_1,x_2}(y) = \mathrm{Tr}\mathbf{M} - \mathrm{Det}\mathbf{M} > 1 \tag{16}$$

(see Appendix 1). $\tilde{W}_{x_1,x_2}(y)$ is a proxy for invasion fitness: Because $\log \tilde{W}$ is sign-equivalent to $\log W$ and has the same smoothness properties, we can find the diallelic singularities and their stability properties using $\tilde{W}_{x_1,x_2}(y)$ (which is easier to calculate) in place of the dominant eigenvalue $W_{x_1,x_2}(y)$ [see Metz and Leimar (2010) for a related approach].

2.3 Monomorphic singularities and evolutionary branching

The mutant allele y, when it appears in a single copy in a large resident population fixed for allele x, has a positive probability of invasion if $W_x(y) > 1$; otherwise the mutant goes extinct with probability 1 (Jagers 1975). By repeated mutations and allele substitutions, the magic trait evolves in the direction of the selection gradient $\partial W_x(y)/\partial y\big|_{y=x}$ until it reaches either an endpoint of the trait space X or an evolutionary singular trait value x^* at which

$$\left.\frac{\partial W_x(y)}{\partial y}\right|_{y=x=x^*} = \frac{1}{2}Q\left.\frac{\partial w_{\hat{E}(\phi_{xx})}(\phi_{xy})}{\partial \phi_{xy}}\right|_{y=x=x^*} = 0 \tag{17}$$

(Geritz et al. 1998; Geritz 2005). Notice that the selection gradient does not depend on function π because $\pi'(0) = 0$, and therefore, the existence, number, and position of evolutionary singularities are independent of assortativity of mating. The singularity is convergence stable (i.e., approached by gradual evolution via small mutation steps) if

$$\left[\frac{\partial^2 W_x(y)}{\partial x \partial y} + \frac{\partial^2 W_x(y)}{\partial y^2}\right]_{y=x=x^*} = \frac{1}{2}Q\left[\frac{\partial^2 w}{\partial \phi_{xx}\partial \phi_{xy}} + \frac{\partial^2 w}{\partial \phi_{xy}^2}\right]_{y=x=x^*} < 0 \tag{18}$$

(Eshel 1983; Christiansen 1991), where we wrote $w = w_{\hat{E}(\phi_{xx})}(\phi_{xy})$ for short. This condition is again independent of mating assortativity, as expected, because convergence stability follows directly from the selection gradient.

The singularity is evolutionarily stable (sensu Maynard Smith 1982) if

$$\left[\frac{\partial^2 W_x(y)}{\partial y^2}\right]_{y=x=x^*} = \frac{1}{4}\left(Q\left[\frac{\partial^2 w}{\partial \phi_{xy}^2}\right]_{y=x=x^*} + q\pi''(0)\right) < 0, \tag{19}$$

where

$$q = \frac{1}{2}\left[1 + \frac{M\mu_{max}(1-\mu_{max})^{M-1}}{1-(1-\mu_{max})^M}\right]. \tag{20}$$

In (19), sexual selection from assortative mating contributes a negative term via $\pi''(0) < 0$. Assortative mating stabilizes x^* against the invasion of mutants because rare phenotypes are at a disadvantage during mating.

The strength of the stabilizing effect of assortative mating depends on parameters μ_{max} and M via quantity q. To interpret the relative weight of ecological and sexual selection in (19), note that Q, the coefficient in front of the term corresponding to ecological selection, simply corrects w for the population dynamical effect of the mating process, i.e., the product Qw is the invasion fitness under random mating [cf. Eq. (14)]. If $M = 1$, then (20) simplifies to $q = 1$ and we recover a result of Schneider (2005): the curvatures of fitness in ecological and in sexual selection contribute equally and additively to the condition of evolutionary stability. If $M > 1$

and $\mu_{max} \to 1$, then $q = \frac{1}{2}$ so that only male sexual selection contributes to (19). This is because the probability that a heterozygote female remains unmated $[(1 - \mu_{max}\pi(\phi_{xx} - \phi_{xy}))^M]$ is in this case a "flat" function of the phenotypic difference with vanishing second derivative at zero, so that for small mutations, female sexual selection is negligible compared to sexual selection on males and to ecological selection. Finally if $M \to \infty$ with arbitrary μ_{max}, then again $q = \frac{1}{2}$; in this case all females are eventually mated and hence there is no sexual selection on females, who are half the parents of the next generation (see also Kirkpatrick and Nuismer 2004; Schneider and Bürger 2006 on evolutionary stability in face of stabilizing sexual selection).

Evolutionary branching occurs in initially monomorphic populations at a singularity that is convergence stable but not evolutionarily stable (Geritz et al. 1998). Assortative mating does not change convergence stability but hinders evolutionary branching via stabilizing sexual selection: increasing assortativity, which corresponds to increasing $\pi''(0)$ in absolute value, can turn an evolutionary branching point [where (19) is violated] into an ESS [where (19) is satisfied].

2.4 Polymorphism near singularities

An important property of a singularity is whether there are pairs of alleles in its neighbourhood such that each of the two alleles can invade the other's monomorphic resident population (*mutual invasibility*) or, to the contrary, there are pairs such that neither can invade the other and therefore the rare allele goes extinct regardless of which of the two alleles is rare (*mutual exclusion*). In the vicinity of x^*, there exist pairs of alleles that exhibit mutual invasibility and hence form a protected polymorphism if

$$\begin{aligned}\left[\frac{\partial^2 W_x(y)}{\partial x \partial y}\right]_{y=x=x^*} &= \frac{1}{4}\left(Q\left[2\frac{\partial^2 w}{\partial \phi_{xx}\partial \phi_{xy}} + \frac{\partial^2 w}{\partial \phi_{xy}^2}\right]_{y=x=x^*} - q\pi''(0)\right) \\ &= -\frac{1}{4}\left(Q\left[\frac{\partial^2 w}{\partial \phi_{xx}^2}\right]_{y=x=x^*} + q\pi''(0)\right) < 0, \end{aligned} \tag{21}$$

where in the last step we used that $w_{\hat{E}(\phi_{xx})}(\phi_{xx}) = 1/Q$ is constant for all x and hence $[\frac{\partial^2 w}{\partial \phi_{xx}^2} + 2\frac{\partial^2 w}{\partial \phi_{xx}\partial \phi_{xy}} + \frac{\partial^2 w}{\partial \phi_{xy}^2}]_{y=x} = 0$; the opposite of (21) implies the existence of allele pairs with mutual exclusion near x^* (Geritz et al. 1998).

There are two aspects of condition (21) to interpret. Concerning ecological selection, recall that in a clonal model of adaptive dynamics, there are strategy pairs with phenotypes ϕ_{xx} and ϕ_{xy} near $\phi_{x^*x^*}$ that mutually invade each other if $[\frac{\partial^2 w}{\partial \phi_{xx}\partial \phi_{xy}}]_{x^*} < 0$. The ecological selection part (derivatives of w in the brackets) in (21) is at variance with this clonal condition. This is because of diploid inheritance: Alleles x and y mutually invade each other if the heterozygote phenotype ϕ_{xy} invades both ϕ_{xx} and ϕ_{yy} (whereas the clonal condition requires that ϕ_{xy} and ϕ_{xx} invade each other). Assume that the singular phenotype $\phi_{x^*x^*}$ can invade all other phenotypes in its vicinity. Then at least alleles placed symmetrically around x^* (such as $x = x^* - \epsilon$ and $y = x^* + \epsilon$)

can invade each other, because due to locally additive allelic effects, they produce the heterozygote phenotype $\phi_{xy} = \phi_{x^*x^*}$, which invades any phenotype in the vicinity, including ϕ_{xx} and ϕ_{yy}. In the opposite case if $\phi_{x^*x^*}$ cannot invade other phenotypes in its vicinity, then alleles near x^* cannot invade each other even if they are symmetrically placed (as easily seen e.g. from pairwise invasibility plots, this is the most favourable configuration for mutual invasibility). Hence the diploid condition of mutual invasibility of alleles coincides with the clonal condition of the singular phenotype being able to invade other similar phenotypes. The latter is given by $[\frac{\partial^2 w}{\partial \phi_{xx}^2}]_{x^*} > 0$ (Geritz et al. 1998), which directly corresponds to the ecological part of (21).

Concerning sexual selection, $\pi''(0) < 0$ makes it more difficult to satisfy (21), i.e., assortative mating hinders mutual invasibility. The weight of sexual selection (q) is the same as in the ESS-condition (19) above. Because rare alleles are at a disadvantage in sexual selection, mutual invasibility can turn into mutual exclusion near the singularity if the assortativity of mating is increased. The remainder of this section explores the consequences of the bifurcation between mutual invasibility and mutual exclusion.

Mutual invasibility yields (protected) polymorphism, but a locally stable polymorphism may occur also without mutual invasibility when the population genetic equations [our Eqs. (1) and (2)] have multiple attractors. We shall refer to a locally asymptotically stable polymorphic equilibrium of two alleles as *unprotected polymorphism* when one or both boundary equilibria (fixation of an allele) are also locally stable. The alleles can coexist indefinitely in an unprotected polymorphism, provided that the population is never subject to large perturbations that would bring the system into the basin of attraction of a boundary equilibrium.

Because sexual selection disfavours rare alleles, it stabilizes the boundary equilibria and for a given pair of alleles, this can lead to loss of mutual invasibility or to mutual exclusion. Sexual selection however weakens when the alleles have more comparable frequencies, i.e., sexual selection does not necessarily destabilize an internal (polymorphic) equilibrium when it does stabilize a boundary equilibrium. One can therefore readily expect that unprotected polymorphisms occur under assortative mating. Below, Fig. 1 illustrates using the Levene model as an example that this is indeed the case. Actually, it is proved in a separate paper (Priklopil in prep.) that unprotected polymorphism generically occurs when mutual invasibility near a singularity turns into mutual exclusion (or vice versa). Therefore, if in the present model ecological selection promotes mutual invasibility near the singularity such that (21) is satisfied for random mating, then strengthening the assortativity of mating [increasing $\pi''(0)$ in absolute value] will cause a bifurcation into mutual exclusion, and unprotected polymorphism necessarily occurs near x^* for some values of $\pi''(0)$. Note however that starting with a monomorphic population, unprotected polymorphism could be reached only if a new allele appears with sufficiently high frequency (e.g. due to secondary contact with a formerly isolated population).

3 Levene model: adaptive dynamics and speciation in polymorphic populations

In the second part of the paper, we aim at investigating the adaptive dynamics of a magic trait when the resident population is already polymorphic. This analysis will show whether the alleles evolve sufficiently far apart such that homozygotes become

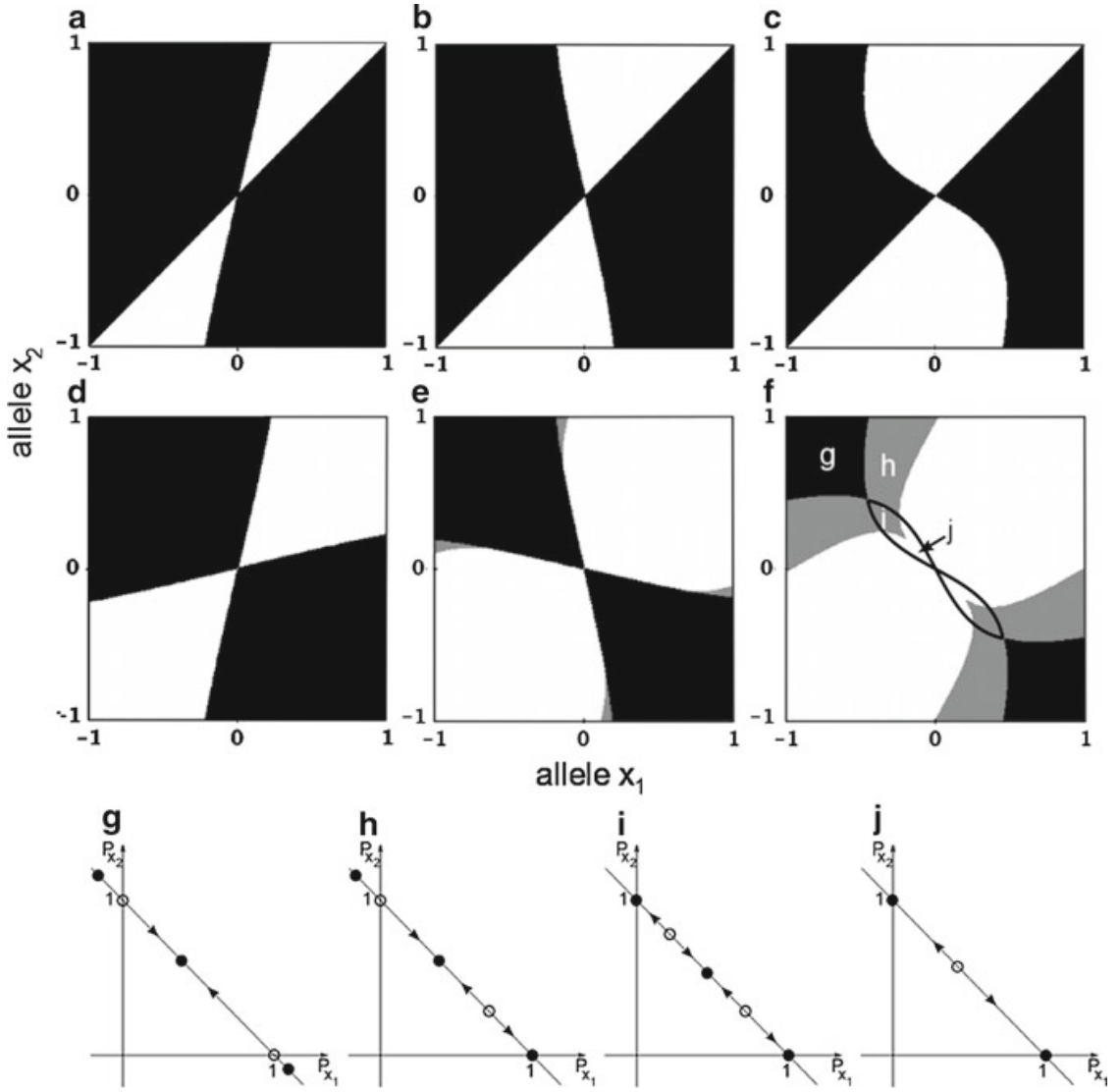

Fig. 1 *Top row* pairwise invasibility plots (*black color* denotes the area where allele x_2 can invade the resident allele x_1 and *white color* where it can not) for $c_1 = c_2 = 0.5, d = 3, \sigma_s = 1$, and **a** $\sigma_m = \infty$ (random mating), **b** $\sigma_m = 0.50$ (moderate assortativness) and **c** $\sigma_m = 0.36$ (strong assortativeness). *Middle row* areas of mutual invasibility (protected polymorphism, *black*) and unprotected polymorphism (*grey*); mutual exclusion occurs in **f** in the area adjacent to the singularity in between the invasion boundaries (*bold lines*). Parameters for **d**–**f** as in **a**–**c**. *Bottom row* dynamics of allele frequencies with **g** protected polymorphism; **h** unprotected polymorphism; **i** unprotected polymorphism with mutual exclusion; and **j** mutual exclusion. The *filled circles* and *empty circles* indicate stable and unstable equilibria, respectively. **g**–**j** represent the dynamics which occurs in the corresponding regions of **f**

reproductively isolated by assortative mating. Because this analysis depends on the global properties of the fitness function, we need to make specific assumptions about the ecological setting underlying $w_{E(t)}(\phi_g)$. We shall thus use a version of Levene's soft-selection model (Levene 1953).

3.1 Assumptions

For the ecological model, we consider a population in which the offspring of each discrete generation are distributed randomly over two habitats (Levene 1953). Within

each habitat, there is first a period of viability selection, which an individual with phenotype ϕ_g survives with probability

$$f_1(\phi_g) = \alpha_1 \exp\left(-\frac{(\phi_g - m_1)^2}{2\sigma_s^2}\right) \tag{22a}$$

in habitat 1 and

$$f_2(\phi_g) = \alpha_2 \exp\left(-\frac{(\phi_g - m_2)^2}{2\sigma_s^2}\right) \tag{22b}$$

in habitat 2, respectively, where α_1 and α_2 are the maximum survival probabilities, m_1 and m_2 are the optimal phenotypes in the two habitats, and $\sigma_s > 0$ controls the strength of stabilizing selection within a habitat. Without loss of generality, we set $m_1 = -d/2$ and $m_2 = d/2$, where d is the distance between the two habitat-specific optima.

Viability selection is followed by non-selective "contest" competition, where a fixed number K_i of individuals survive to adulthood within habitat i ($i = 1, 2$; we assume that fecundity is sufficiently large such that the number of offspring after viability selection exceeds K_i in both habitats and for all phenotypes considered). As a result, a fraction $c_1 = K_1/(K_1 + K_2)$ of the adult population is recruited from habitat 1 and the remaining fraction $c_2 = 1 - c_1$ comes from habitat 2. All adults form a single population where mating is assortative with respect to phenotype but not with respect to habitat.

As we derive in Appendix 2, fitness in ecological selection under these assumptions is given by

$$w_{E(t)}(\phi_g) = c_1 \frac{f_1(\phi_g)}{\sum_h P_h(t)\, f_1(\phi_h)} + c_2 \frac{f_2(\phi_g)}{\sum_h P_h(t)\, f_2(\phi_h)}. \tag{23}$$

Note that the selective environment

$$E(t) = \left(\sum_h P_h(t)\, f_1(\phi_h), \sum_h P_h(t)\, f_2(\phi_h)\right) \tag{24}$$

depends on the frequencies and phenotypes of all genotypes present and changes in time until the genotypic frequencies arrive at an equilibrium, but can always be given by only two variables that are the habitat-specific mean probabilities of survival during viability selection. Contrary to clonal models, in a diploid sexual population the number of environmental feedback variables does not give an upper bound on the number of alleles that can form a polymorphism, except if the alleles act additively on the within-habitat viabilities (Nagylaki and Lou 2001) or there is partial dominance that is constant across habitats in the Levene model (Nagylaki 2009). In our model, allelic effects determine the phenotype ϕ additively but (24) implies nonadditive effects

on the viabilities with variable dominance and possible overdominance; as a consequence, more than two alleles can be present at equilibrium (Kisdi and Geritz 1999; Nagylaki 2009).

For the mating process, in this example we shall assume $M \to \infty$ such that all females are eventually mated. This immediately implies $\bar{Q} = Q = 1$. Eqs. (12) and (13) simplify to $Q_{xy,xx} = 1$ and $Q_{xx,xy} = \pi(\phi_{xy} - \phi_{xx})$, respectively, and in the invasion fitness

$$W_x(y) = \frac{1}{2}(1 + \pi(\phi_{xy} - \phi_{xx}))w_{\hat{E}(\phi_{xx})}(\phi_{xy}), \tag{25}$$

purely ecological selection on females is combined with ecological and sexual selection on males. We adopt the mating function

$$\pi(\phi_h - \phi_g) = \exp\left(-\frac{(\phi_h - \phi_g)^2}{2\sigma_m^2}\right) \tag{26}$$

for the probability that a ϕ_g female accepts a ϕ_h male when they encounter each other (the same has been used e.g. by Doebeli 1996, Matessi et al. 2001; Pennings et al. 2008; Ripa 2009). In (26), decreasing σ_m^2 corresponds to decreasing $\pi''(0) = -1/\sigma_m^2$ and stronger assortativity.

Finally, we assume that the alleles act additively on the phenotype, i.e.,

$$\phi_{x_i x_j} = \frac{x_i + x_j}{2} \tag{27}$$

holds also for large differences between x_i and x_j (local additivity follows already from the smoothness assumptions made in the general model).

By scaling the allelic values, one can set $\sigma_s = 1$ without loss of generality. Hence the adaptive dynamics of alleles depend on three parameters only: the relative size of habitats, c_1 (with $c_2 = 1 - c_1$); the (scaled) difference between the habitat-specific optima, d/σ_s; and the (scaled) strength of assortative mating, σ_m/σ_s. With random mating ($\sigma_m/\sigma_s \to \infty$), this model is identical to the one investigated by Kisdi and Geritz (1999).

3.2 Monomorphic singularities of the Levene model

Before turning to speciation in polymorphic populations, we briefly illustrate our general results on monomorphic singularities in the Levene model. Substituting the ecological model (23) and genotype-phenotype map (27) into Eq. (25), we arrive at

$$W_x(y) = \frac{1}{2}\left(1 + \pi\left(\frac{y-x}{2}\right)\right)\left(c_1\frac{f_1\left(\frac{x+y}{2}\right)}{f_1(x)} + c_2\frac{f_2\left(\frac{x+y}{2}\right)}{f_2(x)}\right) \tag{28}$$

for the the invasion fitness with π and f_i given as in Eqs. (26) and (22), respectively. Using (17), we obtain a unique evolutionary singularity at

$$x^* = (c_2 - c_1)d/2 \tag{29}$$

and this singularity is always convergence stable [condition (18) simplifies to $-1/\sigma_s^2 < 0$]. The ESS condition (19) is not satisfied so that the singularity x^* is an evolutionary branching point if

$$c_1 c_2 (d/\sigma_s)^2 > 1 \quad \text{and} \quad (\sigma_m/\sigma_s)^2 > \frac{1}{2(c_1 c_2 (d/\sigma_s)^2 - 1)}. \tag{30}$$

The first of these inequalities is the condition for evolutionary branching under random mating (cf. Kisdi and Geritz 1999). Assortative mating hinders evolutionary branching so that branching occurs only if it does under random mating, and, in addition, mating is not too assortative so that σ_m is sufficiently large to satisfy the second inequality in (30). In the vicinity of x^*, there are pairs of alleles that mutually invade each other and hence form a protected polymorphism [see (21)] if

$$(\sigma_m/\sigma_s)^2 > \frac{1}{2(c_1 c_2 (d/\sigma_s)^2 + 1)} \tag{31}$$

whereas there are allele pairs with mutual exclusion if the opposite inequality holds. Notice that under random mating ($\sigma_m \to \infty$), the condition for mutual invasibility is always satisfied, but it can be violated if mating is sufficiently assortative.

The resulting bifurcation patterns are illustrated in Fig. 1. The top row contains pairwise invasibility plots [i.e., sign plots of $\log W_x(y)$] for increasing assortativeness, taking parameter values such that evolutionary branching occurs under random mating. The middle row shows the areas of mutual invasibility ($W_x(y) > 1$ and $W_y(x) > 1$) as derived from the pairwise invasibility plots, and areas of unprotected polymorphism as found by numerical continuation of equilibria. As σ_m decreases, x^* first bifurcates from an evolutionary branching point (Fig. 1a) into an ESS with mutual invasibility in its neighbourhood (Fig. 1b); next, mutual invasibility (Fig. 1d, e) bifurcates into mutual exclusion near x^* (Fig. 1f). When mating is sufficiently assortative, unprotected polymorphism appears (Fig. 1e, f). When mutual invasibility has bifurcated into mutual exclusion such that the invasion boundary lines [where $W_x(y) = 1$ and $W_y(x) = 1$, respectively] intersect away from the main diagonal $y = x$, unprotected polymorphism is always found in the neighbourhood of the intersection (Fig. 1f; see Priklopil in prep. for proof). The bottom row of Fig. 1 (where p_{x_i} denotes the frequency of allele x_i) illustrates the dynamics of allelic frequencies in protected and unprotected polymorphisms, and mutual exclusion with no polymorphism.

Figure 2 shows all bifurcations of monomorphic evolutionary singularities for equal habitat sizes ($c_1 = c_2 = \frac{1}{2}$) from (30) and (31). When disruptive selection generated by the contrasting habitats is weak ($d/\sigma_s < 2$), the monomorphic singularity is always an ESS. When the ecological conditions generate stronger disruptive selection ($d/\sigma_s > 2$)

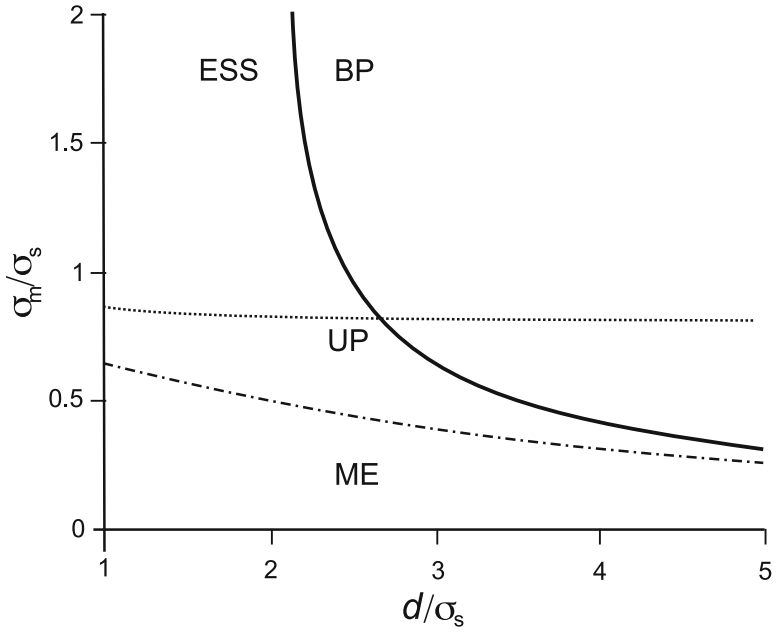

Fig. 2 Bifurcation plot for monomorphic singularities assuming equal habitat size ($c_1 = c_2 = 0.5$). On the *continuous line*, x^* bifurcates between an ESS and an evolutionary branching point BP [equality in the second condition of (30)]; on the *dash-dotted line*, x^* bifurcates between having mutual invasibility and mutual exclusion ME attached to it [equality in condition (31)]. Below the *dotted line*, unprotected polymorphism UP exists for some allelic values (x_1, x_2) (this region is found numerically)

and assortativness is weak (σ_m is sufficiently high), then the singularity is a branching point, but branching is lost if stabilizing sexual selection is too strong (σ_m is small). Strong sexual selection can always generate mutual exclusion near the singularity.

3.3 Adaptive dynamics in polymorphic resident populations and speciation by magic traits

In this section, we investigate the dynamics of evolution after evolutionary branching of a magic trait, with particular attention to whether speciation occurs and whether the existing species are stable in face of changes in the environment. We adhere to the biological species concept, i.e., define species by substantial (although not necessarily complete) reproductive isolation between them (see e.g. Coyne and Orr 2004). The evolution of a magic trait leads to reproductive isolation, and thus to speciation if the phenotypes become sufficiently different relative to the width of the mating function σ_m in Eq. (26), so that separate phenotypes cease to interbreed and heterozygotes ("hybrids" between the homozygote species) become rare. σ_m may depend on environmental factors. A famous example is found in the cichlid fish of Lake Victoria (Seehausen et al. 1997): Mate choice is determined by colour in normal clear water, but since recent eutrophication of the lake has increased water turbidity and made colours difficult to recognize, mating has become less assortative and formerly isolated species started to fuse. Below, we shall also investigate the consequences of environmental changes affecting σ_m.

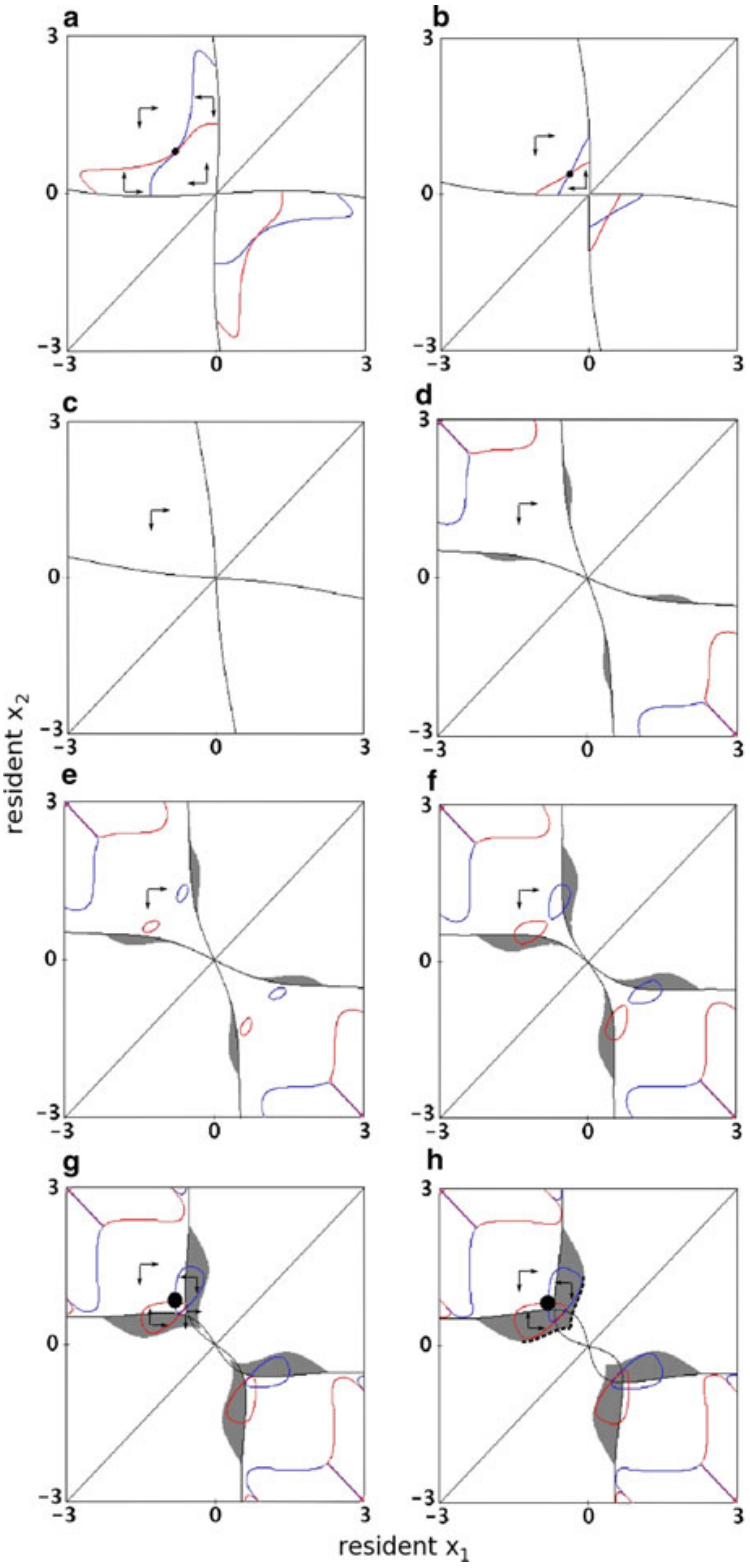

Fig. 3 Trait evolution plots for $d = 2.25$ and **a** $\sigma_m = \infty$, **b** $\sigma_m = 1.6$, **c** $\sigma_m = 1.0$, **d** $\sigma_m = 0.6$, **e** $\sigma_m = 0.57$, **f** $\sigma_m = 0.5$, **g** $\sigma_m = 0.43$ and **h** $\sigma_m = 0.38$. *Arrows* indicate the direction of the selection gradient in allele x_1 (*horizontal*) and allele x_2 (*vertical*); isoclines are the null clines of the selection gradients and diallelic singularities are at the intersection of isoclines. *Dots* indicate convergence stable diallelic singularities (these are also evolutionarily stable). The *size of the dot* indicates the strength of reproductive isolation at the singularity; *smallest dot* denotes $F < 0.9$ (**a**, **b**), *middle sized dot* denotes $0.9 < F < 0.99$ (not in this figure), *biggest dot* denotes $0.99 < F$ (**g**, **h**). *Grey areas* denote unprotected polymorphisms. In **h**, we marked the boundaries across which evolutionary suicide can occur, but analogous boundaries are found in every panel with unprotected polymorphism. The monomorphic singularity at $(x^*, x^*) = (0, 0)$ is an evolutionary branching point for $\sigma_m > 1.37$ (**a**, **b**) and an ESS below this threshold (**c**–**h**); there is mutual invasibility in the neighbourhood of (x^*, x^*) for $\sigma_m > 0.47$ (**a**–**f**) and there is mutual exclusion below this threshold (**g**, **h**)

To analyze the dynamics of evolution after evolutionary branching, we construct so called trait evolution plots, i.e., combined sign plots of the selection gradients $[\partial W_{x_1,x_2}(y)/\partial y]_{y=x_1}$ and $[\partial W_{x_1,x_2}(y)/\partial y]_{y=x_2}$ (Geritz et al. 1998), where the fitness proxy $\tilde{W}$ defined in (16) can be used in place of W. In Figs. 3, 4, 5, arrows

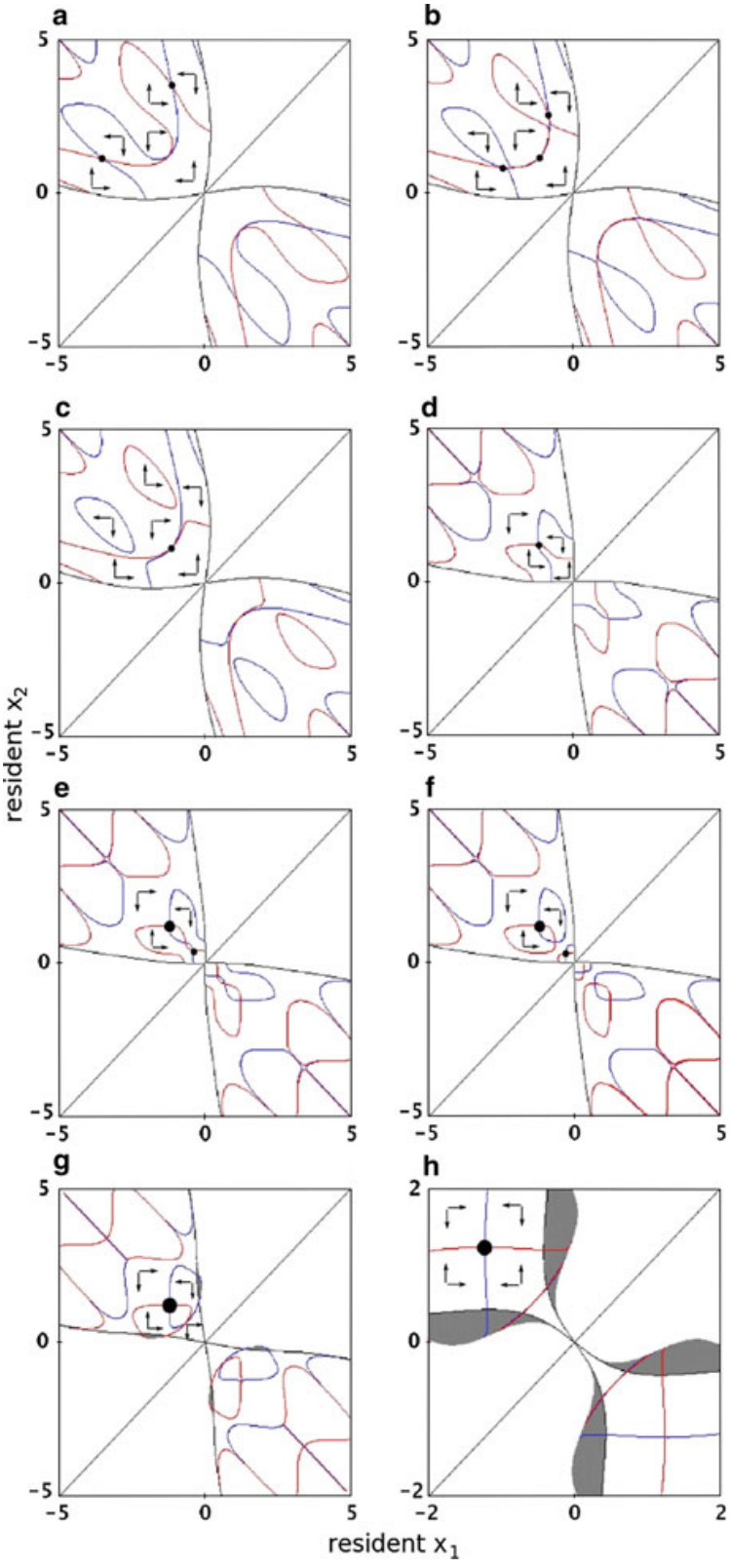

Fig. 4 Trait evolution plots for $d = 2.66$ and **a** $\sigma_m = \infty$, **b** $\sigma_m = 2.9$, **c** $\sigma_m = 2.6$, **d** $\sigma_m = 0.9$, **e** $\sigma_m = 0.83$, **f** $\sigma_m = 0.82$, **g** $\sigma_m = 0.61$ and **h** $\sigma_m = 0.45$. Notations as in Fig. 3. Note that in the *upper left* and *lower right* of each plot, the x_1- and x_2-isoclines are very near to each other especially for small values of σ_m, but there is no isolated singularity in these regions [the outermost symmetric singularity is around $(x_1, x_2) = (-1.2, 1.2)$ in every panel]. Note also the change of scale in **h**. The monomorphic singularity at $(x^*, x^*) = (0, 0)$ is an evolutionary branching point for $\sigma_m > 0.81$ (**a**–**f**) and an ESS below this threshold (**g**–**h**); there is mutual invasibility in the neighbourhood of (x^*, x^*) for $\sigma_m > 0.42$ (in all panels shown here)

show the direction of selection gradients inside the area of coexistence; the boundary lines where the corresponding selection gradient changes sign are referred to as x_1- and x_2-isoclines. Note that the isoclines extend smoothly into the areas of unprotected polymorphism (grey areas). The intersections of isoclines correspond to diallelic

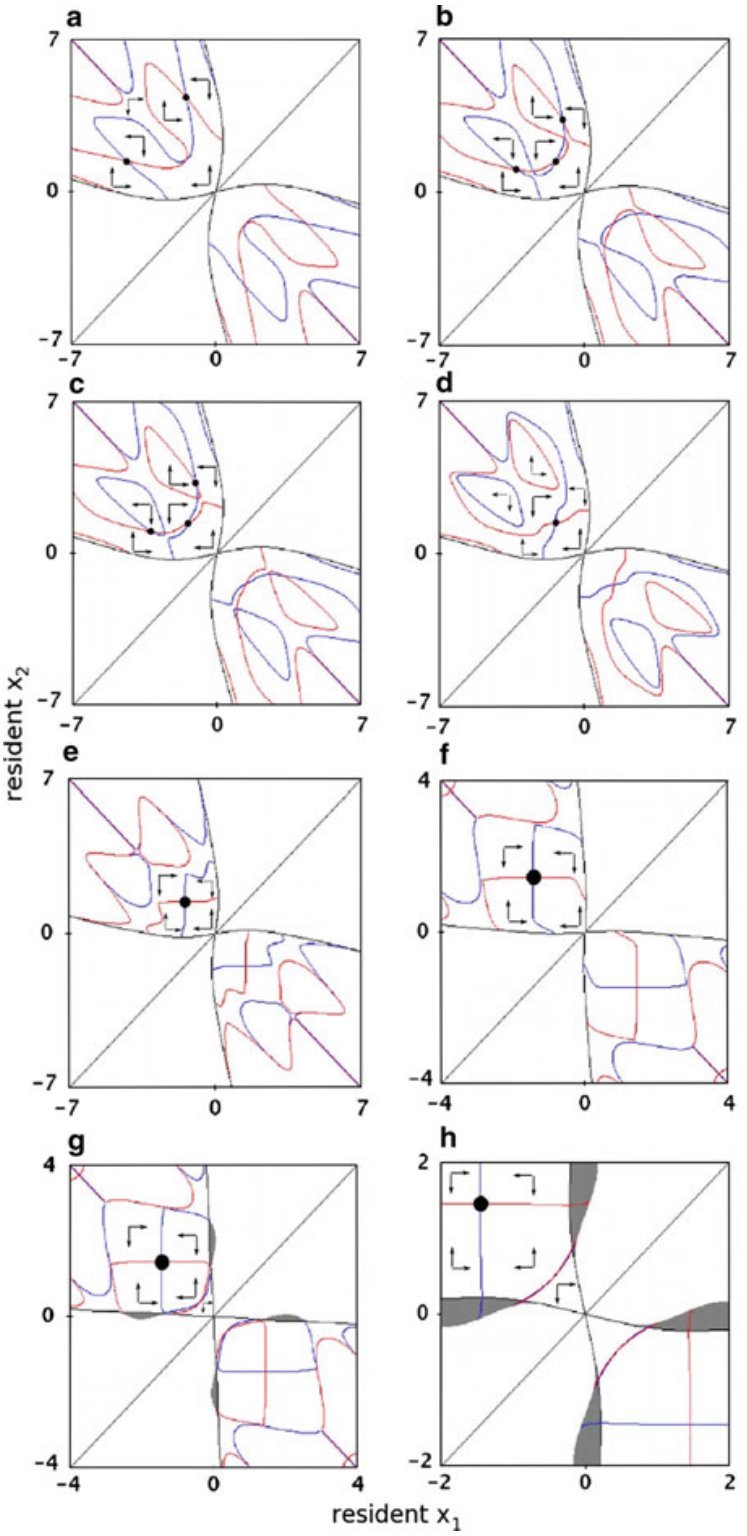

Fig. 5 Trait evolution plots for $d = 3$ and **a** $\sigma_m = \infty$, **b** $\sigma_m = 2.0$, **c** $\sigma_m = 1.95$, **d** $\sigma_m = 1.5$, **e** $\sigma_m = 1.0$, **f** $\sigma_m = 0.7$, **g** $\sigma_m = 0.6$ and **h** $\sigma_m = 0.5$. Notations as in Fig. 3. Note that in the *upper left* and *lower right*, the x_1- and x_2-isoclines are very near to each other but there is no isolated singularity in these regions [the outermost symmetric singularity is around $(x_1, x_2) = (-1.4, 1.4)$ in every panel]. Note also the changes of scale. The monomorphic singularity at $(x^*, x^*) = (0, 0)$ is an evolutionary branching point for $\sigma_m > 0.63$ (**a**–**f**) and an ESS below this threshold (**g**, **h**); there is mutual invasibility in the neighbourhood of (x^*, x^*) for $\sigma_m > 0.39$ (in all panels shown here)

evolutionary singularities [cf. Eq. (39)], and the singularities which are evolutionarily as well as convergence stable (see Appendix 3) are marked with filled circles. Trait evolution plots are symmetric with respect to the main diagonal $x_2 = x_1$ because labelling of the resident alleles is arbitrary. In addition, the trait evolution plots in Figs. 3, 4, 5 are also symmetric with respect to the secondary diagonal $x_2 = -x_1$ because we assume equal habitat size, $c_1 = c_2 = 0.5$ (see Appendix 3 for relaxing

this symmetry). In case of random mating ($\sigma_m = \infty$), we recover the trait evolution plots of Kisdi and Geritz (1999).

To assess the degree of reproductive isolation at the evolutionary singularities, we calculate the deficiency of heterozygotes among newborns (before ecological selection) at the population genetic equilibrium of the two resident alleles of the diallelic singularity, as compared to Hardy-Weinberg equilibrium: $F = 1 - P_{x_1x_2}/(2p_{x_1}p_{x_2})$, where p_{x_i} denotes the frequency of allele x_i ($p_{x_1} = p_{x_2} = \frac{1}{2}$ by symmetry on the secondary diagonal $x_2 = -x_1$). $F = 0$ corresponds to random mating and $F = 1$ implies that the two homozygotes are fully isolated and hence behave as separately evolving species. In Figs. 3, 4, 5 we indicate three different regimes of reproductive isolation with three different sized circles; the smallest circle (as in Fig. 3a) denotes $F < 0.9$, the middle sized circle (as in Fig. 4e) denotes $0.9 < F < 0.99$ and the biggest circle (as in Fig. 4g) denotes $0.99 < F$.

It will be necessary to distinguish between strong discrimination against males even with small phenotypic differences to the female phenotype (small σ_m in Eq. (26)) and strong reproductive isolation due to large phenotypic differences ($|x_1 - x_2|$). Henceforth we shall refer to small σ_m as "strongly assortative", and use "strongly isolated" when the probability of interbreeding between different phenotypes is small and therefore F is close to 1.

Moderate difference between habitat optima. In Fig. 3, there is only a moderate difference between the within-habitat optima ($d/\sigma_s = 2.25$), and therefore disruptive ecological selection is weak. In absence of assortative mating (Fig. 3a), evolutionary branching at $x^* = 0$ is followed by evolution to a unique convergence and evolutionarily stable diallelic singularity, where two alleles segregate in a randomly mating population ($F = 0$). With weak assortativity (Fig. 3b), the qualitative outcome is the same, and mating is nearly random at the singularity ($F = 0.017$). Increasing assortativity by decreasing σ_m leads to the loss of evolutionary branching as the diallelic singularity converges to (x^*, x^*) (Fig. 3c).

At stronger assortativity, an area of unprotected polymorphism appears (grey areas in Fig. 3d–h). At the outer edge of the area of unprotected polymorphism, the diallelic population genetic attractor disappears via a fold bifurcation. This is a "catastrophic" bifurcation where the frequency of an allele drops to zero discontinuously, which makes evolutionary suicide possible (Gyllenberg and Parvinen 2001). In Fig. 3h, we have marked those parts of the boundary of unprotected polymorphism where evolutionary suicide can happen. Above the main diagonal of the figure ($x_2 > x_1$), the selection gradient in x_1 is positive whereas the selective gradient in x_2 is negative along the outer edge of unprotected polymorphism. At the marked boundary above the secondary diagonal ($x_2 > -x_1$), an evolutionary step downwards leads to the loss of polymorphism and fixation of the x_1 allele: Hence an invading mutant of x_2 causes the extinction of the same allele. Below the secondary diagonal, x_1 may be lost via evolutionary suicide in a similar manner; and below the main diagonal, the roles are reversed. Evolutionary suicide events can happen also for other parameter values, whenever unprotected polymorphism is present.

When assortativity is strong, new diallelic singularities are created via a fold bifurcation (Fig. 3g, h). At the convergence stable (outer) singularity, the two homozygotes are strongly isolated and heterozygotes are nearly absent ($F > 0.99$). The convergence

stable singularity thus corresponds to two biological species. Because two isolated species evolve independently and analogously to two clonal strategies, the position of the diallelic singularity is close to the dimorphic singularity of the corresponding clonal model whenever reproductive isolation is strong (cf. Geritz and Kisdi 2000). Note however that in contrast to the clonal model, the convergence stable diallelic singularity in Fig. 3g, h is not attainable via evolutionary branching from an initially monomorphic population: This is because stabilizing sexual selection in the vicinity of the monomorphic singularity $(x^*, x^*) = (0, 0)$ prevents evolutionary branching when mating is strongly assortative. Even though separate species are stable, for these parameters speciation cannot happen by evolving the magic trait from monomorphic populations.

Suppose now that two isolated species exist at the diallelic convergence stable singularity in Fig. 3g, h, but mating assortativity decreases (σ_m increases) due to a change in the environment. If this change is not too large, then the diallelic singularity is lost (cf. Fig. 3c–f): The two species hybridize to some extent, and the hybrid species complex eventually loses genetic polymorphism by evolving to a monomorphic ESS. Note that there may be significant variations in the transient dynamics: If σ_m increases to just above the fold bifurcation point of the diallelic singularity, then the species initially remain reproductively well isolated but start evolving their magic trait towards the common ESS, and reproductive isolation slowly dissolves as the species evolve towards each other. If σ_m increases more dramatically, then reproductive isolation breaks down instantly and the species fuse on the short population genetic timescale, before evolutionary changes occur in the magic trait. Eventually, however, the two species will in both cases be replaced with a single monomorphic species at the ESS, and the system will stay there also if the original small value of σ_m is restored. If the environment changes such that σ_m jumps to a very high value, then the system settles at a diallelic singularity (Fig. 3a, b) where genetic polymorphism is preserved, although the two separate species are fused into a single, nearly randomly mating population. Restoring the small value of σ_m will, interestingly, restore the original two species only if the disturbed value of σ_m was sufficiently large. If, during the environmental disturbance, the alleles evolve according to Fig. 3a, then they remain in the basin of attraction of the convergence stable diallelic singularity of Fig. 3g, h so that if σ_m assumes its original value after the disturbance, the two species become isolated again. If however the disturbed population evolves as in Fig. 3b, then the alleles evolve out of the basin of attraction in Fig. 3g, h such that restoring the original σ_m will not restore the two species; instead, upon reducing σ_m, one species will eventually go extinct and the other will evolve to the monomorphic ESS.

Increasing the difference between habitat optima. With somewhat larger difference between the habitats ($d/\sigma_s = 2.66$, Fig. 4), the symmetric diallelic singularity is a saddle point under random mating (Fig. 4a), but it undergoes a pitchfork bifurcation and becomes convergence stable under weak assortativity (Fig. 4b). In contrast to the previous scenario, this symmetric convergence stable singularity is not lost as σ_m decreases, and there is substantial reproductive isolation maintaining two separate species at this singularity when mating is fairly assortative ($F > 0.9$ at the outermost singularity in Fig. 4e, f and $F > 0.99$ in Fig. 4g, h). However, as mating becomes

more assortative, this singularity becomes isolated from the evolutionary branching point by a new pair of singularities (Fig. 4e, f). There we see an interesting evolutionary bistability: On the one hand, two reproductively almost isolated species exist at the outer singularity, with few heterozygotes present at mating and therefore with stabilizing sexual selection around the two homozygote phenotypes. This singularity is close to the singular coalition of strategies in the clonal model. On the other hand, however, there is an almost randomly mating population at the inner singularity, where heterozygote females are common and exert stabilizing selection on males, thereby preventing the further divergence of alleles. Evolutionary branching thus does not lead to speciation, because the evolution of the magic trait stops at the innermost singularity, where reproductive isolation is very weak ($F < 0.1$ in Fig. 4e, f). Increasing the strength of assortativity further (Fig. 4g, h) leads to the loss of evolutionary branching, although a pair of well isolated species continues to be convergence and evolutionarily stable.

Environmental changes inducing changes in σ_m can destroy species, but here, they will not destroy genetic polymorphism. When mating assortativity drops, then separate species fuse into a single polymorphic population (e.g. in Fig. 4c, $F < 0.1$ at the diallelic singularity); reproductively isolated species appear instantly if the environment is restored such that σ_m assumes a low value again. If mating becomes almost random (σ_m becomes very large), then the magic trait evolves away from the symmetric diallelic singularity (Fig. 4a), but still remains polymorphic. This change is reversible but with a hysteresis effect: σ_m needs to get below the simultaneous fold bifurcation that destroys the asymmetric singularities in order to allow the magic trait to evolve back to the symmetric diallelic singularity.

With fairly large difference between habitats ($d/\sigma_s = 3$, Fig. 5), the evolution of a magic trait leads to speciation provided that assortativity is sufficiently strong, such that it provides sufficient reproductive isolation at the diallelic singularity, but not too strong, such that it does not prevent evolutionary branching. These conditions hold e.g. in Fig. 5f: Evolutionary branching leads to the evolution of two alleles with $F = 0.999$, i.e., to the evolution of two reproductively isolated homozygote species. For stronger assortativity of mating, the monomorphic singularity $x^* = 0$ becomes an ESS and a saddle point bifurcates from x^* that isolates the diallelic convergence stable singularity. Otherwise, similar conclusions hold as for $d/\sigma_s = 2.66$.

In summary, two species can coexist in an evolutionarily stable manner if the habitats are substantially different (d/σ_s is large) and mating assortativity is sufficiently strong (σ_m/σ_s is not too large). Very strong assortativity however prevents evolutionary branching, so that even though there is an evolutionarily stable pair of species, these species cannot evolve from an initially monomorphic population. Speciation by the evolution of a magic trait occurs for intermediate levels of assortativity. We summarize these results in Fig. 6, which shows where the symmetric diallelic singularities correspond to speciation and whether these can be reached via evolutionary branching from an initially monomorphic population. Appendix 3 contains a full bifurcation analysis of the diallelic singularities, which provides a comprehensive overview of the patterns seen in Figs. 3, 4, 5, and discusses how the bifurcation structures unfold when the habitats are not precisely of the same size.

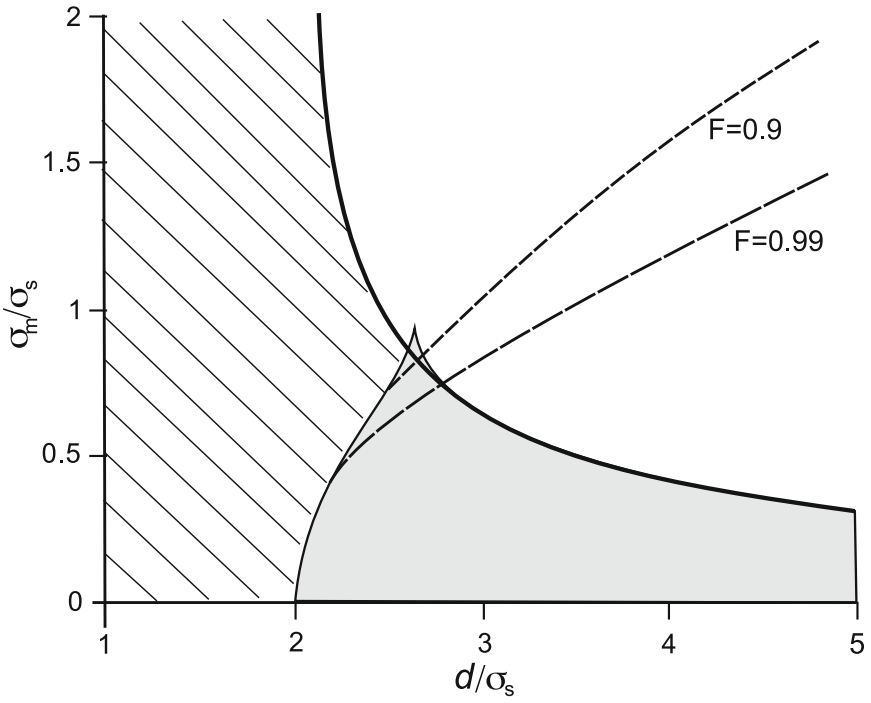

Fig. 6 Strength of reproductive isolation at the symmetric convergence stable diallelic singularity. Such a singularity exists in *white* and *grey areas*. If there is more than one symmetric singularity then F values refer to the outer singularity (see Appendix 3). F increases with decreasing σ_m/σ_s such that $F < 0.9$ above the *upper dashed line*, $0.9 < F < 0.99$ in between the *dashed lines* and $F > 0.99$ below the *lower dashed line*. In the *grey area*, the singularity cannot be reached by evolutionary branching from a monomorphic population. In the *striped area* there are no diallelic singularities

3.4 Species in unequally sized habitats

Analyzing the adaptive dynamics of polymorphic populations with arbitrary habitat sizes (c_1, c_2) is beyond the scope of this paper. However, in Fig. 7 we show one example where the habitats differ substantially in their size $(c_1 = 0.38, c_2 = 0.62)$. In this example, the convergence stable diallelic singularity lies in the area of unprotected polymorphism. Reproductive isolation is strong at this singularity $(F > 0.99)$, i.e., the singularity corresponds to two biological species that coexist at a locally stable equilibrium of their joint population dynamics. This coexistence is however not protected, i.e., not globally stable: If the species harboring allele x_1 becomes rare or goes extinct due to some ecological disturbance, then it is no longer able to invade and recover from low initial population densities. The species loss is therefore permanent, and the remaining species will subsequently evolve to the monomorphic ESS. This is in contrast to the diallelic singularities of Figs. 3, 4, 5, which all occur in the area of protected polymorphism such that any of the two alleles (or species) can invade if rare, provided that it is re-introduced shortly on the evolutionary time scale before the remaining population could evolve away.

4 Discussion

In the first part of this paper, we investigated the evolution of a magic trait under the most commonly used model of assortative mating (originally due to Doebeli 1996;

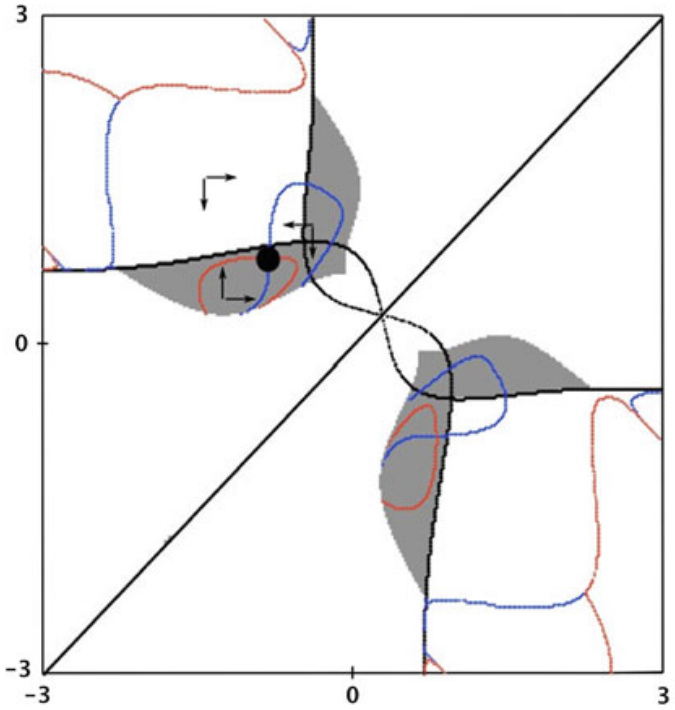

Fig. 7 The convergence stable diallelic singularity lies in the area of unprotected polymorhism when the difference between the habitat sizes is sufficiently large. The parameter values are $d = 2.25$, $\sigma_m = 0.38$ and $c_1 = 0.38$ (the singularity moves to the area of unprotected polymorhism when $c_1 \approx 0.43$)

Gavrilets and Boake 1998; Matessi et al. 2001) and under arbitrary ecological selection. Assortative mating occurs via female preference for males with similar phenotypes to the female herself. Because assortative mating exerts no directional selection, the position and convergence stability of monomorphic evolutionary singularities are not affected by assortativity and coincide with those under random mating (Eqs. (17), (18)); which, in turn, coincide with the monomorphic singularities of the corresponding clonal model of adaptive dynamics (Geritz and Kisdi 2000; Van Dooren in press). Thus as far as the number, position, and convergence stability of monomorphic singularities are concerned, sexual reproduction and assortative mating makes no difference.

In polymorphic populations, however, the abundance of resident genotypes depends on the assortativity of mating, and this affects the selection gradient experienced by rare mutants through both ecological and sexual selection. As ecological and sexual selection interact, the adaptive dynamics of diallelic populations (as shown by the example of the Levene model in Figs. 3, 4, 5) are much richer than adaptive dynamics under random mating (no sexual selection; Kisdi and Geritz 1999) or the adaptive dynamics of clonal phenotypes in the same ecological model (Geritz et al. 1998; Geritz and Kisdi 2000). For the existence and convergence stability of polymorphic singularities, we can draw a general (model-independent) conclusion only for the limiting case where female reproduction is not affected by mating ($M \to \infty$) and assortativity is very strong ($\sigma_m \to 0$), so that heterozygotes are absent and the model reduces to its clonal counterpart.

It is important to recall that if some females remain unmated due to assortative mating ($M < \infty$), then mating also affects ecological fitness via changing the densities of resident genotypes. When relaxing assortativeness ($\sigma_m \to \infty$), the model converges to the corresponding sexual model without mate choice only if all females reproduce ($\mu_{max} = 1$). Likewise, under full reproductive isolation ($\sigma_m \to 0$) but with $M < \infty$

and $\mu_{max} < 1$, females reproduce less on average than in the corresponding clonal model and because this alters the population densities of the residents, ecological selection will be different.

Sexual selection from assortative mating has no directional component but it is stabilizing around the common phenotypes (Kirkpatrick and Nuismer 2004; Schneider 2005; Pennings et al. 2008). At a monomorphic singularity, stabilizing sexual selection counteracts disruptive ecological selection, and therefore may prevent evolutionary branching. Schneider (2005) obtained a similar result in a more specific model, assuming a particular ecological scenario (trait-dependent competition in a Lotka-Volterra model), haploid sexual genetics, and a single opportunity for mating ($M = 1$; $\mu_{max} = 1$); the numerical analysis of two models by Ripa (2009) showed the same. Here, we give the general analytic condition for evolutionary branching to occur in a diploid population in face of assortative mating (Eq. 19).

Stabilizing sexual selection can prevent not only the evolution of polymorphism via evolutionary branching, but also the maintenance of polymorphism of given (fixed) alleles (Matessi et al. 2001; Schneider 2005; Schneider and Bürger 2006; Pennings et al. 2008). We found that under strong assortativity, mutual invasibility near an evolutionary singularity is replaced by mutual exclusion, a situation such that a monomorphic population of either allele resists invasion by the other allele [Eq. (21); see examples in Figs. 1, 2]. Although sexual selection can prevent protected polymorphisms, it can result in an unprotected polymorphism, where there is a stable internal (polymorphic) equilibrium even though both fixation equilibria are also stable (cf. Schneider and Bürger 2006). This is again because assortativity favours the common phenotypes: An allele may be unable to invade when rare but may reach a stable equilibrium when sufficiently common. A set of unprotected polymorphisms appears when mutual invasibility at an evolutionary singularity bifurcates into mutual exclusion (Priklopil in prep.).

To retain analytical tractability (and similarly to previous analytical models of sympatric speciation such as Udovic 1980; Matessi et al. 2001; Schneider 2005; Pennings et al. 2008; Kopp and Hermisson 2008; Otto et al. 2008; Ripa 2009), we assumed throughout that the magic trait is determined by a single gene. While this is a simplification, it may even be close to reality in some cases: The number of genes underlying adaptations and species differences vary and may be as low as one or few (Orr 2001; Woodruff and Thompson 2002). Interestingly, the adaptive dynamics of two loci under random mating in the Levene model leads to loss of genetic variability in one locus, so that eventually the ecological trait is determined by the alleles of a single locus as assumed here (Kisdi and Geritz 1999; Van Doorn and Dieckmann 2006).

4.1 Speciation

In our model, speciation occurs by the evolution of sufficiently large differences between the allelic values of the magic trait, which implies that the homozygotes become reproductively isolated by assortative mating. We studied the evolution of polymorphic populations and in particular speciation in the Levene model as an example. First of all, this model was used as the basis of many classic studies of

speciation assuming non-evolving allelic values (e.g. Maynard Smith 1966; Felsenstein 1981; see Kirkpatrick and Ravigne 2002 and Gavrilets 2004 for reviews); secondly, the adaptive dynamics of allelic values are well understood in this model under random mating (Kisdi and Geritz 1999) and also under linkage disequilibrium with a separate mating locus (Geritz and Kisdi 2000; Kisdi and Geritz in press). In this paper, we explored the evolution of a magic trait rather than separate ecological and mating traits. As recent empirical data demonstrate, this is probably a more common route to sympatric speciation than linkage disequilibrium between separate traits (see Sect. 1).

In the Levene (1953) model, viability selection occurs in two contrasting habitats. The model assumes that adults emerging from both habitats form a single mating population so that mating is independent of habitat origin; in other words, all mating assortativity is due to mate choice by the magic trait and speciation is fully sympatric. This assumption is met if the environment is "fine grained", i.e., if the habitats exist in many randomly placed patches so that distances across different habitats are short relative to the mobility of the organism. A weakness of Levene's soft selection model is that it assumes fully saturated habitats; when the population is maladapted to a habitat, this implies that fecundity must be large. Under random mating and with Gaussian functions as assumed in (22a,b), the monomorphic singularity of the Levene model is always convergence stable, but this does not hold for other choices of functions (Kisdi 2001) and also not in models where the habitats are not always saturated (e.g. Meszéna et al. 1997, Day 2000, Kisdi 2002, Ravigne et al. 2009). If convergence stability of the monomorphic singularity is lost when the habitats differ strongly, then this further limits the possibility of speciation via the evolutionary branching of a magic trait in heterogeneous habitats.

Several models have considered the evolution of mating assortativity, i.e., the evolution of σ_m, with fixed allelic values (x_1, x_2) of the magic trait (Matessi et al. 2001; Pennings et al. 2008; Kopp and Hermisson 2008; Otto et al. 2008; Ripa 2009). These models concluded that for speciation, disruptive ecological selection on the magic trait needs to be strong. As assortativity gradually increases by the evolution of σ_m, stabilizing sexual selection strengthens, and may fully balance disruptive ecological selection: If this happens, then the selection gradient on σ_m vanishes before the prospective species become reproductively isolated, so that the process of speciation stalls (Matessi et al. 2001; Pennings et al. 2008).

In this paper, we made the complementary assumption that only the magic trait x evolves whereas σ_m is fixed (see also Schneider 2005). Our model uncovers other difficulties of speciation by magic traits. Unless ecological selection is strong even for moderately different alleles (i.e., unless d/σ_s is large in the Levene model), an initially monomorphic population cannot evolve into two isolated species because when assortativity is sufficiently strong to provide reproductive isolation at a polymorphic singularity, then evolutionary branching either does not occur (as in Figs. 3g, h, 4g, h) or evolution after branching stops before the alleles become sufficiently different to achieve reproductive isolation (Fig. 4e, f). Ripa (2009) investigated speciation by a magic trait in two other ecological models, where again if assortativity is sufficiently strong for reproductive isolation at the diallelic singularity, then it is likely to prevent evolutionary branching of the monomorphic population. Although the numerical analysis of Ripa (2009) was less complete, it seems that the bifurcation structures of

his models are simpler, and alternative diallelic attractors do not prevent speciation as seen in our Fig. 4e, f.

The above-mentioned problems are particularly relevant for the evolution of species diversity after an extinction event. If initially two species are isolated by assortative mating but one goes extinct due to some environmental disturbance, then mating can remain strongly assortative in the remaining species. The remaining species will evolve to the monomorphic singularity, where assortative mating can prevent evolutionary branching (or prevent the evolution of sufficiently different alleles for obtaining reproductively isolated species after branching) so that the initial species diversity will not be restored. This problem is not particular to the example of the Levene model: If assortativity is sufficiently strong for the given ecological parameters, then the monomorphic singularity is always an ESS [Eq. (19)], and evolutionary branching cannot restore an extinct species. In principle, assortativity could evolve arbitrarily strong between two species, but in practice the selection gradient on assortativity will become weak once the species are well isolated. How strong assortativity does evolve relative to ecological selection determines whether evolutionary branching remains possible after an extinction event or not.

Distinction needs to be made between the questions whether evolutionary branching is possible, whether there is a locally stable polymorphic evolutionary singularity where homozygotes are reproductively isolated, and whether such a singularity can be reached from an evolutionary branching point. Evolutionary branching is possible if assortativity is not too strong relative to disruptive ecological selection [Eq. 19]. A singularity with two separate species exists if assortativity is strong enough and ecological selection is able to maintain two clonal phenotypes in an evolutionarily stable coalition. Note that assortativity needs to be both sufficiently weak so as not to exert too strong stabilising selection on similar alleles near the monomorphic singularity, and also sufficiently strong so as to provide reproductive isolation between distinct homozygotes: Given the Gaussian mating function in Eq. (26), this is possible if disruptive ecological selection is sufficiently strong at the branching point and the homozygotes of the diallelic singularity are sufficiently far apart. In the Levene model, both these requirements hold if d/σ_s is large. But evolutionary branching and the existence of a diallelic singularity with strong reproductive isolation does not yet guarantee speciation (cf. Fig. 4e, f): An initially monomorphic population can evolve into two separate species only if disruptive ecological selection is sufficiently strong to overcome sexual selection for *all* allelic values from the monomorphic singularity to a polymorphic singularity with reproductive isolation. Such a case is shown in Fig. 5f.

In reality, the assortativity of mating (σ_m) can co-evolve with the magic trait (x). We did not investigate the joint evolution of the magic trait and of mating assortativity directly (see Ripa 2009), but we can infer the selection gradient on σ_m using a result of Pennings et al. (2008; see also Otto et al. 2008): Assuming that all females are mated ($M \to \infty$), the heterozygote phenotype is exactly inbetween the homozygotes [as in Eq. (27)], and the mating function is Gaussian [as in Eq. (26); but also under some other mating functions], a mutant gene increasing the assortativity of mating (decreasing σ_m) can invade if and only if both homozygote genotypes of the magic trait have higher fitness than the heterozygote. This condition holds at every symmetric diallelic singularity in Figs. 3, 4, 5, therefore assortativity increases as long as the magic

trait is at or near such a singularity. Assume that σ_m evolves much slower than the magic trait (x), so that the magic trait attains quasi-equilibria at its convergence stable singularities. Under such a separation of evolutionary time scales, speciation occurs by gradual evolution of σ_m in an initially randomly mating population if a symmetric diallelic singularity exists for every σ_m from random mating to full isolation. For moderate values of d/σ_s this is not the case, because the diallelic singularity is lost at intermediate values of σ_m as shown in Fig. 3. Note however that a modifier with large effect on σ_m could cause instant speciation by bringing the population directly from the diallelic attractor of Fig. 3a to that of Fig. 3g, h. A modifier decreasing σ_m can invade a (nearly) randomly mating population independently of the size of its effect (Pennings et al. 2008), although it remains to see if a modifier with large effect goes to fixation. Earlier studies also found that assortment is more likely to evolve in large steps, but for a different reason: If there is an interval of σ_m where assortativeness is not selected for, a large mutation can "jump" over this (e.g. Matessi et al. 2001, Schneider and Peischl, in prep.). At the symmetric diallelic singularities of our model, assortative mating is always selected for, but a large mutation in σ_m could help to jump an interval where a diallelic singularity does not exist.

For larger values of d/σ_s, speciation is prevented by the evolution of the magic trait while the population is nearly randomly mating. In Fig. 4a, the symmetric diallelic singularity is a saddle point and the magic trait evolves to an asymmetric singularity, where the heterozygotes have higher fitness than homozygotes and therefore assortativity is not selected for (cf. Geritz and Kisdi 2000). If however some assortativity exists already in the initial population, then this can stabilize the symmetric singularity [as in Fig. 4b]; from this point, the evolution of assortativity preserves the symmetric singularity of the magic trait and leads to speciation (cf. Figs. 4b–h, 5b–h; Ripa 2009). The symmetric singularity can also be stabilized if migration between the two habitats is somewhat restricted (Kisdi and Geritz in press). Since in a spatially heterogeneous environment a moderate isolation between habitats can easily occur, such a weak initial reproductive isolation by distance can help initializing the process of speciation, which then continues by the evolution of mate choice while the magic trait is at a symmetric singularity. Finally for $d/\sigma_s > 4.03$, the symmetric diallelic singularity is convergence stable in a randomly mating population (Kisdi and Geritz 1999) as well as for any value of σ_m (Appendix 3). This implies that speciation can occur via slow evolution of mating assortativity in an initially randomly mating population. Note however that this requires very strong ecological selection; and for large d/σ_s the assumption of fully saturated habitats breaks down unless fecundity is large.

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Appendix 1

In this Appendix, we derive the invasion fitness of a rare allele y in a resident population harbouring alleles $x_1, \ldots, x_k$. Invasion fitness in a monomorphic population,

Eq. (11), follows directly. We also derive the fitness proxy in Eq. (16), which we use in diallelic resident populations.

Let vector $\mathbf{P}$ be the frequency vector of genotypes containing allele y, $\mathbf{P}^{(t)} = (P^{(t)}_{x_1y}, \ldots, P^{(t)}_{x_ky}, P^{(t)}_{yy})^T$. From (1) and (2), the full dynamics of allele y is given by

$$\begin{aligned}
P^{(t+1)}_{x_ly} = \frac{1}{\bar{Q}\bar{w}^2_{E(t)}} \Bigg[& \frac{1}{4}\sum_{i=1}^{k}\sum_{j=1}^{k} P^{(t)}_{x_iy} P^{(t)}_{x_jx_l} w_{x_iy} w_{x_jx_l} (Q_{x_iy,x_jx_l} + Q_{x_jx_l,x_iy}) \\
& + \frac{1}{4}\sum_{i=1}^{k} P^{(t)}_{x_iy} P^{(t)}_{x_lx_l} w_{x_iy} w_{x_lx_l} (Q_{x_iy,x_lx_l} + Q_{x_lx_l,x_iy}) \\
& + \frac{1}{4}\sum_{i=1}^{k} P^{(t)}_{x_iy} P^{(t)}_{x_ly} w_{x_iy} w_{x_ly} (Q_{x_iy,x_ly} + Q_{x_ly,x_iy}) \\
& + \frac{1}{2}\sum_{i=1}^{k} P^{(t)}_{yy} P^{(t)}_{x_ix_l} w_{yy} w_{x_ix_l} (Q_{yy,x_ix_l} + Q_{x_ix_l,yy}) \\
& + \frac{1}{2} P^{(t)}_{yy} P^{(t)}_{x_lx_l} w_{yy} w_{x_lx_l} (Q_{yy,x_lx_l} + Q_{x_lx_l,yy}) \\
& + \frac{1}{2} P^{(t)}_{yy} P^{(t)}_{x_ly} w_{yy} w_{x_ly} (Q_{yy,x_ly} + Q_{x_ly,yy}) \Bigg]
\end{aligned} \tag{32}$$

$$\begin{aligned}
P^{(t+1)}_{yy} = \frac{1}{\bar{Q}\bar{w}^2_{E(t)}} \Bigg[& \frac{1}{4}\sum_{i=1}^{k}\sum_{\substack{j=1 \\ j\neq i}}^{k} P^{(t)}_{x_iy} P^{(t)}_{x_jy} w_{x_iy} w_{x_jy} (Q_{x_iy,x_jy} + Q_{x_jy,x_iy}) \\
& + \frac{1}{4}\sum_{i=1}^{k} (P^{(t)}_{x_iy})^2 w^2_{x_iy} Q_{x_iy,x_iy} + \frac{1}{2}\sum_{i=1}^{k} P^{(t)}_{yy} P^{(t)}_{x_iy} w_{yy} w_{x_iy} (Q_{yy,x_iy} + Q_{x_iy,yy}) \\
& + (P^{(t)}_{yy})^2 w^2_{yy} Q_{yy,yy} \Bigg]
\end{aligned}$$

where we used the shorthand notation $w_{x_ix_j}$ for $w_{E(t)}(\phi_{x_ix_j})$. To see whether y can invade when rare, we need to investigate the linearized dynamics

$$\mathbf{P}^{(t+1)} = \mathbf{A}\mathbf{P}^{(t)} \tag{33}$$

where $\mathbf{A}$ is the $(k+1)\times(k+1)$ Jacobian derived from (32) and evaluated at the trivial equilibrium $\mathbf{P} = \mathbf{0}$.

It is easily seen from (32) that each element in the last row of $\mathbf{A}$ is zero, but all other elements of $\mathbf{A}$ are strictly positive provided that $w_g > 0$ and $Q_{g,h} > 0$ for all genotypes g, h. With the Gaussian functions (26) and (22) used in our example, these conditions are satisfied if the allelic values are bounded and respectively $\sigma_s > 0$ and

$\sigma_m > 0$. Let $\mathbf{M}$ be the $k \times k$ matrix obtained from $\mathbf{A}$ by deleting its last row and last column. Since

$$\mathrm{Det}(\mathbf{A} - \lambda \mathbf{I}) = -\lambda \mathrm{Det}(\mathbf{M} - \lambda \mathbf{I}), \tag{34}$$

the eigenvalues of $\mathbf{A}$ are the eigenvalues of $\mathbf{M}$ and zero. Because $\mathbf{M}$ is strictly positive, the Perron-Frobenius theorem guarantees that the dominant eigenvalue of $\mathbf{M}$ is simple and strictly positive. The dominant eigenvalue of $\mathbf{A}$, which is the invasion fitness of the rare allele y, is therefore also simple and positive. If the dominant eigenvalue exceeds 1, y can invade.

Notice further that because the last row of $\mathbf{A}$ is zero, the last element of $\mathbf{P}^{(t+1)}$ in Eq. (33) is zero for all $t \geq 0$ irrespectively of the initial frequency vector $\mathbf{P}^{(0)}$. In contrast, the first k elements of $\mathbf{P}$ converge to the strictly positive eigenvector of $\mathbf{M}$ corresponding to its dominant eigenvalue. During the invasion process, therefore, the frequency of mutant homozygotes, which is the last element of $\mathbf{P}$, is negligible compared to the frequency of mutant heterozygotes in the first k elements of $\mathbf{P}$. In the main text, we denote the vector of the first k elements of $\mathbf{P}$ with $\mathbf{P}_{het}$.

Invasion fitness for monomorphic resident populations. When $k = 1$, the resident population consists entirely of homozygotes xx and, using that $Q_{xx,xx} w_{xx} = 1$ in resident equilibrium, (32) simplifies to

$$\begin{aligned}
P_{xy}^{(t+1)} = \frac{1}{w_{xx}} \Bigg[& \frac{1}{2} P_{xy}^{(t)} P_{xx}^{(t)} w_{xy} w_{xx} (Q_{xy,xx} + Q_{xx,xy}) \\
& + \frac{1}{2} (P_{xy}^{(t)})^2 w_{xy}^2 Q_{xy,xy} \\
& + P_{yy}^{(t)} P_{xx}^{(t)} w_{yy} w_{xx} (Q_{yy,xx} + Q_{xx,yy}) \\
& + \frac{1}{2} P_{yy}^{(t)} P_{xy}^{(t)} w_{yy} w_{xy} (Q_{yy,xy} + Q_{xy,yy}) \Bigg] \\
P_{yy}^{(t+1)} = \frac{1}{w_{xx}} \Bigg[& \frac{1}{4} (P_{xy}^{(t)})^2 w_{xy}^2 Q_{xy,xy} \\
& + \frac{1}{2} P_{yy}^{(t)} P_{xy}^{(t)} w_{yy} w_{xy} (Q_{yy,xy} + Q_{xy,yy}) \\
& + (P_{yy}^{(t)})^2 w_{yy}^2 Q_{yy,yy} \Bigg]
\end{aligned} \tag{35}$$

The Jacobian matrix is thus

$$\mathbf{A} = \begin{pmatrix} \frac{1}{2} w_{xy} (Q_{xy,xx} + Q_{xx,xy}) & w_{yy} (Q_{yy,xx} + Q_{xx,yy}) \\ 0 & 0 \end{pmatrix}. \tag{36}$$

$\mathbf{M}$ in this case is the upper left element of $\mathbf{A}$, which is also the dominant eigenvalue of $\mathbf{A}$ and equals the invasion fitness given in Eq. (11).

Invasion fitness for diallelic resident populations. When $\mathbf{M}$ is a 2×2 matrix, we can simplify our calculations using the Routh–Hurwitz criterion. Using also that $\mathbf{M}$ has positive elements, its eigenvalues λ_1 and λ_2 are both less than 1 in absolute value if

$$\mathrm{Det}\mathbf{M} < 1 \quad \text{and} \quad \mathrm{Tr}\mathbf{M} - \mathrm{Det}\mathbf{M} < 1. \tag{37}$$

$\mathbf{M}$ depends continuously on the allelic values (x_1, x_2) and on y. If y equals either x_1 or x_2 such that the mutant allele is neutral, then the dominant eigenvalue of $\mathbf{M}$ equals 1, and hence $\mathrm{Det}\mathbf{M} = \lambda_1\lambda_2 < 1$. By continuity, the first inequality of the Routh–Hurwitz criterion holds also when y is sufficiently close to either resident allele. With small mutations, therefore, it is the second inequality that determines whether a mutant can invade, and we can use $\mathrm{Tr}\mathbf{M} - \mathrm{Det}\mathbf{M}$ as a fitness proxy to see which mutants can invade a diallelic resident population [cf. Eq. (16)].

Appendix 2

Here we derive fitness during ecological selection in the Levene model [Eq. (23)]. The mating population consists of $K = K_1 + K_2$ individuals, who produce a total of $K\bar{Q}B$ offspring, where $\bar{Q}$ is the probability that a female is mated and B is the per capita fecundity (the number of offspring per mated female times the relative frequency of females in the population). Each offspring has probability γ_i to land in habitat i ($i = 1, 2$). An offspring with phenotype ϕ_g survives viability selection with probability $f_i(\phi_g)$ in habitat i, and then becomes one of the K_i individuals who survive till adulthood in habitat i with probability $K_i/[\gamma_i K\bar{Q}B\sum_h P_h f_i(\phi_h)]$ (where the denominator is the total number of offspring surviving viability selection and thus competing for the K_i places of adults). The product of the probability that a newborn survives till reproduction and the per capita fecundity is thus

$$w_{E(t)}(\phi_g) = \sum_i \gamma_i f_i(\phi_g) \frac{K_i}{\gamma_i K\bar{Q}B\sum_h P_h f_i(\phi_h)} B, \tag{38}$$

which, with $\bar{Q} = 1$, simplifies to Eq. (23) of the main text. Note that B is assumed to be large enough such that $K_i/[\gamma_i K\bar{Q}B\sum_h P_h f_i(\phi_h)]$ is always less than one to be a probability; such B can be found if the admissible allelic values are bounded, $\sigma_s > 0$, $\sigma_m > 0$, and $\gamma_i > 0$ for all i. Moreover, γ_i and B cancel in $w_{E(t)}(\phi_g)$ due to "contest" competition (i.e., a fixed number K_i of survivors) within each habitat.

Appendix 3

This Appendix reports the full numerical bifurcation analysis of diallelic singularities in the Levene model with equal habitat size ($c_1 = c_2 = 0.5$), and also shows how the degenerate bifurcations unfold when this symmetry assumption is relaxed.

Analogously to the monomorphic case, a diallelic evolutionary singularity (x_1^*, x_2^*) is determined by

$$\left.\frac{\partial W_{x_1,x_2}(y)}{\partial y}\right|_{\substack{y=x_i \\ x_1=x_1^*, x_2=x_2^*}} = 0 \quad \text{for } i = 1, 2 \tag{39}$$

and it is evolutionarily stable if

$$\left.\frac{\partial W^2_{x_1,x_2}(y)}{\partial y^2}\right|_{\substack{y=x_i \\ x_1=x_1^*,x_2=x_2^*}} < 0 \quad \text{for } i = 1, 2. \tag{40}$$

Convergence stability in more than one dimension may depend on the frequency and size of mutations (e.g. if allele x_1 mutates more frequently or with somewhat larger mutation steps than allele x_2). In this paper, we adopt the concept of "absolute convergence stability" (Leimar 2001, 2009) and rely on the conditions derived by Matessi and Di Pasquale (1996; see also Kisdi 2006). To formulate the criteria for convergence stability of a diallelic singularity, we introduce the notation

$$E_i = \left.\frac{\partial W^2_{x_1,x_2}(y)}{\partial y^2}\right|_{\substack{y=x_i \\ x_1=x_1^*,x_2=x_2^*}} \tag{41}$$

$$M_i = \left.\frac{\partial W^2_{x_1,x_2}(y)}{\partial x_i \partial y}\right|_{\substack{y=x_i \\ x_1=x_1^*,x_2=x_2^*}} \tag{42}$$

$$A_i = \left.\frac{\partial W^2_{x_1,x_2}(y)}{\partial x_j \partial y}\right|_{\substack{y=x_i \\ x_1=x_1^*,x_2=x_2^*}} \quad \text{with } j \neq i \tag{43}$$

for $i = 1, 2$. Matessi and Di Pasquale (1996) classified all generic singularities of a population with two co-evolving resident strategies or alleles into three groups:

(i) All possible allele substitution sequences starting in the neighbourhood of (x_1^*, x_2^*) converge to the singularity if

$$(E_1 + M_1)(E_2 + M_2) > |A_1 A_2| \quad \text{and} \quad E_1 + M_1 < 0, E_2 + M_2 < 0. \tag{44}$$

(ii) There exist both converging and diverging allele substitution sequences from every initial point in the neighbourhood of (x_1^*, x_2^*) if

$$|(E_1 + M_1)(E_2 + M_2)| < |A_1 A_2| \quad \text{and} \quad A_1 A_2 < 0. \tag{45}$$

(iii) In all other cases, every possible allele substitution sequence diverges from a non-zero measure set of initial points.

The above conditions are valid for all possible allele substitution sequences, including those that occur with vanishing probabilities. All singularities we found in the diallelic Levene model are either in (i) or in (iii), i.e., we have evolutionary attractors with absolute convergence stability and evolutionary repellers. We adopt the shorthand names "convergence stable" for singularities with absolute convergence stability and "saddle" for the repellers. The fitness proxy $\tilde{W}$ of Eq. (16) can be used in place of W in all of the conditions given above. We obtained the diallelic singularities of the Levene model by solving Eq. (39) numerically and evaluated the conditions of evolutionary and convergence stability as outlined above.

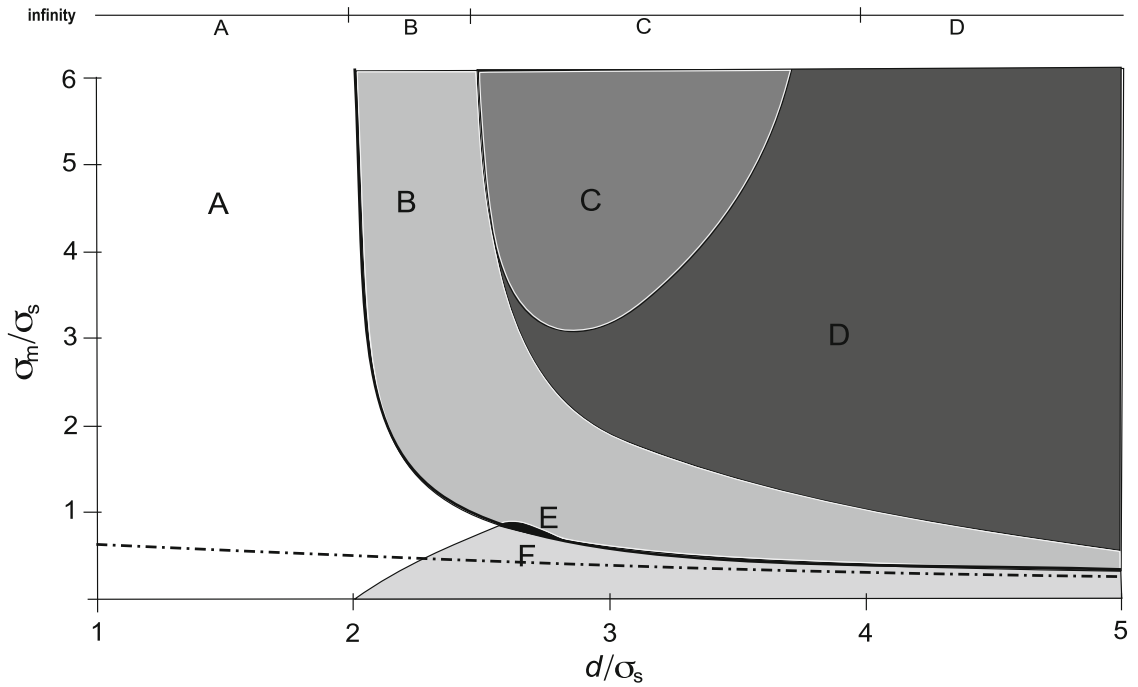

Fig. 8 Bifurcation plot of diallelic singularities for equal habitat size ($c_1 = c_2 = 0.5$). *Region A* no diallelic singularity, *B* one convergence stable singularity, *C* a pair of asymmetric convergence stable singularities separated by a symmetric saddle point, *D* three convergence stable singularities (one in symmetric position and a pair of asymmetric singularities) separated by a pair of asymmetric saddles, *E* two symmetric convergence stable singularities separated by a symmetric saddle, *F* one symmetric convergence stable singularity and one symmetric saddle. *Regions A–D* extend to σ_m/σ_s at infinity as indicated. All convergence stable singularities of this plot are also evolutionarily stable. The *thick line* coincides with the bifurcation line of the monomorphic singularity: $x^* = 0$ is evolutionarily stable in *A* and *F* whereas it is an evolutionary branching point elsewhere. The *dash-dotted line* is same as in Fig. 2

Figure 8 shows the bifurcations of diallelic singularities for equal habitat size ($c_1 = c_2 = 0.5$). There can be up to five diallelic singularities (three convergence stable singularities separated by two saddle points). All convergence stable diallelic singularities found in this bifurcation plot are also evolutionarily stable, hence further branching (leading to tri-allelic polymorphisms) does not occur. (Note however that with substantially unequal habitat sizes, further branching does occur, as found in a narrow range of parameters under random mating by Kisdi and Geritz 1999.)

In region A, there is no diallelic singularity, and two resident alleles are always subject to convergent coevolution (see Fig. 3c for an example). When crossing the bifurcation line to region B, the monomorphic singularity $x^* = 0$ becomes an evolutionary branching point and, simultaneously, a diallelic convergence stable singularity is born with $(x_1^*, x_2^*) = (x^*, x^*)$ at the bifurcation. Within region B, evolutionary branching yields two distinct alleles and these evolve to the unique convergence stable diallelic singularity with symmetric allelic values $x_1^* = -x_2^*$ (as in Fig. 3a, b).

Between regions B and C, a pitchfork bifurcation occurs such that the symmetric convergence stable diallelic singularity of region B becomes a saddle in region C, and two new convergence stable singularities arise, which occupy asymmetric positions but are mirror images of each other such that $x_1^{*(1)} = -x_2^{*(2)}$ and $x_1^{*(2)} = -x_2^{*(1)}$ (compare Figs. 3a, 4a for an example). When crossing from region C to region D, the

symmetric singularity undergoes another pitchfork bifurcation such that it becomes convergence stable again, and two saddles arise that separate the basin of attraction of the symmetric singularity from the two asymmetric convergence stable singularities (compare panels a and b in Fig. 5). The bifurcations described so far occur also with random mating ($\sigma_m/\sigma_s \to \infty$) and are therefore identical to those discussed by Kisdi and Geritz (1999).

At moderate values of σ_m/σ_s, region B (with a single symmetric diallelic singularity) and region D (with three convergence stable singularities and two saddles) have adjacent parts, which are separated by a line of two simultaneous fold bifurcations. Each fold bifurcation involves an asymmetric convergence stable singularity and a saddle point, leaving the symmetric convergence stable singularity unchanged (compare Fig. 5c,d for an example).

There is another fold bifurcation that is independent of all the above, and separates regions A from F and B from E. The convergence stable singularity and the saddle born on this bifurcation line are both in symmetric position. In region F, there are no other singularities but this pair (as in Fig. 3g), whereas in region E, the pair coexists with the symmetric convergence stable singularity also present in region B (as in Fig. 4e). When crossing from E to F, a convergence stable singularity collides with the monomorphic singularity, and simultaneously the latter becomes an ESS (compare Fig. 4f, g), whereas between B and F, a diallelic saddle interacts with the monomorphic singularity (compare Fig. 5f, g).

In the limit of very strong mating assortativity ($\sigma_m \to 0$), the homozygotes are fully isolated and heterozygotes are absent at any diallelic singularity isolated from $(x^*, x^*) = (0, 0)$. Hence the resident population contains two phenotypes just as a dimorphic clonal population; and since assortativity with $M \to \infty$ implies $\bar{Q} = 1$ in Eq. (3), the population densities of the two phenotypes are also the same. Because $\pi'(0) = 0$, the selection gradient is not affected by sexual selection [cf. Eq. (17)]. The two homozygote subpopulations therefore evolve exactly as two clonal phenotypes evolve in absence of sexual reproduction, and the diallelic singularities coincide with the clonal dimorphic singularities. In particular, the clonal version of the Levene model with equal habitat size has a dimorphic singularity for $d/\sigma_s > 2$ (Geritz and Kisdi 2000). Accordingly, in our model, the diallelic singularity in region F disappears at $d/\sigma_s = 2$ when $\sigma_m \to 0$. Note that $d/\sigma_s > 2$ is also the condition for evolutionary branching under random mating ($\sigma_m \to \infty$), which is always the same as the condition for branching in the corresponding clonal model (Van Dooren in press). However, the condition for clonal evolutionary branching coincides with the condition for having a dimorphic singularity only because of equal habitat size (Kisdi and Geritz 1999; see below).

Unequal habitat size. The pitchfork bifurcations found above are degeneracies due to equal habitat size, and they unfold into fold bifurcations producing a convergence stable singularity and a saddle independently of an existing singularity. The simultaneous fold bifurcations between regions B and D separate into two simple fold bifurcations when habitat sizes are not precisely equal. This is shown in Fig. 9: The lines of pitchfork bifurcations (between B and C and between C and D, respectively) and the line of simultaneous fold bifurcation (between B and D) are all replaced by

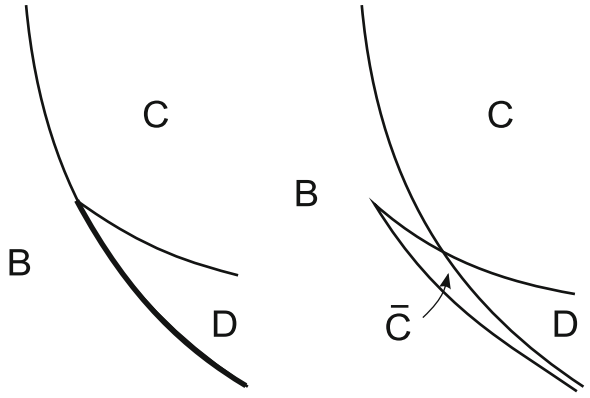

Fig. 9 Detail of the bifurcation plot for equal habitat size in Fig. 8 (*left*) and the same for c_1 close but not equal to 0.5 (*right*). With equal habitat size, pitchfork bifurcations separate *B* from *C* and *C* from *D* (*thin lines*), and two simultaneous fold bifurcations occur between *B* and *D* (*thick line*). With unequal habitat size, the simultaneous fold bifurcations separate and the pitchfork bifurcations are replaced by fold bifurcations. In the new region $\bar{C}$ that opens up between *B* and *D*, there are two convergence stable singularities separated by a saddle. A cusp bifurcation occurs at the peak of $\bar{C}$

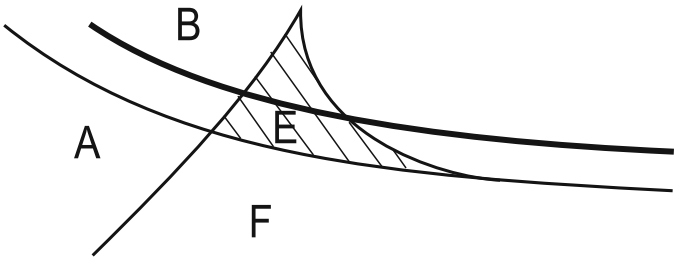

Fig. 10 Detail of the bifurcation plot with slightly unequal habitat sizes. The *thick line* is the bifurcation line of the monomorphic singularity such that $x^* = 0$ is an ESS below and an evolutionary branching point above the line. A convergence stable diallelic singularity appears across the *thin line* and coexists with the monomorphic ESS in the *band-shaped region* below the *thick line*; the ESS-branching point bifurcation of the monomorphic singularity no longer coincides with the boundary between *A* and *B* (see Fig. 8). Another pair of a convergence stable singularity and a saddle are born across the fold bifurcation line on the boundary of regions *E* and *F*

lines of fold bifurcations, and a new region, with two convergence stable singularities and a saddle, emerges between regions B and D.

Another type of degeneracy emerges because equal habitat size implies symmetry about the point $x^* = 0$, such that any diallelic singularities must exist either in symmetric pairs (such as $x_1^{*(1)} = -x_2^{*(2)}$ and $x_1^{*(2)} = -x_2^{*(1)}$) or in a symmetric position (with $x_1^* = -x_2^*$). Singularities which are forced to retain their symmetric position collide with the monomorphic singularity ($(x_1^*, x_2^*) \to (x^*, x^*)$), and therefore changes in the evolutionary stability of the monomorphic singularity x^* are linked to the appearance of a diallelic singularity. As a result, the bifurcation line in Fig. 8 on which the monomorphic singularity changes between an ESS and an evolutionary branching point (thick line) coincides with the boundary between regions A and B, between E and F, and between B and F. The unfolding of this degeneracy is shown in Fig. 10: When habitat size is perturbed, a diallelic singularity exists in a band of the parameter space outside the region where the monomorphic singularity is a branching point (and instead of colliding with the monomorphic singularity, it

appears from across the extinction boundary delineating the set of possible diallelic polymorphisms). This pattern corresponds to what is expected in generic models of adaptive dynamics (see the Appendix of Geritz et al. 1999), and implies an evolutionary hysteresis effect (Kisdi and Geritz 1999): Suppose a polymorphic population exists at the diallelic singularity within the band where x^* is an ESS. If this population loses an allele due to a temporary change in its environment, then polymorphism cannot be regained without a greater change in the environment that not only restores the existence of a diallelic singularity but also makes x^* an evolutionary branching point.

References

Bodmer WF (1965) Differential fertility in population genetic models. Genetics 51:411–424

Bolnick DI, Fitzpatrick BM (2007) Sympatric speciation: models and empirical evidence. Annu Rev Ecol Evol Syst 38:459–487

Bürger R (2010) Evolution and polymorphism in the multilocus Levene model with no or weak epistasis. Theor Pop Biol 78:123–138

Caisse M, Antonovics J (1978) Evolution in closely adjacent populations. Heredity 40:371–384

Christiansen FB (1991) On conditions for evolutionary stability for a continuously varying character. Am Nat 138:37–50

Coyne JA, Orr HA (2004) Speciation. Sinauer Associates Inc., Sunderland

Cruz R, Carballo M, Conde-Padin P, Rolan-Alvarez E (2004) Testing alternative models for sexual isolation in natural populations of Littorina saxatilis: indirect support for by-product ecological speciation? J Evol Biol 17:288–293

Day T (2000) Competition and the effect of spatial resource heterogeneity on evolutionary diversification. Am Nat 155:790–803

Dickinson H, Antonovics J (1973) Theoretical considerations of sympatric divergence. Am Nat 107: 256–274

Doebeli M (1996) A quantitative genetic competition model for sympatric speciation. J Evol Biol 9:893–909

Eshel I (1983) Evolutionary and continuous stability. J Theor Biol 103:99–111

Feder JL (1998) The apple maggot fly, Rhagoletis pomonella: flies in the face of conventional wisdom about speciation? In: Howard DJ, Berlocher SH (eds) Endless forms: species and speciation, Oxford University Press, Oxford, pp 130–144

Felsenstein J (1981) Scepticism towards Santa Rosalia, or why are there so few kinds of animals? Evolution 35:124–138

Gavrilets S (1998) One-locus two-allele models with maternal (parental) selection. Genetics 149: 1147–1152

Gavrilets S (2004) Fitness landscapes and the origin of species. Princeton University Press, Princeton

Gavrilets S, Boake CRB (1998) On the evolution of premating isolation after a founder event. Am Nat 152:706–716

Gegear RJ, Burns JG (2007) The birds, the bees, and the virtual flowers: can pollinator behavior drive ecological speciation in flowering plants? Am Nat 170:551–566

Geritz SAH (2005) Resident-invader dynamics and the coexistence of similar strategies. J Math Biol 50: 67–82

Geritz SAH, Kisdi E (2000) Adaptive dynamics in diploid, sexual populations and the evolution of reproductive isolation. Proc R Soc Lond B 267:1671–1678

Geritz SAH, Gyllenberg M, Jacobs FJA, Parvinen K (2002) Invasion dynamics and attractor inheritance. J Math Biol 44:548–560

Geritz SAH, Kisdi É, Meszéna G, Metz JAJ (1998) Evolutionarily singular strategies and the adaptive growth and branching of the evolutioary tree. Evol Ecol 12:35–57

Geritz SAH, van der Meijden E, Metz JAJ (1999) Evolutionary dynamics of seed size and seedling competitive ability. Theor Pop Biol 55:324–343

Geritz SAH, Metz JAJ, Kisdi E, Meszéna G (1997) Dynamics of adaptation and evolutionary branching. Phys Rev Lett 78:2024–2027

Gyllenberg M, Parvinen K (2001) Necessary and sufficient conditions for evolutionary suicide. Bull Math Biol 63:981–993
Hadeler KP, Liberman U (1975) Selection models with fertility differences. J Theor Biol 2:19–32
Hartl DL, Clark AG (1989) Principles of population genetics. Sinauer Associates Inc., Sunderland
Hatfield T, Schluter D (1999) Ecological speciation in sticklebacks: environment-dependent hybrid fitness. Evolution 53:866–873
Hegde SN, Krishna MS (1997) Size-assortative mating in *Drosophila malerkotliana*. Anim Behav 54: 419–426
Huber SK, De Leon LF, Hendry AP, Bermingham E, Podos J (2007) Reproductive isolation of sympatric morphs in a population of Darwin's finches. Proc R Soc Lond B 274:1709–1714
Jagers P (1975) Branching processes with biological applications. Wiley, London
Jiggins CD, Estrada C, Rodrigues A (2004) Mimicry and the evolution of premating isolation in Heliconius melpomene Linneaeus. J Evol Biol 17:680–691
Jiggins CD, Naisbit RE, Coe RL, Mallet J (2001) Reproductive isolation caused by colour pattern mimicry. Nature 411:302–305
Jones AG, Moore GI, Kvarnemo C, Walker D, Avise JC (2003) Sympatric speciation as a consequence of male pregnancy in seahorses. Proc Natl Acad Sci USA 100:6598–6603
Kingston T, Rossiter SJ (2004) Harmonic-hopping in Wallacea's bats. Nature 429:654–657
Kirkpatrick M, Nuismer SL (2004) Sexual selection can constrain sympatric speciation. Proc R Soc Lond B 271:687–693
Kirkpatrick M, Ravigne V (2002) Speciation by natural and sexual selection: models and experiments. Am Nat 159:S22–S35
Kisdi É (2001) Long-term adaptive diversity in Levene-type models. Evol Ecol Res 3:721–727
Kisdi É (2002) Dispersal: risk spreading versus local adaptation. Am Nat 159:579–596
Kisdi É (2006) Trade-off geometries and the adaptive dynamics of two coevolving species. Evol Ecol Res 8:959–973
Kisdi É, Geritz SAH (1999) Adaptive dynamics in allele space: evolution of genetic polymorphism by small mutations in a heterogeneous environment. Evolution 53:993–1008
Kisdi É., Geritz SAH (in press) Evolutionary branching and speciation: Insights from fewlocus models. In: Dieckmann U, Metz JAJ (eds) Elements of adaptive dynamics. Cambridge University Press, Cambridge
Kopp M, Hermisson J (2008) Competitive speciation and costs of choosiness. J Evol Biol 21:1005–1023
Leimar O (2001) Evolutionary change and Darwinian demons. Selection 2:65–72
Leimar O (2009) Multidimensional convergence stability. Evol Ecol Res 11:191–208
Leimar O (in press) Multidimensional convergence stability and the canonical adaptive dynamics. In: Dieckmann U, Metz JAJ (eds) Elements of adaptive dynamics. Cambridge University Press, Cambridge
Levene H (1953) Genetic equilibrium when more than one ecological niche is available. Am Nat 87: 331–333
Macnair MR, Gardner M (1998) The evolution of edaphic endemics. In: Howard DJ, Berlocher SH (eds) Endless forms: species and speciation. Oxford University Press, Oxford, pp 157–171
Matessi C, Di Pasquale C (1996) Long-term evolution of multilocus traits. J Math Biol 34:613–653
Matessi C, Gimelfarb A, Gavrilets S (2001) Long-term buildup of reproductive isolation promoted by disruptive selection: how far does it go? Selection 2:41–64
Maynard Smith J (1966) Sympatric speciation. Am Nat 100:637–650
Maynard Smith J (1982) Evolution and the theory of games. Cambridge University Press, Cambridge
McPeek MA, Wellborn GA (1998) Genetic variation and reproductive isolation among phenotypically divergent amphipod populations. Limnol Oceanogr 43:1162–1169
Meszéna G, Czibula I, Geritz SAH (1997) Adaptive dynamics in a 2-patch environment: a toy model for allopatric and parapatric speciation. J Biol Syst 5:265–284
Metz JAJ, Leimar O (2010) A simple fitness proxy for structured populations with continuous traits, with case studies on the evolution of haplo-diploids and genetic dimorphisms. J Biol Dyn (in press)
Nagel L, Schluter D (1998) Body size, natural selection, and speciation in sticklebacks. Evolution 52: 209–218
Nagylaki T (2009) Polymorphism in multiallelic migration-selection models with dominance. Theor Pop Biol 75:239–259
Nagylaki T, Lou Y (2001) Patterns of multiallelic polymorphism maintained by migration and selection. Theor Pop Biol 59:297–313

Nagylaki T, Lou Y (2006) Evolution under the multiallelic Levene Model. Theor Pop Biol 70:401–411
Orr HA (2001) The genetics of species differences. Trends Ecol Evol 16:343–350
Otto SP, Servedio MR, Nuismer SL (2008) Frequency-dependent selection and the evolution of assortative mating. Genetics 179:2091–2112
Pennings PS, Kopp M, Meszéna G, Dieckmann U, Hermisson J (2008) An analytically tractable model for competitive speciation. Am Nat 171:E44–E71
Podos J (2001) Correlated evolution of morphology and vocal signal structure in Darwin's finches. Nature 409:185–188
Puebla O, Bermingham E, Guichard F, Whiteman E (2007) Colour pattern as a single trait driving speciation in Hypoplectrus coral reef fishes? Proc R Soc Lond B 274:1265–1271
Ravigne V, Dieckmann U, Olivieri I (2009) Live where you thrive: joint evolution of habitat choice and local adaptation facilitates specialization and promotes diversity. Am Nat 174:E141–E169
Ripa J (2009) When is sympatric speciation truly adaptive? An analysis of the joint evolution of resourve utilization and assortative mating. Evol Ecol 23:31–52
Roughgarden J (1979) Theory of population genetics and evolutionary ecology: an introduction. MacMillan, New York
Rundle HD (2002) A test of ecologically dependent postmating isolation between sympatric sticklebacks. Evolution 56:322–329
Schneider KA (2005) Competitive divergence in non-random mating populations. Theor Pop Biol 68: 105–118
Schneider KA, Bürger R (2006) Does competitive divergence occur if assortative mating is costly? J Evol Biol 19:570–588
Seehausen O, van Alphen JJM, Witte F (1997) Cichlid fish diversity threatened by eutrophication that curbs sexual selection. Science 277:1808–1811
Udovic D (1980) Frequency-dependent selection, disruptive selection, and the evolution of reproductive isolation. Am Nat 116:621–641
Van Dooren TJM (1999) The evolutionary ecology of dominance-recessivity. J Theor Biol 198:519–532
Van Dooren TJM (2000) The evolutionary dynamics of direct phenotypic overdominance: emergence possible, loss probable. Evolution 54:1899–1914
Van Dooren TJM (in press) Adaptive dynamics for Mendelian genetics. In: Dieckmann U, Metz JAJ (eds) Elements of adaptive dynamics. Cambridge University Press, Cambridge
Van Doorn S, Dieckmann U (2006) The long-term evolution of multi-locus traits under frequency-dependent disruptive selection. Evolution 60:2226–2238
Waser NM, Campbell DR (2004) Ecological speciation in flowering plants. In: Dieckmann U, Doebeli M, Metz JAJ, Tautz D (eds) Adaptive speciation. Cambridge University Press, Cambridge, pp 264–277
Wellborn GA (1994) Size-biased predation and prey life histories: a comparative study of freshwater amphipod populations. Ecology 75:2104–2117
Woodruff RC, Thompson JN Jr (2002) Mutation and premating isolation. Genetica 116:371–382

Article II

On invasion boundaries and the unprotected coexistence of two strategies

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On invasion boundaries and the unprotected coexistence of two strategies

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Abstract In this paper we present, in terms of invasion fitness functions, a sufficient condition for a coexistence of two strategies which are not protected from extinction when rare. In addition, we connect the result to the local characterization of singular strategies in the theory of adaptive dynamics. We conclude with some illustrative examples.

Mathematics Subject Classification (2000) 37N25 · 92B05

1 Introduction

Of focal interest in studies on populations is the identification of conditions that guarantee the formation and maintenance of genetic and phenotypic variation. Often, however, the dynamics of populations is so complex that it is unfeasible to determine the existence of attractors at which polymorphisms can be established. This task is substantially simplified if we consider a population of only one or two strategies. For example, a sufficient condition for the coexistence of two alleles can be given in terms of *invasibility*: if both alleles can increase their frequency when rare (mutual invasibility), they will both be maintained in the population. This type of coexistence is known as protected coexistence (Prout 1968; Poulsen 1979), since both alleles are protected from extinction. Note that the condition for protected coexistence of two alleles is obtained using invasion criteria alone, that is, without studying the full genetic dynamical system. This is because the dynamics of two alleles can often be reduced to a one-dimensional genetic state space. However, the concept of protected coexis-

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tence can be extended to populations with two dimensional population state spaces whenever the population size is bounded and each monomorphic (sub)population has a global attractor (Metz et al. 1996). Furthermore, Geritz et al. (2002) and Geritz (2005) showed that if monomorphic populations have similar strategies, generically the coexistence of strategies can *only* be protected.

In this paper, we give a sufficient condition for the stable coexistence of two strategies that *lack* mutual invasibility (unprotected coexistence). This result is derived using invasion criteria alone for the case when monomorphic populations have a global attractor. Further we show that in the characterization of singular strategies (Geritz et al. 1998; Geritz 2005) one particular degenerate case unfolds as an unprotected coexistence of two strategies.

2 Preliminaries

Let the strategy space be $\mathbb{X} \subset \mathbb{R}$, the space of non-negative population sizes $\mathbb{P} = \mathbb{R}_+$ and let the space of environmental conditions $\mathbb{E}$ be a subset of a normed vector space. Taking the space of time $\mathbb{T}$ to be either discrete or continuous, $\mathbb{T} = \mathbb{Z}_+$ or $\mathbb{R}_+$, we can define an environment as a map $E : \mathbb{T} \to \mathbb{E}$.

Consider a population of two strategies $x, y \in \mathbb{X}$ with corresponding densities $N, M \in \mathbb{P}$ in an environment E, and suppose the dynamics is given by a continuous time system

$$\begin{aligned} \dot{N}(t) &= F_\mu(x, E(t))N(t) \\ \dot{M}(t) &= F_\mu(y, E(t))M(t) \end{aligned} \tag{1}$$

or a discrete time system

$$\begin{aligned} N(t+1) &= G_\mu(x, E(t))N(t) \\ M(t+1) &= G_\mu(y, E(t))M(t), \end{aligned} \tag{2}$$

where F_μ, G_μ are some continuous and sufficiently smooth functions and $\mu \in \mathbb{R}^k$ is an auxiliary parameter. An environment E contains all factors that influence population growth, including the effect what population itself has on the environment, and hence it may be a function of strategies x, y and population densities N, M (Metz et al. 1992; Gyllenberg and Metz 2001; Geritz 2005).

Let $E_x(t)$ denote the environment determined by a single strategy x at time t and assume that there exists an invasion fitness function $\sigma_{E_x}(y)$ for a strategy y (Metz et al. 1992). Whether strategy y can invade an environment set by strategy x depends on the sign of the invasion fitness function: when invasion fitness is positive, $\sigma_{E_x}(y) > 0$, strategy y can invade and when invasion fitness is negative, $\sigma_{E_x}(y) < 0$, it can't. The area of invasion can be represented graphically with a so-called pairwise invasibility plot (PIP) (Christiansen and Loeschke 1980; Motro 1982; Matsuda 1985; van Tienderen and de Jong 1986; Kisdi and Meszéna 1993, 1995; Metz et al. 1996; Dieckmann 1997; Geritz et al. 1998; Claessen and Dieckmann 2002; Doebeli et al. 2007). An example of a PIP is illustrated in Fig. 1a. The curve where the sign of the

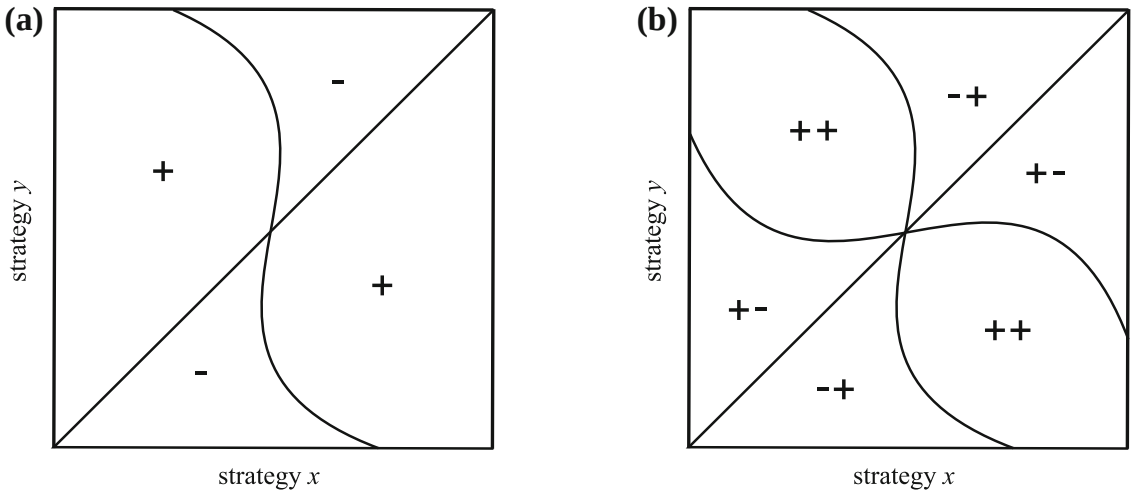

Fig. 1 **a** An example of a pairwise invasibility plot (PIP). The '+' indicates the area where y can invade ($\sigma_{E_x}(y) > 0$) and '−' where it cannot ($\sigma_{E_x}(y) < 0$). The curve where the sign of the invasion fitness function $\sigma_{E_x}(y)$ changes is an invasion boundary I_1. **b** An example of a mutual invasibility plot (MIP). In each pair of symbols '+−', '++', '−+' and '−−' the first symbol indicates the sign of $\sigma_{E_x}(y)$ and the second symbol the sign of $\sigma_{E_y}(x)$. The curves where the sign of $\sigma_{E_x}(y)$ and $\sigma_{E_y}(x)$ changes are the invasion boundaries I_1 and I_2, respectively

invasion fitness function changes is called an *invasion boundary* (Ferriere and Gatto 1993; Rueffler et al. 2004). More precisely, an invasion boundary $I \subset \mathbb{X}^2$ is constructed from all the points $(x, y) \in \mathbb{X}^2$ such that for each point $(x, y) \in I$ there exists a one dimensional manifold T in the strategy plane $\mathbb{X}^2$ which passes (x, y) so that the invasion fitness function changes sign along this manifold T. We denote with I_1 and I_2 the invasion boundaries which are generated by invasion fitness functions $\sigma_{E_x}(y)$ and $\sigma_{E_y}(x)$, respectively. Note that when crossing an invasion boundary between an area of invasion and noninvasion the stability of the corresponding boundary steady state changes. We exclude the highly unlikely case that an invasion boundary coincides with a bifurcation curve of any other (non-boundary) steady state of the system.

The area where strategies x and y can invade each other, that is, the area where both invasion fitness functions $\sigma_{E_x}(y)$ and $\sigma_{E_y}(x)$ are positive, can be visualized by taking the mirror image of a PIP along its main diagonal and superimposing it on the original (see Fig. 1b). This plot is known as a mutual invasibility plot (MIP). When a system lacks mutual invasibility but strategies can nevertheless coexist we call it an *unprotected coexistence*: population is not protected against extinction as it can be perturbed into a basin of a boundary attractor.

From now on, if not mentioned otherwise, we assume that the demographic attractor of a population is an equilibrium point and that strategies x and y define unique environments E_x and E_y, respectively. If an environment $\hat{E}_x$ corresponds to the equilibrium $(N, M) = (\hat{N}, 0)$ we can define

$$s_x(y) = \sigma_{\hat{E}_x}(y) \tag{3}$$

as the invasion fitness of y in a population of x at the equilibrium. As $(\hat{N}, 0)$ and $(0, \hat{M})$ are globally stable equilibria for the dynamics confined to their corresponding

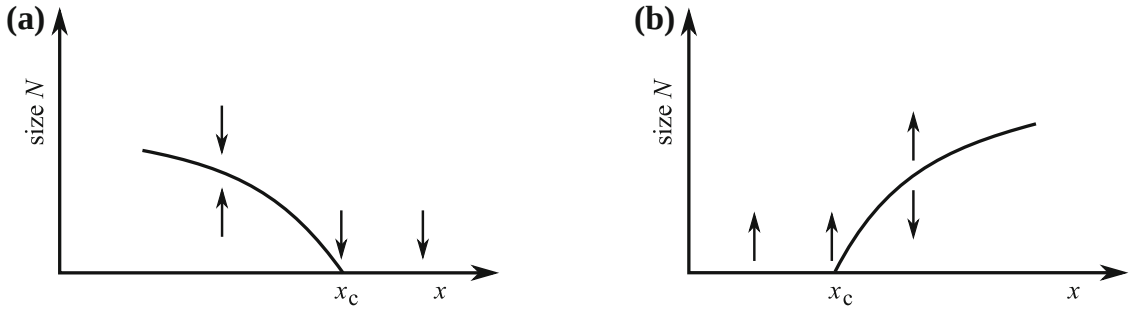

Fig. 2 Examples of two classes of bifurcations of a generic system (1) or (2) at x_c. **a** Supercritical bifurcation: a stable continuous interior (*positive*) equilibrium solution passes transversally a zero equilibrium. **b** Subcritical bifurcation: an unstable continuous interior equilibrium solution passes transversally a zero equilibrium

boundaries, the area of protected coexistence of x and y is defined as a subset of $\mathbb{X}^2$ where $s_x(y) > 0$ and $s_y(x) > 0$.

Finally, system (1) or (2) is called *generic* if at a bifurcation point it satisfies a finite number of genericity conditions (see e.g. Kuznetsov 1998 p. 66). Here it will suffice that the genericity conditions at a bifurcation point guarantee continuity and transversality (non-tangentiality) of equilibrium solutions to (1) or (2). In particular, we say that system (1) or (2) is generic at some point (x, y) on an invasion boundary (or alternatively we say that the point (x, y) itself is generic), if non-boundary equilibrium solutions to (1) or (2) pass the corresponding boundary equilibrium solution at (x, y) continuously and transversally. Now, if the system is generic on an invasion boundary the bifurcations happening on it can be of only two types, *super-* and *subcritical*. We say that the bifurcation is *supercritical* if at the bifurcation point the boundary equilibrium is stable from the interior of the population state space, and *subcritical* if the bifurcation point at the boundary equilibrium is unstable from the interior of the population state space (see Fig. 2). Further, all the bifurcations where at least one stable node and one saddle are created anew we call *saddle-node* after the most generic bifurcation of this type.

3 Results

Our first result gives a sufficient condition for unprotected coexistence of two strategies. The proof is given in the Appendix.

Theorem 1 *Suppose that invasion boundaries I_1 and I_2 of system* (1) *or* (2) *exist and intersect transversally at point $z = (x_0, y_0) \in \mathbb{X}^2$, where $x_0 \neq y_0$. Then, if system* (1) *or* (2) *is generic at z, there exists a neighborhood of z where strategies are in unprotected coexistence.*

In Fig. 3 we give two examples of MIPs where invasion boundaries I_1 and I_2 intersect transversally away from the diagonal $y = x$ (at $y = x$ the system is by definition degenerate). Now, Theorem 1 says, that if the system is generic at the intersection of invasion boundaries I_1 and I_2, then near the intersection point strategies x and y can coexist despite a negative invasion fitness function. Note that the result depends solely

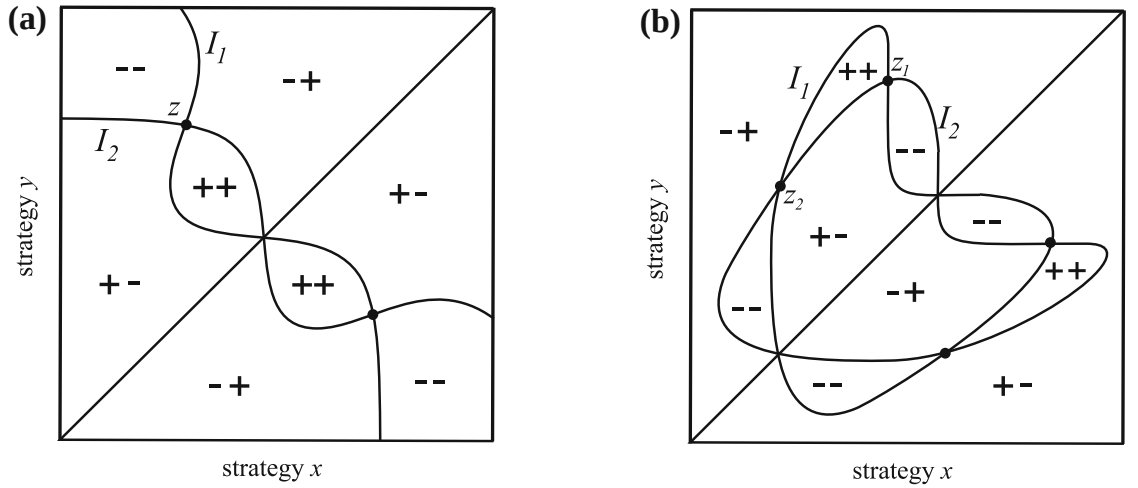

Fig. 3 Two examples of MIPs where invasion boundaries I_1 and I_2 intersect transversally. **a** A single intersection above the diagonal $y = x$, denoted with z. **b** Two intersections above the diagonal $y = x$, denoted with z_1 and z_2. In *both panels* identical intersections are also found below the diagonal because MIPs are symmetric about $y = x$

on the boundary conditions of the population state space, in other words, there is no need for further information on the dynamics in the interior of the population state space (int$\mathbb{P}^2$).

The next result says that if near a singular strategy x^* an area of mutual invasibility MI ($s_x(y) > 0$ and $s_y(x) > 0$) bifurcates into an area of mutual exclusion ME ($s_x(y) < 0$ and $s_y(x) < 0$), or vice versa, then near the bifurcation point invasion boundaries I_1 and I_2 intersect. The proof is given in the Appendix.

Theorem 2 *Suppose that for* (1) *or* (2) *invasion fitness functions* $s_x(y)$, $s_y(x)$ *and a monomorphic singular strategy* x^* *exist, and that there is a model parameter* μ *such that the cross-derivative* $D_{12}s_{x^*}(x^*)$ *changes sign at* $\mu = \mu_0$. *Then there are invasion boundaries* I_1 *and* I_2 *which intersect in the neighborhood of* x^* *and* μ_0. *If* $I_1 \neq I_2$ *at* μ_0, *the intersection is transversal.*

Combining Theorems 1 and 2, it follows that in the neighborhood of a bifurcation point μ_0 between MI and ME near x^* there exists a neighborhood of x^* where strategies x and y are in unprotected coexistence, provided that $I_1 \neq I_2$ at μ_0 and that the system (1) or (2) is generic at the intersection point of I_1 and I_2. Note that the intersection point gets arbitrarily close to x^* as μ approaches μ_0, or alternatively, as $D_{12}s_{x^*}(x^*)$ approaches 0. This fact complements the Classification Theorem of Geritz (2005), which categorizes the possible types of dynamical behavior when x is close to y, given that $D_{12}s_x(x) \neq 0$ (and some minor technical conditions). In particular, the Classification Theorem excludes the possibility of unprotected coexistence in a narrow strip around the main diagonal ($x = y$). However, when $D_{12}s_{x^*}(x^*)$ approaches 0, then near x^* the width of the strip also approaches 0. Thus, Theorems 1 and 2 show under the assumptions made within, that outside of this strip there exists an area of unprotected coexistence whenever $D_{12}s_{x^*}(x^*)$ has changed sign but is still close to 0 (either > 0 or < 0 depending on which side the intersection of invasion boundaries exists).

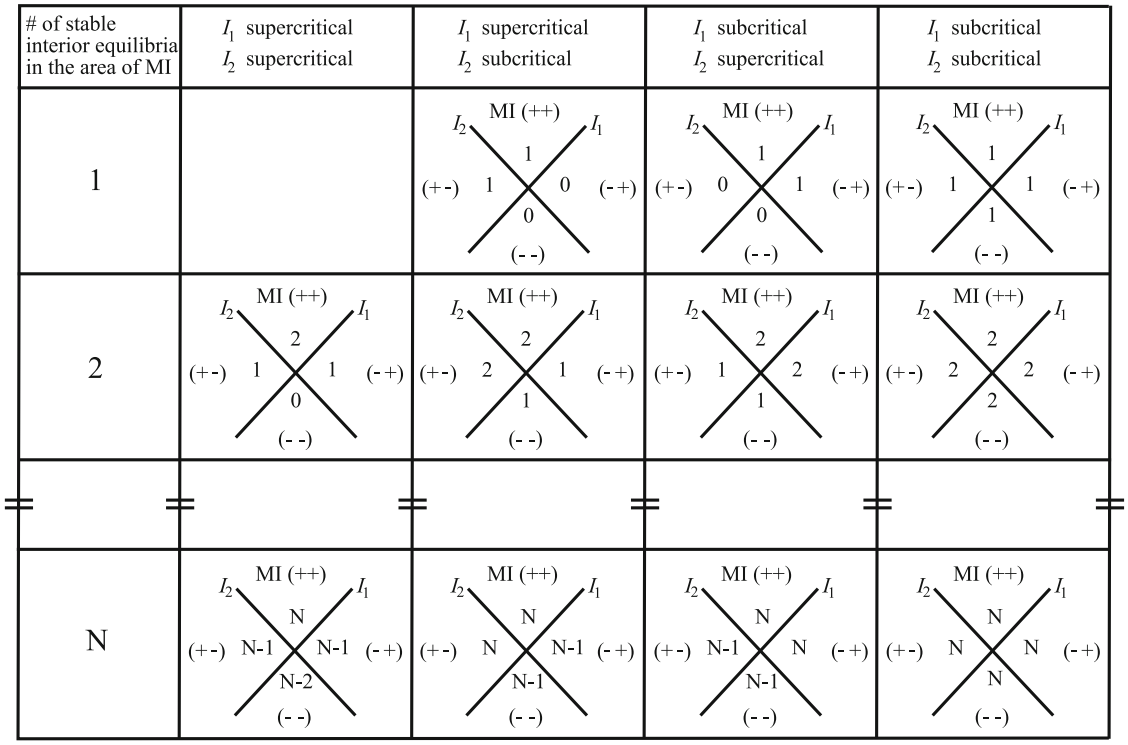

Fig. 4 All the possible cases of unprotected coexistence in the neighborhood of a transversal intersection of invasion boundaries I_1 and I_2, when at the intersection point invasion boundaries are either super- or subcritical. We assume that at most one interior equilibrium passes the boundary equilibrium on each invasion boundary, and that the intersection point is not a bifurcation point between super- and subcritical bifurcations. The *leftmost column* gives the number of interior stable equilibria in the area of mutual invasibility, and in *each row* we show four different cases depending on the type of a bifurcation on I_1 and I_2. Within each frame, the signs in the *brackets* indicate the signs of $s_x(y)$ and $s_y(x)$, respectively, and the *numbers* indicate how many stable interior equilibria exist in the corresponding invasion region. Notice that when there is only one stable interior equilibrium in the area of MI, one of the invasion boundaries must be subcritical (see Appendix)

In Fig. 4 we collected all the configurations of transversal intersections of invasion boundaries I_1 and I_2 for the simplest case where invasion boundaries do not bifurcate between super- and subcritical bifurcations at a generic intersection point z, and where exactly one non-boundary equilibrium bifurcates with the boundary equilibrium. Notice that the upper left frame is empty because that configuration does not exist: when there is exactly one stable interior equilibrium in the area of mutual invasibility at least one of the invasion boundaries must be subcritical (Appendix). In each frame, the numbers around the intersection give the number of interior stable equilibria in the corresponding invasion regions. For example, when there are two stable interior equilibria in the area of mutual invasibility, and I_1 is subcritical and I_2 is supercritical, there exists one interior stable equilibrium in areas $(+-)$ and $(--)$, and two stable interior equilibria in area $(-+)$. All the configurations in Fig. 4 are direct consequences of the continuity of solutions and the boundary bifurcations that lead to Theorem 1.

In Fig. 5 we show bifurcations from MI to ME near a singular strategy x^* for a system with at most one interior stable equilibrium, supposing that with the appearance of ME the two invasion boundaries I_1 and I_2 connected to x^* intersect transversally

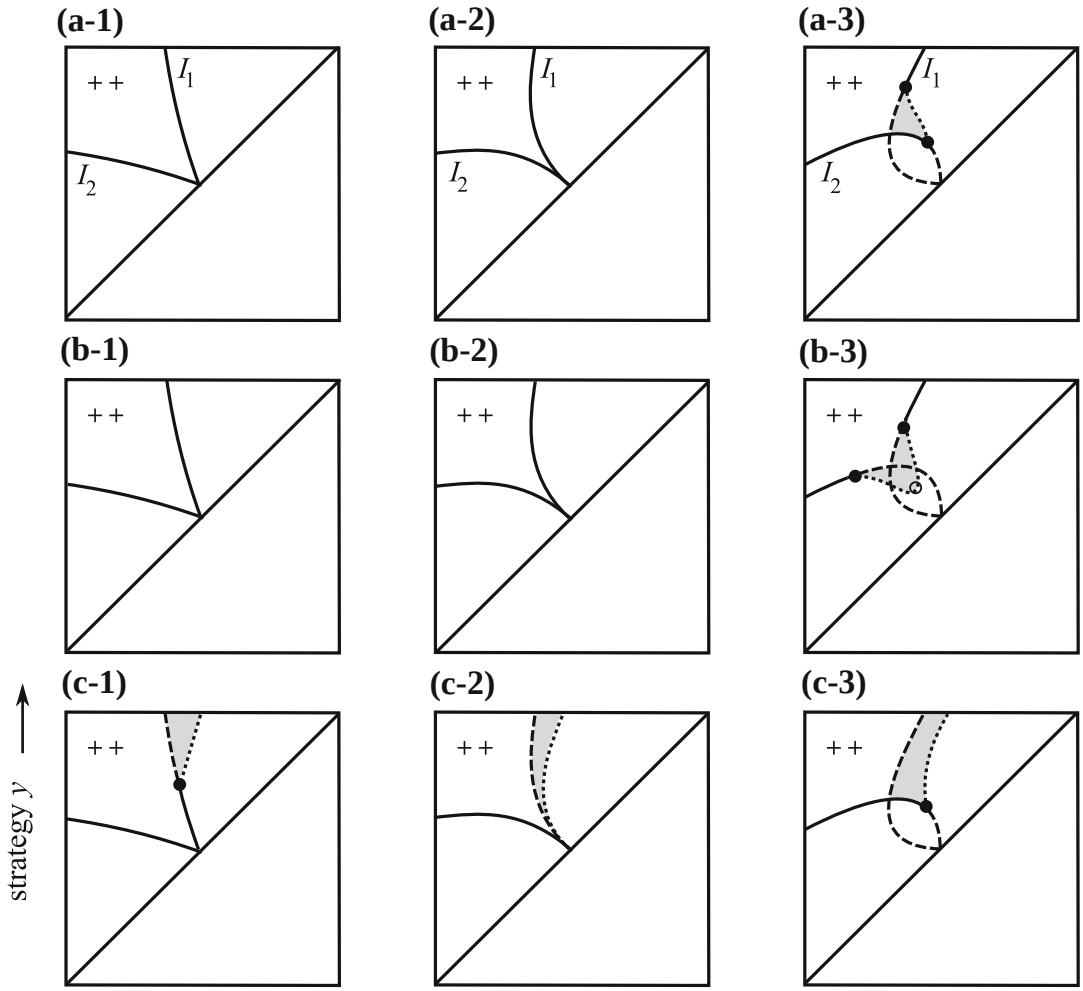

Fig. 5 All qualitatively different bifurcations between MI and ME near a singular strategy x^* with at most one stable interior equilibrium, such that invasion boundaries I_1 and I_2 connected to x^* intersect with the appearance of ME. The intersection point is assumed to be generic. In *each panel* the two curves connected to the main diagonal $y = x$ are invasion boundaries I_1 and I_2 (as marked in **a**). Because identical configurations of invasion boundaries are also found below the diagonal they are not drawn in the figure. In the *left panels* the cross-derivative $D_{12}s_{x^*}(x^*)$ is negative (MI is connected to x^*), in the *middle panels* $D_{12}s_{x^*}(x^*)$ is equal to zero and in the *right panels* it is positive (ME is connected to x^*). The *gray area* gives the region of unprotected coexistence. *Continuous curves* indicate a supercritical invasion boundary and *dashed curves* a subcritical invasion boundary. The *dotted curve* gives a saddle-node bifurcation in the interior of the population state space: this is the catastrophical boundary of an area of unprotected coexistence, by crossing it the population switches to a boundary equilibrium and the corresponding strategy goes extinct. The *filled circle* gives a bifurcation point between a super- and a subcritical bifurcation. The *empty circle* gives the point where two saddle-node bifurcations meet; in the simplest case a pitchfork bifurcation is formed. Note that for simplicity the figures are drawn somewhat symmetric about the diagonal $y = -x$, which they in general are not

at a generic point. The cases (a) to (e) in Fig. 5 are all the possible *qualitatively* different cases; the remaining cases can be constructed by replacing I_1 with I_2 and I_2 with I_1, and by taking the mirror image of the area of unprotected coexistence (gray area) around the diagonal $y = -x$. In Fig. 6 we give a similar classification, but the intersection of invasion boundaries I_1 and I_2 occurs with the appearance of MI. The results in Figs. 5 and 6 are consequences of Theorems 1 and 2 and of the Classification Theorem of Geritz (2005) (see Appendix).

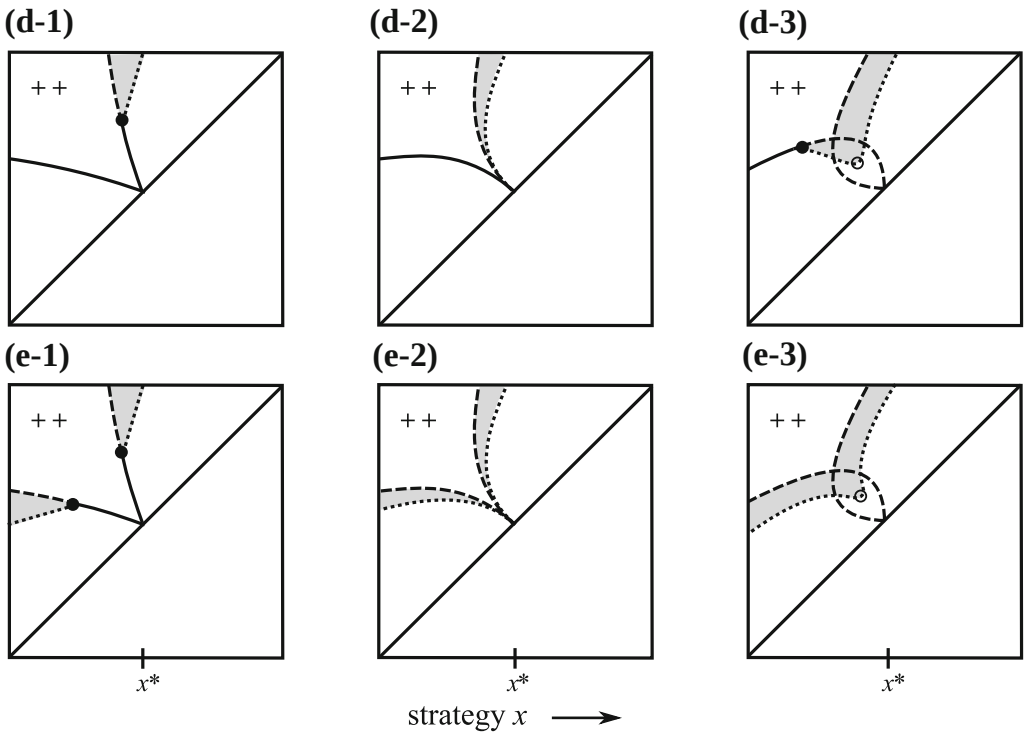

Fig. 5 continued

4 Examples

4.1 Example 1

Kisdi and Priklopil (2010) studied the evolution of a so called magic trait γ (Gavrilets 2004), determined by two additively acting alleles x and y. They presented, in particular, a general condition for the bifurcation between mutual invasibility and mutual exclusion, and used a Levene's soft-selection model (Levene 1953) to demonstrate the presence of unprotected coexistence of x and y.

Consider a sexually reproducing population of diploid individuals with discrete generations. At an encounter between a female and a male, the probability that a female with phenotype γ_g (the subscript indicates the genotype of the individual) accepts a male γ_h for mating is given by

$$p(\gamma_g, \gamma_h) = p_{max} u(\gamma_h - \gamma_g), \tag{4}$$

where $0 < p_{max} \leq 1$ is the maximum probability of mating and u is a sufficiently smooth function that attains its maximum at 0 with $u(0) = 1$. The invasion fitness of allele y in a population of x is

$$W_x(y) = \frac{1}{2}(Q_{xy,xx} + Q_{xx,xy}) w_{\hat{E}(\gamma_{xx})}(\gamma_{xy}), \tag{5}$$

where

$$Q_{xy,xx} = 1 - [1 - p_{max} u(\gamma_{xx} - \gamma_{xy})]^M \tag{6}$$

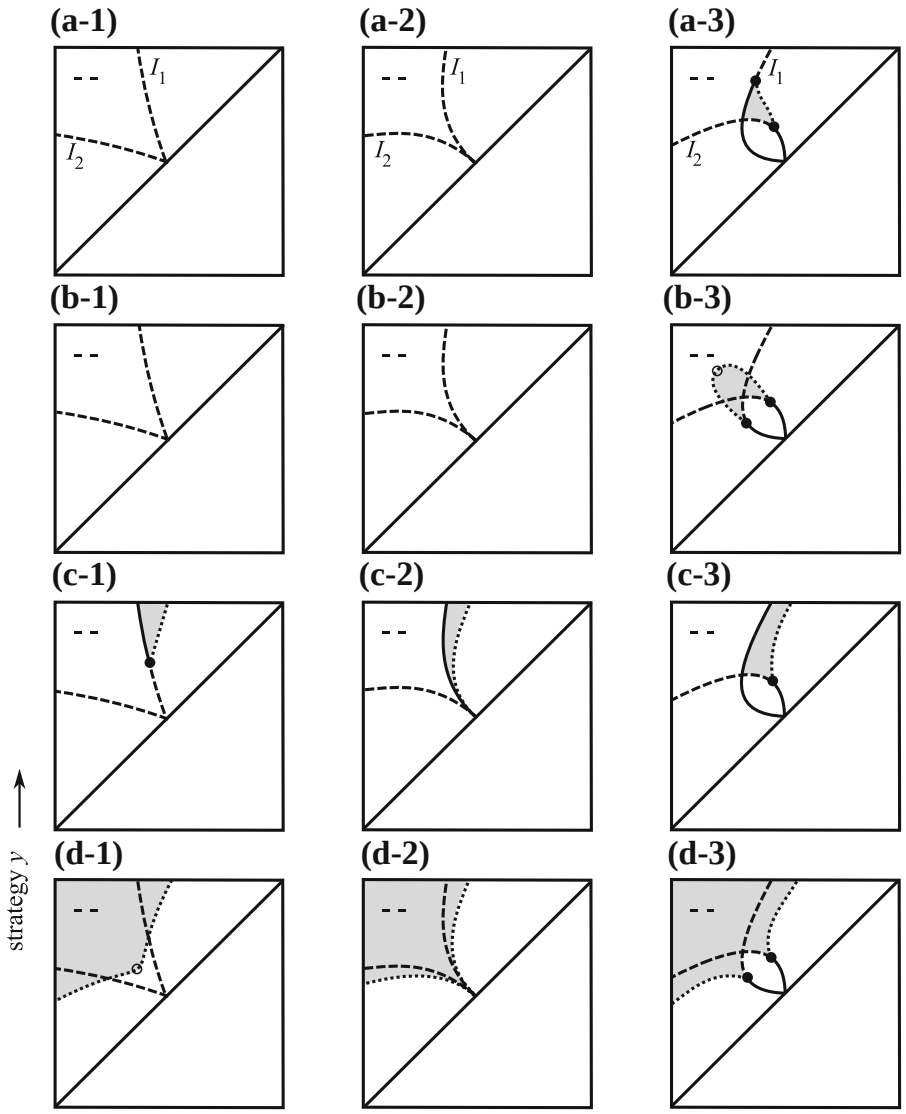

Fig. 6 All qualitatively different bifurcations between ME and MI near a singular strategy x^* with at most one stable interior equilibrium, such that invasion boundaries I_1 and I_2 connected to x^* intersect with the appearance of MI. Notations same as in Fig. 5. In the *left panels* the cross-derivative $D_{12}s_{x*}(x^*)$ is positive (ME is connected to x^*), in the *middle panels* $D_{12}s_{x*}(x^*)$ is equal to zero and in the *right panels* it is negative (MI is connected to x^*). Note that in cases (**d**) to (**f**) the bifurcations on the invasion boundaries are the same, these cases differ only in the location of the saddle-node bifurcation in the interior of the population state space. Note that for simplicity the figures are drawn somewhat symmetric about the diagonal $y = -x$, which they in general are not

describes the effect of sexual selection on females and

$$Q_{xx,xy} = \left[1 - (1 - p_{max})^M\right] u(\gamma_{xy} - \gamma_{xx}) \tag{7}$$

describes the effect of sexual selection on males, where M gives the maximum number of males one female can encounter (see Kisdi and Priklopil 2010 for derivation). Term $w_{\hat{E}_{\gamma_{xx}}}(\gamma_{xy}) = w(\gamma_{xy}, \gamma_{xx})$ is the ecological fitness of phenotype γ_{xy} in an environment set by a population with phenotype γ_{xx}. If a singular strategy x^* exists,

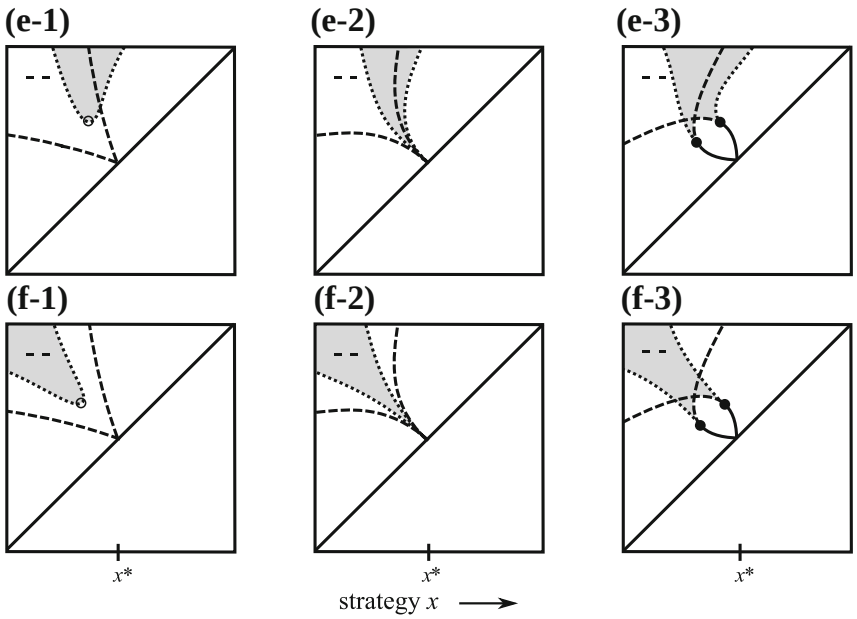

Fig. 6 continued

the cross-derivative of (5) evaluated at x^* is equal to 0 when

$$qD^2u(0) = -Q_{xx,xx}D_{22}w(\gamma_{x^*x^*}, \gamma_{x^*x^*}), \tag{8}$$

where

$$Q_{xx,xx} = 1 - (1 - p_{max})^M \tag{9}$$

and

$$q = \frac{1}{2}\left[1 + \frac{Mp_{max}(1 - p_{max})^{M-1}}{1 - (1 - p_{max})^M}\right]. \tag{10}$$

Because q, $Q_{xx,xx} > 0$, and $D^2u(0) < 0$ whenever individuals mate non-randomly, the term $D_{22}w(\gamma_{x^*x^*}, \gamma_{x^*x^*})$ in (8) must be positive for the bifurcation between mutual invasibility and mutual exclusion to occur. In fact, in diploid populations, $D_{22}w(\gamma_{x^*x^*}, \gamma_{x^*x^*}) > 0$ corresponds to the condition of mutual invasibility under random mating. Now, by an appropriate choice of u and w we get from (5) that $I_1 \neq I_2$ when (8) is satisfied, and hence Theorem 2 guarantees a transversal intersection of the invasion boundaries I_1 and I_2. Next we specify functions u and w for further investigations of the intersection point and unprotected coexistence.

Suppose the ecological selection is given as a Levene's soft-selection with two habitats of size c_1 and $c_2 = 1 - c_1$. In habitat 1, an individual with phenotype ϕ_g survives viability selection with probability

$$g_1(\phi_g) = \alpha_1 \exp\left(-\frac{(\phi_g - m_1)^2}{2\sigma_s^2}\right), \tag{11a}$$

and in habitat 2 with probability

$$g_2(\phi_g) = \alpha_2 \exp\left(-\frac{(\phi_g - m_2)^2}{2\sigma_s^2}\right), \tag{11b}$$

where α_1 and α_2 are the maximum survival probabilities, m_1 and m_2 are the optimal phenotypes in the two habitats, and $\sigma_s > 0$ gives the strength of selection within a habitat. Let d be the distance between habitat optima m_1 and m_2. For the mating process, assume that mating function u is a Gaussian with variance σ_m and that all females are eventually mated, that is, $M \to \infty$.

With these assumptions the singular strategy is $x^* = c_1 m_1 + c_2 m_2$, and condition (8) turns into

$$(\sigma_m/\sigma_s)^2 = \frac{1}{2(c_1 c_2 (d/\sigma_s)^2 + 1)} \tag{12}$$

(see Kisdi and Priklopil 2010). We observe that for any parameter values c_1, c_2, d and σ_s we can tune the width of the mating function σ_m such that the cross-derivative of invasion fitness function (5) changes sign, which implies the existence of unprotected coexistence since non-linearity of the system suggests that the intersection point is generic (see Sect. 5).

In Fig. 7 we give an example on how an area of unprotected coexistence appears and changes for the model with equal habitat size $c_1 = c_2 = 0.5$, and with $d = 3$ and $\sigma_s = 1$, when we decrease the parameter σ_m from infinity (random mating) to high levels of assortative mating. We calculate the location of unprotected coexistence numerically by finding stable equilibria outside the area of mutual invasibility and we complement these results by investigating what types of bifurcations happen on I_1 and I_2. This we do by applying analytical conditions for bifurcations with a low codimension (Wiggins 1990) to our model, and we find that both invasion boundaries are transcritical almost everywhere. More precisely, invasion boundaries are either super- or subcritically transcritical except at isolated points where they bifurcate between the two and undergo a pitchfork bifurcation.

When the individuals mate randomly ($\sigma_m = \infty$), both invasion boundaries are supercritical and no unprotected coexistence is present (Fig. 7a). When $\sigma_m \approx 0.81$, a pitchfork appears on I_1 at $x \approx -0.08$ and $y \approx 3.17$ (on I_2 there is a similar bifurcation due to symmetry around the secondary diagonal $y = -x$). Decreasing the value of σ_m a little, part of the invasion boundary bifurcates to become subcritically transcritical bounded by two pitchfork bifurcations. An area of unprotected coexistence now exists, and is adjacent to the subcritical part of the invasion boundary bounded by these two pitchfork bifurcation points and a curve of saddle-node bifurcations. By decreasing the parameter value to $\sigma_m = 0.50$ (Fig. 7b) we observe the pitchfork bifurcation point which is closer to the singular strategy x^*. This point approaches x^* as σ_m approaches the critical value $\sigma_m \approx 0.39$ where (12) is satisfied, that is, where MI bifurcates to ME near x^* (Fig. 7c). For smaller values of σ_m, the invasion boundaries I_1 and I_2 intersect and are separated by ME from the singular strategy x^*. In Fig. 7d we see an area of unprotected coexistence bounded by an area of MI and two saddle-node bifurcations which coincide at the empty circle forming a pitchfork bifurcation. The bifurcation scheme near x^* follows the case (e) presented in Fig. 5.

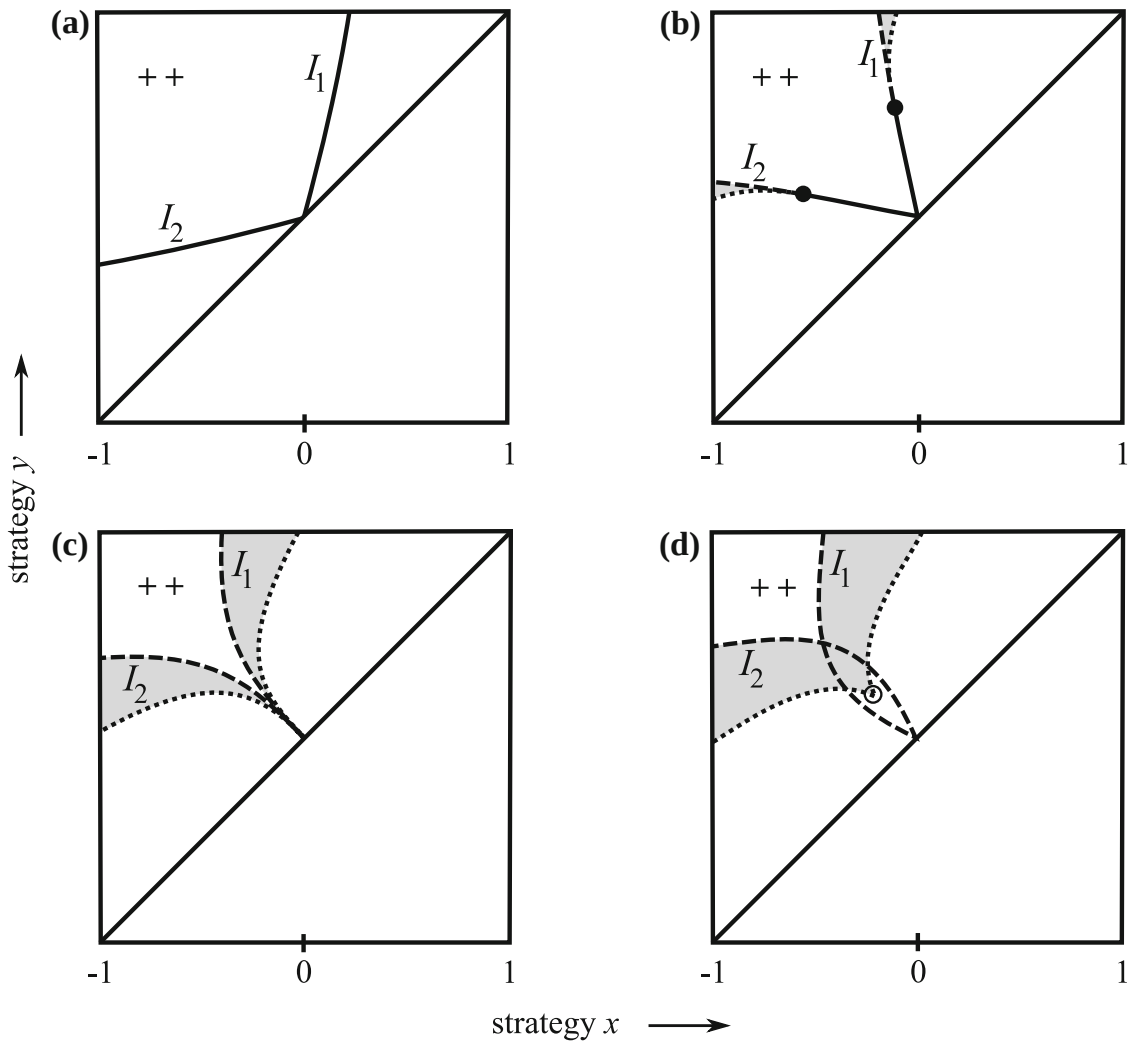

Fig. 7 Mutual invasibility plots and the area of unprotected coexistence for the Levene model with equal habitat size $c_1 = c_2 = 0.5$, and with $d = 3$ and $\sigma_s = 1$. Notations as in Fig. 5. When passing the critical value $\sigma_m \approx 0.81$ from above the area of unprotected coexistence appears. MI bifurcates into ME near $x^* = 0$ at $\sigma_m \approx 0.39$. In (**a**) $\sigma_m = \infty$ (random mating), no area of unprotected coexistence. In (**b**) $\sigma_m = 0.50$, an area of unprotected coexistence exists and is bounded by two pitchfork bifurcations on each invasion boundary I_1 and I_2. A pitchfork closer to $x^* = 0$ on invasion boundary I_1 happens at $x \approx -0.13$ and $y \approx 0.60$ and on I_2 at $x \approx -0.60$ and $y \approx 0.13$ (*filled dots*). The pitchforks that are further away from x^* are not in the range given in the panels. In (**c**) $\sigma_m = 0.39$, a value close to the bifurcation point between MI and ME. The area of unprotected coexistence extends to the singular strategy x^*. In (**d**) $\sigma_m = 0.36$, an intersection of I_1 and I_2 has appeared. The area of unprotected coexistence is bounded by subcritical invasion boundaries and two saddle-node bifurcations (*dotted lines*) which collide to form a pitchfork at the *empty circle*. Bifurcation scheme near x^* follows the case (**e**) in Fig. 5

4.2 Example 2

This example shows that an intersection point of invasion boundaries is not generic in all systems of type (1) or (2).

In a Lotka-Volterra competition model, the population dynamics of strategies $x_1, \ldots, x_k$ with corresponding sizes $N_1, \ldots, N_k$ is given by

$$\frac{d}{dt}N_i = N_i\left[r\rho(x_i) + a\sum_{j=1}^{k}\phi(x_i - x_j)N_j - c\sum_{j=1}^{k} f(x_i - x_j)N_j\right] \tag{13}$$

with $a, c, r > 0$ and given positive functions ρ, ϕ and f which attain their maximum value 1 at 0 and are symmetrical around the origin. Without loss of generality, we can assume $r = 1$ and $c = 1$ by scaling time and density.

Consider system (13) with only two strategies x and y and sizes N and M, respectively. The invasion fitness function for a strategy y in a population with strategy x becomes

$$s_x(y) = \rho(y) + a\phi(y - x)\hat{N}(x) - f(y - x)\hat{N}(x), \tag{14}$$

where $\hat{N}(x) = \frac{\rho(x)}{1-a}$, for $a < 1$, is an equilibrium value of strategy x. The singular strategy x^* can be solved from

$$D\rho(x^*) = 0$$

(Geritz et al. 1998). Since ρ gets its maximum at 0, the singular strategy is $x^* = 0$. Note that there might also be other singular strategies. For the cross-derivative $D_{12}s_{x^*}(x^*)$ we get

$$\frac{1}{1-a}\left(D^2 f(0) - aD^2\phi(0)\right), \tag{15}$$

which is equal to 0 when

$$a = \frac{D^2\phi(0)}{D^2 f(0)}. \tag{16}$$

When choosing appropriate functions ϕ and f, cross-derivative $D_{12}s_{x^*}(x^*)$ changes sign when tuning parameter a. Because $I_1 \neq I_2$ for all reasonable functions in (13), it follows from Theorem 2 that for some parameter values invasion boundaries of (13) intersect transversally near x^*.

The remaining task is to see whether the system (13) is generic at the intersection point. Because the model is symmetric around $y = -x$, the intersection point $(z, -z)$ is given implicitly by

$$\begin{aligned} s_z(-z) &= \rho(-z) + \frac{\rho(z)}{1-a}\left[a\phi(2z) - f(2z)\right] \\ &= \frac{\rho(z)}{1-a}\left[1 - f(2z) - a(1 - \phi(2z))\right] = 0. \end{aligned} \tag{17}$$

Thus, point z satisfies $1 - f(2z) = a(1 - \phi(2z))$. Substituting this into (13), we get that at z the solution is a neutral line of equilibria. Therefore, solutions of (13) are not continuous at the intersection point z and the Theorem 1 can not be applied.

We have shown that in the Lotka-Volterra competition model (13) unprotected coexistence does not follow from the transversal intersection of invasion boundaries.

In fact, every model of type (1) or (2) which is linear in F and G with respect to N and M has at the intersection point neutral line of equilibria and hence does not satisfy the conditions of Theorem 1.

4.3 Example 3

In this example we add to the Lotka-Volterra competition model (13) a non-linear Allee effect. With the assumptions made in the previous example, the population dynamics becomes

$$\frac{d}{dt}N_i = N_i\left[\rho(x_i) + a\frac{\sum_{j=1}^{k}\phi(x_i-x_j)N_j}{1+b\sum_{j=1}^{k}\phi(x_i-x_j)N_j} - \sum_{j=1}^{k} f(x_i-x_j)N_j\right], \tag{18}$$

where $b > 0$. Considering only two strategies x and y, the invasion fitness function of strategy y in a population of x is

$$s_x(y) = \rho(y) + a\frac{\phi(y-x)\hat{N}(x)}{1+b\phi(y-x)\hat{N}(x)} - f(y-x)\hat{N}(x), \tag{19}$$

where $\hat{N}(x)$ is the equilibrium of x. Similarly to the previous example, the singular strategy x^* can be solved from

$$D\rho(x^*) = 0 \tag{20}$$

(Geritz et al. 1998; Geritz 2005), and hence one particular solution is $x^* = 0$. For the cross-derivative $D_{12}s_{x^*}(x^*)$ we get

$$D^2 f(0) - \frac{aD^2\phi(0)}{(1+b\hat{N}(0))^2}, \tag{21}$$

where $b\hat{N}(0) = \frac{1}{2}\left(a+b-1+\sqrt{(a+b-1)^2+4b}\right)$, and it is equal to 0 when

$$\frac{a}{(1+b\hat{N}(0))^2} = \frac{D^2 f(0)}{D^2\phi(0)}. \tag{22}$$

When functions ϕ and f are chosen appropriately, then by Theorem 2, we obtain a transversal intersection of invasion boundaries when tuning the model parameters a and b.

Let's choose ϕ, f and ρ to be Gaussian functions with maximum value 1 at 0, and with variances σ_a, σ_c and σ, respectively. By scaling strategy values we can set σ to 1. The only singular strategy is $x^* = 0$, and the cross-derivative $D_{12}s_{x^*}(x^*)$ is equal to 0 when

$$\frac{a}{(1+b\hat{N}(0))^2} = \left(\frac{\sigma_a}{\sigma_c}\right)^2. \tag{23}$$

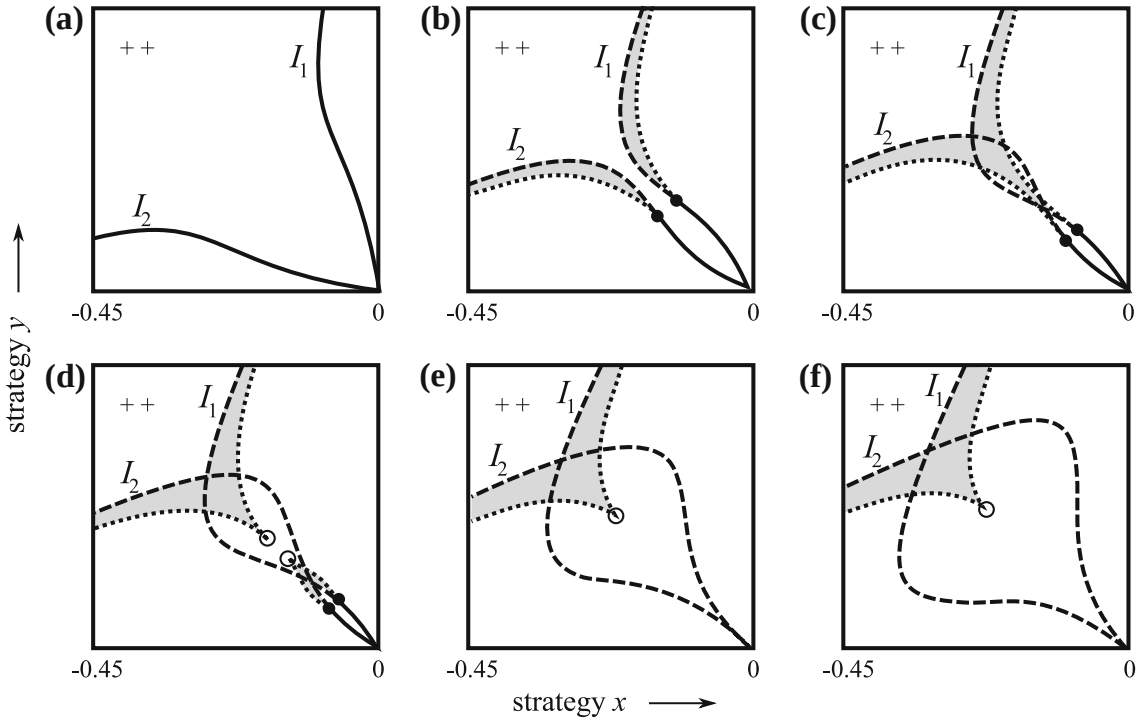

Fig. 8 Mutual invasibility plots and the area of unprotected coexistence for model (18) with $b = 1.25$, $\sigma_a = 0.25$ and $\sigma_c = 0.75$. For clarity, we show only the *upper left* quadrant of each MIP. Notations as in Fig. 5. An area of unprotected coexistence appears when a exceeds the critical value $a_{crit} \approx 0.52$. MI bifurcates into ME near $x^* = 0$ at $a \approx 0.88$. In (**a**) $a = 0.50$, no area of unprotected coexistence. In (**b**) $a = 0.7$, an area of unprotected coexistence has appeared and is connected to a pitchfork bifurcation (*filled dots*). In (**c**) $a = 0.76$, middle parts of I_1 and I_2 have intersected creating two intersection points separated by ME. An area of unprotected coexistence extends right through ME and is connected to pitchfork bifurcation points in the small area of MI near x^*. In ME there is also a region without unprotected coexistence (*small white region* in the area of ME). In (**d**) $a = 0.8$, the area of unprotected coexistence has split into two parts. In (**e**) $a = 0.88$, a value close to a bifurcation point between MI and ME near x^*. In (**f**) $a = 0.95$, an area of ME separates the intersection point from x^*. The bifurcation scheme near x^* follows case (**b**) in Fig. 6

Since $I_1 \neq I_2$ in (18), there exists a transversal intersection of invasion boundaries. After a somewhat lengthy calculation it can also be shown that at the intersection of invasion boundaries the analytical condition for a transcritical bifurcation (Wiggins 1990) is satisfied for almost all model parameter values. Instead of presenting the calculations here, in Fig. 8 we give a summary of numerical results on how an area of unprotected coexistence appears and changes when $b = 1.25, \sigma_a = 0.25$ and $\sigma_c = 0.75$, and when a ranges from 0.50 to 0.95.

5 Discussion

Theorem 1 gives a sufficient condition for the existence of unprotected coexistence of two strategies. Because the condition requires knowledge only about the local stability of the boundary equilibria of the population state space, the existence of unprotected coexistence can be simply observed by constructing a mutual invasibility plot: whenever an intersection of invasion boundaries is present, an unprotected coexistence follows. However, this is true only when the intersection point is generic as defined

in Preliminaries. In fact, a class of models that are linear in F and G with respect to N and M, such as Lotka-Volterra models, have at the intersection point *always* a neutral line of equilibria and thus are in this sense degenerate. By construction, linear models may hence loose a rich bifurcation structure and therefore not contain the biologically meaningful states that would otherwise be present (compare Examples 2 and 3).

Theorem 2 shows that if invasion boundaries intersect in some neighborhood of the singular strategy x^*, the intersection point approaches x^* as cross derivative $D_{12}s_{x^*}(x^*)$ approaches 0. It then follows from Theorem 1 that also an area of unprotected coexistence, and hence multiple equilibria in the interior of the population state space, approaches a singular strategy x^* as $D_{12}s_{x^*}(x^*)$ approaches 0. This complements the Classification Theorem of Geritz (2005) which proves that multiple equilibria do not exist near $y = x$ whenever $D_{12}s_{x^*}(x^*) \neq 0$.

A well known example where invasion boundaries intersect at a generic point is the Levene model (Levene 1953; see Fig. 2 in Hoekstra et al. (1985) for an example of the intersection), and hence from Theorem 1 it follows that the model admits an area of unprotected coexistence (see Novak 2011 for an alternative, but more general proof). Another example where invasion boundaries intersect is in Kisdi et al. (2001), but since the model is a Lotka-Volterra model (linear in F) there is no unprotected coexistence. In Boldin et al. (2009) an explicit condition $D_{12}s_{x^*}(x^*) = 0$ for their model is given, but the existence of unprotected coexistence was not investigated. We conjecture that in their model unprotected coexistence does exist for parameter values where the cross derivative changes sign (using Theorems 1 and 2).

Using concepts developed in this paper, we can also describe the ways how an unprotected coexistence can appear in general. As was earlier shown, an area of unprotected coexistence may emerge adjacent to an area of mutual invasibility as a consequence of either MI having multiple interior stable equilibria in the population state space, or, a supercritical invasion boundary bifurcates to become a subcritical boundary. The only alternative to this is when an area of unprotected coexistence emerges away from invasion boundaries, but since in this scenario no new equilibria move into the interior of the population state space, interior stable equilibria must be created anew through a saddle-node bifurcation (away from the boundary of the population state space).

We would also like to briefly explore the possibility of relaxing some conditions made in this paper. The central idea of Theorem 1 is that if invasion boundaries intersect, at least one stable equilibrium must stay in the interior of the population state space when leaving MI (through a point away form the intersection). This is because not all equilibria can be simultaneously in the neighborhoods of both boundary equilibria. Since this idea can be directly applied to higher dimensional population state spaces, our restriction to two dimensions is not necessary. However, the concepts of protected and unprotected coexistence can't be readily extended to higher dimensions and hence we left the more general setting for future work.

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Appendix

Proof of Theorem 1

Since invasion boundaries I_1 and I_2 are assumed to intersect transversally, the intersection point z divides the neighborhood into four regions A, B, C and MI as depicted in Fig. 9. We denote with I_1^+ and I_2^+ the part of the invasion boundary which is a boundary of the region MI excluding the point of intersection z.

We claim, that in the neighborhood of z there must exist an interior stable equilibrium either in A or B, or in both A and B. If this is not the case, then crossing from MI to A or from MI to B *all* interior stable equilibria in MI must leave the interior of the population state space. Bifurcations on both invasion boundaries I_1^+ and I_2^+ are hence supercritical involving all interior stable equilibria. However, in some small neighborhood of z the neighborhood of $(\hat{N}, 0)$ and the neighborhood of $(0, \hat{M})$ can't *both* contain all equilibria. Therefore, in the neighborhood of z at least one of the invasion boundaries I_1^+ or I_2^+ must be subcritical, *or*, not all interior stable equilibria in MI leave the interior when crossing I_1^+ or I_2^+. In either case, some interior stable equilibria stay in the interior of the population state space when crossing I_1^+ or I_2^+, and hence unprotected coexistence is established either in A or B.

If we assume that there exists only one stable equilibrium in MI, at z this equilibrium must pass *either* $(\hat{N}, 0)$ or $(0, \hat{M})$, but (obviously) not both. Therefore, at z at least one of the invasion boundaries I_1 or I_2 must be subcritical.

Proof of Theorem 2

Lets write $D = D_{12}s_{x^*}(x^*)$, where x^* is a singular strategy. Strategies x and y close to x^* can invade each other for $D < 0$, and for $D > 0$ they cannot (Geritz et al. 1998). Note that since D changes sign, $s_x(y) = 0$ and $s_y(x) = 0$ define I_1 and I_2, respec-

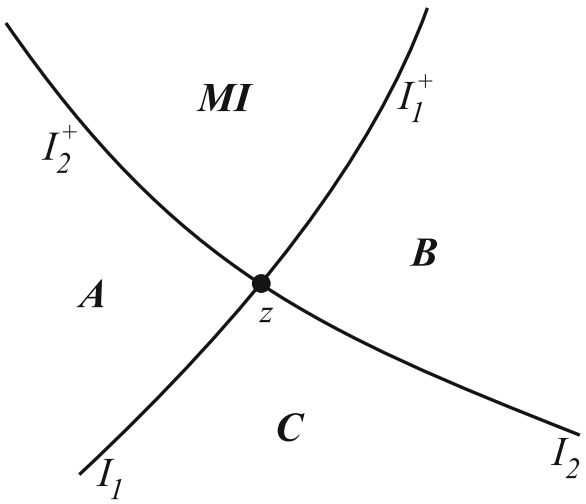

Fig. 9 Transversal intersection point z of invasion boundaries I_1 and I_2 divides a MIP into four regions A, B, C and MI. In MI, $s_x(y) > 0$ and $s_y(x) > 0$; in A, $s_x(y) > 0$ and $s_y(x) < 0$; in B, $s_x(y) < 0$ and $s_y(x) > 0$; in C, $s_x(y) < 0$ and $s_y(x) < 0$. I_1^+ and I_2^+ are the boundaries of MI excluding the point z

tively. Because $x = y$ is always a solution to $s_x(y) = 0$ and $s_y(x) = 0$, there exist functions $i_x(y)$ and $i_y(x)$ such that $s_x(y) = (x - y)i_x(y)$ and $s_y(x) = (x - y)i_y(x)$. Also, there exists a $\delta > 0$, such that for $|D| < \delta$

$$\frac{\partial i_x(y)}{\partial y}\Big|_{x=y=x^*} \neq 0 \neq \frac{\partial i_y(x)}{\partial y}\Big|_{x=y=x^*}. \tag{24}$$

By the implicit function theorem, there are unique functions

$$y_1 = y_1(x), \quad y_1(x^*) = x^*, \tag{25}$$
$$y_2 = y_2(x), \quad y_2(x^*) = x^* \tag{26}$$

defined for $|D| < \delta$ and x sufficiently close to x^*, such that $i_x(y_1(x)) = 0$ and $i_{y_2(x)}(x) = 0$. Now, we construct a distance function $H(x) = y_1(x) - y_2(x)$ in some neighborhood V of x^* and for $|D| < \delta$. Because the first order derivatives of y_1 and y_2 coincide at $D = 0$, the distance function H has the following properties

$$H(x^*) = 0, \tag{27}$$
$$H'(x^*) = 0, \quad \text{for } D = 0, \tag{28}$$
$$H'(x^*) < 0, \quad \text{for } D < 0, \tag{29}$$
$$H'(x^*) > 0, \quad \text{for } D > 0. \tag{30}$$

Because H is a continuous function and it changes its first order derivative at $D = 0$, we can find for an arbitrarily small D a neighborhood $W \subset V$ where $H(x) = 0$ for some value $x \in W \backslash x^*$.

We have proved, that when bifurcating between mutual invasibility ($D < 0$) and mutual exclusion ($D > 0$), the invasion boundaries y_1 and y_2 intersect near x^*. In addition, if $y_1 \neq y_2$ at $D = 0$, the invasion boundaries do not lie on top of each other and hence the intersection is transversal.

Derivation of Figs. 5 and 6

Classification Theorem of Geritz (2005) gives all the possible dynamical behavior for y close to x, and in particular, for strategy values close to a singular strategy x^* whenever $D_{12}s_{x^*}(x^*) \neq 0$. It excludes for $D_{12}s_{x^*}(x^*) \neq 0$ the existence of unprotected coexistence, and hence it defines both invasion boundaries near x^* to be either supercritical, when $D_{12}s^*_x(x^*) < 0$, and subcritical, when $D_{12}s^*_x(x^*) > 0$. Suppose, that invasion boundary near x^* is supercritical, but further away from x^* it is subcritical. There must exist (at least one) point where the type of the bifurcation changes. Because the solutions of system (1) or (2) are continuous and unique, the bifurcation between super- and subcritical bifurcations involves an appearance of a saddle-node bifurcation as depicted in Fig. 10. Supposing that there are unique invasion boundaries I_1 and I_2 connected to x^*, then going through all the possible combinations of super- and subcritical bifurcations (and applying Theorems 1 and 2), we can derive Figs. 5 and 6.

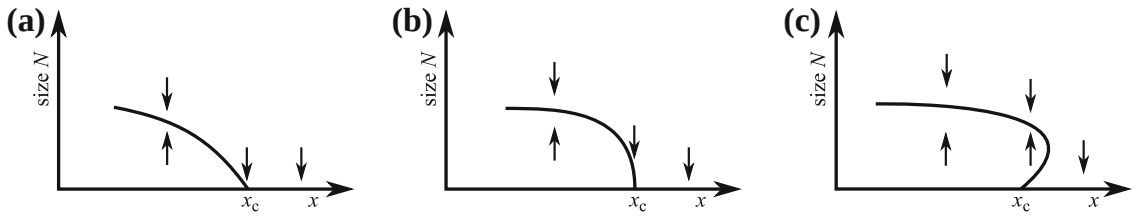

Fig. 10 An example of a transition at x_C from a supercritical (**a**) to a subcritical bifurcation (**c**) involving an appearance of a saddle-node bifurcation. At the transition point (**b**) the bifurcation at x_C is supercritical

References

Boldin B, Geritz SAS, Kisdi E (2009) Superinfections and adaptive dynamics of pathogen virulence revisited: a critical function analysis. Evol Ecol Res 11:153–175

Christiansen FB, Loeschke V (1980) Evolution and intraspecific exploitation competition I. One-locus theory for small additve gene effects. Theor Popul Biol 18:297–313

Claessen D, Dieckmann U (2002) Ontogenetic niche shift and evolutionary branching in structured populations. Evol Ecol Res 4:189–217

Dieckmann U (1997) Can adaptive dynamics invade. Trends Ecol Evol 12:128–131

Doebeli M, Block HJ, Leimar O, Dieckmann U (2007) Multimodal pattern formation in phenotype distributions of sexual populations. Proc R Soc 274:347–357

Ferriere R, Gatto M (1993) Chaotic population dynamics can result from natural selection. Proc Biol Sci 251:33–38

Gavrilets S (2004) Fitness landscapes and the origin of species. Princeton University Press, Princeton

Geritz SAH (2005) Resident-invader dynamics and the coexistence of similar strategies. J Math Biol 50: 67–82

Geritz SAH, Kisdi É, Meszéna G, Metz JAJ (1998) Evolutionarily singular strategies and the adaptive growth and branching of the evolutioary tree. Evol Ecol 12:35–57

Geritz SAH, Gyllenberg M, Jacobs FJA, Parvinen K (2002) Invasion dynamics and attractor inheritance. J Math Biol 44:548–560

Gyllenberg M, Metz JAJ (2001) On fitness in structured metapopulations. J Math Biol 43:545–560

Hoekstra RF, Bijlsma R, Dolman AJ (1985) Polymorphism from environmental heterogeneity: models are only robust if the heterozygote is close in fitness to the favoured homozygote in each environment. Genet Res Camb 45:299–314

Kisdi É, Meszéna G (1993) Density dependent life history evolution in fluctuating environments. In: Clark CW, Yoshimura J (eds) Adaptation in a Stochastic Environment. Lecture Notes in Biomathematics 98:26–62

Kisdi É, Meszéna G (1995) Life history with lottery competition in a stochastic environment: ESSs which do not prevail. Theor Pop Biol 47:191–211

Kisdi É, Jacobs FJA, Geritz SAH (2001) Red queen evolution by cycles of evolutionary branching and extinction. Selection 1–2:161–176

Kisdi É, Priklopil T (2010) Evolutionary branching of a magic trait. J Math Biol. doi:10.1007/s00285-010-0377-1

Kuznetsov YA (1998) Elements of applied bifurcation theory. Springer, New York

Levene H (1953) Genetic equilibrium when more than one ecological niche is available. Am Nat 87:331–333

Matsuda H (1985) Evolutionarily stable strategies for predator switching. J Theor Biol 115:351–366

Metz JAJ, Nisbet RM, Geritz SAH (1992) How should we define "fitness" for general ecological scenarios? Trends Ecol Evol 7:198–202

Metz JAJ, Geritz SAH, Meszéna G, Jacobs FJA, Van Heerwaarden JS (1996) Adaptive dynamics: a geometrical study of the consequences of nearly faithful reproduction. In: van Strien SJ, Verduyn Lunel SM (eds) Stochastic and spatial structures of dynamical systems. Elsevier, North-Holland, pp 183–231

Motro U (1982) Optimal rates of dispersal I. Haploid populations. Theor Popul Biol 21:394–411

Novak S (2011) The number of equilibria in the diallelic Levene model with multiple demes. Theor Popul Biol 79:97–101

Poulsen ET (1979) A model for population regulation with density- and frequency-dependent selection. J Math Biol 8:325–343
Prout T (1968) Sufficient conditions for multiple niche polymorphism. Am Nat 102:493–496
Rueffler C, Van Dooren TJM, Metz JAJ (2004) Adaptive walks on changing landscapes: Levins? approach extended. Theor Popul Biol 65:165–178
van Tienderen PH, de Jong G (1986) Sex ration under the haystack model: polymorphism may occur. J Theor Biol 122:69–81
Wiggins S (1990) Introduction to applied nonlinear dynamical systems and Chaos. Springer, New York

Article III

Chaotic dynamics of allele frequencies in condition-dependent mating systems

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Chaotic dynamics of allele frequencies in condition-dependent mating systems

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ABSTRACT

I study the dynamics of allele frequencies in sexually reproducing populations where the choosy sex has a preference for condition-dependent displays of the opposite sex. The condition of an individual is assumed to be shaped by frequency-dependent selection. For sufficiently strong preferences the dynamics becomes increasingly complex, and periodic orbits and chaos are observed. Moreover, multiple attractors can exist simultaneously. The results hold also when the choosy sex is allowed to maintain a moderate level of assortative mating. Complex dynamics, a well studied phenomenon in a purely ecological setting, has been rarely observed in ecologically motivated population genetic models.

1. Introduction

Female mating preferences generally act on male ornaments that indicate genetic or phenotypic quality and thereby provide the female with additional resources or genetic benefits for their offspring (Andersson, 1994; Møller and Alatalo, 1999; Jennions and Petrie, 2000; Møller and Jennions, 2001; Kokko et al., 2003; Cotton et al., 2004; Andersson and Simmons, 2006). In particular, mate choice based on male displays that indicate condition can bring a considerable advantage to sexual reproduction (Agrawal, 2001; Siller, 2001), and can enhance adaptation (Proulx, 1999, 2001, 2002; Whitlock, 2000; Lorch et al., 2003) and speciation (van Doorn and Weissing, 2009). However, as far as the author is aware, none of the studies up to this date have investigated the role of condition-dependent mating when the condition of an individual is affected by the density and phenotypic structure of the whole population. A frequency- (or density-) dependent condition is observed in cases where individuals face predation, parasitism or intraspecific competition (Clarke and Partridge, 1988; Doebeli, 2011).

In this paper I will make the assumption that female (the choosy sex) preference acts on male ornaments that correlate with frequency-dependent male condition, which is assumed to correlate with male viability, and give my main attention to populations that are under ecologically divergent selection. Because during divergent selection the intermediate phenotypes are at a disadvantage, females prefer males expressing extreme and viable phenotypes (e.g. females prefer small and big traits when intermediate sized traits are at a disadvantage). This setting may lead to disassortative mating when one of the extreme phenotypes is at a greater advantage. Since disassortative mating results in the production of unfit phenotypes, females are assumed to develop an additional preference for phenotypes that are similar to their own (Schluter and McPhail, 1992; Hegde and Krishna, 1997; Snowberg and Bolnick, 2008). Females are hence using multiple cues for mating (Møller and Pomiankowski, 1993; Pomiankowski and Iwasa, 1993; Iwasa and Pomiankowski, 1994; Brooks, 1999; Scheuber et al., 2004; van Doorn and Weissing, 2004).

The main focus of this paper is the dynamics of allele (and genotype) frequencies caused by condition-dependent mating. I show that with sufficiently strong preferences for traits that indicate condition the allele dynamics becomes gradually more complex, and may result in periodic orbits or even chaos. Moreover, multiple attractors can coexist simultaneously. When females are allowed to maintain some level of assortative mating the dynamical behavior remains qualitatively the same. While chaotic dynamics in an ecological setting is a well documented phenomenon (Cushing et al., 2003; Turchin, 2003), previous studies that find chaotic dynamics of allele frequencies in a single species are surprisingly rare and rely largely on so-called pairwise interaction models with arbitrary ecological assumptions (Altenberg, 1991; Gavrilets and Hastings, 1995; but see Yi et al., 1999). Furthermore, Schneider (2008) showed that in the models of Altenberg (1991) and Gavrilets and Hastings (1995) complex behavior can result only when the interaction coefficients have no apparent biological interpretation (but see a multiallelic case of Trotter and Spencer, 2009). At the end of the paper I discuss the general features that lead to the observed complex behavior.

2. The model

I consider a sexually reproducing population of diploid individuals which is well mixed and sufficiently large to ignore random genetic drift. The life-cycle of an individual has two phases. In the first phase, individuals undergo frequency-dependent

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ecological selection that acts on a quantitative character. The surviving individuals then enter the second phase of the life-cycle, a mating season. I assume that females are the choosy sex and that they use multiple cues to select males. Firstly, females prefer traits that signal adaptation to the environment, and secondly, they prefer males that are similar to themselves with respect to ecological character. I will refer to these as condition-dependent and assortative mating assumptions, respectively. After producing offspring the adults die and a new generation begins.

2.1. Ecological and genetic assumptions

I consider one locus with two alleles x and y, so that the three genotypes in the population are xx, xy and yy. The value of the trait that is under ecological selection is denoted with ϕ_g, where g is the genotype of the individual. Alleles contained in g are assumed to act additively on the phenotype, such that $\phi_{xy} = \frac{x+y}{2}$. Denoting with P_g the frequency before selection and with v_g the viability (probability to survive ecological selection) of g, the frequency of genotype g after ecological selection is

$$\tilde{P}_g = \frac{v_g}{\bar{v}} P_g, \tag{1}$$

where $\bar{v} = \sum_h P_h v_h$ is the average survival probability in the population. The numerical results of this paper will be obtained by assuming v to be as in the model of Bulmer (introduced in Bulmer, 1974, 1980) and used e.g. by Bürger (2002), Kopp and Hermisson (2006), Bürger and Schneider (2006) and Peischl and Schneider (2010). However, the main characteristics of the results are independent of the exact choice of the model and require only some form of differential specialization of individuals which results in frequency-dependent disruptive selection (see Section 3).

As in Bulmer (1974, 1980) the viability of an individual g is given as

$$v_g = \psi_g S_g, \tag{2}$$

where ψ_g measures how well individuals survive frequency-dependent competition and S_g represents the effect of frequency-independent selection for an optimal trait value θ. The functions ψ_g and S_g are survival probabilities. S_g is assumed to be a Gaussian function

$$S_g = S_0 \exp[-s(\phi_g - \theta)^2], \tag{3}$$

where S_0 is the maximum probability of survival and s determines the intensity of selection. For example, function (3) can be seen to describe how resources are distributed in the environment (the width of the distribution is regulated with s), such that for individuals with trait value θ the resources are the most abundant and hence their surviving probability attains its maximum S_0 whereas for individuals with trait values away from θ the surviving probability decreases with the amount of available resources.

The function ψ_g is given as

$$\psi_g = \left(\rho - \frac{C_g}{\kappa}\right), \tag{4}$$

where ρ is the maximum probability of survival, C_g gives the effective number of competitors and κ controls how strong the effect of competition is. For ψ to be a probability, $\max\{C_g/\rho\} = N/\rho \leq \kappa$ (see (5) for the definition of C_g). C_g is obtained by summing the strength of competition u over all individuals in the population

$$C_g = N \sum_h u_{g,h} P_h, \tag{5}$$

where N is the total population size, and u is a Gaussian

$$u_{g,h} = \exp[-c(\phi_g - \phi_h)^2], \tag{6}$$

where c can be interpreted as a measure of the degree of resource specialization. Large c means that the competition for resources between individuals is weak (small u), implying high specialization and a strong frequency-dependent effect of competition, whereas the frequency dependence vanishes as c decreases to zero. The parameter c is hence a direct measure of the strength of the frequency-dependent effect of competition. Note that due to the stabilizing component (3), ecological selection acts asymmetrically on the phenotypes ϕ_{xx} and ϕ_{yy} when their trait values are placed at unequal distances from θ.

2.2. Mating assumptions

After the phase of ecological selection, surviving individuals enter a mating season. Let us first consider the assumption of condition-dependent mating. Since well adapted males are likely to be in a better condition, it pays for the female to have heightened preference for traits that correlate with condition (Grafen, 1990; Iwasa et al., 1991; Iwasa and Pomiankowski, 1994). Supposing that the quality of the male sexual trait is proportional to his condition, and that condition is proportional to the survival probability v, the probability to mate with a male h is an increasing function of v_h. In addition, females are also assumed to mate assortatively with respect to the ecological trait (Schluter and McPhail, 1992; Hegde and Krishna, 1997; Snowberg and Bolnick, 2008). Note that the ecological trait and the condition-dependent trait might be one and the same and hence would be controlled by the same set of genes, or alternatively, the condition-dependent trait might be a separate trait which acts purely as an indicator of the condition without affecting the viability of an individual.

When using both mating cues, a female ϕ_g accepts an encountered male ϕ_h for mating with probability

$$\mu_{g,h} = \gamma_h \pi_{g,h}, \tag{7}$$

where γ gives the probability to accept a male based on a condition-dependent trait and π based on their ecological traits. The function that describes condition-dependent mating is taken to be

$$\gamma_h = \gamma_0 \exp[\alpha v_h], \tag{8}$$

where α expresses the level of preference and γ_0 is a constant selected such that γ_h is always less than 1 to be a probability. Note that the proportionality constants are absorbed into α. The assortative mating function is of Gaussian form, i.e.

$$\pi_{g,h} = \pi_0 \exp[-\beta(\phi_g - \phi_h)^2], \tag{9}$$

where π_0 gives the maximum probability and β gives the strength of preference.

The rest of the assumptions on mating follows Gavrilets and Boake (1998). A female encounters males randomly such that the maximum number of males she can meet is M. At each encounter she can either accept him for mating or decline, in which case she moves on to the next male. The probability that she mates with someone at a random encounter is

$$\bar{\mu}_g = \sum_h \mu_{g,h} \tilde{P}_h, \tag{10}$$

and the probability that she will eventually mate with a male ϕ_h is

$$\sum_{i=0}^{M-1} [1 - \bar{\mu}_g]^i \mu_{g,h} \tilde{P}_h. \tag{11}$$

Because I will assume throughout the paper that M is large ($M \to \infty$), from (11) I get that the probability that female g mates with h during a mating season is $Q_{g,h}\tilde{P}_h$, where

$$Q_{g,h} = \frac{\mu_{g,h}}{\bar{\mu}_g}. \tag{12}$$

Note that each female mates only once, but males can participate in multiple matings. Furthermore, all mated females are assumed to produce an equal number of offspring. Monandrous mating systems might be favored when repeated mating poses a cost to females. Costs may include the time and energy waste and increased risk of predation and disease transmission, or even the effect of toxic male ejaculates (Chapman et al., 1995).

2.3. Dynamics and the assessment of parameters

The dynamics of the three genotypes can be described with the recursion equation

$$P_r' = \frac{1}{\bar{Q}} \sum_{g,h} \tilde{P}_g \tilde{P}_h Q_{g,h} R_{g,h\to r}, \tag{13}$$

where $\bar{Q} = \sum_{g,h} \tilde{P}_g \tilde{P}_h Q_{g,h}$ is the mean mating success and $R_{g,h\to r}$ denotes the probability that parents with genotypes g and h produce an offspring with genotype r according to the Mendelian rules. Since all females get mated, $\bar{Q} = 1$. The population size changes according to

$$N' = Fh(N)\bar{v}N, \tag{14}$$

where N and N' are the population sizes in consecutive generations, F is the average fecundity and $h(N)$ contains possible additional factors that regulate the population size (e.g. size of habitat).

In the following section (Section 3) the analytical results are derived for a fairly general class of functions γ, π and v. Numerical results, however, require specific functions and parameter values. I will use the functions defined in (1)–(9), and if not mentioned otherwise, the parameter values will be set as follows. The optimal trait value in (3) is set to $\theta = 0$, and the maximum survival of the frequency-dependent competition in (4) is $\rho = 1$. The probabilities S_0, γ_0 and π_0 in (3), (8) and (9), respectively, can take arbitrary values between 0 and 1. Furthermore, I keep the population size N constant (by assuming, for example, limited size of habitat and high fecundity F), and set the minimum surviving probability of the frequency-dependent competition to $\frac{1}{2}$ (i.e. $\kappa = 2N$ in (4)). Finally, without loss of generality, in (3) I can set $s = \frac{1}{2}$ by scaling trait value ϕ_g. Hence, given allele values x and y, the dynamics of allele frequencies depend on the three remaining parameters: the (scaled) measure of the degree of resource specialization c/s, the (scaled) strength of assortative mating β/s and the strength of condition-dependent mating α. Note that when talking about an increased (decreased) effect of frequency-dependent competition when c is increased (decreased), it is always measured relative to frequency-independent selection. The same holds when changing the value of parameter β. See Table 1 for the glossary of symbols.

3. Results

In the main part of this section, I concentrate on the effect induced by ecological selection and condition-dependent mating alone, that is, without assortative mating ($\beta = 0$). The interplay between assortative and condition-dependent mating will be investigated at the end of the section.

3.1. The effect of condition-dependent mating when $\beta = 0$

Invasion of alleles. Consider γ to be any increasing function of viability v, which at this point doesn't need to be of any specific form (but taking values between 0 and 1 to be a probability). In models of type (13) the invasion of a rare allele y in a resident population of allele x is determined by an invasion fitness function

$$W_x(y) = \frac{1}{2}\left(\frac{Q_{xx,xy} + Q_{xy,xx}}{Q_{xx,xx}}\right)\frac{v_{xy}}{v_{xx}} \tag{15}$$

Table 1
Glossary of symbols. Listing is in the order of appearance in the text; however, the most frequently used symbols are presented first. The references are to the equation closest to the definition of each symbol, such that (1) and (1)- refers to (1) and the text above (1), respectively.

Symbol	Reference	Definition
x, y	(1)-	Allele values
P_g	(1)-	Frequency of genotype g in the beginning of the life-cycle
$\tilde{P}_g$	(1)	Frequency of genotype g after ecological selection
c	(6)	Measure of the degree of resource specialization
α	(8)	Strength of female preference for a condition-dependent trait
β	(9)	Strength of female preference for ecological characters similar to her own
ϕ_g	(1)-	Phenotype of genotype g
v_g	(1)	Viability of genotype g
$\bar{v}$	(1)	Average viability of the population
S_g	(3)	Probability for genotype g to survive selection for an optimal value θ
S_0	(3)	Maximum probability to survive selection for an optimal value θ
θ	(3)	Optimal trait value (trait value for which the resources are the most abundant)
s	(3)	Intensity of selection for an optimal value θ
ψ_g	(4)	Probability for genotype g to survive competition
ρ	(4)	Maximum probability to survive competition
κ	(4)	Controls the strength of competition
C_g	(5)	Effective number of competitors for genotype g
N	(5)	Total population size
$u_{g,h}$	(6)	The strength of competition between genotypes g and h
$\mu_{g,h}$	(7)	Probability that at an encounter between a female g and a male h the female accepts the male for mating
γ_h	(8)	Probability that at an encounter with a male g the female accepts the male based on his condition-dependent trait
γ_0	(8)	Constant regulating the mating probability γ_h
$\pi_{g,h}$	(9)	Probability that at an encounter between a female g and a male h the female accepts the male based on their ecological characters
π_0	(9)	Maximum mating probability in $\pi_{g,h}$
$\bar{\mu}_g$	(10)	Probability that a female g accepts a male at a random encounter
$Q_{g,h}$	(12)	Affinity of a female g towards a male h
$\bar{Q}$	(13)	Mean mating success
$R_{g,h\to r}$	(13)	Probability that genotype r is produced by genotypes g and h
F	(14)	Average fecundity
$h(N)$	(14)	Auxiliary factors that regulate population size

(Kisdi and Priklopil, 2011). Because a rare allele y is only present in heterozygotes xy, it may invade if the fitness of heterozygotes $v_{xy}(\frac{1}{2}Q_{xx,xy} + \frac{1}{2}Q_{xy,xx})F$ is greater than the fitness of homozygotes $v_{xx}Q_{xx,xx}F$, that is, when $W_x(y) > 1$. If mating is purely condition-dependent, $\mu_{g,h} = \gamma_h$ in (7), I get

$$W_x(y) = \frac{1}{2}\left(1 + \frac{\gamma_{xy}}{\gamma_{xx}}\right)\frac{v_{xy}}{v_{xx}}. \tag{16}$$

Notice that when $\frac{v_{xy}}{v_{xx}}$ is greater than 1, then for increasing γ also $\frac{\gamma_{xy}}{\gamma_{xx}}$ is greater than 1 and consequently $W_x(y) > 1$. When $\frac{v_{xy}}{v_{xx}} < 1$ then also $W_x(y) < 1$. The invasion criterion of an allele y is hence independent of condition-dependent mating and depends only on the ratio $\frac{v_{xy}}{v_{xx}}$. When heterozygotes xy have a greater viability than homozygotes xx, the allele y can invade. Moreover, the steeper the function γ, the greater is the reproductive advantage (greater W) of individuals with higher viability. Condition-dependent mating hence reinforces the advantage of fit individuals, and therefore for steep γ big changes in allele frequencies are expected.

Polymorphic attractors. To obtain numerical results, let functions v and γ to be as in (2) and (8), respectively. The region of protected polymorphism (Prout, 1968; Priklopil, 2012), i.e. polymorphism with mutual invasion of alleles x and y, is given in Fig. 1 as the shaded area for allele values $x \in [-1, 1]$, $y \in [-1, 1]$ and for

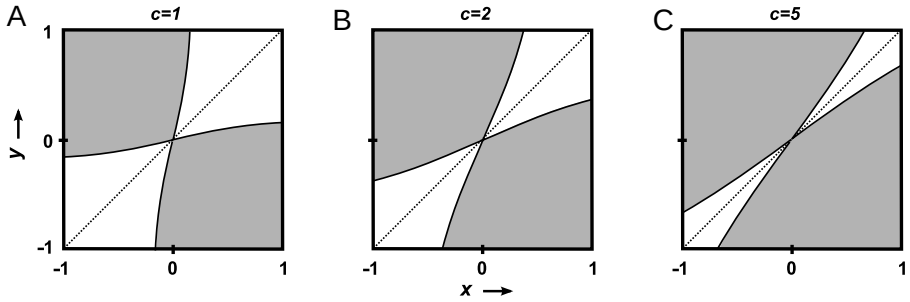

Fig. 1. The area of protected polymorphism of alleles x and y is indicated with gray. Note that the area gets broader with increasing value of the competition coefficient c.

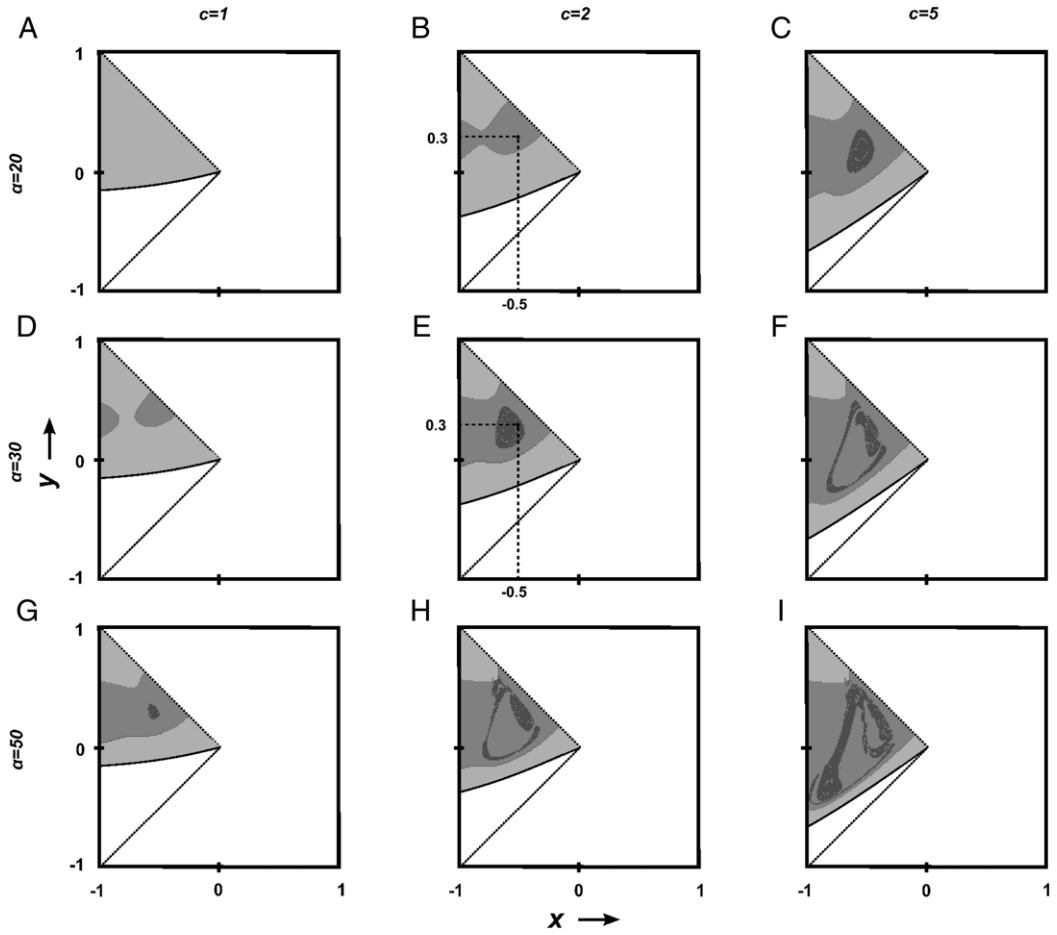

Fig. 2. The attractors of genotypic frequencies in the area of protected polymorphism for the same competition coefficients c as in Fig. 1. Due to symmetries around the main diagonal and off-diagonal only one quadrant is shown. The different shades of gray indicate the type of attractor, where the lightest designates an equilibrium, the intermediate a periodic orbit and the darkest gray a chaotic attractor. For each value of c the preference parameter α takes values 20, 30 and 50.

competition coefficients $c = 1, 2, 5$. Increasing c, or more precisely c/s (i.e. increasing the strength of the frequency-dependent effect of competition), the region of protected polymorphism increases. Furthermore, recall that the preference parameter α doesn't affect the invasion ability of a rare allele (see (16)), and hence the size of the area of protected polymorphism doesn't change with α. Parameter α does, however, affect the magnitude of the reproductive advantage of fit individuals and therefore also the dynamics of coexisting alleles x and y.

Fig. 2 shows the distribution of polymorphic attractors for $c = 1, 2, 5$, $x \in [-1, 1]$, $y \in [-1, 1]$ and $\alpha = 20, 30, 50$. The different shades of gray indicate the type of attractor, where the lightest designates an equilibrium, the intermediate a periodic orbit and the darkest gray a chaotic attractor. Chaotic attractors were identified by having a positive Lyapunov exponent (Guckenheimer and Holmes, 1983). For some allele values a coexistence of two attractors is observed. The two attractors may be two periodic orbits, a periodic orbit and a chaotic attractor, or two chaotic attractors. However, this happens only for a narrow parameter region and therefore it is not shown in Fig. 2 (but see Fig. 3). For all c and for small values of α the alleles x and y can coexist only at an equilibrium point (for $\alpha \lesssim 10$ all diagrams are similar to 2A). Increasing α the equilibrium may first bifurcate to a periodic attractor (see for example 2B for $x = -0.5$ and $y = 0.3$) and with even higher values of α to a chaotic attractor (see for example 2E for the same allele values). Notice that increasing c or α complex attractors spread across the allele space.

The transition to chaos happens via period-doubling bifurcations as shown in the series of panels in Fig. 3. In each plot I fixed all the components that define the strength of ecological

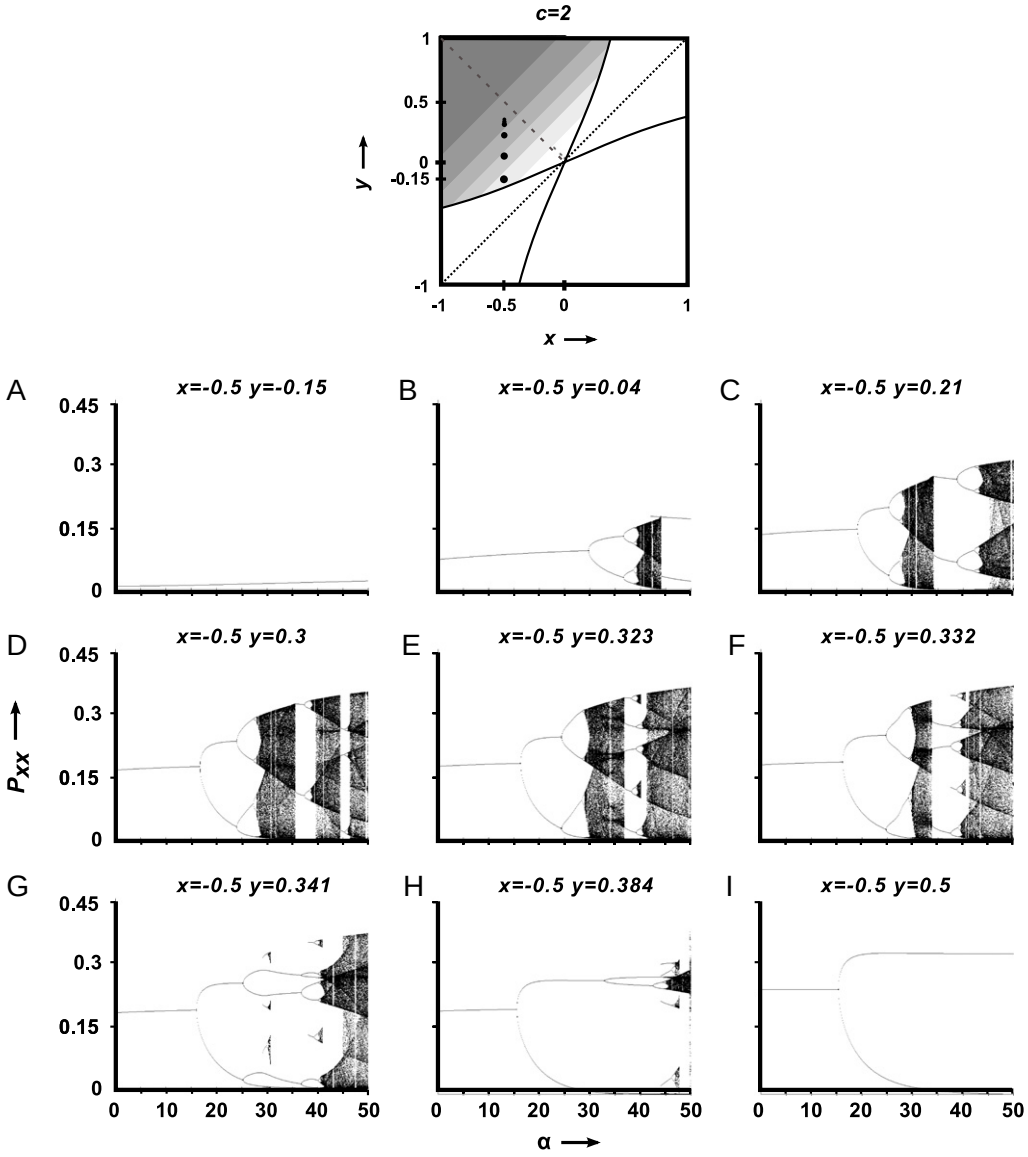

Fig. 3. Bifurcation plots for $c = 2$ and for allele values that are indicated in the top panel as well as above each bifurcation plot. *Top panel*: The biggest dot corresponds to values $x = -0.5$ and $y = -0.15$ used in panel A and the smallest dot to values $x = -0.5$ and $y = 0.5$ used in panel I. Also the strength of competition between homozygotes xx and yy is indicated. The different shades of gray correspond to competition values $u_{xx,yy}$ between [0, 0.1], [0.1, 0.3], [0.3, 0.5], [0.5, 0.7], [0.7, 0.9] and [0.9, 1] from the darkest gray to white, respectively. Note that only the upper left half is drawn in detail as the bottom half is its mirror image. *Panels A–I*: All the existing (and coexisting) attractors are calculated as the bifurcation parameter α varies from 0 to 50, of which the frequency values (that belong to the attractor) of a homozygote xx are plotted. (A) For all α there exists a unique equilibrium. (B)–(H) A period-doubling route to chaos is observed. (I) an equilibrium bifurcates to a period-two cycle. Multiple attractors are observed in (B) period-three cycle and chaotic attractor for $42 \lesssim \alpha \lesssim 44$. (E) Small chaotic attractor and periodic cycles for $\alpha \approx 39$. (F) Two chaotic attractors for $\alpha \approx 40$. (G) A periodic cycle and a chaotic attractor for $\alpha \approx 31$ and two chaotic attractors for $\alpha \approx 41$. (H) Two chaotic attractors for $\alpha \approx 47$.

selection (c, x and y), and used the preference parameter α as the bifurcation parameter. The homozygote frequency P_{xx} of the attractor is drawn. The allele values used in Fig. 3 and the corresponding strength of competition between extreme phenotypes $u_{xx,yy}$ are indicated in the top panel of Fig. 3. For example, in panel 3A where the values $x = -0.5, y = -0.15$ are close to the invasion boundary of x (the lower continuous line on the left of the main diagonal in the top panel of Fig. 3) the strength of competition is strong (i.e. the frequency-dependent effect of competition is weak, see Section 2.1), $u_{xx,yy} \approx 0.78$, while in the other extreme case in panel 3I where $x = -0.5$, $y = 0.5$ the competition $u_{xx,yy} \approx 0.14$ is weaker (i.e. the frequency-dependent effect of competition is stronger). The weak frequency-dependent effect of competition in panel 3A results in an attractor that is an equilibrium for all $\alpha \in [0, 50]$. Because the allele x is close to its invasion boundary, which is in this case also the extinction boundary, the frequency of xx at the equilibrium is close to 0.

In panel 3B where the frequency-dependent effect of competition is slightly stronger an equilibrium bifurcates to a period-two cycle at $\alpha \approx 30$, and after a series of period doubling bifurcations a chaotic orbit emerges at $\alpha \approx 40$. Notice that for $42 \lesssim \alpha \lesssim 44$ a period-three orbit exists simultaneously with the chaotic attractor after which the chaotic attractor is destroyed (the third branch of the period three is close to 0 and hence not visible in the plot). This destruction happens via a so-called boundary crisis (Grebogi et al., 1983), where an unstable period-three saddle on the basin boundary (not visible) collides with the chaotic attractor. After this bifurcation point the trajectories starting from initial values formerly in the basin of attraction of the chaotic attractor show a chaotic transient before eventually settling to the remaining period-three attractor.

In panel 3C another type of crisis occurs. First, a period-doubling route to chaos is observed after which the chaotic attractor is

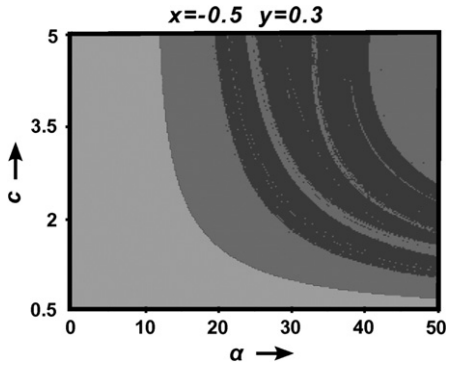

Fig. 4. Attractors for allele values $x = -0.5$ and $y = 0.3$ as α ranges from 0 to 50 and c from 0.5 to 5. The different shades of gray indicating the attractors are as in Fig. 2. Note that increasing α or c the complexity of attractors generally increases.

replaced by a stable and unstable period-three cycle produced by saddle–node bifurcations (a so-called subduction). Then, the stable cycle undergoes a period-doubling route to chaos after which the chaotic attractor collides with an unstable cycle causing a sudden change in the size of the chaotic attractor ($\alpha \approx 45$). Because the collision happens within the basin of attraction it is called an interior crisis (Grebogi et al., 1983). Furthermore, for all $\alpha \in [0, 50]$ the attractor is unique. The same holds for panel 3D while in panels 3E–H a smaller chaotic attractor coexists either with a periodic cycle (panel 3E for $\alpha \approx 39$ and panel 3G for $\alpha \approx 31$) or another chaotic attractor (panel Fig. 3F for $\alpha \approx 40$, panel Fig. 3G for $\alpha \approx 41$ and panel Fig. 3H for $\alpha \approx 47$).

Polymorphic attractors in a symmetric selection. The allele values $x = -0.5$ and $y = 0.5$ in panel 3I are placed symmetrically around $\theta = 0$ where the coefficients of ecological selection are exactly the same for both homozygotes xx and yy. As a consequence, period-doubling bifurcations can't go past period two. The reason is that due to exactly the same selection coefficients of xx and yy the existing attractor is placed symmetrically around the line of equal homozygote frequencies $P_{xx} = P_{yy}$. For instance, an equilibrium lies exactly on the line $P_{xx} = P_{yy}$ and if it bifurcates to a period-two cycle the components of period two $(\hat{P}^1_{xx}, \hat{P}^1_{yy})$ and $(\hat{P}^2_{xx}, \hat{P}^2_{yy})$ lie symmetrically on both sides of $P_{xx} = P_{yy}$ such that $\hat{P}^1_{xx} = \hat{P}^2_{yy}$ and $\hat{P}^2_{xx} = \hat{P}^1_{yy}$. That is, a point $(\hat{P}^1_{xx}, \hat{P}^1_{yy})$ is mapped to a point $(\hat{P}^2_{xx}, \hat{P}^2_{yy})$ and then back again because the selection components are symmetrical $v^1_{xx} = v^2_{yy}$ and $v^2_{xx} = v^1_{yy}$. Now, if a further period-doubling bifurcation would happen there must be one mapping from one point of the (symmetrically placed) attractor to another point $(\hat{P}^i_{xx}, \hat{P}^i_{yy}) \to (\hat{P}^{i+1}_{xx}, \hat{P}^{i+1}_{yy})$ such that $\hat{P}^i_{xx} = \hat{P}^{i+1}_{yy}$ and $\hat{P}^{i+1}_{xx} = \hat{P}^i_{yy}$ (in period-doubling each mapping causes a jump from a point on a branch of the former periodic cycle to a point on another branch, and hence one jump must happen from a point to its symmetrically placed counterpart). This, however, would result in a jump back to the previous point because $v^i_{xx} = v^{i+1}_{yy}$ and $v^i_{xx} = v^{i+1}_{yy}$ and therefore $(\hat{P}^{i+1}_{xx}, \hat{P}^{i+1}_{yy}) \to (\hat{P}^i_{xx}, \hat{P}^i_{yy})$, hence continuing as a period-two cycle. Any periods of higher order are therefore excluded in a situation where homozygotes xx and yy experience exactly the same selection pressures.

The strength of preference for condition-dependent traits. Figs. 2 and 3 seem to suggest that as an increase of c (more precisely c/s) or an increase of the preference parameter α occurs, then the complexity of attractors increases. Fig. 4 showing the interplay between c and α for allele values $x = -0.5$ and $y = 0.3$ confirms this observation. The question arises why this is so. Suppose that alleles x and y are able to invade each other, hence ensuring the existence of a polymorphic attractor, and that $\alpha = 0$ so that the dynamics is governed purely by ecological interactions. For all $c \in [0.5, 5]$ and all x and y in the region of protected polymorphism the polymorphic attractor turns out to be a unique equilibrium. Moreover, the convergence to the equilibrium is monotonic. However, for $\alpha > 0$ the advantage of fit individuals increases so that the jumps in frequency values from generation to generation also increase. For sufficiently high α this may first lead to overshoots and an oscillatory convergence and then to loss of stability. At this critical point a period-two bifurcation happens (note that trajectories further away from the attractor are not affected much and they continue converging). This may be followed by further period-doubling bifurcations until a chaotic attractor is born. Note that this change in dynamics is a consequence of increasing the advantage of fit individuals by increasing the strength of mating preference α. As the advantage of fit individuals increases also with increasing c (i.e. c/s), a stronger frequency-dependent effect of competition reinforces the effect of α.

Another question arises about the necessary level of preference to cause complex dynamics. As a measure of the strength of mating preference I use the relative probability to choose the more fit homozygote male over the less fit homozygote male, which I denote with q. Hence for $v_{xx} > v_{yy}$ I use $q = \frac{\gamma_{xx}}{\gamma_{yy}}$ and for $v_{yy} > v_{xx}$ I use $q = \frac{\gamma_{yy}}{\gamma_{xx}}$. In cases where the magnitude of v changes over time, e.g. periodic cycles, I always choose the biggest value. For the first period-two bifurcation to occur, q is of order 10^0. For example, for $x = -0.5$, $y = 0.3$ and $c \in [1, 5]$ the value is $q \approx 1.3$. On the other hand, the first chaotic orbit emerges for q of order 10^2, that is, during the chaotic cycle the advantage of fit males increases to $q = 100$–1000. For example, for $x = -0.5$, $y = 0.3$ and $c = 2$ the value is $q \approx 770$ and for somewhat weaker competition $c = 5$ it is $q \approx 260$. This shows that the first period-doubling bifurcation requires only a very weak preference for fit males whereas the preference needs to be considerably stronger for chaos to emerge.

3.2. Condition-dependent vs. assortative mating

Finally, I give a brief account of some observations on the model with assortative mating ($\beta > 0$). Kisdi and Priklopil (2011) showed that assortative mating hinders the invasibility of alleles and can cause unprotected coexistence (Priklopil, 2012), that is, a polymorphism lacking mutual invasibility. Indeed, the value of the invasion fitness

$$W_x(y) = \frac{1}{2}\left(1 + \frac{\gamma_{xy}}{\gamma_{xx}}\pi_{xx,xy}\right)\frac{v_{xy}}{v_{xx}} \tag{17}$$

may decrease below 1 as the value of π decreases when assortative mating becomes stronger. Strong condition-dependent mating preference, however, may balance this effect and enable the allele to invade. Condition-dependent mating in this respect works against assortative mating.

In Fig. 5 I show for $c = 2$, $x = -0.5$ and $y = 0.3$ how the bifurcation scheme is affected when β increases from 0 to 8. First, notice that for small α the increase of β causes the homozygotes to mate within their own genotypes (rather than preferring possibly more fit males of other genotypes), hence causing the equilibrium frequency of heterozygotes to decrease and the homozygotes to increase. Surprisingly, homozygotes mate assortatively even after the first period-doubling bifurcation. For example in panel 5B, for $\beta = 2$ and $\alpha = 19.3$ homozygotes mate assortatively during the period-two cycle with $(\hat{P}^1_{xx}, \hat{P}^1_{yy}) \approx (0.27, 0.36)$ and $(\hat{P}^2_{xx}, \hat{P}^2_{yy}) \approx (0.20, 0.44)$. However, in order to reach a more complex cycle or a chaotic attractor α needs to be strong enough to cause the condition-dependent mating to take over and cause disassortative mating so that the frequencies from generation to generation can alter more wildly.

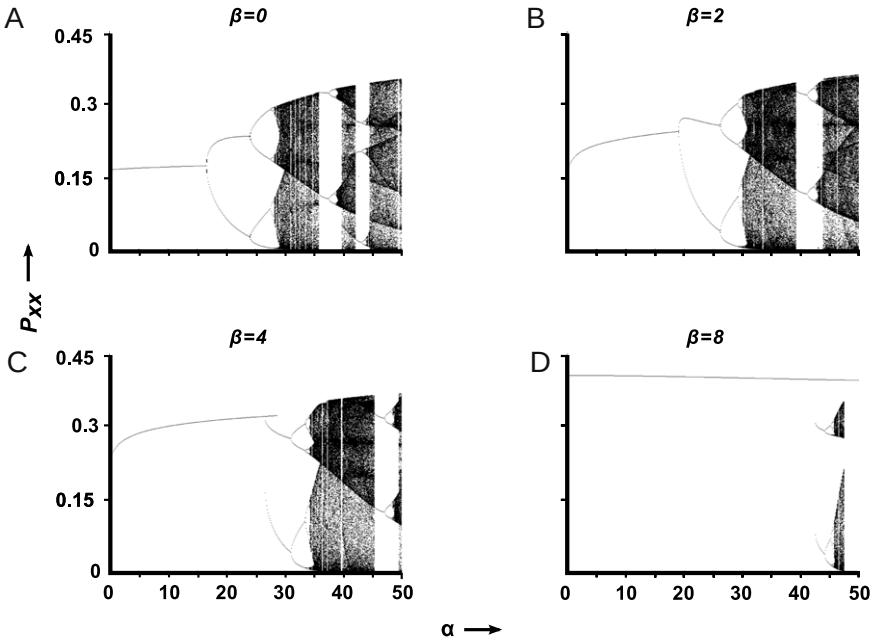

Fig. 5. Bifurcation plots for competition coefficient $c = 2$ and allele values $x = -0.5$ and $y = 0.3$. (A) No assortative mating, period-doubling route to chaos identical to panel Fig. 3D. (B) Period-doubling route to chaos with elevated frequency of an equilibrium value. (C) Discontinuous period-doubling route to chaos such that an equilibrium coexists with a period-two cycle for $26 \lesssim \alpha \lesssim 28$. (D) An equilibrium exists for all $\alpha \in [0, 50]$, and for $42 \lesssim \alpha \lesssim 47.5$ it coexists with a period-doubling route to chaos.

Second, for $\beta > 0$ an equilibrium can coexist with a periodic or chaotic attractor (panels 5C and D). This may result in attractor switching as α increases. For example in panel 5C, a population at the equilibrium experiences a sudden jump to a period-two cycle when the mating parameter passes the value $\alpha \approx 29$. Note further that for $\beta = 8$ in panel 5D alleles x and y (when rare) are not able to invade the resident population for $\alpha \lesssim 0.80$ and $\alpha \lesssim 1.94$, respectively. In this region the corresponding boundary equilibria are stable and hence the coexistence of alleles at the strictly positive equilibrium is unprotected from extinction.

4. Discussion

In this work I studied the allele frequency dynamics at a single diploid locus with two alleles in a model where a population is under disruptive ecological selection and where females choose males for mating based on traits that correlate with frequency-dependent condition. Furthermore, I also considered a case where females have developed an additional preference for ecological traits that are similar to their own. In this setting a complex dynamics of alleles, such as periodic cycling and chaos, is observed when females have sufficiently strong preferences for condition-dependent traits. These results also hold when females maintain, up to a moderate level, a preference for assortative mating. Moreover, multiple attractors can exist simultaneously.

The cause of periodic and aperiodic fluctuations of allele frequencies is the combination of frequency-dependent disruptive ecological selection that acts on phenotypes ϕ_{xx}, ϕ_{xy} and ϕ_{yy} and condition-dependent mating. As the condition correlates with the frequency-dependent viability of the individual, the probability to accept a certain type of male for mating changes with the frequency distribution of the population. Phenotypes that are preferred in one generation might be at a disadvantage in the next. This is the case with negative frequency-dependent selection and strong preference for fit males. Because rare and fit males sire most of the offspring in the next generation, the previously common types become rare (and therefore fit) and the roles are reversed. Indeed, this process may continue indefinitely and result in periodic fluctuations or even chaos (Fig. 3). The necessary prerequisite for aperiodic attractors is an asymmetry in selective forces acting on homozygotes xx and yy. Only in this case can the fluctuations be irregular enough to produce cycles longer than period two, and ultimately lead the dynamics to chaos (compare for example panel 3D where $x = -0.5$ and $y = 0.3$ with a panel 3I where $x = -0.5$ and $y = 0.5$). Chaotic dynamics is in this sense a generic feature which unfolds when the symmetry of selection pressures is relaxed.

Competition (as described by formula (6)) regulates the strength of frequency-dependence and therefore it reinforces the effect of preference for condition-dependent traits (Fig. 4). It turns out, however, that the selective forces need not be very strong for periodic cycles to occur. Suppose for instance that the competition coefficient gets a value $c = 2$ while allele values are $x = -0.5$ and $y = 0.3$ (recall that $s = \frac{1}{2}$, see panel 3D). For $\alpha \approx 16.7$, where the equilibrium bifurcates to a period-two cycle, females prefer the more fit homozygote males only 1.3 times more than the less fit ones. In fact, during a period-two cycle females may even mate assortatively. However, for increasing α the fluctuations in frequencies get wilder, and at values where a chaotic orbit emerges the preference for fit males is substantially stronger. For the same parameter values c, x and y as above the chaotic orbit starts at $\alpha \approx 27.8$ (see panel 3D) where the more fit homozygote males are preferred up to 770 more than the less fit ones. Even for $c = 5$ when the frequency-dependent effect of competition is stronger the preference goes up to 260. It seems that for chaotic dynamics to occur females need to develop a strong preference for condition-dependent traits. Empirical results on condition-dependent mating systems suggest that preference for condition-dependent traits may indeed be strong (Kotiaho et al., 2001; Cotton et al., 2006). For example, the study on stalk-eyed flies (Cotton et al., 2006) shows that females accept nearly all males whose eyespan (a condition-dependent trait) passes a certain threshold while males whose eyespan is smaller than another threshold are always rejected. This indicates an extremely strong preference for fit males. In the experiment of Kotiaho et al. (2001) not a single dung beetle male that had a courtship rate (a condition-dependent trait) past some critical value was rejected for mating, also suggesting strong preferences. It seems that when it pays for

the females to develop a preference for traits that indicate the male's condition the preference can cause a strong advantage for fit males.

A strong preference for condition-dependent traits might also be a by-product of the following simple evolutionary scenario. Suppose that females have developed high values of α and that the selection pressures are such that the population is at an equilibrium state (the scenario might be, for example, as in panel 3A). Even if α is high the viabilities of different phenotypes are similar enough for the advantage of fit homozygote males to be only of the order of 10^0 (in panel 3A for $\alpha = 28$ the advantage is 2.7). Now, consider a beneficial mutation that spreads in the population and substitutes one of the alleles. If the new allele enhances the frequency-dependent effect of selection, the dynamics of allele frequencies might change drastically and can cause periodic fluctuation or chaos (for the $\alpha = 28$ considered above, see for example panels 3H and 3C). Note that the alleles that code for the mating preference α remain the same, and instead, it is the change in the phenotype distribution of the population that leads to a strong advantage of fit males and to abrupt changes in dynamical behavior.

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References

Agrawal, A.F., 2001. Sexual selection and the maintenance of sexual reproduction. Nature 411, 692–695.
Altenberg, L., 1991. Chaos from linear frequency-dependent selection. Am. Nat. 138, 51–68.
Andersson, M., 1994. Sexual Selection. Princeton University Press, Princeton (NJ).
Andersson, M., Simmons, L.W, 2006. Sexual selection and mate choice. Trends Ecol. Evol. 21, 296–302.
Brooks, R., Couldridge,, 1999. Multiple sexual ornaments coevolve with multiple mating preferences. Am. Nat. 154, 37–45.
Bulmer, M.G., 1974. Density-dependent selection and character displacement. Am. Nat. 108, 45–58.
Bulmer, M.G., 1980. The Mathematical Theory of Quantitative Genetics. Clarendon Press, Oxford, UK.
Bürger, R., 2002. On a genetic model of intraspecific competition and stabilizing selection. Am. Nat. 160, 661–682.
Bürger, R., Schneider, K.A., 2006. Intraspecific competitive divergence and convergence under assortative mating. Am. Nat. 167, 190–205.
Chapman, T., Liddle, L.F., Kalb, J.M., Wolfner, M.F., Partridge, L., 1995. Cost of mating in Drosophila melanogaster females is mediated by male accessory gland products. Nature 373, 241–244.
Clarke, B.C., Partridge, L. (Eds.), 1988. Frequency-Dependent Selection. Royal Society, London.
Cotton, S., Fowler, K., Pomiankowski, A., 2004. Do sexual ornaments demonstrate heightened condition-dependent expression as predicted by the handicap hypothesis? Proc. R. Soc. Lond. B 271, 771–783.
Cotton, S., Rogers, D.W., Small, J., Fowler, K., Pomiankowski, A., 2006. Variation in preference for a male ornament is positively associated with female eyespan in the stalk-eyed fly *Diasemopsis meigenii*. Proc. R. Soc. Lond. B 273, 1287–1292.
Cushing, J.M., Costantino, R.F., Dennis, B., Desharnais, R.A., Henson, S.M., 2003. Chaos in Ecology: Experimental Nonlinear Dynamics. In: Theoretical Ecology Series, vol. 1. Academic Press, Elsevier Science, New York.
Doebeli, M., 2011. Adaptive Diversification. Princeton University Press.
Gavrilets, S., Boake, C.R.B., 1998. On the evolution of premating isolation after a founder event. Am. Nat. 152, 706–716.
Gavrilets, S., Hastings, A., 1995. Intermittency and transient chaos from simple frequency-dependent selection. Proc. R. Soc. Lond. B 261, 233–238.
Grafen, A., 1990. Biological signals as handicaps. J. Theor. Biol. 144, 517–546.
Grebogi, C., Ott, E., Yorke, J.A., 1983. Crises, sudden changes in chaotic attractors, and transient chaos. Phys. D 7, 181–200.
Guckenheimer, J., Holmes, P., 1983. Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields. Springer-Verlag, New York.
Hegde, S.N., Krishna, M.S., 1997. Size-assortative mating in *Drosophila malerkotliana*. Anim. Behav. 54, 419–426.
Iwasa, Y., Pomiankowski, A., 1994. The evolution of mate preferences for multiple sexual ornaments. Evolution 48, 853–867.
Iwasa, Y., Pomiankowski, A., Nee, S., 1991. The evolution of costly mate preferences. II. The "handicap" principle. Evolution 45, 1431–1442.
Jennions, M.D., Petrie, M., 2000. Why do females mate multiply? A review of the genetic benefits. Biol. Rev. 75, 21–64.
Kisdi, E., Priklopil, T., 2011. Evolutionary branching of a magic trait. J. Math. Biol. 63, 361–397.
Kokko, H., Brooks, R., Jennions, M.D., Morley, J., 2003. The evolution of mate choice and mating biases. Proc. R. Soc. Lond. B 270, 653–664.
Kopp, M., Hermisson, J., 2006. The evolution of genetic architecture under frequency-dependent disruptive selection. Evolution 60, 1537–1550.
Kotiaho, J.S., Simmons, L.W., Tomkins, J.L., 2001. Towards a resolution of the lek paradox. Nature 410, 684–686.
Lorch, P.D., Proulx, S., Rowe, L., Day, T., 2003. Condition-dependent sexual selection can accelerate adaptation. Evol. Ecol. Res. 5, 876–881.
Møller, A.P., Alatalo, R.V., 1999. Good-genes effects in sexual selection. Proc. R. Soc. Lond. 266, 85–91.
Møller, A.P., Jennions, M.D., 2001. How important are direct benefits of sexual selection. Naturwissenschaften 88, 401–415.
Møller, A.P., Pomiankowski, A., 1993. Why have birds multiple sexual ornaments? Behav. Ecol. Sociobiol. 32, 167–176.
Peischl, S., Schneider, K.A., 2010. Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population. Evolution 64, 561–582.
Pomiankowski, A., Iwasa, Y., 1993. Evolution of multiple sexual preferences by Fisher's runaway process of sexual selection. Proc. R. Soc. Lond. B 253, 173–181.
Priklopil, T., 2012. On invasion boundaries and the unprotected coexistence of two strategies. J. Math. Biol. 64, 1137–1156.
Proulx, S.R., 1999. Matings systems and the evolution of niche breadth. Am. Nat. 154, 89–98.
Proulx, S.R., 2001. Female choice via indicator traits easily evolves in the face of recombination and migration. Evolution 55, 2401–2411.
Proulx, S.R., 2002. Niche shifts and expansion due to sexual selection. Evol. Ecol. Res. 4, 351–369.
Prout, T., 1968. Sufficient conditions for multiple niche polymorphism. Am. Nat. 102, 493–496.
Scheuber, H., Jacoty, A., Brinkho, M.W.G., 2004. Female preference for multiple condition-dependent components of a sexually selected signal. Proc. R. Soc. Lond. B 271, 2453–2457.
Schluter, D., McPhail, J.D., 1992. Ecological character displacement and speciation in sticklebacks. Am. Nat. 140, 85–108.
Schneider, K., 2008. Maximization principles for frequency-dependent selection I: the one-locus two-allele case. Theor. Pop. Biol. 74, 251–262.
Siller, S., 2001. Sexual selection and the maintenance of sex. Nature 411, 689–692.
Snowberg, L.K., Bolnick, D.I., 2008. Assortative mating by diet in a phenotypically unimodal but ecologically variable population of stickleback. Am. Nat. 172, 733–739.
Trotter, M.V., Spencer, G.H., 2009. Complex dynamics occur in a single-locus, multiallelic model of general frequency-dependent selection. Theor. Pop. Biol. 76, 292–298.
Turchin, P., 2003. Complex Population Dynamics: A Theoretical/Empirical Synthesis. Princeton University Press.
van Doorn, S., Weissing, F., 2009. On the origin of species by natural and sexual selection. Science 326, 1704–1707.
van Doorn, S., Weissing, F., 2004. The evolution of female preferences for multiple indicators of quality. Am. Nat. 164, 173–186.
Whitlock, M.C., 2000. Fixation of new alleles and the extinction of small populations: drift load, beneficial alleles, and sexual selection. Evolution 54, 1855–1861.
Yi, T., Cressman, R., Brooks, B., 1999. Nonlinear frequency-dependent selection at a single locus with two alleles and two phenotypes. J. Math. Biol. 39, 283–308.

Article IV

The perfect female's sequential search for males and reproductive isolation by assortative mating

Tadeáš Přiklopil, Éva Kisdi & Mats Gyllenberg

The perfect female's sequential search for mates and reproductive isolation by assortative mating

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Abstract

We find the evolutionarily stable mating strategy of females who have complete information about the size and frequency distribution of the male population as well as about their own condition (e.g. genotype), but are constrained to search for mates sequentially via random encounters within a mating season of limited length. We show that the evolutionarily stable strategy is always a time-threshold strategy that rejects a given type of male if encountered before a time-threshold and accepts after, and give the optimal time-thresholds in terms of the benefits the females receive from various males. Next, we apply the model to the case where males contribute only their genes to the offspring and offspring genotype determines ecological fitness. As an example, we obtain the equilibrium genotypic frequencies under the evolutionarily stable mating strategy in Levene's one-locus-two-allele soft selection model, and establish the conditions for reproductive isolation between homozygotes adapted to contrasting habitats.

Mate choice is a decisive process that shapes the genotypic distribution of populations in the course of evolution. Females, who are often the active sex in mate choice, are faced with an enterprise to select a male that ensures the production and survival of their progeny. In many species the search for males is constrained to happen sequentially in time (Janetos 1980, Real 1990, Bakker and Milinski 1991, Backwell and Passmore 1996, Forsgren 1997, Houde 1997, Ivy and Sakaluk 2006, Lehmann 2007), such that at each encounter with a male the female faces a decision to be either satisfied with the male in which case she accepts him for mating and terminates her search, or to decline him and continue to seek for other males. Ideally, the choice made at each encounter reflects the quality or quantity of benefits the encountered mate is offering, where benefits could be either direct, e.g. high-quality territory, nutrition, parental care or protection (Møller and Jennions 2001, Andersson and Simmons 2006) or indirect, i.e., genes for offspring (Møller and Alatalo 1999, Andersson 2006, Andersson and Simmons 2006). Because greater benefits by definition increase the survival and/or the reproductive succes of the female and/or her progeny, females are under selection pressure to use the best possible search tactic and mate choice criteria.

In this paper we show that the mating strategy that optimizes the benefits for a sequentially searching female is a particular solution of a time-threshold tactic. In time-threshold tactics a male is accepted for mating if he is encountered after a certain time-threshold that depends on his genotype and rejected if he is encountered before (some genotype(s) will have zero time-threshold, i.e., will be accepted from the beginning of the mating season). Motivated by this, we develop a mating model for the time-threshold tactics in a general setting, i.e. where optimality is not necessarily guaranteed. After the general formulation, we give an explicit expression for time-thresholds that are optimal. The optimal time-thresholds are given in terms of the benefits. Because the benefits may depend on the genotypic frequencies of the resident population (for example, this is the case with indirect benefits when sexual

selection operates on the offspring), the time-thresholds that are optimal in the given equilibrium population represent an evolutionarily stable strategy (Maynard Smith 1982).

Time-threshold tactic enables to model various strengths of assortative mating, and importantly, in order to avoid the risk of remaining unmated, it allows for the relaxation of female choosiness as the mating season proceeds (Backwell and Passmore 1996, Thomas et al. 1998, Gray 1999, Kodric-Brown and Nicoletto 2001, Moore and Moore 2001). For example, females might accept right from the beginning of the season only the most desirable males while the least desirable males will be accepted only towards the end. The desirability of the male will be formulated in terms of benefits that males provide to females, or to their offspring, and will depend not only on the quantity or quality of the benefit but also on the suitability of the benefit for the female or to their offspring. This is often a neglected point in the so called good genes models, where the genes of the father are assumed to be good irrespective of the environment he and the recipients of the benefits are adapted to (Iwasa et al. 1991, Iwasa and Pomiankowski 1994, Proulx 2001, Lorch et al. 2003). This becomes problematic if females are allowed to choose males across an adaptive valley (van Doorn et al. 2009, Priklopil 2012) which may result in producing unfit phenotypes and can cause turbulent dynamics of allele frequencies (Priklopil 2012).

The action of mate search in our mating model is described by a Poisson process, where the expected number of males sampled during the mating season depends on the length of the mating season and the rate of encounters, which in turn depends on population density. This, along with the assumptions made above, allows us to avoid some difficulties of many past population genetic models. For example, a widely used mating model (introduced by Gavrilets and Boake 1998 and Matessi et al. 2001 and used e.g. by Kirkpatrick and Nuismer 2004, Schneider 2005, Schneider and Bürger 2006, Pennings et al. 2008, Peischl and Schneider 2009, Ripa 2009, Kisdi and Priklopil 2011) assumes the number of males to encounter to be constant and female choosiness to be fixed throughout the season (but see Kopp and Hermisson 2008). Surely, if a female can't find any males due to low population density it makes sampling $M > 0$ males impossible. Also, being choosy even at the end of the mating season seems biologically unreasonable. Many of these models also assume that assortative mating occurs by self-referent phenotype matching based on a "magic trait" (Gavrilets 2004, Servedio et al. 2011), i.e., females prefer males with some ecological character similar to themselves. The greatest difficulty with this is that it allows maladapted females to prefer maladapted males. In our model, assortative mating, or any other mating scenario, results only if it is beneficial to the female (or her offspring). For optimal time-thresholds, where the benefits are maximized, females thus possess a perfect preference for males. However, the expression for optimal time-thresholds requires the knowledge of some demographic parameters, such as population density and the frequency distribution of genotypes, as well as the length of the mating season. Of course, females don't actually need to hold this information (but see Discussion) but could have evolved their mating preference as a response to the selective environment. Nevertheless, since we consider females who act as if they were omniscient about their selective environment, we refer to them as "perfect females".

Based on the expression for optimal thresholds, we derive the condition under which females should mate randomly in order to gain the maximum benefits. Conversely, if this condition doesn't hold, then females are under a selection pressure to develop a preference for traits that are honest indicators of the benefits males provide (Zahavi 1975, Grafen 1990, Iwasa et al. 1991, Iwasa and Pomiankowski 1994). Finally, and most importantly, we study whether our mating model preserves a polymorphism of alleles, and if so, what type of optimal preferences ecologically different scenarios invoke. To work out the exact mating strategies perfect females should adopt, we need to apply our mating model to a specific ecological setting. We will use a version of the well known Levene model (Levene 1953) and give special attention to ecological scenarios which are conclusive to reproductive isolation. We find that if females are able to sample enough males during the mating season, in our model of the order of magnitude 5 to 20 males, polymorphism with two reproductively isolated clusters can be maintained.

The Model

Consider a large and well-mixed sexually reproducing population of diploid individuals with k different genotypes and non-overlapping generations. Females (the choosy sex) encounter males (that are always ready to mate) sequentially (Janetos 1980, Real 1990, Bakker and Milinski 1991, Forsgren 1997, Houde 1997, Ivy and Sakaluk 2006, Lehmann 2007), such that at each encounter with a male the female has to either accept the male for mating and terminate her search or decline the male and continue to seek for new males. Each encounter is assumed to be independent of the

previous or future encounters (unless the female accepts the male and terminates her search), thus the time interval between consecutive encounters is exponentially distributed with some rate parameter ρ (Ross 1995). The probability to encounter an individual in some time interval Δt is then $1 - \exp[-\rho \Delta t]$. The rate ρ depends on population density and may also depend on the effort invested by the searcher (see Discussion).

In the following section, we derive an expression for the probability of mating between a given female and male genotype, assuming that females use a general time-threshold tactic. In this general setting, we only assume that female preference is expressed as having different (and arbitrary) time-thresholds for different types of males, such that if a male is encountered before a time-threshold connected to his genotype he will be rejected and if encountered after he will be accepted. For each female genotype f there hence exists a time-threshold vector $t_{f,g} = (t_{f,g_1}, t_{f,g_2}, \ldots, t_{f,g_k})$, where male genotypes g are ordered such that $0 \leq t_{f,g_1} \leq \cdots \leq t_{f,g_k} \leq T$, and where t_{f,g_m} is the time-threshold belonging to the male of genotype g_m, for $1 \leq m \leq k$ (see Figure 1). The number of different genotypes that are accepted right from the beginning of the season might be greater than one, and we denote this number with n, where $1 \leq n \leq k$, so that we have $t_{f,g_1} = \cdots = t_{f,g_n} = 0$. Note that whenever the time-threshold depends on the female genotype, we need to use indexing also for female genotypes. To simplify we however often omit the genotype notation and write $t_{f,g_i} = t_i$. Furthermore, for mathematical convenience we set $t_{k+1} = T$.

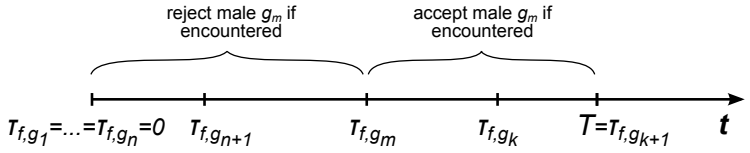

Figure 1: Time-thresholds used by a female f during a mating season of length T. In this example males of type $g_1, \ldots, g_n$ are accepted right from the beginning of the season and males of type g_m only after time t_{f,g_m}.

MATING PROBABILITY

Suppose that the female knows the frequency of genotype g_m at the beginning of the mating season, $\tilde{P}_m$ (the symbol P_m is reserved for the frequency at birth, see below). The probability for a female f and a male g_m to mate during a mating season of length T is obtained from the probability of mating within each time interval $[t_i, t_{i+1}]$ where $i = 1, \ldots, k$, and then adding these up given that the female hasn't terminated her search. Because $1 - e^{-\rho(t_{i+1}-t_i)\sum_{j=1}^{i}\tilde{P}_j}$ is the probability that acceptable males with genotypes $\{g_1, \ldots, g_i\}$ are encountered between t_i and t_{i+1} and $\frac{\tilde{P}_m}{\sum_{j=1}^{i}\tilde{P}_j}$ is the probability that a particular genotype $g_m \in \{g_1, \ldots, g_i\}$ is chosen out of the genotypes $\{g_1, \ldots, g_i\}$, we have that the probability of mating between female f and a male g_m is

$$Q_{f,g_m}\tilde{P}_m = \sum_{i=m}^{k}\Bigg[\underbrace{\frac{\tilde{P}_m}{\sum_{j=1}^{i}\tilde{P}_j}}_{\substack{\text{choose } g_m \\ \text{out of } g_1,\ldots,g_i}} \times \underbrace{\left(1 - e^{-\rho(t_{i+1}-t_i)\sum_{j=1}^{i}\tilde{P}_j}\right)}_{\substack{\text{encounter } g_1,\ldots,g_i \\ \text{between } t_i \text{ and } t_{i+1}}} \times \underbrace{\prod_{j=1}^{i-1} e^{-\rho(t_{j+1}-t_j)\sum_{l=1}^{j}\tilde{P}_l}}_{\substack{\text{remain unmated} \\ \text{until time } t_i}}\Bigg]. \quad (1)$$

OPTIMAL TIME-THRESHOLDS

In this section, we show that females should follow a time-threshold strategy if they are to maximize the benefit they receive, and derive the optimal time-thresholds. Let ε_{f,g_i} denote the benefit that a female of genotype f receives if she mates with a male of genotype g_i. Suppose that at time t in the mating season, a female f encounters a male of genotype g_i. In a sequential search, she has only two possible decisions: either she accepts this male (and hence terminates her search) or rejects this male and continues searching. Thus the most general strategy the female may follow is to accept the male with probability $q_{f,g_i}(t)$ and to reject with probability $1 - q_{f,g_i}(t)$. The benefit that this strategy yields on average is $q_{f,g_i}(t)\varepsilon_{f,g_i} + (1 - q_{f,g_i}(t))E_f(t)$, where $E_f(t)$ denotes the benefit an unmated female of genotype f can expect to receive in the remainder of the mating season. The benefit is thus a simple linear function

of $q_{f,g_i}(t)$, so that the choice of $q_{f,g_i}(t)$ that maximizes the benefit is

$$q_{f,g_i}(t) = \begin{cases} 1 & \text{if } \varepsilon_{f,g_i} > E_f(t) \\ 0 & \text{otherwise} \end{cases} \tag{2}$$

The female thus accepts the encountered male if he provides a greater benefit than what the female can expect if she continues the search; otherwise the female rejects the male and continues searching. The same result is well known in the context of optimal stopping problems. Because each random encounter brings on average the same benefit, the stopping problem is monotone and its solution is to stop when the current offer is better than the future expectation (Chow et al. 1971, p. 54).

Let us index the genotypes in decreasing order of benefits, $\varepsilon_{f,g_1} \geq \varepsilon_{f,g_2} \geq ... \geq \varepsilon_{f,g_k}$. In Appendix 1 we show that the expected benefit of females who are still unmated at time t, $E_f(t)$, changes according to

$$\dot{E}(t) = \rho \left[E(t) - \sum_{i=1}^{k} \tilde{P}_i \max\{E(t), \varepsilon_{f,g_i}\} \right] \tag{3}$$

with $E_f(T) = 0$, which reflects the fact that at the end of the mating season unmated females receive no benefit. Equation (3) is an extended version of the sequential search (or one-step decision) tactic introduced in Janetos (1980) and further developed in Real (1990), Wiegmann et al. (1999), Wiegmann and Angeloni (2007).

Because $E_f(t) \leq \sum_{i=1}^{k} \tilde{P}_i \max\{E_f(t), \varepsilon_{f,g_i}\}$ for all t, the expected benefit of unmated females, $E_f(t)$, decreases throughout the mating season towards $E_f(T) = 0$. If $E_f(0) > \varepsilon_{f,g_i}$ for a given male genotype such that, according to equation (2), the female does not accept males of this type at the beginning of the season, then there is a single time-threshold τ_i such that $\varepsilon_{f,g_i} > E_f(t)$ holds for all $t > \tau_i$ and the female accepts the male at any time after τ_i. If, on the other hand, $\varepsilon_{f,g_i} > E_f(0)$, then ε_{f,g_i} exceeds the expected benefit at all times and the male is accepted right from the beginning of the mating season. Note that, as above, we suppress the genotype indices and write ε_i instead of ε_{f,g_i} and τ_i instead of τ_{f,g_i}; it should however be kept in mind that each female genotype may have its own time-thresholds. The ordering of genotypes with decreasing benefits naturally leads to increasing time-thresholds as assumed in the previous section. Figure 2 shows a particular solution $E_f(t)$ and illustrates the time-thresholds.

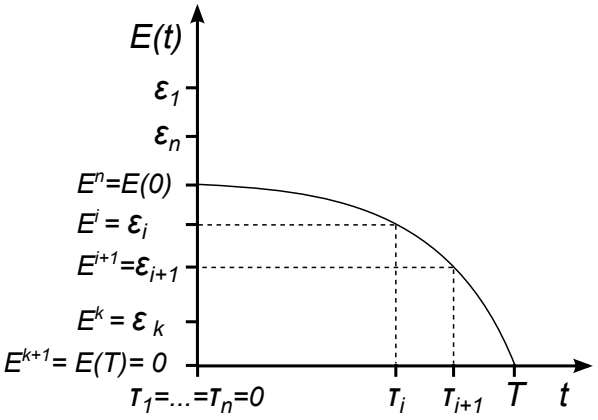

Figure 2: A particular solution to equation (3) which describes how the expectation $E(t)$ changes throughout the mating season of length T. The time-thresholds τ are optimal, i.e., when females accept males only after the optimal time-threshold connected to their genotype, they ensure receiving maximum benefits. For example, males with genotypes $g_1, \ldots, g_n$ offer higher benefits than is the initial expectation $E(0)$, i.e. $\varepsilon_1 \geq \cdots \geq \varepsilon_n > E(0)$, and therefore are accepted for mating right from the beginning of the mating season if encountered. Males with genotype g_i offer higher benefits than the female is expected to receive in the future only after the optimal time-threshold τ_i. To receive the maximum benefits, the female hence should reject male g_i before time τ_i and accept after τ_i. The expression for τ_i is given in equation (4).

Solving equation (3) analytically we obtain the optimal time-thresholds $\tau_1, \ldots, \tau_k$, which can be expressed as

$$\tau_{i+1} - \tau_i = \frac{1}{\rho \sum_{j=1}^{i} \tilde{P}_j} \ln\Big[\frac{E^{i+1} - \bar{E}^i}{E^i - \bar{E}^i}\Big], \qquad \text{for } n < i \leq k, \tag{4}$$

where $\bar{E}^i = \frac{\sum_{j=1}^{i} \tilde{P}_j \varepsilon_j}{\sum_{j=1}^{i} \tilde{P}_j}$ is the average expected benefit provided by the male genotypes $\{g_1, \ldots, g_i\}$ that would be accepted in the time interval $[\tau_i, \tau_{i+1}]$ if encountered, and where $E^i = \varepsilon_i$ for $n < i \leq k$. $E^{k+1} = E(T) = 0$ is the expectation at the end of the season and $E^n = E(0)$ is the initial expectation with $\tau_1 = \cdots = \tau_n = 0$. Recall that $\tau_{k+1} = T$; with this, we can calculate all thresholds from equation (4) explicitly, first substituting $i = k$ and then proceeding backwards with $i = k - 1$, $i = k - 2$ etc. until the time-thresholds are no longer positive, provided that the benefits ε_i are known. Note, however, that the benefits may depend on the mate choice of the resident females, $\tau_1, \ldots, \tau_k$ (see the worked example below). In this case, equation (4) determines the solution only implicitly, and there can be multiple solutions to equation (4). Each solution gives a resident strategy that is the best reply to itself, i.e., an evolutionarily stable strategy.

Once the optimal time-thresholds are found, they can be inserted into equation (1) (substitute $t_i = \tau_i$) to obtain the probabilities of mating between various genotypes, and hence establish the degree of reproductive isolation that follows from the perfect female's sequential mate choice.

WHEN IS RANDOM MATING OPTIMAL?

Females mate at random when the time-thresholds $t_1, \ldots, t_k$ are all 0. Because $t_k = 0$ implies that $t_1 = \cdots = t_{k-1} = 0$, the optimal thresholds in (4) are all 0 when $\tau_k = T - \frac{1}{\rho} \ln\left[\frac{E^{k+1} - \bar{E}^k}{E^k - \bar{E}^k}\right] \leq 0$, i.e.

$$\rho T \leq \ln\Big[\frac{\bar{E}^k}{\bar{E}^k - E^k}\Big], \tag{5}$$

where ρT gives the expected number of individuals encountered during a mating season, $\bar{E}^k$ is the average expected benefit under random mating and $E^k = \varepsilon_k$ is the expected benefit provided by the worst male of type g_k. When inequality (5) is satisfied, the optimal strategy is to mate randomly, that is, to maximize their benefit, females should accept the first encountered male. Conversely, we can say that if $\rho T > \ln\left[\frac{\bar{E}^k}{\bar{E}^k - E^k}\right]$ there exists a selection pressure for females to evolve a mating preference. This condition after a rearrangement of terms is $E^k < \bar{E}^k(1 - e^{-\rho T})$ which expresses that if the worst males benefit E^k is less than the female's expected benefit for the whole mating season (expected benefit in the population $\bar{E}^k$ times the probability to encounter a male,$1 - e^{-\rho T}$), then there is a selection pressure to discriminate against males of type g_k. However, if the worst males' benefit is close to the average benefit (the variance of benefits is small), the term on the right hand side in (5) is large and, unless females are able to sample a great number of males, mating at random is an optimal strategy. Recall, that the time-thresholds might differ for females with different genotypes, and therefore if (5) holds only for some females the population as a whole does not mate randomly.

EXPECTED BENEFITS AND REPRODUCTIVE ISOLATION

To investigate the effects of the above mating model on the dynamics of genotype frequencies, we first give an example of a benefit ε and then find optimal mating strategies in a specific ecological scenario.

Reproductive value of the offspring as benefit

The optimal time-thresholds in (4) are based on the benefits ε that males offer to females. Henceforth we assume that males contribute only their genes to the offspring and females choose their mates to maximize the expected fitness accrued from their offspring. Hence the benefit a female f receives from mating with a male g is the number of her offspring who survive and successfully reproduce, weighted with the benefits the offspring receive from reproduction. The benefit to a female f from mating with a male g is thus given by the expression

$$\varepsilon_{f,g} = K \sum_r R_{f,g\to r} v_r \cdot \tfrac{1}{2}\Big(\sum_h Q_{r,h}\tilde{P}_h \frac{\varepsilon_{r,h}}{2} + \sum_h Q_{h,r}\tilde{P}_h \frac{\varepsilon_{h,r}}{2} \Big), \tag{6}$$

where K is the number of offspring produced per female, $R_{f,g\to r}$ is the probability that parents with genotypes f and g produce an offspring with genotype r according to the Mendelian rules for a diploid autosomal locus, v_r is the probability of survival, $\frac{1}{2}Q_{r,h}\tilde{P}_h$ is the probability that the offspring is a female and that she gets mated with a male h whereas $\frac{1}{2}Q_{h,r}\tilde{P}_h$ is the probability that the offspring is a male times the expected number of females of genotype h he is mated with. The weights attached to the offspring, $\frac{\varepsilon_{r,h}}{2}$ and $\frac{\varepsilon_{h,r}}{2}$, respectively, are halved because the daughters and sons will pass the focal female's gene with probability $\frac{1}{2}$. Technically, the benefit given in (6) is the reproductive value of the couple f, g (see Appendix 2). Equation (6) determines the benefits only up to a constant, but this is irrelevant to the optimal time-thresholds because they depend only on ratios of benefits. Because $R_{f,g\to r} = R_{g,f\to r}$, the benefits are symmetric such that $\varepsilon_{f,g} = \varepsilon_{g,f}$ (but recall that $Q_{f,g}$ is different from $Q_{g,f}$). Notice that in (6), v_r can depend on the size and composition of the population so that the model encompasses arbitrary frequency-dependent ecological selection.

Reproductive isolation in the Levene-Hoekstra model

Description of the life-cycle. Suppose there are two alleles a and A in a single locus so that the possible genotypes are aa, aA and AA. The population undergoes viability (ecological) selection as in a two-habitat version of the Levene model (Levene 1953), where alleles a, A are under symmetrical selection as described by Hoekstra et al. (1985). In the Levene model the population size is assumed to be constant. The relative survival probabilities $w_g = \frac{v_g}{\bar{v}}$ of genotypes aa, aA, AA, where $\bar{v}$ is the average survival probability in the population, are

$$\begin{aligned} w_{aa} &= \frac{1}{2}\Big(\frac{1}{U_1} + \frac{1-s}{U_2}\Big) \\ w_{aA} &= \frac{1}{2}\Big(\frac{1-hs}{U_1} + \frac{1-hs}{U_2}\Big) \\ w_{AA} &= \frac{1}{2}\Big(\frac{1-s}{U_1} + \frac{1}{U_2}\Big), \end{aligned} \tag{7}$$

where s measures the strength of selection homozygotes experience in the habitat they are not adapted to, hs is the selection against heterozygotes in both habitats and $U_i = \sum_g P_g u_g^i$, with u_g^i denoting the viability of genotype g in habitat $i = 1, 2$, is the average survival probability in habitat $i = 1, 2$. The factor $1/2$ accounts for the additional assumption that the two habitats are of equal size. Notice that $\bar{w} = \sum_g P_g w_g = 1$ so that the frequency of genotype g before mating (i.e. after ecological selection) is

$$\tilde{P}_g = P_g \frac{w_g}{\bar{w}} = P_g w_g, \tag{8}$$

where P_g is the frequency of g at the beginning of the life-cycle. With these assumptions and with equal allele frequencies the heterozygotes aA do on average worse than homozygotes aa and AA during the ecological selection phase whenever $1/2 < h \leq 1$ (and better whenever $0 \leq h < 1/2$). Values $1/2 < h \leq 1$ thus describe a situation where a polymorphic population is under disruptive ecological selection and $0 \leq h < 1/2$ corresponds to stabilizing ecological selection. Figure 3 gives a graphical representation of the selection regimes during the ecological selection phase under random mating. Region A in the middle panel gives the parameter region where ecological selection is stabilizing, and regions B-D where it is disruptive. Panels 3A-D (surrounding the middle panel) show how viabilities w_{aa}, w_{aA}, w_{AA} relate to each other in each corresponding region of the middle panel. Regions A-B correspond to protected polymorphism, that is, a stable polymorphism where the fixation equilibria are unstable so that neither allele can go extinct (Prout 1968). Region C, delineated by $h_0 = \frac{1}{2-s}$ and $h_{\text{int}} = \frac{2-\sqrt{4-4s-s^2}}{2s}$, gives the area of unprotected polymorphism (where a stable polymorphic equilibrium exists simultaneously with stable fixation equilibria), and region D shows where polymorphism is not maintained. Expressions h_0 and h_{int} are derived in Appendices 3 and 4, respectively.

The phase of ecological selection is followed by the mating season during which individuals reproduce according to (1). After reproduction parents die and the new generation begins. The genotype frequencies thus change from generation to generation according to

$$P_r' = \frac{1}{\bar{Q}} \sum_{g,h} \tilde{P}_g \tilde{P}_h Q_{g,h} R_{g,h\to r}, \tag{9}$$

where Q is given in (1), $\bar{Q} = \sum_{g,h} \tilde{P}_g \tilde{P}_h Q_{g,h}$ is the mean mating success and $R_{g,h\to r}$ is as in (6).

Optimal mating strategies and reproductive isolation. Here we determine the optimal time-thresholds in the Levene-Hoekstra model for a range of the ecological parameters $s, h, \rho T$. Because the selection coefficients are symmetrical for the homozygotes (see (7)) we restrict our analysis to interior equilibria that are symmetric $\hat{P}_{aa} = \hat{P}_{AA}$ (note that at this equilibrium allele frequencies are also equal) and to time-thresholds that are symmetric for homozygote females aa and AA ($\tau_{aa,aa} = \tau_{AA,AA}$, $\tau_{aa,aA} = \tau_{AA,aA}$ and $\tau_{aa,AA} = \tau_{AA,aa}$). Both homozygote females are hence discriminating equally against the opposite homozygote and heterozygote males.

Notice that as mated females aA always produce offspring on average half heterozygote aA and half homozygote (aa and/or AA depending with which males they mate), it doesn't pay to evolve preference for either genotype since at symmetric equilibria both homozygotes are under equal selection. Therefore, we have $\varepsilon_{aA,aa} = \varepsilon_{aA,aA} = \varepsilon_{aA,AA}$

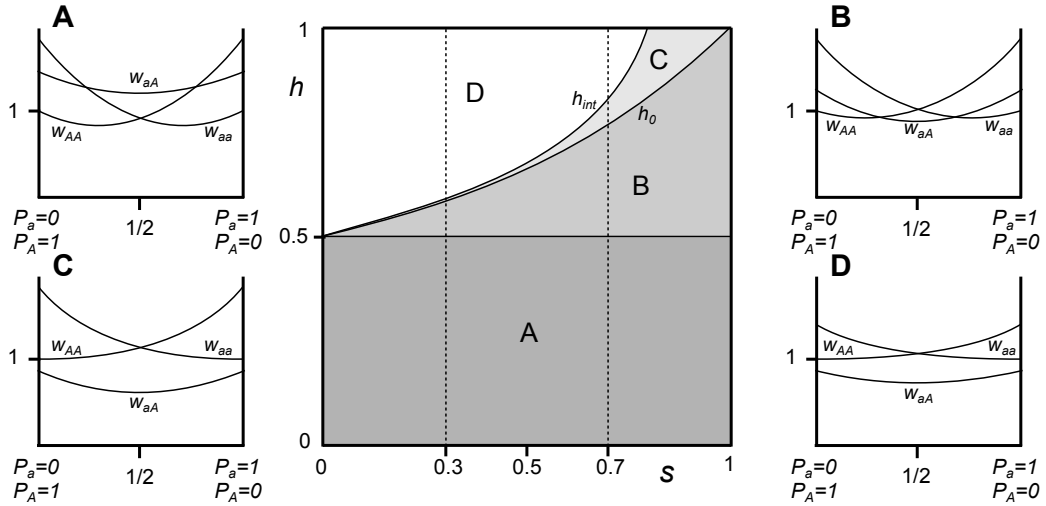

Figure 3: The strength of ecological selection for genotypes aa, aA and AA in the Levene-Hoekstra model when the population mates at random. Panels A-D show the relative viabilities w_{aa}, w_{aA}, w_{AA} for different allele frequencies, where P_a and P_A denote the allele frequencies of alleles a and A, respectively. The big panel in the middle shows for which parameter values s, h the viability configurations A-D exist, and it also indicates the stability of the symmetric interior equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$. In panel A, the heterozygotes do better near the fixation equilibria ($P_a = 0$ or $P_A = 0$) than the common homozygotes so that the polymorphism is protected. Because heterozygotes do also better at equal allele frequencies, this scenario describes stabilizing ecological selection. In panel B selection is similar close to the fixation states, but at equal allele frequencies heterozygotes do worse than homozygotes, hence ecological selection is disruptive. The equilibrium with equal allele frequencies (the symmetric equilibrium) is globally stable in areas A and B. In areas C and D heterozygotes do worse than homozygotes for all allele frequencies. However, while in both areas the fixation equilibria are stable, in area C there exists a stable symmetric equilibrium and in area D the symmetric equilibrium is unstable. In area C, the polymorphism is hence unprotected whereas in area D polymorphism is not maintained. Areas B and C in the middle panel are separated by the line $h_0 = \frac{1}{2-s}$ (see Appendix 3 for derivation) and areas C and D are separated by the line $h_{\text{int}} = \frac{2-\sqrt{4-4s-s^2}}{2s}$ (see Appendix 4 for derivation). The dashed lines at $s = 0.3$ and $s = 0.7$ represent the ecological selection regimes investigated in Figure 5. Note that the figure would look different for mating strategies other than random mating (because the genotype distribution is different).

and $\tau_{aA,aa} = \tau_{aA,aA} = \tau_{aA,AA} = 0$ and females aA mate always at random (see Figure 4, panel D). Because for heterozygotes all time-thresholds are 0, we will reserve the notation τ_i for homozygote females such that τ_1 is the time-threshold for males of their own type ($\tau_1 = \tau_{aa,aa} = \tau_{AA,AA}$), τ_2 is the time-threshold for heterozygote males ($\tau_2 = \tau_{aa,aA} = \tau_{AA,aA}$) and τ_3 is the time-threshold for the opposite homozygote males ($\tau_3 = \tau_{aa,AA} = \tau_{AA,aa}$). Furthermore, because we are interested in ecological scenarios that favor assortative mating (and therefore reproductive isolation), we assume $\varepsilon_{aa,aa} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$ and $\varepsilon_{AA,AA} \geq \varepsilon_{AA,aA} \geq \varepsilon_{AA,aa}$, that is, we assume that homozygote males of the same type as the female provide the highest benefits. Notice that this doesn't necessarily mean that females have an elevated preference only for homozygotes of their own type (Figure 4). If, for example, the length of the mating season is short or the benefits provided by males are similar enough, females may mate randomly (panel 4A) or only the opposite homozygotes might be disfavored (panel 4B). The conditions $\varepsilon_{aa,aa} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$ and $\varepsilon_{AA,AA} \geq \varepsilon_{AA,aA} \geq \varepsilon_{AA,aa}$ hence only exclude the case of disassortative mating, where the production of heterozygotes is favored. Therefore, we consider the following mating strategies for homozygote females: random mating RM ($\tau_1 = \tau_2 = \tau_3 = 0$, panel 4A), partial assortative mating PAM ($\tau_1 = \tau_2 = 0$ and $\tau_3 > 0$, panel 4B) and assortative mating AM ($\tau_1 = 0$ and $\tau_2, \tau_3 > 0$, panel 4C).

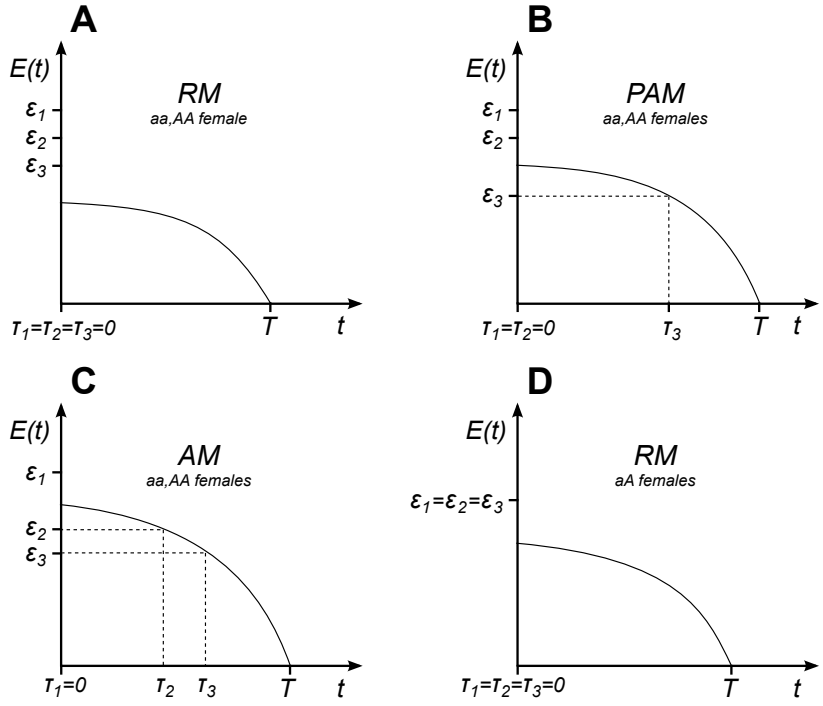

Figure 4: All possible mating strategies for females aa, aA and AA given $\hat{P}_{aa} = \hat{P}_{AA}$, and $\varepsilon_{aa,aa} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$, $\varepsilon_{AA,AA} \geq \varepsilon_{AA,aA} \geq \varepsilon_{AA,aa}$ and $\varepsilon_{aA,aa} = \varepsilon_{aA,aA} = \varepsilon_{aA,AA}$ (see the main text). A-C: Symmetric mating strategies RM, PAM and AM for homozygote females aa and AA, where $\tau_1 = \tau_{aa,aa} = \tau_{AA,AA}$, $\tau_2 = \tau_{aa,aA} = \tau_{AA,aA}$ and $\tau_3 = \tau_{aa,AA} = \tau_{AA,aa}$ are based on benefits $\varepsilon_1 = \varepsilon_{aa,aa} = \varepsilon_{AA,AA}$, $\varepsilon_2 = \varepsilon_{aa,aA} = \varepsilon_{AA,aA}$ and $\varepsilon_3 = \varepsilon_{aa,AA} = \varepsilon_{AA,aa}$ at the symmetric interior equilibrium ($\hat{P}_{aa} = \hat{P}_{AA}$). D: Heterozygote females have equal benefits from all matings ($\varepsilon_1 = \varepsilon_2 = \varepsilon_3 = \varepsilon_{aA,aa} = \varepsilon_{aA,aA} = \varepsilon_{aA,AA}$) at any symmetric equilibrium ($\hat{P}_{aa} = \hat{P}_{AA}$) and therefore $\tau_1 = \tau_2 = \tau_3 = \tau_{aA,aa} = \tau_{aA,aA} = \tau_{aA,AA} = 0$ such that they always have RM strategy. RM is the random mating strategy where no males are discriminated, PAM is the partially assortative mating strategy where homozygote females discriminate the opposite homozygote males and AM is the assortative mating strategy where homozygote females discriminate the opposite homozygote as well as heterozygote males (see also the main text).

The optimal time thresholds τ and equilibria $\hat{P}$ we find for given parameter values $(s, h, \rho T)$ such that we solve three different sets of equations (9), where $Q_{g,h}$ are given by (1) and the time-thresholds in (1) are given by (4). In

the first set of equations we assume that both homozygote females mate randomly (RM; thus we set $n = 3$ in (4)); in the second set of equations we assume that homozygote females mate partially assortatively (PAM; $n = 2$); and in the third set they mate assortatively (AM; $n = 1$). In each set of equations heterozygote females are assumed to mate randomly (see above). As we attempt to solve these three sets of equations for the same parameter values ($s, h, \rho T$), we may find solutions to more than one of the sets of equations; in this case, multiple optimal mating strategies exist simultaneously. Moreover, more than one solution may exist within one mating strategy (multiple optimal time-thresholds and corresponding symmetric equilibria). For each solution, we investigate the stability of the equilibrium by keeping the optimal time-thresholds fixed and calculating the dominant eigenvalue of the system (9) coupled with (1) where we have inserted the fixed τ's. This corresponds to the population genetic stability of the equilibrium given that the population follows the optimal mating strategy. Notice that even if the symmetric equilibrium is stable, there may exist also asymmetric attractors of population genetics for the given mate choice strategy. As said above, we restrict our attention to symmetric equilibria; nevertheless we note that if a perturbation makes the population settle on an asymmetric attractor of genotype frequencies, the symmetric mate choice strategy will no longer be optimal. Numerical computations are done using Maple version 13.0.

The strength of reproductive isolation we measure for each mating strategy by calculating the number of heterozygotes at birth and comparing it to the number of heterozygotes under random mating. The statistic $F = 1 - \frac{\text{\# of heterozygotes}}{\text{expected \# of heterozygotes under random mating}} = 1 - \frac{P_{aA}}{2P_a P_A} = 1 - 2P_{aA}$, evaluated at the equilibrium, measures the amount of "missing" heterozygotes in the population. We consider the homozygotes be reproductively isolated when $F > 0.99$, which corresponds to $\hat{P}_{aA} < 0.005$.

Figure 5 summarizes the results for a range of parameter values h and ρT and for two different values of s. We find various combinations of the mating strategies RM, PAM and AM. Mating strategies where the polymorphism is protected we indicate with subscript *PP*, and with subscript *UP* we denote unprotected polymorphism. Where there are two solutions within the same mating strategy (two equilibria and optimal time-thresholds), we denote with superscripts $-$ and $+$ the solution that has lower/higher homozygote frequency, respectively. When the symmetric interior equilibrium is unstable, hence the polymorphism is biologically unrealistic, we denote this region with NSS (no stable strategies) and do not analyze this region further. We also discard from further analysis the case where only the heterozygotes provide the highest benefits, hence contradicting the assumptions made above ($\varepsilon_{aa,aa} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$ and $\varepsilon_{AA,AA} \geq \varepsilon_{AA,aA} \geq \varepsilon_{AA,aa}$), and we denote this area with $NAMS$ (no assortative mating strategies).

Let us first make some general observations. Firstly, we notice that different mating strategies may indeed exist for the same parameter values. For example, in panel 5B for higher values of ρT various combinations of AM and PAM strategies can coexist. Secondly, if heterozygotes are at an advantage during ecological selection ($h < 1/2$), then there is either AM or no assortative mating at all ($NAMS$ in Figure 5). The reason is that with RM or PAM, heterozygote males are not discriminated against and hence suffer no disadvantage either in ecological selection or in sexual selection. With AM strategies, however, heterozygote males are at a disadvantage, so that when sexual selection is sufficiently strong, it can override ecological selection and lead to an overall disadvantage of heterozygotes, thus making AM an optimal mating strategy.

The condition for RM strategy can be calculated analytically by applying inequality (5) to the Levene-Hoekstra model and using the fact that the equilibrium frequency distribution under random mating is $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$. Given that the interior symmetric equilibrium is stable when $h < h_{\text{int}}$ (see above and Figure 3), we obtain, after some algebra, that mating at random is an optimal strategy for homozygote females if

$$\rho T \leq \ln\Big[\frac{4 - s(2h+1)}{s(2h-1)}\Big], \quad \text{for} \quad \frac{1}{2} < h < h_{\text{int}}. \tag{10}$$

The RM solution is optimal, as expected, when most females sample just a few males (Figure 5), because by refusing a male females run too high a risk to remain unmated, which would lead to receiving no benefits. However, as h approaches $1/2$, surviving the viability selection phase becomes equally likely for all genotypes, so that the RM solution exists for all ρT (although it becomes too narrow to be seen in Figure 5).

Whether the symmetric equilibrium corresponds to a protected polymorphism depends on whether the fixation equilibria are unstable. Since heterozygote males are not being discriminated by homozygote females with either RM or PAM strategies, the stability of the fixation equilibria (which depends on the relative advantage of heterozygotes

over homozygotes) is given by the same condition for PAM as for RM. In Appendix 3, we show that this holds for general time-thresholds and for arbitrary ecological selection. The fixation equilibria are stable if $w_{aA} < 1$, which for the Levene-Hoekstra model corresponds to $h > h_0 = \frac{1}{1-s}$. If there exists a stable interior equilibrium, h_0 thus separates the area of protected and unprotected polymorphism under RM and PAM (see Figure 5). In Appendix 3, we also derive the general condition for the stability of the fixation equilibria when an AM strategy is used,

$$\left[1 - \frac{1}{2} \cdot \frac{1 - e^{-\rho t_2}}{1 - e^{-\rho T}}\right] w_{aA} < 1, \tag{11}$$

where, as mentioned above, $w_{aA} < 1$ is the condition for stability under RM and PAM. $t_2 > 0$, which defines the AM strategy, has a stabilizing effect because the expression in brackets is then strictly less than 1. By stabilizing the fixation equilibria, the AM strategy therefore facilitates unprotected polymorhisms.

Let us now look closer at panel 5A, where selection against homozygotes that are in the "wrong" habitat is relatively weak ($s = 0.3$). Stable polymorphic RM and PAM solutions, which are separated by the line given in (10), exist only close to $h = 1/2$, where heterozygotes are only at a small disadvantage in ecological selection. A greater disadvantage (an increase in h) destabilizes the symmetric interior equilibrium for RM and PAM (area NSS; note

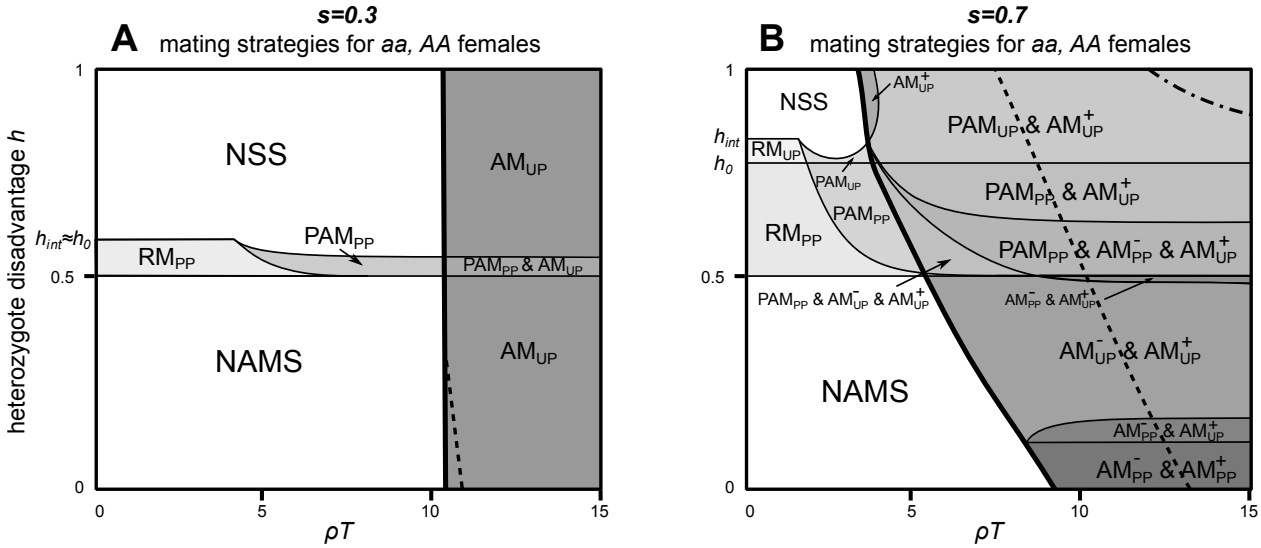

Figure 5: Optimal mating strategies for homozygote females aa and AA for (A) $s = 0.3$ and (B) $s = 0.7$. The horizontal axis shows the expected number of males ρT females are expected to encounter during a mating season and the vertical axis shows the heterozygote disadvantage h during ecological selection (see also Figure 3). The RM, PAM and AM mating strategies are explained in detail in the main text and in Figure 4. Subscripts PP and UP denote whether the polymorphism is protected or unprotected, respectively. The superscripts $-/+$ denote two different equilibria and time-threshold solutions in the same mating strategy, where $-/+$ indicates the equilibrium with smaller/higher homozygote frequencies, respectively. For RM and PAM mating strategies the fixation equilibria change stability at $h_0 = \frac{1}{2-s}$ and for RM the interior symmetric equilibria at $h_{\text{int}} = \frac{2-\sqrt{4-4s-s^2}}{2s}$. The line between RM and PAM mating strategies is given in (10). The line where for AM solutions the fixation equilibria change stability is given in (11). $NAMS$ denotes the area where the assumptions $\varepsilon_{AA,AA} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$ and $\varepsilon_{aa,aa} \geq \varepsilon_{aa,aA} \geq \varepsilon_{aa,AA}$ are violated, and NSS indicates where the interior symmetric equilibrium is unstable and hence biologically irrelevant. The thick continuous line separates the area where AM mating strategies are possible. For the AM/AM^+ mating strategy the heterozygote deficiency F is greater than 0.99 on the right hand side of the dashed line, and for the PAM mating strategy on the right hand side of the dash-dotted line. The shades of gray go from light to dark as the mating strategies that (co-)occur in the different regions increase in complexity (e.g. regions where PAM appears are lighter than where only AM appears) and if in two different regions the same mating strategies appear the one with protected polymorphisms is darker than with unprotected polymorphisms.

that there is a parameter region below NSS and above the horizontal line given by h_0 where RM and PAM solutions are unprotected, but since it is a very narrow strip, i.e. $h_{\text{int}} \approx h_0$, it is not drawn). A greater disadvantage of heterozygotes in ecological selection doesn't affect the polymorphic and stable AM solution. In fact, the value of h seems to have little impact on the existence of an AM strategy in panel 5A (the line in panel 5A which separates the AM strategy is almost vertical), which indicates that AM is maintained mostly by sexual selection against heterozygote males. Condition (11) is always satisfied and hence the polymorphism is unprotected. Moreover, there exists a narrows strip above $h = 1/2$ and for $\rho T \gtrsim 10.5$ where PAM and AM strategies coexist.

Under PAM strategies, the homozygote frequencies increase only little from $\hat{P}_{aa} = \hat{P}_{AA} = 1/4$ because PAM exists close to $h = 1/2$, i.e., where heterozygotes are at only a small disadvantage in ecological selection. Under AM strategies, the frequency of homozygotes approach $1/2$ as ρT increases. On the right side of the dashed line, $F > 0.99$, such that there is nearly perfect reproductive isolation between the two homozygote genotypes.

In panel 5B, ecological selection is stronger ($s = 0.7$). This implies that rare heterozygotes are at a greater advantage near the fixation equilibria for $h > 1/2$, thus protected polymorphism (and hence polymorphism in general) occurs easier. At the symmetric interior equilibrium, however, heterozygotes are at a greater disadvantage during ecological selection so that the PAM and AM strategies appear for smaller values of ρT.

In panel 5B, there exist two AM solutions, $AM^-$with smaller and AM^+ with greater homozygote frequencies. For the AM^+ strategy, increasing ρT increases the optimal thresholds τ_2 and τ_3 so that homozygote females accept only the same homozygote males for increasingly long periods of time. Consequently, the frequency of homozygotes approaches $1/2$ (complete reproductive isolation). For the AM^- strategy, increasing ρT decreases the thresholds τ_2 and τ_3. The thresholds either converge to some small positive values, or τ_2 becomes zero so that the AM^- strategy coincides with PAM (τ_2 becomes zero across the line that separates the regions PAM_{PP} & AM^-_{PP} & AM^+_{UP} and PAM_{PP} & AM^+_{UP}). Under the AM^- strategy, the frequencies of homozygotes do not get close to $1/2$ (depending on h, $\hat{P}_{AA} = \hat{P}_{aa}$ converges to values between ca 0.3 (when τ_2 becomes zero) and ca 0.43 (for $h = 0$) as ρT increases).

When homozygote females adopt the AM/AM^+ strategy, reproductive isolation occurs ($F > 0.99$) on the right side of the dashed line. For $h > 1/2$ reproductive isolation appears for smaller values of ρT when s is greater, as the heterozygotes are at a greater disadvantage during ecological selection. For $h < 1/2$, however, the heterozygotes are at a greater advantage when s is greater and therefore reproductive isolation appears only for higher values of ρT (compare the panels 5A and 5B). For all s, the dashed lines intersect $h = 1/2$ at the same value of ρT, because all genotypes have the same ecological fitness at $h = 1/2$ and hence the equilibrium depends only on ρT. In all cases, reproductive isolation appears at smaller values of ρT as h increases.

Interestingly, at high values of h and s the heterozygotes are at a sufficiently strong disadvantage during ecological selection so that even a PAM strategy can sustain reproductive isolation (this happens on the right side of the dash-dotted line). This is possible because even if heterozygote males are not being disfavored during sexual selection, homozygote females limit the production of heterozygotes by discriminating the opposite homozygote males, and this reinforces the effect of ecological selection.

Discussion

In this article, we introduced a mating model that incorporates several aspects of mate choice that have received little attention in the recent population genetics literature. In addition, we applied the mating model to a specific ecological scenario and found evolutionary stable mating strategies while keeping the main focus on conditions that lead to reproductive isolation between two homozygote groups.

In the highly influential model of Gavrilets and Boake (1998) (used e.g. by Kirkpatrick and Nuismer 2004, Schneider 2005, Schneider and Bürger 2006, Pennings et al. 2008, Peischl and Schneider 2009, Ripa 2009, Kisdi and Priklopil 2011), females are assumed to have fixed choosiness during the entire mating season (but see Kopp and Hermisson 2008). It would however seem more realistic that females become less choosy towards the end of the mating season so as to avoid remaining unmated. Indeed, this has been demonstrated in several natural systems (Backwell and Passmore 1996, Thomas et al. 1998, Gray 1999, Kodric-Brown and Nicoletto 2001, Moore and Moore 2001). For example, female cockroaches that approach the end of their reproductive age require considerably less courtship than their younger conspecifics (Moore and Moore 2001), or, in fiddler crabs, when the temporal constraints increase the costs of sampling towards the end of the sampling period, females become less selective and sample only smaller

males as opposed to the beginning of the sampling period were mainly larger males in the population are sampled (Backwell and Passmore 1996). To incorporate relaxed choosiness towards the end of the mating season, we modeled mate choice with a time-threshold strategy, where a male that is encountered before a time-threshold connected to his genotype will be rejected and if encountered after he will be accepted. By adjusting the time-thresholds, we can obtain any level of female choosiness. For example, if females mate randomly, thus not being choosy at all, we set all the time-thresholds to zero. It turns out that when mate search is sequential, a particular solution of the time-threshold strategy maximizes the benefit the female gains (see below).

Another difficulty with the model of Gavrilets and Boake (1998) is that it assumes a fixed value for the maximum number of encounters with males. In reality, the number of males a given female encounters depends on population density, which can change during the course of evolution, and is also affected by demographic stochasticity inherent in the search process. In our model the sampling is described with a Poisson process such that the rate of encounters is density dependent. In addition, the rate of encounters may depend also on female activity. For example, if mate sampling is costly due to time and energy expenditures (Thornhill 1984, Slagsvold et al. 1988, Alatalo et al. 1988, Milinski and Bakker 1992) or due to increased risk of predation (Sakaluk and Belwood 1984, Forsgren 1992), a searcher that is better adapted to the environment might bear the costs better and sample more mates than weaker individuals (Jennions and Petrie 1997, Cotton et al. 2006).

A third point we address is the assumption of self-referent assortative mating, where females prefer males that are similar to themselves with respect to an ecological trait (Doebeli 1996, Matessi et al. 2001). This assumption is often made to study the evolution of assortative mating under disruptive ecological selection. However, self-referent assortative mating allows maladapted females to prefer maladapted males. Instead, we assume that female choice is based on the benefits males offer (Andersson and Simmons 2006), where benefits might also depend on the female's genotype. The level of preference has evolved according to the quality of these benefits. When males contribute only their genes to the offspring, the benefit is given by the reproductive value of the offspring. Of course, in different situations other benefits may apply and can be used in our framework (Møller and Jennions 2001, Andersson 2006, Andersson and Simmons 2006).

We have shown that in order to maximize the benefits they gain, sequentially searching females should adopt a time-threshold strategy. The optimal time-thresholds are easy to determine if the benefits are fixed (see equation (4)). In many cases, however, the benefits depend on the strategy used by the resident population. In such cases, we find the strategy that is optimal in a population where all other individuals also follow this strategy; this solution represents an evolutionarily stable strategy (Maynard Smith 1982). As Figure 5 illustrates, multiple evolutionarily stable strategies may exist for the same values of the model parameters.

Using the expression for optimal time-thresholds, we have shown that if the expected number of males encountered by a female is less than a certain value, the optimal mating strategy is to mate at random. The opposite condition then implies a selection pressure for females to evolve a mating preference. For the case of two alleles in a single locus, we also give results on how various mating strategies affect the existence of protected polymorphisms. In comparison with the case of random mating, the stability of fixation equilibria (which defines whether polymorphism is protected or unprotected) are affected only if homozygote females discriminate males of all genotypes except their own.

We applied the mating model to a specific ecological scenario with the main goal to establish the conditions for reproductive isolation by assortative mating. Because ecological selection was taken to be symmetric, we focused on symmetric population genetic equilibria and on mating strategies that are symmetric for homozygote females (for heterozygote females, the optimal strategy in this model is to mate at random). Under disruptive ecological selection ($h > 1/2$), reproductive isolation can be achieved if females encounter on average about 10 males during the mating season (Figure 5, dashed line), provided that polymorphism is maintained. This number decreases with increasing ecological selection against heterozygotes (in Figure 5B, it is about 8 when $h = 1$); and with very strong ecological selection ($s = 0.95$ and $h = 1$), the necessary number of encounters is as low as 4. The necessary number of encounters we find is thus roughly in agreement with the results of Kopp and Hermisson (2008), who assumed a fixed number of encounters and self-referent assortative mating. There are, however, two factors that complicate matters. Firstly, genetic polymorphism may be lost due to the positively frequency-dependent sexual selection that is induced by assortative mating (see also Matessi et al. 2001). If ecological selection is weak, then the polymorphism is stable only if a large number of males is encountered; for $s = 0.05$, this raises the necessary number of encounters to about

20. Secondly, the model can have alternative evolutionarily stable strategies without strong reproductive isolation at the same parameter values where reproductive isolation exists. This happens when ecological selection is strong (Figure 5B) so that, surprisingly, weaker selection can in some cases be more conducive to reproductive isolation because the alternative evolutionarily stable strategies are then absent.

There are two other surprising results about the formation of reproductive isolation. Firstly, homozygote females don't need to discriminate against heterozygote males in order to be reproductively isolated. This is the case when disruptive ecological selection is strong, so that discriminating against the opposite homozygote males, thus limiting the production of heterozygote offspring, is sufficient to reinforce the effect of ecological selection against heterozygotes. Secondly, it turns out that assortative mating is a feasible mating strategy, and can even cause reproductive isolation, even if heterozygotes are at an advantage during ecological selection. This is because the benefits for females are the combination of fitness in ecological and sexual selection. Even if homozygotes do on average worse during ecological selection, assortative mating that favors homozygote males counteracts this during sexual selection and can cause reproductive isolation. With strongly stabilizing ecological selection, however, reproductive isolation is possible only if females encounter many males (for $s = 0.95$ and $h = 0$, the necessary number is about 18).

Finally, we wish to highlight some caveats concerning our mating model assumptions. The most important is that females are assumed to have all information about the size and genotypic composition of the population, and act on this information differentially depending on their own genotype. However, time-thresholds might well have evolved as a response to the selective environment of the resident population and hence be genetically determined. Alternatively, females might have acquired at least a rough estimate of some demographic parameters during the phase of ecological selection. As mentioned above, in many species females relax their choosiness toward the end of the mating season. This might hint that they are aware of the time-constraints imposed on them, or it might suggest that females have an estimate (at least a rough one) of the expected number of males to sample throughout the season. Furthermore, many species with filial or sexual imprinting recognize different individuals or traits (Schimmel and Wasserman 1991, Temeles 1994, ten Cate et al. 1993, Kendrick at al. 1998, Irwin and Price 1999, Verzijden and ten Cate 2007). The assessment of the current genotypic distribution might thus follow.

In this paper, we don't consider how the time-thresholds have evolved; we find which mating strategies are evolutionary stable under the various ecological scenarios the population experiences, but we do not investigate the evolutionary dynamics of the time-thresholds (e.g. convergence stability). Indeed, whether females have evolved to be perfect still remains to be seen.

Appendix 1

DERIVATION OF EQUATION (3)

Let $\tilde{\rho}$ be the probability that a female encounters a male in the time interval $[t, t + dt]$. We can write the expected benefit at time t as

$$E(t) = \tilde{\rho} \left[\sum_{\varepsilon_i < E(t+dt)} \tilde{P}_i E(t + dt) + \sum_{\varepsilon_i > E(t+dt)} \tilde{P}_i \varepsilon_i \right] + [1 - \tilde{\rho}]\, E(t + dt), \tag{12}$$

which can be rewritten as

$$\frac{1}{dt} [E(t + dt) - E(t)] = \frac{\tilde{\rho}}{dt} \left[\sum_{\varepsilon_i > E(t+dt)} \tilde{P}_i E(t + dt) - \sum_{\varepsilon_i > E(t+dt)} \tilde{P}_i \varepsilon_i \right], \tag{13}$$

where the summations $\sum_{\varepsilon_i < E(t+dt)}$ and $\sum_{\varepsilon_i > E(t+dt)}$ mean that we sum over all $i = 1, \ldots, k$ for which ε_i is smaller or greater, respectively, than the expected benefit E at time $t + dt$. As $dt \to 0$ we get

$$\dot{E}(t) = \rho \left[\sum_{\varepsilon_i > E(t)} \tilde{P}_i E(t) - \sum_{\varepsilon_i > E(t)} \tilde{P}_i \varepsilon_i \right] \tag{14}$$

$$= \rho \sum_{\varepsilon_i > E(t)} \tilde{P}_i \left[E(t) - \varepsilon_i \right] \tag{15}$$

$$= \rho \left[E(t) - \sum_{i=1}^{k} \tilde{P}_i \max\{E(t), \varepsilon_i\} \right]. \tag{16}$$

Appendix 2

REPRODUCTIVE VALUES AS BENEFITS

Let $N_{fg}(t)$ denote the number of couples where the female and the male have genotypes f and g, respectively and let $\mathbf{N} = [N_{11}, N_{12}, \ldots, N_{1k}, N_{21}, \ldots]^T$. Recall that a female mates only once and is thus part of only one couple, but a male can mate several times and can thus be part of several couples. The population dynamics are given by the matrix model $\mathbf{N}(t+1) = \mathbf{A}(t)\mathbf{N}(t)$, where $\mathbf{A}(t)$ is a $k \times k$ block matrix of $k \times k$ blocks with elements

$$A_{rh,fg} = \tfrac{1}{2} \left[\tfrac{1}{2} K R_{f,g\to r} v_r Q_{r,h} \tilde{P}_h + \tfrac{1}{2} K R_{f,g\to h} v_h Q_{r,h} \tilde{P}_r \right]. \tag{17}$$

The two terms of this expression correspond to the daughters and the sons of the couple (f, g), respectively, who become half of a couple (r, h) a generation later. At equilibrium, the elements of $\mathbf{A}$ are constants and the dominant eigenvalue of $\mathbf{A}$ is 1. The (fg)'th element of the dominant left eigenvector of $\mathbf{A}$,

$$\begin{aligned} \varepsilon_{fg} &= \sum_r \sum_h \varepsilon_{rh} A_{rh,fg} = \\ &= \frac{K}{4} \left[\sum_r \sum_h R_{f,g\to r} v_r Q_{r,h} \tilde{P}_h \varepsilon_{rh} + \sum_r \sum_h R_{f,g\to h} v_h Q_{r,h} \tilde{P}_r \varepsilon_{rh} \right] = \\ &= \frac{K}{4} \sum_r \sum_h R_{f,g\to r} v_r \tilde{P}_h \left(Q_{r,h} \varepsilon_{rh} + Q_{h,r} \varepsilon_{hr} \right) \end{aligned} \tag{18}$$

is the benefit that a female f receives from being part of a couple (f, g) (cf. equation (6) in the main text). The elements of the left eigenvector are the reproductive values of the corresponding couples. The optimal strategy of a female maximizes her reproductive value by choosing to be part of a couple with the highest reproductive value (cf. McNamara 1991).

Appendix 3

STABILITY OF THE FIXATION EQUILIBRIA

Here we calculate the stability of the fixation equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (1, 0, 0)$ for RM, PAM and AM strategies. The calculations for the fixation equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (0, 0, 1)$ are analogous. Note that these results hold for arbitrary ecological models and also for time-thresholds that need not be optimal. When applied to a Levene model and assumed symmetric selection (see main text) the stability conditions for both fixation equilibria are exactly the same.

The fixation equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (1, 0, 0)$ is unstable when

$$W_a(A) = \frac{1}{2} \left(\frac{Q_{aa,aA} + Q_{aA,aa}}{Q_{aa,aa}} \right) w_{aA} > 1, \tag{19}$$

where Q's are evaluated at the fixation equilibrium (Kisdi and Priklopil 2011). This is because a rare allele A is almost only present in heterozygotes aA, so it may invade (i.e. the corresponding fixation equilibrium is unstable) if the fitness of heterozygotes $v_{aA}(\frac{1}{2}Q_{aa,aA} + \frac{1}{2}Q_{aA,aa})K$ is greater than the fitness of homozygotes $v_{aa}Q_{aa,aa}K$, that is, when $W_a(A) > 1$. Here, $w_g = \frac{v_g}{\bar{v}}$ is given in (8), where $\bar{v} = v_{aa}$ is the average viability in the population. To obtain

the stability of the fixation equilibria we thus need to calculate the Q's defined in (1) and use condition (19).

RM: When the population mates randomly, $Q_{aa,aA} = Q_{aA,aa} = Q_{aa,aa} = 1 - e^{-\rho T}$, and condition (19) is reduced to

$$W_a(A) = w_{aA} > 1. \tag{20}$$

Using (8), condition (20) can be written as

$$h > \frac{1}{2-s} = h_0. \tag{21}$$

PAM: Under PAM, $t_1 = t_2 = 0$ and $t_3 > 0$, so that from (1) we get

$$Q_{aA,aa} = 1 - e^{-\rho T} \tag{22}$$

$$Q_{aa,aA} = \frac{1}{\tilde{P}_{aa} + \tilde{P}_{aA}}(1 - e^{-\rho(\tilde{P}_{aa}+\tilde{P}_{aA})(t_3-t_2)}) + (1 - e^{-\rho(t_4-t_3)})e^{-\rho(\tilde{P}_{aa}+\tilde{P}_{aA})(t_3-t_2)} \tag{23}$$

$$Q_{aa,AA} = Q_{aa,aA}, \tag{24}$$

which reduces to $Q_{aa,aA} = Q_{aA,aa} = Q_{aa,aa} = 1 - e^{-\rho T}$ when evaluated at $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (1, 0, 0)$. Stability is thus determined as in the RM case, see (21).

AM: In AM, $t_1 = 0$ and $t_2, t_3 > 0$, so that evaluating the Q's in (1) at $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (1, 0, 0)$, we get

$$Q_{aA,aa} = 1 - e^{-\rho T} \tag{25}$$

$$Q_{aa,aA} = e^{-\rho t_2} - e^{-\rho T} \tag{26}$$

$$Q_{aa,AA} = 1 - e^{-\rho T}, \tag{27}$$

which gives us the condition

$$W_a(A) = \left[1 - \frac{1}{2} \cdot \frac{1 - e^{-\rho t_2}}{1 - e^{-\rho T}}\right] w_{aA} > 1. \tag{28}$$

For $t_2 > 0$ we have $w_{aA} > W_a(A)$ and hence instability of the fixation equilibria occurs easier for RM and PAM than for AM strategies. Note that if the time-thresholds are optimal ($t_2 = \tau_2$), condition (28) depends also on the frequency distribution at the interior symmetric equilibrium via τ_2 (see (4)).

Appendix 4

DERIVATION OF h_{int}

In the main text we defined h_{int} to be the value of h where the interior symmetric equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$ changes its stability under random mating. The expression for h_{int} we obtain by calculating the dominant eigenvalue of the Jacobian of (9) evaluated at $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$. After a little algebra, we get that the dominant eigenvalue is greater than 1 when

$$h > h_{\text{int}} = \frac{2 - \sqrt{4 - 4s - s^2}}{2s}, \tag{29}$$

and hence we have that the equilibrium $(\hat{P}_{aa}, \hat{P}_{aA}, \hat{P}_{AA}) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$ is unstable when $h > h_{\text{int}}$ and stable when $h < h_{\text{int}}$.

References

[1] Alatalo, R.V., A. Carlson, and A. Lundberg. 1988. The search cost in mate choice of the Pied Flycatcher. Anim. Behav. 36: 289–291.

[2] Andersson, M. 2006. Condition-dependent indicators in sexual selection: development of theory and tests. In Essays in Animal Behaviour: Celebrating 50 Years of Animal Behaviour. Elsevier 253–267.

[3] Andersson, M., and L.W. Simmons. 2006. Sexual selection and mate choice. Trends Ecol. Evol. 21: 296–302.

[4] Backwell, P. R. Y., and N.I. Passmore. 1996. Time constraints and multiple choice criteria in the sampling behaviour and mate choice of the fiddler crab, *Uca annulipes*. Behav. Ecol. Soc. 38: 407–416.

[5] Bakker, T. C. M., and M. Milinski. 1991. Sequential female choice and the previous male effect in sticklebacks. Behav. Ecol. and Soc. 29: 205–210.

[6] Chow, Y.S., H. Robbins, and D. Siegmund. 1971. Great expectations: The theory of optimal stopping. Houghton Mifflin, Boston.

[7] Cotton, S., J. Small, and A. Pomiankowski. 2006. Sexual selection and condition-dependent mate preferences. Cur. Biol. 16: 755–765.

[8] Doebeli M. 1996. A quantitative genetic competition model for sympatric speciation. J. Evol. Biol. 9: 893–909.

[9] Forsgren, E. 1992. Predation risk affects mate choice in a gobiid fish. Am. Nat. 140: 1041–1049.

[10] Forsgren, E. 1997. Mate samling in a population of sand gobies. Anim. Behav. 53: 267–276.

[11] Gavrilets, S., and C. R. B. Boake. 1998. On the evolution of premating isolation after a founder event. Am. Nat. 152: 706–716.

[12] Gavrilets, S. 2004. Fitness landscapes and the origin of species. Princeton University Press, Princeton.

[13] Grafen, A. 1990. Biological signals as handicaps. J. Theor. Biol. 144: 517–546.

[14] Gray, D. 1999. Intrinsic factors affecting female choice in house crickets: time of first reproduction, female age, nutritional condition, body size, and size-relative reproductive investment. J. Insect Behav. 12: 691–700.

[15] Hoekstra, R. F., R. Bijlsma, and A. J. Dolman. 1985. Polymorphism from environmental heterogeneity: models are only robust if the heterozygote is close in fitness to the favoured homozygote in each environment. Genet. Res. Camb. 45: 299–314.

[16] Houde, A. E. 1997. Sex, colour, and mate choice in guppies. Princeton University Press.

[17] Irwin, D. E., and T. Price. 1999. Sexual imprinting, learning and speciation. Heredity 82: 347–354.

[18] Ivy, M.T., and S.K. Sakaluk. 2007. Sequential mate choice in decorated crickets: females use a fixed internal threshold in pre- and postcopulatory choice. Anim. Bebav. 74: 1065–1072.

[19] Iwasa, Y., Pomiankowski, A., 1994. The evolution of mate preferences for multiple sexual ornaments. Evolution 48, 853-867.

[20] Iwasa, Y., Pomiankowski, A., Nee, S., 1991. The evolution of costly mate preferences. II. The ”handicap” principle. Evolution 45, 1431-1442.

[21] Janetos, A. C. 1980. Strategies of female mate choice: a theoretical analysis. Behav. Ecol. Sociobiol. 7: 107–112.

[22] Jennions, M.D., and M. Petrie. 1997. Variation in mate choice and mating preferences: a review of causes and consequences. Biol. Rev. 72: 283-327.

[23] Kendrick, K. M., M.R. Hinton, K. Atkins, M. A. Haupt, and J. D. Skinner. 1998. Mothers determine sexual preferences. Nature 395: 229–230.

[24] Kisdi, E., Priklopil, T., 2011. Evolutionary branching of a magic trait. J. Math. Biol. 63, 361–397.

[25] Kirkpatrick, M., and S. L. Nuismer. 2004. Sexual selection can constrain sympatric speciation. Proc. R. Soc. Lond. B 271: 687–693.

[26] Kodric-Brown, A., and P.F Nicoletto. 2001. Age and experience affect female choice in the guppy (*Poecilia reticulata*)Am. Nat. 157: 316–323.

[27] Kopp, M., and J. Hermisson. 2008 Competitive speciation and costs of choosiness. J. Evol. Biol. 21: 1005–1023.

[28] Lehmann, G.U.C. 2007. Density-dependent plasticity of sequential mate choice in a bushcricket (Orthoptera: Tettigoniidae). Australian J. Zool. 55: 123-130.

[29] Levene, H. 1953. Genetic equilibrium when more than one ecological niche is available. Am. Nat. 87: 331–333.

[30] Lorch, P.D., Proulx, S., Rowe, L., Day, T., 2003. Condition-dependent sexual selection can accelerate adaptation. Evol. Ecol. Res. 5, 876–881.

[31] Matessi C., A. Gimelfarb, and S. Gavrilets. 2001 Long-term buildup of reproductive isolation promoted by disruptive selection: How far does it go? Selection 2: 41–64.

[32] Maynard Smith, J. 1982 Evolution and the theory of games. Cambridge University Press, Cambridge.

[33] McNamara ,J. M. 1991. Optimal life histories: A generalisation of the Perron-Frobenius theorem. Theor. Pop. Biol. 40: 230–245.

[34] Milinski, M., and T. C. M. Bakker. 1992. Costs influence sequential mate choice in sticklebacks, *Gasterosteus aculeatus*. Proc. R. Soc. Lond. B 250: 229–233.

[35] Møller, A.P., and R. V. Alatalo. 1999. Good-genes effects in sexual selection. Proc. R. Soc. Lond. 266: 85–91.

[36] Moore, P.J., and A.J. Moore. 2001. Reproductive aging and mating: The ticking of the biological clock in female cockroaches. Evolution 98: 9171–9176.

[37] Møller, A. P., and M.D. Jennions. 2001. How important are direct fitness benefits of sexual selection? Naturwissenschaften 88: 401–415.

[38] Peischl, S., and K. Schneider. 2009. Evolution of dominance under frequency-dependent intraspecific competition in an assortatively mating population. Evolution 64-2: 561–582.

[39] Pennings, P. S., M. Kopp, G. Meszéna, U. Dieckmann & J. Hermisson. 2008. An analytically tractable model for competitive speciation. Am. Nat. 171: E44–E71

[40] Priklopil, T. 2012. Chaotic dynamics of allele frequencies in condition-dependent mating systems. Theor. Pop. Biol. doi:10.1016/j.tpb.2012.06.001.

[41] Proulx, S.R., 2001. Female choice via indicator traits easily evolves in the face of recombination and migration. Evolution 55: 2401–2411.

[42] Prout, T.1968. Sufficient conditions for multiple niche polymorphism. Am. Nat. 102: 493–496.

[43] Real, L. A. 1990. Search theory and mate choice. I. Models of single-sex discrimination. Am. Nat. 136: 376–404.

[44] Ripa, J. 2009. When is sympatric speciation truly adaptive? An analysis of the joint evolution of resource utilization and assortative mating. Evol. Ecol. 23: 31–52.

[45] Ross, S. M. 1995. Stochastic Processes. Wiley.

[46] Sakaluk, S.K., and J.J. Belwood. 1984. Gecko phonotaxis to cricket calling song: a case of satellite predation. Anim. Behav. 32: 659–662.

[47] Schimmel, L., and F. Wasserman. 1991. An interspecific comparison of individual and species recognition in the Passerines *Turdus migratorius* and *Cyanocitta cristata*. Behaviour 118: 115–126.

[48] Schneider K. A. 2005. Competitive divergence in non-random mating populations. Theor. Pop. Biol. 68: 105–118.

[49] Schneider K. A., and R. Bürger. 2006 Does competitive divergence occur if assortative mating is costly? J. evol. Biol. 19: 570–588.

[50] Servedio M. R., G. S. van Doorn, M. Kopp, A. Frame & P. Nosil. 2011. Magic traits in speciation: 'magic' but not rare. TREE 26: 389-397.

[51] Slagsvold, T., J.T. Lifjeld, G. Stenmark, and T. Breiehagen. 1988. On the cost of searching for a mate in female pied flycatchers *Ficedula hypoleuca*. Anim. Behav. 36: 433–442.

[52] Temeles, E. J. 1994. The role of neighbours in territorial systems: When are they 'dear enemies'? Anim. Behav. 47: 339–350.

[53] ten Cate, C., D. R. Vos, and N. Mann. 1993. Sexual imprinting and song learning: two of one kind? Neth. J. Zool. 43: 34–45.

[54] Thomas, F., C. Liautard, F. Czilly, and F. Renaud. 1998. A finite time horizon influences sequential mate choice in male *Gammarus aequicauda* (Amphipoda). Canadian J. Zool. 76: 401–405.

[55] Thornhill, R. 1984. Alternative female choice tactics in the scorpion-fly *Hylobittacus apicalis* (Mecoptera) and their implications. American Zool. 24: 367–383.

[56] van Doorn, S., and F. Weissing. 2009. On the origin of species by natural and sexual selection. Science 326: 1704–1707.

[57] Verzijden, M. N., and C. ten Cate. 2007. Early learning influences species assortative mating preferences in Lake Victoria cichlid fish. Biol. Lett. 3: 134–136.

[58] Wiegmann, D. D., K. Mukhopadhyay, and L. A. Real. 1999. Sequential search and the influence of male quality on female mating decisions. J. Math. Biol. 39: 193–216.

[59] Wiegmann, D. D., and L. M. Angeloni. 2007. Mate search and uncertainty in the decision process. J. Theor. Biol. 249: 654–666.

[60] Zahavi, A. 1975. Mate selection - a selection for a handicap. J. Theor. Biol. 53: 205–214.