Optimization under frequency-dependent selection

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A B S T R A C T

We consider a model of frequency-dependent selection, which we refer to as the Wildcard Model. A variety of more specific models, representing quite diverse biological situations, are covered by the Wildcard Model as particular cases. Two very different particular models that are subsumed by the Wildcard Model are the game theoretically motivated two-phenotype model of Lessard [Lessard, S., 1984. Evolutionary dynamics in frequency-dependent two-phenotype models, Theor. Popul. Biol. 25, 210–234], and the model of selection on a continuous trait due to intraspecific competition of Bürger [Bürger, R., 2005. A multilocus analysis of intraspecific competition and stabilizing selection on a quantitative trait. J. Math. Biol. 50 (4), 355–396] and Schneider [Schneider, K.A., 2006. A multilocus-multiallelic analysis of frequency-dependent selection induced by intraspecific competition. J. Math. Biol. 52 (4), 483–523]. Both these models have been shown in the past to have a global Lyapunov function (LF) under appropriate genetic assumptions. We show that (i) the Wildcard Model in continuous time for a single multiallelic locus, or for multiple multiallelic loci in linkage equilibrium, has a global LF, of which the Lessard and Bürger–Schneider LFs are special cases in spite of their widely different biological interpretations; (ii) the LF of the Wildcard Model can be derived from an LF previously identified for a model of density- and frequency-dependent selection due to Lotka–Volterra competition, with one locus, multiple alleles, multiple species and continuous-time dynamics [Matessi, C., Jayakar, S.D., 1981. Coevolution of species in competition: A theoretical study. Proc. Natl. Acad. Sci. USA, 78 (2, part2), 1081–1084]. We extend the LF with density and frequency dependence to the multilocus case with linkage-equilibrium dynamics. As a possible application of our results, the optimization principle we established can be used as a tool in the study of long-term evolution of various models subsumed by the Wildcard Model based on explicit short-term dynamics.

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1. Introduction

Frequency-dependent selection has been invoked in the explanation of many important evolutionary phenomena. These include the evolution of behavioral traits, the maintenance of genetic variation, and disruptive selection with its possible consequences of ecological character displacement, reproductive isolation and, eventually, speciation (Maynard Smith, 1966, 1982; Bulmer, 1974, 1980; Matessi and Jayakar, 1976, 1981; Clarke, 1979; Slatkin, 1979, 1980; Felsenstein, 1981; Abrams, 1986, 1987; Asmussen and Basnayake, 1990; Dieckmann and Doebeli, 1999; Bürger et al., 2006; Schluter, 2000; Turelli et al., 2001; Gavrilets, 2003, 2004). Frequency-dependent selection occurs if the fitness of a particular phenotype depends on the frequency of its own and other phenotypes in the population. Many mechanisms causing frequency dependence are known, including intraspecific competition, systems of mimicry, host–parasite or predator–prey interactions, aposematic and cryptic coloration, or the evolution of self-incompatibility (cf. Matessi and Cori, 1972; Ayala and Campbell, 1974; Clarke and Partridge, 1988). In general, frequency dependence occurs if changes in the population composition affect the biotic or abiotic environment that induces selection on this population.

Population-genetics theory has included frequency-dependent selection since its conception (Fisher, 1930), but concrete studies remained sporadic (e.g., Wright, 1948). Pioneering work by Haldane and Jayakar (1963) established it as a powerful mechanism for the maintenance of polymorphism in natural populations. Moreover, for the first time in population genetics it showed the possibility of permanent, more or less regular oscillations, a feature that is intimately engrained in this selective mechanism (e.g., Matessi and Cori, 1972; Gavrilets and Hastings, 1995). After the discovery of the impressive and unexpected amount of polymorphism at the molecular level in natural populations (Lewontin and Hubby, 1966), and during the intense debates over the causes...
of its maintenance, frequency-dependent selection has been often indicated as a most likely source of this phenomenon (e.g., Kojima and Yarborough, 1967). Subsequently, the theory of one-locus models under frequency- (and density-) dependent selection has been the subject of a more systematic study (e.g., Clarke, 1972; Cockermham et al., 1972; Matessi and Jayakar, 1976; Matessi and Jayakar, 1981; Asmussen, 1983; Asmussen and Basnayake, 1990), whereas the theory of multilocus models is limited to the study of much less general models (e.g., Bürger, 2005; Schneider, 2006).

The evolutionary consequences of frequency-dependent selection have been investigated within the framework of evolutionary game theory (e.g., Maynard Smith, 1982; Hofbauer and Sigmund, 1998) and, more recently, also within what has been called adaptive dynamics theory (Dieckmann, 1997; Geritz et al., 1998; Doebeli and Dieckmann, 2000). Common to approaches within these frameworks is, with few exceptions, a lack of genetics, i.e., they usually assume asexual reproduction and monomorphic populations. Simple genetics has been incorporated in some ecological models, often, however, resting on a number of assumptions that are not completely specified or justified. In most cases computer simulations have been performed, but only little analytical theory has been developed. However, there are a few exceptions that either allow for more genetic details (Lessard, 1984; Matessi et al., 2001; Kopp and Hermisson, 2006; Matessi and Gimelfarb, 2006), or provide analytical results for multilocus genetics (Schneider, 2007).

A well-known and disappointing feature of frequency-dependent selection is that the mean fitness is in general not maximized, i.e., it is not a Lyapunov function, not even for selection at a single locus for which 'Fisher’s Fundamental Theorem of Natural Selection' holds for constant (frequency-independent) selection (however recent work, Schneider, 2008) establishes some conditions under which mean fitness maximization occurs). On the other hand, alternative maximization principles for frequency-dependent selection have been found in a few cases: Matessi and Jayakar (1976, 1981) were one of the first to provide a global Lyapunov function for a general one-locus multiallele, multispecies model of frequency- and density-dependent selection in continuous time. Lessard (1984) established a Lyapunov function for the discrete-time, one-locus, multiallele dynamics resulting from a two-phenotype selection model in which fitness depends on the payoffs from random pairwise contests. More recently, Bürger (2005) proved, for a particular model of selection on a continuous trait caused by intraspecific competition, that a Lyapunov function exists in continuous time when the selected trait is genetically determined by multiple additive diallelic loci in linkage equilibrium. These results have been further generalized to multiple additive alleles by Schneider (2006).

In this paper we will study a model of frequency-dependent selection that is sufficiently broad to accommodate many models of this type of selection that are present in the literature. We will refer to it as the Wildcard Model. In spite of the different biological scenarios reflected by them, the models of Lessard (1984) and of Bürger (2005) and Schneider (2006) turn out to be particular cases of the Wildcard Model. Assuming one-locus, multiallele genetics, we will show that the continuous-time dynamics of the Wildcard Model forms a generalized gradient system (i.e., a Shashahani gradient system, cf. Hofbauer and Sigmund, 1998, chapter 7)). The potential of such a gradient system is therefore a Lyapunov function for the Wildcard Model. Moreover, it subsumes, as particular cases, the Lyapunov functions already identified for the Lessard and the Bürger–Schneider models, which, from their biological interpretation, would superficially appear to be very different. We then consider the relationship between the Lyapunov function of our model and that found by Matessi and Jayakar (1981) for density- and frequency-dependent selection due to scramble competition and represented by a Lotka–Volterra interaction model. We show that the former can be derived from the latter. Finally, we consider a multilocus, multiallele model and, assuming approximate linkage equilibrium, we show that the Lyapunov function of the Wildcard Model works also in this case. This is done both for a density-independent and a density-dependent version of the model.

Our results generalize all of the above-mentioned models. In order to be able to precisely appreciate these generalizations, first it is necessary to briefly review these models and, if appropriate, also the results that have been previously established. This approach will accompany us throughout this article to guide our intuition for the various biological situations to which the Wildcard Model is applicable.

2. Wildcard Model and particular cases

2.1. The Wildcard Model

The frequency-dependent selection model that we consider assumes that the fitness of an individual of type \( x \in X \), where \( X \) is the (countable) set of all types, is given by the following function of population composition:

\[
W_x = W_x(q) = S_x + \sum_{y \in X} A_{x,y} q_y + C(q),
\]

with \( A_{x,y} = A_{y,x} \) for all \( x, y \in X \),

(1)

where \( q_y \) is the frequency of type \( y \) in the population, so that the vector \( q = (q_x)_{x \in X} \) represents the population composition; \( S_x \) is a frequency-dependent component of fitness; \( A_{x,y} \) is the effect that individuals of type \( y \) have on the fitness of individuals of type \( x \); such an effect is assumed to be the same as that of type \( x \) on type \( y \). Finally, the function \( C(q) \) is arbitrary and represents any effect on fitness that may vary with the population composition but is the same for all types.

It should be mentioned that (1) has a similar structure as the pairwise-interaction model of frequency-dependent selection that is commonly studied in the theoretical literature, e.g., Schütz et al. (1968), Allard and Adams (1969), Cockermham et al. (1972), Asmussen and Basnayake (1990), Altenberg (1991), Gavrilets and Hastings (1995), Asmussen et al. (2004), Trotter and Spencer (2008), and Schneider (2008). However, the pairwise interaction model assumes \( C = 0 \) and, in general, does not require the symmetry conditions \( A_{x,y} = A_{y,x} \).

Assuming that the different types correspond to the genotypes of a single diploid locus with \( n \) alleles, \( a_1, \ldots, a_n \), with respect to which matings occur at random, the fitness of genotype \( a_{i_1}a_{j_2} \) is given by

\[
W_{ij} = W_i(p) = S_{ij} + \sum_{k=1}^{n} A_{ij,k} p_k p_l + C(p),
\]

(2)

where \( p = (p_1, \ldots, p_n) \) denotes the vector of allele frequencies. The dynamics of the allele frequencies in discrete generations, under the selection regime (2), is then given by the following system of recursion equations:

\[
p_i' = \frac{W_i}{\bar{W}_i} \quad \text{for } i \in \{1, \ldots, n\},
\]

(3a)

where the marginal fitness of allele \( i \), \( \bar{W}_i \), and the population mean fitness, \( \bar{W} \), are
\[ W_i = W_i(p) = \sum_{j=1}^{n} W_{ij} p_j = \sum_{j=1}^{n} S_{ij} p_j + C(p), \]

\[ W = W(p) = \sum_{i=1}^{n} W_i p_i = \sum_{i=1}^{n} S_{ij} p_i + C(p), \]

(3b)

(3c)

Note that, since we assume that the effect of a gene does not depend on the parent by which it is transmitted, i.e., a lack of genomic imprinting, it follows that \( S_{ij} = S_j \) and \( A_{ij,kl} = A_{ij,kl} = A_{ij,kl}. \)

2.2. Particular cases

2.2.1. Random pairwise contests

The Lessard model

Lessard (1984) introduced a model of frequency-dependent selection resulting from random pairwise contests, to investigate the population-genetic dynamics implicitly underlying basic principles and results of 'evolutionary game theory', and in particular the notion of evolutionarily stable strategy (ESS) (see Maynard Smith and Price, 1973). This model assumes two phenotypes - or strategies such as, for example, behavioral patterns - \( C_1 \) and \( C_2 \), that determine the outcomes of interactions among pairs of individuals. The fitness payoff accruing to \( C_i \) individuals from interactions with \( C_j \) individuals is given by the matrix \( F = (F_{ij})_{ij \in \{1,2\}} \), which is not necessarily symmetric. Hence, if the population consists of several types of individuals with different probabilities of expressing \( C_1 \) or \( C_2 \), such that type \( x \) has frequency \( q_x \) and expresses \( C_1 \) with probability \( h_x \) and \( C_2 \) with probability \( 1 - h_x \), then the fitness of a type-\( x \) individual is

\[ W^{(1)}_x = hF_{11} + (1 - h)F_{12} h_x + [hF_{21} + (1 - h)F_{22}] (1 - h_x) \]

(6a)

\[ = (F_{12} - F_{22}) h_x + (F_{11} - F_{21} + F_{22} - F_{12}) h_x h_y + [hF_{21} + (1 - h)F_{22}], \]

where \( h = \sum_y h_y \) is the frequency of strategy \( C_1 \) in the population. Comparison of (1) and (6a) immediately shows that this model is a particular case of our Wildcard Model with

\[ S_x = (F_{12} - F_{22}) h_x, \]

(6b)

\[ A_{x,y} = A_{y,x} = (F_{11} - F_{21} + F_{22} - F_{12}) h_x h_y, \]

(6c)

\[ C(q) = hF_{21} + (1 - h)F_{22}. \]

(6d)

The Matessi–Gimelfarb–Gavrilets model

Like Lessard (1984), Matessi et al. (2001) and Matessi and Gimelfarb (2006) considered a model in which frequency-dependent selection results from random pairwise interactions. While Lessard (1984) assumed a discrete strategy set of two elements, \( C_1, C_2 \), the other authors were interested in the case in which the outcome of a confrontation depends on the respective value of some continuous trait of interacting individuals. If interactions with opponents of trait value \( y \) provide the fitness payoff \( f(x, y) \) to individuals of trait value \( x \), then the fitness of these individuals is \( \sum_y f(x, y) q_y \), where \( X \) denotes the countable set of trait values available in the population. In Matessi et al. (2001) and in Matessi and Gimelfarb (2006) the payoff function \( f(x, y) \) is assumed to be

\[ f(x, y) = 1 + \alpha x^2 - (\alpha + \beta) xy + \beta y^2, \quad x, y \in \mathbb{R}. \]

(7)

Note that at the trait value \( x = 0 \), there is an evolutionary singularity (see Geritz et al., 1998) since \( \lim_{x \to 0} f(x, y) = 0 \). This particular point is continuously stable, namely evolution in the phenotypic space tends to proceed in the direction of this point (see Eschel, 1983), provided the continuous stability condition

\[ \alpha < \beta \]

(8a)

holds. It is a monomorphic evolutionarily attainable stable trait (MEAST, see Christiansen, 1991), i.e., a monomorphic continuously stable ESS (monomorphic CSS, see Eschel, 1983), if the MEAST condition

\[ \alpha < \beta \quad \text{and} \quad \alpha < 0 \]

(8b)

holds. In such a case selection in the vicinity of zero is stabilizing. On the opposite, the singularity is a polymorphic evolutionarily attainable stable trait (PEAST, see Christiansen, 1991), or a branching
point (see Geritz et al., 1998), and selection in its vicinity is disruptive, if the PEAST condition
\alpha < \beta \quad \text{and} \quad \alpha > 0 \quad (8c)
holds. The fitness of an individual of trait value \( x \) is its average payoff. Assuming (7) it is given by
\[ W^{(MG)}_x = 1 + \alpha x^2 - (\alpha + \beta) x \bar{x} + \beta (x^2 + v), \quad (9a) \]
where \( \bar{x} = \sum x q_x \) and \( v = \sum (x - \bar{x})^2 q_x \) are respectively the mean and the variance of the trait values in the population. Comparison of (1) and (9a) shows that this model is a particular case of our Wildcard Model with
\[ S_x = 1 + \alpha x^2, \quad A_{x,y} = A_{y,x} = - (\alpha + \beta) xy, \quad C(q) = \beta (x^2 + v). \quad (9b) \]
We note that the payoff function (7) is more general than it superficially appears. Consider any \( C^2 \) payoff function \( v(x,y) \) such that there is an evolutionary singularity at \( x = y = 0 \), i.e., \( \frac{\partial v(x,y)}{\partial s_{ij}} |_{s_{ij}=0} = 0 \). If we define \( F(x,y) := 1 + v(x,y) - v(x,x) \), then it is easily seen that (7) is the second order Taylor approximation of \( F(x,y) \) in a neighborhood of \( x = y = 0 \).

2.2.2. Intraspecific competition. The Bürger–Schneider model
Bürger (2005) and Schneider (2006) formulated a model of density- and frequency-dependent selection, caused by competition for limited resources, where the competitive abilities of individuals are determined by the size of a continuous trait. The explicit intent of this model was that of providing a unified weak-selection approximation for many and diverse models of this kind available in the literature (e.g., Roughgarden, 1972; Slatkin, 1979; Bulmer, 1974, 1980; Christiansen and Loeschcke, 1980; Bürger, 2002a,b). The formulation is based on a rather general model presented by Bulmer (1974), which is then approximated in the limit of selection strength tending to zero. The resulting model assigns to individuals of trait value \( x \) in a population of size \( N \) a fitness given by
\[ W^{(B-S)}_x = F(N) \left[ 1 - \sigma x^2 + \sigma \eta (N)(x - \bar{x})^2 + \psi(N,q) \right], \quad (10) \]
where the trait’s mean is \( \bar{x} \). Here, \( F(N) \) can be any non-negative, monotonically decreasing, continuously differentiable function that is suitable to represent the rate of increase of a population as a function of its size, such as, for example, those employed in the Beverton–Holt or the Hassel model (e.g., Thieme, 2003, Chapter 9) of population regulation. The positive parameter \( \sigma \) is a direct measure of the strength of stabilizing selection. Furthermore, \( \eta(N) = -\frac{NF(N)}{\bar{x}(N)} \), where the positive parameter \( f \) is a measure of the strength of the frequency-dependent effect of intraspecific competition, relative to the strength, \( \sigma \), of stabilizing selection induced by the resource distribution. Finally, \( \psi(N,q) \), which is independent of \( x \), is an arbitrary positive function of the population size and of the composition of the population, \( q \).

This model has also been used in its density-independent version, derived from (10) by assuming that the population size is constant. In such a case both \( F(N) \) and \( \eta(N) \) are positive constants and \( \psi \) depends only on the population composition, \( q \), so that (10) can be rewritten as (Bürger, 2005; Schneider, 2006, 2007)
\[ W^{(B-S)}_x = 1 - \sigma x^2 + \sigma \eta (x - \bar{x})^2 + \psi(q). \quad (11a) \]
Short- and long-term evolution due to frequency-dependent selection as prescribed by the selection regime of (11a) has been studied, mostly assuming continuous time, under a one-locus or multilocus genetic model, with multiple alleles per locus, assuming additivity and, in the multilocus case, quasi-linkage equilibrium (Bürger, 2005; Schneider, 2006, 2007). By rewriting (11a) as
\[ W^{(B-S)}_x = 1 + \sigma (x - 1)x^2 - 2\sigma \eta x + \alpha + \beta x \]
we immediately recognize it as basically equivalent to the fitness function of the selection model of Matessi et al. (2001) and Matessi and Gimelfarb (2006), given by (9a), and therefore as a particular case of the Wildcard Model, (1). Table 1 summarizes the correspondences among the parameters of these two functions and also those of a fitness function derived from the model of Christiansen and Loeschcke (1980) to be discussed below (Section 3.2.2).

3. Optimization by frequency-dependent selection
3.1. The Wildcard Model
Both in the Lessard model (6) and in the Bürger–Schneider model (11) it has been found that certain quantitative population properties increase monotonically in the course of frequency-dependent selection and attain a maximum at a population equilibrium. For the Lessard model, the existence of such a quantity, namely a global Lyapunov function, has been demonstrated by assuming one locus with multiple alleles and discrete time (Lessard, 1984), whereas for the Bürger–Schneider model multiple additive loci and alleles, linkage equilibrium and a continuous-time approximation have been assumed (Bürger, 2005; Schneider, 2006). Given the equivalence of the Bürger–Schneider model and the Matessi–Gimelfarb–Gavrilets model, the Lyapunov function of the former model is of course shared by the latter.

The Lessard model and the Bürger–Schneider model are aimed to represent quite distinct biological situations. Hence, the biological meaning of their respective Lyapunov functions is also very different. The question then arises whether such optimizing properties are only due to special features, peculiar of each of these two models, or can instead be extended to every model that has the structure of our Wildcard Model, (1). We can show that indeed a global Lyapunov function exists for this model, for a one-locus, multiple-allele system, at least in the continuous-time approximation. Moreover, we show (in Section 4) that these results can be extended to multilocus, multiallele genetics, provided linkage equilibrium can be assumed to prevail.

Referring to (5), consider the function
\[ \Lambda(p) = 2s + \bar{s}, \quad (12) \]
where \( p = p(t) = (p_1(t), \ldots, p_n(t)) \). By virtue of the symmetries in the parameters \( s_{ij} \) and \( \bar{s}_{ij} \) we obtain
\[ \frac{\partial \Lambda}{\partial p_l} = 4 \sum_{j=1}^{n} s_{lj} p_j + 4 \sum_{j,k,l=1}^{n} a_{l,j,k} p_{jd} p_{j,l} \]
Hence, the time derivative of \( \Lambda \) is
\[ \dot{\Lambda}(p) = \sum_{l=1}^{n} \frac{\partial \Lambda}{\partial p_l} \]
\[ = 4 \sum_{l=1}^{n} w_l (w_l - \bar{w}) p_l = 4 \sum_{l=1}^{n} (w_l - \bar{w})^2 p_l, \quad (13) \]
which is always positive except at equilibria of (5), where it vanishes. Hence, under (5), \( \Lambda(p) \) increases in time and is maximized at the locally stable equilibria of the population. The above calculations show that (5) is even a generalized (Shashahani) gradient system (see e.g., Hofbauer and Sigmund, 1998; Bürger, 2000). Hence, by the properties of Lyapunov functions (cf. Losert and Akin, 1983) we obtain the following

**Theorem 1.** Let \( \Lambda = 2s + \bar{s} \), where \( s \) and \( \bar{s} \) are given by (5a). Then (5) is a generalized gradient system with potential \( \Lambda/4 \). In particular
A is an (increasing) global Lyapunov function for (5), and its time derivative vanishes only at the equilibrium points. Furthermore, the \(\omega\)-limit of every trajectory is contained in the set of equilibria.

We will show now that the Lyapunov function, \(\Lambda\), of the Wildcard Model includes the Lyapunov functions already identified for the Lessard and the Bürger–Schneider models as particular cases.

3.2. Lyapunov functions previously found in the particular cases

3.2.1. The Lessard model

Lessard (1984) has demonstrated that the one-locus, multiallele, discrete-time dynamics generated by his frequency-dependent selection model, (6), admits a global Lyapunov function. It is given by

\[
\Lambda^{(\text{LD})} (p) = (f_{11} - f_{21} + f_{22} - f_{12}) \left| \bar{h} - h^* \right|,
\]

where \(p = (p_1, \ldots, p_n)\) is the vector of allele frequencies, \(\bar{h}\) is the frequency of strategy \(C_1\) and

\[
h^* = \frac{f_{12} - f_{22}}{f_{21} - f_{11} + f_{12} - f_{22}}.
\]

Whenever \(f_{12} - f_{22} > 0\) and \(f_{21} - f_{11} > 0\), (16) is satisfied, then \((f_{11} - f_{21} + f_{22} - f_{12}) < 0\), which, by the (increasing) Lyapunov function (15) implies that \(\bar{h} - h^*\) decreases across generations, hence the population phenotypic composition approaches the ESS.

As we have seen, the Lessard model is a particular case of the Wildcard Model. Hence, in continuous time, it must admit the global Lyapunov function \(\Lambda\), (12), in the particular version appropriate for the parameters of this model. But in order to derive this version of \(\Lambda\) it is required that we first represent the parameters of the model in the form suitable for the continuous-time (i.e., weak-selection) approximation. In analogy to (4) we write \(F_j = f + \tau f_0\). This yields

\[
h^* = \frac{f_{12} - f_{22}}{f_{21} - f_{11} + f_{12} - f_{22}}.
\]

\[
\Lambda^{(\text{LD})} = \tau (f_{11} - f_{21} + f_{22} - f_{12}) \left| \bar{h} - h^* \right|,
\]

and also, (4) and (6b)–(6d),

\[
S_j = \tau (f_{12} - f_{22}) h_j,
\]

\[
A_{ij,kl} = \tau (f_{11} - f_{21} + f_{22} - f_{12}) h_j h_k h_l h_m.
\]

\[
C (p) = f + \tau \left[ \bar{h} f_{21} + (1 - \bar{h}) f_{22} \right] .
\]

Hence, by applying this parametrization to the general Lyapunov function \(\Lambda\) in (12) we can write it as

\[
\Lambda (p) = (f_{11} - f_{21} + f_{22} - f_{12}) \left( \bar{h}^2 - 2\bar{h}h^* \right) .
\]

Since adding a constant to a Lyapunov function obviously cannot change its nature, by adding the constant \((f_{11} - f_{21} + f_{22} - f_{12}) h^2\) to (18) we obtain the following Lyapunov function for the Lessard model

\[
\Lambda^{(\text{LD})} (p) = (f_{11} - f_{21} + f_{22} - f_{12}) \left( \bar{h} - h^* \right)^2.
\]

It has the same structure as the discrete-time Lyapunov function \(\Lambda^{(\text{LD})}\), (15), originally identified by Lessard (1984). We have therefore proved the following

**Corollary 1.** The continuous-time version of the two-phenotype model of Lessard is a particular case of the Wildcard Model (5). Hence, it forms a generalized gradient system. Moreover, since \(\Lambda^{(\text{LD})}\) given by (19) differs from \(\Lambda\) given by (12) only by a constant, \(\Lambda^{(\text{LD})}/4\) is a potential function of this system. Thus, the (increasing) Lyapunov function \(\Lambda^{(\text{LD})}\) is a special case of \(\Lambda\).

3.2.2. The Bürger–Schneider model

Bürger (2005) and Schneider (2006) have demonstrated that the continuous-time dynamics induced by their frequency-dependent selection model, (11b), on a single locus with multiple alleles that contribute additively to the trait, or even on multiple additive loci with multiple alleles in linkage equilibrium, is governed by an increasing global Lyapunov function. In the one locus case this function is

\[
\Lambda^{(\text{BS})} (p) = (\eta - 1) v - \bar{x}^2,
\]

where the trait’s mean and variance are \(\bar{x}\) and \(v\), respectively. Note that if selection near the evolutionary singularity at \(x = 0\) is stabilizing, i.e., if \(\eta < 1\), the implication of this Lyapunov function is that the population variance of the selected trait, \(v\), tends to be minimized. If, instead, selection is disruptive, i.e., \(\eta > 1\), the population variance tends to be maximized. In either cases the mean trait tends to the singular trait value of zero.

Again, since the Bürger–Schneider model is a particular case of the general model, it must also admit the general Lyapunov function \(\Lambda\), (12), so that we expect \(\Lambda^{(\text{BS})}\) to be a particular version of \(\Lambda\). Indeed, since

\[
s_j = \sigma (\eta - 1) x_j^2 \quad \text{and} \quad a_{ij,kl} = -2\sigma \eta x_i x_k x_l,
\]

we find

\[
\Lambda (p) = 2\sigma (\eta - 1) v - 2\sigma \bar{x}^2 = 2\sigma \Lambda^{(\text{BS})} (p).
\]

Hence, we have

**Corollary 2.** The one-locus, multiallele version of the Bürger–Schneider model in continuous time is a particular case of the Wildcard Model (5). Hence, it is a generalized gradient system. Its potential is \(\frac{\sigma}{\eta} \Lambda^{(\text{BS})}\), where \(\Lambda^{(\text{BS})}\), given by (20) is a global (increasing) Lyapunov function. Thus, \(\Lambda^{(\text{BS})}\) is a particular case of \(\Lambda\) given by (12).

Note that the statement of the above corollary is more general than the results of Bürger (2005) and Schneider (2006) for the one-locus case, since it is not limited by the restriction to additive alleles.

Also, from the correspondence between the Bürger–Schneider and the Matessi–Gimelfarb–Gavrilets models and from (20) we immediately obtain

**Corollary 3.** The one-locus multiallele dynamics in continuous time driven by the Matessi–Gimelfarb–Gavrilets–Gavrilets models forms a generalized gradient system. Its potential function is given by \(\frac{\sigma}{\eta} \Lambda^{(\text{MG})}\), where

\[
\Lambda^{(\text{MG})} (p) = \frac{2\sigma}{\beta - \alpha} v - \bar{x}^2
\]

is an increasing global Lyapunov function.

3.3. An ancestor of the Lyapunov function of the Wildcard Model

3.3.1. The Matessi–Jayakar model

Matessi and Jayakar (1981) analyzed density- and frequency-dependent selection, arising from scramble competition for limited resources, by means of a Lotka–Volterra fitness model applied to a system of multiple species with a single locus and multiple alleles
per species. In the case of just one species, the fitness that this model assigns to individuals of type \( x \) in a population of size \( N \) is

\[
W^{(M)}_x = 1 + r \left( K_x - N \sum_y B_{x,y} y_p \right), \quad B_{x,y} = B_{y,x},
\]

(23a)

where \( r \) is the intrinsic rate of increase of the population, \( K_x/B_{x,x} \) is the carrying capacity the population would have if it consisted only of individuals of type \( x \), and \( B_{x,y}/B_{y,x} \) is the intensity of competition between types \( x \) and \( y \).

We again assume that a single locus with \( n \) alleles determines the types. Accordingly, in discrete time, the changes occurring to a population in one generation are described by the following system of recurrence equations

\[
N' = N \left[ 1 + r \left( K - N \overline{B} \right) \right],
\]

(23b)

\[
p_i' = p_i \frac{1 + r \left( K - N \overline{B} \right)}{1 + r \left( K - N \overline{B} \right)},
\]

(23c)

for \( i \in \{1, \ldots, n\} \), where \( p_i \) is the frequency of \( i \)th allele while

\[
\overline{K}_i = \sum_{j=1}^n K_j p_j, \quad \overline{B}_i = \sum_{j,k,l=1}^n B_{j,k,l} p_j p_k p_l \quad \text{and} \quad \overline{B} = \sum_{i=1}^n \overline{B}_i p_i.
\]

(23d)

If the intrinsic rate of increases, \( r \), is small enough, a continuous-time approximation can be made – even without assuming that differences among genotypes are very slight – transforming (23) into the following system of differential equations (Matessi and Jayakar, 1981)

\[
\dot{N}(t) = \rho N(t) \left[ \overline{K} - N(t) \overline{B} \right],
\]

(24a)

\[
\dot{p}_i(t) = \rho p_i(t) \left[ \overline{K}_i - N(t) \overline{B}_i - \overline{K} + N(t) \overline{B} \right],
\]

(24b)

for \( i \in \{1, \ldots, n\} \), where \( r = \tau \rho \), and \( \tau \) is the duration of the time interval between successive generations. If \( \tau \) tends to zero, (23) becomes (24). Matessi and Jayakar (1981) have demonstrated that (24) admits the following global Lyapunov function

\[
\psi^{(M)}(\mathbf{p}, N) = 2NK - N\overline{B}^2.
\]

(25)

By comparing (23)–(24) to (1)–(5), we immediately notice that these two models would be essentially the same were it not for the density dependence in (23a). Correspondingly, it is clear that there is a close relation among the respective Lyapunov functions \( \psi^{(M)}(\mathbf{p}, N) \), (25), and \( \Lambda(\mathbf{p}) \), given by (12); so close that one would ask how the latter could be derived from the former.

Indeed, from a purely formal point of view, (12) can be immediately obtained from (25) by simply assuming that the population size, \( N \), is a constant, \( N \equiv N_0 \). Hence, by deleting the differential equation (24a) from (24), \( \psi^{(M)} \) and \( \Lambda \) become identical, by the obvious switch of notation

\[
N_0 \dot{B}_{ij} = -a_{ij}, \quad N_0 \delta B_{j,k,l} = -a_{j,k,l}.
\]

(26)

This point of view, although formally correct, has the weakness that it destroys the biological meaning of the Matessi–Jayakar model, (23)–(24), because a model in which competition for limited resources keeps the population size constant would be biologically inconceivable.

However, we can put forward a second argument that leaves the biology of (23)–(24) intact while producing \( \Lambda \) as an approximation of \( \psi^{(M)} \). This argument is rooted in the approach taken by Christiansen and Loeschcke (1980) to analyze evolutionary consequences of competition for limited resources starting from Lotka–Volterra-like equations exactly of the same form as (23).

To this model the authors add two assumptions. The first is implicitly justified by a ‘singular-perturbation’ argument (e.g., Hoppensteadt, 1974). Namely it is stipulated that the population size changes much more rapidly than the population composition. This is reasonable if selection is sufficiently weak relative to the intrinsic rate of increase. Hence, it can be assumed that, on the slow time scale of the population-composition dynamics, the population size is always very close to the equilibrium value corresponding to the current value of the allele frequencies. The second assumption is that the carrying capacity parameters, \( K_x \), and the competition parameters, \( B_{x,y} \), (23a), are determined by the value of a continuous phenotypic trait, according to

\[
K_x = \exp \left[ -\theta x^2 \right] \quad \text{and} \quad B_{x,y} = \exp \left[ -\theta (x-y)^2 \right].
\]

(27a)

In addition, Christiansen and Loeschcke (1980) concentrate mostly on the case in which all individuals have trait values close to zero. A situation that entails weak selection and justifies the further approximation that

\[
K_x \simeq 1 - \theta x^2 \quad \text{and} \quad B_{x,y} \simeq 1 - \theta (x-y)^2.
\]

(27b)

In order to show the kinship of the two Lyapunov functions, \( \psi^{(M)}(\mathbf{p}, N) \) and \( \Lambda(\mathbf{p}) \), we follow Christiansen and Loeschcke (1980) and therefore, first, replace (24a) by

\[
N(t) = \hat{N}(\mathbf{p}(t)) = \overline{K} - \overline{B} \overline{p},
\]

(28a)

Next, we assume there is a small number \( \epsilon \) such that

\[
\overline{K}_i = k_0 + \overline{k}_i \epsilon + o(\epsilon), \quad \overline{B}_i = b_0 + \overline{b}_i \epsilon + o(\epsilon), \quad \overline{B} = b_0 + \overline{b} \epsilon + o(\epsilon),
\]

(28b)

\[
\overline{K} = \sum_{i=1}^n \overline{k}_i p_i, \quad \overline{B} = \sum_{i=1}^n \overline{b}_i p_i.
\]

(28c)

Finally, resting on these assumptions we can immediately conclude with

**Result 1.** (i) The system of differential equations (24) can be approximated by

\[
\dot{N}(t) = \hat{N}(\mathbf{p}(t)) = \overline{K} - \overline{B} \overline{p},
\]

(29a)

\[
\dot{p}_i(t) = \rho p_i(t) \left[ \overline{K}_i - N_0 \overline{B}_i - \overline{K} + N_0 \overline{B} \right],
\]

(29b)

for \( i \in \{1, \ldots, n\} \), where \( N_0 = k_0/b_0 \).

(ii) The Lyapunov function \( \psi^{(M)}(\mathbf{p}, N) \) can be approximated by

\[
\psi^{(M)}(\mathbf{p}) = 2N_0 \overline{K} - N_0 \overline{B}^2.
\]

(29c)

(iii) By changing notation according to (26), (29b) and (5b) coincide as well as the respective Lyapunov functions.

3.3.2. The Christiansen–Loeschcke model

Having recalled above some of the assumptions that characterize the model of Christiansen and Loeschcke (1980), we have the opportunity to uncover another aspect of the network connecting a variety of apparently unrelated models of frequency-dependent selection. In fact, it is easy to verify (cf. Bürger, 2005) that, by its very assumptions, (27b) and (28a), the Christiansen–Loeschcke model reduces to a density-independent selection model equivalent to the models of Matessi et al. (2001), Matessi and Gimelfarb (2000), (9a), and of Bürger (2005), Schneider (2006), (11b).
Table 1
Comparison of the fitness functions of three frequency-dependent selection models.

<table>
<thead>
<tr>
<th>Model</th>
<th>A</th>
<th>B</th>
<th>C(q)</th>
<th>Range</th>
<th>Stabilizing selection</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL</td>
<td>θ - θ</td>
<td>2θ</td>
<td>(θ + θ)(x^2 + v)</td>
<td>θ, θ &gt; 0</td>
<td>θ &lt; θ</td>
</tr>
<tr>
<td>MG</td>
<td>α - (α + β)</td>
<td>β(x^2 + v)</td>
<td>α &lt; β</td>
<td>α &gt; 0</td>
<td></td>
</tr>
<tr>
<td>BS</td>
<td>σ(η - 1) - 2σα</td>
<td>σηx^2 + ψ(q)</td>
<td>σ, η &gt; 0</td>
<td>σ, η &lt; 1</td>
<td></td>
</tr>
</tbody>
</table>

The correspondence between the Christiansen–Loeschcke (CL), the Matessi–Gimelfarb–Gavrilets (MG) and the Bürger–Schneider (BS) model is shown in the table. x, v and q denote the mean trait value, the variance of trait values, and the distribution of types, respectively. The parameters are described in the main text. Moreover, their admissible range is shown and the condition under which selection is stabilizing in the respective models. If the respective condition is violated selection is disruptive.

To see this it suffices to substitute (27b) and (28a) into the fitness function (23a), which coincides exactly with the basic model from which Christiansen and Loeschcke (1980) start. In this substitution only the terms that are up to second order in the trait values are to be retained, because all trait values in the population are assumed to be very close to zero. In this way we find that the fitness function resulting from the assumptions of Christiansen and Loeschcke (1980) is

\[ W_s^{(CL)} = 1 + (θ - θ) x^2 - 2θ x + (θ + θ) (x^2 + v) \]

(30a)

where x and v are respectively the mean and the variance of the trait values in the population. The parameters θ and θ depend on the variances of the (Gaussian) resource spectrum, V_k, and the individual's (Gaussian) resource utilization distribution, V_U, assumed to be the same for all individuals that, depending on their genotype, vary only in the modal position of their utilization distributions. More specifically, the relation among these parameters is

\[ θ = \frac{r}{2V_k + 2V_U}, \quad Δ = \frac{r}{4V_U} \]

(30b)

(cf. Bürger, 2005).

From (30a) it is immediately obvious that W_s^{(CL)} is essentially equivalent to the fitness functions W_s^{(MG)}, of (9a), and W_s^{(BS)} of (11b). Table 1 summarizes the correspondences among the parameters of these three functions. We also conclude that the global Lyapunov function associated to the one-locus, multiallele dynamics in continuous time, generated by frequency-dependent selection with the Christiansen–Loeschcke fitness function W_s^{(CL)}, (30), is

\[ \Lambda^{(CL)}(p) = \frac{θ - θ - v - x^2}{θ} \]

(31)

This Lyapunov function directly informs us that in the Christiansen–Loeschcke model selection is stabilizing and phenotypic variance, v, (the variance among the positions of individual utilization distributions on the resource axis) tends to be minimized if the width of the resource spectrum, V_k, is smaller than that of the individual utilization distribution, V_U. In the opposite case selection is disruptive and the phenotypic variance tends to be maximized. In both cases the mean of the positions of individual utilization functions on the resource axis tends to the modal point of the resource abundance.

4. Multiple loci

Concerning the genetic assumptions, so far we have assumed a single locus with multiple alleles. In this section we want to generalize the results of the previous sections to the case of multiple multiallelic loci in linkage equilibrium. Furthermore, based on the motivation of the last section, we will introduce a density-dependent version of our model.

Before we formulate our model and generalize our results to multiple loci we need some preliminaries. First we will briefly summarize the multilocus notation that we will use.

4.1. The multilocus multiallele framework

For the multilocus dynamics we use the notation introduced by Nagylaki (1993) and Nagylaki et al. (1999). As before, we assume a randomly mating diploid population under viability selection. Now, we consider n loci instead of just one, where at locus k the m_k alleles θ_k^{(n)} can occur (i_k ∈ {1, ..., m_k)). The multi-index i = (i_1, ..., i_n) is used as an abbreviation for the gamete θ_1^{(i_1)}θ_2^{(i_2)}...θ_n^{(i_n)}; its frequency is denoted by p_i. Collectively, these frequencies form the vector p of gamete frequencies, which are elements of the simplex S_{m_1, ..., m_n}. The frequency of θ_k^{(i_k)} is given by

\[ p_{i_k} = \sum_i p_i \theta_k^{(i_k)} \]

(32)

The above sum runs over all multi-indices i with the kth component fixed as i_k. We denote the gene-frequency vector (p_i) of the population by \[ p \in \mathbb{R}^n \] or \[ p \in \mathbb{R}^n \]. The fitness of genotype i is denoted by W_q_i, and since we assume absence of genomic imprinting, W_q = W_q_i holds for all i, j. The marginal fitness of gamete f is given by

\[ W_f = \sum_i W_q_i p_i \]

(33)

and the mean fitness of the population is

\[ \bar{W} = \sum_f W_q p_f \]

(34)

Let \( I, J \) be a nontrivial decomposition of the set \( M = \{1, ..., n\} \), i.e., \( I \cup J = M \) and \( I \cap J = \emptyset \) normalized by \( 1 \in I \). We denote by \( r_{ij} \) the recombination probability between the sets of loci I and J. Throughout we assume \( r_{ij} > 0 \) for every I. Thus, the dynamics of the gametic frequencies are given by the following recurrence relations,

\[ p_i' = p_i W_i - D_i \]

(35)

where

\[ D_i = \frac{1}{W} \sum_j r_{ij} (W_q_{i_j} p_{i_j} - W_{i_{k}j_{k}j_{k}} p_{i_{k}j_{k}j_{k}}) \]

(36)

is a measure for linkage disequilibrium in game i. In the above formula, \( i_{j_{k}} \) signifies the vector with kth component \( i_{k} \) if \( k \in I \) and \( j_{k} \) if \( k \in J \). The allele frequencies in the next generation are

\[ p_{i_k}' = \sum_i p_i W_{i_k}^{(k)} \]

(37)

where the marginal fitness, \( W_{i_k}^{(k)} \), of allele \( \theta_k^{(i_k)} \) is defined by

\[ p_{i_k}' W_{i_k}^{(k)} = \sum_i p_i W_i \]

(38)

The linkage-equilibrium manifold, or Wright manifold, is given by

\[ \mathcal{L}_0 = \{ p : p_{i_1} = p_{i_1}^{(1)} p_{i_2}^{(2)} ... p_{i_n}^{(n)} \text{ for all } i \} \]

(39)

We assume that there is no position effect, i.e., \( W_q = W_{i_{k}j_{k}j_{k}} \) for every \( i_{k}, j_{k}, j_{k} \). Therefore, it follows immediately that \( D_i = 0 \) for
every \( p \in \mathcal{S}_0 \), and we have \( \mathcal{S}_0 \subseteq \{ p \mid D_i = 0 \ \text{for every} \ i \} \). Equality holds in the absence of selection (see Nagylaki et al., 1999).

Fitnesses are frequency dependent and if population regulation is added to the model, they depend also on the population size, \( N \). In such a situation the population size changes according to the following recursion relation:

\[
N' = N \bar{w}.
\]  

(40)

In the following we always assume linkage equilibrium, in which case it is sufficient to consider the dynamics of allele frequencies (37) instead of the dynamics of gamete frequencies (35). Additionally, in analogy to the one-locus case of previous sections, we will assume weak selection and therefore the discrete-time dynamics (37) and (40) will be approximated by a continuous-time dynamics. Our results will hold for specific cases that admit an invariant manifold (the quasi-linkage-equilibrium manifold) sufficiently close to the linkage-equilibrium manifold, where the dynamics behave almost as if they were in linkage equilibrium, which is reached by every trajectory within a few generations. The linkage-equilibrium approximation will not be accurate for any given model. The applicability of such an approximation will heavily depend on the concrete model, i.e., the concrete expressions of the fitnesses, the strength of selection relative to recombination etc. Such results are usually difficult to prove. For instance, the deep results of Nagylaki et al. (1999) prove convergence to quasi-linkage equilibrium for constant selection and weak epistasis, provided recombination is not too weak. Bürger (2005) and Schneider (2006) used these results to prove convergence to quasi-linkage equilibrium in their models.

As in the previous sections, we will concentrate on our Wildcard Model, (1), and also introduce a density-dependent modification of this model, which slightly generalizes the Matessi–Jayakar model (23a).

4.2. The density-independent model

Now, we are able to formulate the multilocus version of our Wildcard Model. In analogy to the single locus case we assume that the phenotypes under selection are identified with their genotypic configuration at the \( n \) loci under consideration. Since we are dealing with a continuous-time approximation, according to an argument analogous to that in Section 2.1, the fitness of the genotype \( \mathbf{i} \) appropriate for the differential equations is given by

\[
w_\mathbf{i} = s_\mathbf{i} + \sum_{k,l} a_{\mathbf{i}kl} p_k p_l + \gamma(\rho).
\]  

(41)

Here, the parameters are defined in a way similar to (4). In particular, the frequency-independent fitness component, \( s_\mathbf{i} \), satisfies

\[
s_\mathbf{i} = s_\mathbf{i}. \tag{42a}
\]

Moreover, the parameters \( a_{\mathbf{ijkl}} \) of the frequency-dependent term must satisfy

\[
a_{\mathbf{ijkl}} = a_{\mathbf{klji}} = a_{\mathbf{ijlk}} = a_{\mathbf{klji}} \tag{42b}
\]

for all \( i, j, k, l \). As in the one-locus case we assume that an individual \( \mathbf{i} \) is affected by an individual \( \mathbf{kl} \) in the same way as \( \mathbf{kl} \) is affected by \( \mathbf{ij} \), which implies

\[
a_{\mathbf{ijkl}} = a_{\mathbf{klji}} \tag{42c}
\]

for all \( i, j, k, l \). Moreover, \( \gamma(\rho) \) is a function that depends only on the frequency distribution of alleles and is the same for all genotypes. We mention here that all our results hold if \( \gamma(\rho) \) in (41) is replaced by \( \psi_\mathbf{i} \) for all \( i,j \), which are arbitrary functions of the allele frequencies \( \rho \) satisfying \( \psi_\mathbf{ij} = \psi_\mathbf{ji} \) and

\[
\sum_j \psi_\mathbf{ij}(\rho)p_j = \sum_j \psi_\mathbf{ji}(\rho)p_j \quad \text{for all} \ i, k.
\]

The marginal fitness of gamete \( i \) is then given by

\[
\bar{w}_i = \sum_j s_\mathbf{ij} p_j + \sum_{j,k,l} a_{\mathbf{ijkl}} p_k p_l + \gamma(\rho),
\]

and the mean fitness is given by

\[
\bar{w} = \sum_{ij} s_\mathbf{ij} p_i p_j + \sum_{ij,kl} a_{\mathbf{ijkl}} p_k p_l + \gamma(\rho). \tag{43a}
\]

The dynamics in linkage equilibrium then become

\[
\dot{p}_\mathbf{ik} = \frac{p_\mathbf{ik}}{w_\mathbf{ik}} (w_\mathbf{ik} - \bar{w})
\]

for \( 1 \leq i_k \leq m_k, \ 1 \leq k \leq n, \) (43b)

where (by using the linkage-equilibrium assumption)

\[
w_\mathbf{ik} = \sum_{l=1}^n p_{l}^{(i)} \prod_{l'=1, l \neq k}^n p_{l'}^{(i)} + \gamma(\rho).
\]  

(43c)

Note that (43b) is independent of \( \gamma(\rho) \).

We are now able to generalize the results of the corresponding one-locus model to the multilocus model in linkage equilibrium.

Theorem 2. Consider the system (43). Moreover, let

\[
\Lambda(\rho) := 25 + \bar{a},
\]

where

\[
\bar{s} = \sum_{ij} s_\mathbf{ij} p_i p_j \quad \text{and} \quad \bar{a} = \sum_{ij,kl} a_{\mathbf{ijkl}} p_k p_l p_i p_j.
\]

Then, the system (43b) is a generalized gradient system with potential \( \Lambda/4 \), i.e.,

\[
\dot{p}_\mathbf{ik} = \frac{p_\mathbf{ik}}{4} \left( \frac{\partial \Lambda}{\partial p_\mathbf{ik}} - \sum_{j=1}^n p_\mathbf{j}^{(i)} \frac{\partial \Lambda}{\partial p_\mathbf{jk}} \right)
\]

for \( 1 \leq i_k \leq m_k, 1 \leq k \leq n, \)

In particular, \( \Lambda \) is a global Lyapunov function, i.e., \( \dot{\Lambda}(\rho) \geq 0 \) and \( \dot{\Lambda}(\rho) = 0 \) if and only if \( \rho \) is an equilibrium, and the \( \omega \)-limit of every trajectory is contained in the set of equilibria.

Proof. We need to calculate the partial derivatives \( \frac{\partial \Lambda}{\partial p_\mathbf{ik}} \). First, note that under the assumption of linkage equilibrium we have

\[
\frac{\partial p_\mathbf{ij}}{\partial p_\mathbf{kl}} = \delta_{\mathbf{ijkl}} \prod_{l'=1, l \neq k}^n p_{l'}^{(i)}
\]

where \( \delta \) denotes the Kronecker-\( \delta \). Now, straightforward calculations yield

\[
\frac{1}{4} \frac{\partial \Lambda}{\partial p_\mathbf{ik}} = \frac{1}{2} \sum_{ij} s_\mathbf{ij} \left( \frac{\partial p_\mathbf{ij}}{\partial p_\mathbf{ik}} p_j + \frac{\partial p_\mathbf{ij}}{\partial p_\mathbf{jk}} p_i \right)
\]

\[+ \frac{1}{4} \sum_{ij,kl} a_{\mathbf{ijkl}} \left( \frac{\partial p_\mathbf{ij}}{\partial p_\mathbf{ik}} p_k p_l + \frac{\partial p_\mathbf{ij}}{\partial p_\mathbf{jk}} p_i p_l \right)
\]
Theo-

Consider the system of differential equations

\[ \frac{\partial p_k}{\partial t} = \sum_{ij} s_{ij} \frac{\partial p_i}{\partial p_k} p_j p_t + \sum_{i,j,k,l} a_{ijkl} \frac{\partial p_i}{\partial p_k} p_j p_k p_l \]

\[ = \sum_{i,j} s_{ij} \frac{\partial p_i}{\partial p_k} \prod_{l=1}^n p_l^{(i)} + \sum_{i,j,k} a_{ijkl} \left( \prod_{l=1}^n p_l^{(j)} \right) \prod_{l=1}^n p_l^{(i)} \]

\[ = \sum_{i,j} \sum_{k=1}^n \left( w_{ik} - \gamma(\rho) \right) \left( w_{jk} - \overline{w} \right) p_i^{(k)} + \sum_{i,j} \sum_{k=1}^n \left( w_{ik} - \overline{w} \right) \left( w_{jk} - \overline{w} \right) p_i^{(k)} \geq 0. \]

Moreover, we have \( \dot{\lambda} = 0 \) if and only if \( \dot{w}_{ik} = \overline{w} \) or \( \dot{p}_k^{(i)} = 0 \), i.e., if and only if \( \dot{w}_{ik} = \overline{w} \) whenever \( \dot{p}_k^{(i)} \neq 0 \). In other words, we have \( \dot{\lambda} = 0 \) only at an equilibrium. Thus, \( \lambda \) is a global Lyapunov function. Its time derivative vanishes only at the equilibria and the \( \omega \)-limit of every trajectory is contained in the set of equilibria. \( \square \)

From similar considerations as in the previous section and from straightforward multilocus formulations of the particular models we immediately obtain the following

**Corollary 4.** Corollaries 1–3 remain valid for underlying multilocus, multiallelic genetics if linkage equilibrium can be assumed.

Note that Bürger (2005) and Schneider (2006) in the proofs of the existence of a gradient system in their model assumed additive genetics, i.e., no dominance or epistasis on the trait level, an assumption that is not required by our more general result (Corollary 4). However, using the additivity assumption, Bürger (2005) and Schneider (2006) were able to prove that the trajectories in their model indeed converge to quasi-linkage equilibrium if selection is sufficiently weak compared with recombination. It is yet unknown to what extent the linkage-equilibrium approximation is valid if the additivity assumption is relaxed, as in our model. Thus, to apply our more general results to a concrete model one has first to ascertain the validity of the linkage-equilibrium approximation.

### 4.3. The density-dependent model

Here, we shall formulate a model that is slightly more general than the Matessi–Jayakar model (see Section 3.3.1) with underlying multilocus genetics. We will refer to it as the density-dependent version of the Wildcard Model. It assumes that the fitness of genotype \( \mathbf{j} \) is given by

\[ w_{ij} = s_{ij} + f(N) \sum_{k,l} a_{ijkl} p_k p_l. \]

where the \( s_{ij} \)'s, and the \( a_{ijkl} \)'s are as in the density-independent case, hence they have the same interpretation, and we impose (42). Moreover, \( f(N) \) is some differentiable function of the population size satisfying \( f(N), f'(N) > 0 \) or \( f(N), f'(N) < 0 \) for all admissible values of \( N \). Note, that \( \Sigma_{i,j}^{(k)} \) is similar to (1) or (41), but it does not allow for a global effect of the population composition on fitness, i.e., an arbitrary function \( \gamma(\rho) \).

The marginal fitness of gamete \( i \) is then given by

\[ \frac{\partial p_i^{(k)}}{\partial t} + \frac{\partial p_i^{(k)}}{\partial p_k} p_j p_t + \sum_{i',j',k',l'} a_{i'j'k'l'} \frac{\partial p_i^{(k)}}{\partial p_k} p_j p_k p_l = \left( w_{ik} - \gamma(\rho) \right) \left( \frac{w_{ik} - \overline{w}}{\overline{w}} \right) p_i^{(k)} + \sum_{i',j',k',l'} \left( w_{i'k} - \overline{w} \right) \left( w_{j'l} - \overline{w} \right) p_{i'}^{(k)} p_{j'}^{(l)} \geq 0. \]

Moreover, we have \( \frac{\partial p_i^{(k)}}{\partial t} = 0 \) if and only if \( \frac{\partial p_i^{(k)}}{\partial p_k} = 0 \), i.e., if and only if \( \frac{\partial p_i^{(k)}}{\partial p_k} = 0 \) whenever \( \frac{\partial p_i^{(k)}}{\partial p_k} \neq 0 \). In other words, we have \( \frac{\partial p_i^{(k)}}{\partial t} = 0 \) only at an equilibrium. Thus, \( \frac{\partial p_i^{(k)}}{\partial t} \) is a global Lyapunov function. Its time derivative vanishes only at the equilibria and the \( \omega \)-limit of every trajectory is contained in the set of equilibria. \( \square \)

The following theorem, a density-dependent analog of Theorem 2, extends the results of the corresponding one-locus results of Matessi and Jayakar (1981) to the multilocus model with linkage equilibrium:

**Theorem 3.** Consider the system of differential equations (45). Let

\[ \Psi(\rho, N) \equiv f(N) \left( 2\overline{s} + f(N) \overline{w} \right), \]

where

\[ \overline{s} = \sum_{i,j} s_{ij} p_i p_j \text{ and } \overline{w} = \sum_{i,j,k,l} a_{ijkl} p_i p_j p_k p_l. \]

Then (45) is a generalized gradient system with potential \( \Psi/4 \), i.e.,

\[ \frac{\partial p_i^{(k)}}{\partial t} = \frac{\partial \Psi}{\partial p_i^{(k)}} \left( \frac{\partial \Psi}{\partial p_k} \right) - \sum_{j,k,l} p_j^{(k)} \frac{\partial \Psi}{\partial p_k^{(l)}} \]

and

\[ \dot{N} = N \frac{\partial \Psi}{\partial N} \left( \frac{\partial \Psi}{\partial \overline{w}} \right) \]
Moreover, we have
\[
\frac{1}{4} \frac{\partial \Psi}{\partial N} = \frac{f'(N)}{4} \left( 2 \sum_{ij} s_{ij} p_i p_j + 2f(N) \sum_{i,j,k} a_{ijk} p_i p_j p_k \right)
\]
\[= \frac{f'(N)}{2} \Psi.\]

The above derivations immediately yield the first statement. By applying the chain rule we obtain
\[
\Psi = \frac{d\Psi}{dt} = \frac{\partial \Psi}{\partial N} \frac{dN}{dt} + \sum_{k=1}^n \sum_{i,k}^m \frac{\partial \Psi}{\partial p_i} p_i \dot{p}_k
\]
\[= N \frac{f'(N)}{2} \omega^2 + f(N) \sum_{i,k=1}^m w^{(k)}_i (w^{(k)}_i - \omega) p_i
\]
\[= N \frac{f'(N)}{2} \omega^2 + f(N) \sum_{k=1}^m \sum_{i=1}^n \sum_{k=1}^m (w^{(k)}_i - \omega)^2 p_i.
\]

Hence, since we assume \( f(N) > 0 \) and \( f'(N) > 0 \) or \( f(N) < 0 \) and \( f'(N) < 0 \), we have \( \Psi \geq 0 \). Moreover, we have \( \Psi = 0 \) if and only if \( \omega = 0 \) and \( w^{(k)}_i = \omega \) provided \( p_i \neq 0 \). In other words, we have \( \Psi = 0 \) only at an equilibrium. This finishes the proof. □

We immediately obtain the following corollary

**Corollary 5.** The multilocus, multiallele version of the Matessi–Jayakar model under the assumption of linkage equilibrium is a generalized gradient system. Its potential function is a global Lyapunov function and is given by

\[
\psi^{(M)}(\rho, N) = N(2\mathcal{R} - \mathcal{B}).
\]

Observe that the density-dependent version of the Christiansen–Loeschcke model, i.e., the model without the singular-perturbation approximation, is a particular case of the Matessi–Jayakar model. Hence, Corollary 5 is also valid for the density-dependent Christiansen–Loeschcke model with obvious modifications.

Note that the density-dependent version of the Bürger–Schneider model is not a particular case of our density-dependent Wildcard Model. The fitness in the Bürger–Schneider model can be written in the form \( w^{(85)}_y = w_y + \gamma(\rho, N) \), where \( w_y \) is given by (44) with adequate choices for \( s_{ij}, a_{ijk}, m, \) and \( f(N) \), and the function \( \gamma(\rho, N) \) is an appropriate function of \( \rho \) and \( N \). Since \( \gamma \) is not a constant, the Bürger–Schneider model is not covered by (44).

### 5. Discussion

In this article we studied a model of frequency-dependent selection (the Wildcard Model) at a single autosomal locus with arbitrarily many alleles. Starting from a discrete-time formulation we switched to the continuous-time analog of the model, which can be regarded as an approximation if selection is weak. We proved that our model forms a generalized gradient system, for which the potential function \( A \) can be derived explicitly. In particular, this function is a global Lyapunov function. Moreover, as a consequence all trajectories approach the set of equilibria. If this is finite, every trajectory converges to an equilibrium. We were able to extend these results to a density-dependent version of the Wildcard Model, which is a slightly more general version of the model introduced by Matessi and Jayakar (1981). Furthermore, under the assumption of linkage equilibrium, we obtained similar results for a multilocus, multiallele version of the density-independent as well as of the density-dependent model. Our model, in both its density-independent and density-dependent versions, subsumes and, in some respects, generalizes several other particular models, and hence admits many different biological interpretations. Furthermore, it extends our results on the Lyapunov function to these other models.

Christiansen and Loeschcke (1980) studied a model of intraspecific competition for a continuous, unimodal resource spectrum, derived from Lotka–Volterra-like dynamics, which is a special case of the Wildcard Model. The competition model studied by Bürger (2005) and Schneider (2006, 2007) is also a special case of the Wildcard Model. Their model was derived as an approximation to, to the best of our knowledge, all models of intraspecific competition for a unimodal resource continuum that are available in the theoretical literature. Initially motivated by the model of Bulmer (1974, 1980), it approximates for instance the models of Roughgarden (1972), Slatkin (1979), Christiansen and Loeschcke (1980), Christiansen (1982) and Loeschcke and Christiansen (1984). Hence, these models can be approximately represented by our model.

Lessard (1984) studied a game theoretically motivated model. In his model individuals join one of two phenotypic pools – or pure strategies – with a certain probability based on their genotype. Although this is not a model of intraspecific competition it also is a special case of the Wildcard Model. Another model that is rooted in evolutionary game theory is that of Matessi et al. (2001), which was later studied by Matessi and Gimelfarb (2006). They introduced a quadratic payoff function to study stabilizing or disruptive selection near an evolutionary singularity, i.e., MEAST or PEAST, respectively (cf. Christiansen, 1991) Such a payoff function can be regarded as the second order Taylor expansion to any payoff function near an evolutionary singularity. Also this model, which has no specific biological interpretation, can be formulated in terms of the Wildcard model.

A commonly studied model of frequency-dependent selection is the pairwise interaction model, e.g., Schütz et al. (1968), Allard and Adams (1969), Cockerham et al. (1972), Asmussen and Basnayake (1990), Altenberg (1991), Gavrilets and Hastings (1994), Asmussen et al. (2004), Trotter and Spencer (2008), and Schneider (2008). The pairwise interaction model can be formulated in terms of our density-independent model, whereas the class of frequency- and density-dependent models studied by Asmussen (1983) is covered by our density-dependent model.

Since all of the above-mentioned models can be represented, at least approximately, by the Wildcard Model, the existence of a global Lyapunov function for each of them follows immediately. Lyapunov functions were already established for the models of Matessi and Jayakar (1981), Lessard (1984), Bürger (2005), and Schneider (2006). They are all special cases of ours. However, the Lyapunov function in the two-phenotype model of Lessard (1984) was established for discrete time.

Bürger (2005) and Schneider (2006) used the assumptions of additivity within and between loci in the control of a quantitative trait to show the existence of a Lyapunov function for the density-independent version of their model. However, our results prove that for their model additivity at the trait level is not required for the existence of a Lyapunov function in the one-locus case. Moreover, in the multilocus case epistasis and dominance do not need to be excluded, as long as the assumption of linkage equilibrium is justified. The existence of a global Lyapunov function for the density-dependent version of the Bürger–Schneider model remains open, since this model is not a special case of our density-dependent Wildcard Model. However, our results do apply to a multilocus generalization of the model of Matessi and Jayakar (1981). Thus, in particular they are valid for the Christiansen–Loeschcke model with density-dependence and Gaussian resource utilization functions, including multilocus generalizations, as studied for instance by Loeschcke.
and Christiansen (1984) in a two-locus context. Hence, for the latter model more analytical results seem possible.

Lessaard (1984) and Schneider (2007) used the existence of a global Lyapunov function to study the long-term evolution of their models by repeated occurrence and possible invasion of new mutations. Thus, our results might be used to study long-term evolution of a variety of models with different interpretations based on explicit short-term results. Therefore, our results have the potential to bridge the gap between traditional population genetic or evolutionary models based on dynamical systems (short-term evolution) and models of adaptive dynamics (long-term evolution) based on invasion dynamics.

Although our results are relevant for many models in the theoretical literature, they are restricted in several respects. First, the Wildcard Model is formulated in continuous instead of discrete time, which implies weak selection. However, most of the above-mentioned models that are special cases of ours were formulated originally in discrete time. Hence, it would be desirable to prove similar results for this case. Since difference equations are usually more difficult to analyze than differential equations, it is uncertain whether similar results can be proved. Second, the symmetry assumption of our model is crucial, i.e., \( a_{ij} = a_{ji} \). Since our results do not hold without this assumption, the biological scenarios that are covered are constrained. For instance, multi-phenotype generalizations of the two-phenotype model of Lessard (1984) can be formulated with our model (e.g., for games such as “rock-scissors-paper” or “hawk-dove-bourgeois”, cf. Hofbauer and Sigmund, 1998), but in general, the symmetry assumption will be violated, unless the payoff matrix is symmetric. Therefore, our results cannot be applied to such generalizations of the Lessard model, indicating that the results of Lessard (1984) cannot be generalized. Anyway, our model will cover multilocus generalizations of the two-phenotype model. Third, our model allows only for a very specific kind of density dependence. Although it is not very general, this kind of density dependence occurs frequently in the theoretical literature. Fourth, in the multilocus case the assumption of linkage equilibrium is crucial. Generalizing our assumptions to linkage disequilibrium seems infeasible. Thus, when applying our results to a particular multilocus model, one has to establish convergence to (quasi-)linkage equilibrium first. Especially if recombination is weak compared with selection, or for strong epistasis, convergence to quasi-linkage equilibrium is likely to fail (cf. Schneider, 2006 for an example).

Summarizing, we studied a population-genetic model of frequency-dependent selection with underlying one-locus or multilocus genetics (that may also allow for population regulation), which covers many particular models that have been studied in the theoretical literature so far. Although, superficially these models seem to be different and have completely different biological interpretations, they turn out to be different disguises of the model studied in this article. Our results are hence applicable to a variety of evolutionary problems, and give hope that many of them can be tackled analytically. Especially the perspective of studying long-term evolution seems inviting for future research.

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