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Intermittency and transient chaos from simple frequency-dependent selection

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SUMMARY

Frequency-dependent selection is an important determinant of the evolution of gametophytic self-incompatibility systems in plants, aposematic (warning) and cryptic coloration, systems of mimicry, competitive interactions among members of a population, mating preferences, predator–prey and host–parasite interactions, aggression and other behavioural traits. Past theoretical studies of frequency-dependent selection have shown it to be a plausible mechanism for the maintenance of genetic variability in natural populations. Here, through an analysis of a simple deterministic model for frequency-dependent selection, we demonstrate that complex dynamic behaviour is possible under a broad range of parameter values. In particular we show that the model exhibits not only cycles and chaos but also, for a more restricted set of parameters, transient chaos and intermittency: alterations between an apparently deterministic behaviour and apparently chaotic fluctuations. This behaviour, which has not been stressed within the population genetics literature, provides an explanation for erratic dynamics of gene frequencies.

1. INTRODUCTION

Frequency-dependent selection is a form of selection where the fitness of an individual depends on the genetic composition of the population to which it belongs. Numerous examples of frequency-dependent selection are well documented. In particular, this form of natural selection is an important determinant of the evolution of gametophytic self-incompatibility systems in plants, aposematic (warning) and cryptic coloration, systems of mimicry, competitive interactions among members of a population, mating preferences, predator–prey and host–parasite interactions, aggression and other behavioural traits (see references in Ayala & Campbell 1974; Clarke & Partridge 1988). Frequency-dependent selection has been widely considered in population genetics literature as a potentially important mechanism for maintenance of genetic variability in natural populations (e.g. Cockerham *et al.* 1972; Clarke 1979; Asmussen & Basnayake 1990).

Frequency-dependent selection has been also demonstrated to be able to produce complex dynamics behaviour including cycles and deterministic chaos in allele frequencies. The best known example are models describing co-evolution of host–parasite systems (e.g. May & Anderson 1983; Seger 1988; Seger & Hamilton 1988). Cycles and chaos arise also in models of frequency-dependent selection describing a single species (May 1979; Hamilton 1980; Altenberg 1991; Holton & May 1993). Unfortunately, the importance of nonlinear phenomena has not been well recognized within the population genetics community.

The purpose of this note is to give additional examples of chaotic dynamics that can be at least potentially important in population genetics. Here we study a simple deterministic population genetics model of frequency-dependent selection describing a single species. We present a complete analysis of conditions for existence and stability of equilibria. We demonstrate that complex dynamic behaviour is possible under a broad range of parameter values. In particular we show that the model exhibits not only cycles and chaos, but also some of the most striking examples of nonlinear behaviour: transient chaos and intermittency. We give a very simple explanation of the mechanisms underlying these dynamic regimes and discuss their biological implications.

2. MODEL

We consider a deterministic model of a large randomly mating diploid population with discrete generations. We assume that there is a single diallelic locus with alleles A and a. Let w_{AA}, w_{Aa} and w_{aa} be the fitnesses (viabilities) of genotypes AA, Aa and aa, respectively, and p be the frequency of allele A, with $q = 1-p$. Under Hardy–Weinberg equilibrium, the frequencies of the three genotypes are p^2, $2pq$ and q^2. The change in p in one generation is described by the standard equation:

$$\Delta p = [p(w_A - \bar{w})/\bar{w}]. \quad (1)$$

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Here $w_A = pw_{AA} + qw_{Aa}$ is the average fitness of allele A and $\bar{w} = p^2 w_{AA} + 2pqw_{Aa} + q^2 w_{aa}$ is the mean fitness of the population. If the fitnesses are constant, the population gradually evolves to a polymorphic equilibrium (with $0 < p < 1$) provided there is overdominance (i.e. if $w_{Aa} > w_{AA}, w_{aa}$) or to a fixation state (with $p = 0$ or $p = 1$), otherwise. Frequency-dependent selection is incorporated in this model by assuming that fitnesses are not constant but depend on the genotype frequencies. In the simplest case, the fitnesses are linear functions of the genotype frequencies (Huang *et al.* 1971; Cockerham *et al.* 1972):

$$w_{AA} = p^2 W_{11} + 2pqW_{12} + q^2 W_{13},$$
$$w_{Aa} = p^2 W_{21} + 2pqW_{22} + q^2 W_{23},$$
$$w_{aa} = p^2 W_{31} + 2pqW_{32} + q^2 W_{33},$$

where W_{ij} are parameters ($i,j = 1, 2, 3$) characterizing the extent to which changes in the frequencies of three genotypes influence their fitnesses. (For examples of models of frequency-dependent selection with non-linear functions see Wright 1969; May 1979; Hamilton 1980). We introduce here a symmetric model of linear frequency-dependent selection, where the matrix of the coefficients W_{ij} has form

$$\begin{pmatrix} \delta & \beta & \alpha \\ \gamma & \eta & \gamma \\ \alpha & \beta & \delta \end{pmatrix}$$

Under this symmetric model, the dependence of fitness of a heterozygote w_{Aa} on the allele frequency p is described by a (quadratic) function symmetric about $\frac{1}{2}$, whereas w_{AA} and w_{aa} considered as (quadratic) functions of p are reflections of each other about $\frac{1}{2}$. The main justification for the symmetry assumption is that it allows to study the system analytically. We expect that in more realistic situations with some asymmetry the spectre of dynamic behaviour will be richer and will be observed under a wider range of parameter values. For this symmetric model to produce feasible (i.e. non-negative) fitnesses, one has to assume that $\alpha, \gamma, \delta > 0, \beta > -\sqrt{\alpha\delta}, \eta > -\gamma$.

The dynamics are not changed if fitnesses are multiplied or divided by a constant. This allows one to assume without loss of generality that $\delta = 1$. For the symmetric model, the dynamic equation (1) can be represented as

$$\Delta p = [pq(p-q)(1-\gamma-\Omega pq)]/\bar{w}, \qquad (2)$$

where the mean fitness can be represented as $\bar{w} = 1 - [2(2-\beta-\gamma)pq] + (2\Omega p^2 q^2)$ with $\Omega = 1 + \alpha - 2\beta - 2\gamma + 2\eta$. Numerous partial cases of this dynamic equation have been studied including those arising in the simplest models of frequency-dependent selection as described in recent textbooks (Hartl & Clarke 1989; Ridley 1993) and in more complicated models (Mallet & Barton 1989; Asmussen & Basnayake 1990; Altenberg 1991). All these references but the last one have concentrated on conditions for existence and stability of equilibria. Altenberg (1991) has broken new ground by demonstrating that there exists a small area of the parameter space, within which none of the equilibria are stable. He has shown that this area produces cycling or chaos and that different cyclic or chaotic attractors can be stable simultaneously. The symmetric model studied by Altenberg is described by (2) with $\Omega = 0$. Here we study the general case of (2). We shall show that the range of parameters that produce cycling or chaos is broad, that cycling or chaos can occur simultaneously with existence of several stable equilibria, and that the model can exhibit transient chaos and intermittency.

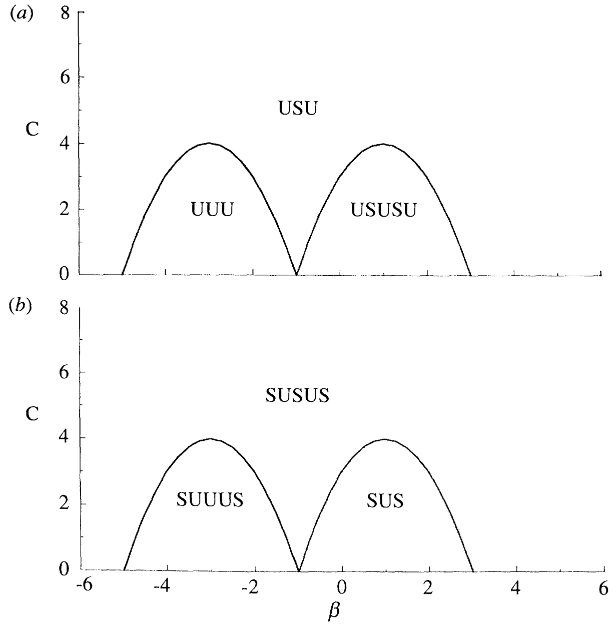

Figure 1. Areas in parameter space corresponding to different patterns of existence and stability of equilibria in a model of linear frequency-dependent selection. (*a*) $\gamma > 1$; (*b*) $\gamma < 1$. Only areas with $C \geqslant 0$ are shown.

As is apparent from (2), the allele frequency p does not change if $p = 0$, $q = 0$, $p = q$ or $pq = (1-\gamma)/\Omega$. Thus equation (2) always has two monomorphic equilibria at $p = 0$ and $p = 1$ and a polymorphic equilibrium at $p = \frac{1}{2}$. If $0 < (1-\gamma)/\Omega < \frac{1}{4}$, it has two additional polymorphic equilibria with allele frequencies satisfying $p(1-p) = (1-\gamma)/\Omega$. We shall denote these equilibria p_- and p_+. An equilibrium of (2) is stable if the corresponding eigenvalue lies between -2 and 0. These eigenvalues can be found in a straightforward manner.

Figure 1 summarizes conditions for existence and stability of different equilibria in terms of γ, β and a parameter C that combines several parameters. The parameter C is defined as $C = c_1 + c_2$ with $c_1 \equiv \alpha - \beta^2$ and $c_2 \equiv 2(\gamma + \eta)$ or, alternatively, as $C = \Omega + 4\gamma - (1-\beta)^2$. Parameter c_2 is the minimal possible value of the fitness of heterozygote. If $\beta < 1$, c_1 determines the minimal possible value of the fitness of homozygotes, $c_1/(c_1 + (1-\beta^2))$, whereas if $\beta > 1$, c_1 determines the maximum possible value of the fitness of homozygotes, $c_1 + \beta^2$. Thus parameter C characterizes the overall strength of selection. For fitnesses to be feasible c_2 must be non-negative and c_1 must be non-negative if $\beta < 0$. These conditions imply that C must be larger or equal to 0 if β is negative, and must be larger or equal to $-\beta^2$ if β is positive. Figure 1 shows areas in parameter space correponding to different patterns of existence and stability of equilibria in a

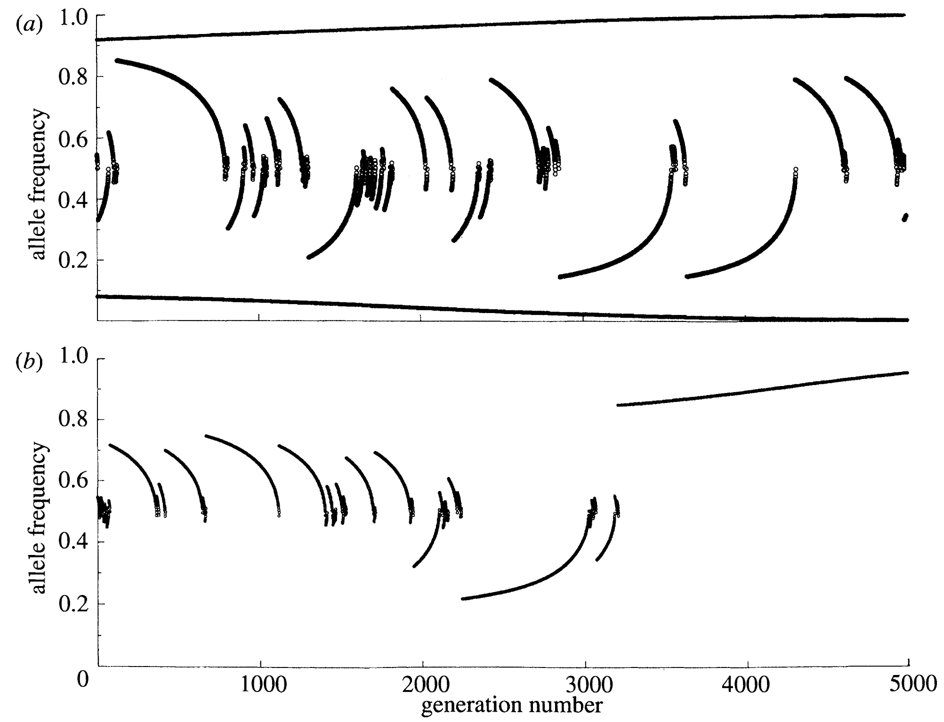

Figure 2. Dynamics of allele frequency with parameter values from the area marked SUUUS in figure 1. Both monomorphic equilibria ($p = 0$ and $p = 1$) are stable to small perturbations. (*a*) $\beta = -1.002$, $\gamma = 0.999$, $C = 0$. Three trajectories are shown with initial allele frequencies $p(0) = 0.09$, $p(0) = 0.55$ and $p(0) = 0.92$. (*b*) $\beta = -1.001$, $\gamma = 0.9985$, $C = 0$, $p(0) = 0.55$.

model of linear frequency-dependent selection. Each pattern is described by a string of S's (for stable) and U's (for unstable). The left-most, the middle and the right-most entries indicate the stability of the monomorphic equilibrium at $p = 0$, the polymorphic equilibrium at $p = \frac{1}{2}$ and the monomorphic equilibrium at $p = 1$, whereas the remaining entries (if any) indicate the stability of the polymorphic equilibria p_- and p_+. The left parabola is described by equation $C = -(\beta+5)(\beta+1)$. The right parabola is described by equation $C = -(\beta+1)(\beta-3)$.

Figure 1 shows that the system can have up to three different stable equilibria simultaneously, that a polymorphic equilibrium can be stable simultaneously with two monomorphic equilibria, and that two different polymorphic equilibria can be stable simultaneously. Simultaneous stability of different equilibria implies that the outcome of evolution strongly depends on the initial conditions and population history. These 'classical' features of the model of linear frequency-dependent selection have been known from previous studies (e.g. Cockerham *et al.* 1971; Asmussen & Basnayake 1990). If parameters change in such a way that the system moves from one area to another, the dynamic system undergoes a bifurcation. For example, a change from USU to USUSU corresponds to a pitchfork bifurcation. Figure 1 also shows that there are two areas with non-standard patterns of stability of equilibria. In the first area (marked UUU), none of the three equilibria (two monomorphic and one polymorphic at $p = \frac{1}{2}$) are stable (cf. Altenberg 1991). If parameters change in such a way that the system moves from USU to UUU, the dynamic system undergoes a period-doubling bifurcation. In the second area (marked SUUUS), the two monomorphic equilibria are stable, whereas none of the three polymorphic equilibria are stable. Numerical iterations of (2) with parameter values corresponding to these areas reveal a variety of complex dynamic behaviours (e.g. cycles and chaos that arises via period-doubling route) similar to those observed in classical ecological models (e.g. May 1974, 1976; May & Oster 1976; Hastings *et al.* 1993). Figure 1 shows that sufficient conditions for the complex dynamic behaviour to occur is sufficiently strong overall selection (i.e. small C) and sufficiently strong deleterious effect of heterozygote on homozygotes (i.e. $\beta < -1$). Altenberg (1991), who makes a similar conclusion for a model representing one case of our symmetric model, has discussed biological situations under which these conditions can be satisfied.

There are also two unusual types of behaviour described in figure 2. In figure 2*a*, depending on the initial conditions, the population evolves to a fixation state or remains polymorphic indefinitely. In the latter case, the gradual changes in the allele frequency towards $p = \frac{1}{2}$ are interrupted by apparently chaotic fluctuations that move p away from $\frac{1}{2}$. Such alterations between an apparently deterministic behaviour and apparently chaotic fluctuations, repeated at apparently random intervals, are called 'intermittency' (Pomeau & Manneville 1979; Olsen & Degn 1985). In figure 2*b*, these alterations end at some time point with the population settling down to a monomorphic state. In this case the system exhibits 'transient chaos' (Grebogi *et al.* 1983; Tél 1990). In the examples presented in figure 2, the deterministic phase of the dynamics can last for hundreds of generations, the chaotic phase is extremely short and transient chaos (in figure 2*b*)

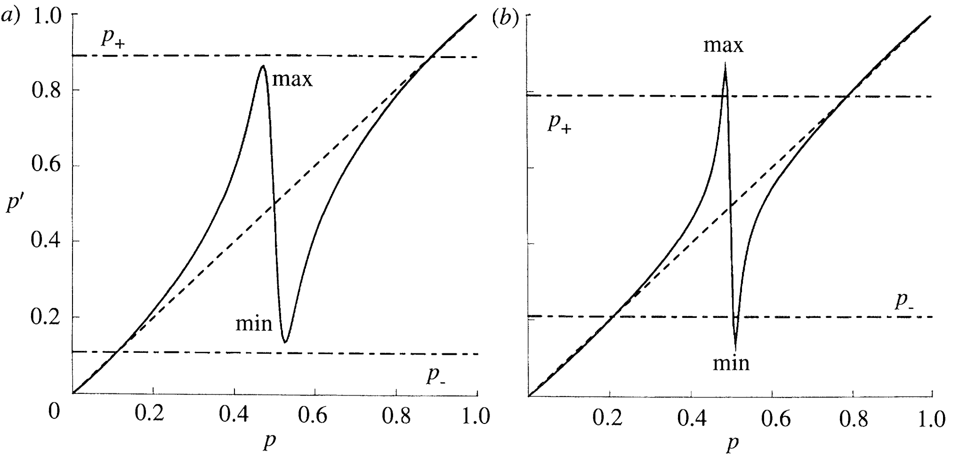

Figure 3. Graphs of the allele frequency in the next generation, $p' = p + \Delta p$, as function of the allele frequency at this generation, p. Also shown are the diagonal and the lines corresponding to the unstable polymorphic equilibria p_- and p_+. Parameter values are $\gamma = 0.9$, $C = 0$, $\beta = -1.15$ (in figure 3*a*) and $\beta = -1.05$ (in figure 3*b*).

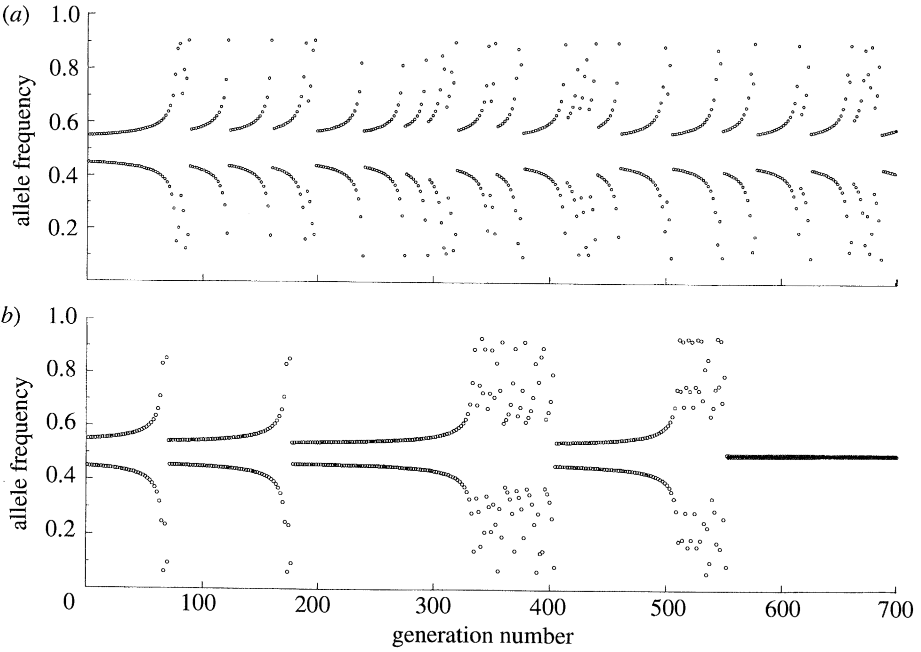

Figure 4. Dynamics of allele frequency with parameter values from the area marked SUSUS in figure 1. Both monomorphic equilibria ($p = 0$ and $p = 1$) and the polymorphic equilibrium at $p = \frac{1}{2}$ are stable to small perturbations. (*a*) $\beta = -5$, $\gamma = 0.9$, $C = 0.1$, $p(0) = 0.55$. (*b*) $\beta = -5$, $\gamma = 0.5$, $C = 0.1$, $p(0) = 0.55$.

persists for a very long time. In general, the durations of all these stages depend both on the parameter values and initial allele frequency (see below the discussion of figure 3). Both intermittency and transient chaos are known to occur in various dynamical models including the simplest nonlinear model, the single logistic map. The potential importance of these types of behaviour in population studies was emphasised in (May 1987). Recently, intermittency and transient chaos were observed in numerical studies of ecological models (Vandermeer 1993; Doebeli 1994; Hastings & Higgins 1994) described by complex systems of coupled equations. In contrast, our model is much simpler and purely frequency-dependent and allows a very simple explanation of the mechanisms underlying intermittency and transient chaos using the graphical 'cobwebbing' method (May & Oster 1976).

Figure 3 shows graphs of the allele frequency in the next generation, $p' = p + \Delta p$, as function of the allele frequency at this generation, p. Also shown are the diagonal and the lines corresponding to the unstable polymorphic equilibria p_- and p_+. First note that for p in the neighbourhood of p_- or p_+, the graph of p' lies very close to the diagonal (at which $p' = p$). That means that in these areas the changes in the allele frequency p are very small. For $p < p_-$, $p' < p$, and p slowly moves towards fixation of allele a. For $p > p_+$, $p' > p$, and the allele frequency slowly moves towards fixation of allele A. For p values slightly larger than p_-, p' is slightly larger than p, and, thus, p slowly moves towards $\frac{1}{2}$. In the neighbourhood of $p = \frac{1}{2}$, however, the dynamics are chaotic as suggested by the fact that the slope of the graph of p' at $p = \frac{1}{2}$ is smaller than -1. It takes many generations to leave the neighbourhood of p_- or p_+ and once the system has left this neighbourhood, the dynamics can abruptly become fully chaotic in the neighbourhood of $p = \frac{1}{2}$, only to get caught in the neighbourhood of p_- or p_+ again, sooner or later. Between 0 and 1, the graph of p' has a minimum, marked 'min', and a maximum, marked 'max'. During the chaotic phase the allele frequency remains between these points that represent the boundaries of the chaotic attractor. In figure 3*a* these boundaries lie close to $\frac{1}{2}$ than the unstable equilibria and the allele frequency cannot cross the values p_+ and p_-. In this case the intermittent chaos in the system (in the form similar to that one in figure 2*a*) is present forever. A different situation is described in figure 3*b*, where the boundaries of the chaotic attractor lie further from $\frac{1}{2}$ than the unstable equilibria p_- and p_+. Now the allele frequency can cross the values p_+ and p_- during the chaotic phase. Once this has happened, p slowly evolves to a fixation state (as in figure 2*b*). The situation when the boundaries of the chaotic attractor coincide exactly with the unstable equilibria is called a 'crisis' (Grebogi *et al.* 1983). In the model considered here, if $C = 0$, the crisis occurs when $\gamma \approx \beta + 2$. Note that in general the dependence of the length of chaotic transients on the system parameter is proportional to $(a - a_c)^{-b}$, where a is the parameter value, a_c is the parameter value at which the crisis occurs, and b is the 'critical exponent,' which is equal to 0.5 for a broad class of one-dimensional systems (Grebogi *et al.* 1987). The parameter values for figure 2 and figure 4 above were chosen to result in long transients. The parameters for figure 3 were chosen slightly different from those for figure 2 in order to produce a 'smoother' graph of p' as function of p.

Numerical analysis of (2) has also shown, perhaps surprisingly, that complex dynamic behaviour occurs even outside areas marked UUU and SUUUS in figure 1. In the areas marked USU and SUSUS, cycles and chaos can not only exist simultaneously with stable equilibria, but the former can have much larger domains of attraction than the latter (see figure 4). For parameter values used in computing the dynamics in figure 4, both monomorphic equilibria and the polymorphic equilibrium at $p = \frac{1}{2}$ are stable to small perturbations. In figure 4*a*, the growing regular oscillations in allele frequency are interrupted by apparently chaotic fluctuations that move p back to the neighbourhood of $\frac{1}{2}$, i.e. one observes intermittency. In figure 4*b*, the alterations between apparently deterministic behaviour and apparent chaos end at some time point with the population settling down to a polymorphic state at $p = \frac{1}{2}$, i.e. one observes transient chaos. The graphical 'cobwebbing' method can be used to understand these kinds of behaviour as well.

A necessary condition for dramatic changes in allele frequency described in figures 2 and 4 is strong (at least occasionally) selection. If selection is very weak (i.e. if the differences among coefficients α, β, γ, δ and η are very small) then the difference equation (2) can be approximated by the corresponding differential equation and the only possible outcome of the dynamics is gradual evolution towards an equilibrium. This is a further illustration of differences between qualitative and quantitative characteristics of population genetic models under strong selection and weak selection which we studied previously (Gavrilets 1993; Gavrilets & Hastings 1993, 1994*a*, *b*). Strong selection seems to be typical in natural populations (Endler 1986). This suggests that nonlinear phenomena similar to those described here may be common in the evolution of populations.

3. DISCUSSION

The results presented here illustrate several important points. The few previous models of frequency-dependent selection within a single species (reviewed in Altenberg 1991) that have been found to produce cycles and chaos, involve either fitnesses that are complex functions of the genotype frequencies or produce the complex behaviour for a very limited range of parameter values. Our first point is that simple population genetics models can have very complex dynamic behaviour (including cycles and chaos) for a broad range of parameter values (e.g. see figure 2), and the even more complex dynamical behaviour of intermittency and transient chaos for more restricted sets of parameter values near bifurcation points.

Population genetics theory has been dominated by the equilibrium approach since its inception. A general implicit assumption is that populations under selection always gradually evolve to an equilibrium. As a consequence, analysis of population genetic models has focused on conditions for existence and stability of equilibria. Our second point is that analysis of equilibria, although necessary, is still incomplete. In some situations it can even be misleading if, for example, domains of attraction of equilibria are very small and the system is never able to get there (as in figure 4*a*) or transient behaviour persists for a very long time.

The general equilibrium paradigm also manifests itself in the fact that the standard explanation of observed erratic fluctuations of allele frequencies is random genetic drift. In situations where this explanation obviously does not work (e.g. Dobzhansky 1943), explanations have typically focussed on non-constant parameter values (for example, as a consequence of climatic changes). Our third point is that selection alone (without external forces) can explain observed regular or random fluctuations in allele frequencies. Ubiquity of frequency-dependent selection make it one possible candidate; another possible explanation is density-dependent selection (Asmussen 1979). Interestingly enough, the very first model of frequency-dependent selection (Wright & Dobzhansky 1946) published almost 50 years ago was proposed in an attempt to explain complex dynamics of the genetic composition observed (Dobzhansky 1943) in some natural populations. Complex behaviour was also observed in a recent experiment (Curtsinger 1990) which looked direct at the dynamics under frequency-dependent selection. Recently developed statistical methods (Sugihara & May 1990; Ellner 1991; Ellner *et al.* 1991; Hastings *et al.* 1993) can be used to decide whether 'deterministic' or 'random' factors better explain the complex dynamics.

Substituting appropriate physical, chemical, economical or ecological terms for the genetical ones in the points we just emphasised would result in claims that have been a subject of numerous studies and discussions in the recent years. In population genetics, however, these ideas have not received the attention they deserve (Ferrier & Fox 1995).

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