CHAOS FROM LINEAR FREQUENCY-DEPENDENT SELECTION

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Abstract.—The simplest diploid model of frequency-dependent selection can generate periodic and chaotic trajectories for the allele frequency. The model is of a randomly mating, infinite diploid population with non-overlapping generations, segregating for two alleles under frequency-dependent viability selection. The fitnesses of each of the three genotypes is a linear function of the frequencies of the three genotypes. The region in the space of the coefficients that yields cycles and chaos is explored analytically and numerically. The model follows the period-doubling route to chaos as seen with logistic growth models, but includes additional phenomena such as the simultaneous stability of cycling and chaos. The general condition for cycling or chaos is that the heterozygote deleteriously effect all genotypes. The kinds of ecological interactions that could give rise to these fitness regimes producing cycling and chaos include cannibalism, predator attraction, habitat degradation, and disease transmission.

The possibilities for complex dynamical behavior from even the simplest models of population growth regulation (May, 1974, 1976) have led to the examination of conditions that produce cycling and chaos in a wide variety of models in population biology. Most studies of chaos in population dynamics have focused on the dynamics of population size. Models in which cycling or chaos is produced by natural selection acting on genetic variation have received less attention.

Asmussen (1979, 1983) and Felsenstein (1979) have examined density-dependent selection in populations exhibiting chaos, but in their models, the chaotic behavior results not from the presence of genetic variability, but from the form of density regulation that is acting. May (1979, 1983) examined a symmetric, two allele model of frequency-dependent selection in which the heterozygote fitness is the geometric mean of the homozygote fitnesses; he showed that when the homozygote's fitness decreases monotonically with its frequency, there could be at most a 2-point limit cycle, but no chaotic dynamics.

The few models of frequency-dependent selection that have been found to produce chaos either involve fitnesses that are complex functions of the genotype frequencies, with steep peaks or non-analytic points, or post hoc choices of fitness functions in order to produce chaos. The latter includes the model of Cressman (1988a), in which the quadratic logistic curve is simply grafted into the recursion, and the model of May (1979, 1983) where the fitness functions are solved to produce a system equivalent to

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the "canonical cubic" recursion: $y_{t+1} = ry_t^3 + (1-r)y_t$, which can produce cyclic and chaotic dynamics. The solution fitness functions are

$$\begin{aligned} w_{AA} &= (1 + 2rx_{aa} - rx_{Aa}/(1 + 2rx_{AA} - rx_{Aa}) \\ w_{Aa} &= 1, \text{ and} \\ w_{aa} &= 1/w_{AA} \end{aligned}$$

(where x_{AA} is the frequency of genotype AA, and so forth), and they do not suggest any obvious biological derivation for their form.

May and Anderson (1983) examined an epidemiological model in which frequency-dependent selection is generated by pathogens that are specific for host genotype. In this two-allele model, infection causes the fitness of each genotype to drop precipitously with increasing frequency, and this drop-off may be severe enough to produce cycling or chaos. May (1979, 1983) examined frequency-dependent selection models in which the heterozygote has a fitness equal to the geometric mean of the two homozygotes and the homozygote fitnesses decrease monotonically with their frequency. These models exhibited stable polymorphisms and two-point limit cycles, but no higher-period cycles or chaos.

These studies may give the impression that complex, non-linear fitness functions are necessary to produce cyclic or chaotic dynamics from frequency-dependent selection. But the general question has remained largely unexamined. Much of the classical work on frequency-dependent selection was done before it was appreciated that nonlinear difference equations could give rise to chaotic dynamics, and therefore focused on the characterization of stable equilibria (Wright, 1955; Li, 1955; Clarke and O'Donald, 1964; Cockerham, et al., 1972), which has also been the similar focus of more recent work (Slatkin, 1979; Curtsinger, 1984a,b; Lessard, 1984; Cressman, 1988b).

In this paper I re-examine the frequency-dependent selection model of Cockerham et al. (1972), in which the fitnesses are linear functions of the genotype frequencies, for the possibilities for cycling and chaos. Their model displays the simplest form of frequency-dependent selection arising from interactions between diploid organisms. Conditions are found for the linear coefficients that produce stable limit cycles of any period and also chaos. The general quality of these conditions is that the heterozygote has a strongly deleterious effect on the fitnesses of all genotypes, and that homozygotes have a milder deleterious effect on their own fitnesses. The strongly deleterious effect is the sort of result that could come out of ecological mechanisms such as cannibalism, predator attraction, habitat degradation, and disease transmission.

The frequency-dependent selection examined here produces the additional feature, described by May (1979) for cubic difference equations, that is not possible for the quadratic logistical growth models: there may be two simultaneously stable limit cycles or chaotic attractors. Furthermore, it is possible to have the simultaneous stability of both cycling and chaos.

It was early recognized that many of the behaviors of one-locus, constant selection models would not necessarily hold for frequency-dependent selection. These include "Fisher's Fundamental Theorem" that the mean fitness increases in time, and the requirement of overdominance in order to have protected polymorphisms. For frequency-dependent selection on two alleles, Wright (1955) and Li (1955) found an "adaptive topography" function which does increase in time, and is maximized at polymorphic equilibria when they are stable. However, we will see that this "evolutionary landscape" (Michod and Abugov, 1980; Curtsinger, 1984a,b) is no predictor of the cyclic and chaotic behavior reported here.

THE MODEL

We consider an infinite, randomly mating population of diploid organisms with non-overlapping generations, segregating for two alleles, A and a, at a single locus. Frequency-dependent viability selection acts on the population. Let p designate the frequency of A, and q designate the frequency of a, and x_{AA} , x_{Aa} , and x_{aa} designate the frequencies of genotypes AA, Aa, and aa. Let w_{AA} , w_{Aa} , and w_{aa} designate the fitnesses of the three genotypes AA, Aa and aa. The fitnesses are defined as linear functions of the genotype frequencies as follows (Cockerham, et al., 1972):

$$\begin{aligned} w_{AA} &= a_1 x_{AA} + b_1 x_{Aa} + c_1 x_{aa}, && (1)\\ w_{Aa} &= a_2 x_{AA} + b_2 x_{Aa} + c_2 x_{aa}, && (2)\\ w_{aa} &= a_3 x_{AA} + b_3 x_{Aa} + c_3 x_{aa}. && (3) \end{aligned}$$

After one generation, the genotype frequencies among zygotes are in Hardy-Weinberg proportions, $x_{AA} = p^2$, $x_{Aa} = 2pq$, $x_{aa} = q^2$, and the recursion on the frequency of allele A has the well known form:

$$p' = p w_A / \overline{w}, \tag{4}$$

where $w_A = p w_{AA} + q w_{Aa}$ is the marginal fitness of allele A and $\overline{w} = p^2 w_{AA} + 2pq w_{Aa} + q^2 w_{aa}$ is the mean fitness of the population. Using (1a,b,c), the mean fitness evaluates to

$$\begin{aligned} \overline{w} = \; & p^4(a_1 - 2a_2 + a_3 - 2b_1 + 4b_2 - 2b_3 + c_1 - 2c_2 + c_3)\\ & p^3(2a_2 - 2a_3 + 2b_1 - 8b_2 + 6b_3 - 2c_1 + 6c_2 - 4c_3)\\ & p^2(a_3 + 4b_2 - 6b_3 + c_1 - 6c_2 + 6c_3) + p(2b_3 + 2c_2 - 4c_3) + c_3. \end{aligned}$$

The behavior of equation (4) with frequency-independent genotypic fitnesses is well known. I retrace the analysis of Lewontin (1958) for the case of frequency-dependent selection. The behavior can be characterized by the fixed points $p' = p$ and their stability properties. The fixed points of equation (4) must satisfy

$$\Delta p = 0 \tag{5}$$

where

$$\begin{aligned} \Delta p &= p' - p = p(w_A - \overline{w})/\overline{w} = pq(w_A - w_a)/\overline{w}\\ &= pq[p w_{AA} + (q-p) w_{Aa} - q w_{aa}]/\overline{w}, && (6) \end{aligned}$$

with $w_a = pw_{Aa} + qw_{aa}$. This occurs for $p = 0, p = 1$, and for $\hat{p}$ such that $w_A = w_a$, and this $\hat{p}$ satisfies

$$\hat{p} = (w_{Aa} - w_{aa})/(2w_{Aa} - w_{AA} - w_{aa}). \tag{7}$$

Since w_A and w_a are cubic in p, there are potentially 3 fixed points (eq. [7]) on the open interval $(0, 1)$ (Cockerham, et al., 1972).

The stability of fixed points of equation (4) is determined by the values of the differential $d\Delta p/dp$ at each fixed point (Lewontin, 1958):

$$\text{if } \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} > 0, \text{ then } \hat{p} \text{ is monotonically repelling;} \tag{8}$$

$$\text{if } -1 < \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} < 0, \text{ then } \hat{p} \text{ is monotonically attracting;} \tag{9}$$

$$\text{if } -2 < \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} < -1, \text{ then } \hat{p} \text{ is attracting with decaying oscillation about it;} \tag{10}$$

$$\text{if } \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} < -2, \text{ then } \hat{p} \text{ is repelling with geometrically increasing oscillations about it;} \tag{11}$$

$$\text{if } \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} = 0 \text{ or } 2 \text{ (nongeneric cases), then } \left.\frac{d^2\Delta p}{dp^2}\right|_{\hat{p}} \text{ must be examined to determine the stability, but } |p - \hat{p}| \text{ changes at most at analgebraic rate near } \hat{p}; \tag{12}$$

$$\text{if } \left.\frac{d\Delta p}{dp}\right|_{\hat{p}} = -1, \text{ then } p \text{ approaches } \hat{p} \text{ rapidly with the possibility of decaying oscillations.} \tag{13}$$

Letting $p(t)$ be the allele frequency after t generations, a geometric rate of change means that $|p(t) - \hat{p}|/|p(0) - \hat{p}| \approx \lambda^t$, for $p(0)$ near enough to $\hat{p}$, where $\lambda > 0$, and $\lambda \neq 1$. An algebraic rate of change means that for $p(0)$ near enough to $\hat{p}$, $|p(t) - \hat{p}|/|p(0) - \hat{p}| \approx 1 + t\lambda$, where $\lambda \approx 0$.

We can ensure that a stable limit cycle or chaotic behavior exists by choosing fitness parameters that yield fixed points which are all unstable, their stability conditions falling under equations (8) or (12) (this possibility was ruled out by Lewontin (1958), due to an error in assuming that equation (12) was inconsistent with equation (4)). Under generic conditions (excluding $d\Delta p/dp|_{\hat{p}} = 0$), the signs of $d\Delta p/dp|_{\hat{p}}$ must alternate for successive fixed points $\hat{p}$. Thus stable cycling or chaos is guaranteed for a protected polymorphism in which condition (8) holds for $p = 0$ and $p = 1$, and where there is one internal fixed point $\hat{p}$, for which condition (12) holds. This situation is illustrated in figure 1. It should be noted that, were there three internal fixed points (which is the other possible generic case for a protected polymorphism, but does not arise in the

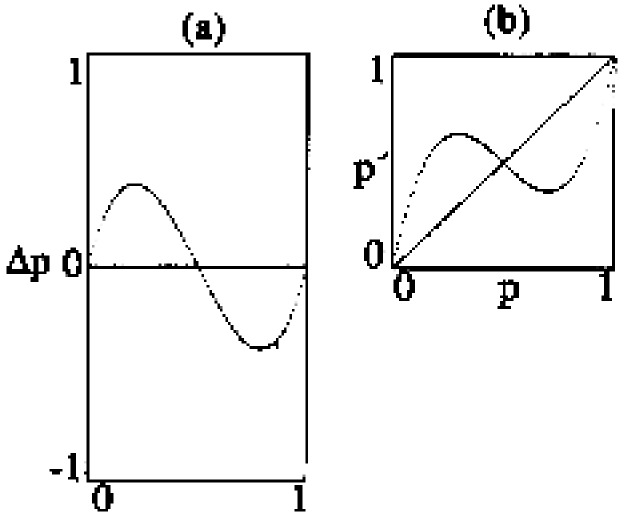

Figure 1: Conditions for cycling or chaos in the case of one internal fixed point. These graphs are for the symmetric model in which $w_{\min} = 0$ and $a_1 = 1/9$, the value at which cycling first appears. (a) $\Delta p = p' - p$ is plotted against p. (b) p' is plotted against p, which is the more familiar format used to represent such recursive mappings (May, 1976).

cases considered below), the outer pair would have to satisfy equation (12) to guarantee cycling or chaos.

These conditions for cycling or chaos readily reduce to the following inequalities on the fitness coefficients, by chain rule differentiation of equation (6):

$$\left.\frac{d\Delta p}{dp}\right|_0 = \frac{w_A - w_a}{\overline{w}} = \frac{c_2 - c_3}{c_3} > 0; \tag{14}$$

$$\left.\frac{d\Delta p}{dp}\right|_1 = \frac{w_a - w_A}{\overline{w}} = \frac{a_2 - a_1}{a_1} > 0; \tag{15}$$

$$\begin{aligned}\left.\frac{d\Delta p}{dp}\right|_{\hat{p}} &= \hat{p}\hat{q}(\mathring{w}_A - \mathring{w}_a)/w_b \\ &= \hat{p}\hat{q}[w_{AA} - 2w_{Aa} + w_{aa} + \hat{p}\,\mathring{w}_{AA}\,(\hat{q} - \hat{p})\,\mathring{w}_{Aa} + \hat{q}\,\mathring{w}_{aa}]/\overline{w} \\ &= \hat{p}\hat{q}\{\hat{p}^2[3a_1 - 4a_2 + a_3 + 2(b_2 - b_1)] \\ &\quad +2\hat{p}\hat{q}[a_2 - a_3|2(b_1 - 2b_2 + b_3) + c2 - c_1] \\ &\quad +\hat{q}^2[3c_3 - 4c_2 + c_1 + 2(b_2 - b_3)]\}/\overline{w} < -2.\end{aligned} \tag{16}$$

The terms $\mathring{w}_i$ stand for dw_i/dp. I now solve for fitness coefficients that satisfy these conditions.

RESULTS

The complete analysis requires searching the nine dimensional parameter space produced by the nine coefficients a_i, b_i, c_i in equations (1), (2), and (3) to find that region in which cycling or chaos occurs. To simplify the problem, the dimensionality of the parameter space can be reduced by making several assumptions. I examine the case where the heterozygote fitness equals the mean of the two homozygote fitnesses, thus,

$$a_2 = (a_1 + a_3)/2, \ c_2 = (c_1 + c_3)/2, \text{ and } b_2 = (b_1 + b_3)/2. \tag{17}$$

This simplifies condition (17) to:

$$\left.\frac{d\Delta p}{dp}\right|_{\hat{p}} = \hat{p}\hat{q}[\hat{p}(a_1 - a_3) + (\hat{p} - \hat{q})(b_3 - b_1) + \hat{q}(c_3 - c_1) < -2. \tag{18}$$

SYMMETRIC CASE:

A further assumption that produces a ready result is that the fitnesses be symmetric with respect to the allele labels, so that

$$c_3 = a_1, \ a_3 = c_1, \text{ and } b_1 = b_3. \tag{19}$$

Then the fitness regime is then determined by the values a_1, b_1 , and c_1 . This regime yields a fixed point $\hat{p} = 1/2$ and:

$$\left.\frac{d\Delta p}{dp}\right|_{0} = \frac{c_1 - a_1}{2a_1} \tag{20}$$

$$\left.\frac{d\Delta p}{dp}\right|_{1} = \frac{c_1 - a_1}{2a_1} \tag{21}$$

$$\left.\frac{d\Delta p}{dp}\right|_{\frac{1}{2}} = \frac{a_1 - c_1}{4\overline{w}} = \frac{a_1 - c_1}{a_1 + 2b_1 + c_1}. \tag{22}$$

To satisfy equations (14) and (15) requires that $c_1 > a_1$. Satisfying equations (14) and (15) also ensures that $\hat{p} = 1/2$ is the only internal fixed point. To satisfy equation (18) requires that

$$b_1 < (3a_1 + c_1)/4. \tag{23}$$

Alternatively, we can specify the fitness regime in terms of a_1, c_1 and w_{min}, the minimal fitness attained by the homozygotes on $0 \leq p \leq 1$. Since equation (23) ensures that the fitness functions w_{AA}, w_{Aa}, and w_{aa} are convex, this entails that

$$b_1 = w_{\text{min}} - [(a_1 - w_{\text{min}})(c_1 - w_{\text{min}}), \tag{24}$$

and

$$w_{\text{min}} = (a_1 c_1 - b_1^2)/(a_1 - 2b_1 + c_1) \tag{25}$$

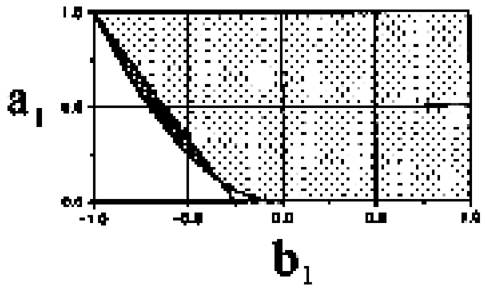

Figure 2: The region of parameter space (a_1 vs. b_1) in which cycling or chaos occurs in the symmetric model is shown by hatched area. The hatched and stippled areas comprise the region of positive fitnesses.

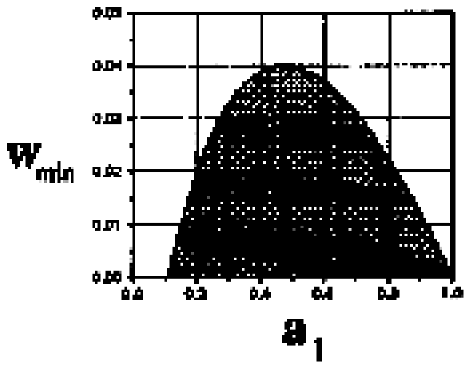

Figure 3: The region of parameter space (a_1 vs. w_{min}) in which cycling or chaos occurs in the symmetric model is shown by hatched area. $c_1 = 1$, $b_1 = w_{\text{min}} - [(a_1 - w_{\text{min}})(c_1 - w_{\text{min}})]^{1/2}$.

(solved by differentiating equation (1)). Therefore the condition (23) for cycling or chaos is equivalent to $w_{\text{min}} < (c_1 - a_1)(9a_1 - c_1)/8(5a_1 + 3c_1)$. The constraints w_{AA}, w_{Aa}, $w_{aa}0$ all be greater that 0 implies that $a_1 \geq 0$, $c_1 \geq 0$, and $b_1 \geq -(a_1c_1)^{1/2}$. Without loss of generality we can set $c_1 = 1$. These constraints are then given by the curves in figures 2 and 3.

The areas enclosed by the curves are the feasible ranges for cycling or chaos. The fitness of the homozygotes must drop below $1/25$ for some range of p in order for cycling or chaos to occur. Figure 4 shows plots of the genotypic fitnesses as functions of p for a regime that produces cycling and a regime that produces chaos.

To investigate the behavior of the allele frequency trajectories for different values of a_1 and w_{min} , we can trace the long term values of p as a function of the parameter a_1 under different choices of w_{min} . Figure 5 shows such diagrams for four different value of w_{min} .

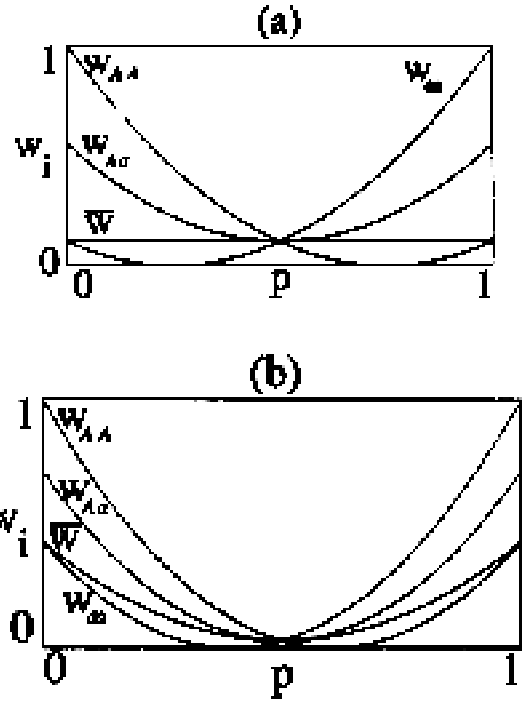

Figure 4: Fitness curves for the symmetric model. The curves for w_{AA} , w_{Aa}, w_{aa} and $\overline{w}$ are shown. (a) $w_{\text{min}} = 0$, and $a_1 = 1/9$, the value for the onset of cycling. (b) $w_{\text{min}} = 0$, and $a_1 = 0.43$, approximately the value for the onset of chaos.

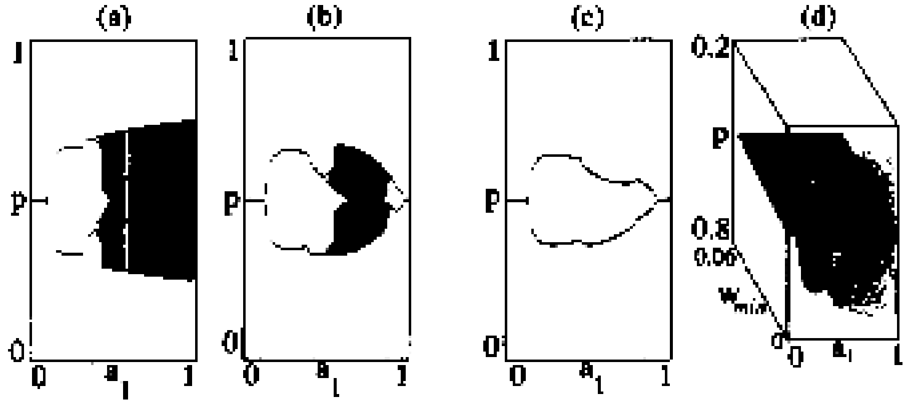

Figure 5: Bifurcation diagrams for the attractors of p in the symmetric model. At each value of a_1, the figure plots the long-term values that p settles into, be they a single stable equilibrium (seen for small a_1), limit cycles (the branched regions) or the cloud of points indicating chaos. For each value of a_1, starting with an initial p, the recursion (4) is iterated 200 times to damp out transients, and then it is iterated another 300 times with the values of p plotted. (a) $w_{\min} = 0$; (b) $w_{\min} = 0.005$; (c) $w_{\min} = 0.01$; (d) A 3-D plot of p values versus a_1 and $w_{\min}$, using fewer plotted points of p for visibility. For each graph there exists a mirror image diagram about $p = 1/2$ for the simultaneously stable second attractor.

These figures are the classical "bifurcation diagrams" that demonstrate the period-doubling route to chaos (Feigenbaum, 1978). Here, the single stable equilibrium frequency $\hat{p}$ bifurcates, as a_1 is increased, into a stable two-point limit cycle, which next bifurcates into two simultaneously stable two-point limit cycles, each of which subsequently bifurcates into limit cycles of 2^n points, with n increasing as a_1 increases, until a_1 reaches the "point of accumulation". Values of a_1 after this point lie in the "chaotic region" in which the allele frequencies can follow trajectories that have any possible period or are aperiodic, depending on a_1. As $w_{\min}$ is increased, the chaotic region for the parameter a_1 lies in an ever shortening interval until, for $w_{\min} \approx 0.008$, only cyclic behavior is observed.

The bifurcation behavior departs from the behavior of recursions such as $p' = rp(1-p)$ which have a single critical point (May, 1974) in that, instead of the period-doubling at the second bifurcation, the attractor itself bifurcates into two simultaneously stable attractors of period two (May, 1979, 1983). The two points in each limit cycle are no longer symmetric about $p = 1/2$.

For example, with $a_1 = 0.39$ and $w_{\min} = 0$, the two limit cycles consist of the pairs of points $(0.306, 0.590)$ and $(0.410, 0.694)$. The values of p that converge to one or the other limit cycle make up a domain of attraction for each cycle. Figure 6 shows how the interval $0 \leq p \leq 1$ is divided up into the two domains of attraction. The domains show a complex interleaving of intervals; as p approaches several critical points, which

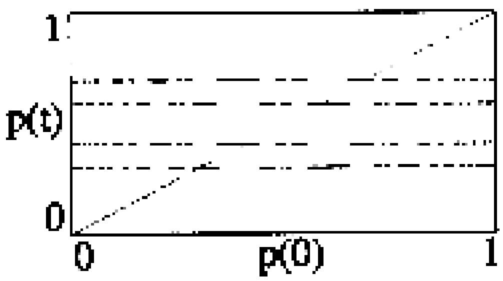

Figure 6: Domains of attraction for the two 2-point limit cycles when $w_{\text{min}} = 0$, $a_1 = 0.39$. The x-axis gives the starting value of p. The y-axis plots the values p takes on after 100 generations have passed, by which time it has settled into one of the 2-point limit cycles. Thus each point $p(0)$ maps to those two points that comprise its attractor. The diagonal $p(t) = p(0)$ is plotted for reference.

include $p = 0$, $1/2$ and 1, there appear to be infinitely many switches between the two domains of attraction, meaning that the slightest change in the initial allele frequency will result in a different final limit cycle. May (1979, 1983) considered the domains of attraction for the similar "canonical cubic" system, but did not note this property, due to an erroneous assumption that each domain of attraction consisted of two simple intervals.

For $w_{\text{min}} = 0$, two simultaneously stable attractors persist for the approximate interval $0.317 \leq a_1 \leq 0.4428$, which includes the "point of accumulation", $a_1 \approx 0.430$, for the onset of chaos. For higher values of w_{min}, the range of values of a_1 that produce cycling or chaos is reduced, and the allele frequencies of the attractors shifts, as is shown in figure 5.

TWO ASYMMETRIC CASES:

Since the assumption of exact symmetry in the above models is biologically unrealistic, it is important to examine the behavior of the model under more general conditions. Keeping $a_3 = c_1$, we can allow c_3 to vary independently, and define a measure of asymmetry: $\alpha = c_3/a_1$. Conditions (14) and (15) for cycling or chaos require that $c_1 > \alpha a_1$ and $c_1 > a - 1$, respectively. Two cases will be considered, the first in which both w_1 and w_3 attain the same minimum value, w_{min} , over p, and the second, in which $b_1 = b_3$.

Case (a): w_1 and w_3 attain the same minimum value over p.

In this case, equations (24) and (25) still hold, and b_3 is now defined as $b_3 = w_{\text{min}} - [(c_1 - w_{\text{min}})(\alpha a_1 - w_{\text{min}})]^{1/2}$. The single internal fixed point is:

$$\hat{p} = \frac{b_3 - b_1 + c_1 - \alpha a_1 - [(b_3 - b_1)^2 + (c_1 - \alpha a_1)(c_1 - a_1)]^{1/2}}{a_1(1-\alpha) + 2(b_3 - b_1)}.$$

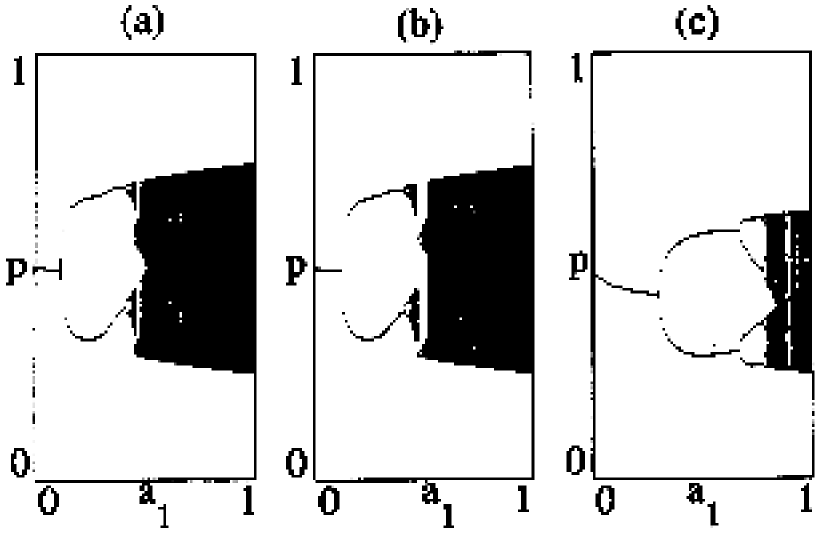

Figure 7: Bifurcation diagrams for the attractors of p in the asymmetrical model, case (a). For each, $w_{\text{min}} = 0$. The measure of asymmetry is $\alpha = c_3/a_1$. (a) $\alpha = 0.9$; (b) $\alpha = 0.8$; (c) $\alpha = 0.1$.

We let $c_1 = 1$ without loss of generality. Condition (18) produces a complex expression that does not clarify the condition. The changes in the behavior of the system as departs from 1 can be seen in the bifurcation diagrams in figure 7. As α gets smaller, there is an increase in value of a_1 at which the attractor bifurcates into two simultaneously stable attractors, and the split moves from the second to later period-doublings. It is unclear whether at some value of α there remains only one stable attractor. This can be seen in figure 8, which is a closeup of figure 7b. It can be seen that over an interval of a , there are two simultaneously stable attractors, one of which is a 2-point limit cycle while the other is chaotic. Thus, depending on the initial allele frequency, the population can either settle into a stable cycle or be chaotic. The domains of attraction for these two attractors are shown in figure 9 for the example $\alpha = 0.8$, $w_{\text{min}} = 0$, and $a_1 = 0.46$.

Case (b): $b_1 = b_3$**.**

In this case, the homozygote fitnesses may attain different minimum values:

$$w_{1\,\text{min}} = (a_1 c_1 - b_1^2)/(a_1 - 2b_1 + c_1),$$

and

$$w_{3\,\text{min}} = (\alpha a_1 c_1 - b_1^2)/(\alpha a_1 - 2b_1 + c_1).$$

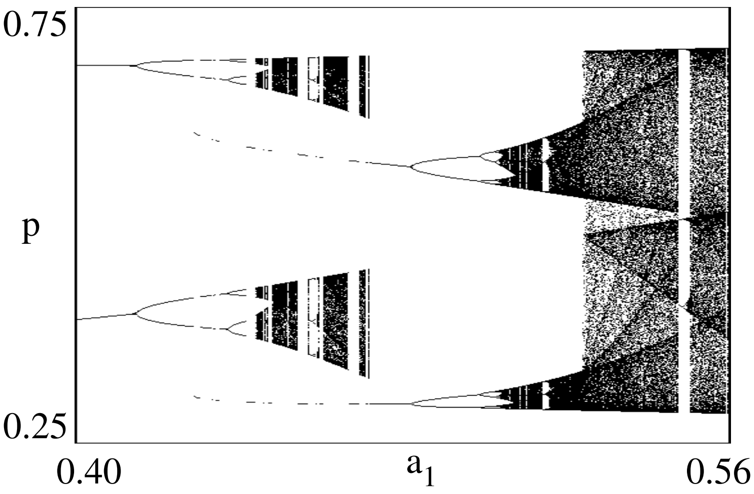

Figure 8: Closeup of figure 7b. Here, the points plotted are the attractor for the initial frequency $p = 10^{-9}$. The bifurcation into the two simultaneously stable attractors occurs through a "tangent bifurcation" (May and Oster, 1976) after a_1 has increased past the point of the second period-doubling.

Figure 9: Domains of attraction for the two simultaneously stable attractors in the asymmetrical case, in which one is a two-point cycle and the other chaotic. The x-axis gives the starting value of p. The y-axis plots the values p takes on from generation 100 to generation 200, by which time it has settled into either the 2-point limit cycle or the chaotic attractor. Each point p(0) maps either to the pair of points $(0.295, 0.577)$ of the 2-point cycle, or the two bands of points that comprise the chaotic attractor. The diagonal, $p(t) = p(0)$, is plotted for reference. $\alpha = 0.8$, $a_1 = 0.46$, and $w_{\text{min}} = 0$.

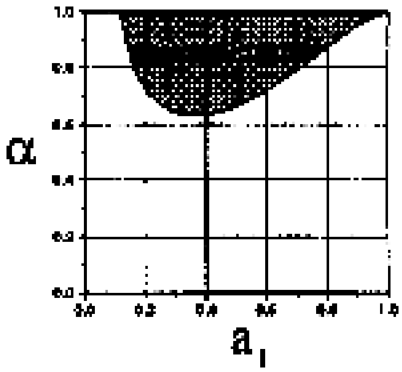

Figure 10: Hatched area shows the region of the parameter space (α vs. a_1) in which cycling or chaos can occur for the asymmetric model in which $\alpha a_1 = c_3$, $a_3 = c_1$, and $b_1 = b_3$.

We may let $\alpha < 1$ without loss of generality, and then $w_{1\,\min} < w_{3\,\min}$. Letting $c_1 = 1$, condition (18) for cycling or chaos reduces to:

$$b_1 < -[1 + a_1 + \alpha a_1(1 - 3a_1)]/4[(1 - \alpha a_1)(1 - a_1)]^{1/2}.$$

The single internal fixed point is

$$\hat{p} = \{1 - \alpha a_1 - [(1 - \alpha a_1)(1 - a_1)]^{1/2}\}/a_1(1 - \alpha).$$

Because the condition $w_{3\,\min} > 0$ entails $b_1 > -[\alpha a_1]^{1/2}$, the range of α that allows satisfaction of equation (18) is restricted to

$$\alpha > \{3a_1^2 - 6a_1 + 7 - 4(1 - a_1)[2(1 - a_1)]^{1/2}\}/a_1(9a_1^2 - 22a_1 + 17).$$

This is shown in figure 10.

The relation of the system's behavior to α is quite complex. The sample of bifurcation diagrams in figure 11 show that changes in α produce large changes in those values of a_1 that produce cycling or chaos. The system can produce simultaneous stability of cycling and chaos, as in figure 9, for at least the cases $0.98 < \alpha < 1$. It can also produce intricate superpositions of period-doubling and period-halving bifurcations, as is shown in figure 12. These figures are shown only as exemplars of the kinds of complexity that emerge from asymmetries in the fitness coefficients.

DISCUSSION

The purpose of this paper has been to examine the frequency-dependent selection model of Cockerham, et al. (1972) for coefficients in the linear fitness functions that produce cycling and chaotic trajectories of the allele frequencies over time. I have

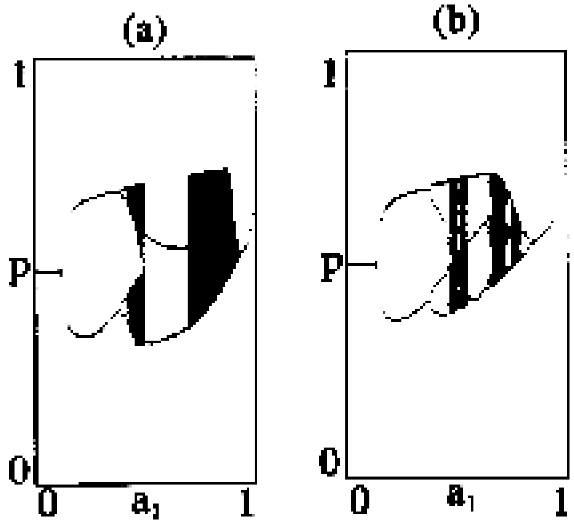

Figure 11: Bifurcation diagrams for the attractors of p in the asymmetrical model, case (b). For each, $w_{3\,\min} = 0$. The measure of asymmetry is $\alpha = c_3/a_1$. (a) $\alpha = 0.97$; (b) $\alpha = 0.9$.

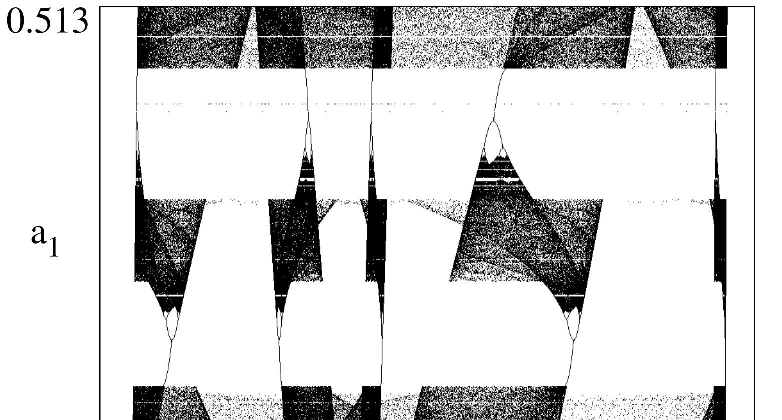

Figure 12: Closeup of figure 11b. Here, the points plotted are the attractor for the initial frequency $p = 10^{-7}$. The complex bifurcation patterns appear to involve superpositions of the generic period-doubling pattern.

considered the special case where the heterozygote fitness is the mean of the homozygote fitnesses. Under these constraints, the requirements for cycling or chaos are 1) that the heterozygote have a strongly deleterious effect on the fitnesses of all genotypes, and 2) that each homozygote have a mildly deleterious effect on its own fitness. The latter condition is the classical situation of "apostatic" frequency-dependent selection (Clarke, 1969), the result of heightened ecological competition or other negative impacts between genetically similar conspecifics. The fitness interactions can be illuminated by writing the fitness functions as

$$w_i = s_i - (s_i - a_i)x_{AA} - (s_i - b_i)x_{Aa} - (s_i - c_i)x_{aa}, \tag{26}$$

where s_i is the "base" fitness of genotype i. Under the assumptions considered here, $s_i = 1$ for $i = 1$, 2, and 3, so antagonistic effects of each genotype upon genotype i correspond to $a_i < 1$, $b_i < 1$, or $c_i < 1$, and beneficial effects correspond to $a_i > 1$, $b_i > 1$, or $c_i > 1$. Thus, apostatic selection among the homozygotes requires $a_1 < 1$ and $c_3 < 1$. This condition is what protects the polymorphism, given the assumptions in the symmetric and asymmetric cases. Figures 5, 7 and 11 show that cycling or chaos do not occur when a_1 (and hence c_3) is too low. In the symmetric case, cycling and chaos occur only for $1/9 < a_1 < 1$.

The conditions for cycling and chaos here are similar to those in May and Anderson (1983) in that the fitnesses of each genotype drop to very low values for intermediate values of the allele frequencies. Cycling or chaos occurs because, at frequencies where the deleterious heterozygote has reduced all fitnesses to very low levels, the apostatic selective advantage to the homozygotes of being rare is greatly magnified, producing an overshoot in the response of the population to displacement from the equilibrium. This association of very low fitnesses with cycling and chaos stands in contrast to the behavior of density-regulation models, in which high fecundities are required to produce cycling or chaos. Thus, the biological situations in which cycling and chaos would arise can be expected to be very different in the two cases.

The necessity that the heterozygote produce a negative effect on others' fitnesses in order to have chaos, derived under the special assumptions considered here, is likely to be a robust condition of chaos for linear fitness functions. This is the general route by which the very low fitnesses at intermediate allele frequencies can be produced when the fitness interaction coefficients between genotypes are fixed as they are here.

Interpretation of the fitness regimes. For field biologists wishing to discern whether fluctuations in population sizes or gene frequencies are cyclic, chaotic, externally driven, or stochastic, a variety of statistical approaches have been explored. These include estimates of the attractor dimension for the system, non-linear forecasting (Sugihara and May, 1990), and estimates of Lyapunov exponents. Ellner (1991) gives a critical review of these techniques, and Ellner et al. (1991) and Nychka et al. (1991) have developed methods of estimating Lyapunov exponents that overcome some of the problems of other methods and offer several advantages. All methods involve trade-offs between sample sizes, statistical power, and computational intensity, and still pose substantial practical problems for field studies.

In the cases of chaos and cycling studied here, interpretation of the fitness regimes can at least point to situations more likely to produce chaos or cycling. Biological conditions that would give rise to the strongly deleterious effect of the heterozygote that is required for cycling and chaos would involve, first, a very antagonistic mode of ecological interaction within the species, and second, a strong overdominance for this mode of interaction.

There are several ecological phenomena in which a phenotype could have a dramatically deleterious effect on the fitness of all genotypes. One of these could be cannibalism. Cannibalism is observed among a wide enough range of taxa (having been reported in protozoa, planaria, rotifers, snails, copepods, centipedes, mites, insects, fish, anurans, birds, and mammals (Fox, 1975)) to make it of interest in this context. Genetic variability for cannibalism has been documented in flour beetles, flatworms, rotifers, spadefoot toads, poeciliid fishes (Fox, 1975) and the moth Heliothis virescens (Gould, 1986).

Using the alternative expression for the fitness functions (26), the interpretation of b as a measure of the heterozygote's cannibalism would mean that, for the symmetric case, each heterozygote would devour $1-b_1$ other individuals on average before reproduction. So, for example, when $a_1 = 0.5$, $c_1 = 1$, a heterozygote phenotype for consuming an average of 1.7 other individuals ($b_1 = 0.7$) would produce chaotic dynamics.

The genetics of cannibalism in the flour beetle (genus Tribolium) has been the subject of several studies. A number of the features that produce chaos in the model studied here are found in the study of cannibalism by Stevens (1989): first, there is genetic variation producing large differences in the levels of cannibalism; second, as an analog to the variation in the mean fitnesses described here, Stevens found ten-fold differences in the equilibrium population sizes of the different strains of Tribolium, which correlated inversely with the amount of cannibalism. In fact, if cannibalism is sufficiently great, it can lead to population extinction (Park et al., 1964). Third, as in the current model, the selective advantage accruing to cannibalistic behavior was slight or nonexistent (Stevens, 1989). However, Stevens's results depart from the model considered here in several ways: first, the genetic variation for cannibalism was found to be polygenic; second, there is only small dominance deviance from additive effects, rather than the strong overdominance investigated here; and third, the populations described by Stevens have interaction between several overlapping demographic stages, while the model here is of strictly non-overlapping, single cohort generations. Therefore, as is usually the case for simple models, there is no ready biological situation documented for which this model directly applies.

Several other ecological interactions can be considered as interpretations of the fitness effects described here. The strongly antagonistic effect required of the heterozygote could be produced by predator attraction, habitat degradation, or disease transmission. Predator attraction has been studied as a likely cause of frequency-dependent selection and found to have diverse modes of action in this regard (Allen, 1988). In the models examined here, the cyclic or chaotic regimes could result from the production of a non-cryptic heterozygote phenotype from two cryptic homozygote phenotypes. The condition that all genotypes be negatively affected by the heterozygote requires a situation in which predators, once attracted to a group of the organisms, are able to prey on them equally. Alternatively, for a population in which the frequency of palatable versus unpalatable individuals determines the rate of predation on the entire population, a palatable heterozygote among two unpalatable homozygotes could produce the conditions for cycling described here.

Habitat degradation as a form of deleterious intraspecific interaction could result from any number of causes. For organisms that relied on hosts for some part of their lifecycle, a heterozygote that caused disease or other mortality or unsuitability in the host could give rise to the fitness regimes that produce cycling and chaos. A variety of mechanisms can be described in which the heterozygote would cause increased disease mortality for all the genotypes in the population. This might include a "Typhoid Mary" phenotype, in which a certain percentage of the heterozygotes were the source of disease transmission to the entire population. Alternatively, some behavior or byproduct of the heterozygote could attract disease bearing vectors to the population, for example, through behavior that contaminated population water supplies, or secondary compounds that attracted disease bearing organisms to the population. For these scenarios to produce the linear form of frequency-dependence, any population dynamics of the disease agent must be assumed to be of negligible effect.

The area of the parameter space that produces cycling or chaos is small, but not negligible. The smallness of the area is due largely to the requirement that the fitnesses be linear functions of the genotype frequencies, which was adopted to demonstrate that complex behavior is possible for the simplest kind of frequency-dependence. The parameter boundary b a c could be extended if we allowed the fitnesses w_{AA}, w_{Aa}, and w_{aa} to simply be truncated at 0 for those frequencies at which they would be negative under the forms in (1a,b,c). This would increase the feasible space for cycling or chaos. Truncation of the fitnesses at 0 would also be the natural expectation for situations in which non-Hardy-Weinberg proportions would make fitnesses negative under the forms (1a,b,c). The general requirement for cycling or chaos is that fitnesses of each homozygote drop to very low values for some allele frequency, as figure 3 shows.

There is no "adaptive topography". In considering how Fisher's "Fundamental Theorem" would apply to populations segregating for multiple loci, Wright developed the idea of the "adaptive topography", in which the space of genotype frequencies was conceived of as a field upon which was defined a fitness function, and the population would tend to move to peaks of this fitness function (Wright, 1969). Models of frequency-dependent selection presented a problem for this heuristic in that fitness peaks might not coincide with stable equilibria. Wright (1955) and Li (1955) produced a "fitness function" for the case of two alleles, recently elaborated upon by Curtsinger (1984a,b) and Michod and Abugov (1980), which preserved the "adaptive topography" property, in that it was maximized at any stable equilibrium. The function is the sum over genotypes i of the indefinite integrals

$$f(p) = \sum_i \int w_i \frac{\partial x_i}{\partial p} dp.$$

Substitution from equation (6) readily shows that

$$f(p) = 2 \int \frac{\Delta p \overline{w}}{pq} dp.$$

For any stable equilibrium point $\hat{p}$, p is positive for $p\hat{p}$ and negative for $p\hat{p}$, therefore $f(p)$ is maximized at $\hat{p}$ by definition. It should be noted that f(p) has no ready biological interpretation.

Wright (1969) recognized that the existence of this function was an artifact of the two-allele model, which made the gene-frequency "field" one-dimensional. He describes cases of frequency-dependent selection with three alleles that produce stable cycling, and hence rule out, in principle, the general existence of a Lyapunov "fitness function".

Wright does not address the possibility of cycling or chaos in the two-allele model which would preclude the maximization of the "fitness function" by the population. Curtsinger (1984b) explored this possibility, and produced conditions on polynomial fitness functions that are sufficient, by excluding cyclic or chaotic behavior, to assure the existence of the "adaptive topography" (i.e. a Lyapunov function) in the two-allele case. So in the cases of cycling and chaos described here, the polynomial fitness functions fall outside these conditions. Although $f(p)$ may still reach a local maximum for fixed points $\hat{p}$, this property no longer reflects the dynamics of the system, so there is no sense in which $f(p)$ can serve as an "adaptive topography". Moreover, the topology of chaotic attractors rules out the use of any other continuous function as a general Lyapunov function for the system.

In summary, these results demonstrate that the complex dynamical behavior reported for models of population density regulation can also be found in a simple model of pure frequency-dependent selection. They are most likely to be found in ecological situations in which certain phenotypes can negatively affect the fitnesses of all genotypes in the population. Complex dynamical behavior has been found for more complicated population genetic models (e.g. Hastings, 1981; May and Anderson, 1983), but the goal here has been to show that it can occur in the simplest as well, for conditions that have a feasible ecological interpretation.

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