Density Dependent Selection 1: A Stable Feasible Equilibrium May not be Attainable†

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The joint evolution of gene frequency \( p \) and population size \( N \) is studied. It is shown that when the genotypic fitnesses are logistic functions of the population size, sets of initial states exist which lead to bizarre behavior. Even though equilibria may be locally stable, these sets of initial conditions eventually produce negative fitnesses. Alternative models are discussed as are some general properties of the mean fitness.

1. Introduction

One of the most natural intersections of ecological and population genetic theories lies in the study of density dependent natural selection. The fitness differences among the phenotypes or genotypes may be viewed as the result of different responses to ecological pressures. These different responses may then be cast in the form of the density dependent effects in classical ecological models. The desirability of a theory accommodating both genetic differences and ecological pressures has been pointed out by many authors (see e.g., Birch, 1960; Turner & Williamson, 1968).

Four recent studies by Roughgarden (1971), Charlesworth (1971), Anderson (1971) and Clarke (1972) have taken a classical one locus, two allele population genetic model, and allowed the fitnesses to be functions of the changing population size. Thus, the deterministic changes over time of the gene frequency and population size were interdependent. Density dependence was incorporated in the choice of the genotypic (Roughgarden, Charlesworth & Anderson) or phenotypic (Clarke) fitnesses as decreasing functions of the population size.

In Clarke's model the density dependent fitnesses of the phenotypes were linear fractional functions of the population size. A complete equilibrium treatment, including local stability analysis was possible. Roughgarden (1971)

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assumed that the genotypic fitnesses were logistic functions of the population size. He produced several numerical iterations of the two-dimensional recurrence system in the gene frequency and population size. Charlesworth followed this with a stability analysis of the equilibria in Roughgarden's model. The major conclusions from this analysis were that the conditions, (1) the mean intrinsic rate of increase at equilibrium of the population \( \hat{r} \) is positive and less than 2, and (2) the carrying capacity \( K_{12} \) of the heterozygote genotype is larger than \( K_{11} \) and \( K_{22} \), those of the two homozygotes, are necessary for a stable non-trivial gene frequency-population size equilibrium to exist. Charlesworth also gave conditions under which one or other allele will be eliminated. Anderson (1971) independently suggested the same model as Roughgarden and obtained a number of the results that Charlesworth reported. The logistic formulation was used by Templeton (1974) in his study of density dependent selection with parthenogenesis.

The appropriateness of the various forms of density dependence has been subject to some debate. Clarke pointed out that density dependent selective values cannot be conveniently represented by Lotka-Volterra functions since these may become negative. Anderson recognized the difficulty but ignored it in his analysis. Nevertheless under the conditions described by Anderson & Charlesworth, the various equilibria of the logistic model are feasible and locally stable. We use feasible here in the sense of Roberts (1974) to mean that at equilibrium the population size is a non-negative number, and the gene frequency is a number in \([0, 1]\). An equilibrium point is locally stable if small perturbations from the equilibrium result in return to the equilibrium. We would like to introduce the term attainable. A feasible locally stable equilibrium is attainable from a set \( S \) of initial values (population sizes and gene frequencies) if, on iteration of the time dependent variables, these pass through only biologically realistic values on their way to the equilibrium. That is, the population size remains positive and the gene frequencies stay between 0 and 1. This definition allows that an equilibrium be attainable from some initial values, but not from others.

The fact that the discrete time logistic equation may have anomalous behavior has been pointed out by May (1974). The iteration of this equation is a particular case of the iteration of general quadratic recursion systems, which was studied in some detail by Chaundy & Phillips (1936). As applied to the classical logistic equation

\[
N_{t+1} = N_t \left[ 1 + r \left( 1 - \frac{N_t}{K} \right) \right]
\]  

with intrinsic rate of increase \( r \) and carrying capacity \( K \), their theorems demonstrate the dependence of the stability behavior on \( r \). For example, if
\( r > 3, N_t \to -\infty \) no matter what the starting population size is. This is a striking example but other anomalies were discussed by May (1974). It is perhaps not surprising that there may also be problems in the interpretation of logistic selection in genetic models.

In this note we shall first be concerned with these difficulties, not as they affect stability but as they relate to whether an equilibrium can be achieved from a given starting population. We do this by a series of examples whose purpose is first to illustrate the particular shortcomings of the logistic model. We then point out the need to carefully scrutinize the total behavior of a given mathematical model, not only the equilibrium behavior, in order to assess how well it portrays the underlying biological phenomenon.

In order to avoid the above difficulty we suggest several alternative models. The first is simply the logistic truncated at zero. A second possibility is the exponential version of the logistic model. This, it seems to us, retains most of the desirable features of the usual logistic form (see e.g. May, 1974) while avoiding the problem of negative fitnesses. May, Hassell, Conway & Southwood (1974) discuss empirical evidence for such exponential population regulation models. Of course there are many other choices. Ayala et al. (1973) claim, on the basis of their experiments with different species of \textit{Drosophila}, that the logistic system (in the guise of the Lotka–Volterra competition equations) is inadequate. Schoener (1973) discusses a hyperbolic model for population growth and claims that evidence from laboratory studies validate this formulation. Our third alternative, then, is a form of hyperbolic fitness.

Since there are so many possible forms for the fitnesses, it is of some importance to aim, as Charlesworth did, towards a theory which, on the one hand, is robust to changes in the functional form of the fitnesses and which, on the other, makes a qualitative classification among models. To this end we present a little theory relevant to more general density dependent fitness schemes and including, as an appendix, a proof of a general maximization principle for this class of models.

2. Logistic Difficulties

We consider the logistic model as studied by Charlesworth. The genotypes \( A_iA_j \ (i, j = 1, 2) \) have intrinsic rates of increase \( r_{ij} \) and carrying capacities \( K_{ij} \). The population size at time \( t \) is \( N_t \), and the gene frequency of \( A_1 \) at time \( t \) is \( p_t \). The fitness of genotype \( A_iA_j \) at time \( t \) is

\[
\psi_i^{(t)} = 1 + r_{ij} \left(1 - \frac{N_t}{K_{ij}}\right). \tag{2}
\]
The marginal fitness of allele $A_i$ is

$$w_i^{(t)} = p_i w_1^{(t)} + (1 - p_i) w_2^{(t)}, \quad (i = 1, 2)$$

(3)

and the mean fitness of the population is

$$\bar{w}^{(t)} = p_1 w_1^{(t)} + (1 - p_1) w_2^{(t)}.$$  

(4)

The basic recursion system in $N$ and $p$ is then

$$p_{t+1} = p_t w_1^{(t)}/\bar{w}^{(t)}$$

(5)

$$N_{t+1} = \bar{w}^{(t)} N_t.$$  

(6)

One obvious problem with this formulation is the meaning to be ascribed to $K_{12}$, the carrying capacity of the heterozygote.

The main problem with which we shall be concerned is that with three carrying capacities, the population size may grow so large that one or more genotype has a negative fitness value. In fact we shall see that this causes very bizarre behavior. It is possible for the model to be well defined initially, but in the course of the evolution, to encounter negative population sizes and negative gene frequencies which prevent the locally stable equilibrium from being reached.

In all of our discussion we shall set $r_{11} = r_{12} = r_{22} = r$. It will be obvious that the qualitative conclusions are not really affected by this and the assumption simplifies many calculations. Let us first consider the case $K_{12} > K_{11}, K_{22}$, i.e. "overdominance" in carrying capacity. The equilibrium is given by

$$N = (\hat{W}_{22} - \hat{W}_{12})/(\hat{W}_{11} + \hat{W}_{22} - 2\hat{W}_{12})$$

$$p = (1/K_{12} - 1/K_{22})/(2/K_{12} - 1/K_{11} - 1/K_{22})$$

(7)

$$\hat{N} = 1/\{\hat{p}^2/K_{11} + 2\hat{p}(1 - \hat{p})/K_{12} + (1 - \hat{p})^2/K_{22}\}$$

$$= (2/K_{12} - 1/K_{11} - 1/K_{22})/(1/K_{11}^2 - 1/K_{11}K_{22}).$$

(8)

This is locally stable if

$$0 < r < 2$$

(9)

and

$$-2 < \hat{p}(\hat{W}_{11} - \hat{W}_{12}) < 0.$$  

(10)

At equilibrium, then, $\hat{W}_{11}$ is negative for instance if $\hat{N} > (1 + r)K_{11}/r$ or

$$K_{11}\{1/K_{11}^2 - 1/K_{11}K_{22}\}/\{2/K_{12} - 1/K_{11} - 1/K_{22}\} < r/(1 + r).$$

A particular numerical example is specified by

$$E_1: \{r = 1.5, K_{11} = 100, K_{12} = 300, K_{22} = 150\}.$$  

In this case $\{\hat{p} = 1/3, \hat{N} = 180\}$ is a locally stable feasible equilibrium with $\hat{W}_{11} = -0.2$. In Table 1 a typical set of trajectories for this parameter set is
DENSITY DEPENDENT SELECTION

**Table 1**

\( r = 1.5, K_{11} = 100, K_{12} = 300, K_{22} = 150 \)

<table>
<thead>
<tr>
<th>( p_0 )</th>
<th>( N_0 )</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>10</td>
<td>( p_t ) increases to ( \tilde{\phi} = 1/3 )</td>
</tr>
<tr>
<td>0.3</td>
<td>10</td>
<td>( N_t ) oscillates to ( \tilde{N} = 180 )</td>
</tr>
</tbody>
</table>
| 0.6 | 10 | \( p_t \) decreases to \( \tilde{\phi} = 1/3 \)
| | | \( N_t \) oscillates to \( \tilde{N} = 180 \) |

Described. Note that \( N_t \) may become larger than \( \tilde{N} \) and may oscillate into equilibrium. Note also that if we take \( r = 1.0 \) rather than 1.5 in \( E_1 \), so that \( \tilde{w}_{11} > 0 \) this oscillation does not occur (see Table 2).

For all starting conditions of example \( E_1 \) listed in Table 1, the analytical equilibrium, equations (7) and (8) is achieved via a biologically realizable trajectory. This is the case even though \( \tilde{w}_{11} < 0 \). Unfortunately, because of this negative fitness, there are regions from which trajectories may pass through biologically inadmissible values. Negative fitness values can force the trajectory into negative gene frequencies, gene frequencies greater than unity, or negative population sizes. It is therefore possible that an equilibrium is feasible and locally stable, but that there exist a set of initial gene frequencies and population sizes from which this equilibrium is not attainable.

**Table 2**

\( r = 1.0, K_{11} = 100, K_{12} = 300, K_{22} = 150 \)

<table>
<thead>
<tr>
<th>( p_0 )</th>
<th>( N_0 )</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>10</td>
<td>( p_t, N_t ) increase to ( \tilde{\phi}, \tilde{N} )</td>
</tr>
<tr>
<td>0.3</td>
<td>10</td>
<td>( p_t ) decreases to ( \tilde{\phi} = 1/3 )</td>
</tr>
<tr>
<td>0.6</td>
<td>10</td>
<td>( N_t ) increases to ( \tilde{N} = 180 )</td>
</tr>
</tbody>
</table>

To demonstrate the various possibilities consider again the overdominant case \( K_{12} > K_{11}, K_{22} \) with \( 0 < r < 2 \). If
\[
\tilde{w}_1^{(n)} < 0 \quad \text{and} \quad \tilde{w}_2^{(n)} > 0 \tag{11}
\]
then obviously \( p_{t+1} < 0 \). For equation (11) we must have
\[
(1+r)r\left\{p_t/K_{11} + (1-p_t)/K_{12}\right\} < N_t < \left(1+r\right)r\left\{p_t^2/K_{11} + 2p_t(1-p_t)/K_{12} + (1-p_t)^2/K_{22}\right\} \tag{12}
\]
which entails that

$$p_t > \beta = \frac{1}{K_{12} - 1/K_{22}}/\left(\frac{2}{K_{12} - 1/K_{11} - 2/K_{22}}\right).$$

(13)

Similarly $p_{t+1} < 0$ if

$$w_1^{(t)} > 0 \text{ and } \bar{w}^{(t)} < 0.$$  

(14)

This is equivalent to

$$(1 + r)/r\{p_t^2/K_{11} + 2p_t(1 - p_t)/K_{12} + (1 - p_t)^2/K_{22}\} < N_t,$$

$$< (1 + r)/r\{p_t/K_{11} + (1 - p_t)/K_{12}\}$$

(15)

for which $p_t < \beta$ is necessary. In other words, if $N_t$ and $p_t$ satisfy equation (12) for any generation $t$, then in the next generation the gene frequency $p_{t+1}$ is negative. If $N_t$ and $p_t$ satisfy equation (15) then not only is $p_{t+1}$ negative, but so is the next population size $N_{t+1}$.

Once a negative population size is achieved in this model, it must stay negative, and in fact proceed to $-\infty$. Note that it is possible for the region (12) to include $N_t$ values less than $K_{12}$ with equation (9) holding if

$$2 > r > [p_t(K_{12}/K_{11} - 1)]^{-1}.$$  

(16)

The same can be said of region (15) if

$$2 > r > [p_t^2K_{12}/K_{11} + 2p_t(1 - p_t) + (1 - p_t)^2K_{12}/K_{22} - 1]^{-1}.$$  

(17)

The regions (12) and (15) provide two examples of bizarre behavior. Starting from region (15) $N_{t+1}$ is negative in the next generation and as a result the population size remains negative and tends to $-\infty$. The gene frequency also appears to remain negative but approaches zero. In contrast, although the gene frequencies of a trajectory originating in equation (12) are also negative at first, the gene frequency appears to suddenly jump completely across $[0, 1]$ to a value larger than one; at the same time the population size becomes negative. In the following generations the population size decreases to $-\infty$, while the gene frequency appears to approach one. Regions analogous to (12) and (15) may be set up for $p_{t+1} > 1$, which require either $w_2^{(t)} < 0$ and $\bar{w}^{(t)} > 0$ or $w_2^{(t)} > 0$ and $\bar{w}^{(t)} < 0$. These regions produce similar unacceptable behavior. The four regions are represented schematically in Fig. 1.

Numerical work indicates that trajectories originating below these regions proceed through biologically admissible values to the locally stable interior equilibrium. In other words, this equilibrium is attainable for initial gene frequencies and population sizes in the region below regions I–IV of Fig. 1. On the other hand, for trajectories starting in any of the four latter regions, the equilibrium is not attainable; in fact, it cannot be reached. This is the case even though the equilibrium is locally stable according to the criteria of Anderson (1971) or Charlesworth (1971). Tables 3 and 4 illustrate some typical trajectories for various initial conditions.
FIG. 1. The four regions from which $\left( \hat{\rho}, \hat{N} \right)$ is not attainable. Region I. Here $w_2^{(1)} < 0$ and $\bar{w}_i^{(1)} > 0$. Once the population enters this region, the gene frequency becomes greater than one in the next generation, although the population size remains positive. In succeeding generations, the gene frequency increases, then suddenly jumps to a negative value. At the same time the population size goes negative. Afterwards the gene frequency appears to increase toward zero, while the population size decreases to $-\infty$. Region II. Here $w_1^{(1)} > 0$ and $\bar{w}_i^{(1)} < 0$ corresponding to (15). In the next generation both the gene frequency and population size will be negative. The gene frequency then appears to increase toward zero while the population size decreases to $-\infty$. Region III. Here $w_1^{(1)} < 0$ and $\bar{w}_i^{(1)} > 0$ corresponding to equation (12). In the next generation $p_{t+1}$ will be negative, $N_{t+1}$ positive. The gene frequency decreases and remains negative for a while, then suddenly takes on a value greater than one. Simultaneously, the population size becomes negative. Thereafter, the gene frequency decreases toward one while the population size decreases to $-\infty$. Region IV. Here $w_2^{(1)} > 0$ and $\bar{w}_i^{(1)} < 0$. In the next generation the gene frequency is larger than one and the population size is negative. The gene frequency then appears to approach one, while the population size decreases to $-\infty$.

It should be emphasized that similar odd behavior is possible from an initial $N_0 < K_{12}$ even when all $\bar{w}_{ij} > 0$ (see Table 4). In a similar fashion, it is possible to derive conditions under which parts of regions I–IV of Fig. 1 lie below $\hat{N}$ and in fact intersect the region, $\bar{w}(\hat{N}) > 1$. An example is given by

$$E_2: \{ r = 1.75, K_{11} = 100, K_{12} = 800, K_{22} = 600 \}.$$  

Trajectories for $E_2$ exhibit the same qualitative behavior described above.

All of these examples occur because the points $\{ \hat{\rho} = 0, \hat{N} = -\infty \}$ and $\{ \hat{\rho} = 1, \hat{N} = -\infty \}$ are locally stable equilibria for the logistic model even
Table 3

\[ r = 1.5, K_{11} = 100, K_{12} = 300, K_{22} = 150 \]

<table>
<thead>
<tr>
<th>( p_0 )</th>
<th>( N_0 )</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>100</td>
<td>( p_t ) increases to ( \hat{p} = 1/3 ), ( N_t ) oscillates to ( \hat{N} = 180 )</td>
</tr>
<tr>
<td>200</td>
<td></td>
<td>( p_t ) increases to ( \hat{p} = 1/3 ), ( N_t ) oscillates to ( \hat{N} = 180 )</td>
</tr>
<tr>
<td>240</td>
<td></td>
<td>( p_t : \hat{N} = 0 ), ( \hat{N} = K_{22} = 150 )</td>
</tr>
<tr>
<td>280</td>
<td></td>
<td>( p_t ) increases to ( \hat{p} = 1/3 ), ( N_t ) oscillates to ( \hat{N} = 180 )</td>
</tr>
<tr>
<td>300</td>
<td></td>
<td>( p_t ) increases to ( \hat{p} = 1/3 ), ( N_t ) oscillates to ( \hat{N} = 180 )</td>
</tr>
</tbody>
</table>

though \( 0 < r < 2 \), and there exists a feasible locally stable finite equilibrium. The results also show that it is possible to go to fixation in this case. It is clear that the local stability of \( (\hat{p}, \hat{N}) \) does not tell the full biological story, and that a wide range of anomalous behavior is possible with overdominant logistic fitnesses.

3. Boundary Equilibria

The difficulties demonstrated in the previous section are not restricted to the case where the interior, polymorphic equilibrium is locally stable. Consider for example the case where fixation at \( \hat{p} = 0, \hat{N} = K_{22} \) is expected
<table>
<thead>
<tr>
<th>$p_t$</th>
<th>$N_t$</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>100</td>
<td>$p_t$, $N_t$ increase to $\hat{\beta} = \frac{1}{3}$, $\hat{N} = 180$</td>
</tr>
<tr>
<td>200</td>
<td></td>
<td>$p_t$ increases to $\hat{\beta} = \frac{1}{3}$, $N_t$</td>
</tr>
<tr>
<td>280</td>
<td></td>
<td>decreases in 1st generation, then increases to $\hat{N} = 180$.</td>
</tr>
<tr>
<td>790</td>
<td></td>
<td>$p_t &gt; \hat{\beta} = \frac{1}{3}$. Thereafter $p_t$, $N_t$ increases to $\hat{N} = 180$, having decreased in 1st generation.</td>
</tr>
<tr>
<td>0.7</td>
<td>100</td>
<td>$p_t$ decreases to $\hat{\beta} = \frac{1}{3}$, $N_t$ increases to $\hat{N} = 180$.</td>
</tr>
<tr>
<td>200</td>
<td></td>
<td>$p_t$ decreases to $\hat{\beta} = \frac{1}{3}$.</td>
</tr>
<tr>
<td>220</td>
<td></td>
<td>$N_t$ decreases in 1st generation, then increases to $\hat{N} = 180$.</td>
</tr>
<tr>
<td>230</td>
<td></td>
<td>$p_t &lt; \hat{\beta} = \frac{1}{3}$. Then $p_t$ increases to $\hat{\beta}$, $N_t$ increases to $\hat{N} = 180$.</td>
</tr>
<tr>
<td>240</td>
<td></td>
<td>$p_t = 0$, $N_t = K_{22}$. $p_t$ remains</td>
</tr>
<tr>
<td>250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>260</td>
<td></td>
<td>$p_t &lt; 0$. Initially $p_t$ decreases, becoming more negative.</td>
</tr>
<tr>
<td>280</td>
<td></td>
<td>Abruptly, $p_t &gt; 1$, $N_t &lt; 0$. Thereafter $p_t$ decreases toward 1, $N_t$ to $-\infty$.</td>
</tr>
<tr>
<td>290</td>
<td></td>
<td>$p_t &gt; 1$, $N_t &lt; 0$. Then $p_t$ decreases</td>
</tr>
<tr>
<td>300</td>
<td></td>
<td>toward 1, $N_t$ decreases to $-r$.</td>
</tr>
</tbody>
</table>

from the stability criteria. Suppose that $K_{12} < K_{11}, K_{22}$ so that $\hat{\beta}$ from (7) is unstable. Then $p_{t+1}$ will be negative if $w_i^{(t)} < 0$ and $w_i^{(t)} > 0$ or $w_i^{(t)} > 0$ and $w_i^{(t)} < 0$. These two regions are represented schematically by the hatched areas in Fig. 2 and are given by

$$p_t < \hat{\beta}$$

$$(1+r)/r\left(p_t/K_{11} + (1-p_t)/K_{12}\right) < N_t <
(1+r)/r\left(p_t^2/K_{11} + 2p_t(1-p_t)/K_{12} + (1-p_t)^2/K_{22}\right)$$

(18)
on the left, and

$$p_t > \hat{\beta}$$

$$(1+r)/r\left(p_t^2/K_{11} + 2p_t(1-p_t)/K_{12} + (1-p_t)^2/K_{22}\right) < N_t <
(1+r)/r\left(p_t/K_{11} + (1-p_t)/K_{12}\right).$$

(19)
on the right of Fig. 2.

Now the boundary equilibrium $\hat{\beta} = 0$, $\hat{N} = K_{22}$ is locally stable if $K_{12} < K_{22} < (2+r)K_{12}/r$, $0 < r < 2$. If these inequalities hold, and $p_t$ and
FIG. 2. Interference with fixation. The vertically lined region corresponds to equation (18). Once a population enters this region the gene frequency in the next generation will be negative. Note that if \( \frac{1+r}{r} K_{12} < K_{22} \) this region surrounds the boundary equilibrium \( \hat{p} = 0, \hat{N} = K_{22} \). The horizontally lined region corresponds to equation (19). From this region both the gene frequency and the population size will be negative in the next generation.

\( N_t \) are in either of the regions (18) or (19) then \( p_{t+1} \) and/or \( N_{t+1} \) are inadmissible. In particular, if in addition \( K_{22} < (1+r)K_{12}/r \), the locally stable fixation point \( \hat{p} = 0, \hat{N} = K_{22} \) is actually surrounded by the region (18) from which fixation is not attainable (see Fig. 2). Table 5 demonstrates that there may be trajectories which converge to \( \hat{p} = 0 \) via damped oscillations between positive and negative gene frequencies. The same qualitative results hold when \( K_{12} \) is intermediate in value.

None of Roughgarden's or Anderson's numerical examples showed the aberrations we have seen above. But one, Roughgarden's last example in his Fig. 10, narrowly misses. The example with \( r_{ij} \) all different is \( E_3: \{r_{11} = 0.8, r_{12} = 0.7, r_{22} = 0.6; K_{11} = 8000, K_{12} = 5000, K_{22} = 12000\} \). By a procedure similar to that we have used above, it can be shown that in this case if \( r_{12} \) had been chosen larger than \( \frac{1}{\hat{p}} \approx 0.714 \), with all other parameters unchanged, the equilibrium \( \hat{p} = 0, \hat{N} = 12000 \) would not be attainable.
Table 5

\[ r = 1.0, \ K_{11} = 200, \ K_{12} = 100, \ K_{22} = 250 \]

<table>
<thead>
<tr>
<th>( p_0 )</th>
<th>( N_0 )</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>10</td>
<td>Initially, ( N ) increases while ( p ) decreases to a negative value. ( N ) reaches fixation at ( p = 0 ), ( N = K_{22} = 250 ) through damped oscillations between positive and negative gene frequencies.</td>
</tr>
<tr>
<td>0.3</td>
<td>10</td>
<td>( N = K_{22} = 250 ) through damped oscillations between positive and negative gene frequencies.</td>
</tr>
<tr>
<td>0.5</td>
<td>10</td>
<td>( N = K_{22} = 250 ) through damped oscillations between positive and negative gene frequencies.</td>
</tr>
</tbody>
</table>

4. Alternative Fitness forms

The logistic model, in which fitnesses decrease linearly with population size, provides perhaps the simplest example of density-dependent selection. It is, of course, this linear construction which allows fitnesses to take negative values. It is of some importance, then, to consider models with more general classes of fitnesses. One obvious model involves truncation of the logistic fitnesses. Thus we might use fitnesses \( w_{ij}^* \) with \( w_{ij}^* = w_{ij} \) if \( w_{ij} \geq 0 \) and \( w_{ij}^* = 0 \) if \( w_{ij} < 0 \). Some numerical study of this was done for the example in Table 5, and trajectories proceeded rapidly to fixation at \( \hat{p} = 0, \hat{N} = K_{22} \), with no oscillation. When these truncated fitnesses were used for the parameters in Table 1, \( p_I \) and \( N_I \) approached a new interior equilibrium with \( \hat{p} = 0.367544 \) and \( \hat{N} = 183.772234 \), as can be obtained by setting \( w_1 = w_2 = 1 \) and \( w_{11} = 0 \). Such truncated fitnesses seem difficult to interpret biologically but might apply to certain threshold situations.

Another particular alternative where the fitnesses decrease asymptotically to zero as the population size grows is the exponential form of the logistic model. In a single species system this produces many of the same qualitative results as the logistic (Macfadyen, 1963; May, 1974). The genotypes \( A_iA_j \) have fitnesses, at time \( t \)

\[
\begin{align*}
w_{ij}^{(t)} &= \exp \left[ r_{ij}(1 - N_t/K_{ij}) \right] \\
i, j &= 1, 2.
\end{align*}
\]

One reason for preferring equation (20) to the logistic scheme has been mentioned above (see also May, 1974), i.e. \( w_{ij}^{(t)} > 0 \). Another is that in the move from the continuous logistic model \( dN/dt = rN(1 - N/K) \) to the discrete model, the appropriate change in the meaning of fitness is the analog of the change from Malthusian to Wrightian fitnesses in classical genetics. The latter are often regarded at least heuristically as exponentiated versions of the former.
In the same way as above, we have equilibrium if and only if \( w_1 = w_2 = 1 \), i.e.

\begin{align*}
1 &= w_1 = pw_1(N) + (1 - p)w_{12}(N) \\
1 &= w_2 = pw_2(N) + (1 - p)w_{22}(N).
\end{align*}

(21)

(22)

Since \( w_{ij}(K_{ij}) = 1 \) and \( w_{ij} \) decreases in \( N \), equation (21) implies \( \hat{N} \) is between \( K_{11} \) and \( K_{12} \) while equation (22) implies that \( \hat{N} \) is between \( K_{12} \) and \( K_{22} \). Hence \( w_1 = 1 \) and \( w_2 = 1 \) intersect giving an interior equilibrium if and only if \( K_{12} > K_{11}, K_{22} \) or \( K_{12} < K_{11}, K_{22} \). The equilibrium is given by equations (23) and (24) below

\begin{align*}
\hat{\beta} &= (\hat{w}_{12} - \hat{w}_{22})/(2\hat{w}_{12} - \hat{w}_{11} - \hat{w}_{22}) \\
\hat{N} &= \left( \frac{\hat{w}_{12} - \hat{w}_{11}}{2\hat{w}_{12} - \hat{w}_{11} - \hat{w}_{22}} \right).
\end{align*}

(23)

(24)

To see that equations (21) and (22) define a unique interior equilibrium, differentiate implicitly in equation (21) to obtain

\begin{align*}
\frac{dN(p)}{dp} &= \left( w_{11} - w_{12} \right) / \left[ -pw_{11}(N) - (1 - p)w_{12}(N) \right].
\end{align*}

(25)

But \( w_{ij}(N) < 0 \) and on \( w_1 = 1 \) with \( 0 < p < 1 \) we must have \( N \) between \( K_{11} \) and \( K_{12} \). Thus, equation (25) implies that for \( 0 \leq p \leq 1 \)

\begin{align*}
\frac{dN(p)}{dp} &< 0 \quad \text{for} \quad K_{12} > K_{11} \\
\frac{dN(p)}{dp} &> 0 \quad \text{for} \quad K_{12} < K_{11}.
\end{align*}

(26)

Similarly,

\begin{align*}
\frac{dN(p)}{dp} &< 0 \quad \text{if} \quad K_{12} > K_{22} \\
\frac{dN(p)}{dp} &> 0 \quad \text{if} \quad K_{12} < K_{22}.
\end{align*}

(27)

It follows that if \( K_{12} > K_{11}, K_{22} \) or \( K_{12} < K_{11}, K_{22} \) there is a unique interior equilibrium. Note that the entire analysis holds for any set of positive \( w_{ij} \)'s which are decreasing differentiable functions of \( N \) and are such that \( w^{-1}_{ij}(1) = K_{ij} > 0 \).

A local stability analysis in the neighborhood of \((\hat{\beta}, \hat{N})\) given by equations (23) and (24) for the general case produces the eigenvalues

\begin{align*}
\lambda_1 &= 1 + \hat{\beta}(\hat{w}_{11} - \hat{w}_{12}) \\
\lambda_2 &= 1 + \hat{N}(\hat{\beta}^2 \hat{w}_{11}(\hat{N}) + 2\hat{\beta}(1 - \hat{\beta})\hat{w}_{12}(\hat{N}) + (1 - \hat{\beta})^2 \hat{w}_{22}(\hat{N})).
\end{align*}

(28)

Hence \( K_{12} > K_{11}, K_{22} \) is necessary for the existence of a stable internal equilibrium. The necessary and sufficient conditions for stability in the exponential model can then be shown to be

\begin{align*}
K_{12} > K_{11}, K_{22} \quad \text{and} \quad K_{12} > K_{11}. \tag{29a}
\end{align*}
At the two boundary equilibria \((0, K_{11}), (1, K_{11})\) the eigenvalues are
\[
\lambda_1 = w_{11}^2(K_{ii}) = e^{R_{ij}(1 - K_{ii}/K_{12})}, \quad \lambda_2 = 1 + K_{ii}w_{ii}'(K_{ii}) = 1 - r_{ii} \quad i = 1, 2
\]
so that these boundaries are stable if and only if \(K_{ii} > K_{12}\) and \(0 < r_{ii} < 2\).

The truncated logistic and exponential logistic models are quite close to the linear logistic model in many respects. A third alternative employs density dependent fitnesses that reflect hyperbolic population growth. The formulation used by Schoener (1973) suggests, in discrete time, fitnesses of the form
\[
W_{ij} = 1 + R_{ij}(I_{ij}/N - C_{ij})
\]
where \(R_{ij}, I_{ij}\) and \(C_{ij}\) are constants. This could again lead to the problem of negative fitnesses. Furthermore \(w_{ij}(0)\) is undefined leaving us with no analog of the birth rate.

We present here two alternative representations of hyperbolic fitnesses. The first, following Skellam (1952), Pielou (1969) and Utida (1967), defines
\[
W_{ij} = \frac{1 + r_{ij}I_{ij}/N - C_{ij}}{1 + r_{ij}I_{ij}/K_{ij}}, \quad i, j = 1, 2.
\]
Then \(w_{ij}^{-1}(1) = K_{ij}\) and \(w_{ij}(0) = 1 + r_{ij}\), so that the biological interpretation of the logistic parameters is preserved. Since the \(w_{ij}\) are non-negative and decreasing functions, the general theory developed above applies. A unique stable interior equilibrium given by equations (23) and (24) exists for this model if and only if \(K_{12} > K_{11}, K_{22}\). In this case equation (24) defines \(N\) implicitly as a root of a complicated cubic equation, after which \(\tilde{p}\) can be computed from equation (23).

An alternative to equation (32), similar in spirit to (31) is another hyperbolic form
\[
W_{ij}(N) = (b_{ij} + K_{ij}/N_i)/(1 + b_{ij}), \quad i, j = 1, 2, \quad \text{if } b_{ij} > 0.
\]
This falls into the general class of density-dependent fitness models analysed above and consequently the same qualitative results hold. Again \(w_{ij}(N) < 0\) and \(w_{ij}^{-1}(1) = K_{ij}\), but \(w_{ij}(0)\) is undefined. Thus there is no direct analog to the logistic intrinsic rate of increase \(r_{ij}\) nor is there an obvious biological meaning to the \(b_{ij}\). However, equation (33) is equivalent to (31) if \(1 - R_{ij}C_{ij} \equiv b_{ij}/(1 + b_{ij})\) and \(R_{ij}I_{ij} \equiv K_{ij}/(1 + b_{ij})\). It should be noted that equation (33) in (24) determines \(\tilde{N}\) explicitly as the root of a simple quadratic function, and \(\tilde{N}(p)\) can be solved explicitly from \(\tilde{w} = 1\). Then \(K_{12} > K_{11}, K_{22}\) is
necessary and sufficient for the existence of the unique stable interior equilibrium \((\beta, N)\) given by equations (23) and (24)\(\dagger\).

Under the general density dependent model in which the fitnesses \(w_{ij}\) are decreasing, non-negative, differentiable functions of \(N\) we have proven analytically that a stable equilibrium corresponds to the maximum of the root \(N^*(\rho)\) of the equation \(\bar{w} = 1\). Here \(N^*(\cdot)\) is the equilibrium function of the gene frequency. The details are in the appendix. For the logistic case since \(N^*(\rho)\) can be obtained explicitly we can obtain the maximization result directly rather than implicitly. However, it should be emphasized that in the logistic case the problem of attainability must be considered in any evaluation of such a principle.

5. Discussion

The analysis we have presented has three objectives. First we have shown that with logistic fitnesses defining the density dependent discrete generation genetic system the problem of attainability must be considered in any equilibrium analysis. The logistic system may produce trajectories which are always admissible; this is certainly the case in Roughgarden's examples. But locally stable, feasible equilibria may not be biologically relevant since the recursion system may pass through inadmissible gene frequency values and/or negative population sizes. It is noteworthy that the birth rates and carrying capacities which occasion this aberration may, when considered as the parameters for a single logistic equation, cause no difficulties. If the logistic fitness system is used then perhaps the relevant "equilibrium", for a starting point from which the standard equilibrium is unattainable should be the last value before the variables become inadmissible. This would be most difficult to predict analytically.

Our second point is that by considering exponential or hyperbolic density dependent fitnesses the problem can in principle be solved and the attainability question does not arise. This is not to say that other difficulties do not remain. For example, there is the complicated issue of regular and chaotic cycling (May, 1974) which arises in the exponential case, as it does in the logistic. In addition the equilibrium \(N\) may not be explicitly extracted even in the hyperbolic case, equation (32). It would be of further interest to study fitnesses in the form, for example, of decreasing rational functions of the population size. Since different forms of the fitnesses may be appropriate to different organisms it is important to develop a classification of the types of qualitative behavior to be expected from categories of density-independent systems.

\(\dagger\)Obviously equations (31) and (33) can be regarded as equivalent, except insofar as, in (33) \(b_{ij}\) is assumed positive, so that \(w_{ij} \geq 0\) for all \(t\) and \(N_t\) has a positive lower bound.
It is interesting to see from the stability conditions (29) and (30) for the exponential model, that even in the overdominant situation \( K_{12} > K_{11}, K_{22} \), if the birth rates \( r_{ij} \) are large enough and in the right configuration, it may be that none of the equilibria are stable. From the work of May (1974) cycling behavior is predicted under these conditions. Dobzhansky (1943) observed pronounced cycling within breeding seasons in the frequencies of gene arrangements standard arrowhead and duracahua of *Drosophila pseudoobscura*. Although this was interpreted as being due to different ecological optima for the different inversions, it seems difficult in the absence of more demographic data on these populations, to exclude a contribution to this cycling by the birth rate configuration in the above density dependent sense.

Finally, the analysis presented in the appendix demonstrates that under broad density dependent conditions, the equilibrium population size, considered as a function of the gene frequency is a maximum at a stable equilibrium. Previous authors have thought this to be an issue of some biological importance. However, with constant selection values, for more than one locus the fundamental theorem of natural selection fails as a general principle. In general, then, we might suspect that the same will be true in density dependent situations.

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REFERENCES

Suppose that the selection functions \( w_{ij}(N) \) are non-negative decreasing and differentiable functions. Define \( K_{ij} \) to be such that \( w_{ij}(K_{ij}) = 1 \) (the \( K_{ij} \) would usually be called carrying capacities). We prove here that a stable equilibrium occurs at the maximum of the root \( N^*(p) \) of the equation 

\[
p^2 w_{11}(N^*) + 2p(1-p)w_{12}(N^*) + (1-p)^2 w_{22}(N^*) = 1. \tag{A1}
\]

To see this we first differentiate equation (A1) implicitly with respect to \( p \) to obtain

\[
\frac{dN^*(p)}{dp} = \frac{2(w_1 - w_2)}{-p^2 w_{11}(N^*) - 2p(1-p)w_{12}(N^*) - (1-p)^2 w_{22}(N^*)} \tag{A2}
\]

By hypothesis, for \( p \) in \([0, 1]\), \([dN^*(p)/dp]_{w=1}\) then has the same sign as \( w_1 - w_2 \), and it can vanish if and only if \( w_1 = w_2 \) (and both equal 1). But this occurs at exactly one point, namely \((\hat{p}, \hat{N})\), provided either \( K_{12} > K_{11}, K_{22} \) or \( K_{12} < K_{11}, K_{22} \).

Now at the boundary point \((p, N) = (0, K_{22})\) we have

\[
\left. \frac{dN^*(p)}{dp} \right|_{(0, K_{22})} = \frac{w_{12}(K_{22}) - 1}{-w_{22}'(K_{22})} > 0 \quad \text{if} \quad K_{12} > K_{22} \\
\left. \frac{dN^*(p)}{dp} \right|_{(0, K_{22})} = -w_{22}'(K_{22}) < 0 \quad \text{if} \quad K_{12} < K_{22}
\]

while at \((1, K_{11})\)

\[
\left. \frac{dN^*(p)}{dp} \right|_{(1, K_{11})} = \frac{-w_{12}'(K_{11}) < 0 \quad \text{if} \quad K_{12} < K_{11}}{-w_{12}'(K_{11}) > 0 \quad \text{if} \quad K_{12} > K_{11}}
\]

Hence if \((\hat{p}, \hat{N})\) is a locally stable interior equilibrium, so that \( K_{12} > K_{11}, K_{22} \), then equation (A3), (A4) and the continuity of \( w_1 - w_2 \) on \( \hat{w} = 1 \) entail

\[
\left. \frac{dN^*(p)}{dp} \right|_{\hat{w}=1} = \begin{cases} 0 & 0 < p < \hat{p} \\ p = \hat{p} & \hat{p} < p < 1 \end{cases}
\]

Hence \( N^*(p) \) has its maximum in \([0, 1]\) at \((\hat{p}, \hat{N})\).

If \( K_{12} \) is intermediate, e.g., \( K_{11} < K_{12} < K_{22} \), then \( d\hat{N}^*(p)/dp \neq 0 \) for \( p \) in \([0, 1]\) then equation (A3) and (A4) require \( dN^*(p)/dp < 0 \) for all \( p \) in \([0, 1]\). The maximum of \( N^*(p) \) therefore occurs at the point \((0, K_{22})\) which is the only possible stable equilibrium in this case. Similar results hold when \( K_{12} < K_{11}, K_{22} \).