Chapter 4

Continuous-time genetics model

The previous two chapters showed that coexistence of the two species, pioneer and climax, is possible under different conditions, either as a stable equilibrium or as a stable periodic solution. In the next chapters, we examine what the effect of genetic variation might have on existence and stability of equilibria. This is an interesting question, and there have been many papers in the past that examined the question in terms of models of systems of differential and difference equations (for example Roughgarden [12], Levin and Udovic[8], Selgrade and Namkoong [15],[17],[16]). Selgrade and Namkoong [16] for example, investigate cases where either populations are excluded competitively in the absence of genetic variation, but a stable polymorphism exists for competition with genetic variation. They use as examples fitness functions that are linear in the population density variables, and both functions that are independent and dependent of the genetic frequency. They find that for fitness functions that are independent of the genetic frequency, stable polymorphic coexistence is possible for a case where there is global exclusion of the genetic population in its fixation planes. In this chapter, I apply the same questions specific to the pioneer-climax case, adding genetic variation to the pioneer species, and using only fitness functions that are independent of the gene frequency. I have been able to demonstrate using an example, that a stable polymorphic interior equilibrium or cycle are possible, although they vanish on either fixation planes.

Basic models are introduced and a theoretical analysis is done in the next section. In the following section, a special assumption allows the use of an invariant manifold
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theorem to show that a stable interior polymorphism and a Hopf bifurcation on the manifold is possible. In the third section a numerical example of this phenomenon is given using XPPAUT.

4.1 Model equations

The following models are extensions of those in chapter 2. With addition of genetic variation in the pioneer species, the dimension of ODE system is increased by one with the introduction of the gene frequency of the A allele, $p$. As explained in the introduction, the system of equations now becomes

\[
\frac{dp}{dt} = p(\mu_A - \mu) = p(1 - p)(\mu_A - \mu_a),
\]

\[
\frac{dM}{dt} = \mu M,
\]

\[
\frac{dN}{dt} = \eta N,
\]

(4.1)

where $\mu_a$ and $\mu_A$ are defined as

\[
\mu_A = p\mu_{AA} + (1 - p)\mu_{Aa},
\]

(4.2)

\[
\mu_a = p\mu_{Aa} + (1 - p)\mu_{aa},
\]

(4.3)

and $\mu$ is the average population fitness defined as

\[
\mu = p\mu_A + (1 - p)\mu_a,
\]

(4.4)

and now there are three distinct fitness functions for each genotype. Only functions of population density are considered in this thesis, although functions involving the genetic frequency $p$ are also possible (see, for example [15]). Thus $\mu_{ij} = \mu_{ij}(M, N)$. Papers such as Selgrade and Namkoong [17] look at a general competition model involving genetic variation, and does most of the analytical ground-work for this chapter. Solutions to
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(4.1) are found in the region

\[ \mathcal{F} = \{(p, M, N) : 0 \leq p \leq 1, M \geq 0, N \geq 0\}. \]

For an interior equilibrium \( ((p, M, N) \in \text{int}(\mathcal{F})) \) to exist, the heterozygote fitness for the pioneer must be either superior or inferior to both homozygote fitnesses at the equilibrium. That is to say, we have over-dominance or under-dominance at the equilibrium. At the equilibrium, we have \( \mu = \eta = 0 \) thus \( \mu_A = \mu_a = 0 \). The last two conditions imply that the following must be satisfied:

\[ p\mu_{AA} + (1-p)\mu_{Aa} = 0, \quad (4.5) \]
\[ p\mu_{Aa} + (1-p)\mu_{aa} = 0. \quad (4.6) \]

Solving the above two equations make it necessary that either \( \mu_{Aa} > 0 > \mu_{AA}, \mu_{aa} \) or \( \mu_{AA}, \mu_{aa} > 0 > \mu_{Aa} \). Since the fitnesses \( \mu_{ij} \) are independent of \( p \), the following identities can be verified easily:

\[ \frac{1}{2} \frac{\partial \mu}{\partial p} = \mu_A - \mu_a, \quad (4.7) \]
\[ \frac{1}{2} \frac{\partial^2 \mu}{\partial p^2} = \mu_{AA} - \mu_{aa} - 2\mu_{Aa}. \quad (4.8) \]

Using these, the Jacobian matrix at an internal equilibrium \( C \) is given by

\[ J(C) = \begin{pmatrix}
\frac{1}{2}p(1-p) & p\frac{\partial (\mu_{AA} - \mu)}{\partial M} & p\frac{\partial (\mu_{Aa} - \mu)}{\partial N} \\
0 & M \frac{\partial \mu}{\partial M} & M \frac{\partial \mu}{\partial N} \\
0 & N \frac{\partial \mu}{\partial M} & N \frac{\partial \mu}{\partial N}
\end{pmatrix}. \quad (4.9) \]

The lower right \( 2 \times 2 \) matrix of \( J(C) \) is called the ecology matrix (from [15]) and is denoted by \( E(C) \). The eigenvalues of \( J(C) \) are \( \lambda_1 = \frac{1}{2}p(1-p) \frac{\partial^2 \mu}{\partial p^2} = p(1-p)(\mu_{AA} + \mu_{aa} - 2\mu_{Aa}) \), and \( \lambda_{2,3} \) are eigenvalues of \( E(C) \). For the stability of \( C \), all eigenvalues must have negative real parts. The first eigenvalue \( \lambda_1 \) is purely real, and for \( \lambda_1 < 0 \), we must clearly have heterozygote superiority: \( \mu_{Aa} > 0 > \mu_{AA}, \mu_{aa} \) at the equilibrium.
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Following Ginzburg [5], we make a non-degeneracy assumption of the fitness functions:

\[
\frac{\partial \mu}{\partial M} \frac{\partial \eta}{\partial N} - \frac{\partial \mu}{\partial N} \frac{\partial \eta}{\partial M} \neq 0,
\]  

(4.10)

which is equivalent to \( \text{det } E(C) \neq 0 \) at an isolated equilibrium \( C \in \text{Int} \mathcal{F} \). Using the Implicit Function Theorem, one can see that the zero fitness curve locally is the graph of a vector function \( g \) of \( p \):

\[
\{(p, M, N) : \mu(p, M, N) = \eta(M, N)\} = \{(p, M, N) : (M, N) = g(p)\}.
\]  

(4.11)

The equilibrium \( C \) is a critical point of \( g \) since \( \partial \mu / \partial p = 0 \).

Now consider the eigenvalues of the ecology matrix \( E(C) \). The trace and determinant of the matrix are:

\[
\sigma = \text{tr} E(C) = M \frac{\partial \mu}{\partial M} + N \frac{\partial \eta}{\partial N}
\]  

(4.12)

\[
\Delta = \text{det } E(C) = MN \left( \frac{\partial \mu}{\partial M} \frac{\partial \eta}{\partial N} - \frac{\partial \mu}{\partial N} \frac{\partial \eta}{\partial M} \right)
\]  

(4.13)

For a Hopf bifurcation to occur from the equilibrium \( C \), one must have complex eigenvalues cross the imaginary axis at nonzero speed. For the trace \( \sigma \) to change sign, \( \frac{\partial \mu}{\partial M} \) and \( \frac{\partial \eta}{\partial N} \) must have opposite signs. Also, since \( \frac{\partial \mu}{\partial M} \) and \( \frac{\partial \mu}{\partial N} \) are negative (all pioneer genotypes have fitnesses that are monotone decreasing, so any nonnegative sum of the functions must also be monotone decreasing), we must have \( \frac{\partial \eta}{\partial M}, \frac{\partial \eta}{\partial N} > 0 \). This again indicates a predator-prey interaction. For the pioneer-climax competition, the competition occurs at small densities where the climax species has not yet climaxed.

4.2 Homozygote equality

It is not possible to solve for the interior equilibria in general. However, if we assume a biologically reasonable simplifying condition [17], \( \mu_{AA} = \mu_{aa} \) (homozygote equality), then
the ODE system can be reduced to two dimensions, and the mean fitness for the pioneer simply becomes the average of the homozygote and heterozygote fitness.

Assuming homozygote equality, it follows that

$$
\mu_A - \mu_a = (2p - 1)(\mu_{AA} - \mu_{Aa}).
$$

(4.14)

So if $p = 0.5$ then $\dot{p} = 0$ for all $M, N \geq 0$. A simple coordinate transformation $p = r + \frac{1}{2}$ gives

$$
\dot{r} = p(1-p)(2p-1)(\mu_{AA} - \mu_{Aa})
$$

(4.15)

$$
= (r + \frac{1}{2})(\frac{1}{2} - r)(2r)(\mu_{AA} - \mu_{Aa})
$$

(4.16)

$$
= r(\mu_{AA} - \mu_{Aa}) + O(r^2).
$$

(4.17)

Thus, for example, if uniform heterozygote superiority holds throughout $intF$ (i.e., $\mu_{AA} < \mu_{Aa} - \varepsilon$ throughout $intF$ for some $\varepsilon > 0$), then the two-dimensional manifold $r = 0$, or equivalently $p = 0.5$, is exponentially stable. Thus the solution to (4.1) reduces to that of

$$
\dot{M} = 0.5(\mu_{AA} + \mu_{Aa})M,
$$

$$
\dot{N} = \eta N.
$$

(4.18)

Note that the mean fitness in (4.18) is just the average of the genotype fitnesses. The space $\mathcal{P} = \{(p, M, N) : p = 0.5, M > 0, N > 0\}$ is an attracting invariant manifold for each interior equilibrium. Thus a periodic solution which is exponentially stable in $\mathcal{P}$ is also exponentially stable in the full 3-dimensional space, since $\lambda_1 < 0$ is assumed by heterozygote superiority. For this to be true, it is not needed that the plane $\mathcal{P}$ be attracting globally, or in other words, it is not necessary to assume global heterozygote superiority. We only need to assume homozygote equality in a neighborhood of $\mathcal{P}$ and heterozygote superiority at the equilibrium. Moreover, since the bifurcation is persistent
under perturbation, and removing homozygote equality will still leave a Hopf bifurcation. Thus if there exists an example with homozygote equality and with a Hopf bifurcation to a stable periodic solution, there also exist other examples without homozygote equality.

Note that if the interaction coefficients for the genotypes of pioneer are the same, that is if \( \mu_{ij} = \mu_{ij}(Z) \), then the reduced system (4.18) can be handled by the analysis of chapter 1 by replacing \( \mu(Z) = 0.5[\mu_{AA}(Z) + \mu_{Aa}(Z)] \), since the average of two pioneer fitnesses are also pioneer. All the conditions for the existence of a Hopf bifurcation hold. If the interaction coefficients are different \( (\mu_{ij} = \mu_{ij}(Z^{ij}) \) and \( Z^{ij} = c_{11}^{ij}M + c_{12}^{ij}N \)), then the conditions become slightly more complicated. It is no longer possible in general to write the coordinates of the interior equilibria explicitly in terms of \( Z^{ij}_1 \) and \( c_{11}^{ij}, c_{12}^{ij}, c_{21}, c_{22} \), where \( \mu_{ij}(Z^{ij}_1) = 0 \). The Jacobian at an equilibrium becomes the ecology matrix mentioned above, and no simple condition like \( \det C < 0 \) can be found. An exception is when the pioneer functions are linear in \( M \) and \( N \), and this case is explored in the next section.

4.3 Linear/Quadratic model

Let us assume that a linear fitness function is used for the pioneer species, and a quadratic for the climax fitness. This is especially simple, because under homozygote equality, the average of two linear fitnesses is also a linear fitness. Let

\[
\mu_{ij}(Z) = a_{ij} - Z^{ij},
\]

\[
\eta(W) = b - (W - c)^2,
\]

then for homozygote equality,

\[
\dot{M} = \bar{a} - \bar{Z},
\]

\[
\dot{N} = b - (W - c)^2,
\]
where \( \tilde{a} = (a_{AA} + a_{Aa})/2 \), and \( \tilde{Z} = (Z^{AA} + Z^{Aa})/2 \). Also let

\[
\begin{align*}
    c_{11} &= \frac{1}{2} (c_{11}^{AA} + c_{11}^{Aa}), \\
    c_{21} &= \frac{1}{2} (c_{12}^{AA} + c_{12}^{Aa}),
\end{align*}
\]

(4.23) (4.24)

then \( Z = \tilde{Z} = c_{11}M + c_{12}N \). So the equilibria \((M_i, N_i)\) are the solutions of

\[
\begin{align*}
    Z &= c_{11}M + c_{12}N = a, \\
    W &= c_{21}M + c_{22}N = \pm \sqrt{b} + c.
\end{align*}
\]

(4.25) (4.26)

It has already been shown in chapter 2, that only the smaller of the two interior equilibria can undergo a Hopf bifurcation. Thus for \( E_1 \)

\[
M_1 = \frac{c_{22}a - c_{12}(c - \sqrt{b})}{\text{det } C}, \quad N_1 = \frac{c_{11}(c - \sqrt{b}) - c_{21}a}{\text{det } C}.
\]

(4.27)

Further, noting that \( \mu'(Z_1) = -1 \) and \( \eta'(W_1) = \sqrt{b} \), the bifurcation occurs, using (2.13), at

\[
c_{11} = \frac{c_{11}c_{22}\sqrt{b}}{-(c_{22}a - c_{12}(c - \sqrt{b})) + c_{22}(c - \sqrt{b})\sqrt{b}}.
\]

(4.28)

Figure 4.1 shows trajectories of the linear/quadratic model using XPPAUT, for homozygote equality. Note that \( p \) approaches 0.5 monotonically, while on the \( MN \)-plane, \( E_1 \) is locally asymptotically stable. As one of the bifurcation parameters, \( c_{11} \) is decreased (recall that \( c_{11} = 1/2 (c_{11}^{AA} + c_{11}^{Aa}) \), so one can decrease either or both of \( c_{11}^{AA} \) and \( c_{11}^{Aa} \) to achieve this), the equilibrium goes through a Hopf bifurcation in \( \mathcal{P} \), and forms a stable periodic orbit.

This simple example already shows the effect of genetic variation. Although both fixations, \( p = 0 \) or \( p = 1 \) are unstable, they are invariant. Thus if, for example, initially \( p = 0 \) then only the \( aa \) genotype is present, and its fitness cannot produce a stable equilibria: the result is exclusion of either species depending on initial values (see figure 4.2 for trajectories on the \( MN \)-plane when \( p(t = 0) = 1 \) or \( p(t = 0) = 0 \)).
Figure 4.1: Trajectories for continuous-time genetics model with homozygote equality. The parameters used are $a_{AA} = a_{aa} = 0.5, a_{Aa} = 1.3, b = 0.4, c = 2.1, c_{22} = 1.1, c_{11} = 0.53$ for top diagrams, $c_{11} = 0.4$ for the bottom diagrams. The diagrams on the right are 2-dimensional $Mp$-plane projections of the graphs on the left. Note that $p$ approaches 0.5 monotonically for both diagrams, and the cycles are on the $MN$-plane.
Figure 4.2: Trajectories under loss of genetic variation. Coexistence of the two species is impossible if only one of the homozygotes is present. Extinction of either species depend on initial values. The parameters used are $a_{AA} = a_{aa} = 0.5$, $a_{Aa} = 1.3$, $b = 0.4$, $c = 2.1$, $c_{22} = 1.1$, $c_{11} = 0.53$. And $p(0) = 0$ ($p(0) = 1$ gives same result for homozygote equality).

Next, to show that the Hopf bifurcation is not restricted to homozygote equality, XPPAUT is used to draw bifurcation diagrams by varying a parameter that will remove the homozygote equality. In figure 4.3 the parameter $a_{AA}$, which initially equaled $a_{aa}$, is varied together with $c_{11}$ to give a two-parameter bifurcation diagram. When homozygote equality is removed, trajectories are attracted to an invariant manifold that is near $p = 0.5$. For the linear/quadratic model, this equilibrium can be sought explicitly by solving the three equations: $\{\mu_A = \mu_a = \eta = 0\}$.

4.4 Summary

In this chapter, I have been able to show that, with genetic variation added to the pioneer species, not only could there be an stable interior polymorphic equilibrium or a cycle, but that the genetic variation is vital for these to exist. The papers referenced in this
Figure 4.3: Two-parameter continuation, using parameters $c_{11}$ and $a_{AA}$, of Hopf bifurcations in genetics model (4.1). The parameters used are $a_{Aa} = 1.3$, $a_{aa} = 0.5$, $b = 0.4$, $c = 2.1$. * indicates where the continuation was started: when $a_{AA} = 0.5$ for homozygote equality. The z-axis shows the gene frequency: it is 0.5 at homozygote equality, but changes as the condition is lost.
chapter only considered fitness functions linear in population density, but also those with gene frequency dependence. The model used here as an example is linear for the pioneer, but quadratic for the climax species, and is specific to pioneer-climax competition, which has not been demonstrated in a previously published paper.

Although the dynamics are more complicated with the added dimension, the assumption of homozygote equality effectively reduced the dimension to two again, and allowed reuse of the theory obtained in chapter 2. Since the pioneer fitness for this special case is simply the average of those of the homozygote and the heterozygote, one can quickly find cases where neither the homozygote nor the heterozygote alone can produce a stable coexistence with the competing climax trees, whereas their average can.