Evolutionary Stability in Lotka-Volterra Systems* 

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Abstract: The Lotka-Volterra model of population ecology, which assumes all individuals in each species behave identically, is combined with the behavioral evolution model of evolutionary game theory. In the resultant monomorphic situation, conditions for the stability of the resident Lotka-Volterra system, when perturbed by a mutant phenotype in each species, are analyzed. We develop an evolutionary ecology stability concept, called a monomorphic evolutionarily stable ecological equilibrium, which contains as a special case the original definition by Maynard Smith of an evolutionarily stable strategy for a single species. Heuristically, the concept asserts the resident ecological system must be stable as well as the phenotypic evolution on the “stationary density surface”. The conditions are also shown to be central to analyzing stability issues in the polymorphic model that allows arbitrarily many phenotypes in each species, especially when the number of species is small. The mathematical techniques are from the theory of dynamical systems, including linearization, centre manifolds and Molchanov’s Theorem.

Keywords: Lotka-Volterra, evolutionary ecology, phenotypic evolution, evolutionarily stable equilibrium, ESS.
1 Introduction

For multi-species systems, ecology clearly plays an important role in any realistic model of phenotypic evolution. However, theoretical ecology has traditionally emphasized analyzing multi-species models where fitness depends only on the density (i.e. population size) of each species and not its phenotypic makeup.¹ On the other hand, the concept of evolutionary stability, that was originally developed to analyze the frequency evolution of phenotypes in a single species (Maynard Smith, 1982), has been extended to multi-species models of phenotypic frequency evolution (Cressman et al., 2001) under the umbrella of what is known as evolutionary game theory (Hofbauer and Sigmund, 1998). From the ecological perspective, the basic problem with these frequency dependent models is that there is no control over species’ densities. In fact, without some artificial assumption on background fitness of the different species, it is quite possible a species may go extinct at the same time its phenotype is evolutionarily stable. Thus, neither the typical approaches of theoretical ecology nor of evolutionary games are satisfactory models of phenotypic evolution in multi-species ecological systems.

The main purpose of this paper is to develop a general concept of evolutionary stability in “evolutionary ecosystems”, where phenotypic fitnesses are density dependent. According to Maynard Smith, a phenotype is evolutionarily stable for a single species if a monomorphic resident population, all of whose members exhibit this phenotype, cannot be successfully invaded by a mutant phenotype (i.e. the mutants neither persist nor spread under the influence of natural selection). Technically, this evolutionarily stable

¹See, for example, Pielou (1977).
phenotype is called an *evolutionarily stable strategy* (ESS). Initially (Section 2), we generalize Maynard Smith’s monomorphic approach. Heuristically, evolutionary ecological stability here refers to a resident ecosystem in which

(i) there are equilibrium densities where no species is extinct and this equilibrium is stable in the underlying ecological model when there are no mutant phenotypes, and

(ii) this equilibrium remains stable when a rare mutant phenotype is introduced in each species and subsequently eliminated.

Technically, the paper follows the “dynamic approach” (e.g. Cressman, 1992) since stability refers to (local) asymptotic stability. We call such an ecological equilibrium *monomorphic evolutionarily stable* (with respect to invasion by these mutants). A complete characterization of this concept is given (Section 2.2) from the mathematical (Theorem 1) as well as the biological (Theorem 2) perspectives.

An equally important model of evolutionary systems is the polymorphic situation where the evolutionarily stable ecosystem can have more than one phenotype present in some or all of the species. Of particular interest from the theoretical evolutionary viewpoint is the “pure strategy” model where each individual exhibits only one behavior. Section 3 shows there is a fundamental connection between stability in the monomorphic and polymorphic scenarios.

\(^2\)Here, mutation is assumed to be a very rare event with selection acting much faster as it eliminates the less fit phenotype before any subsequent mutation. Thus, rare mutations in the single-species monomorphic model mean that the relative frequency of this mutation is small and that the mutant subpopulation is homogeneous (i.e. all individuals in this subpopulation exhibit the same rare phenotype). In general, a phenotype in this monomorphic approach may be a “mixed strategy” (see Section 3.3).

\(^3\)That is, for all initial relevant populations sufficiently close to the equilibrium, evolution remains close to this equilibrium and eventually returns to it.
To illustrate how density and frequency effects can be combined into a single model of an evolutionary ecosystem, we have chosen a standard ecological model (the Lotka-Volterra equation) together with the standard model of evolutionary games with a finite number of possible phenotypes. There are two reasons for this choice. First, both models have similar underlying assumptions. For instance, in the separate models, individual fitness is linearly dependent on the density and on the frequency respectively of the population phenotypes. Moreover, both models ignore spatial and genetic effects, assuming interactions are pairwise and at random since the whole system is totally mixed.

A second reason to restrict our analysis to these well-known elementary models is that the mathematical development is then more straightforward, thereby allowing us to concentrate more fully on biological considerations. However, we hasten to add that the methods we develop (especially the separation of density and frequency effects) as well as the results we obtain apply to more general evolutionary ecosystems where individual fitness functions are not linear. It is interesting to note that a similar separation of density and frequency effects was successfully used to analyze stability in early theoretical research on coevolution; namely, the genetic coevolutionary models for natural selection at a single locus (see Ginzburg (1983) for a summary of both linear and nonlinear fitness models). A discussion of how our evolutionary ecosystem model is related to these models (and others in the literature) is postponed to the final section where the biological usefulness of our results is emphasized.
2 The Monomorphic Model

Consider the $\mathcal{N}$–species Lotka-Volterra equation (1) written, for notational reasons,\(^4\) in the form

\[
\dot{\rho}_k = \rho_k \left( r_k + \sum_{l=1}^{\mathcal{N}} m_{kl}^{RR} \rho_l \right) \quad k = 1, \ldots, \mathcal{N} \tag{1}
\]

where $\rho_k$ and $r_k$ are the density and the intrinsic growth rate of species $k$ respectively and $m_{kl}^{RR}$ is the interaction parameter between species $k$ and species $l$. Underlying this equation is the assumption that every individual in species $k$ has the same fitness (namely, $r_k + \sum_{l=1}^{\mathcal{N}} m_{kl}^{RR} \rho_l$) that depends linearly on the current density vector $\rho = (\rho_1, \ldots, \rho_{\mathcal{N}})$ through the interaction parameters $m_{kl}^{RR}$.

The main purpose of this paper is then to analyze what happens when all individuals in species $k$ do not have the same fitness. To model this situation, we take the perspective of evolutionary game theory where individuals in species $k$ can exhibit different phenotypes (or behaviors).

In the monomorphic model, the $\mathcal{N}$–species ecological system with the original interaction parameters (1) is considered the resident system. Each species is homogeneous in that every individual in it exhibits the same phenotype. A mutant phenotype is then introduced in each species which, through interactions with both resident and mutant phenotypes in all the species, re-

\(^4\)An $\mathcal{N}$–species Lotka-Volterra equation is often written as $\dot{x}_k = x_k \left( r_k + \sum_{l=1}^{\mathcal{N}} a_{kl} x_k \right)$. Our notation avoids possible confusion with variables used later in the paper and the superscripts in $m_{kl}^{RR}$ emphasize that these parameters refer to interactions between residents ($R$). In any notation, population densities are functions of the continuous time variable $t$ and the left-hand side of the differential equation in (1) is the derivative of density with respect to $t$. 

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places each $m_{kl}^{RR}$ with four interaction parameters to produce the monomorphic evolutionary Lotka-Volterra system (2).

\[
\begin{align*}
\dot{\rho}_k &= \rho_k \left( r_k + \sum_{l=1}^{N} \left( m_{kl}^{RR} \rho_l + m_{kl}^{RI} \mu_l \right) \right) \\
\dot{\mu}_k &= \mu_k \left( r_k + \sum_{l=1}^{N} \left( m_{kl}^{IR} \rho_l + m_{kl}^{II} \mu_l \right) \right).
\end{align*}
\] (2)

Here $\rho_k$ and $\mu_k$ respectively are the densities of the resident and mutant strategies respectively in species $k$. It is assumed in (2) that the mutant strategies only affect the interaction parameters, leaving the intrinsic growth rate $r_k$ of each species $k$ unchanged. In the language of evolutionary game theory, $m_{kl}^{RR}$ is called the payoff to a resident phenotype (or strategy) in species $k$ when interacting with a resident phenotype in species $l$. Also, $m_{kl}^{RI}, m_{kl}^{IR}$ and $m_{kl}^{II}$ denote the payoffs involving mutants (e.g. $m_{kl}^{IR}$ is the payoff to an invading (I) mutant in species $k$ interacting with a resident (R) in species $l$). The possible values for these payoffs involving mutants are related to the set of all possible mutants in each species. These are often given by a strategy simplex as in the polymorphic model of Section 3.

### 2.1 The Single Species Monomorphic Model

The stability analysis of (2) contains several mathematical subtleties, especially when there is more than one species. To give some feeling for these

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5 This coupled system of $2N$ ordinary differential equations leaves the nonnegative $2N$-dimensional orthant $R_{\geq 0}^{2N} \equiv \{(\rho,\mu) = (\rho_1,\ldots,\rho_N,\mu_1,\ldots,\mu_N) \mid \rho_k \geq 0 \text{ and } \mu_k \geq 0 \text{ for } 1 \leq k \leq N\}$ invariant as well as its interior (i.e. the positive orthant $R_{>0}^{2N}$) and any of its faces. In particular, on the face where $\mu_k = 0$ for all $1 \leq k \leq N$, this system reverts to the original Lotka-Volterra equation (1) without mutation (i.e. to the resident system).
complexities, we now give a brief but thorough mathematical description of how they apply to the single species model, followed by a summary of the biological interpretation.

Let us rewrite the single species monomorphic evolutionary Lotka-Volterra system as

\[
\begin{align*}
\dot{\rho} &= \rho \left( r + m_{RR} \rho + m_{RI} \mu \right) \\
\dot{\mu} &= \mu \left( r + m_{IR} \rho + m_{II} \mu \right).
\end{align*}
\]

Then, a monomorphic evolutionarily stable ecosystem, as described in the Introduction, is a situation where firstly the resident Lotka-Volterra equation \( \dot{\rho} = \rho \left( r + m_{RR} \rho \right) \) has an asymptotically stable equilibrium \( \rho^* > 0 \). It is easy to show that this one-dimensional dynamic has such a \( \rho^* \) if and only if \( r > 0 \) and \( m_{RR} < 0 \).\(^6\) We assume this ecological condition is true for the remainder of this section.

Secondly, the corresponding equilibrium \((\rho^*, 0)\) of (3) is to be asymptotically stable in the nonnegative first quadrant \( R^2_{\geq 0} \) when a mutant is introduced. Linearization, given by

\[
\begin{bmatrix}
\dot{\rho} \\
\dot{\mu}
\end{bmatrix} \approx
\begin{bmatrix}
m_{RR} \rho^* \\
0
\end{bmatrix}
\begin{bmatrix}
\rho^* & m_{RI} \\
0 & r + m_{IR} \rho^*
\end{bmatrix}
\begin{bmatrix}
\rho - \rho^* \\
\mu
\end{bmatrix}
\]

is also the initial step in the analysis. This has eigenvalues \( \rho^* m_{RR} < 0 \) and \( r + \rho^* m_{IR} = \rho^* (m_{IR} - m_{RR}) \). If \( m_{IR} < m_{RR} \), \((\rho^*, 0)\) is evolutionarily stable since both eigenvalues are negative. If \( m_{IR} > m_{RR} \), \((\rho^*, 0)\) is unstable (in

\(^6\)Linearization will be important throughout the paper and can be used to prove this statement. Here, the eigenvalue at the equilibrium \( \rho^* > 0 \) is \( \rho^* m_{RR} = -r \) and this must be negative for (local) asymptotic stability. In fact, it is well-known that \( \rho^* \) is globally asymptotically stable in this case.
fact, an unstable saddle point). That is, if the mutant phenotype has lower (respectively, higher) payoff than the resident phenotype in their most common interactions with other residents, the invading phenotype goes extinct (respectively, spreads in the population).

The final case, when mutants and residents have equal payoff in their interactions with residents (i.e. \( m^{IR} = m^{RR} \)), is quite important both biologically (since it models the situation where mutants are initially selectively neutral) and mathematically (since there is then a zero eigenvalue and so linearization does not determine asymptotic stability). Higher order terms must be considered in (3). Again, several mathematical techniques can be used to analyze this two-dimensional dynamical system. The technique used in the corresponding situation in the multi species model of Section 2.2 is called centre manifold theory (see the proof of Theorem 1 in the Appendix). When applied to the single species evolutionary system, it asserts there is a smooth one-dimensional curve through \((\rho^*, 0)\) that is invariant under the dynamics and with tangent vector at \((\rho^*, 0)\) the eigenvector of the linearization with eigenvalue 0. Moreover, \((\rho^*, 0)\) is asymptotically stable if and only if it is asymptotically stable for the dynamic restricted to this one-dimensional curve. Calculations using this curve (see Theorem 1 below) show \((\rho^*, 0)\) is asymptotically stable if and only if \( m^{II} < m^{RI} \).

**Summary:** The single species monomorphic evolutionary Lotka-Volterra system has a monomorphic evolutionarily stable ecological equilibrium \((\rho^*, 0)\) (with \( \rho^* > 0 \)) if and only if the following three conditions hold.

\[
r > 0 \text{ and } m^{RR} < 0,
\]  

(4)
\[ m^{IR} \leq m^{RR} \quad \text{and} \quad \text{if } m^{IR} = m^{RR}, \text{ then } m^{II} < m^{RI} \] (5)

From the biological perspective, (4) guarantees the ecological stability of the resident phenotype. Moreover, in the theory of evolutionary games for single species frequency dependent evolution, conditions (5) and (6) are called the (Nash) equilibrium condition and the stability condition respectively. Together, they assert that the resident phenotype is an ESS of the $2 \times 2$ payoff matrix given by

\[
\begin{bmatrix}
  m^{RR} & m^{RI} \\
  m^{IR} & m^{II}
\end{bmatrix}.
\]

That is, $(\rho^*, 0)$ is a monomorphic evolutionarily stable ecological equilibrium of (3) if and only if $\rho^* > 0$ is a asymptotically stable equilibrium of the resident density dynamic (when phenotypes are fixed) and the resident phenotype is asymptotically stable for the frequency dynamic when density is fixed at $\rho^*$. In particular, the single species monomorphic evolutionary Lotka-Volterra system gives a clear separation of frequency and density effects.

2.2 The Multi Species Monomorphic Model

Following the approach of Section 2.1, first consider the resident Lotka-Volterra equation (1) and assume that it has an equilibrium $\rho^* = (\rho^*_1, \ldots, \rho^*_N)$ in the positive orthant $R^N_{>0}$. Moreover, suppose $\rho^*$ is asymptotically stable. By definition, the corresponding equilibrium $(\rho^*, \mu^*) = (\rho^*_1, \ldots, \rho^*_N, 0, \ldots, 0)$ of the monomorphic evolutionary Lotka-Volterra system (2) is monomorphic evolutionarily stable if and only if it is asymptotically stable. The following theorem (proved in the Appendix) is the main stability result.
Theorem 1 Assume \( \rho^* \in \mathbb{R}_{>0}^N \) is an ecologically stable equilibrium of (1) that is hyperbolic.\(^7\) Let \( \lambda_k \equiv r_k + \sum_{l=1}^{N} m_{kl}^{IR} \rho^*_l \) be the expected individual fitness at equilibrium for the mutant phenotype in species \( k \). If \((\rho^*,0)\) is a monomorphic evolutionarily stable ecological equilibrium of (2), then it satisfies the following (Nash) equilibrium condition.

**Equilibrium condition:** \( \lambda_k \leq 0 \) for all \( k = 1,...,N \).

**Stability condition:** Conversely, if \( \lambda_k < 0 \) for all \( k = 1,...,N \), then \((\rho^*,0)\) satisfies the stability condition by default and so is automatically a monomorphic evolutionarily stable ecological equilibrium of (2). Furthermore, if \( \lambda_k = 0 \) for all \( k \), then \((\rho^*,0)\) is a monomorphic evolutionarily stable ecological equilibrium of (2) if and only if \( M \equiv M^{II} - M^{IR} (M^{RR})^{-1} M^{RI} \) is an \( N \times N \) B-matrix.\(^8\)

Here \( M^{RR} \), the \( N \times N \) matrix whose entries are the payoffs \( m_{kl}^{RR} \) for interactions between residents, is invertible. Similarly, \( M^{IR} \) is the \( N \times N \) matrix of payoffs to mutants interacting with residents etc. The statement of the theorem parallels the ecological, equilibrium and stability conditions (i.e. (4), (5) and (6)) of Section 2.1. Our assumption of ecological stability

\(^7\)By definition, an equilibrium is hyperbolic if the Jacobian matrix of the linearization about this equilibrium has no eigenvalue with zero real part. Thus, we suppose \( \rho^* \) is an asymptotically stable hyperbolic equilibrium of (1). Lotka-Volterra equations whose Jacobian has eigenvalues of zero real part, such as the two species predator-prey system, are beyond the scope of this paper.

\(^8\)Precise statements can also be given for the intermediate case where some but not all \( \lambda_k = 0 \) to give a complete description of the stability condition for a monomorphic evolutionarily stable ecological equilibrium. These have been omitted to avoid further mathematical complexities. The \( N \times N \) matrix \( M \) is a B-matrix (see Hofbauer and Sigmund, 1998) if \( \rho_k \sum_{l=1}^{N} M_{kl} p_l < 0 \) for some \( k \) whenever \( \rho \) is a nonzero element of \( \mathbb{R}_{>0}^N \). For \( N = 1 \), \( \lambda_1 = 0 \) is equivalent to \( m^{IR} = m^{RR} \) in Section 2.1 and so \( m^{II} = m^{IR} \left( \frac{1}{m^{RR}} \right) m^{RI} = m^{II} - m^{RI} \). This is a \( 1 \times 1 \) B-matrix if and only if \( m^{II} - m^{RI} < 0 \). That is, we have the stability condition (6).
means the stability of $\rho^*$ is completely determined by the linearization of (1) about $\rho^*$. Since $\lambda_k = \sum_{i=1}^{N} m_{kl}^{IR} \rho_i^* - \sum_{i=1}^{N} m_{kl}^{RR} \rho_i^*$ as well, $\lambda_k$ is also the difference in payoff between the mutant and resident phenotypes. Thus, the equilibrium condition again asserts the residents must be at least as fit as the mutants at $(\rho^*, 0)$. The final statement parallels (6). When the ecologically stable equilibrium is simultaneously invaded by selectively neutral mutants (i.e., $\lambda_k = 0$ for all $k$), the B-matrix condition is a negativity result from Molchanov’s Theorem (Golster, 1997) that replaces $m^{II} - m^{RI} < 0$ for a single species. It also appears naturally in the $\mathcal{N}$–species ESS concept developed for frequency-dependent phenotypic evolution in Cressman et al. (2001) (see also Section 3.3).

This final statement of the theorem is again quite important in the polymorphic model of Section 3. Mathematically, the condition arises from the existence of an $\mathcal{N}$–dimensional centre manifold which determines the stability of $(\rho^*, 0)$ but is difficult to interpret from a biological viewpoint. Fortunately, all the results of the theorem can be given in biological terms (see Theorem 2) by rewriting (2) in terms of species’ densities and phenotypic frequencies (see (14) and (16) in the Appendix).

To this end, let $\epsilon_k$ be the frequency of the mutant phenotype in species $k$ at time $t$ (i.e., $\epsilon_k = \frac{\mu_k}{\mu_k + \rho_k}$) and $N_k = \mu_k + \rho_k$ be the density of species $k$. We then have the following facts (see the Appendix for details). Since $M^{RR}$ is invertible, for $\epsilon \in R_{\geq 0}^N$ sufficiently close to 0, there is an $\mathcal{N}$–dimensional surface given by $(N_1(\epsilon), ..., N_N(\epsilon))$ on which $\dot{N}_k = 0$ for all $k$. In particular, $(N_1(0), ..., N_N(0)) = \rho^*$. We call this surface the stationary density surface for (2) and use the frequency dynamic that (2) induces on this surface in the
The following theorem.\footnote{The stationary density surface is not the centre manifold for (2) when mutants are selectively neutral since it is not invariant. As a consequence, the mathematically precise statement of Theorem 2 must assume the matrix $M$ in Theorem 1 satisfies the technical nondegeneracy condition that it does not possess a zero ray (Golster, 1997). That is, there is no nonzero $X \in R^N_{\geq 0}$ such that $X_k \sum_{l=1}^{N} M_{kl} X_l = 0$ for all $k$. Without this assumption, it is possible that an asymptotically stable equilibrium of the frequency dynamic on the stationary density surface may not be monomorphic evolutionarily stable for (2) (see the final paragraph of the proof of Theorem 2 in the Appendix). From now on, assume each matrix that arises which would imply asymptotic stability if it were a B-matrix does not possess a zero ray.}

**Theorem 2** Assume $\rho^* \in R^N_{>0}$ is an asymptotically stable hyperbolic equilibrium of (1). Then $(\rho^*, 0)$ is a monomorphic evolutionarily stable ecological equilibrium of (2) if and only if 0 is an asymptotically stable equilibrium of the frequency dynamic (written in terms of $\epsilon \in R^N_{\geq 0}$) induced by (2) on its stationary density surface.

Theorem 2 is the analogue, for multi species hyperbolic equilibria of the resident density dynamic, of the last paragraph of Section 2.1. That is, $(\rho^*, 0)$ is a monomorphic evolutionarily stable ecological equilibrium of (2) if and only if the resident ecological system (with fixed phenotypes) as well as the phenotypic evolution (on the stationary density surface) are both stable at this equilibrium.

### 3 The Polymorphic Model

In the polymorphic model, individuals in each species may exhibit an arbitrary (but finite) number of possible phenotypes (i.e. pure strategies). Payoffs are now given through an *interaction system of matrices* $\{A^{kl}\}_{k,l=1,\ldots,N}$.
whereby the individual payoff to the $i^{th}$ phenotype in species $k$ when interacting with the $j^{th}$ phenotype in species $l$ is the entry $A_{ij}^{kl}$ of $A^{kl}$. The (pure strategy) polymorphic evolutionary Lotka-Volterra system is

$$
\dot{n}_i^k = n_i^k \left( r_k + \sum_{l=1}^{N} \sum_j A_{ij}^{kl} n_j^l \right)
$$

(7)

where $n_i^k$ is the number (i.e. density) of the $i^{th}$ phenotype in species $k$.

We will again analyze the stability of (7) in terms of the density $N_k \equiv \sum n_i^k$ and the phenotypic frequencies $p_i^k \equiv n_i^k / N_k$ of each species (e.g. Theorem 3 below). Adopting notation from multi species evolutionary game theory (e.g. Cressman et al., 2001), the frequency vector $p^k$ with components $p_i^k$ is an element of the strategy simplex $\Delta^k$ of species $k$. The vertices of $\Delta^k$ are the unit coordinate vectors $e_i^k$ that have 1 in the $i^{th}$ component and 0 everywhere else. Then, $p^k \cdot A^{kl} p^l \equiv \sum_i \sum_j p_i^k A_{ij}^{kl} p_j^l$ and $e_i^k \cdot A^{kl} p^l \equiv \sum_j A_{ij}^{kl} p_j^l$ are the “average individual payoff” and the “expected payoff of the $i^{th}$ strategy” respectively in species $k$ due to a random interaction with an individual in species $l$. In terms of frequency and density, (7) can be rewritten as

\begin{align*}
(\text{i}) \quad \dot{N}_k &= N_k \left( r_k + \sum_{l=1}^{N} N_l p^k \cdot A^{kl} p^l \right) \\
(\text{ii}) \quad \dot{p}_i^k &= p_i^k \sum_{l=1}^{N} N_l \left( e_i^k - p^k \right) \cdot A^{kl} p^l.
\end{align*}

(8)

Assume there is an equilibrium $n^* = (n_1^*, ..., n_N^*)$ of (7) in the non-negative orthant for which each species is present (i.e. $N_{*k} > 0$ for all $k$). Equivalently, $N^* = (N_1^*, ..., N_N^*)$ and $p^* = (p_1^*, ..., p_N^*)$ is an equilibrium of (8) with all components of $N^*$ positive. For technical reasons, we also
suppose that, when frequencies are fixed at \( p^* \), \( N^* \) is the only equilibrium of (8)(i) in \( R_1^N \). In particular, the \( N \times N \) matrix with entries \( p^*k \cdot A^kl \cdot p^*l \) is invertible. This implies, for \( p \) near \( p^* \), there is a stationary density surface, \( N(p) \equiv (N_1(p),...,N_N(p)) \), on which \( \dot{N}_k(p) = 0 \) for all \( k \).

3.1 The Single Species Polymorphic Model

It turns out the stability of (7) in terms of density and phenotypic frequency is most clear for single species polymorphic evolutionary Lotka-Volterra systems (see Theorem 3 below). We therefore briefly discuss this model here before returning to the multi species model in Section 3.2.

Assume there are \( s \) phenotypes in this species. We rewrite (7) (and similarly (8)) as

\[
\dot{n}_i = n_i \left( r + \sum_{j=1}^{s} A_{ij}n_j \right)
\]

with the \( k, \ell \) parameters that denote the species number omitted.

**Theorem 3** Assume that (9) has an equilibrium \( n^* = (N^*, p^*) \) for which \( N^* > 0 \) and that \( n^* \) is hyperbolic when restricted to the resident system (i.e. to those phenotypes with \( n^*_i > 0 \)). Then \( n^* \) is asymptotically stable for (9) if and only if \( N^* \) is asymptotically stable for the density dynamic at equilibrium frequency and \( p^* \) is asymptotically stable for the frequency dynamic induced by (8)(ii) on the stationary density surface.

**Proof of Theorem 3.** A key to the proof is to introduce the new density variable

\[
\hat{N} \equiv N - N(p)
\]
where $N(p)$ is the stationary density surface. That is, $\dot{N} = 0$ is the stationary density surface. In terms of the variables $\hat{N}$ and $p$, the linearization of (8) is

$$$
\begin{bmatrix}
\hat{N} \\
\dot{p}_1 \\
\vdots \\
\dot{p}_s
\end{bmatrix} 
\approx

\begin{bmatrix}
\frac{\partial \hat{N}}{\partial N} & \frac{\partial \hat{N}}{\partial p_1} & \cdots & \frac{\partial \hat{N}}{\partial p_s} \\
\vdots & \ddots & \ddots & \vdots \\
\frac{\partial \dot{p}_1}{\partial N} & \ddots & \ddots & \frac{\partial \dot{p}_s}{\partial N} \\
J & \ddots & \ddots & \ddots
\end{bmatrix}
\begin{bmatrix}
\hat{N} \\
p_1 - p_1^* \\
\vdots \\
p_s - p_s^*
\end{bmatrix}
$$$

(10)

where $J$ is the $s \times s$ Jacobian matrix of the frequency dynamic on the stationary density surface. Now $\frac{\partial \hat{N}}{\partial N} = \frac{\partial \hat{N}}{\partial N} = \frac{\partial \hat{N}}{\partial N} = N^*p^* \cdot Ap^*$ at equilibrium since $\hat{N} = \hat{N} - \hat{N}(p)$ and, from (8)(i), $\dot{N} = \left(\hat{N} + N(p)\right) \left(r + \left(\hat{N} + N(p)\right) p \cdot Ap\right)$.

More importantly, from (8)(ii), $\frac{\partial \dot{p}_i}{\partial N} = p_i^* \left(e_i - p^*\right) \cdot Ap^* = 0$ for all $i$. Thus, the linearization is upper block diagonal with eigenvalues given by $\frac{\partial \dot{N}}{\partial N}$ (i.e. the linearization of the density dynamic at equilibrium frequency) together with those of $J$.

Since $n^*$ is a hyperbolic equilibrium of the resident system, it is straightforward to show that the only eigenvalues of (9) with zero real part correspond to selectively neutral invading phenotypes (i.e. those invading phenotypes with $r + e_i \cdot An^* = 0$) and then this eigenvalue is zero. Moreover, the only eigenvalues of $J$ in (10) with zero real part correspond to these same selectively neutral invading phenotypes. Thus, if there are no selectively neutral invading phenotypes, the proof is complete since all eigenvalues of (9) have negative real part if and only if both the density dynamic at equilibrium frequency is asymptotically stable (i.e. $\frac{\partial \dot{N}}{\partial N} = N^*p^* \cdot Ap^* < 0$) and the frequency dynamic induced on the stationary density surface is asymptotically stable (i.e. all the eigenvalues of $J$ have negative real part).

Now suppose that $n^*$ is asymptotically stable. If $n$ is initially sufficiently close to $n^*$, then $n$ remains close and eventually converges to $n^*$. Since
$N^* > 0$, $p$ also remains close to $p^*$ and eventually converges to $p^*$ for the frequency dynamic

$$
\dot{p}_i = N p_i (e_i - p) \cdot Ap.
$$

(11)

Now the induced frequency dynamic on the stationary density surface $\dot{p}_i = N(p)p_i (e_i - p) \cdot Ap$ has the same trajectories (by a rescaling of time) with the same direction as the frequency dynamic (11). Thus, $p^*$ is asymptotically stable on the stationary density surface. Since $N^*p^* \cdot Ap^* < 0$, the density dynamic at equilibrium frequency, $\dot{N} = N(r + Np^* \cdot Ap^*)$ has an asymptotically stable equilibrium at $N^*$. Conversely, if $\frac{\partial \dot{N}}{\partial N} < 0$ and $p^*$ is asymptotically stable on the stationary density surface, $N$ remains close to $N^*$ for (9) and so $p$ also remains close to $p^*$ and eventually converges to $p^*$ for (11). That is, $n^*$ is asymptotically stable.

Theorem 3 is the polymorphic analogue, for the single species model, of Theorem 2 for monomorphic systems in that it gives a clear-cut separation of frequency and density effects. In fact, for single species, the dynamic on the stationary density surface is equivalent to the dynamic induced on the “fixed density surface”. To see this, note that the stationary density surface has $r + N(p)p \cdot Ap = 0$ or equivalently, $N(p) = -\frac{r}{p \cdot Ap}$. Thus, the induced frequency dynamic is $\dot{p}_i = -\frac{r}{p \cdot Ap} p_i (e_i - p) \cdot Ap$ and so its trajectories are time-rescaled trajectories of the frequency dynamic $\dot{p}_i = N^*p_i (e_i - p) \cdot Ap$ with the same direction since $-\frac{r}{p \cdot Ap} > 0$. This later frequency dynamic is the one induced on the density surface with $N$ fixed at $N^*$. 
3.2 The Multi Species Polymorphic Model

Unfortunately, Theorem 3 is not true in the multi species setting as can be seen from the following elementary example of two species with 2 and 1 phenotypes respectively.

Example 1 For two species models, we will adopt slightly different notation by defining \( A \equiv A^{11}, B \equiv A^{12}, C \equiv A^{21} \) and \( D \equiv A^{22} \).\(^{10}\)

Suppose species one and two have 2 and 1 phenotypes respectively and that

\[
A = \begin{bmatrix} -1 & -1 \\ -1 & -2 \end{bmatrix}; B = \begin{bmatrix} 0 \\ 1 \end{bmatrix} \\
C = \begin{bmatrix} 3 & 3 \end{bmatrix}; D = [-1].
\]

With \( r_1 = 2 \) and \( r_2 = -5 \), an equilibrium \( n^* \) of (7) is \( n_{11}^* = n_{21}^* = 1 \) and \( n_{12}^* = 1 \). The linearization of (7) has 3×3 Jacobian matrix

\[
\begin{bmatrix}
-1 & -1 & 0 \\
-1 & -2 & 1 \\
3 & 3 & -1
\end{bmatrix}
\]

which has all eigenvalues with negative real part by the Routh-Hurwitz stability conditions (Pielou, 1977). Thus, \( n^* \) is asymptotically stable.

On the other hand, the density dynamic at equilibrium frequency is

\[
\begin{align*}
\dot{N}_1 &= N_1 \left( 2 - \frac{5}{4} N_1 + \frac{1}{2} N_2 \right) \\
\dot{N}_2 &= N_2(-5 + 3N_1 - N_2).
\end{align*}
\]

The linearization about the equilibrium \( N_1^* = 2, N_2^* = 1 \) has Jacobian matrix

\[
\begin{bmatrix}
-\frac{5}{2} & 1 \\
3 & -1
\end{bmatrix}
\]

which is unstable since its determinant is negative.

\(^{10}\)This same notation is also used in the proofs of Theorems 2 and 4 to 6 in the Appendix. We also denote \( p^1 \) and \( p^2 \) by \( p \) and \( q \) respectively as well as \( e_i^1 \) and \( e_i^2 \) by \( e_i \) and \( f_i \) respectively.
By this example, we have an asymptotically stable two species equilibrium of (7) whose corresponding density dynamic (8)(i) is unstable. Similar elementary two species examples show a two species equilibrium of (7) can be unstable even though both the density dynamic and the induced frequency dynamic on the stationary density surface are asymptotically stable.

To see why Theorem 3 does not generalize to two or more species, consider the linearization of (8) at a two species equilibrium

\[ n^* = (N^*, p^*) \]

where species one and two have \( s_1 \) and \( s_2 \) phenotypes respectively. It can be shown that this linearization, which generalizes (10), becomes

\[
\begin{bmatrix}
\dot{N}_1 \\
\dot{N}_2 \\
\dot{p}_1 \\
\vdots \\
\dot{q}_{s_2}
\end{bmatrix} =
\begin{bmatrix}
\frac{\partial N_1}{\partial N_1} & \frac{\partial N_1}{\partial N_2} & -\frac{\partial N_1(p,q)}{\partial p_1} & \cdots & -\frac{\partial N_1(p,q)}{\partial q_{s_1}} \\
\frac{\partial N_2}{\partial N_1} & \frac{\partial N_2}{\partial N_2} & -\frac{\partial N_2(p,q)}{\partial p_1} & \cdots & -\frac{\partial N_2(p,q)}{\partial q_{s_1}} \\
\frac{\partial p_1}{\partial N_1} & \frac{\partial p_1}{\partial N_2} & \cdots & \cdots & \\
\vdots & \vdots & & & \\
\frac{\partial q_{s_2}}{\partial N_1} & \frac{\partial q_{s_2}}{\partial N_2} & & & \\
\end{bmatrix}
\begin{bmatrix}
\dot{N}_1 \\
\dot{N}_2 \\
\dot{p}_1 \\
\vdots \\
\dot{q}_{s_2}
\end{bmatrix} +
\begin{bmatrix}
\dot{N}_1 \\
\dot{N}_2 \\
p_1 - p_1^* \\
\vdots \\
q_{s_2} - q_{s_2}^*
\end{bmatrix}.
\]

(12)

Here \((N_1(p,q), N_2(p,q))\) is the stationary density surface and \(J\) is the \((s_1 + s_2) \times (s_1 + s_2)\) Jacobian matrix of the frequency dynamic on the stationary density surface. The Jacobian of the density dynamic is the \(2 \times 2\) matrix in the upper left-hand corner and \(J\) is in the lower right-hand corner. However, the linearization is not block diagonal since entries such as \(-\frac{\partial N_1(p,q)}{\partial p_1}\) or \(-\frac{\partial q_{s_1}}{\partial N_1}\) need not be zero in general.11

11In Section 3.1 for single species models, we saw an important case where all \(\frac{\partial b_i}{\partial N}\) are zero. It can also be shown that the linearization is lower block diagonal when \(\frac{\partial N_1(p,q)}{\partial p_i} = \frac{\partial N_2(p,q)}{\partial q_i} = 0\) for all \(i, j\) and similarly for \(N_2\). In particular, this is the case when the equilibrium densities \(N_1^*\) and \(N_2^*\) are local maxima (or minima) on the stationary density surface. This situation arises naturally in certain models of natural selection (see, for example, Roughgarden (1979) who exploited this result to great advantage).
On the other hand, the separation of frequency and density effects can be used to analyze the stability of (7) when each species has one resident phenotype but, unlike the monomorphic analysis of Section 2, any number of invading phenotypes. This is summarized in the following theorem whose proof is in the Appendix.

**Theorem 4** Assume $n^*$ is a hyperbolic equilibrium of (7) for which $n_{1}^{*k} > 0$ and $n_{i}^{*k} = 0$ if $i > 1$ for all $k$ (i.e. each species has exactly one resident phenotype at equilibrium). Then $n^*$ is asymptotically stable if and only if $N^*$ is asymptotically stable for the density dynamic at equilibrium frequency (i.e. the resident system is ecologically stable) and $p^*$ is asymptotically stable for the frequency dynamic induced by (8)(ii) on the stationary density surface.

It is now readily apparent that the frequency dynamic on the stationary density surface plays a pivotal role in the stability analysis of evolutionary Lotka-Volterra systems (e.g. Theorems 2, 3 and 4). The following section analyzes this dynamic more completely as it develops a close connection between the monomorphic and polymorphic models. For this purpose, the possibility individuals can play mixed strategies becomes important.

### 3.3 The Mixed Strategy Model

This model has the same interaction system of matrices $\{A^{kl}\}_{k,l=1,...,N}$ and the same strategy simplex $\Delta^k$ for each species $k$ as the polymorphic model introduced at the beginning of Section 3. However, individuals in species $k$ may now play a mixed strategy $p^k$ in $\Delta^k$ where the component $p^k_i$ of $p^k$ specifies the probability this individual uses the $i^{th}$ phenotype in species $k$ at
a given time. Then, $p^k \cdot A^{kl} p'$ becomes the expected individual payoff for an individual playing strategy $p^k \in \Delta^k$ when interacting with strategy $p' \in \Delta^l$.

For instance, suppose there are two possible mixed strategies in use by species $k$, $p^*k$ and $p^k$, and these have densities $\rho_k$ and $\mu_k$ respectively. Then the corresponding mixed strategy polymorphic evolutionary Lotka-Volterra system is none other than the monomorphic evolutionary Lotka-Volterra system with payoff parameters $m_{kR}^{RR} = p^*k \cdot A^{kl} p^*l$, $m_{kR}^{RI} = p^*k \cdot A^{kl} p^l$, $m_{kl}^{IR} = p^k \cdot A^{kl} p^*l$ and $m_{kl}^{II} = p^k \cdot A^{kl} p^l$. If $n^*$ is in the positive orthant, then automatically all invading phenotypes in this monomorphic system are selectively neutral and so the frequency component of the system is monomorphic evolutionarily stable if and only if the associated matrix $M$ is a B-matrix (see Theorem 1). On the other hand, if individuals in species $k$ exhibit only pure strategies, the mixed strategy evolutionary Lotka-Volterra system reverts back to the polymorphic evolutionary Lotka-Volterra system (7).

Thus, the mixed strategy model includes both the monomorphic model of Section 2 and the polymorphic model at the beginning of Section 3. Our discussion of the mixed strategy model emphasizes these two situations. The following result (proved in the Appendix) connects stability of the frequency dynamics for the monomorphic and polymorphic evolutionary Lotka-Volterra systems when there are at most two species.

**Theorem 5** Suppose $p^*$ is the frequency component of an equilibrium of the polymorphic evolutionary Lotka-Volterra system (7) and that $N \leq 2$. If $p^*$ is an asymptotically stable equilibrium of the frequency dynamic induced by the monomorphic evolutionary Lotka-Volterra system (2) on its stationary density surface for any choice of invading (mixed) strategy vector $p$, then $p^*$
is asymptotically stable for the frequency dynamic induced by (8)(ii) on the stationary density surface.

Theorem 5 is not true when there are more than two species. Cressman et al. (2001) produce an explicit counterexample of a three species frequency model (that can be extended to our frequency/density evolutionary systems) to show there exists an equilibrium $p^*$ that is asymptotically stable for all monomorphic models but not asymptotically stable for the polymorphic frequency dynamic corresponding to (8)(ii) on the stationary density surface.\textsuperscript{12}

In the above counterexample, the polymorphic frequency dynamic restricted to the equilibrium resident strategies is unstable. When this resident system is asymptotically stable, we have the following extension of Theorem 5 that is valid for any number of species. Its proof is in the Appendix.

\textbf{Theorem 6} Suppose $p^*$ is the frequency component of an equilibrium for the polymorphic evolutionary Lotka-Volterra system (7) and that $p^*$ is an asymptotically stable hyperbolic equilibrium of the resident frequency dynamic induced by (8)(ii) on the stationary density surface. If $p^*$ is asymptotically stable for the frequency dynamic induced by the monomorphic evolutionary Lotka-Volterra system (2) on its stationary density surface for any choice of invading (mixed) strategy vector $p$, then $p^*$ is asymptotically stable for the frequency dynamic induced by (8)(ii) on the stationary density surface.

\textbf{Remark 1} Theorems 5 and 6 are related to the “ESS program” proposed\textsuperscript{12}They also show that polymorphic stability can be recovered if more stringent conditions are placed on the frequency dynamic than monomorphic evolutionarily stable. One such condition is called \textit{Replicator-Lyapunov stability} in Cressman et al. (2001). By Lyapunov function methods, this condition guarantees global asymptotic stability in their frequency dependent evolutionary model.

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by Cressman (1992). In the single species case, this program starts with the monomorphic dynamic for an invading phenotype and then finds static criteria (such as our Theorems 1 and 2 for the multi species model) that imply stability in these systems for all possible monomorphic invasions. These are the ESS conditions. The basic question then becomes whether these ESS conditions guarantee stability in the polymorphic model. That is, in biological terms, the question is whether the original aim of Maynard Smith (1982) to use static game theory concepts to predict population behavior can be successfully extended to complex ecosystems. Theorems 5 and 6 give positive results for the frequency component of polymorphic Lotka-Volterra systems that can then be combined with an analysis of the density dynamic (as in Theorem 4) to imply polymorphic dynamic stability.

4 Discussion

The evolutionarily stable ecosystem concept developed in this paper generalizes the original ESS concept by combining multi species evolutionary games with density dependent ecological models that have linear payoff functions. It goes further than early treatments of coevolution (e.g. Riechert and Hammerstein, 1983) since we do not neglect simultaneous invasion by several mutants or the effect of evolving densities. It also fills a gap in more recent nongenetic and nonspatial models of phenotypic coevolution (e.g. Marrow et al., 1996; Vincent et al., 1996). In these models, individual fitness is usually assumed to depend nonlinearly on population density and/or phenotypic frequency. The first reference concentrates on a “strategy dynamic” that corresponds in our model to the monomorphic frequency dynamic. The second reference
assumes mutants have strictly lower fitness. In fact, their stability results (especially for equilibria corresponding to polymorphic situations) rely on this nonlinearity so that alternative phenotypes do strictly worse than the residents. When mutants are selectively neutral compared to the residents, we do not ignore the initially extremely rare interactions between mutants since they cannot be considered as negligible in the stability analysis (Garay, 2002) (see also Meszéna et al. (2001) for an analysis of single species “matrix games”).

The dual monomorphic/polymorphic analyses continue the methods initiated by Cressman and co-workers (e.g. Cressman and Dash, 1987; Cressman, 1992; Cressman et al., 2001) for more elementary models. Although monomorphic stability no longer implies polymorphic stability when there are three or more species, the separation of density and frequency dynamics developed in Sections 2 and 3 respectively shows how ecological stability and evolutionary stability effects combine in both models. (It must be noted here that such a separation exists but it is not quite as straightforward as fixing density at equilibrium values when analyzing the frequency dynamic, especially when there are two or more species.) These results suggest the original goal of evolutionary game theory to predict long term population behavior can be implemented in evolutionary ecology as well.

The separation of frequency and density can be applied successfully in general evolutionary ecosystems that do not assume the linearity of our evolutionary Lotka-Volterra system. The methods have the potential to analyze specific ecosystems. For instance, Hammerstein and Riechert (1988) develop a practical single species game-theoretic application to spiders while Levin...
and Muller-Landau (2000) investigate strategy stability in seed dispersal in plants. Both these models contain strategic analyses that ignore the density dynamics which, as we have seen, may lead to incorrect conclusions concerning the ecosystem’s stability.

Applications to epidemic models or to host-parasite interactions can also be regarded as problems in evolutionary ecology. These models often involve nonlinear genetic and/or spatial effects. In particular, the standard multi-species coevolutionary models of density-dependent viability selection at a single autosomal locus summarized by Ginzburg (1983) (see also Levin, 1978) are versions\textsuperscript{13} of our polymorphic dynamic (8) with genotypic fitnesses \(w_{ij}^k\) for species \(k\). In the special case that \(w_{ij}^k\) is frequency independent (i.e. only depends on the species’ densities \(N_1, \ldots, N_N\)), these references show that, generically, \((N^*, p^*)\) is an asymptotically stable equilibrium if and only if \(N^*\) is asymptotically stable for the density dynamic at equilibrium frequencies \(p^*\) and \(p^*\) is asymptotically stable for the frequency dynamic at fixed densities (cf. Theorem 3 above and the discussion at the end of Section 3.1). When each species has exactly one resident allele at equilibrium, Theorem 4 extends their results to the important nongeneric case of simultaneous invasion by selectively neutral mutant alleles. On the other hand, Example 1 shows Ginzburg’s result for polymorphic \(p^*\) does not generalize to genetic coevolutionary models that are both frequency and density dependent, especially

\begin{flushright}
\textsuperscript{13}Explicitly, the continuous-time density-dependent viability selection equations are \\
\(\dot{N}_k = N_k \vec{w}^k, \dot{p}_i^k = p_i^k \left( \sum_j w_{ij}^k p_j^k - \vec{w}^k \right)\) where \(p_i^k\) is the frequency of the \(i^{th}\) allele of species \(k\) at a single locus and \(\vec{w}^k = \sum_{i,j} p_i^k w_{ij}^k p_j^k\) is the expected fitness of species \(k\). Note that \(w_{ij}^k\) is assumed to be equal to \(w_{ji}^k\). In particular, Example 1 can be rewritten as a genetic model by taking, for instance, \(w_{11}^1 = 2 - N_2 - N_1 + N_2 p_1^1, w_{12}^1 = w_{21}^1 = 2 - N_1 + N_2 p_1^1, w_{22}^1 = 2 + N_2 - 2N_1 + N_2 p_1^1,\) and \(w_{11}^2 = -5 - N_2 + 3N_1.\)
\end{flushright}
when there is more than one species.

References


5 Appendix

The Appendix gives mathematical details for the proofs of all our results except Theorem 3. To reduce notational complexities, we restrict the proofs to two species evolutionary systems. However, the essential calculations are all provided in a matrix form that is easily generalized to more species.

**Proof of Theorem 1.** Let us rewrite (2), for $k = 1, 2$, as

$$
\begin{align*}
\dot{\rho}_k &= \rho_k \sum_{l=1}^{2} \left( m_{kl}^{RR} (\rho_l - \rho_l^*) + m_{kl}^{RI} \mu_l \right) \\
\dot{\mu}_k &= \mu_k \sum_{l=1}^{2} \left( m_{kl}^{IR} \rho_l - m_{kl}^{RR} \rho_l^* + m_{kl}^{II} \mu_l \right).
\end{align*}
$$

The linearization about $(\rho^*, 0)$ of this four-dimensional dynamic is

$$
\begin{bmatrix}
\dot{\rho}_1 \\
\dot{\rho}_2 \\
\dot{\mu}_1 \\
\dot{\mu}_2
\end{bmatrix} \approx \begin{bmatrix}
\rho_1^* m_{11}^{RR} & \rho_1^* m_{12}^{RR} & \rho_1^* m_{11}^{RI} & \rho_1^* m_{12}^{RI} \\
\rho_2^* m_{21}^{RR} & \rho_2^* m_{22}^{RR} & \rho_2^* m_{21}^{RI} & \rho_2^* m_{22}^{RI} \\
0 & 0 & \lambda_1 & 0 \\
0 & 0 & 0 & \lambda_2
\end{bmatrix}
\begin{bmatrix}
\rho_1 - \rho_1^* \\
\rho_2 - \rho_2^* \\
\mu_1 \\
\mu_2
\end{bmatrix}
$$

where $\lambda_k = (m_{k1}^{IR} - m_{k1}^{RR}) \rho_1^* + (m_{k2}^{IR} - m_{k2}^{RR}) \rho_2^*$. The $4 \times 4$ upper block diagonal Jacobian matrix has the same eigenvalues as $\text{diag}(\rho^*) M^{RR}$ (which, by
the ecological stability assumption, have negative real part\textsuperscript{14} together with $\lambda_1$ and $\lambda_2$. Standard eigenvalue analysis implies all but the final statement of the theorem.

When $\lambda_1 = \lambda_2 = 0$, centre manifold theory (Carr, 1981; Cressman, 1992) asserts there is a two-dimensional surface through $(\rho^*, 0)$ that is invariant under (2) and whose tangent space at $(\rho^*, 0)$ is the eigenspace corresponding to eigenvalue 0.

To calculate this centre manifold, we first change variables so that the linearization is block diagonal. Specifically, let $X \equiv \begin{bmatrix} \mu_1 \\ \mu_2 \end{bmatrix}$ and $Y \equiv \begin{bmatrix} \rho_1 - \rho_1^* \\ \rho_2 - \rho_2^* \end{bmatrix} + (M^{RR})^{-1}M^{RI}X$. From (13),

\[
\begin{align*}
\dot{X} &= \text{diag}(X) \left( M^{IR}Y - (M^{RR})^{-1}M^{RI}X \right) + M^{II}X \\
\dot{Y} &= \text{diag}(\rho^*)M^{RR}Y + (M^{RR})^{-1}M^{RI}\text{diag}(X) \left( M^{IR}Y - (M^{RR})^{-1}M^{RI}X \right) + M^{II}X
\end{align*}
\]

with linearization

\[
\begin{bmatrix} \dot{X} \\ \dot{Y} \end{bmatrix} = \begin{bmatrix} 0 & 0 \\ 0 & \text{diag}(\rho^*)M^{RR} \end{bmatrix} \begin{bmatrix} X \\ Y \end{bmatrix}
\]

where these 0’s are the $2 \times 2$ zero matrix.

Centre manifold theory implies the invariant surface has the form $Y = h(X)$ where $h(X)$ has no linear terms in $X$ and that $(\rho^*, 0)$ is locally asymptotically stable for (2) if and only if $X = 0$ is locally asymptotically stable for $\dot{X} = \text{diag}(X) \left( (M^{II} - M^{IR}(M^{RR})^{-1}M^{RI})X + M^{IR}h(X) \right)$. Since the term involving $h(X)$ has no linear or quadratic dependence, this can be ignored when applying Molchanov’s Theorem (Golster, 1997) which states that $X = 0$ is locally asymptotically stable if $M \equiv M^{II} - M^{IR}(M^{RR})^{-1}M^{RI}$ is a B-matrix. Conversely, Golster also showed that, if $X = 0$ is locally asymptotically stable for $\dot{X} = \text{diag}(\rho^*)M^{RR}$, then $M^{RR}$ is an invertible matrix if and only if $\text{diag}(\rho^*)M^{RR}$ is. This implies $\rho^*$ is the only equilibrium of the resident density dynamic in $R^N_{>0}$.

\textsuperscript{14}The $N \times N$ matrix $\text{diag}(\rho^*)M^{RR}$ is the matrix product of $\text{diag}(\rho^*)$, the diagonal $N \times N$ matrix with entries $\rho_1^*, ..., \rho_N^*$ down the main diagonal, and $M^{RR}$. In particular, $M^{RR}$ is an invertible matrix if and only if $\text{diag}(\rho^*)M^{RR}$ is. This implies $\rho^*$ is the only equilibrium of the resident density dynamic in $R^N_{>0}$. 
ically stable, then either $M$ is a B-matrix or there is some nonzero $X \in \mathbb{R}^{2}_{\geq 0}$ (called a zero ray) such that $X_k \sum_{l=1}^{2} M_{kl} X_l = 0$ for $k = 1, 2$. In this latter case, take $Y = 0$ and $X = sX_0$ for positive $s$. For $s$ sufficiently small, $(X, Y)$ corresponds to a point $\rho = \rho^* - (M^{RR})^{-1}M^{RI} sX_0, \mu = sX_0$ in the nonnegative orthant $\mathbb{R}^{4}_{\geq 0}$ where $X = 0 = \dot{Y}$ (i.e., to a rest point of (2)). Thus, in this latter case, $(\rho^*, 0)$ cannot be locally asymptotically stable and the proof is complete.

Proof of Theorem 2. From (13), the density dynamic is

$$\dot{N}_k = N_k \left( r_k + \sum_{l=1}^{2} N_l \left[ 1 - \epsilon_k, \epsilon_l \right] \begin{bmatrix} m_{kl}^{RR} & m_{kl}^{RI} \\ m_{kl}^{IR} & m_{kl}^{II} \end{bmatrix} \begin{bmatrix} 1 - \epsilon_l \\ \epsilon_l \end{bmatrix} \right).$$

(14)

For $\epsilon = (\epsilon_1, \epsilon_2) \in \mathbb{R}^{2}_{\geq 0}$ sufficiently close to zero, there is a unique $N(\epsilon) = (N_1(\epsilon), N_2(\epsilon)) \in \mathbb{R}^{2}_{\geq 0}$ with $\dot{N}_1 = \dot{N}_2 = 0$; namely, the solution of the matrix equation

$$\begin{bmatrix} (1 - \epsilon_1, \epsilon_1) \cdot A (1 - \epsilon_1, \epsilon_1) & (1 - \epsilon_1, \epsilon_1) \cdot B (1 - \epsilon_2, \epsilon_2) \\ (1 - \epsilon_2, \epsilon_2) \cdot C (1 - \epsilon_1, \epsilon_1) & (1 - \epsilon_2, \epsilon_2) \cdot D (1 - \epsilon_2, \epsilon_2) \end{bmatrix} \begin{bmatrix} N_1(\epsilon) \\ N_2(\epsilon) \end{bmatrix} = \begin{bmatrix} r_1 \\ r_2 \end{bmatrix}.$$  

(15)

Here $A \equiv \begin{bmatrix} m_{11}^{RR} & m_{11}^{RI} \\ m_{11}^{IR} & m_{11}^{II} \end{bmatrix}$ is the $2 \times 2$ payoff matrix for interactions between phenotypes in species one. Similarly, $D$ is the intraspecific payoff matrix for species two whereas $B$ and $C$ are the appropriate interspecific payoff matrices. Notice that, $N(0) = -(M^{RR})^{-1} \begin{bmatrix} r_1 \\ r_2 \end{bmatrix} = \rho^*$.

The frequency dynamic is

$$\dot{\epsilon} = \text{diag}(\epsilon_1(1-\epsilon_1), \epsilon_2(1-\epsilon_2)) \begin{bmatrix} (-1, 1) \cdot A (1 - \epsilon_1, \epsilon_1) & (-1, 1) \cdot B (1 - \epsilon_2, \epsilon_2) \\ (-1, 1) \cdot C (1 - \epsilon_1, \epsilon_1) & (-1, 1) \cdot D (1 - \epsilon_2, \epsilon_2) \end{bmatrix} \begin{bmatrix} N_1 \\ N_2 \end{bmatrix}.$$  

(16)

On the stationary density surface, the expansion about $\epsilon = 0$ is

\[\text{15} \text{The entries in } A \text{ are denoted by } a_{ij} \text{ (e.g. } a_{11} = m_{11}^{RR} \text{), those of } B \text{ by } b_{ij}, \text{ etc.}\]
\[
\dot{\epsilon}_1 = \epsilon_1 (1 - \epsilon_1) \left\{ (-1,1) \cdot A (1,0) \rho_1^* + (-1,1) \cdot B (1,0) \rho_2^* + \epsilon_1 (-1,1) \cdot A (-1,1) \rho_1^* + \epsilon_2 (-1,1) \cdot B (-1,1) \rho_2^* + (-1,1) \cdot A (1,0) \left( \frac{\partial N_1(\epsilon)}{\partial \epsilon_1} \right) \epsilon_1 + \frac{\partial N_1(\epsilon)}{\partial \epsilon_2} \epsilon_2 \right\} + (-1,1) \cdot B (1,0) \left( \frac{\partial N_2(\epsilon)}{\partial \epsilon_1} \epsilon_1 + \frac{\partial N_2(\epsilon)}{\partial \epsilon_2} \epsilon_2 \right) + \text{h.o.t.}
\]

where the higher order terms are at least quadratic in \( \epsilon \). All partial derivatives here are evaluated at \( \epsilon = 0 \) and there is a similar expression for \( \dot{\epsilon}_2 \) involving the payoff matrices \( C \) and \( D \). Now \( (-1,1) \cdot A (1,0) \rho_1^* + (-1,1) \cdot B (1,0) \rho_2^* = \left( m_{11}^{IR} - m_{11}^{RR} \right) \rho_1^* + \left( m_{12}^{IR} - m_{12}^{RR} \right) \rho_2^* = \lambda_1 \).

If \( \lambda_1 > 0 \) or \( \lambda_2 > 0 \), then \( \epsilon = 0 \) is unstable since at least one eigenvalue is positive. Similarly, if \( \lambda_1 < 0 \) and \( \lambda_2 < 0 \), then \( \epsilon = 0 \) is locally asymptotically stable. By the first paragraph in the statement of Theorem 1, this proves Theorem 2 except in the case where \( \lambda_1 = 0 \) and \( \lambda_2 \leq 0 \) (or the symmetric case with \( \lambda_1 \) and \( \lambda_2 \) interchanged). If \( \lambda_1 = 0 \) and \( \lambda_2 < 0 \), there is a one-dimensional centre manifold which determines local asymptotic stability. Here the manifold is the invariant edge \( \epsilon_2 = 0 \). The stability analysis reduces to a single species model, whereby the results in Section 2.1 imply the theorem.\(^{16}\)

Finally, assume \( \lambda_1 = 0 \) and \( \lambda_2 = 0 \) for the remainder of the proof. By differentiating (15) with respect to \( \epsilon_1 \) and evaluating at \( \epsilon = 0 \), we obtain

\[
\begin{bmatrix}
-2a_{11} + a_{12} + a_{21} & -b_{11} + b_{21} \\
-c_{11} + c_{12} & 0
\end{bmatrix}
\begin{bmatrix}
\rho_1^* \\
\rho_2^*
\end{bmatrix}
+ M^{RR}
\begin{bmatrix}
\frac{\partial N_1(\epsilon)}{\partial \epsilon_1} \\
\frac{\partial N_2(\epsilon)}{\partial \epsilon_1}
\end{bmatrix}
= \begin{bmatrix}
0 \\
0
\end{bmatrix}.
\]

Since \( \lambda_1 = 0 \), \( a_{11} \rho_1^* + b_{11} \rho_2^* = a_{21} \rho_1^* + b_{21} \rho_2^* \) and so

\[
\begin{bmatrix}
\frac{\partial N_1(\epsilon)}{\partial \epsilon_1} \\
\frac{\partial N_2(\epsilon)}{\partial \epsilon_1}
\end{bmatrix}
= (M^{RR})^{-1}
\begin{bmatrix}
-a_{11} + a_{12} & 0 \\
-c_{11} + c_{12} & 0
\end{bmatrix}
\begin{bmatrix}
\rho_1^* \\
\rho_2^*
\end{bmatrix}.
\]

\(^{16}\)When \( N > 2 \), there are more cases where \( \lambda_k \) is zero for some \( k \) and not for others. The proof then relies on an induction method on the number of \( \lambda_k = 0 \).
Similarly, \[ \frac{\partial N_1(\varepsilon)}{\partial N_2(\varepsilon_i)} = (M^{RR})^{-1} \begin{bmatrix} 0 & b_{11} - b_{12} \\ 0 & d_{11} - d_{12} \end{bmatrix} \begin{bmatrix} \rho_1^* \\ \rho_2^* \end{bmatrix} \]. Altogether, on the stationary density surface, we have

\[
\dot{\varepsilon}_1 = \varepsilon_1(1 - \varepsilon_1) \left\{ \varepsilon_1 (a_{11} - a_{12} + a_{22} - a_{21}) + \varepsilon_2 (b_{11} - b_{12} + b_{22} - b_{21}) \rho_2^* + [a_{21} - a_{11}, b_{21} - b_{11}] (M^{RR})^{-1} \begin{bmatrix} a_{11} - a_{12} & b_{11} - b_{12} \\ c_{11} - c_{12} & d_{11} - d_{12} \end{bmatrix} \begin{bmatrix} \varepsilon_1 \rho_1^* \\ \varepsilon_2 \rho_2^* \end{bmatrix} + \text{h.o.t.} \right\} 
= \varepsilon_1(1 - \varepsilon_1) \left\{ [a_{22}, b_{22}] - [a_{21}, b_{21}] (M^{RR})^{-1} \begin{bmatrix} a_{12} & b_{12} \\ c_{12} & d_{12} \end{bmatrix} \begin{bmatrix} \varepsilon_1 \rho_1^* \\ \varepsilon_2 \rho_2^* \end{bmatrix} + \text{h.o.t.} \right\}.
\]

Since \[ \begin{bmatrix} a_{12} & b_{12} \\ c_{12} & d_{12} \end{bmatrix} = M^{RI} \] etc., we obtain from an analogous expression for \( \dot{\varepsilon}_2 \),

\[
\dot{\varepsilon} = \text{diag}(\varepsilon) \left( (M^{II} - M^{IR} (M^{RR})^{-1} M^{RI}) \right) \begin{bmatrix} \varepsilon_1 \rho_1^* \\ \varepsilon_2 \rho_2^* \end{bmatrix} + \text{h.o.t.}
= \text{diag}(\varepsilon) M (\text{diag}(\rho^*)) \epsilon + \text{h.o.t.}
\]

If \( M(\text{diag}(\rho^*)) \) is a B-matrix, then \( \epsilon = 0 \) is locally asymptotically stable by Molchanov’s Theorem. By properties of B-matrices (Hofbauer and Sigmund, 1998), \( M(\text{diag}(\rho^*)) \) is a B-matrix if and only if its transpose \( \text{diag}(\rho^*) M^{tr} \) is a B-matrix if and only if \( M^{tr} \) is a B-matrix if and only if \( M \) is a B-matrix. Conversely, if \( \epsilon = 0 \) is locally asymptotically stable, then either \( M(\text{diag}(\rho^*)) \) is a B-matrix or there is a zero ray as in the proof of Theorem 1. If \( M(\text{diag}(\rho^*)) \) has a zero ray, then so does \( M \) and so we know that \((\rho^*, 0)\) is not locally asymptotically stable for (2) but it is possible that \( \epsilon = 0 \) is locally asymptotically stable for the frequency dynamic. This latter situation can only happen in degenerate circumstances (e.g. a necessary condition for this degeneracy is that \( M \) must not be invertible when restricted to some face of \( R^2_{\geq 0} \)).

**Proof of Theorem 4.** Following the argument in the proofs for Theorems 1 and 2, we can restrict our attention to the resident system together with
the selectively neutral invading system. Suppose there are \( s_1 - 1 \) and \( s_2 - 1 \) selectively neutral invading phenotypes in species one and two respectively. Furthermore, in our notation for polymorphic models, \( M^{RR} \) is now the \( 2 \times 2 \) matrix\(^{17} \) with entries \( M_{ij}^{RR} = A_{i1}^{ij} \), \( M^{II} \) is the \((s_1 + s_2 - 2) \times (s_1 + s_2 - 2)\) matrix with entries \( M_{ij}^{II} = A_{i1,j1}^k \), \( M^{RI} \) is the \( 2 \times (s_1 + s_2 - 2) \) matrix with entries \( M_{ij}^{RI} = A_{i1,j1}^{l} \), and \( M^{IR} \) is the \( (s_1 + s_2 - 2) \times 2 \) matrix with entries \( M_{ij}^{IR} = A_{i1,j1}^{k} \). Here

\[
k \equiv \begin{cases} 
1 & \text{if } i < s_1 \\
2 & \text{if } i \geq s_1
\end{cases}
\quad \text{and} \quad
l \equiv \begin{cases} 
1 & \text{if } i < s_2 \\
2 & \text{if } i \geq s_2
\end{cases}
\]

By the same argument as in the proof of Theorem 1, \( n^* \) is asymptotically stable if and only if all eigenvalues of the resident system have negative real part and the \((s_1 + s_2 - 2) \times (s_1 + s_2 - 2)\) matrix \( M^{II} - M^{IR} (M^{RR})^{-1} M^{RI} \) is a B-matrix.

On the stationary density surface, let \( \epsilon^1_i = p_{i+1} \) and \( \epsilon^2_i = q_{i+1} \). Then \( p^* = e_1 \), \( q^* = f_1 \), \( p = (1 - \sum_{i=2}^{s_1-1} \epsilon_i^1, \epsilon_1^1, \ldots, \epsilon_{s_1-1}^1) \), \( q = (1 - \sum_{i=2}^{s_2-1} \epsilon_i^2, \epsilon_1^2, \ldots, \epsilon_{s_2-1}^2) \) and the induced frequency dynamic is

\[
\dot{\epsilon}_i^1 = \epsilon_i^1 (e_{i+1} - p) \cdot [ApN_1(p,q) + BqN_2(p,q)]
\]
\[
\dot{\epsilon}_i^2 = \epsilon_i^2 (f_{i+1} - q) \cdot [CpN_1(p,q) + DqN_2(p,q)].
\]

There are no linear terms for this \((s_1 + s_2 - 2)\) dimensional dynamic on \( R_{s_1+s_2-2} \) since all invading phenotypes are selectively neutral (e.g. \( e_j \cdot [Ap^*N_1^* + Bq^*N_2^*] = e_1 \cdot [Ap^*N_1^* + Bq^*N_2^*] = 0 \) for all \( j \)). For the same reason, the quadratic terms

\(^{17}\)That is, \( M^{RR} = \begin{bmatrix} p^* \cdot Ap^* & p^* \cdot Bq^* \\ q^* \cdot Cp^* & q^* \cdot Dq^* \end{bmatrix} = \begin{bmatrix} A_{11} & B_{11} \\ C_{11} & D_{11} \end{bmatrix} \). Note that \( M^{RR} \) is again invertible since the resident system is hyperbolic.
\[
\dot{e}_i^1 = e_i^1 (e_{i+1} - e_1) \cdot \left[ \sum_{j=2}^{s_1} \left( A(e_j - e_1)N_1^* + Ae_1 \frac{\partial N_1(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_2(p,q)}{\partial e_j} \right) \right. \\
+ \sum_{j=2}^{s_2} \left( B(f_j - f_1)N_2^* + Ae_1 \frac{\partial N_2(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_2(p,q)}{\partial e_j} \right) \left. \right] e_{j-1}
\]

and an analogous expression for \(\dot{e}_i^2\).

As in the proof of Theorem 2,

\[
\begin{bmatrix}
p \cdot Ap & p \cdot Bq \\
q \cdot Cp & q \cdot Dq
\end{bmatrix}
\begin{bmatrix}
N_1(p,q) \\
N_2(p,q)
\end{bmatrix}
= - \begin{bmatrix}
r_1 \\
r_2
\end{bmatrix}.
\]

Differentiating with respect to \(\epsilon_j^k\) and evaluating at the equilibrium, we obtain

\[
\begin{bmatrix}
\frac{\partial N_1(p,q)}{\partial e_1} \\
\frac{\partial N_2(p,q)}{\partial e_1}
\end{bmatrix}
= -(M^{RR})^{-1} \begin{bmatrix}
e_1 \cdot A (\frac{\partial p}{\partial \epsilon_1}) & 0 \\
f_1 \cdot C (\frac{\partial p}{\partial \epsilon_1}) & 0
\end{bmatrix}
\begin{bmatrix}
N_1^* \\
N_2^*
\end{bmatrix}
\]

\[
= -(M^{RR})^{-1} \begin{bmatrix}
-M_{11}^{RR} + M_{1j}^{RI} & 0 \\
-M_{21}^{RR} + M_{2j}^{RI} & 0
\end{bmatrix}
\begin{bmatrix}
N_1^* \\
N_2^*
\end{bmatrix}
\]

\[
\begin{bmatrix}
\frac{\partial N_1(p,q)}{\partial e_2} \\
\frac{\partial N_2(p,q)}{\partial e_2}
\end{bmatrix}
= -(M^{RR})^{-1} \begin{bmatrix}
0 & -M_{1j}^{RR} + M_{1j+s_1-1}^{RI} \\
0 & -M_{2j}^{RR} + M_{2j+s_1-1}^{RI}
\end{bmatrix}
\begin{bmatrix}
N_1^* \\
N_2^*
\end{bmatrix}
\]

Now \(e_1 \cdot (A(e_j - e_1)N_1^* + Ae_1 \frac{\partial N_1(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_2(p,q)}{\partial e_j})\) and \(e_1 \cdot (B(f_j - f_1)N_2^* + Ae_1 \frac{\partial N_2(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_2(p,q)}{\partial e_j})\) both equal zero since, for instance, \((A_{1j} - A_{11}) N_1^* - [A_{11} \quad B_{11}] (M^{RR})^{-1} [-A_{11} + A_{1j} \quad 0] \begin{bmatrix}
N_1^* \\
N_2^*
\end{bmatrix} = \)
is a B-matrix if and only if
\[(A_{ij} - A_{11})N^*_i + (A_{11} - A_{1j})N^*_j = 0.\]
Thus \(\epsilon_i^1/\epsilon_i^2\) is equal to
\[
epsilon_i^1 \cdot \left( \sum_{j=2}^{s_1} \left( A(e_j - e_1)N^*_j + Ae_1 \frac{\partial N_i^*(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_i^*(p,q)}{\partial e_j} \right) \epsilon_j^1 \right) + \sum_{j=2}^{s_2} \left( B(f_j - f_1)N^*_2 + Ae_1 \frac{\partial N_i^*(p,q)}{\partial e_j} + Bf_1 \frac{\partial N_i^*(p,q)}{\partial e_j} \right) \epsilon_j^2 \right)
\]
\[
= \sum_{j=1}^{s_1-1} \left( M^{II}_{ij} - M^{IR}_{i1} \right) - \left( M^{IR}_{i1} \right. \\
\left. \sum_{j=1}^{s_2-1} \left( M^{II}_{i,j+s_1-1} - M^{IR}_{i2} \right) - \left( M^{IR}_{i2} \right) \right) \right) \right)
\]
\[
= \sum_{j=1}^{s_1-1} \left( M^{II}_{ij} - \left[ M^{IR}_{i1} M^{IR}_{i2} \right] \right) \right) \right)
\]
\[
= \sum_{j=1}^{s_2-1} \left( M^{II}_{i,j+s_1-1} - \left[ M^{IR}_{i1} M^{IR}_{i2} \right] \right) \right) \right)
\]

With \(\epsilon \equiv (\epsilon_1^1, ..., \epsilon_{s_1-1}^1, \epsilon_1^2, ..., \epsilon_{s_2-1}^2)\), we have
\[
\dot{\epsilon} = diag(\epsilon) \left( M^{II} - M^{IR} \left( M^{RR} \right)^{-1} M^{RI} \right) diag(N^*_1, ..., N^*_1, N^*_2, ..., N^*_2) \epsilon
\]
where \(diag(N^*_1, ..., N^*_1, N^*_2, ..., N^*_2)\) has the first \(s_1 - 1\) entries as \(N^*_1\) and the other entries \(N^*_2\). As in the proof of Theorem 2, \( \left( M^{II} - M^{IR} \left( M^{RR} \right)^{-1} M^{RI} \right) diag(N^*_1, ..., N^*_1, N^*_2, ..., N^*_2) \) is a B-matrix if and only if \( M^{II} - M^{IR} \left( M^{RR} \right)^{-1} M^{RI} \) is a B-matrix. This completes the proof.

Proof of Theorem 5. As shown in Section 3.1, the asymptotic stability of the frequency dynamic on the stationary density surface for the single species polymorphic Lotka-Volterra system is equivalent to that on the fixed density surface. By Section 2.1, an equilibrium of the monomorphic frequency dynamic for a single species is asymptotically stable if and only if it is an ESS. It is well-known that an ESS is an asymptotically stable equilibrium of the polymorphic frequency dynamic with fixed density (Cressman, 1992; Hofbauer and Sigmund, 1998). This completes the proof when \(N = 1\).
For \( N = 2 \), it is still true that, at fixed density, an equilibrium of the frequency dynamic \((p^*, q^*)\) that is asymptotically stable for all monomorphic systems is asymptotically stable for the polymorphic frequency dynamic (see Cressman et al, 2001). To complete the proof, it is then sufficient to show that the stationary density surface of each monomorphic Lotka-Volterra system with invading phenotypes \((p, q)\) is tangent to the stationary density surface of the polymorphic model in the direction from \((p^*, q^*)\) to \((p, q)\).

The proof rests on the calculation of the Jacobian of the induced frequency dynamic \( J \) in (12). For instance, from (8)(ii), \( \frac{\partial p}{\partial p_1} \) equals

\[
\begin{align*}
\frac{\partial N_1(p,q)}{\partial p_1} (e_i - p^*) \cdot Ap^* + \frac{\partial N_2(p,q)}{\partial p_1} (e_i - p^*) \cdot Bq^* \\
+ N_1 \frac{\partial (e_i - p) \cdot Ap}{\partial p_1} + N_2 \frac{\partial (e_i - p) \cdot Bq}{\partial p_1}
\end{align*}
\]

\begin{equation}
\begin{align*}
&= - \left[ (e_i - p^*) \cdot Ap^* \ (e_i - p^*) \cdot Bq^* \right] (M_{RR})^{-1} \begin{bmatrix} e_1 \cdot Ap^* + p^* \cdot Ae_1 & e_1 \cdot Bq^* \end{bmatrix} \begin{bmatrix} N_1^* \\ N_2^* \end{bmatrix} \\
&\quad + N_1^* (-e_i \cdot Ap^* + (e_i - p^*) \cdot Ae_1) + N_2^* (-e_i \cdot Bq^*) \\
&= -N_1^* \left[ e_i \cdot Ap^* \ e_i \cdot Bq^* \right] (M_{RR})^{-1} \begin{bmatrix} p^* \cdot Ae_1 \\ q^* \cdot Ce_1 \end{bmatrix} + N_1^* e_i \cdot Ae_1 \\
&\quad - \left[ e_i \cdot Ap^* \ e_i \cdot Bq^* \right] (M_{RR})^{-1} \begin{bmatrix} -r_1 \\ 0 \end{bmatrix}.
\end{align*}
\end{equation}

Since \(-p_1^* \left[ e_i \cdot Ap^* \ e_i \cdot Bq^* \right] (M_{RR})^{-1} \begin{bmatrix} -r_1 \\ 0 \end{bmatrix}\) does not depend on \( p_1 \), when we sum \( \frac{\partial p_i}{\partial p_1} (p_1^* - p_1) + \ldots + \frac{\partial p_i}{\partial p_{s_1}} (p_{s_1}^* - p_{s_1}) \), this term disappears. Thus the linearization for \( J \) is the same as for the frequency dynamic of the polymorphic model with payoff matrices given by the entries from the monomorphic stationary density surface. This completes the proof. \( \blacksquare \)

**Proof of Theorem 6.** By now familiar arguments \((p^*, q^*)\) is an asymptotically stable equilibrium of the frequency dynamic induced by (8)(ii) on the stationary density surface if and only if it is asymptotically stable for
the frequency dynamic restricted to the resident system combined with the selectively neutral invading (pure) strategies. Suppose there are $t_1$ and $t_2$ selectively neutral invading phenotypes in species one and two respectively. Since $(p^*, q^*)$ is asymptotically stable for the resident frequency dynamic on the stationary density surface, the same method used in the proof of Theorem 1 implies $(p^*, q^*)$ is asymptotically stable for the frequency dynamic induced by (8)(ii) on the stationary density surface if the $(t_1 + t_2) \times (t_1 + t_2)$ matrix $\tilde{M}$ corresponding to $M^R - M^R (M^{RR})^{-1} M^{RI}$ is a B-matrix.\textsuperscript{18} This method also shows there is a $(t_1 + t_2)$ dimensional invariant centre manifold in $\Delta^1 \times \Delta^2$ through $(p^*, q^*)$ parameterized by $X \in \mathbb{R}_{\geq 0}^{t_1 + t_2}$ sufficiently close to zero.

To show $\tilde{M}$ is a B-matrix, we must show that $X_i e_i \cdot \tilde{M} X < 0$ for some $i$ whenever $X$ is a nonzero element of $X \in \mathbb{R}_{\geq 0}^{t_1 + t_2}$. Given such an $X$ sufficiently close to zero, let $(p, q)$ be the point in $\Delta^1 \times \Delta^2$ parameterized by $X$. By (17), the B-matrix stability condition for the monomorphic model is that either $\sum_{i=1}^{t_1} X_i e_i \cdot \tilde{M} X < 0$ or $\sum_{i=t_1+1}^{t_2} X_i e_i \cdot \tilde{M} X < 0$. Thus asymptotic stability of every monomorphic model implies $X_i e_i \cdot \tilde{M} X < 0$ for some $i$. \hfill \blacksquare

\textsuperscript{18}Entries in these matrices are expressions such as $\frac{\partial (\dot{p}_i / p_i)}{\partial p_i}$ given in (17).