EVOLUTIONARY DISTRIBUTIONS IN ADAPTIVE SPACE

YOSEF COHEN

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An evolutionary distribution (ED), denoted by $z(\mathbf{x},t)$, is a distribution of density of phenotypes over a set of adaptive traits $\mathbf{x}$. Here $\mathbf{x}$ is an n-dimensional vector that represents the adaptive space. Evolutionary interactions among phenotypes occur within an ED and between EDs. A generic approach to modeling systems of ED is developed. With it, two cases are analyzed. (1) A predator prey inter-ED interactions either with no intra-ED interactions or with cannibalism and competition (both intra-ED interactions). A predator prey system with no intra-ED interactions is stable. Cannibalism destabilizes it and competition strengthens its stability. (2) Mixed interactions (where phenotypes of one ED both benefit and are harmed by phenotypes of another ED) produce complete separation of phenotypes on one ED from the other along the adaptive trait. Foundational definitions of ED, adaptive space, and so on are also given. We argue that in evolutionary context, predator prey models with predator saturation make less sense than in ecological models. Also, with ED, the dynamics of population genetics may be reduced to an algebraic problem. Finally, extensions to the theory are proposed.

1. Introduction

The theory that links evolution and population genetics is well developed (e.g., Hartl and Clark [10]). The theory that links evolution and population ecology is progressing rapidly. Both of these theories are part of a "grander scheme." At the risk of simplifying more than necessary, Figure 1.1 illustrates the interactions among the relevant fields of study and where this manuscript fits. The link between evolutionary ecology and population genetics is important because evolution by natural selection works directly on phenotypes and indirectly on genotypes.

Evolutionary ecology models—that is, models that integrate evolutionary processes with population ecology—usually start with

$$\dot{\mathbf{z}} = \mathbf{H}(\mathbf{x},\mathbf{z})\mathbf{z}, \tag{1.1}$$

where $\mathbf{z}$ is a vector, $\mathbf{H}$ is a matrix of "instantaneous fitness" functions and $\mathbf{x}$ is a vector (possibly of vectors) of adaptive traits—all of appropriate dimensions. Dots denote

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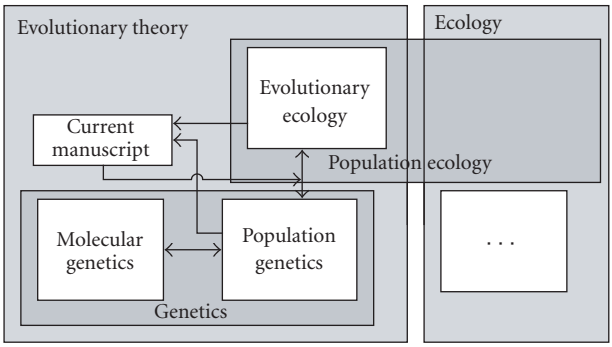

Figure 1.1

derivatives with respect to time. To (1.1) one then adds dynamics of the adaptive traits (called strategies)

$$\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x}, \mathbf{z}). \tag{1.2}$$

With these two equations, ecological and evolutionary dynamics become intertwined (Brown and Vincent [4]; Abrams et al. [1]; Vincent et al. [25]; Vincent et al. [26]; Taylor and Day [23]; Cohen et al. [7]). This starting point—sparked by Maynard-Smith's [13] original work—turns the system into an evolutionary game. The approach above has variations such as matrix games and differential games. One important variation is the inclusion of space. This turns a model of a coevolutionary system from ordinary differential equations to a system of partial differential equations. The edited volume by Dieckmann et al. [9] is a good recent reference on the subject (in particular Chapter 22).

The approach in (1.1) and (1.2)—and its relatives—triggered a profound change in the perception of coevolution—one no longer looks to maximize adaptations under constraints (essentially an optimization criterion), but rather maximize adaptations in the context of other interacting organisms whose adaptations are maximized by coevolution (essentially a game theoretic criterion). These developments in evolutionary ecology parallel those in economics, where starting with Nash's [16] work, emphasis has shifted from optimization to game theoretic solutions of capitalistic market problems.

The game theoretic criterion in evolutionary ecology is called evolutionarily stable strategies (ESS), introduced by Maynard-Smith [13]. According to this criterion, coevolving organisms exhibit a set of genetically-based adaptations that taken together prevent mutants from coexisting with the ESS set. The theory here builds on the foregoing by using the concept of evolutionary distributions (we denote by ED both evolutionary distribution and evolutionary distributions). In fact, stable ED correspond to ESS. Here, reaction-diffusion models are derived from first principles concerning ecological and evolutionary processes. Reaction-diffusion is a large topic both in mathematics and ecology. Smoller [22] and Murray [15] are good references on the subject. The former is more mathematically oriented than the latter. An expository account is given in Britton

[3]. Reaction-diffusion models are of considerable interest in ecology (Segel and Jackson [20]; Levin and Segel [12]; Rosen [19]; Mimura and Murray [14]; Okubo [17]; Conway [8]; Pease et al. [18]; Dieckmann et al. [8]; Alonso et al. [2]). A somewhat related approach to the one taken here was taken by Slatkin [21].

So here is the plan. First, we introduce the important idea of ED. The exposition follows Cohen [5, 6], where detailed proofs are given. Next, we discuss single ED. Here, we analyze the case where mutation rates along the adaptive trait are not equal in all directions. This case is compared to the one in Cohen [5], where equal mutation rates were analyzed. Next, we develop a generic approach to analyze multiple ED. This will be followed by analysis of predator prey interactions. Under predator prey interactions we consider the case where phenotypes within an ED compete and the case where cannibalism occurs. We then talk about mixed interactions where phenotypes in one ED benefit and harm phenotypes in their own ED and in another ED. We close with a discussion.

Before moving on, some notes about notation. $\triangleq$ means equal by definition and $\equiv$ means identically equal. Unless otherwise stated, identical symbols may represent a constant or a function; for example, β or $\beta(\cdot)$. Occasionally, identical symbols are used on both sides of an equation; for example, $\beta(x) \triangleq a\beta(x)$ says "redefines $\beta(x)$." Bold letters denote vectors and matrices (of constants or of functions). $\mathbf{x}$ sometimes represents a particular value, sometimes a space. When $\mathbf{x}$ represents a particular value, this value labels a phenotype. All parameters (constants) are nonnegative real numbers, all densities are nonnegative real numbers and all evolutionary traits are real numbers.

2. The adaptive space and ED

Appendix A includes mathematical definitions of adaptive spaces, evolutionary distributions and other terms used throughout. Heuristically, an adaptive space is constructed as follows: a phenotype is a realization of n values of adaptive traits $\mathbf{x}$. Here $\mathbf{x}$ is a collection of x_i, $i = 1,\ldots,n$ adaptive traits. Each x_i is drawn from a set (bounded or not) of real numbers. The restriction to real numbers is not necessary, but we will stick with it here. A point in the adaptive space, $\mathbf{x} = [x_1,\ldots,x_n]$ represents a phenotype. That phenotype is labeled $\mathbf{x}$. The adaptive traits must be chosen such that they are independent. In other words, we choose the minimum number n that describes the adaptive traits. It should be noted that x may represent a linear combination of some values. For example, if height and weight are perfectly correlated, then they do not represent two adaptive traits. Their combination is a single adaptive trait.

Evolution by natural selection results in changes in the density of phenotypes in the adaptive space. An adaptive trait is a set of values (say height, weight) that a phenotype possesses. The values are heritable and are subject to natural selection. Therefore, trait values are linked to the density of phenotypes that express particular trait values. An evolutionary distribution (ED) is defined as a time dependent distribution of phenotypes in the adaptive space where the dynamics involve evolutionary processes (e.g., competition that results in different phenotypes, mutations and assortative mating). A complete description of the dynamics of ED of a biotic community with m distinct groups of functional organisms is given by $\mathbf{z}(\mathbf{x},\mathbf{y}(t),t)$. Here $\mathbf{z}$ is an m-component vector of ED, $\mathbf{x}$ is

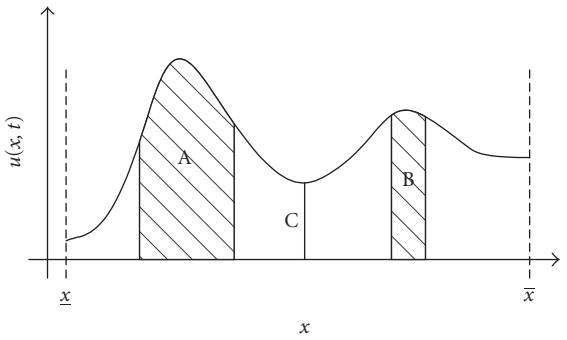

Figure 2.1

a vector of n values of n adaptive traits and $\mathbf{y}$ is a vector of p (possibly time dependent) values of input components. As in Cohen [5, 6], we absorb $\mathbf{y}$ into $\mathbf{x}$. The above are descriptive definitions. Explicit definitions upon which the following theory relies are given in Appendix A.

An ED describes changes in the density of phenotypes $z(\mathbf{x},t)$ along $\mathbf{x}$ for which the (frequency) consequences of changes in the values of $\mathbf{x}$ are similar. This concept corresponds to what Brown and Vincent [4] called evolutionary identical species. These are organisms that function similarly in biotic communities. For example, a single ED corresponds to prey. Predators are described by another ED. Two ED might have seemingly common $\mathbf{x}$. However, because the dynamics on $\mathbf{x}$ for one functional group of organisms differ from that of another, $\mathbf{x}$ in one ED is interpreted differently from that in another ED. For example, if x refers to phenotypes' size in an ED of prey and in an ED of predators, then x should be treated as having two scales. In fact, within an ED scale is comparable among phenotypes, between it is not.

The theory to be discussed does not require a rigid taxonomic classification of phenotypes; for example, species, sub-species and so on. It requires the concept of *evolutionary type* (or simply *type*). A type is defined as a set of individuals whose trait values form an interval on $\mathbf{x}$; this interval contains special points on $\mathbf{z}(\mathbf{x},t)$. Special points are, for example, local extrema. An ED at a particular t and for $n = 1$ and $m = 1$ is illustrated in Figure 2.1 (after Cohen [6]). This definition of types is by no means the most general. Yet, it is adopted here for simplicity.

The areas **A** and **B** in Figure 2.1 isolate two types. **C** isolates another. The classification of organisms into types is arbitrary. It depends on biological and ecological details and the scale of the trait. Scale does not pose a problem because all organisms that belong to a single ED are evolving under the same scale. For example, x may represent tree-height. An observer walking through a forest might surmise that the ED in Figure 2.1 represents a forest with 3 main types: common short and tall trees and rare middle sized trees. The choice of interval width is left to the observer and the biological details.

Only scalar adaptive traits are considered. Examples are height, weight, speed and length. Because x represents a biological trait, it is to be bounded—except where specified—between $\underline{x}$ and $\overline{x}$ (Figure 2.1). Therefore, in considering the dynamics of ED,

we require that the first derivatives of the ED at the boundaries be zero (Neumann conditions). Dirichlet's represent reasonable boundary conditions. The Neumann conditions only are addressed here. To achieve zero derivatives on the boundaries, we will use, without loss of generality, sinusoid functions. These also represent nonhomogeneous perturbations of the ED.

ED trace evolutionary dynamics. As such, understanding them is important. Adaptations by natural selection occur directly through phenotypes and indirectly through genotypes—one can hardly expect environmental vicissitudes, for example, to change the frequency of genes directly. Therefore, by studying the dynamics of ED one can hope to reduce population genetics dynamics to an algebraic problem.

3. Example of ED construction

We mostly deal with a single evolutionary trait and with two ED. This may raise two legitimate questions: how do we derive the ED equations? How applicable is the theory to adaptive spaces with higher dimensions? Here is an example of how ED are constructed from first principles for $n = 2$ and $m = 2$. We are going to show that as in all cases (here and below), we get ED equations of the form

$$\mathbf{z}_t = \mathbf{A}(\cdot)\mathbf{z}_{\mathbf{xx}} + \mathbf{f}(\mathbf{z}), \tag{3.1}$$

where

$$\mathbf{z}_t \triangleq \begin{bmatrix} \partial_t z_1 \\ \partial_t z_2 \end{bmatrix}, \qquad \mathbf{z}_{\mathbf{xx}} \triangleq \begin{bmatrix} \partial_{x_1 x_1} z_1 \\ \partial_{x_2 x_2} z_2 \end{bmatrix}, \qquad \mathbf{f}(\mathbf{z}) \triangleq \begin{bmatrix} f_1(\mathbf{z}) \\ f_2(\mathbf{z}) \end{bmatrix} \tag{3.2}$$

and $\mathbf{A}(\cdot)$ is an appropriate matrix.

3.1. The ED. Consider two ED, $\mathbf{z} \triangleq [z_1, z_2]$. The adaptive trait of z_i is x_i and let $\mathbf{x} \triangleq [x_1, x_2]$. We model coevolution through influence on mortality. Let $\beta_i(\cdot)$ and $\mu_i(\cdot)$ denote the birth and mortality rate functions. The starting point is

$$\partial_t z_i(\mathbf{x}, t) = \beta_i(\cdot) - \mu_i(\cdot) \tag{3.3}$$

with initial conditions

$$\mathbf{z}(\mathbf{x}, 0) = \mathbf{z}_0(\mathbf{x}), \tag{3.4}$$

where $\mathbf{z}_0(\mathbf{x})$ are given nonnegative functions and boundary conditions

$$\partial_{x_1} z_i(\underline{\mathbf{x}}, t) = \partial_{x_2} z_i(\overline{\mathbf{x}}, 0) = 0. \tag{3.5}$$

3.2. Births. Consider birth rates linearly density dependent with mutation rates η_i. Then

$$\beta_1(\cdot) \triangleq \underbrace{(1-\eta_1)\beta_1 z_1(x_1, x_2, t)}_{\text{births no mutations}} + \underbrace{\frac{1}{2}\eta_1\beta_1 z(x_1 - \Delta x_1, x_2, t)}_{\text{births mutations up}} + \underbrace{\frac{1}{2}\eta_1\beta_1 z_1(x_1 + \Delta x_1, x_2, t)}_{\text{births mutations down}}. \tag{3.6}$$

Rearranging, we get

$$\begin{aligned}\beta_1(\cdot) = \beta_1 z_1(x_1,x_2,t) + \frac{1}{2}\eta_1\beta_1\Big[\underbrace{z_1(x_1-\Delta x_1,x_2,t)-z_1(x_1,x_2,t)}_{A}\Big]\\ + \frac{1}{2}\eta_1\beta_1\Big[\underbrace{z_1(x_1+\Delta x_1,x_2,t)-z_1(x_1,x_2,t)}_{B}\Big].\end{aligned} \tag{3.7}$$

Taylor series expansion of A and B give

$$\begin{aligned}A &= -\partial_{x_1} z_1(x_1,x_2,t)\Delta x_i + \frac{1}{2}\Delta x_1^2\partial_{x_1x_1} z_1(x_1',x_2,t),\\ B &= \partial_{x_1} z_1(x_{1i},x_2,t)\Delta x_1 + \frac{1}{2}\Delta x_1^2\partial_{x_1x_1} z_1(x_1'',x_2,t),\end{aligned} \tag{3.8}$$

where $x_1-\Delta x_1 \le x_1'', x_1' \le x_1+\Delta x_1$. With small Δx_1 we write

$$A + B \cong \Delta x_1^2\partial_{x_1x_1} z_i(x_1,x_2,t). \tag{3.9}$$

Therefore,

$$\beta_i(\cdot) = \beta_i z_i(\mathbf{x},t) + \eta_i\beta_i\Delta x_i^2\partial_{x_ix_i} z_i(\mathbf{x},t). \tag{3.10}$$

3.3. Deaths. Suppose that z_2 harms z_1, and z_1 benefits z_2. All interactions are local with respect to the adaptive traits and they affect changes in death rates. Death rates are assumed quadratically density dependent. Then

$$\begin{aligned}\mu_1(\cdot) &\triangleq \big[\mu_1 z_1(x_1,x_2,t) + c_1\big(z_2(x_1,x_2-\Delta x_2,t)\\ &\qquad + z_2(x_1,x_2,t) + z_2(x_1,x_2+\Delta x_2,t)\big)\big]z_1(x_1,x_2,t),\\ \mu_2(\cdot) &\triangleq \Big[\mu_2 z_2(x_1,x_2,t) - c_2\big(z_1(x_1-\Delta x_1,x_2,t)\\ &\qquad + z_1(x_1,x_2,t) + z_1(x_1+\Delta x_1,x_2,t)\big)\Big]z_1(x_1,x_2,t).\end{aligned} \tag{3.11}$$

We take $c_2 < c_1$. Following the procedure for $\beta_i(\cdot)$, we get

$$\mu_1(\cdot) = \big[\mu_1 z_1(\mathbf{x},t) + c_1\big[\Delta x_2^2\partial_{x_2x_2} z_2(\mathbf{x},t) + 3z_2(\mathbf{x},t)\big]\big]z_1(\mathbf{x},t). \tag{3.12}$$

Similarly

$$\mu_2(\cdot) = \big[\mu_2 z_2(\mathbf{x},t) - c_2\big[\Delta x_1^2\partial_{x_1x_1} z_1(\mathbf{x},t) + 3z_1(\mathbf{x},t)\big]\big]z_2(\mathbf{x},t). \tag{3.13}$$

3.4. The ED. Substituting birth and death rates into (3.3), we obtain (3.1) with

$$\mathbf{A}(\mathbf{z}) \triangleq \Delta^2\begin{bmatrix}\eta_1\beta_1 & -c_1z_1\\ c_2z_2 & \eta_2\beta_2\end{bmatrix}, \qquad \mathbf{f}(\mathbf{z}) = \begin{bmatrix}[\beta_1-\mu_1z_1-3c_1z_2]z_1\\ [\beta_2-\mu_2z_2+3c_2z_1]z_2\end{bmatrix}. \tag{3.14}$$

Here $\Delta^2 \triangleq \Delta x_i^2$. The ED discussed below are all constructed similarly.

4. Intra- and inter-ED generic interactions

The starting point is the set of equations

$$\partial_t z_i(\mathbf{x},t) = \beta_i(\mathbf{z}(\mathbf{x},t)) - \mu_i(\mathbf{z}(\mathbf{x},t)), \quad i = 1,\ldots,m,\ \mathbf{x} \in U \subset \mathbb{R}^n \tag{4.1}$$

with data

$$\mathbf{z}(\mathbf{x},0) = \mathbf{z}_0(\mathbf{x}), \qquad \partial_x \mathbf{z}(\underline{\mathbf{x}},t) = \partial_x \mathbf{z}(\overline{\mathbf{x}},t) = \mathbf{0}, \tag{4.2}$$

where m is the number of ED and $\underline{\mathbf{x}}, \overline{\mathbf{x}} \in \partial U$. Here $\beta_i(\mathbf{z})$ and $\mu_i(\mathbf{z})$ reflect addition and subtraction processes from densities of phenotypes $\mathbf{x}$ where $\mathbf{x}$ is the phenotypic space: a collection of say n adaptive traits. $\mathbf{z}_0(\mathbf{x})$ is given. For definiteness, we call $\beta_i(\cdot)$ and $\mu_i(\cdot)$ the birth and death rates. By deriving the additions and subtractions from first principles, we will always end up with a set of R-D equations.

4.1. Births. With notable exceptions (see below), in many ecological interactions one can assume that natural selection acts on a particular $\mathbf{x}$ via mortality, rather than through birth. This is true even if the genetic makeup of a progeny renders it inviable. Death does not occur directly because of genetic makeup but because of some physical force acting on a phenotypic trait (e.g., organ failure). Therefore, we replace $\mathbf{z}$ with z_i in $\beta_i(\mathbf{z})$.

Now suppose that upon birth, a fraction, η_i, of the newborn mutate to $x \pm \Delta x$. As a first approximation, assume that birth rate is a linear function of phenotypes' density, with a constant coefficient β_i. Then

$$\beta_i(z_i) = \beta_i z_i + \eta_i \beta_i \Delta x^2 \partial_{xx} z_i, \tag{4.3}$$

where $z_i \equiv z_i(x,t)$ (see Cohen [5, 6] and the detailed example above).

4.2. Mortality. Now take $\mu_i(\mathbf{z})$ to be additive and proportional to z_i. We consider 3 sources that increase or reduce mortality of phenotype x: density dependent term, $\mu_i(x,t)z_i > 0$, changes due to intra-ED interactions, $\xi_i(z_i) \geq 0$, and changes due to inter-ED interactions $\theta_i(z_j) \geq 0$. Then

$$\mu_i(\mathbf{z}) = \left[\mu_i(x,t)z_i + (-1)^{l_i}\xi_i(z_i) + (-1)^{m_i}\theta_i(z_j)\right] \quad z_i > 0,\ i \neq j. \tag{4.4}$$

External forces (e.g., environment) that change mortality rates are incorporated via $\mu_i(x,t)$ (see Cohen [5, 6]). For now, we assume that interactions are localized. l_i and m_i take on the values 1 or 2. When $l_i = 2$, the intra-ED interactions among phenotypes result in increase in the mortality rate of phenotypes (e.g., competition). When $l_i = 1$, the mortality rate of x decreases (e.g., cannibalism). Similarly, m_i parameterize inter-ED interactions. Thus, various permutations of intra- and inter-ED interactions can be specified with l_i and m_i. This approach of generic interactions does not preclude the possibility that both competition and cannibalism, for example, may occur—because it is unlikely that the effect of both processes on mortality are exactly equal, the net effect within an ED will almost always be one of the 3—competition, cannibalism, or none. Similar comment applies to $\theta(\cdot)$. More generally, any intra- or inter-ED interactions boil down to m_i and l_i

equal either 1 or 2. In an even more general setting we may parameterize l_i and m_i with $l_i(x,t)$ and $m_i(x,t)$. For the time being, we will consider m_i and l_i constants. To a first approximation, take $\xi_i(\cdot)$ and $\theta_i(\cdot)$ to be linear functions. Because we consider localized interactions (on x), we have

$$\xi_i(z_i) = (-1)^{l_i} a_i [z_i(x-\Delta x,t) + z_i(x+\Delta x,t)]. \tag{4.5}$$

When $a_i = 0$ there are no intra-ED interactions. Similar to the derivation of (4.3), we obtain

$$\xi_i(z_i) = (-1)^{l_i} a_i [\Delta x^2 \partial_{xx} z_i + 2z_i]. \tag{4.6}$$

Here a_i scale the strength of the intra-ED interactions. When $a_i = 0$ there are no intra-ED interactions.

Regarding $\theta_i(\cdot)$, we write

$$\theta_i(z_j) = (-1)^{m_i} p_i [z_j(x-\Delta x,t) + z_j(x,t) + z_j(x+\Delta x,t)] \tag{4.7}$$

or

$$\theta_i(z_j) = (-1)^{m_i} p_i [\Delta x^2 \partial_{xx} z_j + 3z_j]. \tag{4.8}$$

Here p_i scale the strength of the inter-ED interactions. When $p_i = 0$, there are no inter-ED interactions.

This formulation encapsulates various interactions that affect the dynamics of ED. For example, with $l_i = 2$, $m_1 = 2$ and $m_2 = 1$ we have predator prey (but see below) coevolution with intra-ED competition.

4.3. The generic equation. With $\xi(\cdot)$ and $\theta(\cdot)$ linear, we have

$$\partial_t z_i = k_1(z_i,l_i)\partial_{xx} z_i - k_2(z_i,m_i)\partial_{xx} z_j + (\beta_i - k_3(z_i,l_i) - (-1)^{m_i} 3 p_i z_j) z_i, \tag{4.9}$$

where

$$\begin{aligned} k_1(z_i,l_i) &\triangleq \Delta x^2 (\beta_i \eta_i - (-1)^{l_i} a_i z_i), \\ k_2(z_i,m_i) &\triangleq (-1)^{m_i} \Delta x^2 p_i z_i, \\ k_3(z_i,l_i) &\triangleq (\mu_i + (-1)^{l_i} 2a_i) z_i. \end{aligned} \tag{4.10}$$

Now to set up the equation for z_i such that the net effect is $z_i(x,t)$ benefiting from $z_i(x \pm \Delta x,t)$, simply let $l_i = 1$. When $z_i(x,t)$ is harmed by $z_i(x \pm \Delta x,t)$, set $l_i = 2$. Set m_i similarly for inter-ED interactions. With no intra-ED interaction, set $a_i = 0$, and with no inter-ED interactions set $p_i = 0$.

For homogeneous equilibria, we have $\mathbf{f}(\mathbf{z}) = 0$ with the solution

$$\widetilde{z}_i \triangleq \frac{\beta_i \mu_j + (-1)^{l_j} 2a_j \beta_i - (-1)^{m_i} 3 p_i \beta_j}{A}, \tag{4.11}$$

where

$$A \triangleq [2(-1)^{l_1}a_1+\mu_1][2(-1)^{l_2}a_2+\mu_2]-9(-1)^{m_1+m_2}p_1p_2. \tag{4.12}$$

The solution of $\tilde{\mathbf{z}}$ does not shed much light on the stability of the homogeneous solution. It is also too complex, and needs to be analyzed numerically. We are interested in pursuing specific relations by building on the generic approach.

5. Predator prey

Let x be the prey's adaptive trait that influences its vulnerability to predators. x is also the predator's adaptive trait—it influences the predator's ability to catch prey. Let z_1 and z_2 denote the prey and predator ED. Predators $z_2(x,t)$ prey on $z_1(\phi x+j\Delta x,t)$, $j=-1,0,1$ where $0<\phi<M<\infty$. Similarly, prey $z_1(x,t)$ are eaten by predators $z_2(\phi^{-1}x+j\Delta x,t)$. For example, if x represents size, then ϕ scales the relation between prey and predator sizes. We will take $\phi=1$. The more general case where $\phi\neq 1$ is much more difficult to analyze.

A notable exception to (4.3) is predator prey interactions. Here, we must approach the effect of changes in the density of prey phenotypes on the density of predator phenotypes through $\beta_i(\mathbf{z})$ as opposed to through $\mu_i(\mathbf{z})$. This is so because we must assume that there could be no births of predators without the presence of prey. Then for the prey we have $\beta_1(z_1)$ as in (4.3). For the predators, we assume that $z_2(x,t)$ gives birth to $z_2(x+j\Delta x,t)$, $j=-1,0,1$ with mutation fraction η_2. Furthermore, $z_2(x,t)$ prey upon $z_1(x\pm j\Delta xt)$. Then

$$\beta_2(\mathbf{z})=p_2(1-2\eta_2)\beta_2[z_1+z_1(x-\Delta x,t)+z_1(x+\Delta x,t)]z_2, \tag{5.1}$$

where p_2 is the proportion of prey biomass that is transformed into predator biomass. This reduces to

$$\beta_2(\mathbf{z})=p_2(1-2\eta_2)[\Delta x^2\partial_{xx}z_1+3z_1]z_2. \tag{5.2}$$

So in predator prey interactions $\beta_1(\cdot)$ is given in (4.3) and $\beta_2(\cdot)$ is given above. What about mortality? The intra-ED portion of mortality is expressed in $\xi_i(z_i)$. The inter-ED portion has been traditionally modeled with saturation effects, *a la* Michaelis-Menten. In the ecological milieu, predator saturation makes sense; not so much in the evolutionary milieu. There is no *a priori* reason to presume that somehow predators will not continue to evolve so as to kill as many prey as available. Thus, we set $m_1=2$ and $\theta_2=0$. For the predators, θ_2 is not necessary as we already included the effect of predation by increasing the predators' birth rate (5.2). Therefore, for linear $\xi(\cdot)$ and $\theta(\cdot)$ we have

$$\begin{aligned}\mu_1(\mathbf{z})&=[\mu_1z_1+(-1)^{l_1}a_1(\Delta x^2\partial_{xx}z_1+2z_1)+p_1(\Delta x^2\partial_{xx}z_2+3z_2)]z_1,\\ \mu_2(\mathbf{z})&=[\mu_2z_2+(-1)^{l_2}a_2(\Delta x^2\partial_{xx}z_2+2z_2)]z_2.\end{aligned} \tag{5.3}$$

Here p_1 is the (biomass) fraction of prey removed by predators. In general, thermodynamic considerations dictate $p_1>p_2$. Furthermore, predators may inflict injuries on the prey and these injuries do not necessarily translate to predator biomass.

Inserting $\beta_i(\cdot)$ and $\mu(\cdot)$ from above into (4.1) and simplifying, we obtain (see Appendix C)

$$\partial_t \mathbf{z} = \mathbf{A}(\mathbf{z})\partial_{xx}\mathbf{z} + \mathbf{f}(\mathbf{z}), \tag{5.4}$$

where

$$\begin{aligned} \mathbf{A}(\mathbf{z}) &= \begin{bmatrix} \Delta x^2(\beta_1\eta_1 - (-1)^{l_1}a_1 z_1) & -\Delta x^2 p_1 z_1 \\ \Delta x^2 p_2(1-2\eta_2)\beta_2 z_2 & -(-1)^{l_2}\Delta x^2 a_2 z_2 \end{bmatrix}, \\ \mathbf{f}(\mathbf{z}) &= \begin{bmatrix} (\beta_1 - (\mu_1 + (-1)^{l_1}2a_1)z_1 - 3p_1 z_2)z_1 \\ (Kz_1 - (\mu_2 + (-1)^{l_2}2a_2)z_2)z_2 \end{bmatrix}, \end{aligned} \tag{5.5}$$

where

$$K \triangleq 3p_2\beta_2(1-2\eta_2). \tag{5.6}$$

Here K scales the conversion of prey biomass to the biomass of predators that do not mutate. With this formulation, we can examine predator prey interactions in evolutionary context with a variety of permutations of net intra-ED interactions. For example, for no intra-ED interactions we set $a_i = 0$; for cannibalism we set $l_i = 1$ and for competition $l_i = 2$. There are 9 permutations in all (e.g., $a_1 > 0$, $a_2 = 0$ and $l_1 = 1$ or 2). We will pursue only three cases: no intra-ED interactions, cannibalism and competition among prey phenotypes. In each case, we will see how some of the parameters influence the stability of the predator prey coevolutionary system.

5.1. No intra-ED interactions. To analyze this case, we set $a_i = 0$ in (5.5). A homogeneous equilibrium solution of (5.4) requires that $\partial_t \mathbf{z} = \mathbf{0}$ and $\partial_{xx}\mathbf{z} = \mathbf{0}$. In this case, the nontrivial equilibrium solution of $\mathbf{f}$ is

$$\widetilde{z}_1 = \frac{\beta_1\mu_2}{D}, \qquad \widetilde{z}_2 = \frac{K\beta_1}{D}, \tag{5.7}$$

where

$$D = 3Kp_1 + \mu_1\mu_2. \tag{5.8}$$

Here

$$\frac{\widetilde{z}_1}{\widetilde{z}_2} = \frac{\mu_2}{3p_2\beta_2(1-2\eta_2)}. \tag{5.9}$$

These equations lead to a few interesting conclusions. First, the equilibrium populations are not controlled by the mutation rate of the prey. Second, as the mutation rate of the predator increases (and remains below 0.5), K becomes smaller and the equilibrium populations of both predator and prey become larger; however, smaller K also results in a larger ratio of $\widetilde{z}_1$ to $\widetilde{z}_2$. In short, higher mutation rates by the predator leave

relatively more prey alive. This is so because large η_2 results in fewer births of $z_2(x,t)$—evolutionarily, a good reason for η_2 to remain small! Third, as expected, larger μ_1 results in smaller equilibrium populations while larger μ_2 results in larger prey populations.

At any rate, at the equilibrium solution the expressions for the eigenvalues of $\partial_{\mathbf{z}}\mathbf{f}(\breve{\mathbf{z}})$ are not pretty. However, the relevant parts of the expression (those that control the sign of the eigenvalues) are

$$\lambda_{1,2} \propto -A \pm \sqrt{B}, \tag{5.10}$$

where

$$\begin{aligned} A &= \sqrt{\mu_2}(\mu_1 + K), \\ B &= \mu_2(\mu_1^2 + K^2) - 2K(6p_1K + \mu_1\mu_2). \end{aligned} \tag{5.11}$$

We can reasonably expect $\eta_2 \ll 0.5$, and therefore $K > 0$. This means that the real part of one of the eigenvalues will always be negative. It turns out that $|A| > |\sqrt{B}|$. To see this, write the last condition as

$$A^2 > B \tag{5.12}$$

and simplify. We thus get

$$4\mu_1\mu_2 + 12Kp_1 > 0. \tag{5.13}$$

With $\eta_i \ll 0.5$, this expression is true for any parameter values. From this we conclude that regardless of the parameter values, we always get homogeneous stable equilibrium. A rather dull model one might say. Is this reason enough to reject the generic system (5.4)-(5.5) as reasonable for coevolutionary predator prey ED? The model, albeit a first approximation, is based on first principles and we may simply have to admit that nature may be sometimes dull (or equivalently that predator prey cycles are controlled by cyclic input to the system). Furthermore, we are yet to examine the system with intra-ED interactions. We purse two specific cases in the next section. Finally, the road to stable equilibrium or unreasonable instability (where phenotypes densities become infinite) is of interest. After all, if that road is taken slowly, we are likely to observe its expression in nature. One might even argue that all extinctions are eventually certain, and therefore the road to stable equilibrium or unreasonable instability may even be more interesting than the stable equilibrium itself. Here is an example.

Example 5.1. We use (5.4) and (5.5) with parameters

$$\begin{aligned} &a_1 = a_2 = 0, \quad \beta_1 = 1, \quad \beta_2 = 0.2, \quad \mu_1 = 0.01, \quad \mu_2 = 0.02, \\ &p_1 = 0.1, \quad p_2 = 0.01, \quad \eta_1 = \eta_2 = 0.01, \quad \Delta x = 0.001 \end{aligned} \tag{5.14}$$

(the parameter values, albeit in the range expected, are set for illustrative purposes only) and data

$$z_1(x,0) = 9 + 2\cos(x), \qquad z_2 = 3 + \cos(x), \qquad \partial_x\mathbf{z}(0,t) = \partial_x\mathbf{z}(4\pi,t) = 0. \tag{5.15}$$

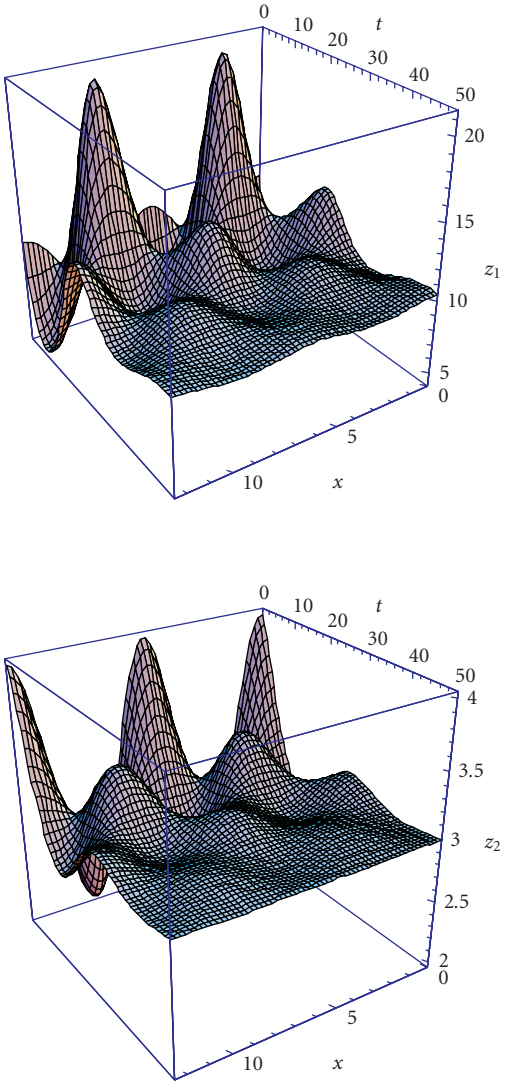

Figure 5.1

Figure 5.1 illustrates the road to stable equilibrium. A particular cross section ($t = 15$) is compared to the initial data (Figures 5.2 and 5.3).

The example illustrates interesting dynamics. One might start with 5 types of prey and 5 types of predators, but along the way the predators cause increase in biodiversity and we observe 9 types of prey, while the predators remain with 5 types. Note also that in some cases, the densities of types for prey and predators are reversed (with some phase shifts

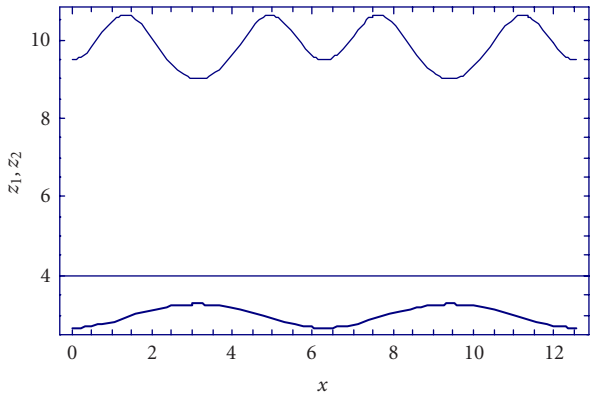

Figure 5.2

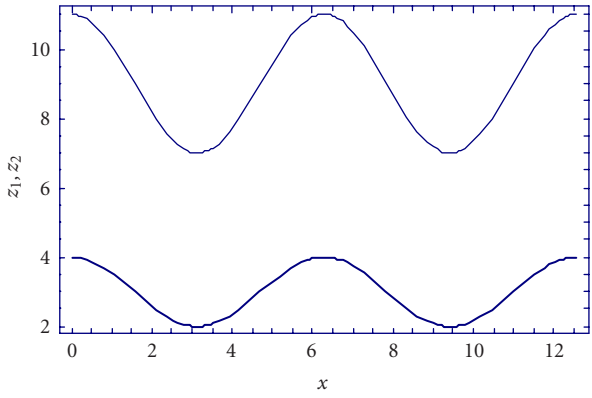

Figure 5.3

along x). So along the way to homogeneous equilibrium we start with certain biodiversity. It increases (due to predation) and then ends up with homogeneous equilibrium.

5.2. Cannibalism among preys. For $a_1 > 0$, $a_2 = 0$, and $l_1 = 1$, we obtain

$$\tilde{z}_1 = \frac{\beta_1 \mu_2}{D'}, \qquad \tilde{z}_2 = \frac{K\beta_1}{D'}, \tag{5.16}$$

where

$$D' = D - 2a_1\mu_1. \tag{5.17}$$

Here again $\widetilde{z}_1/\widetilde{z}_2$ is as in (5.9). However, the equilibria in this case are larger because of the relation between D' and D. At the equilibrium solution the expressions for the eigenvalues of $\partial_{\mathbf{z}}\mathbf{f}(\widetilde{\mathbf{z}})$ are

$$\lambda_{1,2} = \frac{\beta_1\sqrt{\mu_2}}{2D'}\left[-\left(A - 2a_1\sqrt{\mu_2}\right) \pm \sqrt{B'}\right], \tag{5.18}$$

where B' is an ugly expression. However, things can be simplified considerably. For the system to be stable, we require that

$$\left(A - 2a_1\sqrt{\mu_2}\right) > 0, \qquad \left(A - 2a_1\sqrt{\mu_2}\right)^2 > B'. \tag{5.19}$$

The last equation simplifies to the requirement that

$$D' > 0 \tag{5.20}$$

or

$$9p_1p_2\beta_2(1 - 2\eta_2) + \mu_1\mu_2 - 2a_1\mu_2 > 0. \tag{5.21}$$

All of the parameters are positive fractions. In particular, $p_1 \cdot p_2$ is on the order of 10^{-2}. Therefore, a_1 must be small for the above condition to be satisfied. In other words, the scale of cannibalism must be small.

All else being equal, as a_1 increases from 0 (the case of no intra-ED interactions), the system moves from stability to instability as one of the eigenvalues moves from negative to positive values.

5.3. Competition among the preys. Here we take $a_1 > 0$, $a_2 = 0$ and $l_1 = 2$. The equilibrium solution is as in (5.16), except that now

$$D' = D + 2a_1\mu_1. \tag{5.22}$$

In other words, $\widetilde{\mathbf{z}}$ with competition $< \widetilde{\mathbf{z}}$ with no intra-ED interactions $< \widetilde{\mathbf{z}}$ with cannibalism (where interactions are among prey phenotypes). The eigenvalues of $\partial_{\mathbf{z}}\mathbf{f}(\widetilde{\mathbf{z}})$ are now

$$\lambda_{1,2} = \frac{1}{2D'}\left(A \pm \sqrt{B'}\right), \tag{5.23}$$

where

$$A = -\beta_1\mu_2(2a_1 + K + \mu_1) \tag{5.24}$$

and B' not pretty. However,

$$-A^2 + B' = -4\beta_1^2\mu_2\left[3Kp_1 + \mu_2(2a_1 + \mu_1)\right]. \tag{5.25}$$

This expression will always be negative and therefore $\lambda_{1,2}$ are negative and the system is stable. In other words, competition stabilizes the system compared to cannibalism.

Time to examine a different case.

6. Mixed interactions

Let x be the adaptive trait of z_1 and of z_2. Mutations η_i occur at birth to $x \pm \Delta x$. Assume that $z_1(x,t)$ benefit from $z_2(x,t)$. However, $z_1(x,t)$ are harmed by $z_2(x \pm \Delta x, t)$. Similarly, $z_2(x,t)$ benefit from $z_1(x,t)$ but are harmed by $z_1(x \pm \Delta x)$. Ecologically, this is a version of specific symbiotic relations. If z_i do not exactly fit, they actually harm each other. Then from Appendix C,

$$\begin{aligned} \mathbf{A}(z) &= \Delta \begin{bmatrix} \beta_1\eta_1 - (-1)^{l_1}a_1z_1 & -p_1z_1 \\ -p_2z_2 & \beta_2\eta_2 - (-1)^{l_2}a_2z_2 \end{bmatrix}, \\ \mathbf{f}(z) &= \begin{bmatrix} (\beta_1 - (\mu_1 + (-1)^{l_1}2a_1)z_1 - p_1z_2)z_1 \\ (\beta_2 - (\mu_2 + (-1)^{l_2}2a_2)z_2 - p_2z_1)z_2 \end{bmatrix} \end{aligned} \tag{6.1}$$

where $\Delta \triangleq \Delta x^2$ (compare to (5.5)). Again, several combinations can be used to reflect intra-ED interactions by setting $l_i = 1,2$. The analytical expressions for $\tilde{\mathbf{z}}$ and λ are messy and not very revealing. However, when values are assigned to the parameters, it turns out that for $a_i \geq 0$ and a variety of combinations of $l_i = 1,2$ we get a pair of real eigenvalues; one negative and one positive. The dynamics of the ED are truly remarkable. Over time, we get a complete separations of phenotypes along x. Here is an example.

Example 6.1. The parameter values are:

$$\begin{aligned} a_i = 0, \quad \eta_i = 0.01, \quad \beta_1 = 0.4, \quad \beta_2 = 0.5, \\ \mu_i = 0.01, \quad \Delta x = 0.001, \quad p_i = 0.1. \end{aligned} \tag{6.2}$$

The data are

$$\begin{aligned} z_1(x,0) &= 4.6 + 0.46\cos(4x), \\ z_2(x,0) &= 3.5 + 0.35\cos(4x), \\ \partial_x\mathbf{z}(0,t) &= \partial_x\mathbf{z}(4\pi,t) = \mathbf{0}. \end{aligned} \tag{6.3}$$

With these and the assumption of homogeneous equilibrium, we get $\tilde{\mathbf{z}} = (4.6, 3.5)$ and $\lambda_{1,2} = (-0.6, 0.2)$. The evolutionary dynamics of the ED are shown in Figures 6.1 and 6.2. The cross sections at $t = 0$ and $t =$"∞" are shown in Figure 6.3.

Figures 6.1 and 6.2 show the coevolution to stable ED. The coevolution to stable ED are also illustrated in Figure 6.3. Here the cross sections at $t = 0$ (thick curve) and $t = 1000, 2000$. For $t = 1000$ and 2000 the cross sections are indistinguishable for both ED.

The results of this example merit a few comments. The coevolutionary process spaces z_1 and z_2 such that phenotypes with similar traits values from different ED do not overlap. If the amplitudes b in the initial conditions $a + b\cos(4x)$ are sufficiently small, one ends with a homogeneous equilibrium ED. In other words, the peculiar biodiversity here can be produced only with sufficiently nonhomogeneous (along x) perturbations. This conclusion was first suggested and observed by Turing [24]; see also discussion in Levin [11]. Based on the mutual benefits of specific phenotypes and mutual harm to other phenotypes, one would have expected the ED of both functional types to overlap considerably.

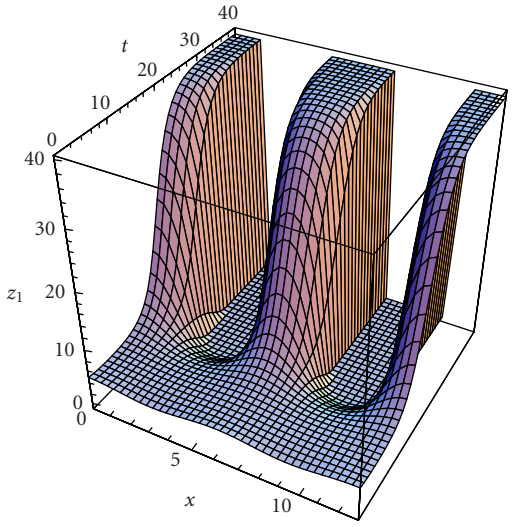

Figure 6.1

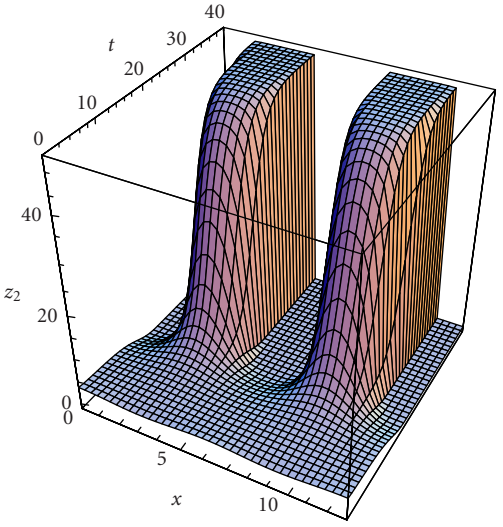

Figure 6.2

In fact, the opposite happens—a form of "coevolutionary exclusion." Finally, because interactions are local, we end with 3 types of z_1 that do not interbreed and 2 types of z_2 that do not interbreed.

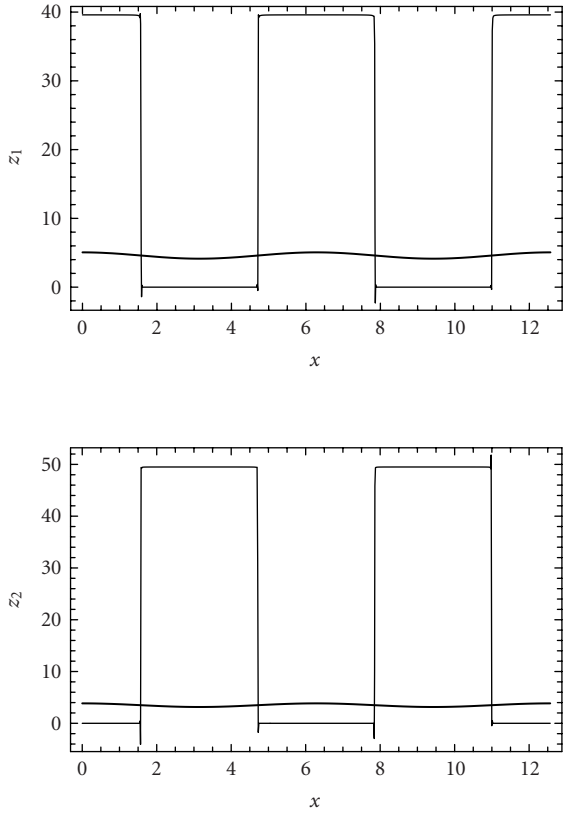

Figure 6.3

7. Discussion

The ED approach raises legitimate questions. One might argue that a population is composed of discrete units (individuals) and thus there are no individuals of phenotype $x \pm \Delta x$ when Δx is sufficiently small. Like any other model of diffusion, the continuous approach is based on small scale averaging. This is justified only for large populations. The approach taken here does not apply to small populations, where individual based stochastic models are appropriate.

Three important issues are challenging. First, the dimensionality of the adaptive space (i.e., the dimensionality of $\mathbf{x}$) is imposed on the system *a priori*. A fundamental generalization of the theory should provide the means to decide on the appropriate dimensionality. One may even go further and say that the ED dimensionality is in fact a dynamic feature of the system. This boils down to a fundamental question in the theory of evolution: what is the meaning of "innovative" adaptation? Perhaps it is a new dimension in the adaptive space. Of course what might look as an innovative adaptation on one scale

(say organismal) may not be on another (say molecular). Furthermore, without clear definitions (e.g., as in Appendix A) of concepts such as evolutionary innovation , I do not know how to begin addressing this question.

Second, how would assortative mating and intra-ED interactions—among phenotypes that are not necessarily close—affect the interactions among evolutionary distributions? One can go even further and ask: how can the theory lead to the emergence (evolution) of assortative mating and specific intra-ED rules? Indications are that competition among distant phenotypes, for example, end in smoothing the nonhomogeneous equilibrium of predator prey relations compared to x-local competition (see Cohen [6]).

Third, inter-ED interactions—such as predator prey—among functionally different organisms are specified arbitrarily. How can the theory give rise to such interactions? In other words, can the theory give rise to the emergence of one ED from another? Of course uncovering such generalizations will lead to ever more fundamental questions and eventually to the "theory of everything" (and perhaps of nothing). Needless to say, at this time, I have no answers to these questions.

A flicker of how some of these questions may be addressed appears in (4.9): one may consider $k_i(\cdot)$ to be unknown functions whose solutions give rise to intra- and inter-ED interactions. This requires articulation of some game-theoretic or optimization criteria. Solving for the criteria, with (4.9) as a constraint, should result in appropriate $k_i(\cdot)$. Also, one may approach ED as a single hyper-distribution with added dimension x_{n+1}. This requires a fundamental shift in our concept of phenotypes function in ecosystems. They may be prey at one time and predators at another—all in a single adaptive space $\mathbf{x}$.

The approach taken here lends itself to extensions with little conceptual (but otherwise much) effort. For example, x can be taken as $\mathbf{x}$ of arbitrary dimension and $\mathbf{z}$ can have a dimension larger than 2. In fact, spatial coordinates can be viewed as values of adaptive characters, particularly for sessile organisms. Yet, even with low dimensional ED we already get rich results. Of course the examples are limited to the specific models of intra- and interspecific interactions. Yet, they establish possible consequences of the theory.

Appendices

A. Definitions

The theory discussed in this manuscript relies, implicitly, on the following formal definitions. $\mathbb{R}^n$ denotes an n-dimensional Euclidean space. Each dimension is represented by the extended real line (but see Comments below).

Adaptive space and phenotypes. Each dimension in $\mathbb{R}^n$ represents an adaptive trait. Because of physical and biological constraints, adaptive traits take an open bounded set of real values, $\Omega \subset \mathbb{R}^n$. The set Ω is said to be the *adaptive space*. Elements of Ω, denoted by $\mathbf{x} = [x_1, \ldots, x_n]$, are called *points*. An organism that possesses a point, say $\mathbf{x}$, is called a *phenotype* and is labeled by $\mathbf{x}$.

Adaptive trait. Let $\mathbf{e}$ be the basis of Ω. Then each $\mathbf{e}_k$, $k = 1, \ldots, n$ represents an *adaptive trait*. Let x_k be the value of the kth adaptive trait. x_k are heritable (with mutations). x_k, as expressed in phenotypes are subject to natural selection.

Natural selection. Phenotypes are characterized by $\mathbf{x} = \sum_{k=1}^{n} x_k \mathbf{e}_k$ where x_k is a value of the adaptive trait k for the phenotype $\mathbf{x}$. The otherwise (biologically) inherent mortality of each $\mathbf{x}$ is influenced by three factors: $\mathbf{x}$'s interactions with the environment, $\mathbf{x}$'s interactions with phenotypes and chance. The contribution of the first two factors to the mortality of $\mathbf{x}$ constitute *natural selection*. So natural selection can be decomposed into *external* and *internal*. External is due to physiological and other constraints (e.g., resistance to extreme heat and cold). Internal is due to interactions among phenotypes (e.g., competition).

Heritable trait. A trait is said to be heritable if a progeny of $\mathbf{x}$ becomes $\mathbf{y}_m = \mathbf{x} + \sum_{k=1}^{n} (j_{mk}\Delta x)\mathbf{e}_k$ where Δx is small and $j_{mk} = -1$, 0 or 1. Here $m = 1,\ldots,$ *total number of progeny of* $\mathbf{x}$.

Mutation. The term $\sum_{k=1}^{n} (j_{mk}\Delta x)\mathbf{e}_k$ is said to be *mutation*. If, for a given m, $j_{mk} = 1$ or -1 for at least one k, then $\mathbf{y}_m$ is said to be the mth *mutant* with respect to the $\mathbf{x}$ phenotype.

Type. Choose points of interest $\mathbf{p}_i \in \Omega$, $i = 1,\ldots,s$. Now define a *type* A_i, $i = 1,\ldots,s$ as follows:

$$A_i = \{\mathbf{x} : d(\mathbf{x},\mathbf{p}_i) < \delta_i, \text{ where } \delta_i \text{ are chosen such that } A_i \text{ are disjoint}\}. \tag{A.1}$$

Here $d(\mathbf{a},\mathbf{b})$ denotes the distance (based on an appropriate norm) between $\mathbf{a}$ and $\mathbf{b}$ and $\delta_i > 0$. Phenotypes $\mathbf{x}_p \in A_i$ are said to be of type A_i, where $A_i \subset \Omega$.

Evolutionary distribution (ED). Let $z(\mathbf{x},t)$, $\mathbf{x} \in \Omega$, $z \in \mathbb{R}_{0+}$ be the density of z with adaptive traits $\mathbf{x}$ at t. If the dynamics of z give rise to changes in the relative frequencies of $z(\mathbf{x},t)$ due to natural selection, then $z(\mathbf{x},t)$ is said to be an *evolutionary distribution*. If $\lim_{t\to\infty} z(\mathbf{x},t) = \widetilde{z}(\mathbf{x})$ exists, then $\widetilde{z}(\mathbf{x})$ is said to be *equilibrium ED*. If, in addition, $\widetilde{z}(\mathbf{x})$ is stable, then $\widetilde{z}(\mathbf{x})$ is said to be *stable ED*.

Evolutionary system. A collection of m ED, $\mathbf{z}(\mathbf{x},t)$, $\mathbf{z} \in \mathbb{R}_{0+}^{m}$, is called an *evolutionary system.*

Genotype. Let Γ be the set of all unique genes, g, among phenotypes that belong to a single ED z. We say that Γ is the genome of z. Let $\mathcal{P}(\Gamma)$ denote the power set of Γ. Choose $G \subset \mathcal{P}(\Gamma)$ such that $f^{-1} : G \to \Omega$ where f is one-to-one. G is said to be the *genetic space* that corresponds to the genome Γ. Now $f(A_i)$, $i = 1,\ldots,s$ induces a partition of G into disjoint subsets, $\{G_k\}$, where $\{G_k\}$ is said to be a *genotype* of z in the adaptive space Ω.

Comments. (i) To the best of my knowledge, this is the first time that such definitions are formalized in the above terms. Therefore, they may not be widely accepted.

(ii) A phenotype displays many traits. Not all of them adaptive. Because f is one-to-one (but not necessarily onto), the definitions allow for $g \notin G$. These are genes that do not have phenotypic expressions or genes whose expressions are not subject to natural selection. The reverse is not true. That is, all phenotypic traits are, by definition, heritable.

(iii) From the definitions, it is clear that G is constructed from Γ using the definitions of A_i and invoking f on A_i. The existence of f reduces the dynamics of population genetics to an algebraic problem whose solution is obtained from $\mathbf{z}$.

(iv) Because A_i are disjoint, $\bigcup A_i = \sum A_i$.

(v) $\sum_{i=1}^{s} A_i$ do not necessarily completely cover Ω. Here s is a particular measure of biodiversity.

(vi) From the definitions one concludes that $\mathbf{x}$ may be classified as one and only one type or as no-type at all. *No type* is defined as $\mathbf{x} \in (\sum_{i=1}^{s} A_i)^c$ where c denotes a complement.

(vii) A type is an abstract taxonomic classification. Depending on the biological details of the adaptive space Ω, a type may be a species, a subspecies, a phenotype $\mathbf{x}$, no type and so on.

(viii) Every ED has its own adaptive space Ω_z and genetic space G_z. When dealing with a single ED, the subscript is dropped. For example, in a predator prey system, one has two ED, z_1 and z_2 and two adaptive spaces, Ω_{z_1} and Ω_{z_2}. Furthermore, even if both ED are measured for identical traits (say size of phenotypes), x in Ω_{z_1} should be interpreted differently from x in Ω_{z_2}. This is so because the consequences of evolutionary changes (e.g., adaptation) in x in the two spaces may be different for the organisms in these spaces. $\cup\Omega_{z_i}$ may be interpreted as the *coevolutionary space*.

(ix) Stable ED correspond to ESS.

(x) The preceding definitions are not the most general. In nature, adaptive traits need not cover a contiguous interval on $\mathbb{R}$. Many adaptive trait values are not real numbers. For example, male-female is a binary trait. There are other traits that may be represented by integers. Yet others may be categorical. The definitions can be generalized by defining Ω in abstract metric spaces, instead of in $\Omega \subset \mathbb{R}^n$.

B. Derivation of (5.5)

We have

$$\begin{aligned}
\beta_1(z_1) &= \beta_1 z_1 + \eta_1\beta_1\Delta x^2\partial_{xx}z_1,\\
\beta_2(\mathbf{z}) &= p_2(1-2\eta_2)\beta_2\left[\Delta x^2\partial_{xx}z_1 + 3z_1\right]z_2,\\
\mu_1(\mathbf{z}) &= \left[\mu_1 z_1 + (-1)^{l_1}a_1\left(\Delta x^2\partial_{xx}z_1 + 2z_1\right) + p_1\left(\Delta x^2\partial_{xx}z_2 + 3z_2\right)\right]z_1,\\
\mu_2(\mathbf{z}) &= \left[\mu_2 z_2 + (-1)^{l_2}a_2\left(\Delta x^2\partial_{xx}z_2 + 2z_2\right)\right]z_2.
\end{aligned} \tag{B.1}$$

Thus,

$$\begin{aligned}
\partial_t z_1 &= \beta_1 z_1 + \eta_1\beta_1\Delta x^2\partial_{xx}z_1\\
&\quad - \left[\mu_1 z_1 + (-1)^{l_1}a_1\left(\Delta x^2\partial_{xx}z_1 + 2z_1\right) + p_1\left(\Delta x^2\partial_{xx}z_2 + 3z_2\right)\right]z_1\\
&= \Delta x^2\left(\eta_1\beta_1 - (-1)^{l_1}a_1 z_1\right)\partial_{xx}z_1 - p_1\Delta x^2 z_1\partial_{xx}z_2\\
&\quad + \left(\beta_1 - \mu_1 z_1 - 2(-1)^{l_1}a_1 z_1 - 3p_1 z_2\right)z_1,\\
\partial_t z_2 &= p_2(1-2\eta_2)\beta_2\left[\Delta x^2\partial_{xx}z_1 + 3z_1\right]z_2\\
&\quad - \left[\mu_2 z_2 + (-1)^{l_2}a_2\left(\Delta x^2\partial_{xx}z_2 + 2z_2\right)\right]z_2\\
&= p_2(1-2\eta_2)\beta_2\Delta x^2 z_2\partial_{xx}z_1 - (-1)^{l_2}a_2\Delta x^2 z_2\partial_{xx}z_2\\
&\quad + \left(3\beta_2 p_2(1-2\eta_2)z_1 - \mu_2 z_2 - (-1)^{l_2}a_2 2z_2\right)z_2.
\end{aligned} \tag{B.2}$$

Thus, we obtain (5.5).

C. Derivation of the mixed interaction equation

As before,

$$\beta_i(z_i) = \beta_i z_i + \beta_i \eta_i (\Delta x)^2 \partial_{xx} z_i. \tag{C.1}$$

Let x be the adaptive trait of z_1 and of z_2. Mutations η_i occur at birth to $x \pm \Delta x$. Assume that $z_1(x,t)$ benefit from $z_2(x,t)$. However, $z_1(x,t)$ are harmed by $z_2(x \pm \Delta x, t)$. Similarly, $z_2(x,t)$ benefit from $z_1(x,t)$ but are harmed by $z_1(x \pm \Delta x)$. Ecologically, this is a version of specific symbiotic relations. If z_i do not exactly fit, they actually harm each other. Based on the assumptions in Section 6 we obtain

$$\begin{aligned} \mu_i(\mathbf{z}) &= [\mu_i z_i + (-1)^{l_i} \xi_i(z_i) + (-1)^1 \theta_i(z_j)] z_i \\ &= [\mu_i z_i + (-1)^{l_i} a_i [\Delta x^2 \partial_{xx} z_i + 2 z_i] - \theta_i(z_j)] z_i. \end{aligned} \tag{C.2}$$

Here

$$\begin{aligned} \theta_i(z_j) &= p_i(z_j - z_j(x - \Delta x, t) - z_j(x + \Delta x, t)) \\ &= -p_i(z_j(x - \Delta x, t) - z_j + z_j(x + \Delta x, t) - z_j + z_j) \\ &= -p_i(\Delta x^2 \partial_{xx} z_j + z_j). \end{aligned} \tag{C.3}$$

So

$$\begin{aligned} \partial_t z_i &= \beta_i z_i + \beta_i \eta_i (\Delta x)^2 \partial_{xx} z_i - [\mu_i z_i + (-1)^{l_i} a_i (\Delta x^2 \partial_{xx} z_i + 2 z_i) + p_i (\Delta x^2 \partial_{xx} z_j + z_j)] z_i \\ &= (\Delta x)^2 (\beta_i \eta_i - (-1)^{l_i} a_i z_i) \partial_{xx} z_i - p_i \Delta x^2 z_i \partial_{xx} z_j + [\beta_i - (\mu_i + (-1)^{l_i} 2 a_i) z_i - p_i z_j] z_i. \end{aligned} \tag{C.4}$$

In matrix notation:

$$\partial_t \mathbf{z} = \mathbf{A}(z) \partial_{xx} \mathbf{z} + \mathbf{f}(z), \tag{C.5}$$

where $\mathbf{A}$ and $\mathbf{f}$ are given in (6.1).

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Yosef Cohen: Department of Fisheries and Wildlife, University of Minnesota, St. Paul, MN 55108, USA

Current address: Department of Desert Ecology, Institute for Dryland Environmental Research, Ben-Gurion University of the Negev, Sede Boqer 84490, Israel

E-mail address: yosefcohen@comcast.net

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Celso Grebogi, Department of Physics, King's College, University of Aberdeen, Aberdeen AB24 3UE, UK; grebogi@abdn.ac.uk