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Evolutionary distributions

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ABSTRACT

Aims: (i) Based on first principles, develop partial differential equation models of evolution by natural selection operating on phenotypic traits. (ii) Use the models to draw conclusions about possible outcomes of evolution, their stability, resistance to invasion, and co-existence of phenotypes.

Assumptions: (i) Populations are large. (ii) Mutations are random – they are introduced at birth and by immigration. (iii) Selection operates through mortality and emigration. (iv) The selection unit is a phenotype.

Conclusions: (i) Stable evolutionary distributions represent evolutionarily stable strategies in that the co-existing set of phenotypes cannot be invaded by mutants. (ii) Because adaptive traits are bounded, phenotypes evolving on the boundaries are subject to less mortality due to competition than those in the interior of the adaptive space. (iii) Phenotypic plasticity allows the increase in density of a prey phenotype that would otherwise be depressed – the density increases because prey evolve lower susceptibility to predation. (iv) Host–pathogen co-evolution can lead to stable (possibly Turing) pattern formation of a phenotype's density in the adaptive space. (v) Phenotypic co-evolution in model ecosystems can stabilize a potentially local chaotic dynamics.

Keywords: adaptive traits, competition, evolutionary distributions, host–parasite, predator–prey.

1. INTRODUCTION

Evolutionary distributions were first introduced by Cohen (2003a, 2003b). The concept is a generalization of some aspects of smooth evolutionary games (see Vincent and Brown, 2005). The main idea is that phenotypes evolve in a space of continuous adaptive traits. This means that mathematical models that encapsulate evolution must include a derivative of time with respect to the density of phenotypes. Now with small mutations, phenotypes also 'move' incrementally along their adaptive traits. This means that one must introduce derivatives (as many as the number of adaptive traits) with respect to the adaptive traits. Thus, one obtains

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a partial differential equation that describes evolution of phenotypes in time and adaptive space. Such an equation describes 'evolutionary distributions' (ED). If functionally different phenotypes participate in co-evolution, then a system of evolutionary distributions describes co-evolution.

Smooth (dynamical) evolutionary games represent a point process where one follows species co-evolution. The strategy dynamics then represent changes in the average phenotypes of a species. So behind strategy dynamics there is a distribution of phenotypes. ED capture these distributions.

This article is arranged as follows: Section 2 introduces some notation and provides the basis for the motivation that is introduced in Section 2.1. Section 2.3 moves from smooth evolutionary games to evolutionary distributions. This section also outlines how to derive ED from first principles. Section 3 places a number of ecological relationships (competition, predator–prey, host–pathogen, and ED embedded in ecosystems) in the context of ED. Section 4 summarizes interesting consequences of ED.

The main focus of this article is to introduce a theoretical framework. This framework is less specific and more precise than ED as introduced by Cohen (2003a, 2003b). One should realize that the theory did not arise out of thin air. Darwinian smooth evolutionary games, as introduced by Vincent and Brown, are the basis from which the ED framework has been implemented.

2. BACKGROUND

Notation := denotes equality by definition. $z \equiv z\,(t)$ says that z equals $z\,(t)$ for all t. So,

$$z' := \frac{\mathrm{d}z(x)}{\mathrm{d}x}, \quad \partial_{xx} z\,(x, t) := \frac{\partial^2 z\,(x, t)}{\partial x^2}.$$

$x \in \mathcal{X}$ means that x is a member of the set $\mathcal{X}$ and $\mathcal{X} \subset \mathbb{R}$ means that $\mathcal{X}$ is a subset of $\mathbb{R}$, where $\mathbb{R}$ denotes the real numbers. $\mathbb{R}^n$ denotes the n-dimensional space of real numbers, $\mathbb{R}_{0+}$ denotes the set of non-negative real numbers, and $\mathbb{R} \times \mathbb{R}$ denotes the Cartesian product of two real lines (a plane). $f: \mathcal{X} \to \mathcal{Y}$ means that f maps elements in $\mathcal{X}$ to elements in $\mathcal{Y}$ (i.e. f is a function). Boldface notation indicate vectors; for example, $\boldsymbol{f}(x)$ is an n-dimensional vector valued function $[f_1(x), \ldots, f_n(x)]$. A is an operator – a rule that applies to functions; for example, $Az\,(x, t) = z\,(x, t) + \partial_{xx} z\,(x, t)$. Δ is a (very) small quantity. $\partial\mathcal{X}$ is the boundary of $\mathcal{X}$. $\mathcal{X} \cup \partial\chi$ is the union of the open set $\mathcal{X}$ with its boundary $\partial\mathcal{X}$. This closed set is denoted by $\overline{\mathcal{X}}$. The notation

$$\frac{\partial}{\partial v} G\,(v, \boldsymbol{x}, \boldsymbol{z})\,|_{v = x_i}$$

says 'take the derivative of G with respect to v and then replace v with x_i'. Boundary conditions sometimes require the so-called unit outward normal. This means that one needs a directional derivative that is perpendicular to the tangent to the boundary at a particular point. To fully specify a system of partial differential equations, we need both initial conditions ($t = 0$) *and* boundary conditions. Both the initial and boundary conditions are called data. Often we need boundary conditions with no flows across the boundary and non-uniform initial surface. We achieve this with the *sin* or *cos* functions and with appropriate size of the solution space.

2.1 Introduction

A large class of population models in ecology and epidemiology boils down to

$$\boldsymbol{z}'(t) = \boldsymbol{f}(\boldsymbol{z}(t)), \quad \boldsymbol{z}(0) = \boldsymbol{f}_0\,, \tag{1}$$

where $'$ denotes derivatives with respect to time, z is a vector of species population densities, $\boldsymbol{f}$ is a vector of real valued smooth functions, t is time, and $\boldsymbol{f}_0$ is given. All vectors are of dimension n. The implicit assumptions in such models are: (i) reproduction is by cloning; (ii) $\boldsymbol{z}$ is large; (iii) $\boldsymbol{z}$ represents some moment of the population dynamics; (iv) stochastic effects can be faithfully represented by $\boldsymbol{z}$; and (v) $\boldsymbol{z}$ are smooth functions of t.

To cast (1) in the context of evolution by natural selection, one needs to encapsulate the following principles: (i) phenotypic traits are inherited with some mutations; (ii) different values of these traits result in differences in the frequency of these values in future populations; and (iii) natural selection acts directly on phenotypic traits, not on genotypic traits.

To proceed from (1), we identify a set of adaptive traits and write (1) as

$$\boldsymbol{z}'(\boldsymbol{x}_i, \boldsymbol{x}, t) = \boldsymbol{f}(\boldsymbol{z}(\boldsymbol{x}_i, \boldsymbol{x}, t)), \quad \boldsymbol{z}(0) = \boldsymbol{f}_0, \tag{2}$$

where $\boldsymbol{z}$ is the vector of population (species) densities, and $\boldsymbol{x}_i$ is the set of adaptive heritable traits of species i. The dimension of $\boldsymbol{x}_i$ is m_i and $\boldsymbol{x}$ is the set of all adaptive traits of all species with dimension $m := \sum_{i=1}^{n} m_i$. The dynamics of $\boldsymbol{x}$, called the strategy dynamics, can be constructed by different methods, which involve additional assumptions (see Murray, 1989; Weibull, 1995; Fudenburg and Levine, 1998; Hofbauer and Sigmund, 1998, 2003; Samuelson, 1998; Gintis, 2000; Cressman, 2003; Vincent and Brown, 2005). Strategy dynamics usually take on the form

$$\boldsymbol{x}_i'(t) = \boldsymbol{g}_i(\boldsymbol{v}, \boldsymbol{x}, t), \quad \boldsymbol{x}(0) = \boldsymbol{x}_0, \quad i = 1, \ldots, n, \tag{3}$$

where $\boldsymbol{g}_i$ is a vector valued function of dimension m_i and $\boldsymbol{v}$ is a vector (of dimension m_i) of the so-called virtual strategies. Here v is the strategy of some focal individual and $\boldsymbol{g}_i$ is a gradient of fitness of the focal individual. Both $\boldsymbol{f}$ and $\boldsymbol{g}$ are assumed to be at least twice differentiable.

The basic achievement in deriving $\boldsymbol{g}_i$ is that it gives invasion-ability criteria, through a maximum principle (Vincent *et al.*, 1996). Specifically, from $\boldsymbol{g}_i$ we derive values of $\boldsymbol{x}$, call them $\tilde{\boldsymbol{x}}$, that give negative gradient on $\boldsymbol{g}_i$ for all i and for all $\boldsymbol{x} \neq \tilde{\boldsymbol{x}}$ (with respect to $\boldsymbol{z}$). It should be noted that in the case of Darwinian dynamics (Vincent and Brown, 2005), (3) becomes

$$\boldsymbol{x}_i'(t) = \boldsymbol{g}_i(\boldsymbol{v}, \boldsymbol{x}, \boldsymbol{z}, t), \quad \boldsymbol{x}(0) = \boldsymbol{x}_0, \quad i = 1, \ldots, n \tag{4}$$

and the strategy dynamics is no longer divorced from the population dynamics. Because of the effect of $\tilde{\boldsymbol{x}}$ on the dynamics of all other values of $\boldsymbol{x}$, we call $\tilde{\boldsymbol{x}}$ the evolutionarily stable strategies (ESS) of the system (3) or the system (2) and (4). So far, the strategies are assumed to be unbounded. Bounded strategy spaces introduce complications that can be avoided if one assumes that $\tilde{\boldsymbol{x}}$ is in the interior of the strategy space.

Behind each $\boldsymbol{x}$ hide distributions of the density of phenotypes of $\boldsymbol{z}$. In fact, the dynamics of $\boldsymbol{x}$ represent the dynamics of moments of these distributions (usually the mean). This raises the possibility that we may be following dynamics of phenotypes that do not exist (Fig. 1). The stability of $\tilde{\boldsymbol{x}}$ cannot be determined by traditional stability analysis – hence the

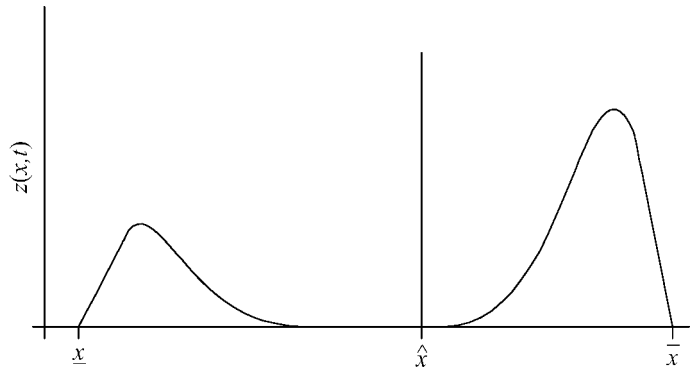

Fig. 1. A bounded distribution (between $\underline{x}$ and $\bar{x}$) of phenotypes at time t. Note that there are no phenotypes at the mean adaptive trait, $\hat{x}$. The figure illustrates a snapshot of some evolutionary distribution at t.

proliferation of stability criteria for ESS (Eshel, 1996; Apaloo, 1997; Taylor and Day, 1997). Stable evolutionary distributions (ED[1]) represent a set of ESS because all possible mutants are included in the ED.

The roots of the theory developed here can be found in the following: Kimura (1965), who was interested in population genetics and characterizing moments of the distribution of genotypes. Levin and Segel (1985) referred to the adaptive space as aspect (see also Keshet and Segel, 1984). Slatkin (1981) used a diffusion model to discuss species selection. Ludwig and Levin (1991) examined the dispersal of phenotypes in plant communities. A different approach to characterizing evolutionary related distributions is to use moments. In fact, some distributions can be characterized by a finite (and small) number of moments (see, for example, Barton and Keightley, 2002).

2.2 Smooth evolutionary games

Continuous games are different from smooth games in the following sense: Continuous means that in some places there may be 'kinks' where, at a point, the limits of the derivative from the right and from the left are different. No such points are allowed in smooth games.

There are different ways to study evolutionary games. Two distinct approaches are through matrix games and through games that involve smooth functions (Hofbauer and Sigmund, 1998, 2003). The latter is implemented with ordinary differential equations (ODE) and the approach to formulating such games was discussed in Section 2. To illustrate the connection between smooth evolutionary games and ED, we shall follow the approach taken by Vincent and Brown (2005) and apply it to co-evolution under competition (for further details, see Cohen *et al.*, 2000b, 2001).

The Lotka-Volterra competition model with a single adaptive trait x (see Vincent *et al.*, 1993) is

$$z_i' = z_i G(x_i, \boldsymbol{x}, \boldsymbol{z}), \quad i = 1, \ldots, n.$$

[1] ED is singular or plural.

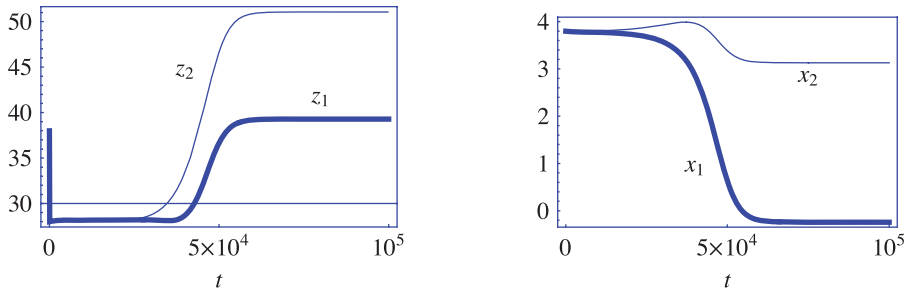

Fig. 2. Two-species ESS: population dynamics (left) and strategy dynamics (right).

Here $\boldsymbol{x} = [x_1, \ldots, x_n]$ and $G(x_i, \boldsymbol{x}, \boldsymbol{z})$ is the instantaneous individual fitness function for species i. The explicit form of G is

$$G(\cdot) = r - \frac{r}{k(x_i)} \sum_{i=1}^{n} \alpha(x_i, \boldsymbol{x})\, z_i,$$

where $k(\cdot)$ and $\alpha(\cdot)$ are the carrying capacity and competition function respectively. The ability of i to adjust to its carrying capacity is a function of the value of the adaptive trait. The ability of i to compete is a function of the value of its adaptive trait and the values for all other species. The explicit forms of k and α for i are

$$k(x_i) = k_m \exp\left[-\frac{1}{2}\left(\frac{x_i}{\sigma_k}\right)^2\right], \tag{5}$$

$$\alpha(x_i, x_j) = 1 + \exp\left[-\frac{1}{2}\left(\frac{x_i - x_j + b}{\sigma_\alpha}\right)^2\right] - \exp\left[-\frac{1}{2}\left(\frac{b}{\sigma_\alpha}\right)^2\right].$$

Thus, we obtain the strategy dynamics

$$x_i' = \sigma \frac{\partial}{\partial v} G(v, \boldsymbol{x}, \boldsymbol{z})|_{v = x_i}$$

(see Vincent *et al.*, 1993). We use the parameter values

$$k_m = 100, \quad \sigma_k = \sqrt{12.5}, \quad b = 2, \quad \sigma_\alpha = 2, \tag{6}$$
$$\sigma = \sqrt{0.04}, \quad r = 0.25$$

with $n = 2$. These values lead to ESS (Fig. 2).

2.3 Evolutionary distributions

We shall translate the smooth games approach into a single ED with a single adaptive trait. Later, we state the results more generally. We do not identify z with a species density, but with the distribution of phenotype densities (see Fig. 1). On the distribution, one might identify species (see discussion in Cohen, 2003a, 2005). So we write

$$z' = f(z, x, t),$$

where $z \equiv z\,(x, t)$. Next, we decompose f into the two components: one that results in growth in the density of phenotypes and the other in a decline:

$$f\,(z, x, t) = \tilde{\beta}\,(z, x, t) - \mu(z, x, t).$$

As Darwin (1859) surmised, when resources are plenty, populations grow exponentially. In our vernacular, $\tilde{\beta}$ is a linear function of z. As resources are exhausted, $\beta(\cdot) - \mu(\cdot)$ decreases. We usually assume that mutations occur at birth (i.e. on $\tilde{\beta}$) and selection on processes that lead to decline in the density of phenotypes; namely μ. Thus we write

$$\tilde{\beta}\,(z, x, t) = \beta z(x, t), \tag{7}$$

where β is a rate coefficient. Now assume a mutation rate coefficient η. Then

$$\partial_t z(x, t) = (1 - \eta)\,\beta z(x, t) + \frac{1}{2}\,\beta\eta[z(x + \Delta) + z(x - \Delta)] - \mu(z, x, t).$$

Using Taylor series expansion of z around x, we obtain approximately

$$\partial_t z = \beta z + \frac{1}{2}\,\Delta^2\beta\eta\partial_{xx} z - \mu(z, x, t).$$

Now for a single ED with m independent adaptive traits, we obtain

$$\partial_t z = \beta z + \frac{1}{2}\,\Delta^2\beta \sum_{i=1}^{m} \eta_i \partial_{x_i x_i} z - \mu(z, x, t). \tag{8}$$

For convenience, we define the linear operator ${}_m A$ thus:

$${}_m A = 1 + \varepsilon \sum_{i=1}^{m} \eta_i \partial_{x_i x_i}, \tag{9}$$

where $\varepsilon := \Delta^2\beta/2$. To emphasize its role, we call ${}_m A$ the *m-order mutation operator*.

For the case of n ED, each with m_i independent adaptive traits, we have

$$\partial_t z_i(\boldsymbol{x}_i, t) = \beta_i m_i A z_i(\boldsymbol{x}_i, t) - \mu_i(\boldsymbol{z}, \boldsymbol{x}, t), \quad i = 1, \ldots, n. \tag{10}$$

In (10) we assume that all traits are independent and that mutations are random. The selective effects on trait values are embodied in $\mu(\cdot)$. Note that we shall usually assume that the mutation operator depends on the set of adaptive traits within a specific ED, z_i. Thus we write ${}_{m_i} A z(\boldsymbol{x}_i, t)$. Declines in densities of phenotypes on an ED depend on the adaptive traits of all ED. Therefore, we write $\mu_i(\boldsymbol{z}, \boldsymbol{x}, t)$.

The problems we deal with require specific boundary conditions. In general, there is no *a priori* reason to set the boundary conditions to a particular value, so we shall use the Neumann boundary conditions – any dissipation across the boundaries disappears from the system. Because we deal with rectangular boundaries and orthogonal traits, our boundary conditions remain simple. We shall always set the initial conditions such that they are compatible with the boundary conditions. Also, physical constraints dictate that $\boldsymbol{x}$ is bounded.

To formally define ED, we need the following. Let $z_i \in \mathbb{R}_{0+}$, $i = 1, \ldots, n$ be the distribution of the density of phenotypes with m_i adaptive traits $\boldsymbol{x}_i$ and let $\boldsymbol{x} := [\boldsymbol{x}_1, \ldots, \boldsymbol{x}_n]$ (the dimension of each $\boldsymbol{x}_i$ is m_i). Define the bounded open set $\mathcal{X} \subset \mathbb{R}^M$ (where $M = \Sigma_{i=1}^{n} m_i$) with boundary $\partial\mathcal{X}$. Then,

Definition. *A semi-linear ED*, $z_i(\boldsymbol{x}, t)$, *is the solution of the system*

$$\partial_t z_i(\boldsymbol{x}, t) = \beta(\boldsymbol{x})_{m_i} A z_i(\boldsymbol{x}_i, t) - \mu_i(\boldsymbol{z}, \boldsymbol{x}, t)$$

(*where A is the mutation operator*) *with the data*

$$z_i(\boldsymbol{x}, 0) = z_{i0}(\boldsymbol{x})$$

(*where* $z_{i0}(\boldsymbol{x})$ *given*) *and*

$$\partial_n z_i(\boldsymbol{x}, t)\,|_{x=\partial\mathcal{X}} = 0, \quad i = 1, \ldots, n$$

where ∂_n *denotes the directional* (*outward normal*) *derivative on* $\partial\mathcal{X}$.

The definition refers to a semi-linear ED because μ_i is usually non-linear. In certain cases (e.g. predator–prey), we have $\beta(\boldsymbol{z}, \boldsymbol{x}, t)$, which makes the ED quasi-linear (for definitions, see Evans, 1998). It is important to realize that the mutation operator is a function of $\boldsymbol{x}_i$ only. This imparts a unique structure to the system of ED.

The definition implies that: (i) mutations occur on the processes that lead to growth in z_i; (ii) growth rate is a linear function of z_i; (iii) selection occurs on the processes that lead to decline in z_i; (iv) selection depends on all ED with which z_i interacts; and (v) adaptive traits are bounded with dissipation across boundaries. These are simplifying assumptions. They can be relaxed at the expense of more elaborate models and there is nothing in the ED approach that limits us to these simplifications.

The set $\overline{\mathcal{X}} = \mathcal{X} \cup \partial\mathcal{X}$ defines the so-called *adaptive space* and $\overline{\mathcal{X}} \times \mathbb{R}^n_{0+}$ defines the *evolutionary space*. The restriction to linear growth can be relaxed. In such cases, the Taylor series approximation becomes more involved.

Because we model physical systems, we shall not be concerned with the existence and uniqueness of solutions – if solutions do not exist, then there is something fundamentally wrong with the model. We do not require uniqueness because there is nothing in the theory of evolution that requires unique solutions. Next, we translate the continuous game in Section 2.2 to ED. In general, solutions of ED will depend on initial conditions and on the size of the adaptive space.

3. APPLICATIONS

Here we apply ED to ecological interactions and an ecosystem model.

3.1 Competition

First, we examine mutations with selection. Next, we apply ED with a single adaptive trait. Finally, we examine ED with two orthogonal adaptive traits. This section is based primarily on Cohen (2003a) with some extensions. It is presented here for the sake of completeness.

3.1.1 Single adaptive trait without selection

Returning to the model in Section 2.2, we write

$$\partial_t z = rAz - \frac{r}{k_m} z^2. \tag{11}$$

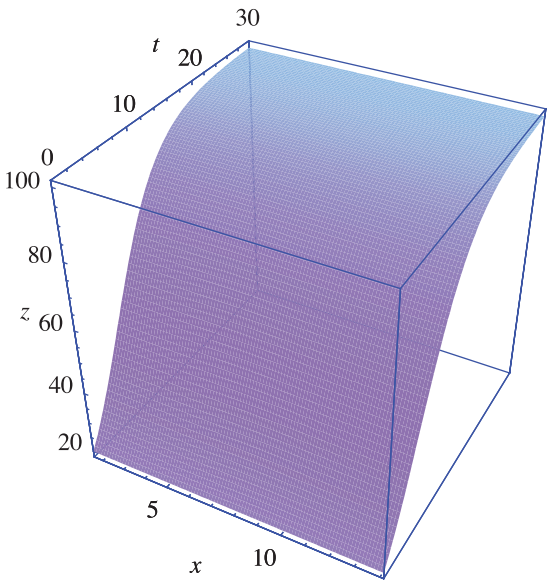

Fig. 3. Single trait ED with no evolution.

This is the well-known Fisher equation (Fisher, 1930; Smoller, 1982). As in (6), we use

$$r = 0.25, \quad k_m = 100, \quad \Delta = 0.01, \quad \eta = 0.01$$

with the data

$$z(x, 0) = 20,$$
$$\partial_x z(\pi/2, t) = \partial_x z(9\pi/2, t) = 0.$$

This results in a homogeneous stable ED, $\bar{z}(x) = 100$ (Fig. 3).

3.1.2 Single adaptive trait with selection

We assume that phenotypes with $x = 5\pi/2$ are most adapted to the environment and write

$$k(x) = k_m \left(1 + \exp\left[-\frac{1}{2}\left(\frac{x - 5\pi/2}{\sigma_k}\right)^2\right]\right). \tag{12}$$

For competition, we write

$$\alpha(x, \xi) = k_\alpha \left(1 + k \exp\left[-\frac{1}{2}\left(\frac{x - \xi}{\sigma_\alpha}\right)^2\right]\right). \tag{13}$$

The parameters k_α and k_m scale the influence of competition and carrying capacity on the ED. The parameters σ_α and σ_k reflect the plasticity of phenotypes with respect to the adaptive traits that influence competition and carrying capacity, respectively. Thus, (11) becomes

$$\partial_t z = rAz - \frac{r}{k(x)} z(x, t) \int_{\pi/2}^{9\pi/2} \alpha(x, \xi) z(\xi, t) \mathrm{d}\xi$$

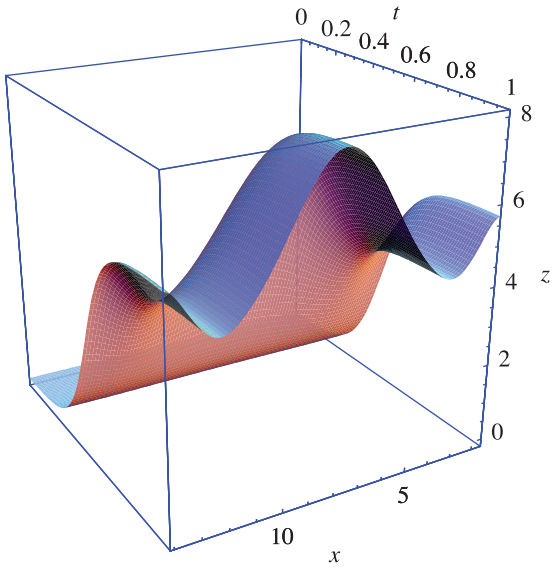

Fig. 4. ED with a single adaptive trait and with competition.

with

$$z(x, 0) = 0.005,$$
$$\partial_x z(\pi/2, t) = \partial_x z(9\pi/2, t) = 0.$$

With the parameter values

$$r = 0.25, \quad \sigma_k = 100, \quad k_a = 1, \quad \sigma_a = 1,$$
$$k_m = 10^4, \quad \Delta = 0.01, \quad \eta = 0.01,$$

the effect of adding selection through both carrying capacity and competition is to depress the phenotype densities in the ED (Fig. 4). Mutations and selection end up with a concentration of phenotypes around the most adaptive value of the carrying capacity and at the boundaries of the adaptive space because phenotypes on the boundary experience half the competition that phenotypes in the interior do.

The theory predicts that where competition dominates, we should find the most fit phenotypes on the boundaries of the adaptive space. For example, in places where competition among plants for light is strong (e.g. no nutrient limitations and uniform carrying capacity with respect to the values of the adaptive trait), we should find – at evolutionary stability of ED – a higher density of short and tall plants than intermediate plants, a common phenomenon in forested plant communities.

3.1.3 Two adaptive traits with selection

Assume two orthogonal traits, $\boldsymbol{x} := [x_1, x_2]$. x_1 is selected for carrying capacity and x_2 for competitive ability. So we write

$$\partial_t z = r_2 A z - \frac{r}{k(x_1)} z \int_{\pi/2}^{9\pi/2} \alpha(x_2, \xi) z(x_1, \xi, t) \mathrm{d}\xi$$

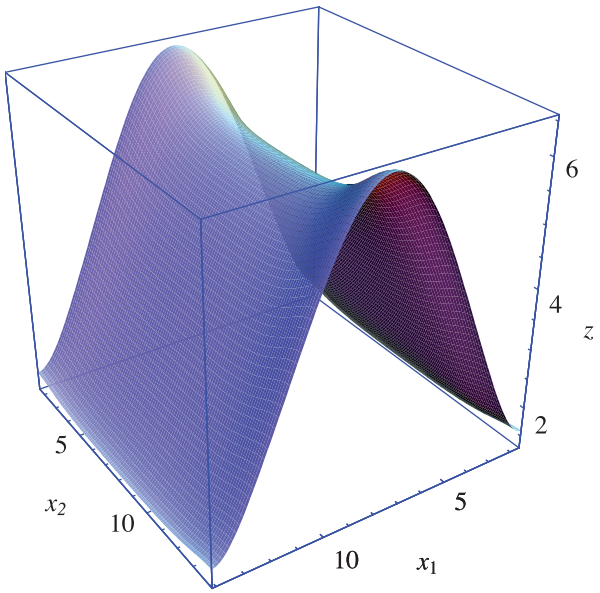

Fig. 5. Stable ED with two adaptive traits.

with data

$$z(\boldsymbol{x}, 0) = 20,$$

$$\partial_{x_1} z(\pi/2, x_2, t) = \partial_{x_1} z(9\pi/2, x_2, t) = 0,$$

$$\partial_{x_2} z(x_1, \pi/2, t) = \partial_{x_2} z(x_1, 9\pi/2, t) = 0,$$

where $k(x_1)$ and $\alpha(x_2, \xi)$ are defined in (12) and (13) and $z \equiv z(x_1, x_2, t)$. With the same parameter values as before, we obtain a stable solution (Fig. 5). With two traits, the effect of competition on the ED becomes apparent – at both ends of the adaptive trait that reflects competitive ability (x_2), we have a higher density of phenotypes on the stable ED. This is because at the boundaries, phenotypes are faced with half as much competition as phenotypes in the interior of the adaptive space.

Apparently, the highest fitness (most abundant phenotypes at stable ED) is with respect to the carrying capacity adaptive trait (x_1). Among these phenotypes, the highest fitness is that of phenotypes on the boundaries of the adaptive trait that reflect competitive ability (x_2). In the evolutionary games context, at ESS, the *G*-function (Vincent and Brown, 2005) would have two distinct values at its maximum = 0. One can trivially modify the parameters to produce separation of maxima in the adaptive space and thus a sort of sympatric 'speciation'.

3.2 Predator–prey

Here we use the logistic model for the prey and add to it mortality due to predation with saturation (the Rosenzweig-MacArthur model). A different predator–prey model in the context of ED was analysed in Cohen (2003b). Thus we write:

$$z_1' = rz_1 - \frac{r}{k} z_1^2 - \frac{az_1}{b + cz_1} z_2, \tag{14}$$

$$z_2' = d \frac{az_1}{b + cz_1} z_2 - \mu z_2^2.$$

With

$$r = 0.25, \quad k = 100, \quad a = 1, \quad b = 10, \tag{15}$$

$$c = 1, \quad d = 0.1, \quad \mu = 0.01$$

we obtain a limit cycle (Fig. 6). With the tendency to produce evolutionary cycles, models such as (14) were criticized by Abrams (2000) as not especially general. Interestingly, as we shall see in a moment, these cycles, ubiquitous as they are in point process models, disappear in our case for a large space of parameters values.

Let $\boldsymbol{x} := [x_1, x_2]$. Assume that the prey phenotypes density, z_1, evolves on the adaptive trait x_1 and the predator phenotypes density, z_2, evolves on the adaptive trait x_2. For example, x_1 may represent the running speed of prey phenotypes and x_2 the running speed of predator phenotypes. We also assume that predation is at its maximum when $x_1 = x_2$ with some phenotype plasticity (variance) with respect to the adaptive traits, σ. We model the interaction between the prey adaptive trait x_1 and the predator adaptive trait x_2 with

$$\alpha(\boldsymbol{x}) = \exp\left[-\frac{1}{2}\left(\frac{x_1 - x_2}{\sigma}\right)^2\right].$$

Let the mutation operators of the prey and predator be

$$Az_1 := z_1 + \frac{1}{2}\Delta^2 \eta_1 \partial_{x_1 x_1} z_1$$

and

$$Az_2 := z_2 + \frac{1}{2}\Delta^2 \eta_2 \partial_{x_2 x_2} z_2.$$

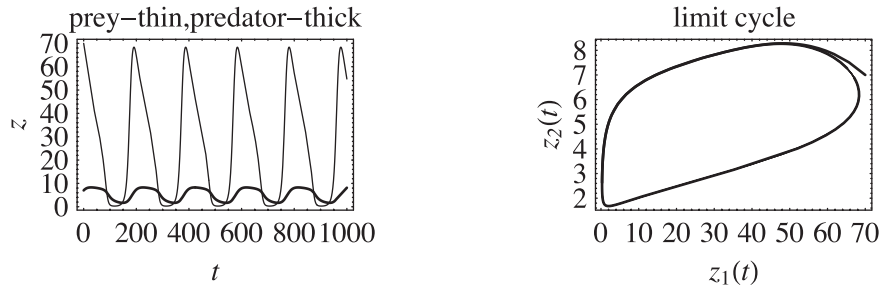

Fig. 6. Predator–prey limit cycle.

We now have two ED (for prey, z_1 and predator, z_2) with two adaptive traits

$$\partial_t z_1 = rAz_1 - \frac{r}{k} z_1^2 - \alpha(\boldsymbol{x}) \frac{az_1}{b + cz_1} z_2,$$

$$\partial_t z_2 = d\alpha(\boldsymbol{x}) \frac{az_1}{b + cz_1} Az_2 - \mu z_2^2.$$

We choose initial conditions

$$z_1(\boldsymbol{x}, 0) = 10,$$

$$z_2(\boldsymbol{x}, 0) = 1$$

and boundary conditions

$$\partial_{x_1} z_1(\pi/2, x_2, t) = \partial_{x_1} z_1(9\pi/2, x_2, t) = 0,$$

$$\partial_{x_2} z_2(x_1, \pi/2, t) = \partial_{x_2} z_2(x_1, 9\pi/2, t) = 0.$$

In addition to the parameters in (15) we use

$$\Delta = 0.01, \quad \eta_1 = \eta_2 = 0.01, \quad \sigma = \pi/3.$$

The stable ED are shown in Fig. 7. The densities of prey phenotypes along the positive diagonal (with slight shifts in opposite directions for prey and predator) of the evolutionary space, $x_1 = x_2$, are depressed. The densities of predator phenotypes are nearly a mirror image. The shapes of the stable ED depend on the interplay between the size of the adaptive space and phenotypic plasticity (Fig. 8). For fixed plasticity, an increase in the size of the adaptive space means a decrease in the effective phenotypic plasticity. Both Figs. 7 and 8 indicate that in the evolutionary space, low fitness of prey phenotypes is associated with high fitness of predator phenotypes. The density of low fitness prey phenotypes is maintained (fed) by prey with high fitness phenotypes. To end with stable ED, it is necessary to have low fitness phenotypes to 'feed' the prey and this is done by the high fitness prey

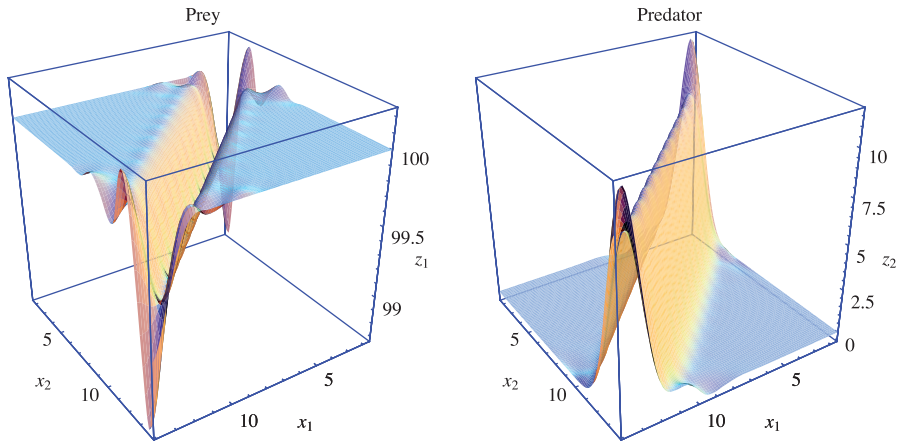

Fig. 7. Stable ED with phenotype plasticity with respect to the adaptive traits $\sigma = \pi/3$.

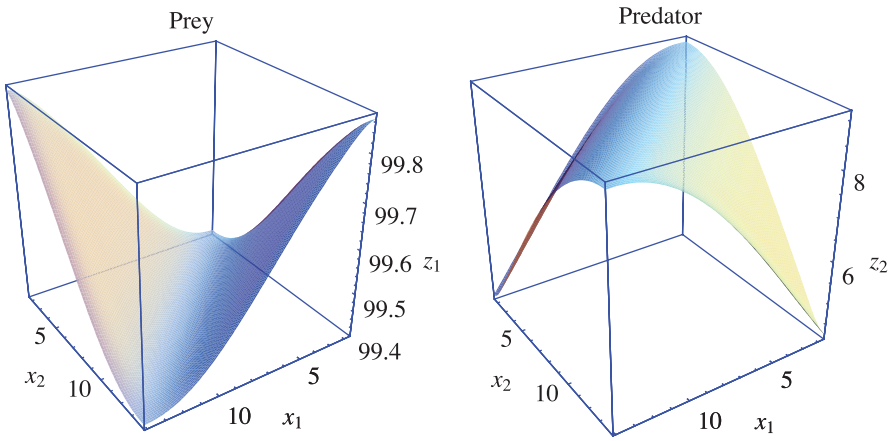

Fig. 8. Stable ED with phenotype plasticity with respect to the adaptive traits $\sigma = \pi$.

phenotypes. This is a good example where a spectrum of fitnesses co-exist and in fact is necessary to maintain evolutionary stability. One might object to showing the adaptive space of prey, for example, with the adaptive trait of the predator (e.g. Fig. 7). Yet the solutions of both ED are linked to both adaptive traits through $\boldsymbol{f}$.

If x_1 and x_2 represent the prey and predator running speed, for example, then we interpret Fig. 7 as follows. Fast prey (large x_1) that co-evolve with fast predators (large x_2) show low fitness at evolutionary stability. In these circumstances, predator phenotypes show high fitness. At low values of x_1 and x_2, we have the same co-evolutionary outcome. This pattern is maintained by the continuous evolution of prey phenotypes of high fitness (off the diagonal) into the diagonal region of the adaptive space. Off-diagonal prey phenotypes co-evolve to be either fast runners associated with slow predator phenotypes or slow runners associated with fast predator phenotypes. So if you are a slow prey (e.g. hiding), you are fit against fast (chasing) predators. If you are fast (e.g. running), you are fit against slow (ambushing) predators. These outcomes are conditioned on the existence of low fitness prey phenotypes (along the diagonal). The picture for predators is a mirror image of the picture for the prey. Overall, we find high diversity of prey phenotypes occupying the adaptive space and low diversity of predator phenotypes – a common feature in nature.

The predator–prey model we use did not result in evolutionary cycles. This is not to say that they are not possible. The fact that point process evolutionary models are cyclic does not mean that the cycles will remain when the models are translated to ED. Because the boundaries are not smooth, the ED warps in the corners (Fig. 7). We use orthogonal adaptive traits. Therefore, we are not concerned with smooth boundaries.

3.3 Host–pathogen

The evolution of host–pathogen and infectious diseases is of much interest (Dieckmann *et al.*, 2002). For example, witness recent news about resistant pathogens and pathogen mutations that may cause pandemics (such as the bird flu). Following Anderson and May (1980, 1981), we define the densities of host population z_1, infected hosts z_2, and pathogens z_3. The parameters are the coefficients of individual host birth rate, a; natural death rate, b; additional death rate due to infection, $\tilde{\alpha}$; transmission efficiency, ν; recovery rate, γ; death

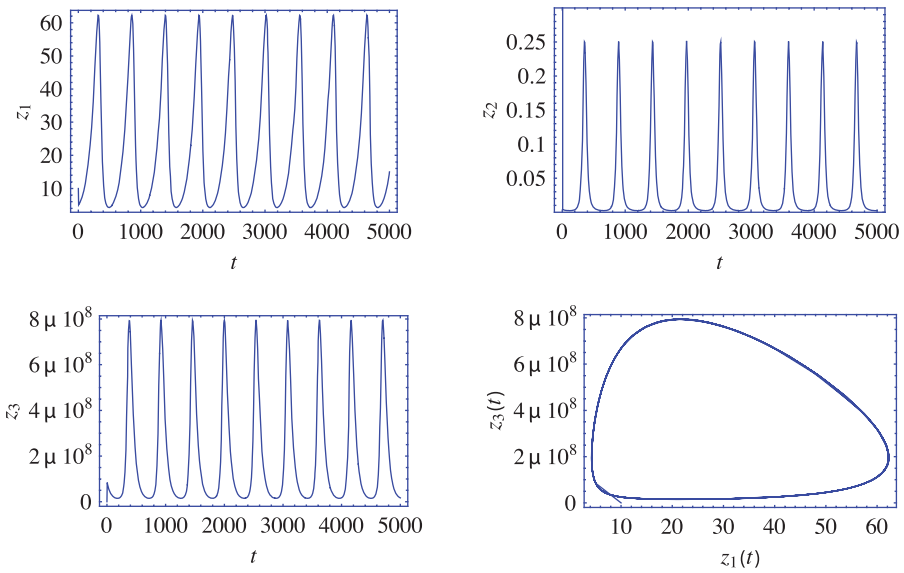

Fig. 9. Host–pathogen limit cycle (after Anderson and May, 1980). z_1, z_2, and z_3 denote the host, infected and pathogen population, respectively.

rate of infective stages, $\tilde{\mu}$; and the rate at which an infected host produces infective stages of the parasite λ. The model is from Anderson and May (1980, equations 3, 5, and 6):

$$z_1' = (a - b)\, z_1 - \alpha z_2,$$
$$z_2' = \nu z_3 (z_1 - z_2) - (\alpha + b + \gamma)\, z_2,$$
$$z_3' = \lambda z_2 - (\mu + \nu z_1)\, z_3.$$

The parameter values

$$a = 5.3, \quad b = 5.29, \quad \tilde{\gamma} = 10^{-20}, \quad \alpha = 5.5,$$
$$\tilde{\lambda} = 10^{8}, \quad \mu = 0.02, \quad \nu = 10^{-10}$$

produce a limit cycle (Fig. 9).

Let x_1 be the adaptive trait that affects the host death rate coefficient due to infection. The adaptive trait of the pathogen, x_2, affects the pathogen death rate of infective stages. Assume that at some value of x_1, say $5\pi/2$, the death rate of hosts due to infection is at its minimum. Thus we write

$$\alpha(x_1) = \tilde{\alpha}\left(1 - 0.1 \exp\left[-\frac{1}{2}\left(\frac{x_1 - 5\pi/2}{\sigma_\alpha}\right)^2\right]\right). \tag{16}$$

We also assume that at some value of x_2 (= $5\pi/2$) the death rate of pathogen infective stages is at its maximum:

$$\mu(x_2) = \tilde{\mu}\left(1 + 0.1 \exp\left[-\frac{1}{2}\left(\frac{x_2 - 5\pi/2}{\sigma_\mu}\right)^2\right]\right). \tag{17}$$

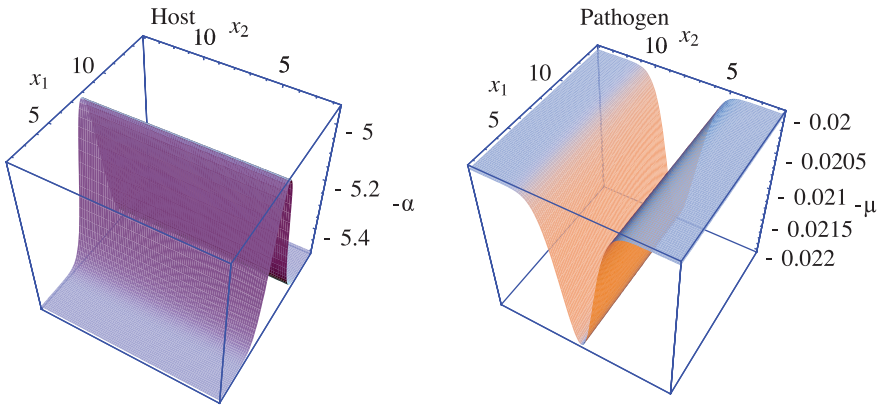

Fig. 10. Anticipated effect of α and μ on the host and pathogen ED in the adaptive space (x_1, x_2).

The anticipated effects of α and μ on the host and pathogen ED are shown in Fig. 10.

Now the ED become

$$\partial_t z_1 = aAz_1 - bz_1 - \alpha(x_1)z_2,$$
$$\partial_t z_2 = vAz_3(z_1 - z_2) - (\alpha(x)_1 + b + \gamma)z_2,$$
$$\partial_t z_3 = \lambda z_2 - (\mu(x_2) + vz_1)z_3,$$

with data

$$z_1(\boldsymbol{x}, 0) = 20,$$
$$z_2(\boldsymbol{x}, 0) = 0,$$
$$z_3(\boldsymbol{x}, 0) = 2 \times 10^9,$$
$$z_i(\pi/2, x_2, t) = z_i(9\pi/2, x_2, t),$$
$$z_i(x_1, \pi/2, t) = z_i(x_2, 9\pi/2, t),$$
$$i = 1, 2, 3.$$

The parameters are set to

$$a = 5.3, \quad b = 5.29, \quad \gamma = 10^{-20},$$
$$k_\alpha = 5.5, \quad \lambda = 10^8, \quad k_\mu = 0.02,$$
$$v = 10^{-10}, \quad \Delta = 0.01, \quad \eta_1 = \eta_2 = \eta_3 = 0.01,$$
$$\sigma_a = \sigma_\mu = \pi/3$$

(note the periodic boundary conditions; they were set to produce a desired effect). Figure 11 (and the animation of the dynamics at https://wiki.umn.edu/view/Main/YosefCohen) show the limit-cycle ED at a particular t. Because of co-evolution, the anticipated ED of the host and pathogen ED (Fig. 10) are different from the evolutionary ED.

Several interesting features emerge from the co-evolution of the host–pathogen system. Foremost is the fact that we obtain a limit cycle. This cycle can be observed only via

animations (at https://wiki.umn.edu/view/Main/YosefCohen). High densities of phenotypes are pushed to the corners of the adaptive space. These are separated by zero densities. In the model, reproduction is localized. So if one chooses to identify species with phenotypes that co-produce, then the process results in the emergence of four common host 'species' and rare species in between. Phenotypes at the inner boundaries of the high densities fare well with the pathogen (their densities are the highest). This is the case because we find low densities of pathogen phenotypes corresponding to these boundaries (z_3 in Fig. 11). The infected phenotypes (z_2 in Fig. 11) follow the ED of the host (z_1) with more pronounced densities of phenotypes along the inner edges – being at the extreme inner edges of the adaptive space imparts extra protection to the infected phenotypes. At other times of the cycle (not shown), the corners infected phenotypes and pathogen disappear and a single peak of both appears in the middle.

These results indicate that we may observe cycles in the co-evolution of host–pathogen. In these cycles, resistant hosts and virulent pathogens co-evolve from single peaks in the

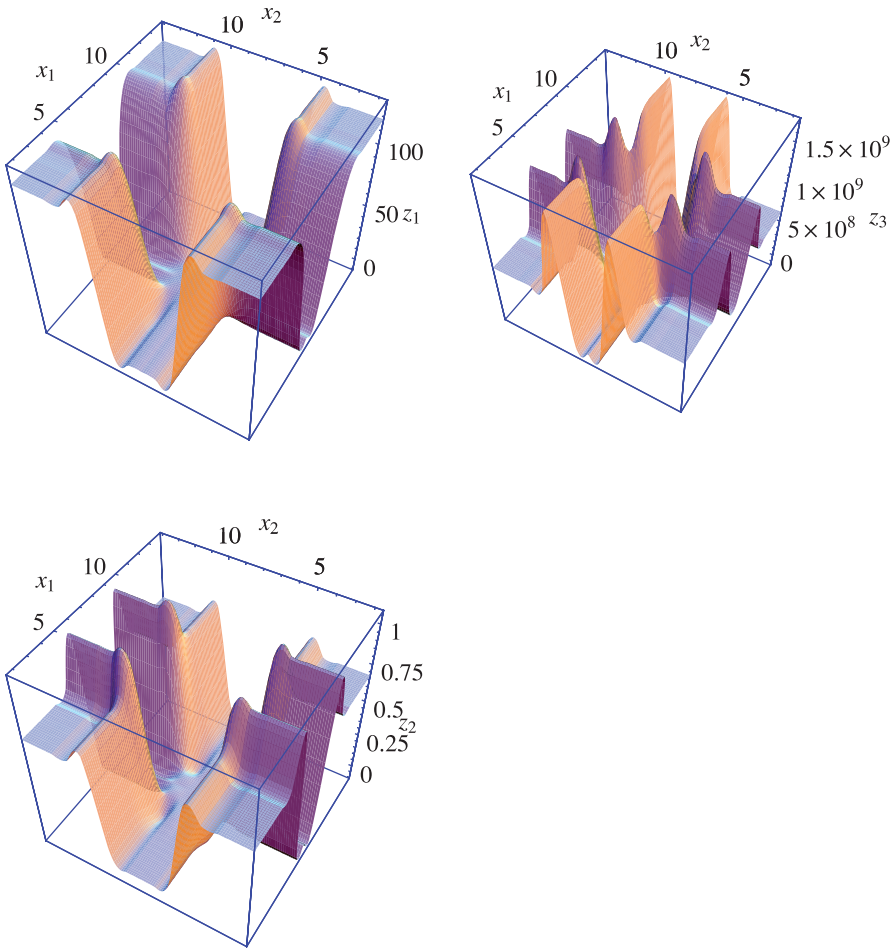

Fig. 11. Limit-cycle ED for host and pathogen in the evolutionary space (compare with Fig. 10), at $t = 2\times10^5$.

middle of the adaptive space to peaks in the corner of the adaptive space. The approach we take to the co-evolution of host–pathogen is different from traditional views (e.g. Levin, 1996). First, the adaptive traits of the host and the pathogen are independent and certain parameter values are not fixed; they are functions (given in 16 and 17) whose values are determined by co-evolution. The dependency between the values of these functions surfaces through mutation and selection. Second, ours is an infinite dimensional system. Therefore, we do not need to introduce a fixed mutant with a certain parameter value and examine the outcome of co-evolution (as in, for example, Anderson and May, 1982). Finally, and perhaps most importantly, we do subscribe to the view that natural selection is a local phenomenon (e.g. Levin and Bull, 1994; Levin, 1996). However, fitness is a distributional phenomenon. Thus, we may observe a spectrum of fitnesses at evolutionary stability (of ED).

The results depend on the initial conditions. When we use

$$z_1(\boldsymbol{x}, 0) = 20 + 2\cos(x_1),$$
$$z_2(\boldsymbol{x}, 0) = 0,$$
$$z_3(\boldsymbol{x}, 0) = 2 \times 10^9 + 2 \times 10^8 \cos(x_2),$$

we obtain a pathogen and infected ED very different from those obtained from homogeneous initial surfaces (compare Fig. 11 with Fig. 12). This is a particularly convincing example of the problems that arise if one follows the dynamics of the mean of the strategies, as opposed to following the dynamics of the ED. Following the mean of the strategy dynamics leads one to believe that one is following dynamics of typical phenotypes when in fact no phenotypes with adaptive trait values that represent the mean density of phenotypes exist. This application of the theory also illustrates the much richer behaviour of solutions of PDE compared with ODE and thus brings us closer to the richness of phenomena we see in nature.

The results in this section indicate that ED may result in Turing instabilities and pattern formation (see, for example, Murray, 2003) in the evolutionary space. Such analysis is pursued elsewhere (Cohen, 2008).

3.4 ED in ecosystems

So far, we have discussed ED from the perspective of co-evolutionary interactions within and between ED. Here, we discuss ED in the context of ecosystems. Our fundamental tenet is that ecosystems are open. Thus, in addition to abiotic considerations, we must include input and output to the system. Extending ED to incorporate the abiotic environment opens the door to applications that include nutrient cycling in the context of co-evolution. This section builds upon smooth evolutionary games in ecosystems (Cohen *et al.*, 2000a). Note that the number of permutations of the dynamics of ecosystem input and output may lead to different results. For example, a constant (no oscillations) input will probably result in different numerical solutions of the ED than oscillating inputs (as is the case in may ecosystems). We outline a single example.

3.4.1 A simple ecosystem model

We shall use a simple system (Fig. 13) with nitrogen cycling through three compartments. All units in the model are expressed in N-equivalent units (e.g. density of N). We model the system with saturation and from Fig. 13 derive the model as:

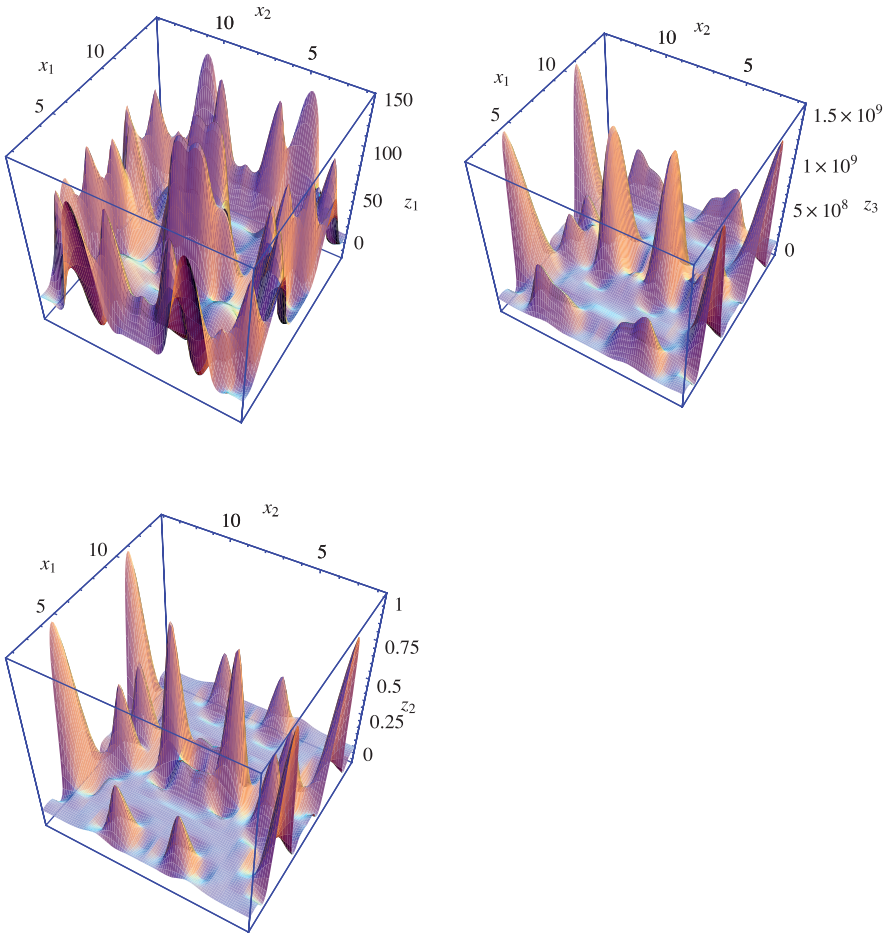

Fig. 12. Stable infected and pathogen ED.

$$
\begin{aligned}
z_1' &= v_3 v_1 \frac{z_3}{1 + v_2 z_3} z_1 - \chi_1 \frac{z_1}{1 + \chi_2 z_1} z_2 - (\lambda_1 + \mu_1) z_1^2, \\
z_2' &= \chi_3 \chi_1 \frac{z_1}{1 + \chi_2 z_1} z_2 - (\lambda_2 + \mu_2) z_2^2, \\
z_3' &= \iota(t) + \mu_1 z_1^2 + \mu_2 z_2^2 - v_1 \frac{z_3}{1 + v_2 z_3} z_1 - \lambda_3 z_3,
\end{aligned}
\tag{18}
$$

where the scaling coefficients v_3 and χ_3 are between zero and one. Assume that the nitrogen input rate, $\iota(t)$, oscillates with one cycle per year. So we write

$$
\iota(t) = A + B \sin\left(\frac{2\pi t}{12}\right). \tag{19}
$$

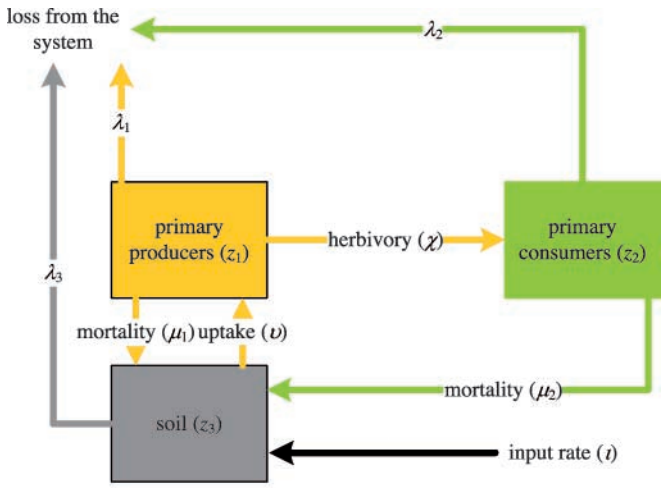

Fig. 13. A simple ecosystem model.

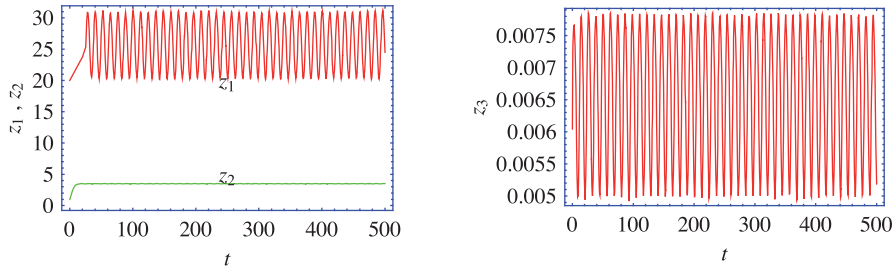

Fig. 14. Time trajectories of system (18) with parameters values in (20) and initial conditions (21). z_1, z_2, and z_3 are the density of nitrogen in primary producers, primary consumers, and soil, respectively.

With the parameter values

$$\begin{aligned} &A = 10, \quad B = 4, \quad \lambda_1 = 0.01, \quad \lambda_2 = 0.1, \quad \lambda_3 = 0.01, \\ &\mu_1 = 0.01, \quad \mu_2 = 0.01, \quad v_1 = 100, \quad v_2 = 1, \quad v_3 = 1, \\ &\chi_1 = 1, \quad \chi_2 = 1, \quad \chi_3 = 0.4 \end{aligned} \tag{20}$$

and initial conditions

$$z_1(0) = 20, \quad z_2(0) = 1, \quad z_3(0) = 0.006 \tag{21}$$

we obtain Fig. 14. The time trajectories indicate that the system may be either chaotic or resonant (Fig. 15). We shall not pursue this issue.

3.4.2 Co-evolution in ecosystems

From (18) and Fig. 13 we conclude that reproduction occurs on the uptake (v) and consumption (χ) terms. To simplify the system, we shall ignore competition within ED

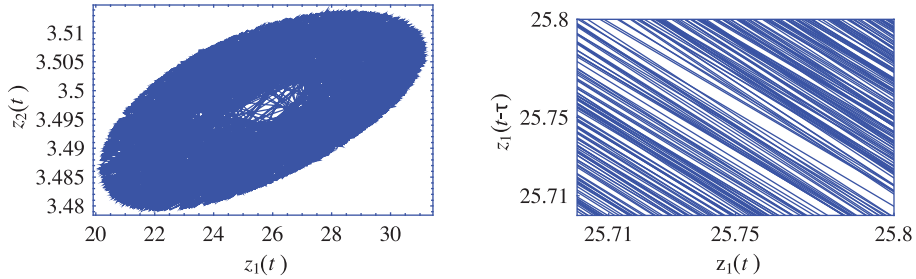

Fig. 15. Phase portraits of system (18) with parameters values in (20) and initial conditions (21). The delay $\tau = 6$. z_1 and z_2 denote nitrogen density in primary producers and primary consumers, respectively.

and assume that x_1 is the primary producers' adaptive trait that endows them with the ability to resist consumption (e.g. tannin and lignin concentrations in plant tissue). Similarly, x_2 is the adaptive trait that allows consumers to handle these compounds. Assume that an increase in x_1 causes a decrease in consumption – the χ related term in Fig. 13. The price to pay for an increased resistance to consumption is an increase in death rate – the μ_1 related term in Fig. 13. An increase in resistance to the primary producers' defences results in a decrease in the primary producers' mortality rate – the μ_2 related term in Fig. 13. The price is a decrease in the consumption efficiency – the χ_3 related term in (18); for example, a decrease in the conversion efficiency from plant to herbivore biomass.

These considerations lead us to modify (18) and write

$$\chi_1(x_1) = \chi_4 \exp\left(-\frac{x_1}{\sigma_1}\right), \quad \mu_1(x_1) = \mu_3\left(1 - \exp\left(-\frac{x_1}{\sigma_2}\right)\right),$$

$$\mu_2(x_2) = \mu_4 \exp\left(-\frac{x_2}{\sigma_3}\right), \quad \chi_3(x_2) = \chi_5 \exp\left(-\frac{x_2}{\sigma_4}\right)$$

and

$$\begin{aligned}
\partial_t z_1 &= v_3 v_1 \frac{z_3}{1 + v_2 z_3}\ Az_1 - \chi_1(x_1)\frac{z_1}{1 + \chi_2 z_1} z_2 - (\lambda_1 + \mu_1(x_1))z_1, \\
\partial_t z_2 &= \chi_3(x_2)\chi_1(x_1)\frac{z_1}{1 + \chi_2 z_1} Az_2 - (\lambda_2 + \mu_2(x_2))z_2, \qquad (22) \\
\partial_t z_3 &= \iota(t) + \mu_1(x_1)z_1 + \mu_2(x_2)\ z_2 - v_1 \frac{z_3}{1 + v_2 z_3} z_1 - \lambda_3 z_3,
\end{aligned}$$

where

$$Az_i = z_i + \frac{1}{2}\Delta^2 \eta_i \partial_{x_i x_i} z_i \quad i = 1, 2.$$

We let $\boldsymbol{x} := [x_1, x_2]$ and $z_i \equiv z_i(\boldsymbol{x}, t), \quad i = 1, 2, 3.$

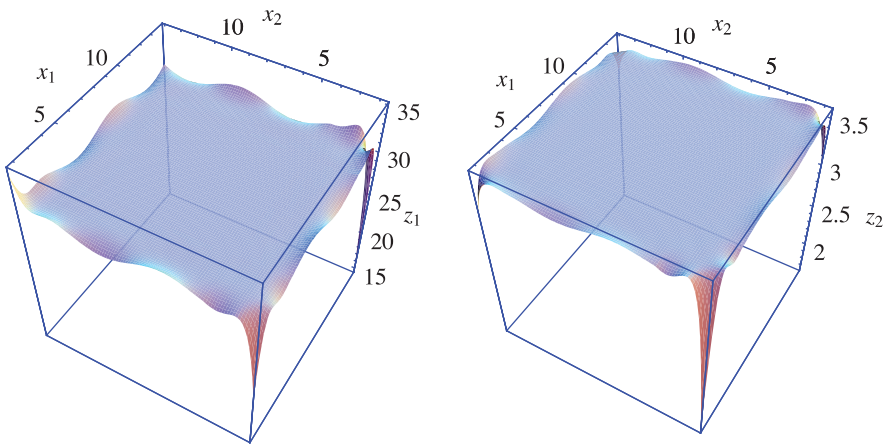

Fig. 16. Stable cyclic solution of (22) with parameter values (23), Neumann boundary conditions and initial conditions (24).

We use the following parameter values

$$\begin{aligned} &A = 10, \quad B = 4, \quad \lambda_1 = 0.01, \quad \lambda_2 = 0.1 \quad \lambda_3 = 0.01, \\ &\mu_3 = 0.01, \quad \mu_4 = 0.01, \quad \nu_1 = 100, \quad \nu_2 = 1, \quad \nu_3 = 1, \\ &\chi_2 = 1, \quad \chi_4 = 1, \quad \chi_5 = 0.4, \quad \sigma_1 = \sigma_2 = \sigma_3 = \sigma_4 = 10^6, \\ &\Delta = 0.01, \quad \eta_1 = \eta_2 = 0.01, \end{aligned} \tag{23}$$

with the Neumann boundary conditions and initial data

$$\begin{aligned} z_1(\boldsymbol{x}, 0) &= 20 + 2\cos(x_1 + x_2), \\ z_2(\boldsymbol{x}, 0) &= 1 + 0.1\cos(x_1 + x_2), \\ z_3(\boldsymbol{x}, 0) &= 0.006 \end{aligned} \tag{24}$$

to obtain a stable cyclic solution (Fig. 16). For the most part, the stable surface preserves the initial conditions except in the extremes of the adaptive traits.

The theory provides interesting predictions. The ED of the primary producers illustrates a marked decrease in fitness (density at stability) of those producer phenotypes that are at the extremes (high or low) of their adaptive traits. These low densities correspond to consumer phenotypes at the extreme high values of tolerance to the producers' resistance to consumption (high values of x_2). In other words, if you are a producer co-evolving with a consumer in a resistance-tolerance (to consumption) adaptive space, you want to be neither devoid of resistance nor too resistant because in either case your death rate is too high. However, the disadvantage of being in the extreme of your adaptive trait disappears when you co-evolve with consumers with low tolerance to your defences – there are no dips in abundance of x_1 phenotypes at the extremes of resistance to consumption by consumers with low tolerance to your defences (small values of x_2).

4. DISCUSSION

Albeit applicable to a large class of population models, ED are not applicable to discrete models. Deriving analytical criteria for stability for a system of non-linear PDE is tedious, but in principle follows the usual procedure in ordinary differential equations, such as through common linearization techniques. Stability should not be viewed as the holy grail of mathematical biology. The often made claim that unstable systems are not likely to be observed in nature may not be true, particularly if transients are slow. After all, all species are doomed to extinction sooner or later. Transient phenomena in mathematical models can produce rich results that mimic nature. Also, stability in ED does not mean that the ED will collapse to a point process and eventually will not allow mutations. In fact, mutations are the very reason that we obtain stable surfaces that are not necessarily homogeneous.

The significant contributions of the theory of ED to evolutionary ecology boil down to: (i) ED do not require a game-theoretic approach to the study of co-evolution in adaptive space; (ii) because ED covers all of the adaptive space, invasion of mutants to a stable ED is possible for a short time only – we thus do not need to invoke the ESS concept; (iii) the theory admits that fitness of phenotypes at stable ED can be positive, negative or zero – the fitness of phenotypes must be viewed in the context of fitness of the whole distribution. Thus, as in nature, ED allow phenotypes of various fitness values to co-exist and resist invasion.

As with all differential equation models, ED are limited to large, yet finite populations. There is a common misconception that continuous models in bounded domains imply infinite population sizes. This is not the case simply because the integrals of phenotype densities over bounded domains *are* finite.

Stochastic effects are ignored. For small populations, evolutionary ecology systems should be modelled with probabilities. Ignoring stochastic effects is more difficult to justify. However, we are often interested in the range of phenomena that deterministic models exhibit. For example, deterministic models that produce chaotic behaviour are interesting precisely because they produce stochastic-like behaviour. In the case of ED, the fact that entirely different solutions emerge from different initial conditions (Figs. 11 and 12) indicates that perturbations (stochastic or otherwise) can move a system of ED to different regions in the solution space.

One difficulty with ODE models is that they assume smooth functions and therefore the transition from an individual-based approach to the continuous approach needs to be justified (as in Dieckmann and Law, 1996, for example). In the case of PDE, this problem vanishes because the theory of PDE, from its foundations, relies on measure theory and on generalized functions (Renardy and Rogers, 1993; Evans, 1998). Thus, the extension from points (individuals) to intervals is natural. Because of the functional space within which PDE operate, solutions that are not smooth and even not continuous are acceptable (for examples, see Smoller, 1982; Cohen, 2005).

The richness of solutions that ED can produce, and the fact that they need not be unique, raise tantalizing possibilities in modelling evolution via ED. For example, Abrams *et al.* (1993) raised the possibility of stable ESS where fitness is at its minimum (see also Cohen *et al.*, 2000b, 2001). The results illustrated in Fig. 7 support Abrams and co-workers' (1993) finding. These results also indicate that phenotypes with a spectrum of fitness values can co-exist in stable ED. The whole issue of maximizing fitness becomes irrelevant when we deal with stable ED. For example, in the predator–prey system (Fig. 7), there are many regions where the slope of

the surface (instantaneous fitness) is zero and others where it is positive or negative. Yet, the surface is stable. We must therefore revise the notion that stable evolutionary systems should reflect special points in the fitness space.

We invoke orthogonality of traits in the mutation operator (9). This might appear as a restrictive assumption. However, many adaptive traits are (or seem to be) unrelated: it is difficult to see the connection between the length of an elephant's trunk and the colour of his skin. Furthermore, if adaptive traits are not orthogonal, then they can be made so (with rotation methods akin to principal components analysis). Finally, there is nothing in the theory of ED that restricts one from using non-orthogonal traits. The mutation operator will simply need to include mixed partial derivatives.

In developing the framework for ED, we claimed that birth is a linear function (see equation 7). Incorporating non-linear birth processes introduces some notational difficulties, but no conceptual difficulties. In using the Taylor series expansion, one will need to invoke the chain rule for the Taylor series approximation to hold (see Cohen, 2008).

The fact that solutions on the boundaries of the adaptive space differ from those in the interior (where we may find zero densities in some regions of the adaptive space) should come as no surprise. For certain classes of PDE, there are maximum principles that establish that the maxima of solutions should always be on the boundaries (see, for example, Evans, 1998). From an evolutionary perspective, this makes sense – natural selection in a bounded domain is expected to push the most fit organisms to the boundaries of the adaptive space. A good example is the body temperature of birds, which is close to the temperature of protein denaturation – possibly because based on thermodynamic principles, it is easier to warm up than to cool down. Another possible example is the tremendous size of the Irish elk antlers that eventually led to its extinction (Moen *et al.*, 1999).

The application of ED to ecosystem models opens the door to (at least theoretical) investigations of how co-evolution responds to inanimate components of systems in nature. Such applications offer one way to address the issues of global change in the context of co-evolution.

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