

Distributed predator–prey co-evolution

Yosef Cohen*

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

ABSTRACT

An evolutionary distribution $z(\mathbf{x}, t)$ is a distribution of the density of organisms along the values of adaptive traits \mathbf{x} at time t . Predator and prey evolutionary distributions with competition, assortative mating and mutations display dynamics that include cyclic waves and other esoteric phenomena. Such phenomena depend on the parameter space, on the instability of the predator–prey dynamics, on initial conditions and on the nature of both intra- and inter-distribution interactions. Traditional reaction-diffusion models treat polynomial reaction terms. Here the reaction terms are rational. Diffusion and reaction terms in the reaction-diffusion equations emerge from first principles, including selective predation, assortative mating, mutations and competition. Non-homogeneous distributions of phenotypes along trait values emerge from non-homogeneous perturbations. The non-homogeneity, albeit stable, depends on the specific non-homogeneous perturbation. The emerging patterns are internal to population processes (predator–prey). When external, non-homogeneous selection is imposed, the predator–prey dynamics exhibit high-frequency changes in evolutionary types on the boundaries of the smooth transitions of external selective pressures.

Keywords: evolutionary distributions, evolutionary games, predator–prey co-evolution, reaction diffusion equations.

INTRODUCTION

The dynamics of predator–prey populations have been of major interest in ecology and mathematics. It started with Volterra's (1926) celebrated model (named after both Lotka and Volterra; see Lotka, 1920, 1925) and continued through the striking Lynx-Hare cycles (Elton and Nicholson, 1942). Currently, much interest is focused on predator–prey systems in space (e.g. Smoller, 1982; Murray, 1989; Leung, 2001). The starting model is usually the point process

$$\begin{aligned}\dot{\mathbf{u}} &= \mathbf{f}(\mathbf{u}, \mathbf{v}) \\ \dot{\mathbf{v}} &= \mathbf{g}(\mathbf{u}, \mathbf{v})\end{aligned}\tag{1}$$

where $\dot{\mathbf{u}}$ and $\dot{\mathbf{v}}$ are the time derivatives of say n prey and m predator populations. The vector valued functions \mathbf{f} and \mathbf{g} describe species interactions (Abrams and Ginzburg, 2000). To this model one adds diffusion terms, usually via constant diagonal matrices, to get

* e-mail: cohen006@tc.umn.edu

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$$\begin{aligned}\dot{\mathbf{u}} &= \mathbf{D}\Delta\mathbf{u} + \mathbf{f}(\mathbf{u}, \mathbf{v}) \\ \dot{\mathbf{v}} &= \mathbf{E}\Delta\mathbf{v} + \mathbf{g}(\mathbf{u}, \mathbf{v})\end{aligned}\quad (2)$$

where \mathbf{D} and \mathbf{E} are the diffusion matrices and Δ is the Laplacian (when Δ refers to a small quantity, it will always be accompanied by x ; i.e. Δx). Such systems are called reaction-diffusion (R-D) equations (see, for example, Smoller, 1982). The first term on the right-hand side is the diffusion and the second is the reaction. These have received considerable interest in ecology (Segel and Jackson, 1972; Levin and Segel, 1976; Rosen, 1977; Mimura and Murray, 1978; Okubo, 1980; Conway, 1984; Murray, 1989; Dieckmann *et al.*, 2000; Alonso *et al.*, 2002).

Regarding R-D equations, two issues are of particular interest. The first refers to the stability of the system and the second to the homogeneity of the distribution of the predators and prey across space. It has been shown that the predator-prey R-D system may result in what one might call spontaneous spatial patterns. Spontaneous because the patterns emerge from the nature of the interaction terms; they are not imposed externally on the system (Mimura and Murray, 1978; Okubo, 1980; Conway, 1984; Arditi *et al.*, 2001; Leung, 2001; Alonso *et al.*, 2002).

Both the point process system (1) and the R-D system (2) incorporate underlying assumptions that are routinely addressed in population ecology models (e.g. structured populations), but rarely in evolutionary ecology models. First, all organisms are considered identical; alternatively, one assumes that there is no loss of generality in treating the dynamics via an average individual. Second, reproduction is by cloning; alternatively, one assumes that an average individual produces an average individual. Third, the diffusion term is superimposed on the system arbitrarily and often without reference to the underlying bioecological mechanisms. One can hardly expect the diffusion terms to be independent in biological communities where species interact. Fourth, species are considered discrete; the number of equations in the model is arbitrary. Finally, in treating the system from a strictly mathematical perspective, one usually takes \mathbf{f} and \mathbf{g} to be polynomials of appropriate degrees. Murray (1989) argued that polynomial predator-prey models are not very 'realistic'. These assumptions lead to difficulties in incorporating evolution in the system (but see Brown, 1987; Vincent *et al.*, 1993; Cohen *et al.*, 2000, 2001). Cohen (2003) addressed some of these issues in the context of a single evolutionary distribution.

To fix ideas, let us start with some definitions. An evolutionary distribution is defined as a time-dependent distribution of individuals with respect to a set of adaptive traits. For example, for z scalar and \mathbf{x} vector of n adaptive traits, an n -dimensional evolutionary distribution is described by $z(\mathbf{x}, t)$. Adaptive traits are defined as phenotypic traits that are subject to evolution by natural selection. Examples of adaptive traits might be height, weight, sex, colour and behavioural categories. A single evolutionary distribution describes changes in the density of organisms of phenotype \mathbf{x} for which the consequences of changes in the values of \mathbf{x} are similar. These are organisms that function similarly in biotic communities. For example, to an ecological community there corresponds a single evolutionary distribution for all prey. Predators will have another distribution. Thus, a complete description of the evolution of distributions 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 densities and \mathbf{y} is a vector of p (possibly time-dependent) input components.

To avoid the pitfalls inherent in classifying organisms into, say, species, sub-species and so on, I use the concept of types. A type is defined as a set of individuals whose trait values

on the evolutionary distribution form a continuous interval that contains special points (continuity is not necessary, but it simplifies the discussion). Special points are local minima or maxima. An evolutionary distribution at a particular t is illustrated in Fig. 1. The areas A and B isolate two types. So does the rectangle C. From this perspective, the classification of organisms into types is arbitrary. It depends on biological and ecological details and the scale of the traits. Scale here is not as important as it may seem because all organisms are evolving on the same scale, x . For example, x may represent tree-height. An observer walking through a forest might surmise that the distribution in Fig. 1 represents a forest with three 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.

To keep the mathematics from clouding the issues at hand, I deal with scalar traits only. Because traits are phenotypic, there are good reasons to consider bounded trait values between, say, \underline{x} and \bar{x} (see Fig. 1). Traits are therefore represented by a section on the real line (again, this requirement is not necessary, but it simplifies the discussion). The first derivatives of the evolutionary distributions at the boundaries are taken to be zero – there is no boundary crossing.

Cohen (2003) discussed competition and other aspects of evolutionary distributions for a single distribution. Here I deal with two interacting distributions. Evolution is incorporated via mutations, assortative mating, predation and competition. Mutations refer to the phenomena where organisms differ in the value of their adaptive trait x from their progenitors. Assortative mating refers to the tendency of types to mate with types in ways other than uniformly random. Predation is defined as processes that increase the mortality of prey and decrease the mortality of predators. Competition increases the mortality of the same functional organisms (i.e. those who share a single evolutionary distribution). Thus, predation operates across evolutionary distributions and competition within. Cannibalism operates like predation, but within a distribution.

The R-D equations are derived from first principles. My purpose is to show the rich set of evolutionary consequences that relatively simple and parameter-specific evolutionary models produce. The derivations of appropriate models will always result in reaction-diffusion equations. The evolutionary principles that drive the models may or may not result in distribution-independent diffusion terms. Furthermore, the diffusion itself is derived from the logic of the models.

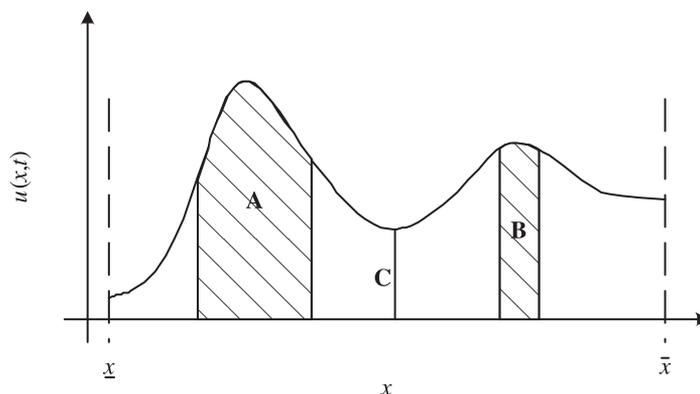


Fig. 1

About notation. **Bold face** letters denote vectors and matrices. Functions are denoted with parentheses; so are vector valued functions. Letters without parentheses denote vectors or scalars. For example $\boldsymbol{\beta}(\cdot)$ is a vector valued function of some variables, $\boldsymbol{\beta}$ is a vector of constants, $\beta(\cdot)$ is a function of some variables and β is a scalar. All scalars are positive real numbers. Partial derivatives are denoted with subscripts (e.g. $z_{xx} = \partial^2 z / \partial x^2$). To simplify the notation, for $\mathbf{a} = [a_1, a_2]$ and $\mathbf{b} = [b_1, b_2]$, for example, the product \mathbf{ab} is interpreted as $[a_1 b_1, a_2 b_2]$.

DISTRIBUTED PREDATOR-PREY CO-EVOLUTION

Denote by $\mathbf{z}(x, t) = [u(x, t), v(x, t)]$ the population densities of prey and predators. Here x is the adaptive trait. Assume that $v(x, t)$ preys upon $u(x + j\Delta x, t)$, $j = -1, 0, 1$ where Δx is small. Similarly, $u(x + j\Delta x, t)$ are preyed upon by $v(x, t)$. For example, if the adaptive trait is size, then v preys upon u of the same size and upon slightly larger and slightly smaller prey. If the adaptive trait is location, then v preys upon nearby u . Denote the mutation rates from x to $x \pm \Delta x$ by prey and predators by $\boldsymbol{\eta} = [\eta_1, \eta_2]$. Mutations occur in newborns. Let $\boldsymbol{\beta}(\cdot) = [\beta_1(\cdot), \beta_2(\cdot)]$ and $\boldsymbol{\mu}(\cdot) = [\mu_1(\cdot), \mu_2(\cdot)]$ be the rates of additions to and subtractions from populations for the prey and the predators. To simplify notations, \cdot denote variables that will be specified later. Assortative mating is limited to nearly like phenotypes – organisms with trait value x reproduce with organisms with trait values $x + j\Delta x$. Now

$$\mathbf{z}_t(x, t) = \boldsymbol{\beta}(\cdot) - \boldsymbol{\mu}(\cdot) \tag{3}$$

As a first approximation, assume that the rate of addition is due to birth and that it is proportional to the population density with proportionality constants $\boldsymbol{\beta}$. Then

$$\boldsymbol{\beta}(\cdot) = \boldsymbol{\eta} \boldsymbol{\beta} \Delta x^2 \mathbf{z}_{xx} + \boldsymbol{\beta} \mathbf{z}$$

(see Cohen, 2003). Subtractions occur due to mortality and predation. Mortality is assumed proportional to \mathbf{z} , with the mortality coefficients functions of \mathbf{z} . To a first approximation consider mortality coefficients that are linear functions of \mathbf{z} . The mortality coefficients include a saturation effect. Then

$$\boldsymbol{\mu}(\cdot) = \boldsymbol{\mu}(x, t) \mathbf{z}(x, t)$$

where

$$\mu_1(x, t) = \mu_1 u(x, t) + \frac{a}{u(x, t) + c} [v(x - \Delta x, t) + v(x, t) + v(x + \Delta x, t)]$$

$$\mu_2(x, t) = \mu_2 \frac{b}{u(x - \Delta x, t) + u(x, t) + u(x + \Delta x, t)} v(x, t)$$

Here a is the maximum consumption rate, b is the conversion efficiency and c is the saturation constant (Ruxton *et al.*, 1992; Huisman and de Boer, 1997). Therefore

$$\boldsymbol{\mu}(x, t) = \begin{bmatrix} \mu_1(x, t) \\ \mu_2(x, t) \end{bmatrix} = \begin{bmatrix} \mu_1 u + \frac{a}{u+c} (\Delta x^2 v_{xx} + 3v) \\ \mu_2 \frac{b}{\Delta x^2 u_{xx} + 3u} v \end{bmatrix}$$

(see Cohen, 2003). Here u and v are to be understood as dependent on x and t . Assume that $\Delta x^2 u_{xx} + 3u > 0$ (with $u > 0$ this assumption is always satisfied if one chooses Δx small enough). Therefore

$$\mathbf{z}_t = \mathbf{k} \mathbf{z}_{xx} + \mathbf{f}(\cdot) \mathbf{z} \tag{4}$$

where

$$\mathbf{k} \triangleq \begin{bmatrix} k_1 \\ k_2 \end{bmatrix} \triangleq \begin{bmatrix} \eta_1 \beta_1 \Delta x^2 \\ \eta_2 \beta_2 \Delta x^2 \end{bmatrix}$$

$$\mathbf{f}(\cdot) \triangleq \begin{bmatrix} f_1(\cdot) \\ f_2(\cdot) \end{bmatrix} \triangleq \begin{bmatrix} \beta_1 - \mu_1 u - \frac{a}{u+c} [\Delta x^2 v_{xx} + 3v] \\ \beta_2 - \mu_2 \frac{bv}{\Delta x^2 u_{xx} + 3u} \end{bmatrix} \tag{5}$$

The solutions of these equations, $\mathbf{z}(x, t)$, represent the evolutionary distributions of the predator and prey with respect to the adaptive trait. Without evolution, this is precisely the model proposed by Murray (1989, pp. 70–77) as being realistic. The ordinary differential equation version of the model can produce predator–prey cycles. The evolution of the predator and prey distributions (4) is different from standard reaction-diffusion models:

$$\mathbf{z}_t = \mathbf{k} \mathbf{z}_{xx} + \mathbf{f}(\mathbf{z})$$

(where \mathbf{k} is usually a diagonal diffusion matrix with constant coefficients). In (4), the dynamics of u depend on the evolutionary changes in v (as expressed via v_{xx} in f_1). Similarly, the dynamics of v depend on evolutionary changes in u . These arise from selective predation. The mutation-dependent evolutionary changes (as expressed via $k_1 u_{xx}$ and $k_2 v_{xx}$) are independent for u and v (as they should be). Of course, the trait x may represent location. Because of (4)'s mathematical peculiarities (there are rational as opposed to polynomial terms), a general mathematical analysis of the solutions will marginalize the interpretation of the evolutionary dynamics of (4). The next example is needed for baseline comparisons (see Murray, 1989).

Example 1 (no evolution). *The dynamics of (4) with the dependence on x removed (that is without evolution) and with*

$$a = \frac{1}{3}, b = 3, c = 0.2, \boldsymbol{\eta} = \begin{bmatrix} 0.01 \\ 0.01 \end{bmatrix}$$

$$\boldsymbol{\beta} = \begin{bmatrix} 1.01 \\ 0.02 \end{bmatrix}, \boldsymbol{\mu} = \begin{bmatrix} 1.00 \\ 0.02 \end{bmatrix}, \mathbf{z}(0) = \begin{bmatrix} 0.20 \\ 0.35 \end{bmatrix} \tag{6}$$

exhibit a limit cycle (Fig. 2).

EQUILIBRIUM AND STABILITY

At equilibrium, $\mathbf{z}_t \equiv 0$. Therefore, (4) becomes

$$\mathbf{0} = \mathbf{g}(\cdot) \mathbf{z}'' + \mathbf{f}(\cdot) \mathbf{z} \tag{7}$$

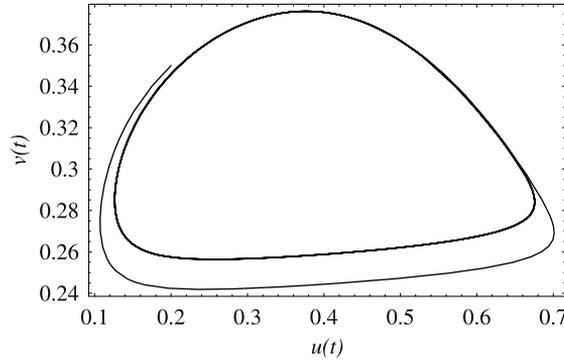


Fig. 2

where $\mathbf{z}'' = d^2\mathbf{z}/dx^2$ and the functions \mathbf{g} and \mathbf{f} are

$$\mathbf{g}(\cdot) = \begin{bmatrix} (u+c)k_1 - a\Delta x^2 u \\ \beta_2 \Delta x^2 v \quad (\Delta x^2 u'' + 3u)k_2 \end{bmatrix}$$

$$\mathbf{f}(\cdot) = \begin{bmatrix} [(\beta_1 - \mu_1 u)(u+c) - 3av] \\ 3\beta_2 u - \mu_2 bv \end{bmatrix} \tag{8}$$

The ‘diffusion’ functions in the matrix $\mathbf{g}(\cdot)$ are not constants. Therefore, the analysis of (7) will be local. From the mathematical theory of reaction diffusion equations (Smoller, 1982), the stability of the solutions of (7) depends on \mathbf{f} . First, note that f_1 is cubic and, therefore, has 3 zeros. f_2 is quadratic and has two zeros. It turns out that of the six solutions of $\mathbf{f} = 0$, only one results in both u and v positive and real. This remains true for a range of parameter values. To show that solutions may exhibit periodic travelling waves, one needs to show that the equilibrium solutions of (7) are unstable.

Example 2 (instability of the equilibrium solution). For \mathbf{f} in (8) with parameter values as in Example 1,

$$\tilde{u} = 0.364, \quad \tilde{v} = 0.364$$

($\tilde{\cdot}$ indicate equilibrium values). The eigenvalues of this solution are complex conjugates with positive real parts

$$\lambda_{1,2} = 0.00387 \pm 0.085463i$$

and the equilibrium solution is unstable.

Next, one needs to show that there is an invariant region that contains the equilibrium point. These are regions that once the solution enters, it stays in. If upper and lower bounds on u'' are assumed, then the existence of an invariant region can be shown by one of several methods (Leung, 2001). An invariant region in this case is easy to show numerically. Simply start from various initial conditions on a box that contains the limit cycle in Fig. 2 and observe the trajectories entering the region.

Next, let us turn to the analysis of perturbations of the predator–prey system (4).

HOMOGENEOUS PERTURBATIONS

As Turing (1952) noted, homogeneous perturbations to partial differential equations may not reveal salient dynamics of a system (see also Britton, 1986). The dynamics of (4) with parameter values as in Example 1 and the additional parameter and data (boundary and initial conditions)

$$\Delta x = 0.001$$

$$\mathbf{z}(x, 0) = \begin{bmatrix} 0.20 \\ 0.35 \end{bmatrix}, \mathbf{z}_x(0, t) = \mathbf{z}_x(\pi, t) = \mathbf{0}$$

are shown in Fig. 3 for the prey. Note that, based on the stability analysis, these are the parameter values that should produce interesting evolutionary dynamics. The boundary conditions reflect the fact that the trait values are constrained between 0 and π . The dynamics of the predators' evolutionary distribution are similar (with a phase delay). From an evolutionary perspective, one concludes that all organisms are equally distributed. Populations fluctuate, but the distributions of phenotypes over the adaptive trait remain uniform. In other words, no types are recognized.

To an observer, these dynamics appear as if no evolution occurs. Populations fluctuate, but the relative frequencies of types remain unchanged. One can hardly expect homogeneous perturbations of trait values to be common.

NON-HOMOGENEOUS PERTURBATIONS

Now use (4) with data as before except for the initial conditions:

$$\mathbf{z}(x, 0) = \begin{bmatrix} 0.20 + 0.05 \cos(2x) \\ 0.35 + 0.08 \cos(2x) \end{bmatrix}, \mathbf{z}_x(0, t) = \mathbf{z}_x(\pi, t) = \mathbf{0} \tag{9}$$

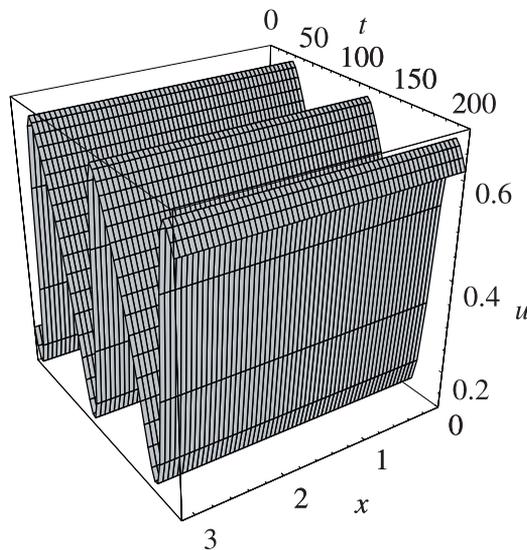
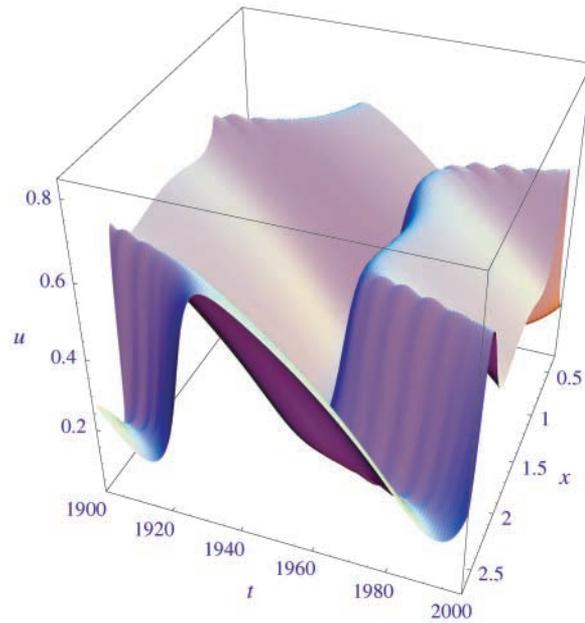
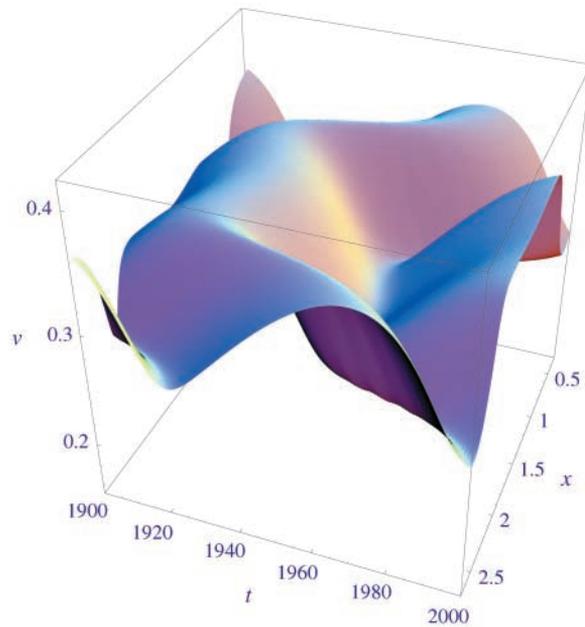


Fig. 3

The trajectory of the distributions remains cyclical. However, the shape of the evolutionary distribution is more elaborate. Figures 4 and 5 show about one cycle of the distributions. An observer looking at a cross-section over time sees a complex periodic front. Cycles are

**Fig. 4****Fig. 5**

synchronized with a phase delay between the prey and the predator (Fig. 6). Before discussing the evolutionary implications of these predator-prey dynamics, it is instructive to interpret typical cross-sections of the dynamics from an observer standpoint.

In the early 1940s, an observer would see a range of trait values in which predators are more abundant than prey, although they prey on prey of roughly the same trait values (say size). At the extremes, prey are more abundant (Fig. 7). If special points along the distribution (say minima and maxima) are identified with some biological classification (see Fig. 1), then the observer would conclude that there are roughly four types of prey (two common and two rare). Similarly with predators. However, the abundance of these types are reversed for the predator and prey. In the mid-1940s, prey types become more distinct ('speciation' if you will). Not so the predators. The prey become relatively (to the predators) more abundant. In the 1950s, prey are more abundant and clear types emerge. The relative

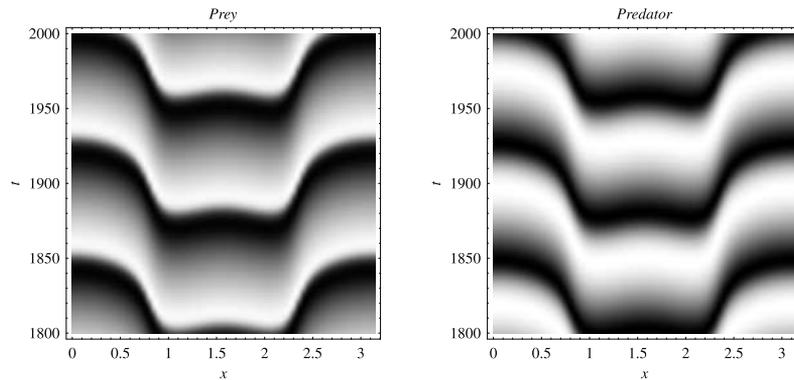


Fig. 6. Light = high intensity, dark = low intensity.

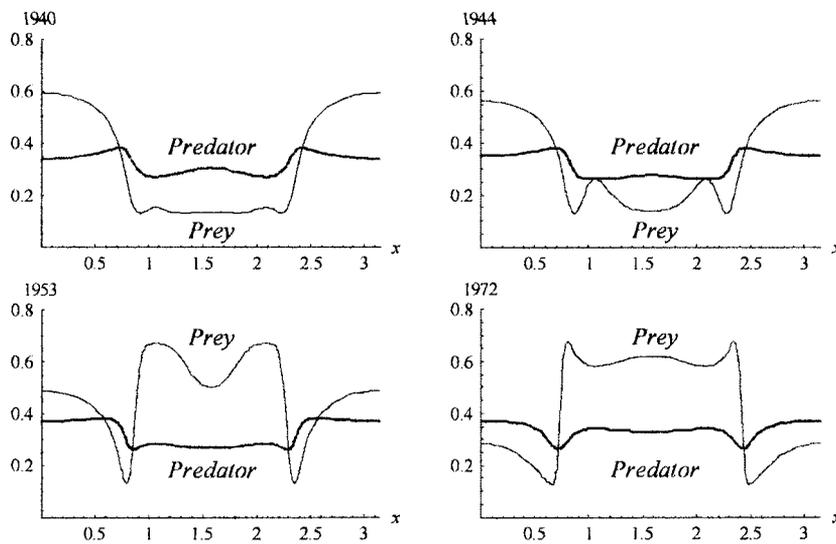


Fig. 7

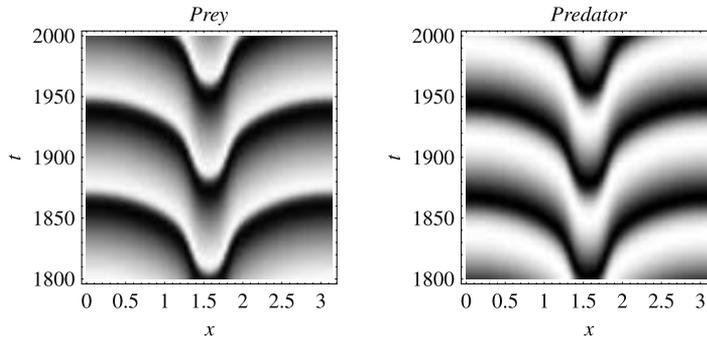


Fig. 8. Light = high intensity, dark = low intensity.

abundances of types are reversed for the prey and predator. In the 1970s, both prey and predators seem to become distinct – their densities along some values of the trait are narrowly peaked. Abundance of phenotypes in the context of adaptive traits associate the changes above with evolutionary dynamics.

Common to all of these dynamics is the fact that although predators feed on prey with similar trait values, the abundances of types are reversed in time. This is due to the lag in the evolution of the predator dynamics behind the prey. The lag is both in time and along the evolutionary trait values. This may be interpreted wrongly if the observer is not aware of the evolutionary dynamics. For example, if the trait is location, the observer might conclude that prey and predators seem to ‘evolve’ to occupy different spaces.

The gallery of dynamics depends within a certain range (continuously!) on the initial conditions. There is a threshold amplitude of the non-homogeneous perturbations. Below this threshold the dynamics settle to those of homogeneous perturbations shown in Fig. 3. As the amplitude of the perturbations in (9) increase, the evolutionary distributions change. For example, when the initial data in (9) change to

$$z(x, 0) = \begin{bmatrix} 0.30 + 0.08 \cos(2x) \\ 0.50 + 0.16 \cos(2x) \end{bmatrix}$$

Fig. 8 is obtained (cf. Fig. 6). As the amplitude of the perturbations (9) continues to increase, stability eventually breaks down. The Dirichlet boundary conditions give qualitatively similar results.

PREDATION AND COMPETITION

The term $\mu_1 u$ in (5) may incorporate competition with own type. Suppose that u of type x compete with types $u(x + j\Delta x)$, $j = -1, 0, 1$. Then, the prey equation in (4) becomes

$$u_t = g(u) u_{xx} + u f_1(\cdot)$$

where

$$g(u) = k_1 - \mu_1 \Delta x^2 u$$

$$f_1(\cdot) = \beta_1 - \mu_1 u - 3u - \frac{a}{u + c} (\Delta x^2 v_{xx} + 3v)$$

This amounts to increasing the death rate for u , and slowing the ‘diffusion’ term along values of the evolutionary trait. With the same parameter and data as in (6) and (9), the evolutionary distribution becomes homogeneous. However, increasing the birth rate and decreasing the death rate brings the dynamics back to non-homogeneous evolutionary distribution. For example,

$$\beta = \begin{bmatrix} 2 \\ 0.053 \end{bmatrix}, \mu = \begin{bmatrix} 0.01 \\ 0.02 \end{bmatrix}$$

$$\mathbf{z}(x, 0) = \begin{bmatrix} 0.4 + 0.2 \cos(2x) \\ 0.4 + 0.2 \cos(2x) \end{bmatrix}, \mathbf{z}_x(0, t) = \mathbf{z}_x(2\pi, t) = 0$$

(and the remaining parameters as before) generate Fig. 9. To an observer of the evolutionary distributions at the turn of the millennium (Fig. 10), it seems that there are more types of predators than of prey. In addition to decreasing prey abundance, it seems that competition smooths the evolutionary distribution of types.

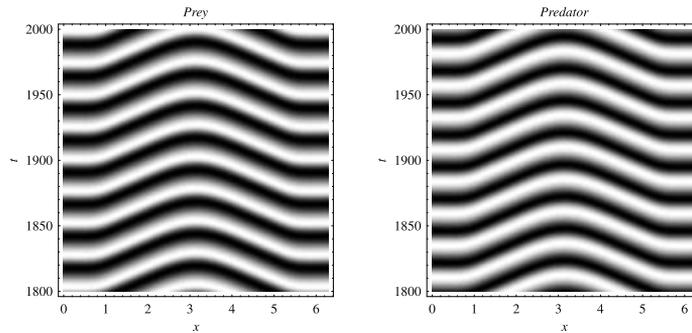


Fig. 9. Light = high intensity, dark = low intensity.

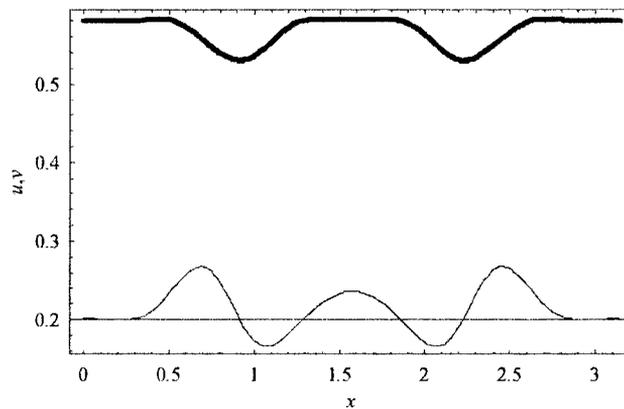


Fig. 10. Bold curve = predator, thin curve = prey.

COMPETITION WITH MORE THAN JUST THY NEIGHBOUR

Suppose that prey compete with all types, not just nearby ones. Then $\mu_1(\cdot)$ in (3) becomes

$$\mu_1(x, t) = \mu_1 \int_x^{\bar{x}} \alpha(x, \zeta) u(\zeta, t) d\zeta + \frac{a}{u+c} (\Delta x^2 v_{xx} + 3v)$$

where $x \in [\underline{x}, \bar{x}]$. From the mean value theorem, there exists $q(t)$, $\underline{x} \leq q(t) \leq \bar{x}$, such that

$$\mu_1(x, t) \stackrel{\Delta}{=} \int_x^{\bar{x}} \alpha(x, \zeta) u(\zeta, t) d\zeta = \alpha(x, q(t)) u(q(t), t) (\bar{x} - x)$$

In other words, there is a unique solution to the equations

$$u_t = k_1 u_{xx} + u f_1(\cdot)$$

$$v_t = k_2 v_{xx} + v f_2(\cdot)$$

where

$$f_1(\cdot) = \beta_1 - \mu_1 \gamma - \frac{a}{u+c} (\Delta x^2 v_{xx} + 3v)$$

and f_2 is given in (5). The function $\alpha(x, \zeta) > 0$ need not be monotonic. It does not even need to be smooth. In other words, almost any conceivable scheme of competition among organisms who share an evolutionary distribution may be applied. Of course, one can also use α that has negative values. In such cases, one has cooperation among phenotypes that share an evolutionary distribution.

Example 3 (predator and prey with competition). *As a specific example choose*

$$\alpha(x, \zeta) = \alpha_1 (1 + \exp[-\alpha_2 (x - \zeta)^2]) \quad (10)$$

This choice models an ever-decreasing competition among dissimilar types. The parameter α_1 scales the competition and α_2 controls the rate of decrease in the intensity of competition as organisms' trait values become more dissimilar. With $\alpha_i = 1$ and the remaining parameters as previously, results similar to those shown in Figs 9 and 10 are obtained.

The significance of competition with potentially distant types is that the dynamics now become more like an evolutionary game. Equation (10) says: 'If you wish to minimize the effect of competition on your mortality, be as different as possible from everybody else; however, keep in mind that this is what everybody else wants to do'. This case was analysed by Cohen (2003). So far, I have addressed the important issue of how heterogeneity in the evolutionary distributions arise from the evolutionary process itself. Selection was assumed to be homogeneous along the evolutionary trait. Frequently, environmental, physiological and biochemical constraints impose non-homogeneous selection within the domain of x . One such constraint is reflected in the bounds of the adaptive traits (between 0 and 2π in the examples above). Next I address non-homogeneous selection within the domain of the adaptive trait, as opposed to on its boundary. Non-homogeneous selection can, and often is, imposed by environmental heterogeneity.

NON-HOMOGENEOUS SELECTION

Suppose that, for some reason, there are special sections (not necessarily connected) along the values of the adaptive trait. Along these sections, selection is different from other sections. For example, the adaptive trait might be height and grazers live in a habitat where browse is either low or high. Those browsers with intermediate height are selected against through higher mortality rates than short or tall herbivores. Another example might be the environment where prey live in. The environment is structured such that dark and light colours provide camouflage from predators. Intermediate colours are more visible. This should lead to the evolution of colour phases of the prey types. Let us now see the consequences of these considerations.

To keep things simple, assume that the environmentally imposed selection operates on the mortality of the prey. Then the dynamics are those of (4) with $f_1(\cdot)$ in (5) modified to

$$f_1(\cdot) = \left[m_1 - \mu_1(x)u - \frac{a}{u+c} (\Delta x^2 v_{xx} + 3v) \right] u$$

Here $\mu_1(x)$ expresses the externally imposed selection. To fix ideas, let us use

$$\mu_1(x) = \mu_1 \left(1 + \exp \left[-d \left(x - \frac{\bar{x}}{2} \right)^2 \right] \right)$$

This imposes a maximum selective pressure of $2\mu_1$ focused at the middle of the range of x . The parameter d dictates how sharp the focus of selection is – the larger it is, the more focused the selection is. With large d , the mortality rate on both sides of the selection focus is roughly μ_1 .

Example 4 (non-homogeneous selection). *The parameters are given in (6), the data in (9) and take $d=1$. To examine details, increase the range of x from $[0, \pi]$ to $[0, 4\pi]$ (similar increase in the standard model (4) has no effect on the results above). The distribution of external selection is shown in Fig. 11. Thus, for $x < 4$ and $x > 8$, one is back to the standard model (4). The cross-section of the evolutionary distribution at $t = 2000$ is shown in Fig. 12. The density of the distribution is shown in Fig. 13.*

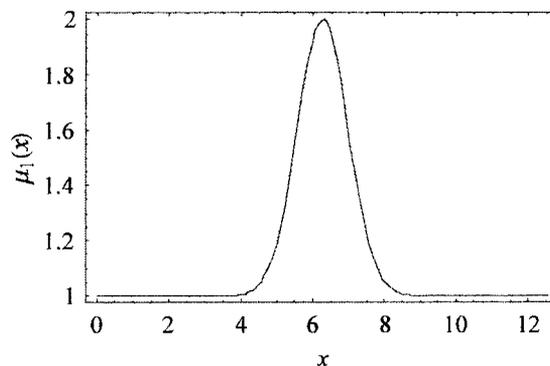


Fig. 11

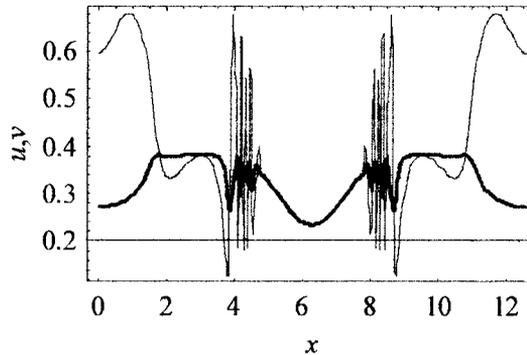


Fig. 12. Bold curve = predator, thin curve = prey.

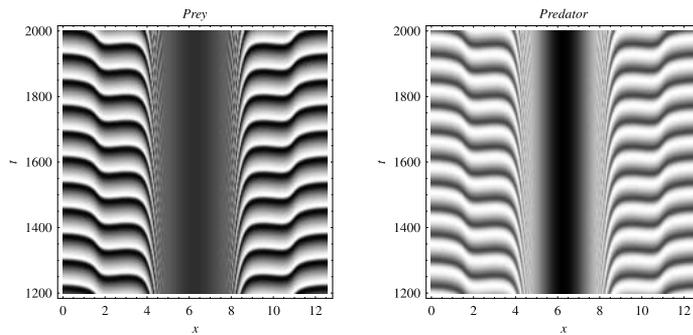


Fig. 13

The net effect of imposing external selection on an otherwise similar dynamics is more than just the increase in local selection along the adaptive trait (compare Figs 6 and 13). The transition from homogeneous to increased selection is smooth (Fig. 11). Yet, the predator–prey dynamics react quite differently. The transition zone is now occupied with high-frequency changes in both predator and prey distributions (Figs 12 and 13). In fact, there are local prey extinctions and many types of both predators and prey can be identified. This is an area of high ‘diversity’ along the evolutionary distribution. Non-homogeneous selection seems to disrupt the smooth transition among types along the evolutionary distributions.

An observer might conclude that there is a certain range of values of the adaptive trait in which one finds many types. A keen observer might conclude that this is so because of changes in selective pressure (however gradual it may be!)

DISCUSSION

The approach here was anticipated by Levin and Segel (1985). In particular, their approach to modelling long-range effects on redistribution (their equation 4.10) corresponds to Example 3. Keshet and Segel (1984) analysed a different predator–prey model. Their solution exhibited periodic non-uniform steady state where spikes in the distribution of

prey and predators were isolated by areas where the prey were not present (see also Mimura *et al.*, 1979).

The development above uses symmetric discrete jumps on the order of $\pm \Delta x^2$ to approximate a continuous to discrete process. The same discussion terms arise for any symmetric mutation process with variance Δx^2 (I am indebted to Steve Ellner for pointing this out to me). When selection, associative mating, predation and other processes are asymmetric (as they must be in many evolutionary processes), different results may emerge. This will be pursued elsewhere.

The approach here can be generalized to multi-trait evolutionary distributions and to more than two distributions with no new conceptual difficulties (and as usual with considerable numerical difficulties). The approach taken here suffers from some shortcomings. First, I did not introduce explicit genetic mechanisms. I have shown how this can be accomplished specifically (Y. Cohen, submitted). Second, I use continuous processes. This means that populations composed of discrete organisms are approximated. Thus, the approach here is not appropriate for small populations consisting of discrete organisms. Such populations are subject to extinctions by chance alone, and a probabilistic approach is called for.

This paper, together with Cohen (submitted), represents my effort to inch evolutionary ecology and population genetics closer together. It seems to me that once we have gone through the process of sequencing DNA, we are going to be faced with the much more difficult task of mapping these sequences to phenotypes. After all, selection operates directly on phenotypes. Furthermore, except for some spectacular successes in molecular genetics in identifying, for example, sex linked diseases, one can expect that most phenotypic traits are expressions of sets of genes. At some point, we will have to be concerned with more organismic-centric (as opposed to genetics-centric) evolution than we have thus far. For example, expressing mutations in terms of phenotypic differences along adaptive traits.

The analysis is based on numerical experiments of a specific system, as opposed to on general mathematical analysis (I did choose parameter values that make biological sense). This was done for two reasons. First, the theory of rational PDEs is not well developed and has received little attention. Polynomial reaction terms in reaction-diffusion equations have been treated in the literature extensively. One can make progress in the mathematics of rational PDE as presented here with simplifying assumptions. This can be done at the expense of bioecological insights. Second, my main objective here is to point out the range of possible evolutionary phenomena based on relatively simple models. These shortcomings cannot be swept under the rug. Yet, one has to start somewhere.

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REFERENCES

- Abrams, P.A. and Ginzburg, L.R. 2000. The nature of predation: prey dependent, ratio dependent or neither? *Trends Ecol. Evol.*, **15**: 337–341.
- Alonso, D., Bartumeus, F. and Catalan, J. 2002. Mutual interference between predators can give rise to Turing spatial patterns. *Ecology*, **83**: 28–34.

- Arditi, R., Tyutyunov, Y., Morgulis, A., Govorukhin, V. and Senina, I. 2001. Directed movement of predators and the emergence of density-dependence in predator-prey models. *Theor. Pop. Biol.*, **59**: 207–221.
- Britton, N.F. 1986. *Reaction-Diffusion Equations and Their Applications to Biology*. New York: Academic Press.
- Brown, J. 1987. Predator-prey co-evolution as an evolutionary game. In *Applications of Control Theory in Ecology* (Y. Cohen, ed.), pp. 83–101. Berlin: Springer-Verlag.
- Cohen, Y. 2003. Distributed evolutionary games. *Evol. Ecol. Res.*, **5**: 1–14.
- Cohen, Y., Vincent, T.L. and Brown, J.S. 2000. A G -function approach to fitness minima, fitness maxima, evolutionarily stable strategies and adaptive landscapes. *Evol. Ecol. Res.*, **1**: 923–942.
- Cohen, Y., Vincent, T.L. and Brown, J.S. 2001. Does the G -function deserve an F ? *Evol. Ecol. Res.*, **3**: 375–377.
- Conway, E.D. 1984. *Partial Differential Equations and Dynamical Systems*. Berlin: Springer-Verlag.
- Dieckmann, U., Law, R. and Metz, J., eds. 2000. *The Geometry of Ecological Interactions: Simplifying Spatial Complexity*. Cambridge: Cambridge University Press.
- Elton, C.S. and Nicholson, M. 1942. The ten-year cycle in numbers of lynx in Canada. *J. Anim. Ecol.*, **191**: 215–244.
- Huisman, G. and de Boer, R.J. 1997. A formal derivation of Beddington functional response. *J. Theor. Biol.*, **185**: 389–400.
- Keshet, Y. and Segel, L.A. 1984. Pattern formation in aspect. In *Modelling of Patterns in Space and Time* (W. Jager, ed.), pp. 188–197. Berlin: Springer-Verlag.
- Leung, A.W. 2001. Positive solutions for systems of PDE and optimal control. *Nonlinear Anal.*, **47**: 1345–1356.
- Levin, S.A. and Segel, L.A. 1976. Hypothesis for origin of planktonic patchiness. *Nature*, **259**: 659.
- Levin, S.A. and Segel, L.A. 1985. Pattern generation in space and aspect. *SIAM Rev.*, **27**: 45–67.
- Lotka, A.J. 1920. Updated oscillations derived from the law of mass actions. *J. Am. Chem. Soc.*, **42**: 1595–1599.
- Lotka, A.J. 1925. *Elements of Physical Biology*. Baltimore, MD: Williams & Wilkins.
- Mimura, M. and Murray, J.D. 1978. On a diffusive prey-predator model which exhibits patchiness. *J. Theor. Biol.*, **75**: 249–262.
- Mimura, M., Nishiura, Y. and Yamaguti, M. 1979. Some diffusive prey and predator systems and their bifurcation problems. *Ann. NY Acad. Sci.*, **316**, 490–510.
- Murray, J.D. 1989. *Mathematical Biology*. New York: Springer-Verlag.
- Okubo, A. 1980. *Diffusion and Ecological Problems: Mathematical Models*. Berlin: Springer-Verlag.
- Rosen, G. 1977. Effects of diffusion on the stability of the equilibrium in multispecies communities. *Bull. Math. Biol.*, **39**: 373–383.
- Ruxton, G.D., Gurney, W.S.C. and de Roos, A.M. 1992. Interference and generation cycles. *Theor. Pop. Biol.*, **42**: 235–254.
- Segel, L.A. and Jackson, J.L. 1972. Dissipative structure: an explanation of an ecological example. *J. Theor. Biol.*, **37**: 545–559.
- Smoller, J. 1982. *Shock Waves and Reaction-Diffusion Equations*. New York: Springer-Verlag.
- Turing, A.M. 1952. On the chemical basis of morphogenesis. *Phil. Trans. R. Soc. Lond. B*, **237**: 37–72.
- Vincent, T.L., Cohen, Y. and Brown, J.S. 1993. Evolution via strategy dynamics. *Theor. Pop. Biol.*, **44**: 149–176.
- Volterra, V. 1926. *Variations and Fluctuations of a Number of Individuals in Animal Species Living Together*, pp. 409–448. Translation in R.N. Chapman, 1931, *Animal Ecology*. New York: McGraw Hill.