

Distributed evolutionary games

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ABSTRACT

Standard population models in ecology and evolutionary ecology assume that all individuals are identical and reproduce by cloning. Often, the connection between evolutionary ecology, genetics and mating systems is not explicit. Functionally similar species are modelled with separate equations (e.g. ‘competing’ species). Here, a theory is developed that: (i) views a population as a distribution of individuals over adaptive character values; (ii) identifies various evolutionary types (e.g. species) with special points along this distribution; (iii) treats individuals as different with respect to the adaptive character value; (iv) incorporates ecological interactions among individuals along distributions; and (v) incorporates assortative mating and mutations.

A stable equilibrium distribution of individuals over the adaptive character (the strategy) is a distributed evolutionarily stable strategy (ESS). At the distributed ESS, individuals may be equally distributed over all values of the adaptive character. Conditions that give rise to a non-uniform distributed ESS are analysed. Organisms are classified into types – an abstract classification for which species, phenotypes, and so on are special cases. With distributions, when the stable distribution is not homogeneous with respect to the adaptive character, it makes no sense to talk about the fitness of an individual in isolation from that of others. We talk about a fitness distribution. At a distributed ESS, the distribution of fitness is stable and different individuals have different values of the adaptive character.

The theory is extended to multiple characters and functionally different organisms (predators, prey, etc.). It remains intact with evolution of multiple characters with multiple ecological interactions in space and with mutations and assortative mating. A way to link distributed evolutionarily stable strategies to genetic mechanisms is outlined.

Keywords: assortative mating, distributed ESS, distributed fitness, evolutionary games, mutations, partial differential equations.

INTRODUCTION

One of the major goals of evolutionary game theory is to determine the existence of evolutionarily stable strategies (ESSs). Strategies are identified with organisms’ character values that are subject to natural selection and therefore affect the outcome of the ‘game of evolution’. With this stability (convergent and otherwise), one can establish that invaders (mutants) cannot co-exist indefinitely with populations whose member phenotypes exhibit

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the ESS values (Eshel, 1996; Vincent *et al.*, 1996; Taylor and Day, 1997). A major point of departure in the analysis of evolutionary games is the so-called strategy dynamics. The argument runs as follows. Take a collection of populations, modelled as

$$\begin{aligned}\dot{z} &= zH(z, u) \\ \dot{u} &= f(z, u)\end{aligned}\tag{1}$$

where z is a vector of species, u is a vector of strategy values (we shall call it adaptive character values), H is a matrix of functions that represent instantaneous fitness, and f is a vector of functions that determine how strategies change over time. With appropriate initial conditions, we can examine the trajectories of populations and strategies. Stability analysis, both on z and u , determines the stability of the system. At an ESS, the instantaneous fitness of each individual of a species must be zero and so is f .

This approach relies on some fundamental assumptions. First, with $\dot{z}$, we assume that reproduction is by cloning. Therefore, the potential effect of assortative mating on the outcome of the game of evolution is ignored. Second, we either assume that all organisms are identical or that variability among individuals (with respect to the values of the adaptive character u) can be ignored. Third, $\dot{u}$ represents the dynamics of the average strategy values. The validity of these assumptions is rarely questioned (but for examples, see Law, 1996; Dieckmann and Doebeli, 1999; Y. Cohen, submitted). Furthermore, the values of the adaptive character are often unbounded (but see Vincent and Vincent, 1996).

Now if u represents a population's average of strategy values, then

$$u_i(t) = \frac{1}{z_i(t)} \sum_{j=-\infty}^{\infty} j(\Delta u) z_{ij}(t)$$

where z_{ij} is the population of phenotype j of species i and Δu is a small fixed value. In other words, u do not have dynamics that are independent of z . However, because variation of types within a population is ignored, u cannot be accessed directly (algebraically) and we must rely on (1). Therefore, a direct approach that gives rise to the dynamics of u is needed. This requires articulation of mechanisms that may give rise to such dynamics (mutations, for example). With such articulation, evolutionary game theory and population genetics inch one step closer. This, then, is the aim of this paper. *About notation:* unless otherwise specified, identical symbols may represent a constant or a function; e.g. β or $\beta(\cdot)$, where $\cdot$ are some variables.

POPULATION DYNAMICS WITH MUTATIONS

Let us view a population as a distribution of individuals over trait values. This we shall call the population distribution. The trait is adaptive and is identified with strategy. To develop a theory that does not treat all individuals as being equal, we start from first principles. Let $z(u, t)$ denote the density of the population of types u at time t . That is, $z(u, t)$ is the population distribution at t . Then

$$z(u, t + \Delta t) = z(u, t) + \Delta t (\beta(\cdot) - \mu(\cdot) z(u, t))\tag{2}$$

where Δt is a short time interval, $\beta(\cdot)$ is a birth function of some variables, and $\mu(\cdot)$ is a death rate function of some variables. Rearranging terms and taking $\Delta t \rightarrow 0$, we have

$$\dot{z}_t(u, t) = \beta(\cdot) - \mu(\cdot) z(u, t)\tag{3}$$

where z_t denotes the partial derivative of z with respect to t . Let us use subscripts to denote partial derivatives. To a first approximation, we assume that $\mu(\cdot)$ is a linear function of $z(u, t)$ and write

$$\mu(\cdot) = \mu z(u, t)$$

where μ is a constant. Now, assume that upon birth a mutation rate, η , changes u by a small amount, Δu . Mutations by amounts greater than Δu are negligible. Then, for a fixed birth rate β :

$$\beta(\cdot) = (1 - \eta) \beta z(u, t) + \frac{1}{2} \beta \eta (z(u + \Delta u, t) + z(u - \Delta u, t))$$

In words, the birth function equals a rate constant times the density of individuals with character value u . This birth rate must be adjusted by the mutation rate to individuals with character values other than u . We add the birth of mutants from individuals $u + \Delta u$ and individuals $u - \Delta u$. These mutants contribute to $z(u, t)$. Now Taylor series expansion gives

$$\begin{aligned} z(u + \Delta u, t) - z(u, t) &= (\Delta u) z_u(u, t) + \\ &\quad \frac{1}{2} (\Delta u)^2 z_{uu}(u, t) + o(z(u', t)), \\ u \leq u' \leq u + \Delta u, \end{aligned} \quad (4)$$

$$\begin{aligned} z(u - \Delta u, t) - z(u, t) &= (-\Delta u) z_u(u, t) + \\ &\quad \frac{1}{2} (-\Delta u)^2 z_{uu}(u, t) + o(z(u'', t)), \\ u - \Delta u \leq u'' \leq u \end{aligned}$$

where $o(\cdot)$ denotes negligible amounts. Adding the last two equations, substituting into (3), rearranging and ignoring small values, we obtain

$$z_t = k_1 z_{uu} + (k_2 - \mu z) z \quad (5)$$

where

$$k_1 = \beta \eta (\Delta u)^2 \quad k_2 = \beta \quad z = z(u, t)$$

Equation (5) belongs to a large class of partial differential equations called reaction diffusion equations (Smoller, 1982). Conway (1984a,b), Britton (1986) and Murray (1989) provided detailed exposition of the subject from an ecological perspective. The edited volume by Dieckmann *et al.* (2000) is a good recent reference on the subject (in particular Chapter 22). See also the recent article by Alonso *et al.* (2002). Equation (5) describes the dynamics of the population distribution with respect to time and values of the adaptive character. It includes a mechanism for genetically motivated evolution with respect to the adaptive character u . To completely specify the dynamics, we need initial and boundary conditions. Let $u_{\min} < u < u_{\max}$. Then, we use the Neumann boundary conditions with initial conditions

$$z(u, 0) = a(u) \quad z_u(u_{\min}, t) = 0 \quad z_u(u_{\max}, t) = 0 \quad (6)$$

The boundary conditions ensure that the dynamics remain within the range $u_{\min}$ to $u_{\max}$. Equations (5) and (6) completely specify the system dynamics.

The equilibrium solution of (5) and (6) is achieved when $z_t = 0$. When this equilibrium is stable, we have an ESS distribution. We call this the distributed ESS. This is an ESS simply because mutants represent perturbation to the distributed ESS. Because it is a distributed ESS, mutants disappear by dying or by their progeny merging into the distributed ESS. Thus, no mutant can co-exist with the distributed ESS. This corresponds to the original definition of ESS as given by Maynard-Smith (1982). Because we use distributions, strategy and populations are inseparable. We therefore do not need the concept of convergent stability (Abrams *et al.*, 1993; Eshel, 1996; Taylor and Day, 1997; Cohen *et al.*, 2000) – a stable distribution is a distributed ESS.

The equilibrium solution of (5) and (6) dictates that the fitness distribution at the ESS is zero. The individual fitness distribution is simply the right-hand side of (5) divided by $z(u, t)$. It thus makes no sense to talk about individual fitness alone. The fitness of an individual with character value u is just a point on the distribution. This is so because, to the usual definition of individual instantaneous fitness ($= k_2 - \mu z$), we must add the relation of the individual to its neighbours (via $k_2 z_{uu}/z$).

Skipping technical details (see Appendix 1), we assert that the model (5) with (6) is stable [this was proved in an entirely different context by Sattinger (1972) for a different version of the model]. Furthermore, the distribution of z at equilibrium, $\bar{z}(u)$, is homogeneous with

$$\bar{z}(u) = \frac{\beta}{\mu} \quad (7)$$

Example 1. We take (5) with the initial and Neumann boundary conditions:

$$z(u, 0) = 10 + 5 \cos(4u) \quad z_u(0, t) = z_u(\pi, t) = 0$$

These boundary conditions model mutations on u within the range $[0, \pi]$. With parameter values

$$\beta = 0.2 \quad \eta = 0.001 \quad \Delta u = 0.001 \quad \mu = 0.01$$

we get Fig. 1. As it should, the value of $z(u, 50) = 19.98$ to within 5 decimal digits.

From an evolutionary standpoint, the initial distribution starts with a non-homogeneous distribution of individuals over character values, u . It ends with a homogeneous equilibrium distribution. That is, at the distributed ESS all individuals are equally distributed across all values of the adaptive character. This example assumes reproduction by ‘almost’ cloning (almost because of mutations). I will address this issue in a moment.

From an individual’s perspective, the environment is rarely constant. Let us address this issue next. To keep things simple, suppose that certain values of u lead to higher mortality than others. Equation (5) then becomes

$$z_t(u, t) = k_1 z_{uu}(u, t) + (k_2 - \mu(u) z(u, t)) z(u, t)$$

with the initial and boundary conditions (6). The dynamics are stable with the equilibrium distribution bounded (see Appendix 1):

$$\frac{\beta}{\max_u \mu(u)} \leq \bar{z}(u) \leq \frac{\beta}{\min_u \mu(u)} \quad (8)$$

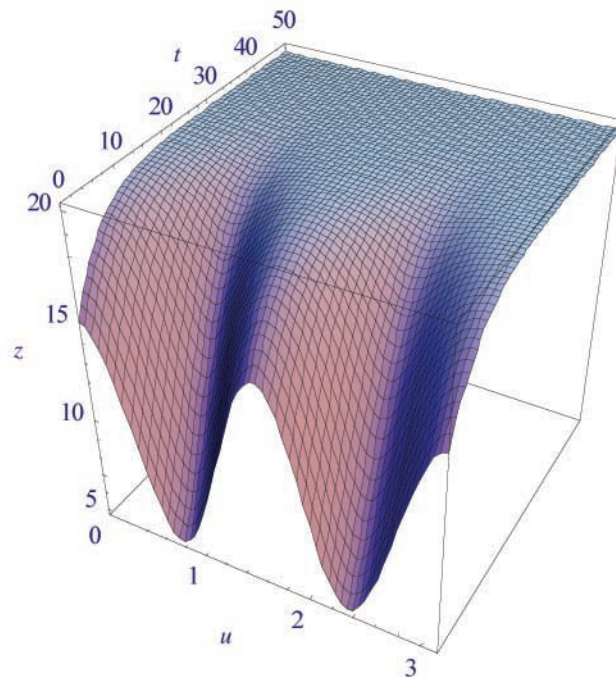


Fig. 1.

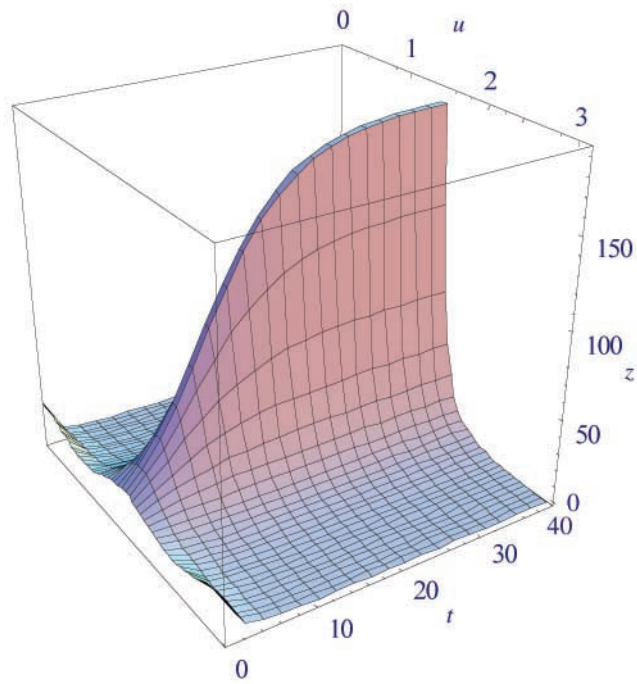


Fig. 2.

In other words, we assert that the distributed ESS is bounded within a range. The specific shape of the distribution depends on the mortality as a function of the adaptive character, $\mu(u)$. This raises the possibility that the distributed ESS is not homogeneous.

Example 2. Suppose that there is a natural mortality rate of 0.001. Furthermore, at $u = \pi/2$ mortality is minimal. This can be modelled with

$$\mu(u) = 0.001 + 0.1(1 - e^{-0.5(u - \pi/2)^2})$$

for example (parameters values are not important – they were chosen to produce pretty pictures). Then, with the same parameter values as in Example 1 (except for μ), the trajectory of the distribution of the population over the selective character results in a non-homogeneous distributed ESS (Fig. 2). The slices of $z(u, t)$, at $t = 0, 50$ and 100 , are shown in Fig. 3. The slices at 50 and 100 are indistinguishable.

How would a naturalist perceive the results in Fig. 3? Suppose that u represents tree heights. Then he would conclude that there are many trees of medium height. Tall and short trees are rare, but trees between the extremes and the medium height are even rarer. From an evolutionary standpoint, this is the distributed ESS – that is, the stable distribution of individuals over the selective character values. The introduction of mutants perturbs the distributed ESS. Such mutants would, of course, disappear with time. The usual instantaneous individual fitness is now $k_2 - \mu(u)z(u, t)$. It is not equal among all individuals at the distributed ESS, $k_2 - \mu(u)\bar{z}(u)$, because now fitness at equilibrium is a function of u . When the equilibrium distribution is homogeneous with respect to u (as in Fig. 1), the fitness of all individuals is equal, and the distributed case collapses to the point process case (as in, for example, Brown and Vincent, 1987; Hines, 1987; Abrams *et al.*, 1993; Vincent *et al.*, 1993, 1996; Rand *et al.*, 1994; Taylor and Day, 1997). When adjusted for the second-order relations (with respect to u) of individuals to their neighbours ($k_1 z_{uu}$), the distributed fitness is zero. The relations are second-order because individuals of type u interact with both individuals of type $u - \Delta u$ and individuals of type $u + \Delta u$.

In the standard approach to evolutionary games, the single peak in Fig. 2 is interpreted as a single species (Cohen *et al.*, 2000, 2001) – provided that the distribution is symmetric (for examples, see Abrams *et al.*, 1993; Vincent *et al.*, 1993). Here, we produce a distribution and

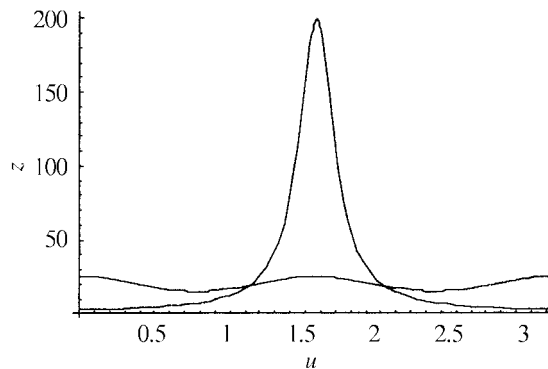


Fig. 3.

it is left to a specific case to interpret the boundaries and classification of individuals along the trait u . In one case, the peak $\pm$ some distance from it may be interpreted as a species. In another as a subspecies. The emphasis here is on the dynamics of the distribution (e.g. as in Fig. 2) that may lead to a distributed ESS. These dynamics encapsulate changes in both populations and strategies and emphasize the fact that they are inseparable.

MUTATIONS AND COMPETITION

The derivation of (5) shows that as long as μ is a function of u and $z(u, t)$, and not $u \pm \Delta u$, the dynamics of the population remain unchanged with respect to z_{uu} . Suppose that competition among types affects survival, but neither birth nor mutation rates. Let the function $\alpha(u, \zeta)$ model the competition between individuals of type u and individuals of type ζ . Then (5) becomes

$$z_t(u, t) = k_1 z_{uu}(u, t) + [k_2 - (\mu + \gamma(u, t)) z(u, t)] z(u, t) \quad (9)$$

where

$$k_1 = \beta \eta (\Delta u)^2 \quad k_2 = \beta \quad \gamma(u, t) = \int_{u_{\min}}^{u_{\max}} \alpha(u, \zeta) z(\zeta, t) d\zeta \quad (10)$$

The competition function α emphasizes neutrality with respect to classification of individuals into types. Competition is viewed as a process among all individuals, not between species where the competition within a species is constant. Thus, like z , the competition function is a distribution. Without competition $\gamma = 0$ and (9) reduces to (5). The initial and boundary conditions (6) remain unchanged.

Mutations with competition allow us to investigate a fundamental issue. In Example 2, we saw the emergence of a non-homogeneous distributed ESS due to environmental forcing. If one so desires, this leads to identification of types with respect to u . Now the question is: Can internal processes, such as competition, produce a non-homogeneous distributed ESS? If yes, then we know that internal population mechanisms can lead to a sympatric increase in the number of types. Recall that types are defined as special points (peaks of high and low density) in the distribution of individuals over the adaptive character. The next example demonstrates that non-homogeneous distribution of competing types is possible.

Example 3. Assume that competition with individuals of your own type is strongest. The strength of competition decays as individuals of type u encounter individuals of type ζ when the difference between u and ζ increases. This can be modelled with

$$\alpha(u, \zeta) = k_3 e^{-k_4(u - \zeta)^2} \quad (11)$$

Here k_3 is a scaling parameter. k_4 scales how fast the competition with others decreases with the increase in the difference between types. We also assume that competition is density dependent.

As in the previous examples, we take $u \in (0, \pi)$ (the notation $\in$ means belongs to). To avoid mathematical discourse, assume that (9) with the boundary conditions (6) has a stable equilibrium. Thus, to determine the shape of the distributed ESS, we need to solve (see Appendix 2):

$$k_1 \bar{z}_{uu}(u) + [k_2 - (\mu + \gamma(u)) \bar{z}(u)] \bar{z}(u) = 0$$

where γ is given in (A1). The boundary conditions are

$$\bar{z}(0) = \bar{z}(\pi) = 20 \quad \bar{z}_u(0) = \bar{z}_u(\pi) = 0$$

We choose the following parameters (again, parameter values should not be taken too seriously; they are used to demonstrate qualitative results):

$$k_1 = 0.9 \quad k_2 = 10 \quad \mu = 0.01 \quad k_3 = 0.01 \quad k_4 = 0.05 \quad c = 1.55$$

(see equation A1 for explanation of c). This produces a non-homogeneous distributed ESS (Fig. 4).

The example demonstrates that if mutation with competition, as modelled by (9), results in a stable equilibrium, then it is possible that the distributed ESS is non-homogeneous. For example, in Fig. 4 we may identify (arbitrarily) three types, at both maxima and at the minimum. Depending on the nature of the adaptive character u and its relation to the genetic mechanisms that produce it, such types may be classified as needed. The example also demonstrates that the non-homogeneous distribution of types is produced by competition – an internal population mechanism. We know that the surface in Fig. 4 is an equilibrium solution of (9). We also assume that it is stable. Thus, it is a distributed ESS for (9).

Example 3 models an evolutionary game. In fact, (11) adjusted for the densities of $z(u, t)$ and $z(\zeta, t)$ says, heuristically: ‘If you are different in character from others, your mortality risk goes down, but beware, everybody wants to be different!’ This is the reason for the dip in the middle of the distributed ESS. In the evolutionary game, players are not required to be rational. They are not even required to be aware that they are playing the game. Thus, a rational player (say a human) who is aware that he is playing the game should choose a strategy that is off the centre (in this case) on either side. The problem is that collectively, as humans, we are not fully aware (or simply deny) that we are part of the evolutionary game.

MUTATION, COMPETITION AND ASSORTATIVE MATING

So far, the theory has dealt with reproduction (almost) by cloning. The ‘almost’ stands for mutations. Time to move on. Here we discuss the case where individuals with character value u mate with other individuals of nearby character values. The case where there are different assortative mating systems (with types mating with distant types as one possibility) was analysed in Cohen (submitted). Starting from first principles (see Appendix 3), we end with

$$z_t(u, t) = k_1 z_{uu}(u, t) - [k_2 - (\mu + \gamma(u, t)) z(u, t)] z(u, t)$$

where

$$k_1 = \frac{1}{6} \beta (\Delta u)^2 \quad k_2 = \beta \left(1 - \frac{1}{3} \eta\right) \quad \gamma(u, t) = \int_{u_{\min}}^{u_{\max}} \alpha(u, \zeta) z(\zeta, t) d\zeta \quad (12)$$

The initial and boundary conditions (6) remain unchanged. Qualitatively, this is identical to the model with mutation and competition (see equation 9). The parameters k_1 and k_2 are different. In the case here, the effect of the magnitude of mutation is diminished compared with the model with mutation and competition only – compare (10) with (12). This is so

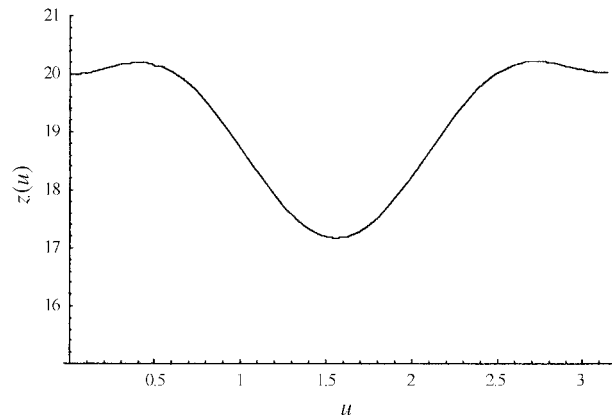


Fig. 4.

because some of the progeny that are bound to have character values of $u - \Delta u$ or $u + \Delta u$ end up having u for their character value because of mutations. Were we to assume that the rate of mutation is a function of the character values u , the model would have been different from the model with mutation and competition only.

Thus, all the comments about the qualitative behaviour of the system with mutation and competition remain intact for the case here. Note that the model uses specific minimalist assumptions about assortative mating. Namely:

1. Mating occurs among individuals that are almost alike.
2. Because the functions we use are smooth, we assume that $z(u \pm \Delta u, t) \approx z(u, t)$.
3. The proportions of u that mate with u and with $u \pm \Delta u$ are equal.

Other, more elaborate assortative mating mechanisms, are discussed in Cohen (submitted).

DISCUSSION AND EXTENSIONS TO THE THEORY

The distributional approach to the theory of evolution raises some legitimate questions. For example, one might argue that a population is composed of discrete units (individuals) and thus there are no individuals of type $u + \Delta u$ when Δu is small enough. Like any other model of diffusion, the continuous approach is based on small-scale averaging. This is justified only for large populations. The approach taken here does not apply to small populations, when individual-based models are appropriate (as in DeAngelis and Gross, 1992, for example).

Even with this justification, one might ask: Why not discrete models? Why all the mathematical fanfare? The answer is simply that discrete models do not lend themselves to generalizations. One invariably ends up with specific examples and resorts to simulations. The attempt here is to draw general conclusions. For example, it will be next to impossible to draw 'parametric' conclusions about the shape of the stable equilibrium distribution (the distributed ESS); for example, as in equations (7) and (8) and Figs 3 and 4.

In the next few paragraphs, I outline extensions of the theory to multiple characters and to a collection of functionally different individuals such as predators and prey. Let u denote a collection of n characters (u is now a vector). Then (5) is written as

$$z_t = k_1 \Delta z + (k_2 - \mu z) z \quad (13)$$

where now

$$\Delta z \triangleq \sum_{i=1}^n \frac{\partial^2 z}{\partial u_i^2}$$

(the notation $\triangleq$ means equal by definition) and we are dealing with a multivariate distribution of individuals over n characters. The comparison theorem for non-linear partial differential equations (see Appendix 1) still applies and the theory can be uplifted to a higher dimension. Of course, we require k_1 to be an n -vector of birth and mutation parameters.

With competition and mutations, (9) becomes

$$z_t = k_1 \Delta z + [k_2 - (\mu + \gamma) z] z$$

Now

$$\gamma(u, t) = \int_{u \in U} \alpha(u, \zeta) z(\zeta, t) d\zeta$$

where U is an n -dimensional hypercube that encloses the minimum and maximum for each of the n adaptive characters. The integration is done n times. Other than changing the values of k_i , including assortative mating requires no extra effort.

Suppose that we have a collection of organisms, with m different ecological functions: prey, predators, parasites, primary producers, and so on. For each, we identify a vector, u_i , each composed of n_i elements that represent important selective characters. Then z is a vector of m elements. Now (5) is written as equation (13) with

$$\Delta z \triangleq \sum_{j=1}^m \sum_{i=1}^{n_j} \frac{\partial^2 z}{\partial u_{ij}^2} \quad (14)$$

with the appropriate proliferation of the parameters k_i . In addition, α becomes a vector of m functions, α_i , each representing the different interactions. To analyse the shape of the distributed ESS distributions, for example, we end up with m distributions, each with n_i dimensions. Special points in these distributions identify types. These points cannot be visualized for $n_i > 2$. However, special points can be identified (e.g. where the derivatives of the distributions are zero and the second derivatives are positive or negative). Incorporating directional selection poses no new conceptual problems. The notation for Δz in (14) needs to be modified to include other orders of derivatives and new parameters will surface. The solutions, however, may behave quite differently. For example, with first derivatives in the reaction diffusion equations, we may be faced with phenomena such as shock waves (see, for example, Smoller, 1982; Murray, 1989).

There is no distinction between u as selective characters and u as spatial coordinates. After all, the location of an individual in space is one of its characters. In other words, the theory remains intact with evolution of multiple characters for multiple ecological interactions in space and with mutations and assortative mating.

How does the theory relate to genetic mechanisms? Via details that link values of u to genetics. For example, suppose that the mutation rate η is a function of u . We may imagine a function $g(u)$ that maps character values to a location of a DNA sequence. Then, instead of η , we may write $\eta(g(u))$. In other words, the mutation rate depends on the correspondence between the value of the adaptive character and the location of the sequence in the DNA. As discussed in Cohen (submitted), $g(u)$ need not be a smooth function. It may even be represented by a set of specific locations on the DNA with the Kronecker's delta function (Y. Cohen, unpublished).

How does the theory relate to point process, continuous evolutionary games? None of the results here contradict results from point processes (e.g. Brown and Vincent, 1987; Abrams *et al.*, 1993; Vincent *et al.*, 1993; Rand *et al.*, 1994; Law, 1996; Dieckmann and Doebeli, 1999). The theory is simply a generalization of the point process, with the explicit inclusion of evolutionary mechanisms such as mutations. For example, a trajectory $z(t, u)$ for each value of u in Fig. 1 settles to a uniform surface $\bar{z}$, which depends neither on t nor on u . It turns out that this is exactly the ESS solution for the point process with the G -function approach (as in Vincent *et al.*, 1996, for example) – adjusted for the mutation rate of course (I am indebted to Joel Brown for pointing this out).

REFERENCES

- Abrams, P., Matsuda, H. and Harada, Y. 1993. Evolutionary unstable fitness maxima and stable fitness minima of continuous traits. *Evol. Ecol.*, **7**: 465–487.
- Alonso, D., Bartumeus, F. and Catalan, J. 2002. Mutual interference between predators can give rise to Turing spatial patterns. *Ecology*, **83**: 28–34.
- Britton, N.F. 1986. *Reaction-Diffusion Equations and Their Applications to Biology*. New York: Academic Press.
- Brown, J.S. and Vincent, T.L. 1987. A theory for the evolutionary game. *Theor. Pop. Biol.*, **31**: 140–166.
- 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. 1984a. *Diffusion and Predator–Prey Interaction: Pattern in Closed Systems*, pp. 85–133. Boston, MA: Pitman.
- Conway, E.D. 1984b. *Partial Differential Equations and Dynamical Systems*. Berlin: Springer-Verlag.
- DeAngelis, D.L. and Gross, L.J. 1992. *Individual-Based Models and Approaches in Ecology: Populations, Communities and Ecosystems*. New York: Chapman & Hall.
- Dieckmann, U. and Doebeli, M. 1999. On the origin of species by sympatric speciation. *Nature*, **400**: 354–357.
- Dieckmann, U., Law, R. and Metz, J., eds. 2000. *The Geometry of Ecological Interactions: Simplifying Spatial Complexity*. Cambridge: Cambridge University Press.
- Eshel, I. 1996. On the changing concept of population stability as a reflection of a changing problematics in the quantitative theory of evolution. *Math. Biol.*, **34**: 485–510.
- Hines, W.G.S. 1987. Evolutionary stable strategies: a review of basic theory. *Theor. Pop. Biol.*, **31**: 195–272.
- Law, U.D.R. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. *Math. Biol.*, **34**: 579–612.
- Maynard-Smith, J. 1982. *Evolution and the Theory of Games*. Cambridge: Cambridge University Press.

- Murray, J.D. 1989. *Mathematical Biology*. New York: Springer-Verlag.
- Rand, D.A., Wilson, H.B. and McGlade, J.M. 1994. Dynamics and evolution: evolutionary stable attractors, invasion exponents and phenotype dynamics. *Phil. Trans. R. Soc. Lond. B*, **343**: 261–283.
- Sattinger, D. 1972. Monotone methods in nonlinear elliptic and parabolic equations. *Indiana Univ. Math. J.*, **21**: 979–1000.
- Smoller, J. 1982. *Shock Waves and Reaction-Diffusion Equations*. New York: Springer-Verlag.
- Taylor, P. and Day, T. 1997. Evolutionary stability under the replicator and the gradient dynamics. *Theor. Pop. Biol.*, **11**: 579–590.
- Vincent, T. and Vincent, T.L. 1996. Using the ESS maximum principle to explore root–shoot allocation, competition and coexistence. *Theor. Biol.*, **180**: 111–120.
- Vincent, T.L., Cohen, Y. and Brown, J.S. 1993. Evolution via strategy dynamics. *Theor. Pop. Biol.*, **44**: 149–176.
- Vincent, T.L., Van, M.V. and Goh, G.S. 1996. Ecological stability, evolutionary stability, and the ESS Maximum Principle. *Evol. Ecol.*, **10**: 567–591.

APPENDIX 1: A PROOF OF (7)

To prove (7), we invoke the comparison theorem for non-linear partial differential equations (Smoller, 1982: 94–95). We take the ordinary differential equations

$$\frac{dv}{dt} = [(1 - \eta) \beta - \mu v] v, \quad v(0) = M$$

$$\frac{dw}{dt} = [(1 - \eta) \beta - \mu w] w, \quad w(0) = m$$

where $m \leq z_0(u) \leq M$. Now z , v and w satisfy (5) and (6) and

$$w(t) \leq z(u, t) \leq v(t)$$

homogeneously in $u \in (0, \pi)$. Also, dv/dt tends to $(1 - \eta) \beta - \mu v$ and dw/dt tends to $(1 - \eta) \beta - \mu w$ as $t \rightarrow \infty$. Therefore, $z(u, t) \rightarrow (1 - \eta) \beta - \mu w$ as $t \rightarrow \infty$. Thus, all solutions of (5) and (6) having bounded initial values tend to $z(u, t) \rightarrow (1 - \eta) \beta - \mu z$ homogeneously in u as $t \rightarrow \infty$. Furthermore,

$$z = \frac{\beta}{\mu}$$

as required.

APPENDIX 2: EQUILIBRIUM DISTRIBUTION WITH MUTATION AND COMPETITION

We seek the equilibrium solution for

$$z_t(u, t) = k_1 z_{uu}(u, t) + \left(k_2 - \left(\mu + \int_{u_{\min}}^{u_{\max}} \alpha(\mu, \zeta) z(\zeta, t) d\zeta \right) z(u, t) \right) z(u, t)$$

with

$$z(u, 0) = z_0(u) \quad z_u(u_{\min}, t) = 0 \quad z_u(u_{\max}, t) = 0$$

At equilibrium, if it exists,

$$k_1 \bar{z}_{uu}(u) + [k_2 - (\mu + \gamma(u)) \bar{z}(u)] \bar{z}(u) = 0$$

where

$$\gamma(u) = \int_{u_{\min}}^{u_{\max}} \alpha(u, \zeta) \bar{z}(\zeta) d\zeta$$

From the mean value theorem, we know that there exists a c such that

$$\gamma(u) = (u_{\max} - u_{\min}) \alpha(u, c) \bar{z}(c) \quad u_{\min} \leq c \leq u_{\max} \quad (\text{A1})$$

where c is a constant. The initial and terminal conditions are

$$\bar{z}(u_{\min}) = a \quad \bar{z}(u_{\max}) = b \quad \bar{z}_u(u_{\min}) = \bar{z}_u(u_{\max}) = 0$$

This is a two point boundary value problem. Example 3 demonstrates a numerical solution.

APPENDIX 3: COMPETITION, MUTATION AND ASSORTATIVE MATING

We start with (3), namely

$$z_t(u, t) = \beta(\cdot) - \mu(\cdot) z(u, t)$$

with

$$\mu(u, t) = \left(\mu + \int_{u_{\min}}^{u_{\max}} \alpha(u, \zeta) z(\zeta, t) d\zeta \right) z(u, t)$$

Assume that assortative mating works as follows. One-third of types u mate with types $u - \Delta u$, one-third with themselves and one-third with types $u + \Delta u$. Each of these reproduce with a rate coefficient of $\beta/2$. Of each of these, $1 - \eta$ do not mutate and η mutate (we use equal fractions to avoid proliferation of parameters). Therefore,

$$\begin{aligned} \beta(\cdot) = & \frac{1}{6} \beta (1 - \eta) (z(u, t) + z(u - \Delta u, t)) + \frac{1}{6} \eta (z(u, t) + z(u - \Delta u, t)) + \\ & \frac{1}{6} \beta (1 - \eta) (z(u, t) + z(u + \Delta u, t)) + \frac{1}{6} \eta (z(u, t) + z(u - \Delta u, t)) + \\ & \frac{1}{6} \beta (1 - \eta) (z(u, t) + z(u + \Delta u, t)) \end{aligned}$$

Note that the coefficients of β sum to 1, as they should. Expanding and collecting terms, we get

$$\beta(\cdot) = \frac{1}{6} \beta [2z(u, t) + z(u - \Delta u, t) + z(u + \Delta u, t)] + \frac{1}{3} \beta (1 - \eta) z(u, t)$$

Add and subtract $2z(u, t)$ to the term in square brackets, take the Taylor series expansion and drop small terms (see 4) to get

$$\beta(\cdot) = \beta \left(1 - \frac{1}{3} \eta \right) z(u, t) + \frac{1}{6} \beta (\Delta u)^2 z_{uu}(u, t)$$

Thus, the desired result

$$z_t = \frac{1}{6} \beta (\Delta u)^2 z_{uu}(u, t) - \left[\beta \left(1 - \frac{1}{3} \eta \right) - \left(\mu + \int_{u_{\min}}^{u_{\max}} \alpha(u, \zeta) z(\zeta, t) d\zeta \right) \right] z(u, t)$$