

Evolutionary distributions and competition by way of reaction-diffusion and by way of convolution

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May 1, 2013

Abstract

Evolution by natural selection is the most ubiquitous and well understood process of evolution. We say *distribution* instead of *the distribution of the density of populations of phenotypes across the values of their adaptive traits*. A phenotype refers to an organism that exhibits a set of values of adaptive traits. An adaptive trait is a trait that a phenotype exhibits and that is subject to natural selection. Natural selection is a process by which populations of different phenotypes decline at different rates. An evolutionary distribution (ED) encapsulates the dynamics of evolution by natural selection. The main results are: (i) ED are derived by way of PDE of reaction-diffusion type and by way of integro-differential equations. The latter capture mutations through convolution of a kernel with the rate of growth of a population. The kernel controls the size and rate of mutations. (ii) The numerical solution of a logistic-like ED driven by competition corresponds to a bounded traveling wave solution of population models based on the logistic. (iii) Competition leads to increase in diversity of phenotypes on a single ED. Diversity refers to change in the number of maxima (minima) within the bounds of values of adaptive traits. (iv) The principle of competitive exclusion in the context of evolution depends, smoothly, on the size and rate of mutations. (v) We identify the sensitivity—with respect to survival—of phenotypes to changes in values of adaptive traits to be an important parameter: increase in the value of this parameter results in decrease in evolutionary-based diversity. (vi) Stable ED corresponds to Evolutionary Stable Strategy; the latter refers to the outcome of a game of evolution.

1 Introduction

[Darwin and Wallace \(1858\)](#) articulated the idea of evolution by natural selection (evolution for short) as we know it. For a biological system to evolve, it must obey the following rules: Inheritance of values of some traits of phenotypes with small variation and natural selection operating on this variation. Natural selection then results in change of the frequency (and potentially density) of subpopulations of phenotypes that exhibit some values of adaptive traits.

The said variation in values of the adaptive traits is around the trait values of progenitors. It is expressed in progeny. We call this variation mutation.

Of all the disciplines in the life sciences, in addition to *how*, evolution is perhaps the only discipline that can answer the question of *why*. For instance, some mathematical models that mimic dynamics

of populations illustrate the emergence of patterns; most prominently from models that capture interactions among prey and their predators (for example Nunes et al., 1999; Tokita, 2004; Ji and Li, 2006). Another example is the differentiation of cells in embryos (Murray, 2003). These patterns emerge as intrinsic properties of such models. Because of their similarity to patterns that are observed in nature, links are made between patterns from models and in nature. All such models do not answer the quintessential question: Why do we observe such patterns in the first place? Answers to such a question may unravel through inquiries into the dynamics of *evolution*.

There exist other (than natural selection) modes of evolution: (i) Neutral Theory and genetic drift (Crow and Kimura, 1970; Kimura, 1983) and (ii) evolution through acquired characters. The characters are presumably acquired during the life-time of an individual and then inherited. The mode of evolution through acquired characters was first articulated by Lamarck (Jablonka and Lamb, 1995) and have been accepted by a small minority biologists. The richness of genomic variation, its implications and the growing body of knowledge about epigenetics (Gissis and Jablonka, 2011; Shapiro, 2011) have contributed to renewed interest in the so-called Lamarckian evolution.

Soft-selection is a variant of evolution by natural selection. It refers to the process by which densities of phenotypes are affected by natural selection, but not by its magnitude (Slatkin, 1978; Lenormand, 2002; Saccheri and Hanski, 2006). As we shall see, when the selection function in ED is constant, we obtain soft-selection. With generalized functions and weak solutions (see for example Smoller, 1982; Renardy and Rogers, 1993; Cantrell and Cosner, 2004), one may implement abrupt changes in soft selection with a step function.

There are also claims of causes of evolution other than those discussed above. As far as we know, one cannot refute these claims with the scientific method. Such claims are therefore outside the purview of science.

In taxonomy, a collection of phenotypes may be classified as belonging to a single species, or occasionally to a subspecies. There exist a number of definitions of species (Futuyma, 2005). In spite of its quintessential role in biology, there is no universal definition of species. We are not concerned with such definitions. We are concerned with the distribution of populations of phenotypes across values of some traits—such as the length of proboscis of some insects.

We discuss evolution with respect to subpopulation densities of phenotypes, the values of their adaptive traits and the effect of natural selection on such densities. A phenotype is an organisms that displays values of some adaptive traits. A trait is adaptive if it is subject to natural selection. Natural selection effects changes in the frequency, and potentially density, of subpopulations of phenotypes. Apart from small random variation, all members of a population of phenotypes are affected by natural selection similarly. This must be so because natural selection acts *directly* upon phenotypes; not upon genotypes. It follows that the unit of selection is a subpopulation of phenotypes. A phenotype can be any object that is a member of a population that obeys the rules of evolution by natural selection: viruses, genes, DNA, cell organelles, cells and so on.

Examples of adaptive traits are speed of running of prey or a predator, the concentration of oxygen that a red blood cells carries, a color pattern of skin that enhances camouflage. The fact that sickle cell anemia is inherited through genes is well established. However, a person dies not because of the genetic mechanisms that brings it about. He dies because there is lack of supply of oxygen to vital organs.

The roots of the the theory developed here can be found in Kimura (1965) and in Crow and Kimura (1970), where the dynamics of genes in populations was emphasized (the connection to ED is discussed further below). These were developed in the milieu of continuous approximation of small populations and discrete mutations to large populations and continuous mutations. The theory was

extended to include frequency-dependent selection (Nagylaki, 1979; Slatkin, 1979; Bulmer, 1980). These extensions were discussed further in Champagnat et al. (2006).

Bürger (2005), extended the theory to include quantitative traits, frequency-dependent selection and population demography.

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 (e.g., see Barton and Keightley, 2002).

Some relevant articles related directly to ED are as follows. Cohen (2003) introduced the concept of ED. Cohen (2009) discussed ED in general and its applicability to a variety of systems. Cohen (2011a) considered ED with time delays. And Cohen (2011b) established conditions under which the dynamics of an evolving system of producer-consumer display patterns whose emergence is an internal property of the system. These patterns reflect regular changes in the density of populations of phenotypes along values of adaptive traits.

To fix ideas, we adopt (for the most part) the following rules of notation: Small roman letters denote functions; albeit functions themselves, we denote state variables with upper roman letters. Parameters are denoted by Greek letters. Parameters and state variables are nonnegative real numbers. Occasionally we may write *a density* instead of *density of a population, evolution* instead of *evolution by natural selection* and *selection* instead of *natural selection*. We denote by x the set of values that an adaptive trait may take—for example, $\underline{x} \leq x \leq \bar{x}$. Sometimes x denotes a specific value that an adaptive trait might take—for example $x \in (\underline{x}, \bar{x})$. The distinction between the two meanings of x should be clear by the context in which x appears.

In Section . . .

2 Derivation of ED

A mathematical model that captures the dynamics of a population according to evolution by natural selection must identify, explicitly, terms that account for: (i) growth of a population; (ii) inheritance with variation; and (iii) decline of a population and selection.

Albeit not entirely, the definitions we use correspond to those that are accepted widely.

We say that growth of a population occurs at the instant when a member of a population is exposed to natural selection. A member of a population is one which might contribute progeny at some time in the future, to the growth of a population. A contribution may be indirect—as is the case with worker bees. Decline is identified with the instantaneous event at which a progeny is no longer considered a member of a population. Membership is forfeited when an individual can no longer contribute to the growth a population (to which it belongs). Immigration and emigration are identified with growth and decline. Decline may occur because of random events or selection. Decline due to random events is such that, on the average, it does not lead to changes in the frequency of subpopulations of phenotypes in a single ED. If such changes occur, they are, by definition, caused by natural selection—whether its agents have been identified or not.

It follows that growth and decline are independent and therefore additive. Because inheritance and mutations are associated with growth, they are independent of natural selection. Thus we delineate

items (i), (ii) and (iii) above.

Here is an example. In a population of organisms that reproduce sexually, a fertilized egg becomes a member of the population. If, for instance, the fertilized egg fails to become an embryo—or if it results in an organism that will not be able to contribute to the growth of the population to which it belongs—then it is not a member of the population (a mule belongs to the population of neither horses nor donkeys whereas his progenitors do). The failure to become an embryo occurs because of natural selection. Ostensibly, our vernacular is moral-less.

Being of biological and physical nature, values of adaptive traits must be bounded. A phenotype on the boundary vanishes from the population. For example, lest proteins freeze or denature,¹ core body temperature of most organisms must be bounded approximately between 0°C and 42°C. Therefore, one must consider absorbing boundaries (the so-called Dirichlet boundary conditions).

We use two approaches to derive the dynamics of ED. In Section 2.1, we use Taylor’s Theorem to obtain the dynamics of ED that are encapsulated by a reaction-diffusion equation. In Section 2.2 we obtain an integro-differential equation whose solution is an ED. We also show that these approaches are interchangeable.

We expound systems with a single adaptive trait only. Extension to multiple adaptive traits is laborious (Cohen, 2011a,b) and brings forth unexpected conclusions (Y. C. manuscript). Also, we examine adaptive traits whose values are real (\mathbb{R}).

2.1 Reaction-diffusion

Our development of an R-D system of equations that capture evolution follows the approach taken by Crow and Kimura (1964) and Kimura (1965). Champagn et al. 2006 put Crow and Kimura’s derivation of the R-D equations (with mutations) on solid foundations. They started with individual based model, where the dynamics of a small populations can be followed in detail. They then formally stated the conditions under which continuous models may be used to approximate discrete individuals models.

In the sequel, we concentrate on linear rate of growth and smooth functions of both decline and selection. With respect to evolution by natural selection, Darwin (1859) recognized the principal importance of resources that limit growth of populations. In his view (which was influenced by Verhulst, 1844), when small (compared to availability of resources), populations should grow exponentially. In fact, in the most ubiquitous model of the dynamics of populations, the logistic, growth is exponential: when small, one might consider U^2 in $U' = \alpha U - \beta U^2$ to be approximately zero. It follows that growth is exponential; namely $U' \approx \alpha U$.

Denote by $U(t)$ the density of a population at time t with rates of growth and decline encapsulated by

$$U'(t) = \alpha U(t) - d(U(t)). \tag{1}$$

Let $[\underline{x}, \bar{x}] \subset \mathbb{R}$ be the set of values of some adaptive traits. On the scale of $x \in [\underline{x}, \bar{x}]$, the coefficient of mutations is small and related to some $\varepsilon_0 < 1$ which determines the mutation rate. Following Kimura (1965) and with respect to phenotypes, we suppose that the following events occur in a

¹as in fried eggs

small neighborhood of x :

$$\begin{aligned} \text{no mutations} &= \alpha(1 - \varepsilon_0^2)U(x, t), \\ \text{mutations from } U(x + \Delta x, t) &= \frac{1}{2}\alpha\varepsilon_0^2U(x + \Delta x, t), \\ \text{mutations from } U(x - \Delta x, t) &= \frac{1}{2}\alpha\varepsilon_0^2U(x - \Delta x, t). \end{aligned} \quad (2)$$

Using Taylor's Theorem, we deduce the following expression that approximates growth rate that is linear

$$\alpha \left(U(x, t) + \frac{\varepsilon^2}{2} \partial_{xx} U(x, t) \right)$$

where $\varepsilon^2 = \varepsilon_0^2(\Delta x)^2$. It follows that the dynamics of the distribution of phenotypes across x , that corresponds to (1), is

$$\partial_t U(x, t) = \alpha \left[U(x, t) + \frac{\varepsilon^2}{2} \partial_{xx} U(x, t) \right] - s(U(x, t), x)U(x, t), \quad (3)$$

where s embodies natural selection. In general, s is a density dependent function that captures the effect of selection on the dynamics of U . The diffusion term in (3) accounts for inheritance and variation of values of adaptive traits among subpopulations of phenotypes. Therefore, (3) epitomizes the dynamics of evolution by natural selection.

We address independent adaptive traits only (but see (14) in Section 4). Therefore, the boundaries of a set of adaptive trait define an n -dimensional box. We define ED thus:

Definition 1 (Evolutionary Distribution) *Let $\mathcal{X} \subset \mathbb{R}^n$ be an open set and denote by $\partial\mathcal{X}$ its closure. Also, let $\bar{\mathcal{X}} = \mathcal{X} \cup \partial\mathcal{X}$ and define $\mathbf{x} = [x_1, \dots, x_n]$ to be the vector of values of n adaptive traits. Then we say that the solution of*

$$\begin{aligned} \partial_t U(\mathbf{x}, t) = \alpha \left[U(\mathbf{x}, t) + \frac{\varepsilon_i^2}{2} \sum_{i=1}^n \partial_{x_i x_i} U(\mathbf{x}, t) \right] - \\ s(U(\mathbf{x}, t), \mathbf{x})U(\mathbf{x}, t), \end{aligned} \quad (4)$$

with data

$$\begin{aligned} U(\mathbf{x}, 0) = g(\mathbf{x}), \quad \mathbf{x} \in \mathcal{X}, \\ U(\underline{\mathbf{x}}, t) = U(\bar{\mathbf{x}}, t) = 0, \quad \mathbf{x} \in \partial\mathcal{X} \end{aligned} \quad (5)$$

is an *Evolutionary Distribution*.

Cohen (2011a) gives a definition of a system of ED. Such a system has a peculiar structure: the diffusion that arises from mutations occurs in different coordinates for each ED.

2.2 Generalization to non local dynamics

In Section 2.1 we assume that the density of the rate of growth of $U(x, t)$ at x is affected by mutations produced at $x \pm \Delta x$ only (see equation 2). Here we assume that mutations occur continuously on the whole interval of adaptive trait values. Thus the dynamics of the ED is expressed by an integro-differential equation (see equation 7 below).

Let $\zeta \geq 0$ be some smooth function defined on \mathbb{R} such that $\int_{\mathbb{R}} \zeta(x) dx = 1$ and consider the *mutation kernel* $\zeta_\varepsilon(x) = \frac{1}{\varepsilon} \zeta\left(\frac{x}{\varepsilon}\right)$ for $\varepsilon > 0$. Then the term of the rate of growth of the dynamics of ED is given by

$$(U(\cdot, t) * \zeta_\varepsilon)(x) := \int_{\mathbb{R}} U(z, t) \zeta_\varepsilon(x - z) dz.$$

Here, the value of the parameter ε is such that the value of ζ_ε is negligibly small for z outside an ε -neighborhood of x . A frequent choice for ζ is the Gaussian with mean zero and variance ε^2 ; ergo

$$\zeta_\varepsilon(z) = \frac{1}{\varepsilon\sqrt{2\pi}} \exp\left(-\frac{1}{2}\left(\frac{z}{\varepsilon}\right)^2\right). \quad (6)$$

Being a variance, ε^2 encapsulates the size of mutations centered at z . Thus we obtain from (1) that

$$\partial_t U(x, t) = \alpha \int_{\mathbb{R}} U(z, t) \zeta_\varepsilon(x - z, t) dz - s(U, x) U(x, t), \quad (7)$$

for $x \in \mathbb{R}$ and $t > 0$. Because $\zeta_\varepsilon \rightarrow \delta$ as $\varepsilon \rightarrow 0$, where δ is the Dirac Delta, the transition from the ED to its corresponding ODE stated in (1) is smooth.

Recall that values of adaptive traits must be bounded. However, with convolution, they are not. One can achieve boundaries by incorporating a penalizing term in (7). We apply this idea in Section 3.

Next, we show that both approaches to the dynamics of ED (through reaction-diffusion and through integro-differential equations) give similar results for (i) small intervals of time with (ii) Gaussian kernel (6) and for (iii) unbounded values of adaptive traits.

Set $\varepsilon := \sqrt{2K\tau}$ for $K > 0$ and $\tau \geq 0$. Then $w(x, \tau) = \int_{\mathbb{R}} V(z) \zeta_\varepsilon(x - z) dz$ is the solution of the heat equation

$$\begin{aligned} \partial_\tau w(x, \tau) &= K \partial_{xx} w(x, \tau), \\ w(x, 0) &= V(x). \end{aligned}$$

Because $\tau \rightarrow 0$ implies $\varepsilon \rightarrow 0$, we have

$$\lim_{\tau \rightarrow 0} \partial_{xx} w(x, \tau) = \lim_{\varepsilon \rightarrow 0} \int_{\mathbb{R}} \partial_{xx} V(x - z) \zeta_\varepsilon(z) dz = \partial_{xx} V(x).$$

For τ small, we deduce the first order approximation for the convolution

$$w(x, \tau) \approx w(x, 0) + \tau \partial_\tau w(x, 0) = V(x) + K\tau \partial_{xx} V(x).$$

Noting that $K\tau = \varepsilon^2/2$ and replacing $V(x)$ by $U(x, t)$ above, we conclude from (7) that

$$\begin{aligned} \partial_t U(x, t) &= \alpha \int_{\mathbb{R}} U(z, t) \zeta_\varepsilon(x - z) dz - s(U, x) U(x, t) \\ &\approx \alpha \left[U(x, t) + \frac{\varepsilon^2}{2} \partial_{xx} U(x, t) \right] - s(U, x) U(x, t), \end{aligned} \quad (8)$$

which approximates (3). This approximation holds for convolution with a Gaussian kernel—it is not necessarily valid for convolution with other kernels.

Extension of (8) from a single adaptive trait x to n adaptive traits \mathbf{x} is straightforward.

As a canonical example, consider (8) with the following parameter values:

$$\begin{aligned} \underline{x} = 1, \quad \bar{x} = 3, \quad \alpha = 1, \quad s(U) = 0.01U, \\ U(x, 0) = \zeta_{0.001} \left(x - \left(\frac{\underline{x} + \bar{x} - x}{2} \right) \right). \end{aligned} \quad (9)$$

To achieve the desired boundary conditions, in the numerical integration we pad \underline{x} to the left and \bar{x} to the right with 10 grid points. Thus we obtain Figure 1 which illustrates the usual traveling wave

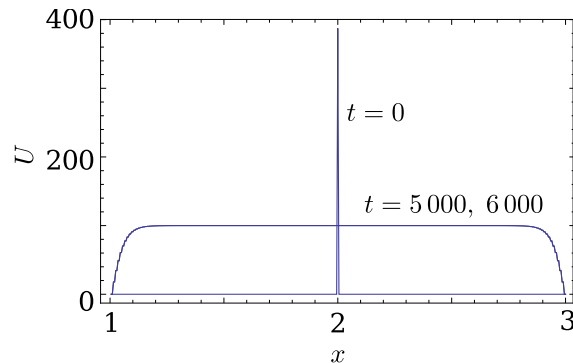


Figure 1: Numerical solution of (8) with parameter values given in (9).

solution (Murray, 2003) with Dirichlet boundaries added.

The convolution (8) corresponds to the Fisher equation (Fisher, 1937) where the motivation for the term s in (8) is different from ours. In fact, the Fisher's equation is derived from the logistic with a constant coefficient of the diffusion term; namely $U_t = \rho U (1 - U/\kappa) + \mu U_{xx}$ where μ is constant. It refers to the spread of an advantageous gene in a population. Also, the Fisher's equation is unbounded.

There are numerous generalizations of the Fisher equation. For example Gourley et al. (2001) studied a scalar reaction-diffusion equation which contains a nonlocal term in the form of an integral convolution in the spatial variable. None of these formulations consider a mutation term. They typically modify the diffusion term of the logistic.

Note that upon reaching \underline{x} and \bar{x} , the moving front (that is the density of U on the boundary) should decline vertically. This is not the case here because ζ is smooth (Figure 1). We shall address this matter soon.

3 Numerical experiments: competition-driven diversity

For a while, ecologists held to the notion that speciation cannot occur in a single (sympatric) population. Speciation, it was claimed, can occur only in allopatric situations, where there is some barrier among subpopulations of a species. This view, championed primarily by Mayr (1963) faded away and by the 1990s the notion that both types of speciation occur was taking hold; see Dieckmann and Doebeli (1999) and Via (2001) and review by Bolnick and Fitzpatrick (2007). Here we show that natural selection, imposed by competition—a common interaction among entities in nature—leads to apparent isolation of the density of population of some phenotypes across their distribution on values of an adaptive trait.

In the following, we assume that $U(x, t)$ obtains equilibrium, $\widehat{U}(x)$. In the numeric expositions, we presume that equilibrium has been reached when the relative error between two consecutive iterations is

$$\max_{x \in U} |U(x, t_{k+1}) - U(x, t_k)| < \text{tol}$$

where $\text{tol} = 1.e - 10$.

We address the topic of like-competes-most-with-like with the strength of competition decaying uniformly as phenotypes become disparate on the their values of the adaptive trait. Competition is likely to influence the profile of ED. Hence, we examine its consequences: first, with respect a *kernel*—a modified version of (6)—of competition (Section 3.1); second, for reaction-diffusion type ED (Section 3.2); third, for ED as derived from convolutions (Section 3.3); and fourth, competition within and among two populations (Section 3.4).

3.1 Like competes most with like

We assume that the strength of competition between phenotypes x and z decays uniformly *and symmetrically* as $|x - z|$ (for $\underline{x} \leq x, y \leq \bar{x}$) increases. The assumption that the kernel that encapsulates competition is symmetric is important. Without it, the validity of our argument in this section collapses.

Suppose that s in (8) is given by $s(U(x), x) = \beta \zeta_\sigma(x) U$. The share of natural selection assumed by competition between x and z for all $z \in [\underline{x}, \bar{x}]$ is then

$$c(x) = \int_{\underline{x}}^{\bar{x}} \zeta_\sigma(x) \zeta_\sigma(x - z) dz = \frac{1}{2\sigma} \left(\text{erf} \left[\frac{\bar{x} - 2x}{2\sigma} \right] - \text{erf} \left[\frac{\underline{x} - 2x}{2\sigma} \right] \right) \quad (10)$$

where the so-called error function is defined thus:

$$\text{erf}(y) = \frac{2}{\sqrt{\pi}} \int_0^y e^{-\tau^2} d\tau.$$

Figure 2 illustrates the distribution of the share of selection in mortality due to competition between x and $0 \leq z \leq 4$ according to (10). The figure illustrates the sensitivity of this distribution with respect to the location of x and magnitude of σ . Ostensibly, phenotypes on the boundaries do not compete with phenotypes (that do not exist) outside the boundaries. It follows that the mortality due to competition exhibits the pattern illustrated in the figure; it decreases for phenotypes on the boundaries of the values of adaptive traits.

The parameter σ reflects the rate of the decay of like-competes-most-with-like: As σ increases, the rate of this decay decreases. Hence, the distinction between phenotypes on the boundaries and in the interior blurs. This phenomena is reminiscent to the term *edge effect* as it is used in ecology to refer to edges of habitats—say the transition from forest to grassland.

If the sign of c is switched, one obtains a model of *cooperation* with attributes that mirror those owing to competition.

The results in this section do not consider the dynamics of selection that accompany the dynamics of ED. We address this matter next.

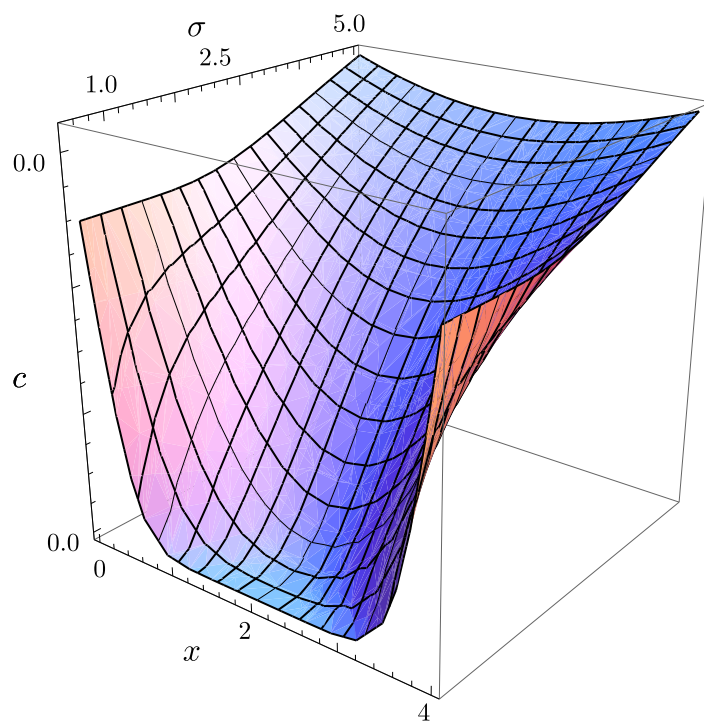


Figure 2: Distribution of selection owing to competition between x and $\underline{x} \leq y \leq \bar{x}$ according to (10) with $\underline{x} = 0$, $\bar{x} = 4$ and $0.5 \leq \sigma \leq 5$.

3.2 Dynamics of evolution with competition

In being an agent of natural selection, competition effects decline of U only: It does not affect mutations and growth explicitly. In general, competition may occur among individuals of all phenotypes. Because the unit of selection is a subpopulation of a single phenotype, competition also occurs among individuals of a single phenotype. Albeit an oxymoron, one might call it self competition. Hence we choose

$$s(U(x)) = \int_{\underline{x}}^{\bar{x}} \mu(x, z)U(z, t)dz, \quad (11)$$

where $\mu : [\underline{x}, \bar{x}] \times [\underline{x}, \bar{x}] \rightarrow \mathbb{R}_+$ is a smooth function. For the following numerical experiments, we assume that like competes most with like and that as the distance between phenotypes x and z increases, competition decreases uniformly. These considerations permit the kernel $\mu(x, z) = \zeta_\sigma(x - z)$, where ζ is defined in (6).

Implementing (4) and (5) for a single adaptive trait and with an atom-type initial condition at the center the interval $[\underline{x}, \bar{x}]$, we write:

$$\begin{aligned} \partial_t U(x, t) = & \alpha \left[U(x, t) + \frac{1}{2} \varepsilon^2 \partial_{xx} U(x, t) \right] - \\ & (\beta + 1) U(x, t) \int_{\underline{x}}^{\bar{x}} \zeta_\sigma(x - z) U(z, t) dz \end{aligned} \quad (12)$$

where $x \in (\underline{x}, \bar{x})$ and $t > 0$. For data we specify

$$\begin{aligned} U(x, 0) = & \zeta_{0.001} \left(x - \frac{\bar{x} + \underline{x}}{2} \right), \quad \text{and} \\ U(\underline{x}, t) = & U(\bar{x}, t) = 0. \end{aligned}$$

In the pursuit of general conclusions about the role that parameters play in shaping ED, we carry numerical experiments. With

$$\begin{aligned} \alpha &= 1, \quad \sigma = 1, \quad \beta = 0 \\ \varepsilon^2 &= 0.005, \quad \underline{x} = 0, \quad \bar{x} = \pi \end{aligned}$$

in (12), we generate Figure 3.

As evolution progresses, the distribution of the density of phenotypes changes from uni- to bi-modal. Those phenotypes that are subject to stronger competition than others, *ceteris paribus*, end up with a density smaller than otherwise.

This phenomena comes to view in Figure 3. The parameter σ in (12) influences the rate at which competition declines from like competes most with like (this feature is evident in Figure 2 where no dynamics are used). In the the lingo of statistics, σ^2 denotes variance. As $\sigma \rightarrow \infty$, competition approaches “all compete equally with all”. As $\sigma \rightarrow 0$, like compete with like only.

Denote the stable equilibrium of U by $\hat{U}(x)$. Figure 4 illustrates the effect of σ on the splitting of the ED to different types of phenotypes. We define type as a set of phenotypes that are—loosely speaking—distinguishable from nearby phenotypes by concentration of density around a local maximum or a local minimum. In taxonomy, such sets may be defined as subspecies. For example for $\sigma = 0.5$ in Figure 4 we identify five types. Because adaptive traits are inherited, such types should correspond to five *unique* genotypes. It is well known that many genes are pleiotropic

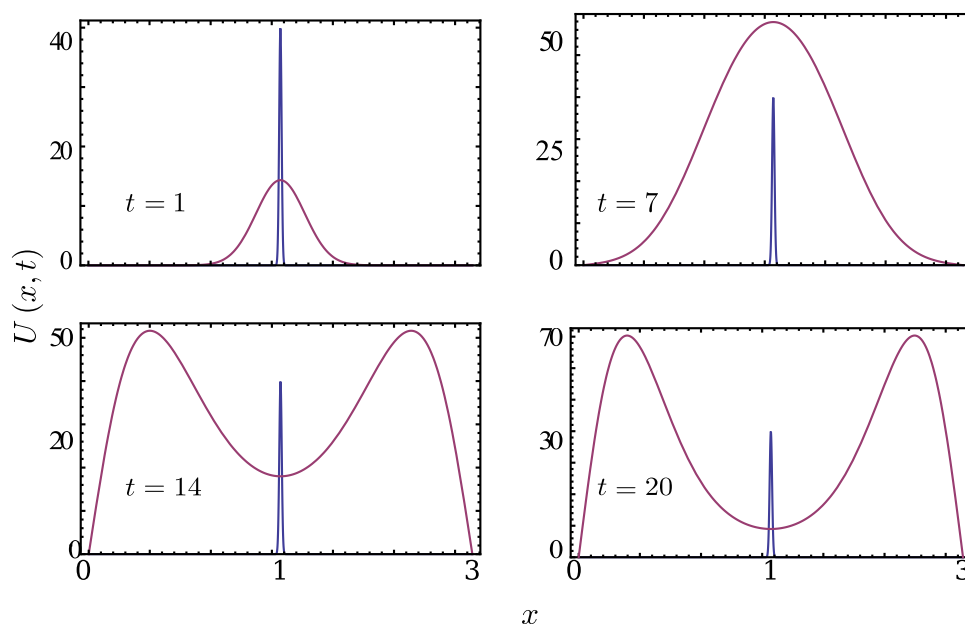


Figure 3: Dynamics of evolution with competition.

(Lenski, 1988; Foster et al., 2004). Yet, we claim unique genotypes. We shall address this claim in Section 4.

The shape of the profile of ED that emerges with evolution, expounded in (12) differ from that without it (see 10). It reflects competition that is density and phenotype dependent.

Our numerical experiments epitomize the influence of competition of σ on the profile of the ED. In the context of evolution, we interpret σ with the following heuristic argument in mind.

Poisonous chemicals, named tannins, are found in the cell walls of foliage of plants in a wide range concentrations. Suppose that within a single ED there exist subpopulations of phenotypes whose tolerance to such concentrations differ. Those with a *wide-range* of tolerance can consume a wider variety of plants and thus obtain more food—particularly in winter—compared to other phenotypes whose *range* of tolerance is narrower. The important point here is that from the point of view of natural selection (ergo mortality), it is not the mean tolerance that matters, it is its variance. Food is a limiting resource, phenotypes compete for it, and therefore the diversity of diet is an adaptive trait.

Let us contemplate Figure 4 again. Suppose that the adaptive trait, x , represents the variety of plants (with varying concentrations of tannins in their foliage) that a phenotype can consume. The diet reflected in the top left frame of Figure 4 is narrower—with respect to the variance of tannins in the foliage of plants— than that of phenotypes represented in the bottom right. Following our ad-hoc (intuitive) definition of diversity, for some range of σ , a set of phenotypes specialized to a diet narrow in the concentration of tannins results in lower diversity. Lower compared to an ED where the breadth of diet of phenotypes is larger. Thus we arrive at a counter-intuitive conclusions, which we state in ecological terms: *Diversity in ED composed of specialists is lower compared to that of generalists.* Why? Because when σ is large, phenotypes on the boundaries experience less competition compared to when σ is small. In other words, when σ is small, the overlap in the distribution of phenotypes due

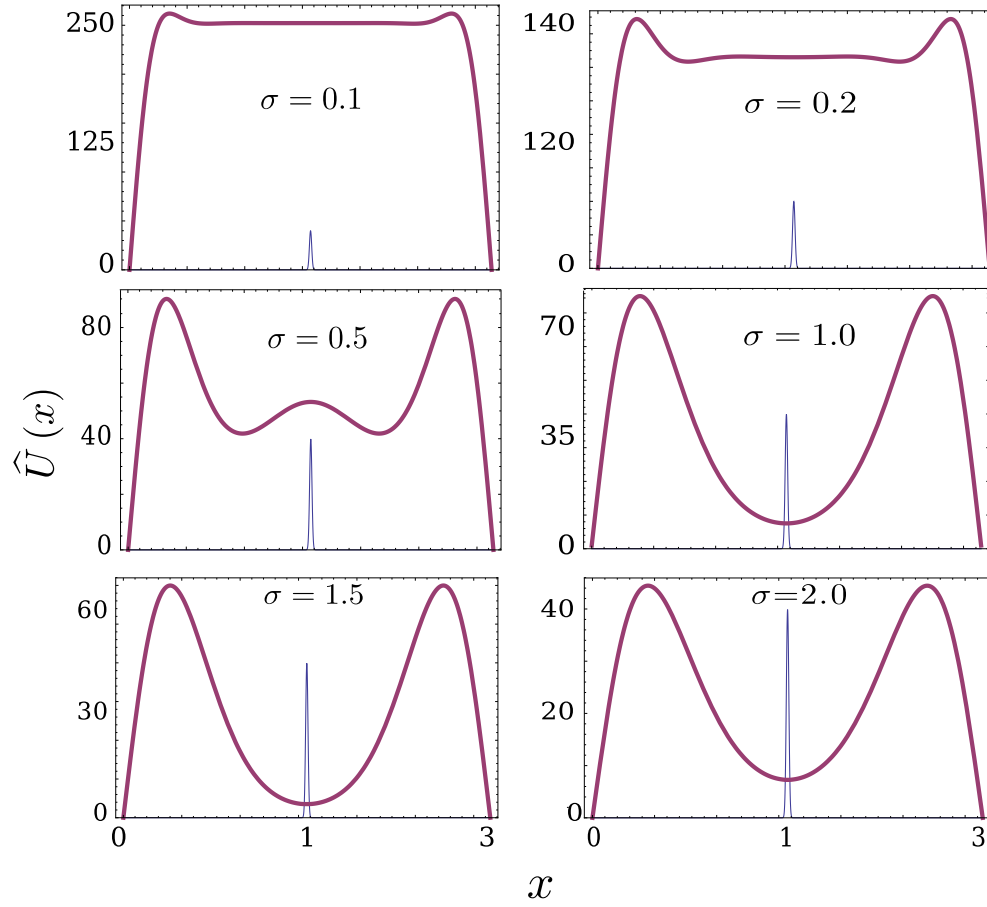


Figure 4: Effect of σ in (12) on splitting the distribution of the density of phenotypes. Narrow thin curves are the initial distribution. Thick curves indicate ED at numerical equilibrium ($t = 40$).

to competition equalizes. Another counter intuitive conclusion follows: The density of phenotypes is split among phenotypes at both extremes of the diversity of diet.

In the case, of say, efficiency of microorganisms in metabolizing glucose, the theory predicts the following: Under competition for resources, populations of two phenotypes end up coexisting—those that are at the boundary of high efficiency those that are at the boundary of low efficiency. Alas this prediction of the theory cannot be confirmed from existing experiments, such as those by Lenski and coworkers (Bennett and Lenski, 1996; Barrick et al., 2009; Crozat et al., 2011) on evolution because the average phenotype from a single chemostat is determined. To reject the augury of the theory, one would have to determine the distribution of phenotypes in a single chemostat (or a single repetition of an experiment)—a difficult task indeed.

Now if we flip the sign of the integral in (12), competition turns into cooperation. In such a case, when, for example, $\sigma = 2$ (Figure 4), the density of phenotypes around the middle value of the adaptive trait becomes the maximum (we venture to propose that the maximum is global) of the distribution.

It follows that for some range of σ , competition enriches diversity while cooperation pauperizes it. Colloquially speaking, one might say that under competition, it is good to be eccentric while under cooperation it is good to be conformist.

In passing, we note the following observation: Distinguishing competition from cooperation in cultures of microorganisms is difficult (Foster and Bell, 2012). Perhaps the profile of ED can help.

3.3 Competition according to convolution type ED

We now return to (7). The Gaussian kernel (6) is smooth. It follows that both the decay in the size of mutations away from the progenitor (whose value on the adaptive trait is x) and in competition with increasing distance from x are smooth. The rate of these decays are controlled by ε ; the smaller it is, the steeper the decay. When the convolution (of competition) reaches the boundaries, the Dirichlet condition requires that $U(x, t)$ declines precipitously to zero. Unless $\varepsilon \rightarrow 0$, this requirement cannot be met with the smooth Gaussian kernel. Hence, in addition to implementing competition (as in 11) to the dynamics of ED (as in 7), we make use of a penalizing function thus:

$$\partial_t U(x, t) = \int_{\underline{x}}^{\bar{x}} U(z, t) (\alpha \zeta_\varepsilon(x - z) - U(x, t) \zeta_\sigma(x - z)) dz - U(x, t) P(x).$$

Here $P : U \rightarrow \mathbb{R}$ is a smooth function such that

$$\lim_{x \rightarrow \underline{x}} P(x) = \lim_{x \rightarrow \bar{x}} P(x) = \infty$$

so that $P(x)$ is small for x far from the boundaries. For instance, in the numerical experiments we used the following penalty:

$$P_\delta(x) = \zeta_\delta(\underline{x} - x) + \zeta_\delta(x - \bar{x}),$$

for $\delta > 0$ small. The consequence of such penalty is to steepen the decline of U at \underline{x} and at \bar{x} .

Would we obtain similar dynamics under competition with, say, equal number of types, with different kernels for competition? Based on the analysis accompanied by Figure 2, the answer is yes (Figure 5). The thin blue lines in the figure show the ED at equilibrium with the Gaussian competition

kernel only. As expected, the profile of the ED (thin blue lines) corresponds the profile shown in Figure 4.

However, with the compactly supported competition kernel

$$\zeta_\varepsilon(x) = \begin{cases} 1/\varepsilon + x/\varepsilon^2 & \text{for } x \in (-\varepsilon, 0), \\ 1/\varepsilon - x/\varepsilon^2 & \text{for } x \in (0, \varepsilon), \\ 0 & \text{elsewhere,} \end{cases} \quad (13)$$

(which satisfies $\int_{\mathbb{R}} \zeta_\varepsilon = 1$) the similarity of the profile of ED at equilibrium collapses (compare thin blue curves to thick red curves in Figure 5). In Figure 5 we show that different kernels (equations 6

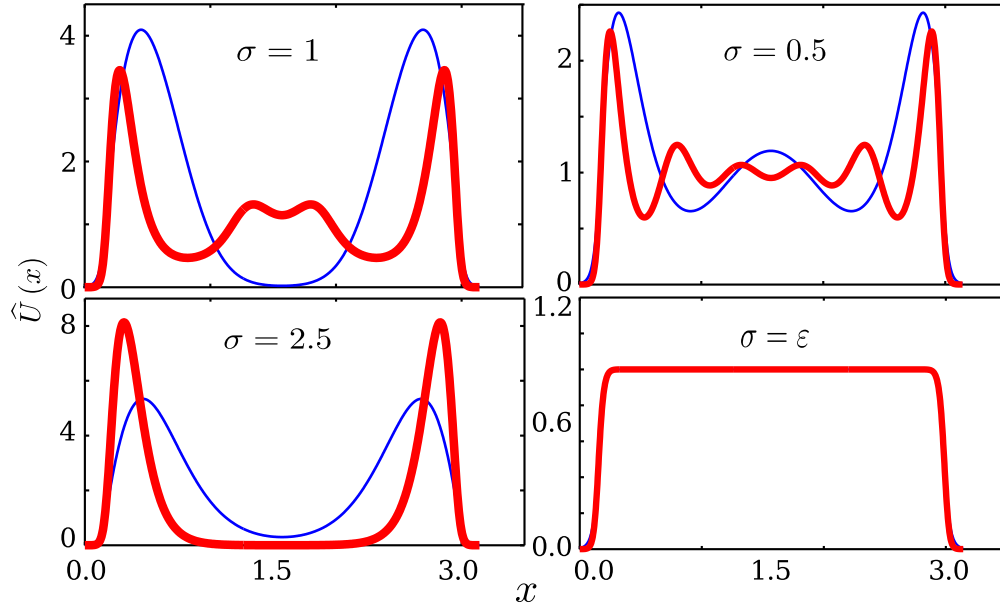


Figure 5: Thin (blue) show ED at equilibrium with Gaussian kernel while thick (red) with the kernel given in (13)

and 13) obtain different profiles of ED at equilibrium. The salient point is this: with a kernel that approximates the Dirichlet boundary condition closer than another kernel, the effect of competition becomes more prominent. Observe the different profiles in the left panel of Figure 5.

In the right panel of Figure 5 one can hardly distinguish the profile according to (6) and according to (13). This is so because the steepness of decline of U at the boundaries converge as $\sigma = \varepsilon \rightarrow 0$.

3.4 Competition within and among two populations

Imagine a system with a single adaptive trait, x , but with two populations that differ in the value of σ while competing in the same trait-space:

$$\begin{aligned} \partial_t U_i(x, t) = & \int_{\underline{x}}^{\bar{x}} U_i(z, t) (\alpha_i \zeta_{\varepsilon_i}(x-z) - U_1(x, t) \zeta_{\sigma_{i,1}}(x-z) - U_2(x, t) \zeta_{\sigma_{i,2}}(x-z)) dz \\ & - U_i(x, t) P(x). \end{aligned}$$

Such a case raises the specter of covariance among σ . This would be the circumstance when like compete most with like but “most” relates to two dimensions on σ . The fact that we use two subpopulations in the same adaptive space does not contradict the definition of ED because values of σ are not considered adaptive.

For values of the parameters, we chose $[\underline{x}, \bar{x}] = [0, 1]$, $\alpha_1 = \alpha_2 = 1$, $\varepsilon_1 = \varepsilon_2 = 0.004$. For the Gaussian kernel we use

$$\sigma = \begin{pmatrix} 0.25 & 0.5 \\ 0.001 & 0.001 \end{pmatrix}. \tag{14}$$

Thus the left panel of Figure 6. With

$$\sigma = \begin{pmatrix} 0.5 & 0.5 \\ 0.001 & 0.001 \end{pmatrix}$$

we obtain the right panel of Figure 6. In addition, we enforce the homogeneous Dirichlet boundary

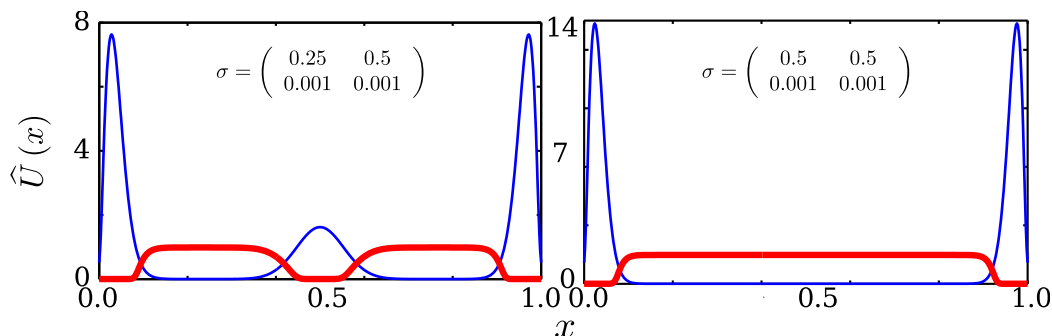


Figure 6: Equilibrium solutions for two populations: thin curves (blue) vis à vis thick curves (red).

data by redefining the kernel close to the boundary which should be equivalent to (12).

Now we add competition and obtain Figure 7 with parameter values as in (9) and with $\sigma = 1$ in

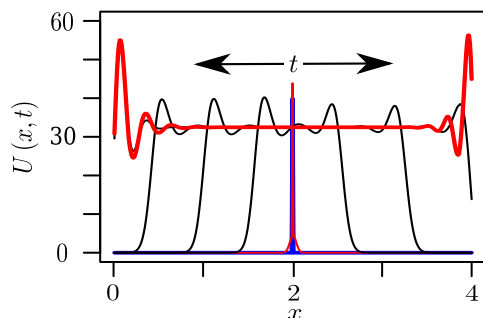


Figure 7: Numbers account for the simulation time for which the boundaries are shown. Note that what seems like decaying periodicities in the center and increasing on the boundaries are in fact different boundaries as they move in time.

(11).

The pattern of the distribution of phenotypes shown in Figure 7 corresponds roughly to the pattern shown in Figure 4 for $\sigma = 0.1$ or 0.2 . The distribution at equilibrium emerges at $t = 200$. This may be interpreted as follows: At the beginning (say up to $t = 30$) density of the distribution of phenotypes decreases from the initial distribution. Then a moving wave can be detected visibly with the leading phenotypes at higher densities than the remaining. This is so because of the smaller amount of competition at the forefronts of the traveling wave. Once the wave reaches its boundaries, the density of the phenotypes at the edges is higher because there is no competition with phenotypes outside the boundaries. There is a distribution of phenotypes with different density at the edges as one moves towards the boundaries. This is so because of the gradual decay in the strength of competition as one moves away from the edges. The upshot is that with $\varepsilon \rightarrow 0$ we end with two phenotypes with high densities at the edges and the remaining phenotypes with even distribution of density.

In the case of ED, a moving wave is not to be interpreted as invasion of phenotype into formerly inhospitable values of adaptive trait. In fact, the range of adaptive traits is set such that mutations allow positive densities where they have been zero. In other words, as long as $x \in [\underline{x}, \bar{x}]$, $U(x, t) = 0$ is part of the ED.

4 Discussion

Adaptive landscapes Early mathematical models of evolution pictured the mutation-variation-selection process from the perspective of the dynamics of hill-climbing on a so-called *adaptive landscape*—the higher the position on the landscape, the higher the fitness. Adaptive landscapes were perceived as a rigid topography upon which the genetic make-up of an evolving population wanders, driven by selection due to factors controlled by environments (Wright, 1969). The next step in the theory was to recognize that the adaptive landscape metaphor misses one-half of the dynamics of evolution: although the environment imposes selection and thereby adaptations, the latter can shape the environment (Haldane, 1932; Pimentel, 1968; Stenseth, 1986; Metz et al., 1992). Ergo, an initial (and rigid) adaptive landscape is undefined; in fact, the fitness of a phenotype depends upon the distribution of the density of phenotypes' subpopulations (Metz et al., 1992; Heino et al., 1998). Kirkpatrick and Rousset (2005) reviewed applications of mathematical models of adaptive landscapes. The shape of the distribution of subpopulations of phenotypes across values of adaptive trait falls under the topic of adaptive landscape. In the case of ED, phenotypes do not wander across the landscape: Progeny are born into subpopulations of phenotypes (by local mutations of values of traits of progenitors). Here, the metaphor is a shape of a surface changing by flipping a blanket with fixed (Dirichlet) boundaries.

Evolutionary games, evolutionary stable strategies and ED Applications of theory of games to assemblages of animals in the context of animal conflict began with Maynard-Smith and Price (1973) and were encapsulated in Maynard-Smith (1982). A central concept in the game-theoretic approach to ecology and evolution is that of evolutionary stable strategies (ESS). Roughly, ESS is defined as a set of values of strategies (phenotypic traits) that assemblages of phenotypes will emerge such that a small mutation (carried by a small population of phenotypes) cannot coexist with such assemblages indefinitely.

The conditions of invasability are achieved through variety of approaches. A widely used one is that of Dieckmann (1997) which then followed by numerous papers; this approach is known as *adaptive dynamics*. Vincent and Brown (2005) approach the continuous dynamics of games of evolution through the so-called *G-function* which is wrapped with ODE.

According to ED, the sets of adaptive traits cover all possible values of adaptive traits. It then follows that (i) locally stable ED cannot be invaded by small populations of phenotypes that mutate from the locally stable ED; in other words, invasion in dynamics games is represented by perturbations of stable ED. (ii) The concept of invasability is not applicable. If ED is globally stable, then the condition that correspond to ESS is global.

Phenotypes as units of selection A phenotype is an organism that displays a specific set of values on the set of adaptive traits. Some variation within a population of a phenotype is expected. We do not deal with such variation, which one might call intra-phenotypic variation. Consequently, one should view our definition of a phenotype in the limit. The magnitude of the intra-phenotypic variation is delimited by the effect of natural selection: within a population of identical phenotypes, each individual has an equal probability of death. Hence, *the unit of selection is a population of a single phenotype*.

We address models of the dynamics of evolution of phenotypes only. Yet, genetics plays a quintessential role in such dynamics. First, genetics is involved in producing a phenotype. Second, if we are to understand evolution and use it pragmatically—for example for “fooling” microorganisms that are pathological and evolve—the underlying influence of genetics on the dynamics of evolution must be understood. In this context, the experiments conducted by Lenski and his collaborators (Lenski, 1988; Bennett and Lenski, 1996; Barrick et al., 2009) are particularly relevant. Their research revolves around linking evolution, phenotypes and genotypes.

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