Synthesis

Theoretical Approaches in Evolutionary Ecology: Environmental Feedback as a Unifying Perspective

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abstract: Evolutionary biology and ecology have a strong theoretical underpinning, and this has fostered a variety of modeling approaches. A major challenge of this theoretical work has been to unravel the tangled feedback loop between ecology and evolution. This has prompted the development of two main classes of models. While quantitative genetics models jointly consider the ecological and evolutionary dynamics of a focal population, a separation of timescales between ecology and evolution is assumed by evolutionary game theory, adaptive dynamics, and inclusive fitness theory. As a result, theoretical evolutionary ecology tends to be divided among different schools of thought, with different toolboxes and motivations. My aim in this synthesis is to highlight the connections between these different approaches and clarify the current state of theory in evolutionary ecology. Central to this approach is to make explicit the dependence on environmental dynamics of the population and evolutionary dynamics, thereby materializing the eco-evolutionary feedback loop. This perspective sheds light on the interplay between environmental feedback and the timescales of ecological and evolutionary processes. I conclude by discussing some potential extensions and challenges to our current theoretical understanding of eco-evolutionary dynamics.

Keywords: Price equation, adaptive dynamics, weak selection, selection gradient, separation of timescales, eco-evolutionary feedback loop.

> Hence, as more individuals are produced than can possibly survive, there must in every case be a struggle for existence, either one individual with another of the same species, or with the individuals of distinct species, or with the physical conditions of life. Darwin (1859, 63)

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Introduction

Evolution is rooted in ecology (Hutchinson 1965; McPeek 2017). Natural selection and genetic drift, the two forces that sort genetic variation, are fundamentally ecological processes that result from the population dynamics of genetically diverse populations. Among the two forces that create variation (mutation and gene flow), gene flow is the resultant, at the population level, of the different dispersal strategies of individuals. Importantly, evolution in turn causes changes in the ecological conditions, leading to a permanent feedback loop between ecological and evolutionary dynamics. Understanding this feedback is a major focus of interest in evolutionary biology, particularly in the field known as evolutionary ecology.

Ecology and evolutionary biology both having strong theoretical foundations, a rich theoretical literature has been devoted to this objective. Over the years, a variety of modeling approaches have been proposed, all sharing the common goal of formulating metrics of evolutionary change that incorporate the feedback of the environment. My aim in this synthesis is to clarify the connections and purposes of these different approaches, using the notion of environmental feedback as a unifying concept.

The notion of environmental feedback is central to the field of adaptive dynamics (AD), where fitness is explicitly defined as a function of environmental variables (Metz et al. 1992; Mylius and Diekmann 1995). In this perspective, the environment collects all the relevant information necessary to calculate the vital rates of individuals, such as the density of different types of conspecifics, the density of resources or predators, or other abiotic factors. As illustrated by the epigraph from *The Origin of Species*, this notion already played a key role in Darwin's largely ecological theory. I will show in this synthesis that this perspective can also be usefully adapted to other theoretical frameworks, such as the Price equation and quantitative genetics (QG).

A central assumption of AD is that evolution unfolds on a much slower timescale than ecological processes. This assumption, which is also at the core of evolutionary game theory and inclusive fitness theory, is justified by limiting arguments on the mutation process (Metz et al. 1992; Geritz et al. 1998; Rousset 2004; Lehmann and Rousset 2014; Van Cleve 2015). This has fostered the development of a rich set of techniques to deal with evolution in potentially complex population dynamics, which has now de facto become the standard for studies of long-term evolution. In the following, I will regroup these models under the AD umbrella.

In general, however, teasing apart ecology and evolution is a challenging task, as reflected by recent evidence of rapid genetic and phenotypic changes in natural populations (Thompson 1998; Hairston et al. 2005; Duffy et al. 2009). To study rapid or short-term evolution, a variety of approaches have been proposed, which I will discuss under the QG umbrella in this synthesis. These models do not necessarily assume that ecological and evolutionary timescales can be decoupled (Roughgarden 1971; Slatkin 1980; Taper and Case 1992; Abrams and Matsuda 1997; Abrams 2001; Day 2005). Although most QG models have considered either simple population dynamics or specific assumptions on the distribution of traits, recent models of parasite evolution have successfully extended this approach to account for the interplay between epidemiological and evolutionary dynamics (Day and Proulx 2004; Day and Gandon 2006; Gandon and Day 2007). The fundamental equation underlying QG models is the Robertson-Price equation (Robertson 1966; Price 1970; Queller 2017), which is encountered in the literature under a variety of disguises depending on additional assumptions on the distribution of traits.

Unfortunately, these different approaches have been developed by largely independent groups of researchers, and the dialogue between theoreticians belonging to different schools is often hampered by the focalization on a particular technique. This is particularly surprising as QG and AD models use similar assumptions (such as weak selection) and share key concepts (such as the selection gradient). Among practitioners of AD, the usefulness of the Price equation for eco-evolutionary questions is debated, while many quantitative geneticists will question the usefulness of AD as a model of evolution. My contention is that this apparent dissonance can be resolved by putting environmental feedbacks at the center of our theoretical models. In particular, a key objective of this synthesis will be to highlight that the timescales of ecological and evolutionary processes affect the feedback of the environment.

Because of constraints on space, I have made some deliberate choices. For instance, I will consider only continuous-time models (although extension to discrete-time models is straightforward). In addition, although evolution is an inherently stochastic process, I will focus on deterministic ecological models described by systems of ordinary differential equations (ODEs) and ignore the traditional population genetics approach of analyzing stochastic processes. However, the two approaches are not disconnected, as the systems of ODEs can be thought of as describing the expected dynamics of a reasonably large population. As a matter of fact, most of the results described in this article can be obtained as suitable large-population limits of an individual-based stochastic process (Champagnat et al. 2001, 2006; Rousset 2004; Méléard 2011; Lehmann and Rousset 2014; Lehmann et al. 2016). Moreover, ODEs represent a versatile tool to model complex ecological scenarios and are an essential component of classical ecological theory.

The article is organized as follows. I start with a general model of population and environmental dynamics. I then discuss how this model can be used to derive a dynamical equation for the change in allele frequencies and the change in a mean trait of interest. This framework allows me to revisit classical results, such as the Price equation and Fisher's fundamental theorem of natural selection, with an explicit coupling with environmental dynamics. The next section shows that, under some limiting assumptions on the mutation regime, one can recover the now classical AD approach. The standard toolbox for analyzing long-term evolution is reviewed briefly, paying particular attention to the connections with the Price equation approach. Finally, I discuss some conceptual implications for our understanding of evolutionary processes and environmental feedbacks and some challenges for future studies.

Ecological Dynamics

Populations and Environments

My starting point will be a population of individuals. In general, the population will be structured in types and states. Types can represent the genotypes or phenotypes of interest. Individuals of a given type may still differ by their demographic or ecological state, but for now I will consider that there is no other source of heterogeneity in the population.

Individuals live in an environment, which is defined from a purely individually centered perspective (Metz et al. 1992; Mylius and Diekmann 1995). The environment collects all the relevant information necessary to compute the reproduction and survival of individuals. This includes any effects external to the focal population, such as the density of a resource, and any direct effects of conspecifics, through the densities of the different types of individuals.

Consider, for instance, a population of N types. The dynamics of each type will depend on an environmental vector, $\mathbf{E}$, that collects the population densities of the types, $\mathbf{n}$,

and a vector of external variables, **e**, such as the density of a resource, the densities of other species (e.g., competitors, predators, parasites), or any relevant biotic or abiotic factor. The environmental vector **E** can then be written as

$$\mathbf{E} = \begin{pmatrix} \mathbf{n} \\ \mathbf{e} \end{pmatrix}. \tag{1}$$

Turning to the epigraph I have chosen for this synthesis, it is clear that this is merely a mathematical reformulation of a key notion of Darwinian evolution: **n** corresponds to the struggle with individuals "of the same species," while **e** regroups the "individuals of distinct species" and the "physical conditions of life." For our purpose, "species" should simply be replaced with "focal population."

Because population densities and external variables change over time, the environmental vector is a function of time. Throughout this synthesis, the vector **E** will materialize the feedback between ecology and evolution.

Population Dynamics

I write the per capita growth rate of type i as a function $r_i(\mathbf{E})$, therefore making explicit the dependence of reproduction and survival on the environment. By definition, the density of type i changes as follows:

$$\frac{\mathrm{d}n_i}{\mathrm{d}t} = r_i(\mathbf{E})n_i. \tag{2}$$

Note that equation (2) is a simple consequence of the definition of a per capita growth rate and does not rely on assumptions about how type i individuals are created (e.g., clonally or through the mating of other types).

Similarly, the dynamics of the external variables can be written as an ordinary differential equation:

$$\frac{\mathrm{d}\mathbf{e}}{\mathrm{d}t} = \mathcal{D}(\mathbf{E}). \tag{3}$$

Let $n = \sum_{i=1}^{N} n_i$ be the total density of the focal population. Then, the frequency of type i in the population is given by

$$f_i = \frac{n_i}{n}. \tag{4}$$

The dynamics of the total density n can then be written as

$$\frac{\mathrm{d}n}{\mathrm{d}t} = \bar{r}(\mathbf{E})n, \tag{5}$$

where $\bar{r}(\mathbf{E}) = \sum_{i=1}^{N} r_i(\mathbf{E})f_i$ is the average growth rate.

Equations (2) and (3) give a complete description of the population's dynamics. If we are interested in purely ecological questions, it is often sufficient to track the dynamics of the total density n (eq. [5]) along with the dynamics of external variables (eq. [3]). Typical ecological models tend to possess one or several ecological attractors, such as fixed points (e.g., equilibria, which are the most common case, at least in the literature), limit cycles, or even chaotic attractors.

Examples

To fix the ideas, I now give two illustrating examples that will be used in the remainder of the article.

Example 1: Lotka-Volterra Competition Model. Consider a population of N types exploiting a resource continuum. Let n_i be the density of type i. Following previous works (see, e.g., Kisdi 1999), we write the dynamics of type i as

$$\frac{\mathrm{d}n_i}{\mathrm{d}t} = \left[\rho(z_i) - \sum_{j=1}^{N} a(z_i - z_j)n_j\right] n_i,$$

where z_i is the trait value of type i; $a(z_i - z_j)$ is the competition kernel between types i and j, which is assumed to be a function of the trait difference; and $\rho(z_i)$ is the intrinsic growth rate of type i. In this model, the environment is totally specified by the vector **n**, and we do not need to track the dynamics of any external variable.

Example 2: Host-Parasite Interactions. Consider now a simple SIR epidemiological model (Kermack and McKendrick 1927; Hethcote 2000) where the host population is infected by N parasite strains. The focal population is the population of infected hosts, and we denote by n_i the density of hosts infected by strain i. The dynamics of infected hosts will depend on the densities of susceptible (S) and recovered (R) hosts, so that **e** may be thought of as the vector $(S \quad R)^{\top}$.[1] We thus have the following dynamics for **e**:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b - dS - \bar{\beta}Sn + \rho R,$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma n - \rho R,$$

where n is the total density of infected hosts, b and d represent background reproduction and mortality, ρ is the rate at which recovered hosts lose immunity, and $\bar{\beta}$ and $\bar{\gamma}$ are the mean transmission and recovery rates. Furthermore, the total density of infected hosts changes as

$$\frac{\mathrm{d}n}{\mathrm{d}t} = [\bar{\beta}S - (d + \bar{\alpha} + \bar{\gamma})]n,$$

where $\bar{\alpha}$ is the mean virulence. The dynamics of type i is

1. Throughout the text, the symbol $\top$ denotes the transpose operation.

given by

$$\frac{\mathrm{d}n_i}{\mathrm{d}t} = [\beta_i S - (d + \alpha_i + \gamma_i)]n_i.$$

In this model, we thus have $r_i(\mathbf{E}) = r_i(S) = \beta_i S - (d + \alpha_i + \gamma_i)$ and $\bar{r}(\mathbf{E}) = \bar{\beta} S - (d + \bar{\alpha} + \bar{\gamma})$.

In the following, I will often refer to example 2, which is a classical epidemiological model. The reason for focusing on host-parasite interactions as a running example is that it works well as a minimal but not too simple ecological model, taking into account realistic features such as interspecific interactions, demographic changes in population densities, resource dynamics (the density of susceptible hosts from the parasite's point of view), and potentially complex dynamics such as the extinction of either species.

Evolutionary Dynamics

Evolutionary change is given by the change in the frequencies of types, which can be tracked either directly or, by proxy, through the change in the average value of a trait in the population. In this section, I show how dynamical equations for the change in frequency or mean trait can be derived in a general ecological setting. This approach has close links with traditional population or quantitative genetics frameworks and relies on a version of the Price equation while keeping the dependence of fitness on the environment as general as possible. It can be seen as a generalization of the framework introduced by Day and Proulx (2004) and Day and Gandon (2006, 2007) to deal with evolution in epidemiological models.

Change in Frequency

A first approach to tracking evolutionary change is to focus on changes in frequency. Because $f_i = n_i/n$, the change in frequency of type i is

$$\begin{aligned}\frac{\mathrm{d}f_i}{\mathrm{d}t} &= \frac{1}{n}\frac{\mathrm{d}n_i}{\mathrm{d}t} - \frac{n_i}{n^2}\frac{\mathrm{d}n}{\mathrm{d}t} \\ &= f_i(r_i(\mathbf{E}) - \bar{r}(\mathbf{E})).\end{aligned} \tag{6}$$

Equation (6) tells us that if the growth rate of type i is larger than the mean growth rate of the population, type i will increase in frequency. Equation (6) is a version of the replicator equation (Taylor and Jonker 1978; Schuster and Sigmund 1983), with explicit environmental dependence. It is also a continuous-time version of a classical result of population genetics linking the change in allele frequency to the allelic marginal fitnesses (Fisher 1930; Rice 2004; Queller 2017).

Because all densities n_i can be written as $f_i n$, the coupled ecological and evolutionary dynamics take the form of the self-contained system

$$\begin{cases} \dfrac{\mathrm{d}\mathbf{e}}{\mathrm{d}t} = \mathcal{D}(\mathbf{E}) \\ \dfrac{\mathrm{d}n}{\mathrm{d}t} = \bar{r}(\mathbf{E})n \\ \dfrac{\mathrm{d}f_i}{\mathrm{d}t} = f_i(r_i(\mathbf{E}) - \bar{r}(\mathbf{E})), \qquad \text{for all types } i, \end{cases} \tag{7}$$

where the environment $\mathbf{E}$ can be written in function of the total density, the vector of frequencies $\mathbf{f}$, and the external variables as $E = (n \quad \mathbf{f} \quad \mathbf{e})^{\top}$. This decomposition of the environmental feedback shows that it includes the classical models of density- and frequency-dependent selection but also the dependence on external environmental variables due to the interaction with other species or abiotic factors.

The dynamical system (7) illustrates the tangled feedback loop between ecology and evolution: the change in the genetic or the phenotypic composition of the population is affected by the environmental variables, but in turn these evolutionary changes lead to modifications of the environment. Unravelling this feedback loop has been a key challenge of recent theoretical developments. Note that at an abstract level, the dependence on the environment of the coupled system (7) is sufficient to capture processes such as niche construction or ecological inheritance (Odling-Smee et al. 2003).

Classical models of population genetics tend to focus on equation (6) instead of the full coupled eco-evolutionary system. The derivation of equation (6) from population dynamical first principles shows that this cannot be expected to hold true unless some specific form of environmental feedback is assumed. A common assumption is that there is no frequency-dependent density dependence (Heino et al. 1998; Rice 2004; Day 2005). For instance, if the environmental feedback is such that $r_i(\mathbf{E}) = m_i - c(n)$, where m_i is a constant Malthusian parameter and $c(n)$ quantifies density dependence, the change in frequency is simply proportional to a constant selection coefficient $s = m_i - \bar{m}$, and ecological dynamics may be safely ignored. However, with only a slight change in the environmental feedback, such as assuming that the total density affects the growth rates multiplicatively, $r_i(\mathbf{E}) = m_i c(n)$, the change in frequency becomes proportional to $sc(n)$ and is therefore dependent on the dynamics of the total density. Density-dependent migration rates are another biological mechanism that may cause the standard ecological assumptions of population genetics models to break down (Holt and Gomulkiewicz 1997).

A simpler expression of the replicator equation can be found if we consider that the population contains only

two types, a wild type (w) and a mutant type (m). Then, the change in frequency of the mutant type, f_m, takes the following simple form:

$$\frac{\mathrm{d}f_m}{\mathrm{d}t} = f_m(1 - f_m)(r_m(\mathbf{E}) - r_w(\mathbf{E})). \tag{8}$$

The frequency of the mutant type increases if its per capita growth rate is higher than the wild type's. Equation (8) shows that the rate of frequency change is scaled by the population variance, $f_m(1 - f_m)$, and thus neatly separates the effect of genetic variance from the effect of selection, measured by the difference in per capita growth rates.

Change in Mean Trait

We can also translate the change in frequency into a quantity that is more easily measurable, such as the mean phenotype in the population. For a given trait z, with value z_i for type i, we track the average trait value, $\bar{z} = \sum_i z_i f_i$. Assuming that the trait values z_i do not vary with time (e.g., there is no seasonal or circadian variation in phenotypic expression), the change in mean trait depends only on the dynamics of frequencies and is given by the following equation:

$$\begin{aligned}\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} &= \sum_i z_i \frac{\mathrm{d}f_i}{\mathrm{d}t} \\ &= \sum_i z_i r_i(\mathbf{E}) f_i - \bar{z}\,\bar{r}(\mathbf{E}) \\ &= \mathrm{cov}(z, r(\mathbf{E})).\end{aligned} \tag{9}$$

Equation (9) shows that directional changes in the mean trait will be observed if there is a nonzero covariance between the trait and the per capita growth rate across all strains. In other words, if strains with a higher value of the trait tend to have a higher per capita growth rate, then the covariance will be positive and natural selection will cause the mean trait to increase.

We can equivalently express this covariance as the product of the population variance and the regression of the per capita growth rate on the trait. We then have by definition

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \sigma_{zz}\beta_{zr}, \tag{10}$$

where σ_{zz} is the population variance in the trait and

$$\beta_{zr} = \frac{\mathrm{cov}(z, r(\mathbf{E}))}{\sigma_{zz}}$$

is the least-square regression coefficient between the trait and the per capita growth rate. Equation (10) was first formalized by Robertson (1966). It gives the effect of natural selection on the change in mean trait and highlights two key ingredients of Darwinian evolution. First, for natural selection to operate, we need some variability in the population ($\sigma_{zz} > 0$). The rate of evolutionary change by natural selection is scaled by the variance. Second, it shows that the direction of selection is determined by a linear relationship between the per capita growth rate, $r_i(\mathbf{E})$, and the trait. This may seem surprising at first, because we lose a fair bit of information by focusing on this regression. However, as long as we are interested in the change in mean trait and not on the change in other moments of the distribution of the trait (such as the variance), this is all we need (for a more complete discussion, see Rice 2004, chap. 6).

As mentioned above, equation (9) holds true only for traits that do not change over time. For many ecologically relevant traits, such as photosynthetic rates, hormonal secretion, or the infectivity of parasites, circadian or seasonal rhythms are likely to cause variations in phenotypic expression over time. Other traits, such as biomass production or total population growth rate, are also necessarily time dependent. When considering time-dependent traits $z_i(t)$, an additional term $\sum_i f_i \mathrm{d}z_i/\mathrm{d}t$ must be added to equation (9). We shall see an example of this term later on in the discussion of Fisher's fundamental theorem.

The Price Equation

Equation (10) is not a complete description of evolutionary change, because it does not take into account changes occurring during reproduction through mutation or recombination. Consider, for instance, that $r_i(\mathbf{E})$ can be written as $b_i(\mathbf{E}) - d_i(\mathbf{E})$, with $b_i(\mathbf{E})$ being the birth rate and $d_i(\mathbf{E})$ being the death rate. Assuming that mutation occurs with probability μ during reproduction and that with probability m_{ji} type i may mutate to type j, the per capita growth rate of type i takes the form $(1/n_i)dn_i/dt = (1 - \mu)b_i(\mathbf{E}) + \mu\sum_j m_{ij}b_j(\mathbf{E}) - d_i(\mathbf{E})$, and using the same approach as above, one may write the change in the trait as

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \mathrm{cov}(z, r(\mathbf{E})) + \mu\sum_i \left(\sum_j m_{ji}z_j - z_i\right) b_i(\mathbf{E}) f_i \tag{11}$$

or more simply as

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \mathrm{cov}(z, r(\mathbf{E})) + \mu\overline{b_i(\mathbf{E})\delta_i}, \tag{12}$$

where $\delta_i = \sum_j m_{ji}z_j - z_i$ is the difference between the trait of an individual of type i and the mean trait calculated over the mutation distribution, $\sum_j m_{ji}z_j$. This is a specific version of the Price (1970, 1972) equation, which generally partitions the change in mean trait into a part due to natural

selection (the covariance term) and a part due to changes occurring during reproduction (the second term). The second term in equation (12) is proportional to the mutation rate and can be rewritten as $\mu(\bar{z}_m - \bar{z}_n)$ in this simple genetical context: here, $\bar{z}_m$ is the mean trait among new offspring in the presence of mutations, $\bar{z}_m = \sum_i b_i(\mathbf{E})\sum_j m_{ji} z_j f_i$, while $\bar{z}_n = \sum_i b_i(\mathbf{E}) z_i f_i$ is the mean trait among offspring in the absence of mutation. Thus, the second term in equation (12) measures the additional directional effect on the mean trait due to mutation bias.

In contrast to most treatments of the Price equation (e.g., Gardner 2008; Frank 2012; Queller 2017), equation (12) explicitly incorporates the dependence on the environment, through the vector **E**. Doing so emphasizes that the Price equation, as a description of evolutionary change, needs to be coupled with a set of dynamical equations describing the dynamics of **n** and **e**. Of course, because the Price equation is precisely derived from these equations, it does not provide additional information. However, it still allows one to translate population dynamics into phenotypic change, which is useful for biological applications. Moreover, with additional assumptions on the trait distribution (e.g., weak selection or Gaussian approximation of the traits) or the dependence of the vital rates on the trait, it is possible to decouple to some extent the dynamics of the mean trait from the environmental dynamics, as we shall see in the next sections. Hence, provided we are explicit on environmental feedbacks and the approximations we make, we can use the Price equation as a meaningful description of evolutionary change, with an explicit coupling with ecological dynamics. For instance, an application of this approach to evolutionary epidemiology has been used to study the short-term dynamics of virulence evolution (Gandon and Day 2007).

In more abstract treatments, the effect of environmental change is often incorporated into the second term of the Price equation, which tends to be treated as negligible compared to the more fundamental covariance term (Queller 2017). In contrast, the approach I follow here represents the change in the environment as a coupled set of dynamical equations. I view this as a useful representation because it forces us to be explicit on the ecological basis of selection and makes no a priori assumption on the relative forces of ecological and evolutionary processes.

Fisher's Fundamental Theorem

A particular trait one may want to look at is the net growth rate, $r_i(\mathbf{E})$. Because this trait is a function of the environment, which depends on time, we need an additional term to account for environmental change. Slightly generalizing Gandon and Day's (2009) approach, this gives the following equation for the dynamics of the mean growth rate, $\bar{r}(\mathbf{E})$:

$$\frac{\mathrm{d}\bar{r}(\mathbf{E})}{\mathrm{d}t} = \underbrace{\mathrm{var}(r(\mathbf{E}))}_{\Delta r_{\mathrm{ns}}} + \underbrace{\mu\overline{b_i(\mathbf{E})\delta_i}}_{\Delta r_{\mathrm{m}}} + \underbrace{\nabla_{\mathbf{E}}\bar{r}(\mathbf{E}) \cdot \frac{\mathrm{d}\mathbf{E}}{\mathrm{d}t}}_{\Delta r_{\mathrm{e}}}. \qquad (13)$$

In the population genetics literature, $\bar{r}(\mathbf{E})$ would be interpreted as mean fitness. Equation (13) thus partitions the change in mean fitness into three components. The first component, Δr_{ns}, is the change in mean fitness due to natural selection, which is simply the variance in the growth rate $r_i(\mathbf{E})$. The second term, Δr_{m}, is the change in mean fitness due to mutation or recombination, which takes the same form as in the Price equation, except that δ_i is now calculated using $r_i(\mathbf{E})$ as the trait z_i. The third term, Δr_{e}, represents the change in mean fitness due to environmental change (Frank and Slatkin 1992; Gandon and Day 2009). This term is the product of $\nabla_{\mathbf{E}}\bar{r}(\mathbf{E})$, the gradient of the mean growth rate with respect to the environmental vector **E** multiplied by the time derivative of the environment.

If Δr_{m} and Δr_{e} are 0, equation (13) collapses to Fisher's (1930) fundamental theorem, which states that the part of the change in mean trait due to natural selection is equal to the variance in fitness. Because a variance cannot be negative, this implies that natural selection always leads to an increase in mean fitness. The emphasis on natural selection is important because otherwise this statement would be wrong. In fact, mean fitness does not necessarily increase, because of mutation (Δr_{m}) or environmental change (Δr_{e}; Frank and Slatkin 1992). Fisher (1930) referred to the Δr_{e} term as the "deterioration of the environment": at evolutionary equilibrium ($d\bar{r}/dt = 0$) and in the absence of mutational bias, the increase in mean fitness due to the selection term ($\Delta r_{\mathrm{ns}} > 0$) is necessarily opposed by a decrease due to the environmental term ($\Delta r_{\mathrm{e}} > 0$). More generally, depending on the magnitude of the various terms, mean fitness can even decrease until the population becomes extinct (Matsuda and Abrams 1994; Webb 2003). The crucial point is that Fisher defined the environment from an individual- or gene-centered perspective, so that environmental change encompasses both changes in external abiotic or biotic factors and changes in population densities or allele frequencies. Thus, Fisher's concept of environment is consistent with the definition used in this synthesis.

Hence, Fisher's fundamental theorem emphasizes a rather restrictive definition of natural selection, which excludes environmental feedbacks. It is important to note that while this definition is very much in line with the tradition in population genetics, evolutionary ecologists generally consider environmental feedbacks as part and parcel of the action of natural selection. The key to understanding this apparent discrepancy is to recognize that theoretical evolutionary

ecology generally relies on an argument of separation of timescales: environmental change is assumed to take place on a fast timescale compared to evolutionary dynamics (Gandon and Day 2009). Separations of timescales are key ingredients in modeling evolutionary change and will be discussed in Long-Term Evolution and Limiting Mutation Regimes.

Change in Trait Variance

In most of the literature, the Price equation formalism is used to describe the change in the mean trait and not the change in other moments of the trait distribution. However, the approach is more general, and equation (12) is best seen as the first of an infinite hierarchy of moment equations giving the dynamics of the variance and of other moments. Various assumptions have been used to close the system of moment equations, the most common one being the Gaussian approximation (Lande 1976, 1982; Lande and Arnold 1983). If we are ready to assume that the distribution of traits is (and remains) Gaussian, only the mean and variance are needed to describe the full distribution. We can then either assume that the variance is constant and treat it as a parameter or explicitly track the dynamics of the variance (Slatkin 1980; Taylor and Day 1997; Day and Proulx 2004; Rice 2004; Débarre and Otto 2016).

In our general model, a Price equation for the dynamics of the variance can be obtained using the definition $\sigma_{zz} = \sum_i z_i^2 f_i - \bar{z}^2$. Dropping the mutation term for simplicity, we obtain

$$\begin{aligned}\frac{\mathrm{d}\sigma_{zz}}{\mathrm{d}t} &= \mathrm{cov}((z-\bar{z})^2, r(\mathbf{E})) \\ &= \mathrm{cov}(z^2, r(\mathbf{E})) - 2\bar{z}\frac{\mathrm{d}\bar{z}}{\mathrm{d}t}.\end{aligned} \tag{14}$$

Equation (14) is valid for any distribution of the trait and shows that even when there is no directional change in the mean trait, the variance can still change due to the covariance between the squared trait and the per capita growth rate. An increase in variance while the mean stays constant could, for instance, arise due to symmetric disruptive selection around the mean. Figure 1 provides an illustration using the Lotka-Volterra model described in example 1. In the presence of mutation, a similar analysis shows that even when there is no mutation bias in the trait (i.e., the second term of eq. [12] is 0), any nonzero mutation bias in the squared trait will affect the dynamics of the variance.

Multivariate Traits: An Example from Evolutionary Epidemiology

My treatment so far focuses on the evolution of single traits, but the joint evolution of several traits brings other challenges to the modeler. Provided the mutation events are rare, we can drop the mutation term from the Price equation, and the change in a vector of traits $\mathbf{z}$ takes the form

$$\frac{\mathrm{d}\mathbf{z}}{\mathrm{d}t} = \mathbf{G}(\mathbf{z}) \cdot \mathbf{S}(\mathbf{z}), \tag{15}$$

where $\mathbf{G}$ is a genetic (co)variance matrix and $\mathbf{S}$ is a vector of selection gradients. The structure of equation (15) illustrates two additional difficulties with evolution in multidimensional trait spaces. First, the change in a focal mean trait will depend on the selection gradient of all other traits, and therefore interactions between the fitness effects of the traits may affect the direction of evolution. Second, these fitness effects are weighted by trait-specific measures of genetic variation that can be correlated due to pleiotropic effects. This should be contrasted with one-dimensional traits, where genetic variance affects only the magnitude of selection, while its direction can be determined using the selection gradient only.

As an example of multivariate evolution, let us look at the joint evolution of transmission and virulence in a parasite population. The coupled ecological and evolutionary dynamics of the host-parasite interaction following the SIR epidemiological model can be written as follows. Assuming that the only variable traits are transmission and virulence, we have for the epidemiological dynamics

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b - dS - \bar{\beta}SI + \rho R, \tag{16a}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = [\bar{\beta}S - (d + \bar{\alpha} + \gamma)]I, \tag{16b}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I - \rho R. \tag{16c}$$

Furthermore, we can expand the various covariance terms $\mathrm{cov}(z, r(\mathbf{E}))$ in function of second moments of the distribution of traits. This gives us the following equation for the change in mean traits (Day and Gandon 2006):

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix}\bar{\alpha}\\ \bar{\beta}\end{pmatrix} = \mathbf{G} \cdot \begin{pmatrix}-1\\ S\end{pmatrix} + \text{mutation bias}. \tag{17}$$

Equation (17) partitions the change in the vector of mean traits into two components. The first component is the product between the genetic covariance matrix $\mathbf{G}$ and the selection gradient $(-1 \quad S)^\top$. The genetic covariance matrix has the variances $\sigma_{\alpha\alpha}$ and $\sigma_{\beta\beta}$ on its diagonal and the covariances $\sigma_{\alpha\beta}$ and $\sigma_{\beta\alpha}$ on its antidiagonal. The second term represents the effect of mutation bias, which depends on the mutation model. Equation (17) shows that potential equilibria are determined by the balance between selection and mutation. Note the striking similarity between equa-

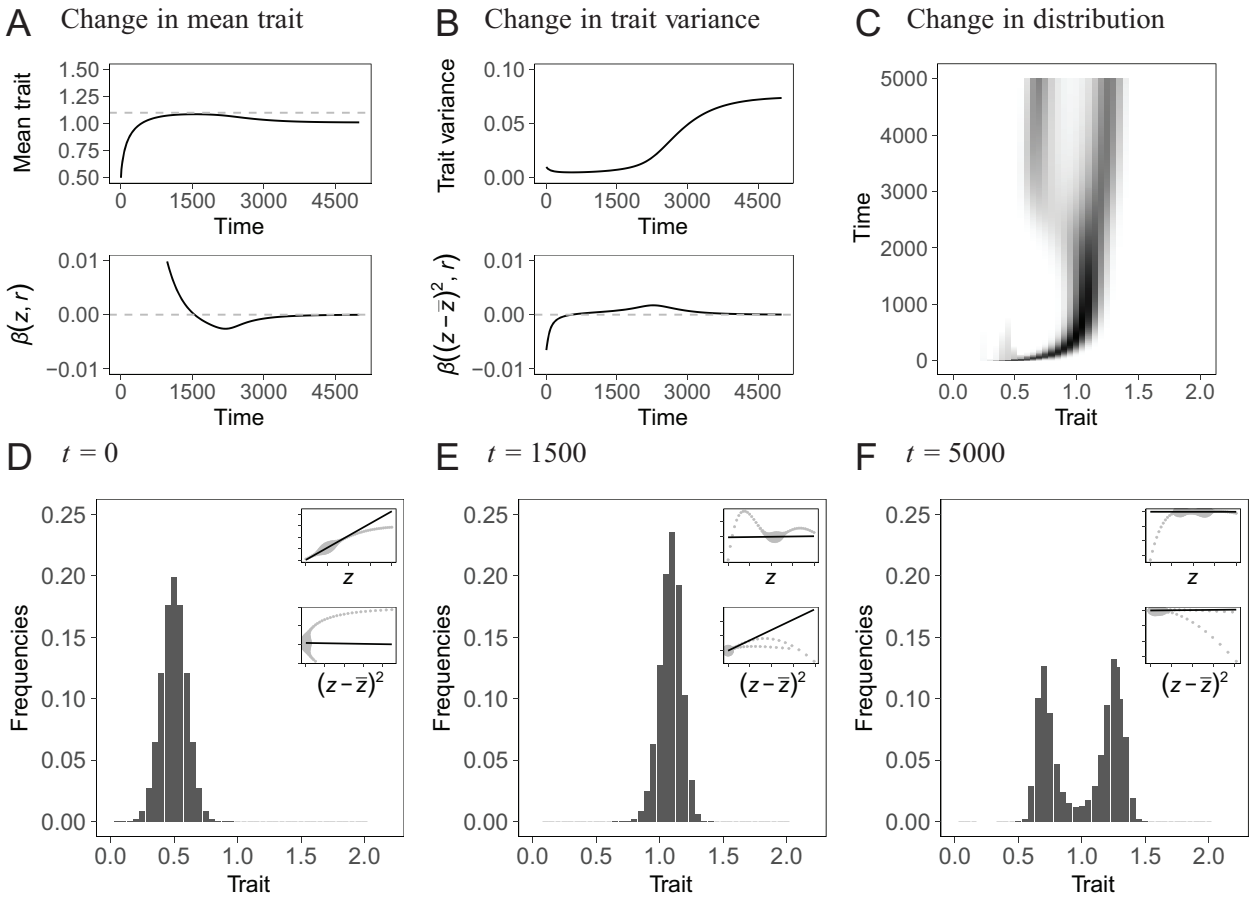

Figure 1: Dynamics of the mean and variance of a trait distribution. This figure presents results of a numerical integration of the Lotka-Volterra competition model studied by Kisdi (1999; example 1 described in the main text). I use $\rho(z) = \exp(-z^2/2)$, $a(x) = 2(1 - 1/(1 + \exp(-2.2x)))$, which corresponds to figure 3 in Kisdi (1999). At $t = 0$, the population is initiated with a Gaussian distribution of the trait, with mean 0.5 and standard deviation 0.1. There is no mutation, so selection operates solely on the standing variation. *A*, Change in the mean trait (*top*) and the regression of $r_i(\mathbf{E})$ on the trait (*bottom*). *B*, Change in the trait variance (*top*) and the regression of $r_i(\mathbf{E})$ on $(z_i - \bar{z})^2$ (*bottom*). *C*, Time evolution of the trait distribution (darker shades indicate higher strain frequencies). *D–F*, Trait distribution at $t = 0$, 1,500, and 5,000. Until about $t = 1{,}500$, the mean trait increases, but the distribution stays Gaussian with an approximately constant variance. After $t = 1{,}500$, the variance increases and the distribution splits into two phenotypic clusters, while the mean stays approximately constant. *D–F*, *insets*, weighted linear regressions of the per capita growth rates $r_i(\mathbf{E})$ on z_i and $(z_i - \bar{z})^2$ (each dot corresponds to one type, and the size of each dot is proportional to the frequency of the type). Selection becomes disruptive near $\bar{z} = 1.1$ (dashed line in *A*), which is the value predicted by the adaptive dynamics model of Kisdi (1999, fig. 3). See video 1 (available online) for an animated version of *D–F*.

tion (17) and classical quantitative genetics models (Lande and Arnold 1983). In both cases, the change in mean trait is determined by the product of the covariance matrix and the selection gradient. Crucially, equation (17) does not depend on the assumption that the distributions of the traits are Gaussian and does not assume that population variance is small. It does, however, rely on the assumption that the dynamics linearly depend on the traits.

Equations (16) and (17) can be used to investigate two conceptually different questions about the coupled eco-evolutionary dynamics of the host-parasite interaction. First, they can be used to study the short-term evolution of the traits. In general, numerical integration is the only way to obtain results. However, as discussed above, equation (17) gives only the dynamics of the first moment of the trait distribution and depends on higher-order moments (the variances and covariances in matrix **G**). To make some progress, we therefore need additional assumptions on the distribution of traits (Day and Proulx 2004). The quantitative genetics literature typically relies on the Gaussian or small variance approximations (box 1), in which case the gradient dynamics of equation (17) can be approximated using partial derivatives and coupled to a closed equation for the dynamics of the variance.

Box 1: Environmental Feedback and Classical Quantitative Genetics Models

A major contribution of early quantitative genetics theory was to consider the joint dynamics of ecological and evolutionary processes (Lande 1976; Slatkin 1979; Taper and Case 1985; Abrams et al. 1993). This is particularly apparent in models of ecological character displacement, where fitness $W(z, n, p(z, \bar{z}))$ is assumed to depend on the individual's trait z, the total density n, and the distribution $p(z, \bar{z})$ of the trait in the population (Slatkin 1980; Taper and Case 1985, 1992). Such fitness functions therefore explicitly depend on the environmental feedback, as discussed in the main text. However, it was immediately realized that these coupled models are often too complex to analyze. As a result, various analytical approximations have been sought, using either the Gaussian approximation or a small variance approximation. These assumptions yield two useful gradient expressions for the change in mean trait.

The Gaussian approximation assumes that the trait is (and remains) normally distributed. It can then be shown that the dynamics of the mean trait is

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \sigma_{zz}\overline{\frac{\partial w}{\partial z}}, \tag{a}$$

where the partial derivative $\partial w/\partial z$ is averaged over the trait distribution (Lande 1976, 1982; Lande and Arnold 1983; Taylor 1996; Taylor and Day 1997; Day and Proulx 2004). However, forcing the distribution to remain normal implicitly assumes a specific form of environmental feedback. Figure 1 shows that, in general, environmental feedbacks may alter the shape of the trait distribution. Whether this is relevant on the timescale of observation depends on the question at hand.

Alternatively, under the small variance approximation, the trait dynamics take the form (Charlesworth 1990; Iwasa et al. 1991; Abrams et al. 1993)

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \sigma_{zz}\left.\frac{\partial w}{\partial z}\right|_{z=\bar{z}}. \tag{b}$$

This result does not assume any particular shape of the trait distribution, except that it should be relatively narrow around the mean (although this condition can be relaxed if we make additional assumptions on the distribution; see, e.g., Abrams et al. 1993).

In many studies, an additional assumption is made about the fitness function, typically defined as a function $w(z, \bar{z})$ of the individual's trait and of the mean trait. It is worth highlighting that this formulation assumes a restrictive form of environmental feedback, because frequency dependence is handled only through the mean trait instead of the full distribution of the traits. However, different distributions with the same mean need not have the same ecological effect. Moreover, it is not always possible to write the fitness as a closed function of the mean trait, for instance, when frequency dependence is mediated by other ecological variables. For this reason, the environmental feedback formulation is in general conceptually more satisfying.

Equation (17) can also be used to investigate the long-term evolution of the traits. For instance, neglecting mutation, equation (17) shows that at evolutionary equilibrium the following relationship holds (Day and Gandon 2007):

$$\frac{\sigma_{\alpha\beta}}{\sigma_{\alpha\alpha}} = \frac{1}{\hat{S}}, \tag{18}$$

where $\sigma_{\alpha\alpha}$ is the variance of virulence and $\sigma_{\alpha\beta}$ is the covariance between virulence and transmission. In this specific example, maximizing the regression between virulence and transmission at evolutionary equilibrium is equivalent to minimizing the equilibrium density of susceptible hosts, $\hat{S}$. As we shall see later, a similar result can be derived from invasion analyses (box 2). Equation (18) can be viewed as the quantitative genetics equivalent of the pessimization principle (Mylius and Diekmann 1995; see Optimization, Polymorphism, and the Dimension of the Environmental Feedback Loop).

Long-Term Evolution and Limiting Mutation Regimes

Although the above equations yield an exact deterministic description of the joint ecological and evolutionary dynamics of the population, they are usually not analytically tractable and are typically studied using numerical integration or under restrictive assumptions on the trait distribution. Various approximations have been used to make further

progress, often taking the form of quasi-equilibrium approximations based on a separation of timescales. I will consider two limiting regimes for the mutation process: mutation limitation (low mutation rate) and weak selection (small phenotypic effects of mutations).

Mutations Are Rare

If the mutation rate is sufficiently low, one may assume that the population reaches its ecological attractor before a new type appears in the population (Metz et al. 1992; Geritz et al. 1998). In other words, we assume a separation of timescales so that ecological dynamics takes place on a fast timescale, while evolutionary dynamics is much slower.

For simplicity, let us assume that this attractor is a fixed point, characterized by a constant environment, $\mathbf{E} = \hat{\mathbf{E}}$. In this environment, the population is stable, and

$$\bar{r}(\hat{\mathbf{E}}) = 0. \tag{19}$$

A rare focal strain, m, will not affect this equilibrium, and from equation (6) we can write the initial dynamics of the rare mutant as

$$\frac{\mathrm{d}f_m}{\mathrm{d}t} = f_m r_m(\hat{\mathbf{E}}). \tag{20}$$

Hence, whether the mutant will grow depends on the sign of its per capita growth rate, $r_m(\hat{\mathbf{E}})$, in the environment set by the resident population on its ecological attractor. This is the definition of invasion fitness (Metz et al. 1992, 1996; Geritz et al. 1998), which is a core concept of the adaptive dynamics framework. Note that while the above discussion focuses on equilibria, the concept of invasion fitness is valid for other types of ecological attractors, such as periodic attractors (cycles), in which case one needs to consider the ergodic average of the per capita growth rate of the mutant on the attractor of the resident dynamics. For instance, for a periodic environment, one needs to integrate the per capita growth rate over one period of the cycle.

Because the environment on the resident attractor is solely determined by the resident dynamics, we can write $\hat{\mathbf{E}}$ as a function of the different resident traits z_i. Hence, invasion fitness can be written equivalently as a function of resident and mutant traits, $s(z_m; \mathbf{z})$, and we may write the invasion condition of the mutant type equivalently as

$$r_m(\hat{\mathbf{E}}) > 0 \quad \text{or} \quad s(z_m; \mathbf{z}) > 0. \tag{21}$$

For monomorphic resident populations, the signs of the invasion fitness $s(z_m; z_w)$ for different combinations of mutant and resident traits can be synthesized into a pairwise invasibility plot (PIP; Geritz et al. 1998), which allows the practitioner to graphically gauge the potential evolutionary outcomes of the model (see fig. 2 for an example).

Mutations Have Small Phenotypic Effects (Weak Selection)

Alternatively, we may assume that the mutation step is very small, so that the population variance in the trait is vanishingly small. This leads to another separation of timescales. If the trait value for each type is close to the mean trait in the population (i.e., we write $z_i = \bar{z} + \varepsilon\delta_i$ for a small parameter ε), it can be shown that the environmental dynamics are given by the zeroth-order terms of equations (3) and (5), while the dynamics of the frequencies f_i and the mean trait $\bar{z}$ depend on the first- and second-order terms of equations (8) and (10), respectively (see appendix; see also Meszéna et al. 2005). Therefore, under weak selection one may assume that ecological dynamics unfold on a fast timescale compared to the slower evolutionary dynamics. Under this separation of timescales, one may therefore assume that $\mathbf{e}$ and n reach a quasi equilibrium and use this quasi-equilibrium value to compute the first-order effect of selection on either the change in frequency or the change in mean trait.[2] This result holds irrespective of the frequency of each type and does not depend in particular on an assumption of rarity for a focal mutant strain.

The Adaptive Dynamics Toolbox

If we assume that mutations are rare and have small phenotypic effects, we recover the standard toolbox of adaptive dynamics or evolutionary game theory, which I will now review briefly. Because mutations are rare, it is typical to focus (at least initially) on a rare mutant in a monomorphic resident population on its ecological attractor. Under weak selection, we can write the change in mutant frequency as

$$\frac{1}{f_m}\frac{\mathrm{d}f_m}{\mathrm{d}t} = s(z_m; z_w) = \varepsilon\frac{\partial s}{\partial z_m} + \frac{\varepsilon^2}{2}\frac{\partial^2 s}{\partial z_m^2} + O(\varepsilon^3), \tag{22}$$

where $\varepsilon = z_m - z_w$ and the partial derivatives are evaluated at $z_m = z_w$. The direction of selection is given by the first-order effect on the change in frequency or equivalently by the selection gradient. The mutant frequency increases if

$$\mathcal{S}(z_w) = \left.\frac{\partial s}{\partial z_m}\right|_{z_m = z_w} > 0. \tag{23}$$

The zeros of the selection gradient, $\mathcal{S}(z)$, allow us to identify the evolutionary singularities (i.e., the potential evolution-

2. More generally, one may compute the ergodic average of the per capita growth rate (Meszéna et al. 2005).

Box 2: Adaptive Dynamics of a Host-Parasite Interaction

To illustrate the adaptive dynamics methodology, let us consider a simple host-parasite interaction with density-dependent birth and vertical transmission (Lipsitch et al. 1996; Ferdy and Godelle 2005). The dynamics of the monomorphic resident population is given by the following system of ODEs:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(S + (1-\delta)f(\alpha)I)\left(1 - \frac{N}{K}\right) - \beta(\alpha)SI - dS;$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \delta bf(\alpha)I\left(1 - \frac{N}{K}\right) + \beta(\alpha)SI - (d+\alpha)I.$$

Susceptible hosts reproduce at rate b and die at rate d. Infected hosts die at rate $d + \alpha$, where α is the virulence of parasites, and reproduce at rate $bf(\alpha)$, where $f(\alpha)$ is the relative fecundity of infected hosts, which is assumed to be a function of parasite virulence. Vertical transmission of the parasite occurs on reproduction with probability δ. Horizontal transmission occurs at rate $\beta(\alpha)$, where we assume a trade-off between transmission and virulence. Depending on the parameter values, the model can lead to the extinction of both species, a disease-free equilibrium with $\hat{S} = K(1 - d/b)$, or an endemic equilibrium $(\hat{S}, \hat{I})$.

Horizontal Transmission Only ($\delta = 0$)

In the absence of vertical transmission, the invasion fitness of a rare mutant parasite with virulence α_m in a resident population at endemic equilibrium takes the following simple form:

$$s(\alpha_m; \alpha) = \beta(\alpha_m)\hat{S} - (d + \alpha_m). \tag{a}$$

In this textbook example, invasion fitness depends on only one environmental variable ($\hat{S}$) in a monotone way. This model therefore admits an optimization principle (see Optimization, Polymorphism, and the Dimension of the Environmental Feedback Loop). For an increasing, concave function $\beta(\alpha)$, the evolutionarily stable α^* can be obtained by simply maximizing the function $\beta(\alpha)/(d + \alpha)$, which can be interpreted as the lifetime infectivity of a parasite. This is equivalent to minimizing the equilibrium density of susceptibles $\hat{S}(\alpha) = (d + \alpha)/\beta(\alpha)$. In the end, the parasite strategy that can survive with the lower possible density of susceptible hosts is selected for. This is an example of the pessimization principle (Mylius and Diekmann 1995).

Two Routes of Transmission

When δ is nonzero, the invasion fitness is given by

$$s(\alpha_m; \alpha) = \delta bf(\alpha_m)\left(1 - \frac{\hat{N}}{K}\right) + \beta(\alpha_m)\hat{S} - (d + \alpha_m). \tag{b}$$

Equation (b) shows that invasion fitness now depends on two environmental variables, $\hat{S}$ and $\hat{N}$. In this case, there is no optimization principle, and it is possible to find conditions where evolutionary branching occurs at an evolutionary singularity, which is a zero of the selection gradient,

$$\left.\frac{\partial s}{\partial \alpha_m}\right|_{\alpha_m = \alpha} = \delta bf'(\alpha)\left(1 - \frac{\hat{N}}{K}\right) + \beta'(\alpha)\hat{S} - 1.$$

The stability of an evolutionary singularity, α^*, is given by the sign of the second-order derivative,

$$\left.\frac{\partial^2 s}{\partial \alpha_m^2}\right|_{\alpha_m = \alpha = \alpha^*} = \delta bf''(\alpha^*)\left(1 - \frac{\hat{N}}{K}\right) + \beta''(\alpha^*)\hat{S}.$$

Thus, for a concave-down trade-off $\beta(\alpha)$, the stability of α^* depends on the convexity of $f(\alpha)$. If $f''(\alpha)^*$ is positive, it is possible to find some parameters such that α^* is an evolutionarily stable strategy for low values of δ and a branching point for higher values of δ, as illustrated in figure 2.

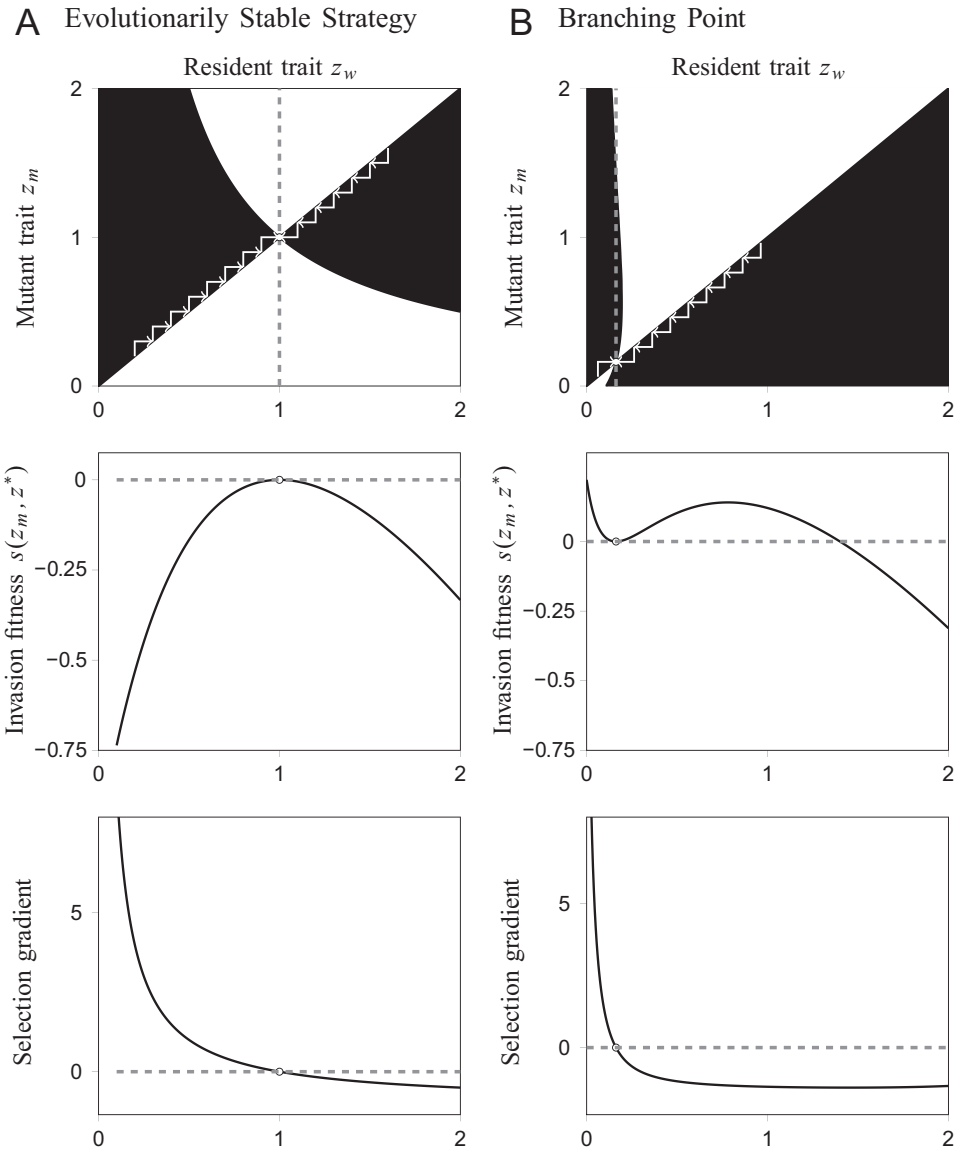

Figure 2: Examples of evolutionary outcomes for mutation-limited evolution. The panels are drawn using the model of box 2. *A*, Virulence is predicted to evolve toward an evolutionarily stable strategy. *B*, Virulence is predicted to evolve toward a branching point. *Top*, pairwise invasibility plot (invasion fitness is positive in black regions). *Middle*, fitness profile around the evolutionary singularity (corresponding to the dashed vertical line in the pairwise invasibility plot). *Bottom*, selection gradient (giving the sign of selection along the diagonal). Parameters: $\delta = 0$ (*A*), $\delta = 0.5$ (*B*). The following trade-off functions are used: $\alpha(z) = z$, $\beta(z) = \beta_0 z/(1 + z)$, $b_l(z) = b\kappa/(1 + cz)$. Other parameters: $b = 3$, $d = 1$, $K = 5$, $\beta_0 = 10$, $c = 6$, $\kappa = 15$. See videos 2, 3 (available online) for animations showing the change in the fitness landscape resulting from changes in the resident trait.

ary end points). Two questions may be asked regarding these singularities. First, one may ask whether a given singularity, z^*, can be attained through small mutation steps (convergence stability; Eshel 1983, 1996; Taylor 1989; Geritz et al. 1998). An evolutionary singularity is convergence stable if the selection gradient $\mathcal{S}(z)$ is a locally decreasing function of $z(\mathrm{d}\mathcal{S}(z)/\mathrm{d}z < 0)$, which can be written in terms of second-order derivatives (Geritz et al. 1998) as

$$\left.\frac{\partial^2 s}{\partial z_w^2}\right|_{z_m = z_w = z^*} > \left.\frac{\partial^2 s}{\partial z_m^2}\right|_{z_m = z_w = z^*}. \tag{24}$$

Convergence-stable strategies are called evolutionary attractors.

Second, the evolutionarily stability of these singularities can be determined by the sign of the second-order term of the Taylor expansion of the invasion fitness (Taylor 1989; Christiansen 1991; Eshel 1996; Geritz et al. 1998). In particular, a singularity z^* will be called evolutionarily stable (ESS) if

$$\left.\frac{\partial^2 s}{\partial z_m^2}\right|_{z_m=z_w=z^*} < 0. \tag{25}$$

A singularity that is both an ESS and convergence stable is called a continuously stable strategy (Eshel and Motro 1981; Eshel 1983) and represents a monomorphic end point of the evolutionary process. In contrast, a singularity that is convergence stable but not ESS is called a branching point: an initially monomorphic population is attracted toward this singularity, but in the vicinity of the singular point the population becomes dimorphic and splits into two diverging subpopulations owing to disruptive selection (Geritz et al. 1998). We have already encountered this situation in figure 1, and another example is described in box 2. Figure 2 illustrates how an ESS and a branching point can be identified using a PIP or by looking at the shape of invasion fitness around the singularity.

The Canonical Equation of Adaptive Dynamics

As for the Price equation, we can derive a dynamical equation for the change in the trait z under this limiting mutation process. The resulting ODE has been named the canonical equation of adaptive dynamics and was introduced by Dieckmann and Law (1996). In its basic form for one-dimensional traits in unstructured population, it reads:

$$\frac{\mathrm{d}z}{\mathrm{d}t} = \frac{1}{2}\mu(z)\sigma(z)\hat{n}(z)\left.\frac{\partial s}{\partial z_m}\right|_{z_w=z_m=z}, \tag{26}$$

where $\mu(z)$ is mutation rate per birth event, $\sigma(z)$ is the variance of the mutation distribution, and $\hat{n}(z)$ is the equilibrium size of the resident population. This equation shows that the direction of the change in the trait z is given by the selection gradient and scaled by a positive factor that gives the rate of evolutionary change. This equation is derived as a deterministic approximation of a stochastic model under the assumptions that mutations are rare and have small steps (Dieckmann and Law 1996; Champagnat et al. 2001, 2006; Proulx and Day 2001). The canonical equation has been extended to class-structured and physiologically structured populations as well as diploid and haplodiploid genetics (Durinx et al. 2008; Méléard and Tran 2009; Metz and de Kovel 2013). A general form has been given by Metz (2011) for the dynamics of a vector of traits:

$$\frac{\mathrm{d}\mathbf{z}}{\mathrm{d}t} = N_e\mu(\mathbf{z})\mathbf{C}(\mathbf{z})\mathbf{S}(\mathbf{z}). \tag{27}$$

Here, $\mu(\mathbf{z})$ is mutation rate per birth event, $\mathbf{C}(\mathbf{z})$ is the mutational covariance matrix, $\mathbf{S}(\mathbf{z})$ is the vector of selection gradients, and N_e is the effective size of the population. Note the similarity with the multidimensional Price equation (15). The dynamics of mean traits is also given by the product of a matrix, $\mathbf{G} = N_e\mu\mathbf{C}$, multiplied by a vector of selection gradient $\mathbf{S}$. However, equation (15) does not assume a separation of timescales.

As noted previously, the direction of selection in a multidimensional trait space will in general be affected by pleiotropic effects, which the matrix $\mathbf{C}$ captures. Thus, defining convergence stability for vector traits is not as straightforward as for scalar traits. A summary of known results on multidimensional convergence stability can be found in Leimar (2009), where the notion of strong convergence stability is introduced. He shows that if the Jacobian matrix of the selection gradient is negative definite at a singular point (where $\mathbf{S} = 0$), this singular point is an asymptotically stable point of equation (15), irrespective of the genetic matrix $\mathbf{G}$. In this situation, pleiotropy does not matter. Similarly, evolutionarily stability can be evaluated from the Hessian matrix (Leimar 2009; Débarre et al. 2014; Svardal et al. 2014; Mullon et al. 2016). These latter articles provide a good entry point into the topic of multidimensional trait space, with particular reference to the consequences on evolutionary branching. It should also be noted that, mathematically speaking, the coevolution of two species is not a different question from the joint evolution of two traits, and a similar toolbox can be applied (see, e.g., Marrow et al. 1996; Best et al. 2010; Débarre et al. 2014).

To better understand the connection between the canonical equation and the quantitative genetics framework, an alternative formulation introduced by Champagnat et al. (2001), Proulx and Day (2001), and Day (2005) is useful. Instead of using a branching process approximation, they derive an equation for the mean trait in function of a gradient of fixation probability instead of a gradient of invasion fitness. They obtain the following equation, under the assumption that the trait distribution is tightly centered around the mean $\bar{z}$ (which is equivalent to assuming small mutational steps):

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \mu(\bar{z})\sigma(\bar{z})b(\bar{z})\hat{n}(\bar{z})\left.\frac{\partial U}{\partial z}\right|_{z=\bar{z}} + \mu(\bar{z})b(\bar{z})\mathcal{M}. \tag{28}$$

The first term on the right-hand side of equation (28) bears a close resemblance to the right-hand side of equation (26).

The extra $b(\bar{z})$ factor, which represents the per capita birth rate, comes from the substitution of the fixation probability U for invasion fitness. However, there is a second term that is proportional to $\mathcal{M}$, the first moment of the mutation distribution. A nonzero value of $\mathcal{M}$ indicates mutational bias in phenotype space. Although frequently ignored in the theoretical literature, evidence of systematic distortions of the mutation machinery suggests that the directional effect of mutations on phenotypic evolution may be important (Stoltzfus and Yampolsky 2009; Lynch 2010). Thus, equation (28) partitions the change in mean trait into a component due to selection and a component due to mutation bias, as in the Price equation, but does so with the extra assumptions of a separation between ecological and evolutionary timescales and of weak selection.

Finally, it is worth emphasizing that, as in the Price equation, the canonical equation of adaptive dynamics is only the first of a series of moment equations. An equivalent dynamical equation could be written for the mutational variance of the covariance matrix and for higher moments of the mutation distribution.

Implications

The focus on environmental feedbacks helps to lay bare the formal connections between the Price equation and adaptive dynamics formalisms. It is also helpful when discussing the implications of both theoretical frameworks for our understanding of eco-evolutionary processes.

Environmental Feedback, Population Variance, and Timescales

Most models in evolutionary theory assume a separation of timescales between ecological and evolutionary processes. This is true of classical population genetics models, where population size is treated as a constant parameter, and of more elaborate ecological models studied using adaptive dynamics. A key insight of the large body of work that is synthesized here is that the tightness of the coupling between ecology and evolution crucially depends on the variance in the trait distribution. This can be seen by writing each trait as a deviation from the mean trait, $z_i = \bar{z} + \varepsilon\delta_i$. As a result, the variance is proportional to ε^2, and the scaling parameter ε can be used to investigate the impact of variance on the coupling between ecological and evolutionary dynamics. If ε is small, the full ecological-evolutionary dynamics can be described by the following system:

$$\text{environmental dynamics } \frac{\mathrm{d}\mathbf{E}}{\mathrm{d}t} = \mathcal{F}^{\circ}(\bar{z}, \mathbf{E}) + O(\varepsilon), \quad (29a)$$

$$\text{evolutionary dynamics } \frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \sigma_{zz}[\mathcal{S}^{\circ}(\bar{z}, \mathbf{E}) + O(\varepsilon)]. \quad (29b)$$

In equation (29a), the function $\mathcal{F}^{\circ}$ represents the zeroth-order term of the environmental dynamics with respect to the parameter ε. In other words, $\mathcal{F}^{\circ}(\bar{z}, \mathbf{E})$ represents the part of environmental dynamics that results from a monomorphic population where all types have the same value of the trait, $z_i = \bar{z}$. Similarly, equation (29b) shows that the evolutionary dynamics depends on the variance σ_{zz} multiplied by the selection gradient $\mathcal{S}^{\circ}(\bar{z}, \mathbf{E})$. Again, to evaluate the selection gradient, we retain only the environmental feedback due to a monomorphic population with the mean trait. The errors resulting from substituting this simplified environment for the true environment are of order ε.

This perturbation analysis allows us to identify three main regimes of interest for evolutionary ecologists. First, when the variance is vanishingly small, a full separation of timescales is possible because the mean trait changes on a much slower timescale compared to the environmental dynamics. This is because the dynamics of the mean trait is scaled by the variance, which is $O(\varepsilon^2)$ whereas ecological dynamics are $O(1)$. Then, it is possible to assume that the environmental dynamics (29a) have reached an attractor and to calculate the selection gradient as $\mathcal{S}^{\circ}(\bar{z}, \hat{\mathbf{E}})$, where $\hat{\mathbf{E}}$ is calculated on the (monomorphic) ecological attractor. Furthermore, because the variance changes on an even slower timescale, it is possible to treat the variance as a constant, at least away from evolutionary singularities.

Second, when the variance is larger but remains sufficiently small for the approximations (29) to remain accurate, it is no longer possible to assume that the environmental dynamics have reached an ecological attractor before a change in the mean trait can be observed. Hence, a full separation of timescales is not possible, but it is still possible to summarize a potentially complex environmental feedback using the monomorphic environment. This greatly simplifies the problem, because the full distribution of the trait then becomes irrelevant to calculate the dynamics. The environmental feedback is determined only by the mean trait (see, e.g., Abrams and Matsuda 1997; Day and Proulx 2004). This can be seen as a generalization of the standard quantitative genetics approach where fitness is assumed to depend only on the individual's trait and the mean trait (box 1).

Finally, when the variance is too large for approximations (29) to hold, there is no escaping the complex problem of jointly tracking the entangled dynamics of the trait distribution and the environmental variables. In most cases, analytical insight will be harder to obtain, and researchers will typically have to turn to numerical approaches.

An interesting perspective for future research would be to investigate the domains of validity of these three regimes. When a particular shape for the fitness function is assumed, as in many quantitative genetics models, a condition for the accuracy of the small variance approximation can be obtained that depends on the higher-order derivatives of fitness (Abrams et al. 1993; Abrams and Matsuda 1997). Other conditions have been derived in more complex ecological models (see, e.g., Day and Proulx 2004), but most of our current understanding comes from models with a full separation of timescales. We lack a clear picture of the interplay between population variance and environmental feedback in general ecological scenarios.

When Does Invasion Imply Fixation?

Equation (29b) shows that, provided selection is weak enough, the direction of selection depends only on the sign of the selection gradient. In particular, the change in frequency of a focal mutant type in an equilibrium monomorphic resident population with trait value z_w can be written as

$$\frac{\mathrm{d}f_m}{\mathrm{d}t} = \varepsilon f_m(1 - f_m)\mathcal{S}(z_w) + O(\varepsilon^2). \tag{30}$$

The selection gradient has been found to be independent of the frequency of the mutant type in a large class of haploid models with a single demographic attractor (Rousset 2004, 2006; Meszéna et al. 2005; Lehmann and Rousset 2014; Lehmann et al. 2016). Frequency dependence, then, appears only in the second-order terms of the change in frequency (Rousset 2004; Meszéna et al. 2005). This implies that, away from an evolutionary singularity, the direction of selection will not change as the mutant frequency increases, and as a consequence, invasion implies fixation. The long-term evolutionary process can thus be described as a trait-substitution sequence (Gillespie 1983; Eshel 1996; Hammerstein 1996; Metz et al. 1996; Weissing 1996; Eshel et al. 1998; Geritz et al. 1998).

However, there are many interesting cases where invasion need not imply fixation. First, in models where a separation of timescales does not hold, a focal type may be selected only transiently. This has been observed in epidemiological models and experiments where the initial abundance of susceptible hosts favors virulent strains that are subsequently counterselected when the endemic equilibrium is reached (Lenski and May 1994; Gandon and Day 2007; Bull and Ebert 2008). Second, even when the population has reached an ecological attractor, it is not always possible to predict the outcome of the competition between the mutant and the resident using only an invasion criterion. This may occur in ecological models with multiple demographic attractors, where an initially successful invasion by a mutant type may cause the resident population to switch to another attractor at which the mutant cannot invade, resulting in the failure of the invasion (a scenario known as "the resident strikes back" in the literature; see, e.g., Doebeli 1998; Diekmann et al. 1999; Mylius and Diekmann 2001). In such situations, invasion will imply fixation only if the resident and mutant traits are sufficiently close (Geritz et al. 2002; Geritz 2005) and if the population is away from a bifurcation point where the resident attractor undergoes an abrupt change (see, e.g., Matsuda and Abrams 1994; Doebeli 1998). Hence, it is important to bear in mind that the focalization on relatively simple ecological models and a particular set of approximations (weak selection) may cause us to miss important features of real ecological communities.

The Ubiquity of Gradient Dynamics

The adaptive dynamics and quantitative genetics frameworks both show that it is possible to capture the effect of selection through a gradient formulation: the change in mean trait is given by a measure of genetical variation, which depends on the mutational regime, multiplied by a selection gradient that gives a first-order (linear) approximation of fitness. In the absence of mutational bias, this is all we need to evaluate the direction of selection and potential evolutionary end points.

This result has been repeatedly found in the literature. This is a cornerstone of many key results in quantitative genetics (see box 1). In fact, the label "canonical" was attached to equation (26) by Dieckmann and Law (1996) precisely to refer to the ubiquitous nature of such gradient dynamics in evolutionary theory. There is, however, a subtle difference between the interpretations of the measure of genetic variation in quantitative genetics and in adaptive dynamics. In quantitative genetics, the factor in front of the selection gradient measures the standing variation in the population on which selection can operate. In the canonical equation of adaptive dynamics, the factor depends on the probability and variance of new mutations. This difference arises because the canonical equation of adaptive dynamics is obtained as a particular limit of the selection-mutation balance leading to mutation-limited evolution.

Gradient dynamics are ubiquitous because theoreticians tend to use the same approximations to describe evolution. The environmental feedback perspective emphasizes that gradient dynamics implicitly assume a specific form of feedback where the effect of selection is affected only by the contribution of environmental dynamics resulting from a monomorphic population with the mean trait.

Optimization, Polymorphism, and the Dimension of the Environmental Feedback Loop

A popular misconception is that evolution necessarily leads to the maximization of a population- or individual-level quantity. In our discussion of Fisher's fundamental theorem, we have seen that mean population growth rate is not necessarily maximized through evolution, due to the effect of environmental change. This mirrors the well-known result in population genetics that frequency dependence prevents the maximization of mean fitness (see, e.g., Rice 2004; Day 2005 for discussions). But is it nonetheless possible to predict the outcome of long-term evolution by maximizing some well-chosen measure of absolute fitness, such as individual lifetime reproductive success?

The Rarity of Optimization Principles. Recent theoretical advances have shown that the answer to this question lies in the effective dimension of the environmental feedback loop, defined as the number of independent variables needed to characterize the sign of the invasion fitness $r(z_m, \hat{\mathbf{E}})$ of a mutant with trait z_m in the resident environment $\hat{\mathbf{E}}$ (Mylius and Diekmann 1995; Heino et al. 1998; Metz et al. 2008; Gyllenberg et al. 2011; Metz and Geritz 2016). In short, if the effect of the environment on the sign of invasion fitness can be summarized by a single number with monotonic effect, it is possible to find a function of the trait $\phi(z)$ that is maximized at an ESS (optimization principle) or equivalently a function of the environment $\psi(\hat{\mathbf{E}})$ that is minimized at an ESS (pessimization principle). In some special cases, the function ϕ coincides with the lifetime offspring number R_0 or the intrinsic rate of natural increase r (Mylius and Diekmann 1995; Metz et al. 2008; Gyllenberg et al. 2011), but this is not a general property.

Most realistic ecological models will require more than one real number to accurately describe the action of the environment. Think of a predator feeding from multiple prey species, a forager exploiting a time-fluctuating resource, or a size-structured population where intraspecific competition depends on the size distribution. However, many textbook results are simple enough to allow a one-dimensional representation of the environment. For instance, models satisfying some form of marginal value theorem (Charnov 1976) admit an optimization principle. An example is provided in box 2 using our running example for parasite evolution, where, in the absence of vertical transmission, invasion fitness (eq. [a]) is an increasing function of the density of susceptible hosts. This focalization on simple models may partly explain the long tradition of using optimization approaches in evolutionary theory.

Environmental Feedbacks and Evolutionary Branching. An immediate consequence of the existence of an optimization principle is that the evolutionary process is of the simplest kind: any evolutionarily stable strategy is an evolutionary attractor and vice versa (Metz et al. 2008). Consequently, evolutionary branching is impossible, and at most one type can persist in the population. In contrast, evolutionary branching can occur in more general models where the effective dimension of the environment is higher than 1. This can be seen as a generalization of Levin's competitive exclusion principle (Levin 1970). Properly assessing the dimensionality of the environmental feedback is therefore a critical step in the formulation of an evolutionary model, especially if one is interested in the origin and maintenance of diversity.

Once the population becomes dimorphic, new tools are needed to analyze the evolutionary dynamics. These tools rely on the concept of invasion fitness of a rare mutant in a coalition of coexisting strategies (eq. [21]). For instance, one may use $s(z_m; z_1, z_2)$, the invasion fitness of a mutant with trait z_m in a resident population consisting of two types with traits z_1 and z_2, or more generally $r_m(\hat{\mathbf{E}}(C))$, the invasion fitness of a mutant calculated on the ecological attractor determined by the resident community C (Geritz et al. 1998, 2004; Metz 2008). It is then possible to determine dimorphic singularities and check whether they are evolutionarily stable or whether further branching is possible (see Kisdi 1999 for an illuminating introductory example). Quantitative genetics models provide an alternative approach to disruptive selection based on the dynamics of the variance (Wakano and Iwasa 2013; Débarre and Otto 2016). In finite populations, this approach has been used to show that small population sizes tend to be less conducive to evolutionary branching (Claessen et al. 2007; Wakano and Iwasa 2013; Débarre and Otto 2016).

Models for the Real World?

Over the years, the development of the AD and QG approaches has provided us with a strong theoretical basis to analyze the feedback loop between ecology and evolution. However, for many realistic ecological scenarios, further extensions are needed, and we still face some unresolved challenges. In this last section, I will briefly describe various refinements of the AD and QG approaches to tackle physiologically and spatially structured populations and to study the evolution of responsive or plastic traits. I then sum up four key challenges for future theory, namely, the integration of complex genetics, the development of new techniques beyond the weak selection or normal approximations, the consequences of stochasticity, and the coupling of evolutionary change with complex, nonstable population dynamics.

Natural Populations Are Structured

In nature, individuals of a given type may still differ by their state (heterogeneity, or h-state; Metz et al. 1992; Metz

2008). The h-state of an individual collects all sources of heterogeneity, such as the physiological state of the individual (age, size, infection status, etc.) or even its spatial location. A population structured with a discrete h-state is often referred to as class structured (Taylor 1990; Caswell 2001; Rousset 2004), but age- or size-structured populations provide examples of a continuous h-state (Metz and Diekmann 1986; Charlesworth 1994; de Roos 1997; Diekmann et al. 2003).

The description of populations used in the above sections ignores population structure by aggregating all individuals of a given type together. However, this may be misleading or uninformative because the different h-states need not have the same evolutionary value. This occurs, for instance, in species with both reproducing and sterile stages, in populations exploiting habitats of different qualities, or in age-structured populations where the strength of selection may vary with age. In the literature, these intrinsic differences in quality among h-states are generally quantified using the notion of reproductive value, which measures the relative contribution of each class to the future of the population (Fisher 1930; Taylor 1990; Frank 1998; Rousset 2004; Rousset and Ronce 2004). Reproductive values are best viewed as a vector of weights that give the relative importance of selective effects in each class (Taylor 1990; Frank 1998; Gardner et al. 2011; Lehmann and Rousset 2014).

Equipped with this concept, extensions of the theory to account for class structure have been formulated, either in models with separation of timescales (Taylor 1990; Metz et al. 1992; Frank 1998; Rousset 2004; Rousset and Ronce 2004; Metz 2008) or using the Price equation (Day and Gandon 2006; Gandon and Day 2007). Related approaches exist for continuous h-states (Durinx et al. 2008; Day et al. 2011). In particular, integral projection models have become a popular tool for modeling structured populations and can be coupled with both the AD and the QG approaches (Rees and Ellner 2016). In most studies, analytical insight is typically gained with the additional help of the now familiar weak selection assumption.

Spatial Structure and Inclusive Fitness Theory

Spatial structure is another important component of realistic ecological dynamics. In spatially structured populations, selection will be determined by the interplay between demographic and genetic structures (Lion et al. 2011). Demographic structure describes the spatial distribution of individuals, while genetic structure describes the spatial distribution of types. Genetic structure may generally be neglected when we have infinite local population sizes, but typically local fluctuations and limited dispersal will lead to the buildup of genetic structure in the population. For instance, under local dispersal, one may expect similar types to be more clustered together than they would be in a well-mixed population. Deviations from random spatial distributions of types can be quantified using relatedness coefficients, which typically measure the probability of identity between pairs of genes sampled in different individuals (Rousset 2004; Lehmann and Rousset 2014).

Spatial structure therefore complicates our vision of environmental feedback in two important ways. First, we need many more variables to accurately describe the spatial structure of the environment, which leads to high-dimensional models. Second, a proper description of spatial dynamics requires one to take into account local demographic stochasticity. It is therefore not obvious that the above framework is helpful when studying spatially structured populations.

However, provided the total population is large enough, the Price equation and adaptive dynamics approaches can be extended to account for spatial structure. The idea is to use a moment expansion to express the rate $r_i(\mathbf{E})$ in function of higher-order spatial moments (van Baalen and Rand 1998; Bolker and Pacala 1999; van Baalen 2000; Ovaskainen et al. 2014; Lion 2016). The differential equations describing the dynamics of the densities n_i should now be viewed as representing the expected dynamics derived from a microscopic description of the spatial individual-based process and must be coupled with a suite of equations describing the expected dynamics of various spatial moments (Ovaskainen et al. 2014; Lion 2016). Spatially structured versions of the Price equation and adaptive dynamics methodologies can then be derived (Lion 2016; Lion and Gandon 2016). The downside of this approach is that the environment $\mathbf{E}$ is now an infinite vector collecting all relevant spatial moments of the population. As a result, moment closure approximations are needed to close the system and obtain a low-dimensional approximation. Unfortunately, the accuracy of those approximations is currently poor. Alternative perturbation methods have been proposed as a replacement (Ovaskainen and Cornell 2006), but their application to evolutionary theory has so far been limited (but see North et al. 2011).

There is a close relationship between models based on spatial moment equations and more classical deme-structured populations, often analyzed using inclusive fitness methodology (Rousset and Billiard 2000; Metz and Gyllenberg 2001; Ajar 2003; Parvinen et al. 2003; Rousset 2004; Lehmann et al. 2016). From an ecological perspective, inclusive fitness is perhaps best viewed as an expansion of invasion fitness to take into account the indirect fitness effects generated by spatial demographic and genetic structuring (see also Lehmann and Rousset 2014; Van Cleve 2015; Lehmann et al. 2016 for a more technical treatment). However, many discussions of inclusive fitness, kin selection, or

group selection focus on simplified models with little ecological realism (Lion et al. 2011). An unfortunate consequence is that the ecological underpinning of inclusive fitness theory is not apparent. It should be noted, however, that although the inclusive fitness approach to evolution in space has been extended to models with fluctuating demography (Rousset and Ronce 2004; Lehmann and Rousset 2014), the application of these methods to complex ecological scenarios is not straightforward. In some cases, such as models of host-parasite interactions, spatial moment equations have been used to derive compact analytical expressions for invasion fitness while retaining the useful interpretation in terms of inclusive fitness and kin selection theory (Lion and Gandon 2016).

In general, deriving good approximations of evolutionary change in complex spatial ecologies is still a challenge and an active area of research. An important objective of a research program in spatial evolutionary ecology is to formulate new methods to derive deterministic approximations of spatial models and to check the range of validity of approximations. It is possible that in many real-life examples, taking into account spatial structure will bring little added value compared to standard well-mixed models, but we still lack a good understanding of when spatial structure can and cannot be neglected.

Beyond Traits: Evolution of Responsive Phenotypes

I have previously discussed the evolution of fixed traits, following standard practice in evolutionary ecology. However, most traits we are interested in are not constant but change in response to another continuous variable. For instance, the vital rates of an individual generally depend on their age and can also be affected by environmental conditions (pH, temperature, salinity, etc.). Such responsive traits are best viewed as functions (Stinchcombe et al. 2012). Common examples of function-valued traits are growth trajectories (e.g., mean body size as a function of age) or the reaction norms of plastic phenotypes (giving the expected phenotype of a given genotype as a function of an environmental variable).

To study the evolution of responsive traits, an extension of the classical QG machinery was proposed by a number of authors (Kirkpatrick and Heckman 1989; Kirkpatrick and Lofsvold 1992; Beder and Gomulkiewicz 1998; Kingsolver et al. 2001). Although these models considered very simple ecological dynamics, more realistic environmental feedbacks can be taken into account. Using an epidemiological dynamics, Day et al. (2011) derived a Price equation for function-valued traits. As for fixed traits, additional assumptions (such as normal distribution of phenotypes, weak selection, or mutation limitation) can be used to rewrite the covariance term as a gradient dynamics (Kirkpatrick and Heckman 1989; Dieckmann et al. 2006; Day et al. 2011; Stinchcombe et al. 2012). Thus, although mathematically more challenging, the evolution of responsive phenotypes can be studied through the same methods as fixed traits. Depending on the context, responsiveness and plasticity can also be studied using more simple class-structured models, for instance, when modeling the eco-evolutionary consequences of animal personalities (Wolf et al. 2007; Wolf and Weissing 2012).

Further Challenges

The body of theoretical literature I synthesize here appears to be sufficiently robust to handle population heterogeneity, spatial structure, and even function-valued traits used to model responsive phenotypes. In my opinion, these extensions raise technical (and often mathematically difficult) problems but do not pose a real conceptual challenge to theoreticians.

However, the above discussion highlights an important difficulty of current eco-evolutionary theory. Indeed, although the Price equation approach is very general, the resulting models are often difficult to analyze without recourse to numerical methods. Both the AD and the QG approaches rely on a small variance approximation to make analytical progress, but as we have seen, the timescales between ecological and evolutionary dynamics are affected by the magnitude of population variance. When the population variance is sufficiently small, the environmental feedback can be simplified and decoupled from the evolutionary dynamics, but to my knowledge little attention has been devoted to the domain of validity of this approximation. In particular, it is unclear to what extent we can use this approximation to study rapid evolutionary dynamics caused by high standing variation and/or mutation rates. Progress will likely come from new perturbation methods that go beyond the common assumption of tightly clustered unimodal trait distributions or Gaussian trait distribution. The oligomorphic approximation introduced by Sasaki and Dieckmann (2011) seems a promising step in that direction. This approach extends the standard theoretical toolbox through a decomposition of multimodal trait distributions into a sum of unimodal distributions, each corresponding to a morph (oligo = "a few"). It would be interesting to conduct an extensive investigation of the coupling with environmental feedbacks within this framework.

Another limitation of current theory is the focus on ecological attractors. Broadly speaking, analytical predictions for evolution under complex ecological scenarios are possible only if the ecological dynamics are poised at an attractor (in practice, often an equilibrium). As we have seen, this requires a separation of timescales. However, the

concept of an ecological attractor or stationary ecological states has been criticized for being unrealistic, especially when studying rapid environmental changes such as climate fluctuations. Recently, Chesson (2017) suggested that the classical attractor concept could be usefully replaced in ecology by an asymptotic environmentally determined trajectory, which is a time-dependent function of the environment toward which the population converges. This could provide an interesting starting point for an extension of eco-evolutionary theory to handle rapid, nonstationary dynamics for which the standard separation of timescales fails. In particular, this could shed new light on the mechanisms causing evolution toward extinction (evolutionary suicide; Matsuda and Abrams 1994; Parvinen 2005) or away from extinction (evolutionary rescue; Bell and Gonzalez 2009).

The deterministic models I have discussed assume large population sizes and neglect environmental stochasticity. Stochastic models of population dynamics, as well as individual-based simulations, often show systematic deviations from deterministic predictions (Lande et al. 2003). Evolutionary dynamics can also be strongly affected by stochasticity, for instance, because of genetic drift (Lenormand et al. 2009). In particular, in finite populations, the "invasion implies fixation" principle breaks down and the probability of fixation will typically be a better measure of selection than invasion fitness (Proulx and Day 2001). However, we still lack a clear picture of the ecological conditions under which deterministic approximations fail and stochasticity needs to be explicitly taken into account. In particular, because most studies of evolution in finite populations assume some kind of separation of timescales, often based on a weak selection assumption (Rousset 2004, 2006; Van Cleve 2015; Débarre and Otto 2016), we are still far from a full understanding of the joint effect of stochasticity and environmental feedback on evolutionary dynamics.

The last challenge is perhaps more fundamental. As is common practice in evolutionary ecology, I have chosen to simplify the genetics to focus on the ecology. Clearly, for many ecologically relevant traits in nature, integrating genetics and ecology is a necessary challenge, and the task may seem daunting. There are still very few studies that couple complex genetics or developmental pathways with ecologically realistic models. For instance, the adaptive dynamics methodology can be extended to diploids, provided the initial frequency of mutant homozygotes is negligible (Kisdi and Geritz 1999; Van Dooren 1999, 2006; Metz 2008; Metz 2011; Metz and de Kovel 2013). For structured populations, extensions of inclusive fitness theory to account for dominance or multilocus genetics have been derived (Roze and Rousset 2003, 2008; Lehmann and Rousset 2014). The Price equation formalism has also been used to analyze multilocus epidemiological models (Day and Gandon 2012). However, a full synthesis currently remains out of reach. Nonetheless, it is encouraging to note that the methods that I have described in this synthesis are conceptually similar to those used to analyze multilocus models, which also rely on arguments of separation of timescales. This suggests that a tighter integration of these different fields may be technically possible with our current theoretical toolbox.

Conclusion

There are a diversity of approaches to modeling evolutionary dynamics in an explicit ecological context. In this synthesis, I have tried to highlight the connections between different schools of thought. Central to this perspective is the requirement that any eco-evolutionary model needs to incorporate an explicit dependence of vital rates on an environmental vector, thereby materializing the eco-evolutionary feedback loop. This requirement is central to adaptive dynamics theory but can usefully be adapted to produce ecologically explicit versions of the Price equation. This environmental feedback perspective forces us to adopt a mechanistic, rather than a phenomenological, view of evolution, which is based on population dynamical processes and is refreshingly close to Darwin's original and deeply ecological insight.

In most cases, analytical progress is possible only through additional methodological assumptions, generally taking the form of a separation of timescales. Typically, the aim is to decouple ecological and evolutionary timescales, either by focusing on the invasion of a rare mutant type or by assuming that mutations have small phenotypic effects (weak selection). Although this has proven to be a very successful toolbox to study long-term evolution, a key challenge for future studies is to take into account rapid or short-term transient evolutionary dynamics in realistic ecological scenarios. This has already motivated new approaches to model evolutionary epidemiology, based on the Price equation formalism I discuss above (Day and Proulx 2004; Day and Gandon 2006; Gandon and Day 2007). It is certainly possible to hope that the nascent dialogue between these different theoretical frameworks will foster new understanding of the tangled loop between ecology and evolution.

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APPENDIX

Separation of Timescales under Weak Selection

Under weak selection, the dynamics of the environment can be decoupled from the dynamics of the trait and from the change in frequency. To see why, let us write the per capita growth rate of type i as a function of the vector of traits and of the environment, $r_i(\mathbf{z}, \mathbf{E})$. We are interested in understanding the behavior of the coupled ecological and evolutionary system when all traits are close to the mean value. We thus write $z_i = \bar{z} + \varepsilon\delta_i$, where ε is a small parameter. To first order, we have

$$r_i(\mathbf{z}, \mathbf{E}) = r_i(\mathbf{z}^\circ, \mathbf{E}^\circ) + \varepsilon \frac{dr_i}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) + O(\varepsilon^2),$$

where $r_i(\mathbf{z}^\circ, \mathbf{E}^\circ)$ is the per capita growth rate of type i when $\varepsilon = 0$ (i.e., all types have trait value $\bar{z}$). Hence, $z^\circ = (\bar{z} \quad \dots \quad \bar{z})^\top$, and $\mathbf{E}^\circ$ is the neutral component of the environmental vector. The dynamics of the densities n_i are therefore described by a perturbation of the monomorphic dynamics when all types have the same trait value $\bar{z}$.

This allows us to calculate the dynamics of the total density n as

$$\begin{aligned}\frac{\mathrm{d}n}{\mathrm{d}t} &= \left(\sum_i r_i(\mathbf{z}, \mathbf{E}) f_i\right) n \\ &= \left[\sum_i f_i r_i(\mathbf{z}^\circ, \mathbf{E}^\circ) + \varepsilon f_i \frac{dr_i}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) + O(\varepsilon^2)\right] n \\ &= \bar{r}(\mathbf{z}^\circ, \mathbf{E}^\circ) n + \varepsilon \frac{d\bar{r}}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) n + O(\varepsilon^2).\end{aligned}$$

To zeroth order, the dynamics of the total density is given by the dynamics of the population when all individuals have the same value $\bar{z}$. A similar perturbation can be applied to the dynamics of the external variables, **e**, and we finally have

$$\frac{\mathrm{d}\mathbf{E}}{\mathrm{d}t} = \mathcal{F}^\circ(\mathbf{z}^\circ, \mathbf{E}^\circ) + O(\varepsilon).$$

The environmental dynamics are therefore $O(1)$.

We now Taylor expand the change in frequency as

$$\begin{aligned}\frac{\mathrm{d}f_i}{\mathrm{d}t} &= f_i(r_i(\mathbf{z}) - \bar{r}(\mathbf{z})) \\ &= f_i(r_i(\mathbf{z}^\circ, \mathbf{E}^\circ) - \bar{r}(\mathbf{z}^\circ, \mathbf{E}^\circ)) + \varepsilon f_i \frac{d(r_i - \bar{r})}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) \\ &\quad + O(\varepsilon^2).\end{aligned}$$

In a population where all types have the same trait value, we should not observe any frequency change. This requirement of neutrality implies that we have necessarily $r_i(\mathbf{z}^\circ, \mathbf{E}^\circ) = \bar{r}(\mathbf{z}^\circ, \mathbf{E}^\circ)$ and as a result

$$\frac{\mathrm{d}f_i}{\mathrm{d}t} = \varepsilon f_i \frac{d(r_i - \bar{r})}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) + O(\varepsilon^2).$$

The change of frequency is therefore $O(\varepsilon)$.

Finally, the change in mean trait is given by

$$\begin{aligned}\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} &= \sum_i z_i \frac{\mathrm{d}f_i}{\mathrm{d}t} \\ &= \sum_i \bar{z} \frac{\mathrm{d}f_i}{\mathrm{d}t} + \varepsilon \sum_i \delta_i \frac{\mathrm{d}f_i}{\mathrm{d}t}.\end{aligned}$$

The first term cancels out because $\sum_i f_i = 1$, and we have

$$\frac{\mathrm{d}\bar{z}}{\mathrm{d}t} = \varepsilon^2 \sum_i \delta_i f_i \frac{d(r_i - \bar{r})}{d\varepsilon}(\mathbf{z}^\circ, \mathbf{E}^\circ) + O(\varepsilon^3).$$

The dynamics of the mean trait is therefore $O(\varepsilon^2)$. This shows that under weak selection we can decouple the change in the environment (which is $O(1)$) from the change in frequencies and mean trait (which are $O(\varepsilon)$ and $O(\varepsilon^2)$, respectively).

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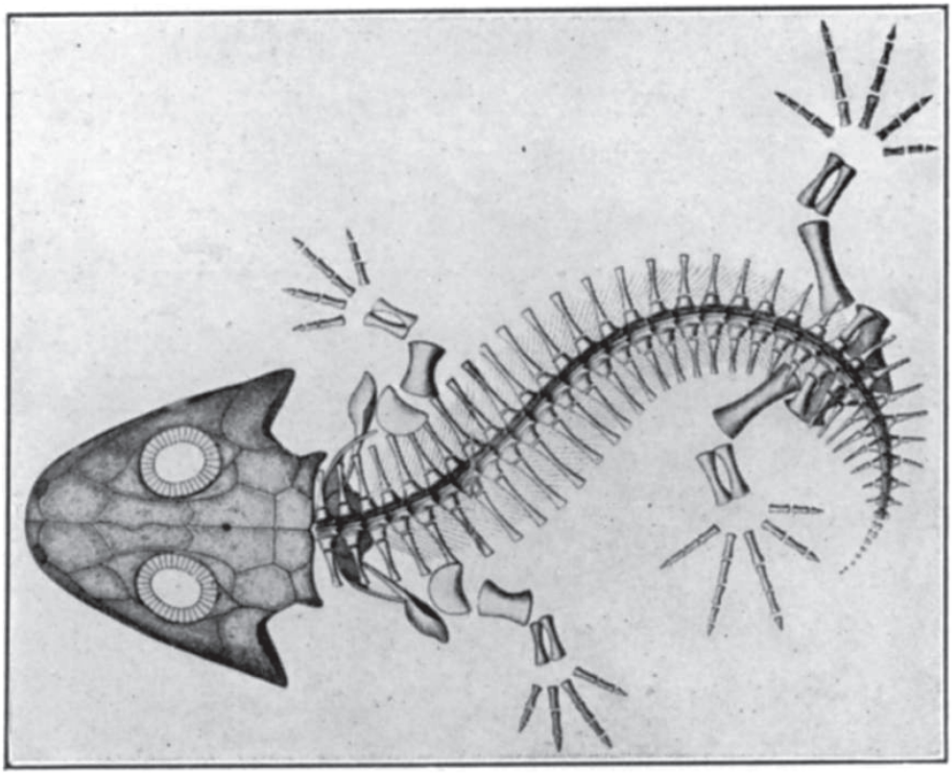

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