Does rapid evolution matter? Measuring the rate of contemporary evolution and its impacts on ecological dynamics

**Abstract**

Rapid contemporary evolution due to natural selection is common in the wild, but it remains uncertain whether its effects are an essential component of community and ecosystem structure and function. Previously we showed how to partition change in a population, community or ecosystem property into contributions from environmental and trait change, when trait change is entirely caused by evolution (Hairston et al. 2005). However, when substantial non-heritable trait change occurs (e.g. due to phenotypic plasticity or change in population structure) that approach can mis-estimate both contributions. Here, we demonstrate how to disentangle ecological impacts of evolution vs. non-heritable trait change by combining our previous approach with the Price Equation. This yields a three-way partitioning into effects of evolution, non-heritable phenotypic change and environment. We extend the approach to cases where ecological consequences of trait change are mediated through interspecific interactions. We analyse empirical examples involving fish, birds and zooplankton, finding that the proportional contribution of rapid evolution varies widely (even among different ecological properties affected by the same trait), and that rapid evolution can be important when it acts to oppose and mitigate phenotypic effects of environmental change. Paradoxically, rapid evolution may be most important when it is least evident.

**Keywords**

Contemporary evolution, countergradient selection, dynamic heterogeneity, evolutionary rates, phenotypic plasticity, Price Equation, rapid evolution.

**INTRODUCTION**

In the decades since Endler (1986) and Thompson (1998) we have learned that rapid contemporary evolution due to natural selection in the wild is widespread (Hendry & Kinnison 1999; Reznick & Ghalambor 2001; Schoener 2009). Underlying causes include invasion of new habitats, the rise and fall of natural enemies, resources or competitors, anthropogenic harvesting or habitat modifications and environmental variability on year-to-year or longer time scales (e.g. Hendry & Kinnison 1999; Kinnison & Hendry 2001; Reznick & Ghalambor 2001; Palumbi 2002; Svensson & Gosden 2007; Siepielski et al. 2009). In contrast, little is known about how important these rapid evolutionary changes are for ecosystem structure and function (Schoener 2011). If strong ecological interactions often lead to intense selection and rapid response, can ecologists safely continue to study local interactions and their consequences (Paine 2010) while ignoring rapid heritable changes in traits that determine the strength and outcome of interactions, or will we lose critical understanding by doing so?

Our goal in this paper is to present an approach to answering this question quantitatively. Previously (Hairston et al. 2005) we showed how to relate changes in an evolving trait $z$ (e.g. finch beak size) and an ecological or environmental variable $k$ (e.g. mean seed size) to an ecologically relevant response variable $X$ (e.g. finch per-capita population growth rate). Using the chain rule, the rate of change in $X$ can be expressed as a term proportional to $dz/dt$ plus a term proportional to $dk/dt$. The $dz/dt$ term is interpreted as the contribution of evolution to change in the response variable. Ezard et al. (2009) applied this approach to five ungulate populations, finding that in four of the five, variation in annual population growth was affected more by variation in individual size, a heritable trait, than by environmental variability.

However, our previous approach tacitly assumed that trait change was caused solely by evolution (Coulsdon & Tuljapurkar 2008; Ezard et al. 2009; Tuljapurkar et al. 2009), whereas traits also exhibit substantial non-heritable change (e.g. Bolker et al. 2003; Coulson & Tuljapurkar 2008; Osgul et al. 2009). Tallying all trait change as evolution leads to incorrect conclusions about the importance of ecological and evolutionary change when non-heritable trait change is substantial.

In this paper, we expand Hairston et al’s (2005) approach to account for non-heritable trait change, focusing on:

1. Phenotypic plasticity (effects of environment on trait expression), so that genotype determines a norm of reaction to environmental conditions rather than a fixed trait value.

2. Changes in population structure, e.g. age or size structure, with organism development resulting in non-heritable trait change.

We start with the simplest case, an unstructured population with phenotypic plasticity, in continuous time. We then build up to structured populations, discrete time (e.g. before vs. after an experimental manipulation or environmental change), and to cases where the ecological effects of trait change in the focal species are mediated through interactions with other species.

The approach here is a synthesis of our previous approach with a decomposition of trait change using the Price Equation (Price 1970,
It was inspired by recent, innovative applications and extensions of the Price Equation to decompose changes in a population, community or ecosystem attribute into contributions from multiple factors, including evolution (Fox 2006, 2010; Coulson & Tuljapurkar 2008; Fox & Harpole 2008; Collins & Gardner 2009; Ozgul et al. 2009). Our first contribution here is to extend those innovations to situations where interest centers on how trait change impacts an ecologically important response variable, rather than on trait change per se (Fig. 1), as in Hairston et al. (2005). Instead of asking ‘how much did evolution contribute to trait change?’ we ask ‘how large was the ecological impact of trait evolution?’ in the context of multiple factors that jointly determine the ecological response (Fig. 1).

We also extend previous approaches by considering indirect trait effects mediated through other ecosystem variables or species, for example if an herbivore’s trait affects a primary producers’ birth and death rates, thereby changing the producer’s abundance and ecosystem primary productivity.

Our second contribution is to demonstrate how the analyses can be done without having to model the selection process, and with more limited data than were available in many previous applications of the Price Equation. We present empirical studies of rapid evolution in: sexual vs. asexual reproduction in rotifers; juvenile growth rate in guppies. These examples also illustrate how our previous approach sometimes led to incorrect conclusions. The largest differences occur when evolution has compensated for effects of environmental change, such that environmental and evolutionary forces ‘pulling’ in opposite directions produce a small net phenotypic change, analogous to countergradient selection (Levins 1969; Conover & Present 1990; Conover & Schultz 1995). These examples show that rapid evolution can be important when it leads to stasis rather than change.

**OUR PREVIOUS APPROACH**

We consider an ecological response variable of interest, $X$, which (in the simplest setting) is a function of a heritable evolving trait $z(t)$ and an ecological variable $k(t)$ which change smoothly over time. By the chain rule,

$$\frac{dX}{dt} = \frac{\partial X}{\partial z} \frac{dz}{dt} + \frac{\partial X}{\partial k} \frac{dk}{dt}. \tag{1}$$

In Hairston et al. (2005) the first term on the right in eqn 1 was interpreted as the change in $X$ due to trait evolution, while the second term was interpreted as the change in $X$ due to ecological or environmental change. The relative absolute magnitude of the two terms, averaged over the time period of interest, measures the relative importance of ecological and evolutionary change for the response variable. Because evolution has in the past been assumed to occur slowly (e.g. Slobodkin 1961; Pianka 2000), we defined evolution to be ‘rapid’ if the ‘evolution’ term in eqn 1 approached or exceeded the ‘ecology’ term in magnitude.

In general, equations like (1) should be interpreted as referring to the underlying processes, rather than to values estimated from data. For example, differences in the estimated trait mean from one sample to the next will include random sampling error. Therefore, estimation of $\frac{dz}{dt}$ and similar terms should begin with smoothing the data to remove random variability unrelated to the processes being considered.

Two assumptions underlying eqn 1 should be noted:

1. The evolving trait is represented by a single quantity $z$, not by the distribution of trait values in the population. In effect, the response is assumed to be a function of the trait mean, $\bar{z}$.

2. All trait change is interpreted as evolution. The traits considered by Hairston et al. (2005) were all known to have high heritability, but this is not always the case.

Our goal in this paper is to get rid of the second assumption. We retain the first, assuming that any effects of changes in trait distribution are a function of the change in trait mean; we call this the ‘Population Mean Assumption’ (PMA). The PMA will hold if trait variation at any time is small and evolution results from strong selection on a small amount of heritable variation, as is often assumed in models for quantitative trait evolution (see Lande 1982). However, the PMA will also be a good approximation if all consequences of the trait are nearly linear functions of trait value within the range of variation at any time (Fig. 2).

We also assume that the trait mean can be predicted from the genotype distribution and the mean effect of other variables affecting trait expression. That is, the population must be large enough that we can neglect the deviation between the population mean and the theoretical trait mean in an infinite population with the same genotype distribution. The deviation will have standard deviation $\sqrt{V_p/N}$ where $N$ is population size and $V_p$ is the ‘environmental variance’ in the sense of quantitative genetics (the deviation between individual phenotype and breeding value), so the deviation should be negligible except in very small populations. We can therefore attribute any transmission bias to factors such as environmental change or demographic processes.

**PRICE EQUATION**

Price (1970, 1972) showed how changes in a quantitative trait $z$ due to selection could be expressed in terms of the covariance between trait value and fitness. Price (1970) noted that his equation applied to
The first term on the last line of eqn 3 is:

$$\sum_j p_i (m_i - \bar{m}) \dot{z}_j = \sum_j p_i m_i z_j - \bar{m} \sum_j p_i \dot{z}_j$$

$$= \bar{m}_m - \bar{m} \ddot{z} = \text{Cov}(m, z),$$

where the means and covariance are computed with respect to the probability distribution $p(t)$. The second term on the last line of eqn 3 is the average rate of change in the genotype-specific trait value, which we denote by $E[\Delta z]$. Substituting these into eqn 3 gives the continuous-time Price Equation:

$$\frac{d\ddot{z}}{dt} = \text{Cov}(m, z) + E[\Delta z].$$

Comparing eqn 3 and eqn 4 we see that $\text{Cov}(m, z)$ is the change in $\ddot{z}$ due to changes in genotype frequencies $p_i$, i.e. the change due to evolution. $E[\Delta z]$ is the change in $\ddot{z}$ due to changes in genotype-specific trait means, often called ‘transmission bias’ (especially when generations are non-overlapping; Frank 1997; Okasha 2006); it will generally be non-zero whenever trait change occurs without genetic change.

**GENOTYPE–PHENOTYPE–ENVIRONMENT EQUATION**

Combining the Price Equation with our previous approach, by substituting eqn 4 into eqn 1, gives an accounting of how genotype dynamics ($\text{Cov}(m, z)$), phenotype dynamics unrelated to genetic change ($E[\Delta z]$) and environmental dynamics $k(t)$ interactively affect the response variable $X$:

$$\frac{dX}{dt} = \frac{\partial X}{\partial \ddot{z}} \text{Cov}(m, z) + \frac{\partial X}{\partial \ddot{z}} E[\Delta z] + \frac{\partial X}{\partial k} \frac{dk}{dt}.$$  

$$\text{Cov}(m, z)$$ does not actually need to be evaluated, because we can equivalently write

$$\frac{dX}{dt} = \frac{\partial X}{\partial \ddot{z}} \left( \frac{d\ddot{z}}{dt} - E[\Delta z] \right) + \frac{\partial X}{\partial \ddot{z}} E[\Delta z] + \frac{\partial X}{\partial k} \frac{dk}{dt}.$$  

We will refer to eqn 5 and eqn 6, which separate change in $X$ into components caused by genotype, phenotype and environmental change, as the GPE equation. The first term on the right-hand side of both equations is the effect of gene frequency change on $X$; the last term on the right-hand side is the direct effect of $k$ on $X$. The interpretation of the middle term, involving transmission bias, depends on the situation. Transmission bias that occurs because $k$ affects trait expression would be an ‘effect of ecology’. If transmission bias is strictly the result of endogenous dynamics (e.g. changes in age structure), or imposed on individuals by an external factor (disease, partial predation, etc.), it might instead be regarded as a third distinct factor. Purely random transmission bias is possible in small populations.

Although eqn 5 and eqn 6 are mathematically equivalent, they differ in an important way. Equation 5 is prospective, predicting how $X$ will change by combining the processes driving change in $\ddot{z}$ eqn 6 is retrospective, with the trait dynamics $d\ddot{z}/dt$ a ‘given’ and the selection-driven component computed by subtracting off the transmission bias. As a result, eqn 6 can be used without modelling selection, and without data on how trait values were correlated with realized fitness components (of the sort used by Coulson & Tuljapurkar 2008 and Ozgul et al. 2009). This is especially important when the response $X$...
is affected by several evolving traits (possibly in several different species). The Price Equation still applies to each trait separately, because it does not require any assumptions about the cause of fitness differentials. With multiple traits the selection and transmission bias terms in eqn 5 and eqn 6 are just replaced by sums of those terms over all traits, as in eqn. 3 of Hairston et al. (2005); the same is true if multiple environmental factors affect X. But prospective analysis with eqn 5 has the complication that calculation of \( \text{Cor}(u, z) \) has to include correlated selection, e.g. a trait may correlate with fitness only because it is genetically correlated with a functionally unrelated trait that is undergoing selection. For these reasons, eqn 6 will usually be preferable for analysing data on trait evolution and its effects.

However, eqn 5 can be used for forecasting if \( \text{Cor}(u, z) \) can be calculated from a mechanistic selection model, which may be useful for integrating evolutionary and ecological components of predicted responses to environmental change (e.g. prospective versions of the retrospective analyses in Collins & Gardner 2009). Equation 5 also provides one way of incorporating the dynamics of species- or genotype-specific trait values into trait-based approaches to community assembly and function (e.g. Shipley 2009, 2010; Webb et al. 2010).

The rest of this paper focuses on demonstrating how eqn 6 can be applied to real data (Table 1). We concentrate on cases where transmission bias occurs due to phenotypic plasticity or demographic processes. Under the PMA (Fig. 2), the transmission bias equals the rate of change in trait mean for a typical individual, whose trait value equals the population mean. We also discuss two situations in which eqn 6 must be modified: first, if data are only available on the net change over an extended period of time, rather than on short-term rates of change; second, if the trait’s effect on X is indirect and mediated through effects on the dynamics of other components of the community or ecosystem.

### Table 1

<table>
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<th>Link</th>
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<td>( g \rightarrow z )</td>
<td>Heritable component of trait variation</td>
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<tr>
<td>( k \rightarrow z )</td>
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<td>( z \rightarrow X )</td>
<td>Direct effect of trait change on ecological response</td>
<td>Peak dimensions → population growth rate in finch, Geospiza fortis (H05)</td>
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<td>( k \rightarrow X )</td>
<td>Direct effect on ecological response of change in environmental conditions or ecological variables</td>
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<tr>
<td>( s \rightarrow X )</td>
<td>Direct effect of individual-level dynamic state variable on ecological response</td>
<td>Tree size and height diversity → productivity of spruce forest stands (Lei et al. 2009)</td>
<td>O</td>
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<tr>
<td>( z \rightarrow y )</td>
<td>Direct effect of trait change on other species, or other community or ecosystem processes</td>
<td>Guppy phenotype → abundance of algae, invertebrates, etc</td>
<td>E</td>
</tr>
<tr>
<td>( y \rightarrow X )</td>
<td>Direct effect of intermediate variables on ecological response</td>
<td>Changes in community composition resulting from guppy evolution → net primary productivity, respiration rate, etc</td>
<td>E</td>
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For the response variable X we choose the per-capita rate of amictic offspring production by amictic females; this is the birth rate that determines rotifer population growth, because mictic daughters produce only males which have zero fitness. The ecological variable k affecting X is C, the abundance of the rotifers' algal prey. We have \( X(\xi, C) = (1 - \xi)F(C) \), where F is the per-capita fecundity of amictic females as a function of C.

Trait values were not directly observable because mictic and amictic females are morphologically indistinguishable (Fussmann and et al. 2003). Instead, trait dynamics were inferred by fitting a differential equation model for the dynamics of a and of the algal and rotifer abundances to the available data: the total abundance of algae and rotifers, and the numbers of rotifers carrying mictic vs. amictic eggs. Combining the fitted a(t) and rotifer fecundity function F, with the experimental data on R and C (Fig. 1, left column), gives estimates for \( \xi(t) = a(t)R(t) \) and \( X(t) = (1 - \xi(t))F(C(t)) \).

Next we compute the transmission bias \( E[\Delta x] \), which is the rate at which the trait mean would change if genotype frequencies were held constant. With \( \xi = aR \) and genotype determining a, we have \( E[\Delta x] = a \frac{dx}{dt} \). The contribution of evolution to the change in X is therefore

\[
\frac{\partial X}{\partial \xi} \left( \frac{dx}{dt} - a \frac{dR}{dt} \right) = -F(C) \left( \frac{dx}{dt} - a \frac{dR}{dt} \right) = -F(C)R \frac{da}{dt}.
\]

In this case the transmission bias is plasticity driven by an ecological factor, the change in rotifer abundance. Therefore, eqn 7 is the only evolutionary contribution to the change in X, and the ecological contribution can be computed as \( \frac{dx}{dt} - a \frac{dR}{dt} \) minus the value of eqn 7. The response and the transmission bias are both linear functions of \( \xi \) (for all else held constant), so the PMA holds.

Figure 3 shows the population data and resulting estimates of evolutionary and ecological contributions to change in X for the four experimental replicates, using the data on changes in C and R, the inferred changes in a, and the fitted fecundity F. Even though the experimental design created very rapid ecological dynamics following rotifer introduction, the contribution of evolution to changes in rotifer per-capita fecundity was roughly 25% as large as the effects of the ecological dynamics (EVO : ECO = 0.25). The turbulence driving selection against mixis is always present, but the selection is weak when rotifer density is very low (because the trait is not expressed then), or when food is too scarce for much reproduction to occur. The contribution of evolution was therefore small at the start of each replicate because rotifer densities were low, large while rotifers reproduced rapidly and reached high density, and small again when food and rotifer densities declined.

Plasticity is central to this example, because mixis is a plastic response and evolution results from genetic variation in the degree of plasticity. If the importance of evolution were estimated by our previous approach which ignored trait plasticity, the results would be very small again when food and rotifer densities declined.

The role of evolution in all replicates was to partially counteract the maladaptive plastic response to increased rotifer density, an increase in mixis. Although the plastic response was dominant (ECO > EVO), in all replicates the evolution of a reduced propensity to mixis was fast and large enough to temporarily reverse the decline in rotifer numbers as their food supply was exhausted.

**STRUCTURED POPULATIONS AND DYNAMIC HETEROGENEITY**

Another widespread cause of non-heritable trait change, whose importance has recently been emphasized (Coulson & Tuljapurkar 2008; Ozgul et al. 2009; Tuljapurkar et al. 2009), is dynamic heterogeneity: changes in trait distributions caused by the endogenous dynamics of population structure. For example, non-heritable changes in a population’s size structure will inevitably occur due to development, reproductive allocation, and changes in resource availability or other environmental factors, even without changes in allele frequency at loci affecting growth trajectories.

Equation 6 still applies to trait changes caused by dynamic heterogeneity (either alone or in combination with plasticity), but to use it we first need to describe how to compute the transmission bias. We assume that individuals are characterized by two things:

1. A non-heritable attribute s that we will call ‘size’, though it could be any attribute that changes during an individual’s life (or some property of the local environment affecting a plant or sessile animal).
2. A heritable trait b, which could be a genotype index (i.e. \( b = 1, 2, 3, \ldots, m \) for the m genotypes in the population), or a quantitative character such as specific metabolic rate.

An individual’s value of the focal trait \( \xi \) is assumed to be a function of \( b, s, \) and environmental variable \( k \), i.e. \( \xi = F(b, s, k) \), so the trait has both size-dependence and plasticity in response to \( k \). The population state is specified by the distribution function \( n(b, s, t) \) giving the abundance of \( (b, s, t) \)-type individuals at time \( t \).

A general expression for the transmission bias of any one genotype can be derived by applying the Price Equation to individuals classified by size rather than by genotype. To simplify notation, we drop the genotype index \( b \), and write \( \xi(s, t) \) for \( \xi(s, k(t)) \). Let \( m(s, t) = B(s, t) - D(s, t) \), where \( B \) and \( D \) are the per-capita (state-dependent) birth and death rates, respectively, at time \( t \). The result (derived in Appendix S1) is

\[
E[\Delta x] = C \omega_C(\mu, \xi) + E_i[\Delta x] + E_i[B(s, t)(\xi(s, t) - \xi(s, t))]
\]

where the subscript \( s \) indicates that the covariance and expectation are computed with respect to the size-frequency distribution.

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Equation 9 expresses one transmission bias in terms of another. The gain from this is that the terms on the right-hand side of eqn 9 correspond to distinct processes and are straightforward to calculate from the size dynamics. The first term is the change in the population trait mean due to size-dependent differential survival and reproduction, and the last term is change due to trait differences between parents and offspring. $Es[Dz]$ is the rate of change in trait mean among survivors, and $z_0 (s, t)$ is the trait mean in offspring of size-$s$ individuals at time $t$.

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Two important features of eqn 9 are first, that it applies regardless of why or how the size distribution changes, and second, that it involves quantities that can be estimated directly from suitable data: the size-dependent rates of birth, death and change in trait value.
individuals due to dynamic heterogeneity (e.g. natural growth as individuals age) and to plasticity in response to environmental change.

Consider, for example, a population where size-$s$ individuals grow deterministically at rate $g(s, t)$. The trait value of a survivor then changes at rate $\frac{d}{dt}E[g(s, t)] + \frac{d}{dt}\frac{\partial}{\partial g}E[g(s, t)]$, so

$$E;[\Delta z] = \int \left( \frac{\partial E[g(s, t)]}{\partial s} + \frac{\partial}{\partial k} \frac{\partial E[g(s, t)]}{\partial k} \right) p(s, t) ds. $$

The first term inside the integral is the change in mean trait due to individual growth, and the second is the change due to phenotypic plasticity.

Ideally, transmission bias would be computed separately for each genotype $b$ using genotype-specific vital rates, and then averaged according to current genotype frequencies. But under the PMA (Fig. 2), we can just evaluate the transmission bias for a ‘typical’ individual, by fitting demographic models in which individual growth, survival, and fecundity depend on individual size but not on genotype. Transmission bias calculated from those rates using the methods in this section approximates the average genotype-specific transmission bias.

**DISCRETE TIME: COMPARING ENDPOINTS INSTEAD OF INSTANTANEOUS RATES**

So far we have considered instantaneous rates of change in the trait, environment and response, but in many cases it will be necessary or preferable to consider the amount of change that occurs between the endpoints of a time interval:

1. Experiments typically measure the outcomes of different treatments rather than following processes in continuous time, and continuous monitoring is often not feasible.
2. If measurements are strongly affected by sampling variability, it may be more accurate to estimate long-term trends and total effects over an extended observation period.
3. For species with non-overlapping generations or from highly seasonal environments, it is more natural to measure the effects of evolution between one year (or generation) and the next.

Extending our analysis to discrete-time data is therefore important for empirical applications. In addition, comparison of ‘before’ and ‘after’ is conceptually the same as comparison of ‘here’ and ‘there’ (two spatial locations), so the same analysis can be used to evaluate the importance of local adaptation.

In continuous-time analyses there are no $\xi \times k$ interaction terms, which would show up as mixed second-order partial derivatives in eqns 1, 5 and 6. The reason for this is that over a short time period $dt$, the changes in $\xi$ and $k$, and their separate main effects on $X_s$ are both proportional to $dt$, so their interaction is proportional to $(dt)^2$. In the continuous-time limit $dt \to 0$ the interactions therefore become vanishingly small compared to the main effects.

But in discrete time, $dt$ is not small and we need to account for $\xi \times k$ interactions. Here, we show how our previous approach can be extended to account for non-heritable trait change. In Appendix S2 we discuss how our approach relates to Okasha’s discussion of causal interpretations of the Price Equation [Okasha (2006), sec. 1.4].

The basis for our approach is ANOVA, or equivalently using a linear model to estimate the main effects of changes in $\xi$ and $k$. Returning for simplicity to an unstructured population where transmission bias occurs due to phenotypic plasticity, consider a change from genetic state $p_1$ and ecological state $k_1$ at time $t_1$, to genetic state $p_2$ and ecological state $k_2$ at time $t_2$. Let $F$ denote the norm of reaction that gives $\xi$ as a function of the genetic and ecological states, and define $\tilde{\xi}_g = F(p, k), X_g = X(\tilde{\xi}_g, k)$, the trait mean and response variables resulting from genetic state $\xi$ in environment state $k$. Define indicator variables $\tilde{p}, \tilde{k}$ each having the value 0 or 1 corresponding to times $t_1$ and $t_2$, respectively.

To estimate the main effects of ecological and environmental change, we fit a linear model

$$X = x_0 + x_p \tilde{p} + x_k \tilde{k} + \text{error}$$

so that $x_p$ is the main effect of change in $p$, $x_k$ is the main effect of change in $k$. Alternatively, an interaction term can be estimated by fitting

$$X = x_0 + x_p \tilde{p} + x_k \tilde{k} + y \tilde{p} \tilde{k} + \text{error}. $$

Because an interaction term is omitted in eqn 10, the fitted main effects of genotype and environment are averages over the ‘background’ values of the other factor. By solving the least-squares normal equations for the model coefficients, we find that eqn 10 divides the interaction term evenly (half to ‘evolution’, half to ‘ecology’), and that the resulting contributions of evolution and ecology are

Evolution:

$$\frac{1}{2}[(X_{g2} - X_{g1}) + (X_{s2} - X_{s1})].$$

Ecology:

$$\frac{1}{2}[(X_{s2} - X_{s1}) + (X_{g2} - X_{g1})].$$

In the Evolution line of eqn 12, the effect of changing only genetic state is calculated for environment states 1 and 2, and then averaged. These are the changes that would occur with transmission bias removed (because transmission bias here is entirely due to changes in $k$), so they correspond to the first term on the right-hand side of eqn 6. In the ‘Ecology’ line of eqn 12, the effect of changing only environment state is calculated for genetic states 1 and 2, and then averaged. These effects include both the indirect effect of $k$ through its effect on the trait, corresponding to the second term on the right-hand side of eqn 6, and the direct effect of $k$, corresponding to the last term on the right-hand side of eqn 6. So although eqn 12 looks nothing like eqn 6, in concept it is exactly the same.

Equation 12 is identical in form to eqn 8 in Hairston *et al.* (2005); the difference is in how the response values $X_g$ are defined. Here, we assume that the trait is jointly determined by genotype and environment through the reaction norm $F$, so eqn 12 involves four different trait means resulting from all possible combinations of genetic states and environment states. Hairston *et al.* (2005) did not consider phenotypic plasticity, so they arrived instead at eqn 12 with response values $X_g = X(\tilde{\xi}_g, k), where tilde{\xi}_g = \tilde{\xi}$ is the observed trait at time $i$. Thus, their calculations involve only the two trait means that were actually observed at the initial and final times.

The four trait values needed to compute eqn 12 raise the data requirements relative to Hairston *et al.* (2005). Based on the traits observed in particular environmental conditions, we need to infer what the traits of those same individuals would have been under other conditions. Exactly the same is true in continuous time, in order to estimate the transmission bias. In the rotifer mix example (above), this was done using the empirically supported relationship between the trait value and environmental conditions, i.e. $\xi = aR$. Observing mean trait value $z_1$ at rotifer density $R_1$, we can infer that the same genotypes experiencing rotifer density $R_2$ would instead have mean trait value $z_2R_2/R_1$. 

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Example: rapid evolution of juvenile growth rate in Daphnia

Hairston et al. (1999, 2001) studied how the juvenile growth rate of Daphnia galeata in Lake Constance evolved in response to a period of eutrophication during the 1960s and 1970s. Eutrophication caused an increase in the abundance of cyanobacteria, which are poor quality food for Daphnia, or even toxic at high concentration. Genotypes from before and after the onset of eutrophication were obtained by hatching dormant eggs buried in different sediment strata. These were assessed in the laboratory for juvenile growth rates on ‘good’ and ‘poor’ food sources (i.e. without or with cyanobacteria respectively) that were formulated to represent ambient food quality before and during the period of eutrophication. Experiments showed that juvenile growth rate and food quality affect adult weight at first reproduction, which is ecologically important because body size determines both clutch size and the risk of predation by fish.

For this example we take adult body mass as our response variable, juvenile growth rate as the evolving trait affected by genotype and environment, and food quality as the dynamic ecological variable. The reaction norm $F$ is juvenile growth rate as a function of genotype and food quality. The experimental data on genotype-specific juvenile growth rates on each food type (Fig. 4a) allow us to compute the mean juvenile growth rate for the four possible combinations of ‘before’ ($p$) and ‘after’ ($p’$) genotypes with ‘good’ ($k$) or ‘poor’ ($k’$) food. These values, and the fitted models for adult mass in relation to juvenile growth rate and food quality, allow us to calculate the response values $X_{ij}$ (Fig. 4b) and evaluate eqn 12. This gives

\[
\text{Evolution: } +4.0 \mu g \ (\pm 1.5 \mu g) \\
\text{Ecology: } -11.9 \mu g \ (\pm 1.5 \mu g) \\
\text{Evolution/Total: } 24\% \ (\pm 7.5\%)
\]

so that the estimated evolutionary contribution is 1/3 as large as the estimated ecological contribution (values in parentheses are bootstrap standard deviations, obtained by repeating the calculations for 10 000 bootstrap samples from the set of sampled genotypes; Total is the summed absolute values of Evolution and Ecology effects).

As expected, the ecological contribution is negative – poorer food reduced juvenile growth and adult size, while the contribution from evolution is positive, offsetting a third of the effect of food quality on adult size. Alternatively, the three-way partition using eqn 11 gives

\[
\text{Evolution: } +3.8 \mu g \ (\pm 1.6 \mu g) \\
\text{Ecology: } -12.1 \mu g \ (\pm 1.4 \mu g) \\
\text{Interaction: } +0.3 \mu g \ (\pm 0.3 \mu g)
\]

We can compare these to a calculation using the Hairston et al. (2005) method that ignores phenotypic plasticity, which gives:

\[
\text{Evolution: } -1.1 \mu g \ (\pm 1.2 \mu g) \\
\text{Ecology: } -6.9 \mu g \ (\pm 1.9 \mu g)
\]

In this calculation the slower juvenile growth during the period of eutrophication is ascribed entirely to evolution, which yields a small negative contribution of evolution to adult size. In contrast, eqn 13 correctly accounts for the fact that evolution of the reaction norm for juvenile growth rate partially counteracted the decrease in food quality, giving a larger, positive contribution of evolution.

Note that the sum of contributions is the same for all decompositions, because the sum always equals the change in response. So as in the rotifer mixis example, the difference between our current analysis and Hairston et al. (2005) is that a component of the total change shifts from one category to another. But unlike the rotifer mixis example, in this case accounting for plasticity leads to a much higher estimate for the importance of evolution.

Example: fledging mass in great tits

Another example, with many similarities, is provided by Garant et al. (2004) who studied trends in fledging mass of great tits (Parus major)

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**Figure 4** Illustration of the process used to determine the contribution of evolution to changes in adult size of Daphnia galeata in Lake Constance following eutrophication. (a) Juvenile growth rates (day$^{-1}$) of sediment-derived genotypes from times before or during the period of eutrophication, when provided with good or poor quality food (‘before’ genotypes: green triangles; ‘during’ genotypes: red circles). Data replotted from Hairston et al. (2001). (b) The four values of the response variable (adult weight) needed to compute the ecological and evolutionary contributions to the changes in the response variable. The dashed curves are the fitted model for adult weight as a function of juvenile growth rate, for individuals provided with poor quality (black curve) or good quality food (blue curve). The fitted models are linear regressions of log10 adult body mass on log10 juvenile growth rate with intercept depending on food quality, fitted to the data on adult weight vs. juvenile growth rate plotted in Figure 6 of Hairston et al. (2001). The plotted symbols are the predicted mean adult body size for ‘before’ and ‘during’ genotypes [symbols as in panel (a)], growing with poor or good quality food, based on their mean juvenile growth rates in each food quality environment. For these calculations, and those reported in the text, the response (mean adult weight) is computed for the mean trait values (juvenile growth rate as a function of food type) of the assayed genotypes from each time period (before: $n = 12$, during: $n = 10$). Nearly identical results are obtained if instead the response is computed separately for each genotype, and the responses are then averaged, which confirms that the Population Mean Assumption is valid in this case (this is also evident from the near-linearity of the fitted response curves).
between 1965 and 2000, and its effects on fledgling survival. Some methods used by Garant et al. (2004) have recently been critiqued by Hadfield et al. (2010), but the re-analysis by Hadfield et al. (2010) supports the trends from the original analysis that we discuss here. We use fledgling survival as our response variable $X_t$ and fledging mass as the evolving trait. Garant et al. (2004) found that fledgling survival was affected both by the individual’s fledgling body mass $m$, and by its breeding value for body mass, $b$ (fledging body mass is actually ‘the residual mass from a general linear model including lay date, clutch size and egg weight’, and breeding value was estimated by pedigree analysis). The independent effects of $b$ and $m$ suggest that $b$ is, in part, an indicator for many factors affecting viability. The deviation between actual mass and breeding value was explained in part by environmental factors including the density of the breeding bird population and temperatures in the current and preceding spring, the remainder being unexplained variation.

Using their published data on annual fledgling survival (their Table 1), mean fledging mass and mean breeding value (their Figs 2 and 3), we fitted a logistic regression model for fledgling survival as a function of fledging mass and breeding value. Linear trends in mean fledging mass and estimated breeding value for the period 1965–2000 (shown in their Figs 2 and 3) are

$$\dot{b} = 0.026 + 0.00192(t - 1965)$$
$$\dot{m} = 0.21 - 0.0096(t - 1965)$$

Combining these gives the overall trend in the ‘environmental deviation’ $e = b - m$, which is caused partially by increases in the density of breeding birds (50% increase between 1965 and 1970) and spring temperatures (15% increase in the temperature index (sum of daily maximum temperature from March 1 to April 25)). These trends smooth over year-to-year variation in the explanatory variables and unexplained variation, so the calculations here compare the contributions of the overall evolutionary and ecological trends to fledgling survival.

We can now apply eqn 12 with $t_1 = 1965$ and $t_2 = 2000$ as the initial and final times, and the response values $X_{i,j}$ being the survival of a fledgling with fledging mass $m_{ij} = b_i + e_i$ and breeding value $b_i$ predicted by our logistic regression model. The response function is very close to linear over the range of the data [adjusted $R^2 > 0.99$ for 1000 randomly generated points in the rectangle defined by the ranges of eqn 16], so the PMA is valid. The results are

$$\text{Evolution: } +4.0\% \pm 1.5\%$$
$$\text{Ecology: } -2.4\% \pm 1.5\%$$
$$\text{Evolution/Total: } 62\% \pm 18\%$$

(values in parentheses are bootstrap standard deviations based on 10,000 bootstrap samples from the set of study years). As in the Lake Constance example, evolution acted to oppose the effects of declining environmental quality (more competitors, higher temperature). The relatively small total change in survival again represents the balance between two larger but opposing forces, with evolution making the larger contribution in this case.

**INDIRECT EFFECTS OF TRAIT CHANGE**

So far we have assumed a direct and immediate link between the trait $\xi$ and the response variable $X$, but this may not always be true: the link may pass through intermediate variables, and the effect of trait change may be delayed. For example, traits affecting a consumer’s ability to capture and consume different types of prey will affect the relative abundance of prey types, which in turn could determine a community-level response variable (e.g. total biomass, total N, or C fixation rate).

Immediately acting indirect effects can be addressed much like direct effects (A. Hendry, personal communication). ‘Immediate’ means in practice that intermediate variables respond fast enough to trait change that their measured values predominantly reflect the trait value at the same sampling time. For example, suppose that the response variable $X$ is directly affected by a trait $\xi$ with phenotypic plasticity, and also by the abundance $N$ of some other species which is affected by $\xi = X(N(\xi), \gamma_k)$. So we still have $X = F(\xi, k)$, with the additional ability to separate out direct and indirect effects of evolution and plasticity:

$$\frac{dX}{dt} = \frac{\partial X_d}{\partial X} \frac{dN}{dt} + \frac{\partial X_d}{\partial X} \frac{d\xi}{dt} + \frac{\partial X_d}{\partial k} \frac{dk}{dt}$$

The first two terms on the right-hand side of eqn 18 are the direct and indirect effects of trait change, and by expanding $d\xi/dt$ using the Price Equation we can separate these into contributions from evolution vs. plasticity.

But to reach eqn 18 we had to assume an immediate effect of the trait on the level of the intermediate variable $N$. A different approach is needed when the trait affects intermediate variables by altering the rates of processes that affect their dynamics. That is, the response variable is a function of ecosystem state, while the trait affects ecosystem dynamics. A general continuous-time model for this situation is

$$X = X(y, k)$$

where $y$ is the ecosystem state vector. Then

$$\frac{dy}{dt} = f(y, \xi, k)$$

(or appropriate sums of such terms if $y$ or $k$ are vectors), so $d\xi/dt$ does not directly affect the instantaneous rate of change in $X$. Therefore, effects of evolution involving delayed indirect effects always have to be evaluated in terms of net effects over an extended period of time, rather than through an equation like (18).

As in our previous discrete-time analysis, we assume an unstructured population with phenotypic plasticity, with $\xi = F(p, k)$ denoting the trait’s reaction norm as a function of genotype and environment. We again consider a change from genetic state $p_1$, environment state $k_1$ at time $t_1$, to genetic state $p_2$, environment state $k_2$ at time $t_2$. In this case, however, change in $y$ and therefore in $X$ is expected to occur even without any evolutionary or environmental change. The baseline for evaluating ecological and environmental contributions to change in $X$ is therefore not $X(t_1)$, but the value of $X(t_2)$ that would have resulted from ecosystem dynamics with $p$ and $k$ held constant.

As before, we can estimate the main effects of evolutionary and ecological change by fitting a linear model. Define $X_{2,i}$ to be the value of $X$ at the final time $t_2$, with $i = 1$ if the genetic state $p$ is allowed to proceed along its observed time-course between $t_1$ and $t_2$, and $i = 1$ if evolution is blocked so that $p$ remains ‘frozen’ at $p_1$. Similarly, let $j = 1$ or 2 according to whether $k$ is held fixed at $k_1$ or allowed to proceed to $k_2$. $X_{2,2}$ is therefore the observed value of $X$ at the final
time, but all other $X_j$ are counterfactuals. For example, $X_{11}$ is calculated from
\[ \frac{dy}{dt} = f(y, F(p_1, k_1), k_1), \quad y(t_1) = y_1 \\
X_{11} = X(y(t_2), k_1). \tag{20} \]

To compute $X_{21}$, $p_1$ in eqn 20 is replaced by $p(t)$ or the best available approximation (e.g., a line connecting initial and final values if all intermediate information is lacking). To compute $X_{21}$, $k_1$ is replaced by $k(t)$ or the best available approximation. Fitting the linear model (eqn 10) without interaction then estimates the main effects of allowing evolution to proceed, and of allowing ecological change to proceed. The result is again given by eqn 12 using the definitions of the $X_j$ stated above for this case.

**Ecosystem-level indirect effects of predator-induced evolution in guppies**

Bassar et al. (2010) used mesocosm experiments to quantify how local adaptation of Trinidadian guppies to different levels of predation altered several ecosystem-level properties including algal and invertebrate biomass, benthic organic matter, primary productivity, and biogeochemical fluxes. Exposure to predators had (at least) three effects on the guppy population: rapid evolution from low-predation (LP) to high-predation (HP) phenotypes; a roughly twofold reduction in guppy density (number of individuals per m$^2$); and a change in population size structure causing an additional roughly twofold decrease in guppy biomass (Bassar et al. 2010). Bassar et al. (2010) varied guppy phenotype and abundance independently while holding size-structure constant. This design provides the information needed to estimate the relative impacts of the ecological variable guppy population density and the heritable guppy phenotype on the measured ecosystem-level properties. For this example, we consider the experiment as mimicking the consequences of predator introduction, which causes a change from high guppy density ($k_1$) and LP phenotypes ($p_2$) to low guppy density ($k_2$) and HP guppy phenotypes ($p_2$).

We used eqn 12 to compute the ecological and evolutionary contributions to changes in all ecosystem properties measured by Bassar et al. (2010), using the mean values for each density-phenotype combination (obtained from their Supplementary Table 6). Doing so presumes that the PMA is valid because LP and HP phenotypes are distinctly different, with small within-type variability relative to the difference in means.

Half of the ecosystem response variables were affected more by guppy evolution than by the change in guppy density (Fig. 5). In some cases the evolutionary contribution was substantially larger, and in the opposite direction from the ecological contribution. These calculations provide further support for Bassar et al.’s conclusion that evolution on ecological timescales can significantly affect important ecosystem properties.

Some caveats should be noted, however. First, Bassar et al. (2010) conducted their experiments at roughly double the observed guppy densities in both habitats, which would exaggerate density effects. We also don’t know to what extent changes in guppy density reflect life-history evolution rather than elevated predation, so treating density as a purely ecological factor may also over-estimate the ecological contribution. On the other hand, holding size-structure constant reduced the ecological consequences of predators. Second, guppy reproduction during the experiment increased the variation in guppy numbers and biomass among treatments. Third, Bassar et al. (2010) initiated their mesocosms with LP and HP guppies from streams, so the actual time-scale of ecosystem response to predator introduction would be lengthened by the time required for guppy evolution. Many of these issues are consequences of using mesocosms rather than manipulations within streams, with the benefits of replication and control but unavoidably some loss of realism.

**DISCUSSION**

This paper could have been much longer. We have not discussed dynamic heterogeneity in discrete time, or in combination with phenotypic plasticity (Coulson & Tuljapurkar 2008; Ozgul et al. 2009). We have not considered situations where the response variables are affected by evolution in multiple traits, by multiple environmental factors that change over time, or by the many possible interactions amongst these. You can probably think of some other things that we...
left out. What we have done, following the lead of recent applications of the Price Equation to trait dynamics (Fox 2006; Coulson & Tuljapurkar 2008; Collins & Gardner 2009; Ozgul et al. 2009), is develop a versatile extension of our previous approach, and illustrate how to apply it in theory and in practice to situations where ecologically important variables are jointly determined by trait and environmental variability (Table 1). However, identifying which traits and environmental variables are important, and how genotype and environment combine to determine trait expression, depends on each investigators’ intimate understanding of the natural history of their study system. Each will have its own strong interactions, and we cannot provide an all-encompassing recipe.

We now know that traits can exhibit rapid, heritable change in the wild, due to changes in the strength or direction of selection (see Introduction). We also know that trait-mediated effects on ecological interactions often equal or exceed in importance the abundance-mediated interactions that have classically been the focus of population and community ecology (Bolker et al. 2003). Putting those together, there is every reason to believe that rapid evolution of traits mediating ecological interactions may be an essential aspect of how ecosystems function. Our case-studies have revealed an intriguing additional reason: the effect of rapid evolution may be important when it acts to oppose and reduce the effects of environmental change on traits, a temporal analogue of 'countergradient selection' in response to spatial environmental gradients (Levins 1969; Conover & Present 1990; Conover & Schultz 1995). These findings suggest that, paradoxically, rapid evolution may be most important when it is least evident. If so, rapid evolution may be even more common than we presently imagine, and a key to understanding how species and communities persist in the face of constant change in their biotic and abiotic environments.

As Schoener (2011) has emphasized, however, these are still hypotheses, and we are short on hard data. Getting these data for natural systems won’t be quick and easy. We need to track trait evolution and then (to evaluate its importance) create the counterfactual in which traits had not evolved, either by experimental manipulations, mechanistic modelling, or analysis of long-term data (Hast, N.G., Jr, Lampert, W., Caceres, C.E., Holtmeier, C.L., Weider, L.J., Fischer, J.M. & Hairston, N.G. (1999). Perspective: the pace of modern life: measuring rates of contemporary microevolution. Evolution, 53, 1637–1653.

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REFERENCES


SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Transmission bias for structured-population models.

Appendix S2 Ecology–evolution interactions and causal analysis of the Price Equation.

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