Review

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The concept of fitness in fluctuating environments

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Fitness is a central concept in evolutionary biology, but there is no unified definition. We review recent theoretical developments showing that including fluctuating environments and density dependence has important implications for how differences among phenotypes in their contributions to future generations should be quantified. The rate of phenotypic evolution will vary through time because of environmental stochasticity. Density dependence may produce fluctuating selection for large growth rates at low densities but for larger carrying capacities when population sizes are large. In general, including ecologically realistic assumptions when defining the concept of fitness is crucial for estimating the potential of evolutionary rescue of populations affected by environmental perturbations such as climate change.

Does ecology really matter?

A recent central focus in evolutionary biology has been to link phenotypic evolution acting at longer time-scales to current ecological processes [1]. These advances have also been motivated by the question of the extent to which populations subject to stress caused by various types of human activities can be rescued by rapidly evolving new adaptations [2]. However, a problem in these analyses has been that many classical models in population genetics typically are based on simplifying assumptions such as constant population size and a stable environment, or at least constant relative fitnesses. These assumptions seriously restrict the applicability of classical models to the accurate description of evolution in fluctuating environments. Recently, several advances have been made through models that relax these assumptions, for example, by introducing environmental stochasticity [3–6] and density dependence [7,8]. While evolution in a constant environment will maximize mean fitness and mean relative fitness in the population, this is not necessarily the case when populations fluctuate in size [9]. As a consequence, the specific assumptions about the ecological processes involved have profound influences on which quantities genotypic or phenotypic evolution tends to maximize, and how the contribution of an individual to future generations, or fitness, should then be assessed [10]. This problem of understanding the effect of environmental variation has a long history in evolutionary biology, as

Glossary

Bet-hedging: selection for increased long-run growth rate (geometric mean fitness) by a decrease in deterministic growth rate and environmental variance.
Conservative bet-hedging: bet-hedging with no effect on phenotypic variance within genotypes.
Demographic stochasticity: stochastic variation in vital rates among individuals with the same genotype or phenotype within a year.
Density dependence: change in population size depends on current and/or previous population sizes.
Density-dependent selection: relative fitnesses or the Malthusian fitness of genotypes depend on population size.
Deterministic growth rate: the growth rate of the expected population size on the logarithmic scale, or, the logarithm of the expected multiplicative growth rate $\lambda = E(N_{t+1}/N_t|N_t)$.
Diversified bet-hedging: bet-hedging generated by selection for increased phenotypic variance within genotypes.
Environmental stochasticity: random environmental variation that affects vital rates of all individuals, or groups of individuals with the same phenotype, similarly.
Expected relative fitness: fitness measure relevant for describing change in expected allele frequency in a stochastic environment.
Fitness of a phenotype or genotype: the expected individual fitness with respect to demographic noise conditioned on the phenotype or genotype.
Frequency-dependent selection: the contribution of a genotype or phenotype to the following generation relative to other genotypes or phenotypes varies with the frequency in the population.
Geometric mean fitness: (i) measure of observed growth over a period of time such that the initial number N_0 of genotypes or phenotypes with mean fitness W_t at time t, grows to $N_t = N_0 W_0 W_1, \ldots\ldots, W_{t-1} = N_0 \hat{W}^t$, where $\hat{W} = \left(\prod_{u=0}^{t-1} W_u\right)^{1/t}$ is the geometric mean fitness. Because $\ln N_t = \ln N_0 + t \ln \hat{W}$, the t average growth rate at the logarithmic scale is $(\ln N_t - \ln N_0)/t = \ln \hat{W} = \sum \ln W_u / t$, which is the mean value of the $\ln W_u$. (ii) Measure of the *expected* growth over a period of time of genotypes or phenotypes with the same distribution of individual fitnesses $W_0, W_1 \ldots\ldots W_{t-1}$, which is the expected growth at the logarithmic scale, equal to the long-run growth rate $s = \mathrm{E}(\ln N_t - \ln N_0)/t = \mathrm{E} \ln \hat{W} = \mathrm{E} \ln W$, where E denotes the expectation.
Individual fitness: the realized contribution from an individual to the next time-step or generation including a component of random genetic drift.
Long-run growth rate: the mean population growth rate on a logarithmic scale in a stochastic environment in the absence of demographic noise and density-regulation.
Malthusian fitness: the mean growth rate on the logarithmic scale in continuous time of a hypothetical population of individuals with a given phenotype and population size.
Phenotype: the observable characteristics or traits of an organism.
***r*- and *K*-selection:** different phenotypes are selected for at small and large population sizes.
Trade-offs: population parameters, for example, $\bar{r}$ $\bar{y}$, and σ_e^2, depend on the same phenotype and therefore cannot vary independently.

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illustrated by the difficulties interpreting Fisher's [11] fundamental theorem of natural selection under density regulation and in a variable environment (Box 1).

In this review we outline, based on recent theoretical developments, approaches for integrating ecology and evolution to obtain a better understanding of how current population dynamics affect evolution over shorter and longer periods of time. We propose that this can be achieved by including specific assumptions about how different phenotypes or genotypes are affected by changes in population size as well as by fluctuations in the environments. We argue that this can be done in practice using individual fitness rather than lifetime reproductive success or net reproductive rate as a measure of fitness. The advantage of this observed measure of fitness is that it enables, by a stochastic extension of the famous Robertson–Price equation [12–15], statistical analyses of how phenotypes respond to phenotypic selection, and how this response is affected by changes in the environment and random genetic drift. Another advantage of this measure of fitness is that over a short time-period its expected value is equal to the observed multiplicative growth rate. However, a central point is that the expected individual fitness is not the quantity maximized by evolution in a fluctuating environment but instead the geometric mean fitness, or the log of this which equals the expected value of the logarithm of the mean individual fitness. This illustrates the importance of working at a logarithmic scale in empirical analyses of evolutionary processes. We highlight the importance of studying trade-offs between the expected value of individual fitness and environmental variance, for example, to understand the evolution of bet-hedging strategies (see Glossary) [10]. Finally, we emphasize that under density dependence evolution tends to maximize the expected value of some more complex functions of population size, which poses some challenges in predicting evolutionary change in density-dependent populations.

Box 1. Fisher's fundamental theorem of natural selection

One of the most important general insights in evolutionary biology was provided through Fisher's [11] fundamental theorem of natural selection. It states that 'the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time'. Accordingly, fitness always increases in a constant environment, either towards infinity or more realistically towards an equilibrium where its additive genetic variance is zero. The full implications of Fisher's [11] theorem were perhaps not fully realized until a formal proof was given by Ewens [81], showing in discrete time that the additive genetic variance in fitness divided by the squared mean fitness is equal to a partial change in the mean fitness from one generation to the next due to natural selection. In accordance with this, Edwards [82] made Fisher's theorem more accurate, rephrasing the continuous time version as 'the rate of increase in the mean fitness of any organism at any time ascribable to natural selection acting through changes in gene frequencies is exactly equal to its genetic variance in fitness at that time'. Thus, the theorem expresses a general principle for how natural selection alone affects the mean fitness [43].

One major problem in applying Fisher's result in studies of evolutionary dynamics is that variation in fitness is affected by a variety of processes other than natural selection. One of the most important of those is environmental change that Fisher [11,83] expressed as deterioration of the environment. In a fluctuating environment the fitness function (fitness as function of phenotype or genotype) is not constant but varies through time, not only due to changes in physical and biological environments but also due to variation in the population itself. Thus, environmental changes generate variation in the fitness function, implying that fitness in the next environmental state may even be less than before [84]. Therefore, in a fluctuating environment there is no single fitness function and no simple maximum that is reached by evolution.

Fisher's fundamental theorem of natural selection still represents one of the few general laws in evolutionary biology [43,82]. However, it is not a model for evolution, and hence alone does not provide a valid description of how variations in environmental conditions in natural populations will act as important selective agents [85] and influence the mean fitness of the population and evolution over longer periods.

Measures of fitness

In natural populations, measuring variation in fitness among genotypes is usually extremely difficult even with access to the most advanced toolkit of molecular genetic methods, for example, because of difficulties in capturing a sufficient proportion of individuals in the population and constructing reliable pedigrees, and because genes coding for different fitness related traits usually have small effects and are generally unidentified. Quantitative genetics provides an approach that enables analyses of even genetically complex traits without any knowledge of the genetics involved. A key concept is individual fitness, which is the contribution in numbers of individuals to the next generation from individual i with phenotype z_i. Individual fitness can be defined as $C_i = B_i/2 + J_i$, where B_i is the number of offspring produced during a given time-step (divided by 2 because in a sexually reproducing species there are two parents per offspring) and J_i indicates whether the individual has survived ($J_i = 1$) or not ($J_i = 0$). The fitness of an individual i with a given genotype or phenotype is then $W_i = \mathrm{E}(C_i)$, where the expectation E refers to demographic stochasticity in C_i, conditioned on genotype or phenotype (Figure 1A). The relative individual fitness of individual i is then $c_i = C_i/\bar{C}$, where $\bar{C} = \sum_i C_i/N$ is the mean individual fitness in a population of N individuals.

The concept of individual fitness provided the foundation for Alan Robertson [13] and George R. Price [12,14] when they derived exact expressions for the change $\Delta(\bar{z})$ in mean phenotype ($\bar{z}$) during a time-step that, for example, can be a year or a generation,

$$\Delta(\bar{z}) = \frac{1}{N}\sum_i (c_i - \bar{c})(z_i - (\bar{z})) + \frac{1}{N}\sum_i (c_i \overline{\Delta z_i}), \qquad [1]$$

where $\overline{\Delta z_i}$ is the mean difference between the phenotype z_i of individual i and that of its offspring, including the individual itself if it survives. The first term in Equation 1 is the selection differential $S = \mathrm{cov}(c, z)$. The second term is a transmission term generated by the difference in phenotypes between adults and their offspring. Thus, the Robertson–Price equation originally formulated using fitness across generations enables statistical analyses of how phenotypes respond to phenotypic selection and how this response is affected by changes in the environment (e.g., as a consequence of trophic interactions [16,17]).

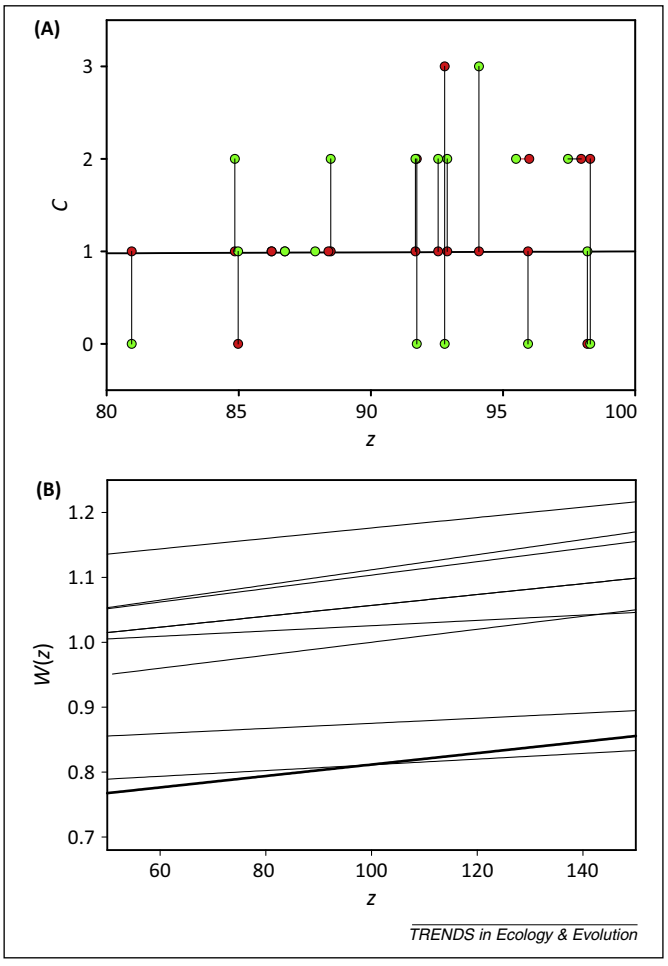

Figure 1. Stochastic variation in individual fitness. **(A)** Realization of individual fitness C in two different years (green and red circles) for two sets of individuals with the same phenotype z. The individual fitness for a haploid phenotype is $C = I + B$, where I is a binomially distributed [bin(1,0.5)] indicator variable for survival and B is the number of recruits produced to the next season, assumed to be Poisson-distributed with mean 0.4 + 0.001z. This gives that the fitness of an individual of phenotype z is W(z) = EC(z) = 0.9+0.001z averaged over individual demographic stochasticity. The lines connect the variation in fitness of individuals with the same z caused by demographic stochasticity. **(B)** Differences between years in fitness $W(z)$ as function of phenotype z with some environmental stochasticity in survival and fecundity. The thick line denotes the function used in the 2 years shown in (A).

The selection differential can be decomposed [15] into a mean value, a genetic drift component caused by demographic stochasticity due to the binomial variation in survival or similar stochastic variation in the number of offspring produced (Figure 1A) as well as a component due to temporal variation in the environment (Figure 1B). In general, individual fitness C_i will vary stochastically among individuals with the same phenotype (Figure 1). This stochastic variation in individual fitness will induce random genetic drift [18]. The fitness W_i = E(C_i) will fluctuate in time due to variation in the environment, inducing fluctuating selection. The relative size of these two components will depend on population size: the contribution of the variation in individual fitness caused by demographic stochasticity (Figure 1A) relative to the influence of fluctuations in the environment (Figure 1B) will decrease with increasing population size [15,19–21].

A measure of fitness frequently used in empirical studies of age-structured populations is lifetime production of offspring (LRS), which is the number of offspring produced by a female during her lifetime. However, this approach assumes that the population does not grow or decline rapidly and assumes a stable age-distribution [22]. Another measure closely related to LRS is the net reproductive rate $R_0 = \sum_a l_a f_a$, which is the expected value of LRS with respect to demographic stochasticity, where l_a is the mean survivorship to age a and f_a is the mean fecundity rate at age a. The net reproductive rate provides the deterministic population growth rate per generation but is an expectation ignoring several sources of variability, including permanent heterogeneity among individuals [23] and individual variation in the pathways taken through the life cycle [24–27], which is influenced by demographic and environmental stochasticity [15]. Thus, R_0 is not defined for stochastic environments. Two other approaches, which may provide the foundation for empirical studies of fitness variation in age-structured populations, were proposed by McGraw and Caswell [28] who defined individual fitness in an age-structured population as the growth rate of a group of individuals with the same realized life history, and by Engen *et al.* [29] who used the concept of individual reproductive value to define individual fitness for any individual alive in the population reflecting the expected contribution to the total reproductive value in future generations.

Coulson *et al.* [30] employed another approach to estimate individual fitness, which they termed 'de-lifing', to calculate the influence of each individual to realized changes in population size and age distribution from one year to the next, and then related these contributions to variation in different fitness-related traits. Their analyses are based on individual contributions in a large population but do not show how to separate out the effect of random genetic drift which is important at small population sizes.

Long-run growth rate as measure of fitness

All ecological systems are to a varying extent subject to stochastic variation in environmental conditions, which may strongly affect the fitness of genotypes or phenotypes (Box 2). An important general principle for evolution in stochastic environments is that the long-run growth rate is maximized over long time-periods. Although the mean fitness $\overline{W}$ of the haplotype describes the growth from one year to the next, the temporal expected value EW = $\lambda = e^r$ does not determine its number over a long period of time. This is because the type with the largest value of the long-run growth rate s = E ln W always will be the winner, such that evolution maximizes the geometric mean fitness e^s [31,32] (Box 2). It follows from Jensen's inequality that s $\leq r$ [10]. In large populations where demographic stochasticity can be ignored $s \approx r - \sigma_e^2/2\lambda$ [33–36], where $\sigma_e^2 = \mathrm{var}(W)$ is the environmental variance describing the temporal fluctuations in W.

The importance of using the logarithm of population size can be illustrated by comparing the distributions of N_t and lnN_t. From the central limit theorem it follows that lnN_t approaches a normal distribution with mean ln $N_0 + st$ and variance (to the first order) $\sigma_e^2 t/\lambda^2$ as the time t increases. Accordingly, the population size N_t is approximately

Box 2. Long-run growth rate and geometric mean fitness

To understand how fluctuations in the environment affect evolution we consider a population with N_t identical individuals of the same haplotype at time t that grows without density regulation. However, mean fitness varies across years depending on environmental conditions. If the environmental variation is uncorrelated among years, it is realistic to assume that the fitness W_t are independent variables with a given distribution. The log population size $X_t = \ln N_t$ at time t for a given initial population size is then $X_t = X_o + \sum_{u=1}^{t} \ln W_u$, such that the mean slope on the log scale is $t^{-1}(X_t - X_0) = t^{-1}\sum_{u=1}^{t} \ln W_u = \overline{R}$, and the mean value across years of the logarithm of the fitness $R_u = \ln W_u$. As time approaches infinity, this mean converges to the constant long-run growth rate [33,34,86] $s = \mathrm{E} \ln W$, where the expectation E refers to the temporal fluctuations in W. Writing $\mathrm{E}W = \lambda = e^r$, the deterministic growth rate r of the haplotype is the rate of growth of the expected number of individuals $\mathrm{E}N_u$, or, more precisely, $\ln \mathrm{E}N_t = \ln(N_0) + \ln[\prod_{u=1}^{t} \mathrm{E}W_u] = X_o + rt$. Thus, $\mathrm{E}N_u$ grows exponentially with the deterministic growth rate equal to the logarithm of the mean fitness $r = \ln \mathrm{E}W$, whereas the actual growth over long time-intervals is also exponential but with growth rate $s = \mathrm{E} \ln W$ (Figure I). If the sequence of individual fitnesses are correlated in time, the long-run growth rate is still s due to the fact that expectations are additive regardless the covariances between the variables [87]. However, the growth of the expected population size is affected by such auto-covariances.

The growth of an age-structured population affected by environmental stochasticity, but not subject to density regulation, can be described by a stochastic projection matrix L. With no temporal autocorrelations in the environment such populations will, by analogy with populations with no age structure, have a long-run growth rate which is the appropriate fitness measure over long periods of time [34]. This growth rate is given approximately [35] as $s = r - \sigma_e^2/(2\lambda^2)$, where $r = \ln \lambda$ and λ is the dominant eigenvalue of the expected projection matrix EL with elements π_i describing the growth of the expected population size similarly to models without age structure (see above). The environmental variance is $\sigma_e^2 \simeq \sum_{ij} \frac{\partial \lambda}{\partial \pi_i}\frac{\partial \lambda}{\partial \pi_j} cov(\pi_i, \pi_j)$, where $\frac{\partial \lambda}{\partial \pi_i}$ denotes how sensitive λ is to a unit change of element π_i [88]. Tuljapurkar's [35] approximation shows that the impact of a given environmental perturbation on the long-run growth rate is dependent on the sensitivity of the element it affects directly and also on the extent to which it is correlated with environmental influences on other elements.

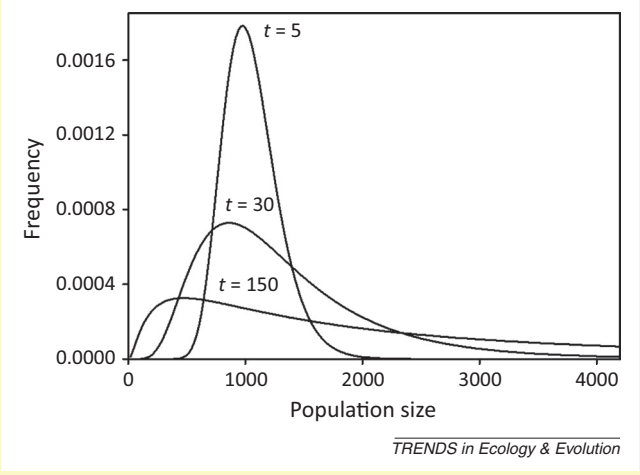

Figure I. The distribution of the number of individuals over time t in an exponentially growing population subject to a fluctuating environment. The initial number of individuals was 1000, the deterministic growth rate $r = 0.005$, and $\sigma_e^2 = 0.01$, giving $s = 0$. The expected number of individuals at times 5, 30, and 150 years are 1025, 1161, and 2117, respectively, while the median is constant equal to 1000.

lognormally distributed. As the variance of $\ln N_t$ increases through time, the skewness of the distribution of N_t increases dramatically, with a long heavy positive tail, making the expectation $N_0 e^{rt}$ much larger than the median $N_0 e^{st}$ (Box 2).

Fitness at different time-scales

An important advance in our understanding of how fluctuating environments affect evolution was provided by Lande [3]. He used a model in continuous time to analyze selection among haplotypes with correlated environmental noise and density regulation affecting all types equally, which then can be ignored when describing the genetic dynamics. The genotypic numbers n_i, $i = 1,2,...$ are then assumed to have infinitesimal mean and covariances $\mathrm{E}dn_i/dt = r_i n_i$ and $\mathrm{cov}(dn_i, dn_j)/dt = \tau_{ij} n_i n_j$ (details are given in Box 3). Regardless covariances, the type with the largest long-run growth rate $s_i = r_i - \tau_{ii}/2$ is still the evolutionary stable strategy [37].

So far we have considered definitions of fitness that apply at long time-scales. It is now generally realized that evolution can occur rapidly even over shorter periods of time [1,16,38–40]. Wright [41,42] proposed the shifting balance theory to describe evolution over shorter time-periods and suggested, assuming constant fitness differences among genotypes in time as well as random mating, that natural selection in a constant environment would cause gene frequencies to change in such a way that the mean fitness of the population increases. This concept of adaptive topography, together with Fisher's theorem (Box 1), represent central paradigms in evolutionary biology. The major difference between Fisher and Wright was Wright's emphasis on the influence of genetic drift on gene frequency changes in small subpopulations [43], which constitutes the first stage of Wright's [42,44] shifting balance theory.

In Lande's model [3] with two haplotypes logit (p_i), where p_i is the proportion of individuals of type i, is aWiener process [45] such that logit (p_i) is normally distributed and $\mathrm{E}(p_i)$ can easily be computed. Lande [3] showed that the infinitesimal change in $\mathrm{E}(p_i)$ is $p_1 p_2(\tilde{s}_1 - \tilde{s}_2)$, where $\tilde{s}_i = s_i - [p_i\tau_{i1} + (1 - p_i)\tau_{i2}]$. The expression subtracted from s_i is the infinitesimal covariance between the growth rate dn_i/n_i and the growth rate $(dn_1+dn_2)/(n_1+n_2)$ of the population. By analogy with the corresponding selection in a constant environment, Lande proposed that $\tilde{s}_i$ may be the relevant fitness measure for type i, thus generalizing Wright's [42,44] shifting balance theory to a fluctuating environment. Box 3 shows an example with very weak selection producing considerable difference between the median of p_1, corresponding to using the long-run growth rate, and the mean of p_1. This illustrates that it is not obvious which of these fitness measures are most relevant for predicting evolution of allele frequencies even over long time-intervals [10]. Although the sign of the long-run growth rate of logit (p_1) determines whether p_1 eventually

Box 3. Lande's models for genotype and phenotype evolution in fluctuating environments

For Lande's [3] haploid model that is defined in the main text with initial values $x_{i,0}$ (and $p_{i,0}$), $i = 1, 2$, the distribution of logit(p_1) = $x_1 - x_2$ after time t is normal with mean $\mu_t = (x_{1,0} - x_{2,0}) + (m_1 - m_2)t$ and variance $\sigma_t^2 = (\tau_{11} - 2\tau_{12} + \tau_{22})t$, also determining the distribution of $p_1 = e^{x_1 - x_2}/(1 + e^{x_1 - x_2})$ and the expected value of p_1. Using the transformation formulas for diffusions it appears that the infinitesimal mean of p_1 is $p_1 p_2(\bar{s}_1 - \bar{s}_2)$, where $\bar{s}_i = s_i - [p_i\tau_{i1} + (1 - p_i)\tau_{i2}]$. Lande called $\bar{s}_i$ the expected relative fitness of type i. The expression subtracted from s_i is seen to be the infinitesimal covariance between the growth rate dn_i/n_i and the growth rate $(dn_1+dn_2)/(n_1+n_2)$ of the population. The same type of formula applies for multiple genotypes [3] and sexually reproducing organisms [66].

The median of p_1, which is the backward transformation of μ_t, may be very misleading and completely disregards the fact that the frequency may be small (or large) with a large probability, while the expected frequency to a large extent takes the whole distribution into account. After some time, the distribution of p_1 is typically U-shaped (Figure I).

Lande [3,66] also analyzed a model for phenotype evolution allowing for phenotype-dependent effects of fluctuations in the environment writing $\tau(\mathbf{y},\mathbf{z})$ for the temporal covariance between the Malthusian fitness of individuals with phenotype $\mathbf{y}$ and $\mathbf{z}$. The infinitesimal covariance in the evolutionary response is $v(\bar{\mathbf{z}}) = cov[d\mathbf{z}, d\mathbf{z}^T]/dt = \mathbf{G}\mathbf{P}^{-1}cov\left[\tau(\mathbf{y},\mathbf{z}), (\mathbf{y} - \bar{\mathbf{z}})(\mathbf{z} - \bar{\mathbf{z}})^T\right]\mathbf{P}^{-1}\mathbf{G}$, where ^{T} denotes matrix transposition, and the covariances are taken with respect to $p(\mathbf{y})p(\mathbf{z})$, where p(•) is the multinormal phenotypic distribution and **G** and **P** are the additive genetic and phenotype matrices, respectively. The environmental variance in the ecological process is $\sigma_e^2 = \int\int \tau(\mathbf{y},\mathbf{z})\, p(\mathbf{y})\, p(\mathbf{z}) dydz$. Figure II shows an example in one dimension that illustrates the effect of environmental stochasticity with a parameter I describing the scaling of the covariance. If all phenotypes are affected similarly, and thus the scaling I is large, the mean will change (for a given heritability) more rapidly than if fluctuations in environment show phenotypic-specific effects corresponding to small values of I (Figure IIA). Furthermore, the infinitesimal variance of $(\bar{z})$ will also be strongly affected by I (Figure IIA). As a consequence, the phenotypic evolution will proceed with variable pace depending on the degree of phenotypic similarity of the environmental influences (Figure IIB).

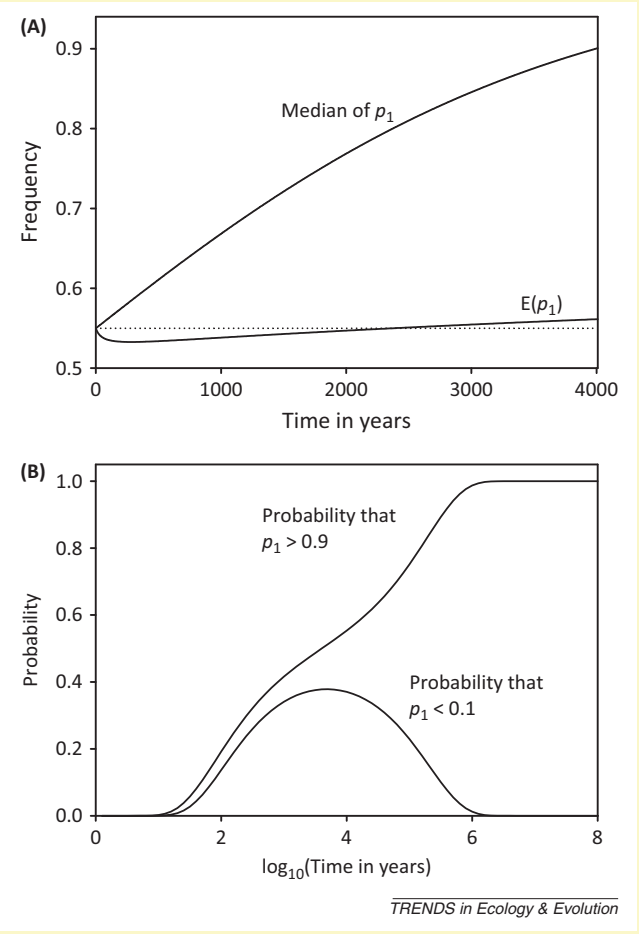

Figure I. Different measures of fitness in a stochastic environment. **(A)** The expected frequency E(p_1) of genotype 1 and the median of the distribution of p_1 (the backward transformation of E[logit(p_1)]) as functions of time. The dotted line indicates the initial frequency of p_1 at time t_0 which is 0.55. The difference in long-run growth rates between type 1 and 2 is $m_1 - m_2$ = 0.0005. **(B)** The probabilities that the allele frequency p_1 are smaller than 0.1 (solid line) and larger that 0.9 (dashed line) against log$_{10}$(t), where t is time in years.

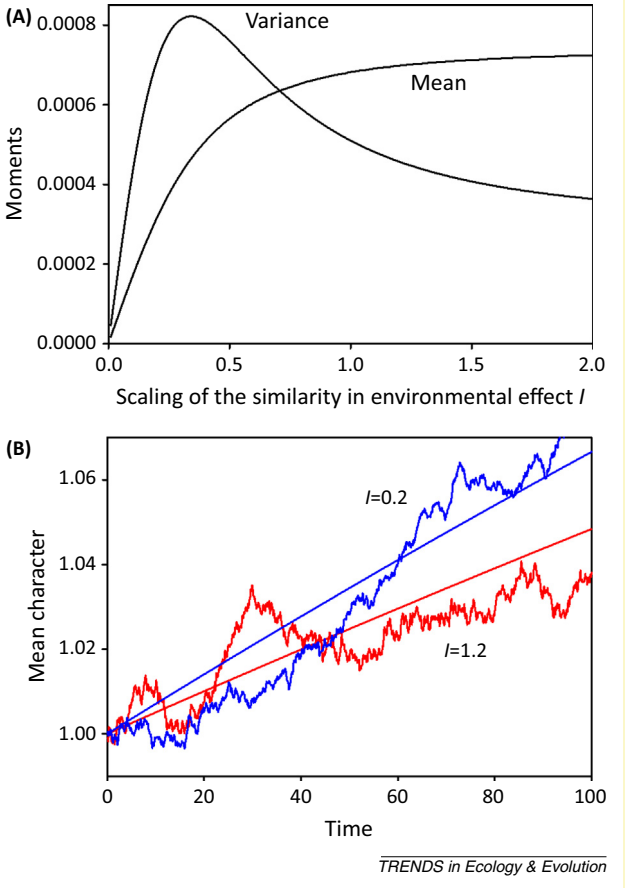

Figure II. Temporal change in mean phenotype. **(A)** The infinitesimal mean and variance for the change in mean phenotype as function of the scaling I defined along the phenotypic axis, and **(B)** simulated change of the mean character with time for two different scales of environmental effects. The autocorrelation function for the environmental noise between the Malthusian parameter of phenotype z and y is $\rho_g(y,z) = e^{-(y-z)^2/(2I^2)}$, while $\tau(z,z) = \alpha^2 e^{-2\beta z}$ such that $\tau(y,z) = \alpha^2 e^{-\beta(y+z)-(y-z)^2(2I^2)}$. The phenotypic variance var(z) = 0.09, α^2 = 0.01 and β = 1. In the simulations the mean growth rate and the heritability is held constant ($\nabla\bar{r}(\bar{z})$) and h^2 = 0.5, respectively) such that the expected evolution acts only through the environmental variance $\sigma_e^2(\bar{z})$ with infinitesimal mean $\mu(\bar{z}) = \beta h^2 \nabla\sigma_e^2(\bar{z})$.

will approach zero or one, such a process may be very slow. For a long period of time there may be a significant probability that p_1 is close to zero even if it eventually approaches one as explained in some detail and illustrated in Box 3. Thus, these examples clearly illustrate that appropriate definition of fitness in fluctuating environments is strongly influenced by how environmental variation affects the distribution of genotypes or phenotypes in the population, and that the shape of this distribution is important to consider when making inferences about evolutionary changes in stochastic environments [32]. An implication of this is that environmental stochasticity induces frequency-dependent selection [8,46] through the covariance term between the growth rate of the genotype and that of the population as a whole [3].

Bet-hedging

Bet-hedging was proposed as a strategy by which genotypes could hedge their bets by reducing the fitness costs of occasionally poor environmental conditions at the expense of smaller fitness contributions during favorable periods [47], and in this way decrease the negative effect of temporal variation in mean fitness. The advantage of such a strategy was well illustrated by Seger and Brockmann [48] and by Phillipi and Seger [49] using a simple example in which the reproductive success of a bird mother susceptible to flooding was compared when laying her eggs either in the same nest or spreading her eggs among different nests. This provided a practical illustration of Gillespie's [50] model showing that selection could favor a reduction in mean fitness if this leads to a decrease of the environmental variance.

Based on the same example, Seger and Brockmann [48] also proposed that there may be two forms of bet-hedging. In general, a bet-hedging strategy reduces the impact of extreme events happening over time [50]. Diversified bet-hedging refers specifically to a strategy in which the long-run growth rate can be increased by increasing the phenotypic variation within genotypes within a generation. For instance, in a system with two environmental states, a diversified strategy may involve production of a mixture of a poor- year and a good-year specialist within a given year. A conservative bet-hedging strategy represents a strategy that increases geometric mean fitness by reducing the deterministic growth rate other than through an influence on the phenotypic variance. For instance, in great tits *Parus major* the influence of bad years on the number of new recruits produced are larger in females laying a large number of eggs than in those with small clutch sizes [51]. However, Olofsson *et al.* [52] provided an example where both types of bet-hedging were included in the same model.

Both these types of bet-hedging strategies are a consequence of geometric mean fitness or long-run growth rate being the relevant fitness measure (Box 2), which induces some trade-off between r and σ_e^2 [53]. This means that evolution tends neither to maximize r nor minimize σ_e^2, but instead maximizes the long-run growth rate s. The variance in fitness across years depends on the magnitude of stochastic fluctuations in the environment and how the organisms can adjust to these fluctuations [54,55]. Provided that environmental stochasticity is present, conservative bet-hedging represents trade-off between r and σ_e^2 in which r is reduced at the expense of decreasing σ_e^2 to increase s [50,56]. However, in some situations this trade-off may be achieved by the phenotypic variation in fitness among genetically identical individuals within years. Strategies that include variation among individuals with the same genotype represent diversified bet-hedging, and are often popularly expressed as 'not putting all your eggs in the same basket' [10]; thus, being prepared for coping with different types of environmental conditions.

An important component of the theory of bet-hedging, as highlighted by Starrfelt and Kokko [10], is that diversified bet-hedging can be considered as evolution promoting small correlation in fitness among phenotypes of a given haplotype the same year. Bull's [57] model demonstrates that evolution of this strategy may depend on the strength of stabilizing selection around the optimal phenotype (Box 4). If the variance of the optimal phenotype is much larger than the strength of stabilizing selection (the squared width of the fitness function), evolution of diversified bet-hedging strategy is favored (Box 4).

Environmental stochasticity in quantitative genetics

To understand how environmental stochasticity affects phenotypic evolution, Lande [3] also derived, using a continuous time model and assuming a large population, a gradient formula for the expected response

$$\mu(\bar{\mathbf{z}}) = \frac{1}{dt}\mathrm{E}[d\bar{\mathbf{z}}|\bar{\mathbf{z}}] = \mathbf{G}\nabla\bar{s}(\bar{\mathbf{z}}) \qquad [2]$$

for a multinormally distributed character, where ∇ is the gradient with respect to $\bar{\mathbf{z}}$, $\bar{s}(\bar{\mathbf{z}})$ is the mean long-run growth rate for a given $\bar{\mathbf{z}}$, and $\mathbf{G}$ is the additive genetic variance-covariance matrix. The infinitesimal covariance matrix and relationship to the environmental variance is given in Box 3, which then yields a stochastic version of Lande's [58] classical deterministic gradient formula for the response to selection on a quantitative character $d\bar{\mathbf{z}}/dt = \mathbf{G}\nabla\bar{\mathbf{r}}(\bar{\mathbf{z}})$. Evolution now depends on how phenotypes are affected differently by environmental variation (Box 3).

Fitness in populations subject to density regulation

In a density-dependent population, where mean vital rates depend on density, the absolute and relative fitnesses of genotypes or phenotypes within a population change with total population size or density. Absolute fitness and population growth rate both decrease as population size increases. In a constant environment MacArthur [59] showed that evolution maximizes the equilibrium population size, or the carrying capacity K, a result extended to age-structured populations by Charlesworth [60]. Most populations are, however, affected by stochastic fluctuations in the environment that generate fluctuations in population size and Malthusian fitness with an effect on the strength and direction of natural selection. This problem was initially dealt with by MacArthur and Wilson [61] introducing the concept of r- and K-selection referring to the natural selection occurring at small and large population sizes, respectively. They argued that high population

Box 4. Bull's model of bet-hedging

As an illustration of how evolution of phenotypic variance is influenced by stochastic fluctuations in the environment we consider the model of Bull [57]. The fitness of an individual with phenotype z in environment θ is assumed to be a Gaussian function $W(z,\theta) = W_0 e^{-(z-\theta)^2/(2\tau^2)}$ [89,90], with mean $\overline{W}(\theta) = \int W(z,\theta)\, p(z)\, dz$, where $p(z)$ is the normal phenotypic distribution with mean a (the breeding value) and variance σ_z^2. Integration then yields $\ln\bar{W}(\theta) = \ln W_0 - \frac{(a-\theta)^2}{2(\sigma_z^2+\tau^2)} + \frac{1}{2}\ln\frac{\tau^2}{(\sigma_z^2+\tau^2}$. The long-run growth rate is the mean value of $\ln\bar{W}(\theta)$ with respect to the environmental fluctuations in the optimal value θ of z, which is $s = E\ln W_0 - \frac{\sigma_\theta^2 + (a-\mu_\theta)^2}{2(\sigma_z^2+\tau^2)} + \frac{1}{2}\ln(\frac{\tau^2}{\sigma_z^2+\tau^2})$, where μ_θ and σ_θ^2 are the temporal mean and variance of θ, respectively. Now, consider all possible haplotypes characterized by their breeding value a and phenotypic variance σ_z^2. It then appears that the type maximizing s has breeding value $a = \mu_\theta$ regardless of stochastic fluctuations in the environment. The optimal phenotypic variance, however, is zero if $\sigma_\theta^2 < \tau^2$, but positive and equal to $\sigma_\theta^2 - \tau^2$ if the variability in the environment given by σ_θ^2 is larger than the strength τ^2 of stabilizing selection. Hence, large phenotypic variation may evolve as a consequence of large fluctuations in the environment.

In this model there is a temporal correlation $\rho = \mathrm{corr}[W(y,\theta), W(z,\theta)]$ between fitnesses of two randomly chosen individuals measured in the same year, which then experiences the same θ, as well as a total temporal variance in fitness $\sigma^2 = \mathrm{var}[W(z,\theta)]$. The temporal variance σ_e^2 in mean fitness is then $\mathrm{var}[\overline{W}(\theta)] = \rho\sigma^2$. Accordingly, diversified bet-hedging results from selection for small values of ρ due to a trade-off between r and σ_e^2. In Bull's [57] model one can show, provided that W_0 is independent of θ, that $\rho = \tau^2/\sigma_z^2$, such that $\rho = 1$ for $\sigma_\theta^2 < \tau^2$ and $\rho = \tau^2/\sigma_\theta^2$ for $\sigma_\theta^2 > \tau^2$. Figure I illustrates how $\mathrm{var}[W(z,\theta)]$, $\mathrm{var}[\overline{W}(\theta)]$, and ρ depend on the environmental fluctuations σ_θ^2: large environmental fluctuations promote evolution of diversified bet-hedging.

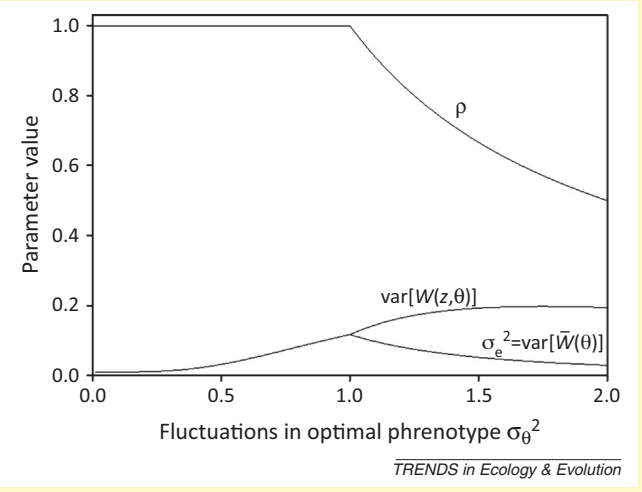

Figure I. Bet-hedging under increasing environmental variability. The two lower lines are the temporal variance in fitness $\mathrm{var}[W(z,\theta)]$ and the variance in mean fitness $\mathrm{var}[\bar{W}(\theta)]$, all as functions of the environmental noise σ_θ^2 under the optimal value of σ_z^2 in Bull's [57] model. The upper line describes the correlation in fitness $\rho = \mathrm{var}[\bar{W}(\theta)]/\mathrm{var}[W(z,\theta)]$ expressing the degree of diversified bet-hedging. The parameters are $\tau^2 = 1$, $E(W_0) = 1.2$, and $\mathrm{var}(W_0) = 0.01$ where W_0 is assumed to be independent of θ.

growth rates should be favored when the number of individuals was small (r-selection). By contrast, at large population sizes selection should result in increased carrying capacity (K-selection). One important implication of r- and K-selection is that evolution does not maximize mean individual fitness. In fact, when no environmental stochasticity is present, evolution will maximize K [62–64], and the Malthusian fitness and long-run growth rate at equilibrium are zero.

The first general model for r- and K-selection in a stochastically-fluctuating environment was analyzed by Lande *et al.* [7] using an asexual haploid model. In this model the evolutionary stable strategy (ESS), the single genotype that cannot be invaded by any other type [37], is that with the largest value of $\left[1 - \sigma_e^2/(2r)\right] f(K)$, where $f(N)$ is some increasing function of the total population size N describing the density regulation ($f(K) = K$ in a logistic model). Because the type that is established in the population has zero average growth rate, it is an ESS only if any invader has negative long-run growth rate when it is rare. This evolutionary maximization principle shows that in constant environments $(\sigma_e^2 = 0)\, f(K)$, and hence K is maximized, confirming the conclusion of MacArthur [59]. Furthermore, for small environmental variances evolution tends to produce large values of K. As environmental variance increases, this yields an ESS with larger values of r, producing r-selection (Figure 2).

Engen *et al.* [65] generalized the Lande *et al.* [7] haploid model of density-dependent selection to include sexual reproduction, assuming weak selection and multinormally distributed phenotypes $\mathbf{z}$ with mean $\bar{\mathbf{z}}$ that is changed by natural selection, and a constant phenotypic covariance matrix. The Malthusian parameter was expressed in the form $m(\mathbf{z}, N) = s(\mathbf{z}) - \gamma(\mathbf{z})\, f(N)$, where $s(\mathbf{z})$ is the long-run growth rate of a hypothetical population of individuals with phenotype $\mathbf{z}$ in the absence of density regulation, and $\gamma(\mathbf{z})$ is the strength of density regulation in such a population. Thus, Fisher's fundamental theorem (Box 1) is applicable for given N, but N fluctuates and changes the fitness function continuously [9]. With reasonable trade-offs preventing unlimited growth of fitness, evolution approaches a stationary distribution of $\bar{\mathbf{z}}$. The center of the stationary distribution of the mean phenotype is the

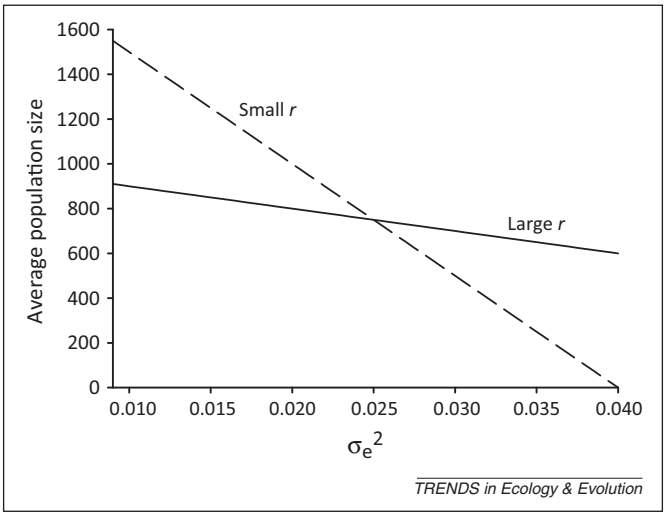

Figure 2. r- and K-selection in a variable environment. The average population size $\left[1 - \sigma_e^2/(2r)\right]K$ with respect to the stationary distribution of population sizes, which is the quantity maximized by r- and K-selection in the Lande *et al.* [7] haploid model with logistic density regulation, as a function of the environmental variance σ_e^2. The two cases shown are $r = 0.05$ and $K = 1000$ (solid line) and $r = 0.02$ and $K = 2000$ (dashed line). The type with the larger r is favored when $\sigma_e^2 > 0.025$.

phenotypic value $\mathbf{z}^*$ maximizing $Q(\bar{\mathbf{z}}) = \bar{s}(\bar{\mathbf{z}})/\bar{\gamma}(\bar{\mathbf{z}})$, which is the mean population size for a given $\bar{\mathbf{z}}$, where $\bar{s}(\bar{\mathbf{z}})$ is the mean of $s(\mathbf{z})$ over all individuals in the population and $\bar{\gamma}(\bar{\mathbf{z}})$ is the mean strength of density regulation. This maximum will not be reached because stochastic variation in the environment and population size tends to move $\bar{\mathbf{z}}$ away from $\mathbf{z}^*$ [9,65].

Concluding remark and discussion

We have presented theoretical evidence that population dynamics affect the rate of evolution, depending on how genotypes or phenotypes are influenced by stochastic variation in environment and density dependence. In many cases this causes stabilizing selection around an intermediate value of the phenotype [3,7,65,66]. Consequently, evolution tends to maximize other quantities than mean population fitness [18,67].

Eco-evolutionary dynamics is generally considered to be a feed-back process in which the organism modifies the effects of its environment and in this way changes the selection pressures it experiences [68,69]. This approach has been especially useful in describing rapid evolution in species subject to strong trophic interactions because demographic alterations caused by selection can modify the supply of available resources or the demography of a competitor or predator [16,17,38,69]. We have shown that such feedback loops are not a prerequisite for eco-evolutionary dynamics [70]. For instance, variation in population size will cause *r*- and *K*-selection towards an intermediate phenotype [65], provided that phenotypes favored at high population sizes have smaller growth rates at low population numbers, as exemplified in Figure 2. The phenotypic evolution will also be affected by environmental stochasticity: stronger *r*-selection will occur in more variable environments [7,65].

A conclusion appearing from this review is that we recently have experienced considerable advances in our understanding of how fluctuations in the environment affect phenotypic evolution. Nevertheless, general principles for how differences in ecological dynamics are expected to influence the rate of evolution are still not available. This will probably involve analyses of how fitness contributions of genotypes or phenotypes are affected by density dependence or environmental fluctuations because these affect which quantities evolution will maximize (Box 3). Such analyses may require that the contributions from demographic stochasticity that generate random genetic drift and errors in estimates of mean individual fitness are accounted for. Several promising approaches using advanced statistical methods are now becoming available that can partition different stochastic effects on selection [4,71–73]. However, such analyses require long time-series of reliable data on individual differences in production of new recruits to future generations. It will therefore be of paramount importance for understanding the process of evolution that studies providing such data [74] are maintained in the future.

Evolutionary biology is currently experiencing a period of great synthesis where several important contributions have been made in our understanding of evolution as a process [75–77]. We emphasize that an important pathway for understanding why rates of evolution show large variation [78–80] may be to include specific assumptions about how ecological dynamics affect the quantities maximized by evolution. This will provide valuable insight about which conditions are expected to produce rapid responses to environmental changes, thus providing the potential for evolutionary rescue [2] of populations facing risk of extinction.

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