

NATURAL SELECTION, VARIATION, ADAPTATION, AND EVOLUTION: A PRIMER OF INTERRELATED CONCEPTS

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Natural selection is an elegantly simple concept but one that can manifest in complex ways. I review how the basic model of single-trait viability selection has been extended to more complex forms of selection on multiple traits and on reaction norms. Fitness is defined as the expected lifetime reproductive success for individuals with a given genotype or phenotype over a given range of environments. Since the reproductive success realized by any individual will include a stochastic departure from this expectation, selection is therefore a consistent difference in fitness between organisms with different characteristics. A clear distinction is drawn between selection, which can act on any phenotypic difference, and the response to selection, which can occur only if phenotypic differences are heritable. This distinction separates the action of natural selection in filtering variation from the origin of the novel variants on which selection acts. Since selection frequently acts on standing genetic variation or on conditionally neutral variation, both of which accumulate in populations before the imposition of selection, such variation accumulates independently of its fitness effects under the subsequent selection regime. Recent discussions of “Lamarckian” inheritance must be carefully circumscribed to avoid the implication of directed mutation, for which there is no evidence.

Keywords: variation, selection, response, reaction norm, mutation.

Evolution by natural selection is an elegantly simple concept. As few as four sentences (sometimes called Darwin’s postulates) are needed to describe the essence of the process: (1) There is variation among conspecific organisms. (2) Some of the variation is heritable and passes to offspring, causing offspring to resemble their parents. (3) Some of the variation confers variation in the survival and/or reproductive ability of the parents. (4) Therefore, those variants that enjoy greater lifetime reproductive success will produce more offspring in the next generation, and their heritable traits will become more common in the population over generations. Many detailed field studies have documented the heritability of variation (review in Mousseau and Roff 1987; Roff 1997; Lynch and Walsh 1998), the correlation of variation with fitness (Endler 1986; Kingsolver et al. 2001) and the subsequent change across generations (Hendry and Kinnison 1999). For example, Galen (1989, 1996) documents heritable variation in the corolla size of alpine skypilots (*Polygonium viscosum*) and shows that individuals with larger corollas receive more pollinator visits from bumblebees (*Bombus* spp.), thus setting more seeds. The progeny of the plants that underwent this round of selection exhibited a 9% increase in corolla size relative to the progeny of unselected controls. This result is closely concordant with a 12% difference seen between wild populations recently exposed to bumblebee pollination and ancestral populations pollinated by other insects (Galen et al. 1987).

This same simplicity can at times foster a misunderstanding of natural selection. For like many simple processes, there is

a wide range of ways that natural selection can play out under different circumstances. Most biologists are familiar with the “breeder’s equation,” a mathematical expression of Darwin’s postulates for the simple case of viability selection on a single trait. The response to selection is the product of selection pressure and heritability (h^2):

$$R = h^2S, \quad (1)$$

where S is the trait difference between survivors and the population average before selection and R is the change in trait mean between generations. This simple relationship is taught in many undergraduate classes. Yet Darwin’s postulates often unfold in more complex ways than the single-trait viability selection of the breeder’s equation, and when this happens, there can be a tendency to infer that natural selection presents an insufficient explanation of evolutionary change. Dennett (1995, p. 263), for example, describes a then-widespread perception among nonbiologists that natural selection had been overturned by ideas such as punctuated equilibrium. Such misperceptions arise when novel findings or perspectives are presented as bold challenges against “orthodox” evolutionary theory, a charge that connotes an established order, willfully ignoring compelling new evidence, even though this connotation may not be the author’s intent (Sepkoski 2008). That numerous authors have asked whether evolutionary theory is insufficient, “gene-centric,” or in need of extension (e.g., Eldredge 1985; West-Eberhard 2005; Pigliucci 2007; Gilbert and Epel 2009), does little to dispel this image.

It is true that an introductory-level understanding of differential viability (“survival of the fittest”) driving change in single traits does not account for all evolutionary phenomena—but

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neither is it a complete description of modern evolutionary theory, which has been continually extended and refined since Darwin's original formulation. Perhaps the earliest extension of natural selection was Darwin's (1859, 1871) own hypothesis of sexual selection. The elaborate male characteristics of many species (the peacock's tail is the archetypal example) seem to be detrimental to survival. Yet such characteristics are clearly beneficial through enhancing the mating success of males (Gontard-Danek and Møller 1999). Indeed, Galen's (1996) example above hinges on the mating success of floral characteristics rather than survival and thus fits within the definition of sexual selection (Wilson and Burley 1983; Skogsmyr and Lankinen 2002). Altruistic behaviors provide another well-known example of phenomena that have been explained by an extension of natural selection despite seeming at odds with simple selection on viability (Hamilton 1963).

In this article, I hope to provide researchers in other fields of biology with an overview of adaptation by natural selection that goes beyond simple single-trait viability selection. In this context, I will also examine the recurrent notion that natural selection is an insufficient explanation for evolutionary phenomena. Many of the issues I will discuss are those I have encountered in my discussions with colleagues in other branches of evolutionary biology, or other biological disciplines. My theme will be that the "basic" concept of natural selection captured in the breeder's equation is only a starting point. I will trace how the concept of selection has been extended across multiple forms of selection and variation, within a unified framework, and argue that this framework explains a wide variety of evolutionary phenomena. Yet the strength of the breeder's equation as a central concept is the distinction it draws between natural selection and the variation on which selection acts. This crucial distinction represents something of a "division of labor" within evolution. Natural selection remains the only well-supported mechanism of adaptation in evolution. New variation—the "fuel" on which natural selection acts—arises independently of selection.

Defining and Measuring Fitness

To discuss natural selection, it is crucial to have a definition of fitness (for a glossary of key terms, see box 1). The word "fitness" was applied in an evolutionary context originally to describe the suitability or appropriateness of a trait to a given set of ecological circumstances (Darwin 1859). This is the same meaning as in the phrase "a feast fit for a king"; that is, it is appropriate to be served to royalty. Darwin (1859) refers, for example, to winged seeds being "fitted for dispersal" or the parts of the flower "fitted for different purposes" (for the attraction of different pollinators). The concept is that those individuals with traits most appropriate to their environments will be best able to survive and reproduce in those environments.

Operationally, however, evolutionary biologists measure fitness in a given environment by that very ability to survive and reproduce. Fitness can be generalized to the ability of any entity to replicate itself and leave copies in a future generation. Such entities might be molecules (Lincoln and Joyce

2009), or protocells (Chen et al. 2004), relevant to theories about the origin of life, as well as genes (cf. kin selection: Hamilton 1963; or selfish genetic elements: Charlesworth and Langley 1989; Hatcher 2000); populations (e.g., in phase III of the shifting balance hypothesis of Wright [1931]), and species or clades (Gould and Eldredge 1977; Vrba 1989, in the context of large-scale evolutionary changes). But many of these levels of selection remain controversial (Williams 1992), and most biologists focus on the offspring of individuals, as I will here.

The quantitative measure of fitness used in studies of selection is lifetime reproductive success. This was formalized by Fisher (1930), who imagined a life table describing the mortality and reproductive schedule of a hypothetical population of identical individuals. The population growth rate of that population in a given environment is a measure of the fitness of the traits of that imaginary population, which could then be compared with similar measures for other theoretical populations with different traits. Sober (1984) then succinctly defines the fitness of organisms with a particular trait(s) as

$$W = k \sum_{i=0}^{i=\infty} i \Pr(i), \quad (2)$$

where $\Pr(i)$ is the probability of producing i offspring and the summation is over all possible numbers of offspring left by an individual with the trait(s) being considered. (The constant k describes the proportion of genes the individual passes to each offspring—0.5 for an outcrossed offspring, 1 for a cloned or selfed offspring, and so on.) So for example, if 100 individuals with, say, red coloration are monitored and 50 die before reproduction, 30 leave 1 offspring, 15 leave 2, the final 5 leave 10 offspring each, the fitness of red coloration is $(0.5 \times 0 + 0.25 \times 1 + 0.2 \times 2 + 0.05 \times 10 =)$ 1.1, if reproduction is asexual. On average, red individuals leave 1.1 offspring across the range of possible environments being considered. If the individuals are outcrossers, then the fitness is only 0.55.

Where parents and offspring have the same genotype, as with clonally reproducing species, isolates of each genotype can have their population growth rates directly measured to quantify fitness (e.g., Bell 1990; Bennett et al. 1990; Elena and Lenski 2003). However, in panmictic populations, each individual is unique, and fitness is usually estimated as the lifetime reproductive success realized by individuals. Applied to individuals, equation (2) integrates over a probability distribution of separate possible outcomes (numbers of offspring) from which only one will be realized for any given individual (Mills and Beatty 1979). Individual fitness by itself is therefore typically uninformative because it contains a substantial environmental and stochastic component. It is the comparison of fitness among many different individuals that reveals the action of selection (see "Selection and Response").

It is frequently impossible to measure total lifetime reproductive success for many individuals under relevant environments, and researchers must often content themselves with measuring as many of the major components of fitness as they can. Lifetime reproductive success can first be subdivided into two major components, survival and reproduction, and equation (2) can be conceptually recast as the product

Box 1

Glossary

Fitness: the lifetime reproductive success of an individual or of a class of individuals sharing some characteristic.

Absolute fitness: the number of offspring an individual leaves during its lifetime.

Relative fitness: the lifetime reproductive success of an individual (or class of individuals with a particular trait) relative to the fitness of other individuals (or classes); for example, in one common formulation, the absolute fitnesses are scaled so that the average over the population is 1.

Fitness component: an element of a life table that contributes to total lifetime reproductive success; fitness components are usually chosen such that they multiplicatively determine fitness (e.g., survival \times fecundity).

Natural selection: a systematic association between fitness and some characteristic of organisms within a population occupying a specific environment (or range of environments).

Directional selection: selection on a trait that consistently favors one end of the phenotypic distribution within a population over the other.

Stabilizing selection: selection on a trait that consistently favors the middle of the phenotypic distribution within a population over the tails.

Diversifying selection: selection on a trait that consistently favors the extremes of the phenotypic distribution over the middle.

Correlational selection: selection on multiple traits which favors particular combinations of traits over the alternatives (technically directional selection on the cross products of the traits)

Adaptation: (1) the process by which the characteristics of a population of individuals change over generations in response to natural selection in such a way as to better fit the organisms to their environment; (2) a trait that increases the fitness of individuals that possess it relative to other individuals that do not. Some authors restrict the term adaptation to only those adaptive traits that arose through the process of adaptation for its current function.

Breeder's equation: the classic description of the relationship between selection (change, S , in population mean within a generation due to differential survival) and the change in trait mean between generations (response, R). These two are linearly related by the heritability (h^2) as $R = h^2 S$.

Response to selection: the change in mean characteristics of the population across generations as a result of inheriting genetic variation that is associated with greater fitness.

Correlated response to selection: a response to selection that occurs in a trait that is not directly under selection but that is genetically correlated with another trait on which selection is acting.

Exaptation: an adaptive trait that originally evolved for some reason other than its current function. As most commonly used, exaptation assumes the trait originated as an adaptation for some other function, but traits that originally emerged through nonadaptive mechanisms have also been considered exaptations.

Heritability: the proportion of the total phenotypic variance that is accounted for by genetic effects. Narrow-sense heritability is the proportion of phenotypic variance made up of only additive genetic variance, and it determines the response to selection in panmictic populations

Gene: in evolutionary theory, a gene is a piece of information affecting some aspect (biochemical, developmental, behavioral, morphological, etc.) of the organism's phenotype, occurring at a particular place (locus) on the DNA molecule and passed from parent to offspring. This is a somewhat broader definition than that given in many genetic textbooks that restrict the term to DNA sequences coding polypeptides. In Williams's (1992, p. 11) words, "A gene is not a DNA molecule; it is the transcribable information coded by the molecule."

Additive genetic variance: that component of the total variation in the phenotype that is determined by the cumulative (linear, additive) effects of alleles within and across loci. In a randomly mating population, additive genetic variance determines the correlation between parents and offspring.

Dominance: The nonadditive (nonlinear) interaction between alleles at a single locus in their effect on the phenotype, such that the phenotype of the heterozygote differs from the midpoint between the homozygotes.

Epistasis: the nonadditive (nonlinear) interaction between genotypes at separate loci in their effect on the phenotype.

Environmental variance: that component of the total phenotypic variance that is not influenced by the genotype and which is therefore influenced by variations in some aspect (usually unidentifiable) of the microenvironment (Went 1953).

Standing genetic variation: the pool of genetic variation within a population. Such variation is available to respond to novel selection without the need for new mutation to arise.

Genetic covariance matrix: a square matrix in which each row/column represents a trait and the diagonal elements contain the genetic variances of traits, while the off-diagonal elements contain the genetic covariance between each pair of traits.

Mutation: a change in the genotype that is not caused by recombination. Typically viewed as a change in the DNA base sequence.

Phenotypic plasticity: the ability of an organism to express different phenotypes in different environmental conditions. Plasticity is a form of environmental variation in which the response to the environment is specific and consistent, as, for example, when seedlings grow taller under shaded conditions.

Acclimation: a form of plasticity that involves the adjustment of an individual's phenotype in response to a change in the environment within the individual's lifetime.

Reaction norm: the pattern of phenotypic plasticity expressed by a given genotype (or class of genotypes such as a family of related individuals) across a given range of environments.

Genotype by environment interaction: the condition where different genotypes respond (alter their phenotype) in different ways across a given range of environments.

Neutral variation: variation which is uncorrelated with fitness (the term is more commonly applied to genetic variation than to phenotypic).

Conditionally neutral variation: variation that is neutral under certain circumstances (e.g., environments or genetic backgrounds), but that is associated with fitness under other circumstances.

Neutral Theory: the hypothesis that the majority of DNA-level mutations are either deleterious or neutral. Neutral theory predicts that since the deleterious mutations will be rapidly removed by selection, most DNA polymorphisms are neutral.

Darwinian: refers to the hypothesis that evolutionary adaptation proceeds by the differential fitness of heritable variants.

Lamarckian inheritance: the passing to offspring of phenotypic characters that are acquired during the parent individual's lifetime.

Box 1*(Continued)*

Lamarckian evolution: Lamarck's hypothesis that evolutionary adaptation proceeds by the directed change of phenotypic characters in a direction that increases fitness (through "use and disuse"—the elaboration of characters that are more frequently used by the individual).

Modern Synthesis: the central understanding of evolution that emerged from the synthesis of Darwin's theory of natural selection with Mendelian genetics. The term has been used by many authors to mean "orthodox" evolutionary thought.

Punctuated Equilibrium: the hypothesis that over geological time, evolution is characterized by long periods of stasis (no change in morphology) interspersed with rare rapid bursts of evolutionary change (which may be associated with speciation events).

Recombination: the creation of novel combinations of alleles at different loci. Applies to both crossing over within a chromosome and independent assortment of genes on different chromosomes.

Trade-off: a form of constraint on adaptation caused by selection acting on two traits in opposition to the correlation between them (e.g., selection favoring increases in each of two negatively correlated traits). Some authors reserve the term "trade-off" for an inevitable functional constraint, while others also apply the term to traits that are correlated by linkage and that can thus be broken up by recombination.

W = survival \times reproduction (of survivors; fig. 1). Survivorship can be further reduced into components, such as the probability of surviving particular life stages (germination, establishment, etc.), which multiply together to give total probability of survival. Reproduction can also be subdivided, for example to examine the fitness effects of different reproductive allocation patterns (fig. 1). It is often beyond the scope of a study to measure all of these components, so researchers will choose those that are most relevant and amenable to study. If the researcher chooses wisely and/or measures the majority of fitness components, a reasonable estimate of natural selection is likely, but there is always the possibility that variation in an unmeasured component of fitness will confound the study (most commonly where fitness components experience a trade-off; see "The Form of Natural Selection").

There is a second reason to use fitness components even when lifetime reproductive success can be directly measured. Subdividing fitness into components can help to isolate and study the mechanisms of selection, by identifying the particular fitness component with which a trait is associated (Arnold 1983; Conner 1996). One fitness component can be problematic, however. To study selection on certain traits, such as parental investment, it can be helpful to include the early survival of the offspring as a component of the fitness of the parents. For example, seed size investment by the parent can have substantial effects on the performance of the offspring (Stanton 1984; Leishman et al. 2000). If one wishes to measure the lifetime reproductive success of a parent plant, does the fitness component "juvenile survival" refer to the survival of the parent before reproducing or to the offspring in which the parent invested? Equation (2) applies to a single generation and is straightforward when the generation is defined from zygote formation to death (for diploid organisms), since the genotype does not change over this period. For reproductive characters, one might modify the definition of a generation such that fitness might be calculated as the number of surviving adult offspring a living adult can be expected to leave. However, considerable care must be taken, because this combines fitness components across multiple individuals

(the parent and the offspring), who usually have different genotypes. Wolf and Wade (2001) offer a detailed analysis of the strengths and weaknesses of such a definition of fitness.

Selection and Response

With fitness operationally defined as lifetime reproductive success, selection is straightforward to define as a systematic association between variation in fitness and variation in the genotypes or phenotypes of individuals. So while the fitness of each individual contains substantial stochastic influences, a consistent pattern of association across many individuals with different characteristics is the essence of natural selection. For most continuously varying traits, natural selection is the correlation between fitness and the value of the trait (cf. Galen's correlation of seed set with corolla size, described above). Selection thus turns on the fitness of different types relative to one another more than on the absolute lifetime reproductive success of the phenotypes.

(Such "absolute" fitness does have one important consequence in evolutionary theory. Since the mean absolute fitness of a population corresponds to the growth rate of that population, populations cannot sustain an absolute fitness less than 1 offspring per individual for many generations without going extinct; see Lynch and Lande 1992. Therefore, the maximal strength of selection that can be sustained must maintain an absolute mean fitness greater than 1 [Haldane 1957].)

There are many forms that the relationship between traits and fitness might take, but assuming a simple linear relationship with slope β , the evolutionary change in trait Z ($\Delta\bar{Z}$) in a panmictic population can be predicted as

$$\Delta\bar{Z} = \sigma_a^2 \beta, \quad (3)$$

where σ_a^2 is the additive genetic variance. Equation (3) is a form of the breeder's equation. Both forms of the equation highlight the distinction between selection (the correlation between fitness and the trait) and the response to that selection—

the change in the trait between one generation and the next. This distinction simply recognizes the fact that not all of the variation in a trait is necessarily heritable, and selection and response are linearly related by the amount of heritable variation. Conceptually this distinction is useful because it allows us to separate the discussion of what fits an organism to its environment from the presence or absence of genetic variation. It is particularly useful to recognize that genetic variation for a trait is expected to decline as selection adapts populations to their environments (by removing the less fit types [Falconer 1989; Mousseau and Roff 1987] this variation can be replaced by recurrent mutation [Barton and Keightley 2002]). Such a reduction in genetic variation, and thus, a slowing of the response to selection does not imply that the trait is becoming less important in adapting the organism to the environment, and a continued association of fitness with the phenotype will remain, even if genetic variation is completely eliminated by selection.

There is one circumstance, however, in which it can be operationally useful to conflate selection and response by examining only the association between fitness and the genetic variation of the trait (e.g., by correlating fitness with an estimate of breeding value or family mean). The expression of many traits is influenced by the environment, especially in plants (Went 1953; Bradshaw 1965). Fitness similarly has a strong environmental influence—some environments are intrinsically easier to survive than others. This joint effect of the environment on both the trait and individual fitness can bias the estimate of selection (Rausher 1992). Such environmental influences on many traits (particularly fitness) are virtually impossible to remove (see Potvin et al. 1990 for an illustration of the environmental variation present even within high-tech growth chambers). Therefore, one must eliminate the spurious environmental correlation between the trait and fitness by actually estimating the growth rate of multiple populations, each with different genotypes. This is done by taking family means, where the individuals within each family have been reared in randomized locations across the range of environments under consideration. This approach can do much to eliminate the problem of a spurious correlation between traits and fitness (Mauricio and Mojon-

nier 1997; Stinchcombe et al. 2002), but the ability to detect selection with this method is dependent on the presence of genetic variance, and the presence of genetic variation is not a prerequisite of selection but only of the response (eq. [3]).

The breeder's equation is typically presented in terms of the additive genetic variance in panmictic populations. This is not because of any preconceived assumption about the mode of gene action in nature. A host of studies in both domestic and natural populations reveals that dominance and epistatic interactions are widespread and have clear fitness implications (e.g., Charlesworth and Charlesworth 1987; Whitlock et al. 1995). However, the presence of such interactions does not invalidate equation (3), for two reasons. First, regardless of the actual mode of gene action, in a panmictic population, it is only the additive variance that creates a correlation between parents and offspring (because only one allele per locus is passed to the offspring). Since it is the correlation between parents and offspring that drives the response to selection, additive variance is thus the crucial determinant of short-term evolutionary change. Dominance and interaction variance are disrupted between generations by recombination and mating. (By extension, for clonally reproducing or strictly selfing populations, the entire genome is passed to offspring, and the correlation between parents and offspring is driven by the total genetic variance. In these cases, total genetic variance can be substituted in eq. [3].) Second, whether variance is additive or nonadditive (in the statistical sense relevant to the correlation between parents and offspring) is somewhat independent of the actual mode of gene action. Genes that have a dominant or epistatic action will nevertheless (usually) contribute an additive component to the variance (see Falconer 1989; Roff 1997; Lynch and Walsh 1998) and therefore can contribute to an evolutionary response to selection.

The Form of Natural Selection

To this point I have been discussing directional selection on a single trait. Intuitively the most straightforward mode of selection, this is the case where one extreme of the pheno-

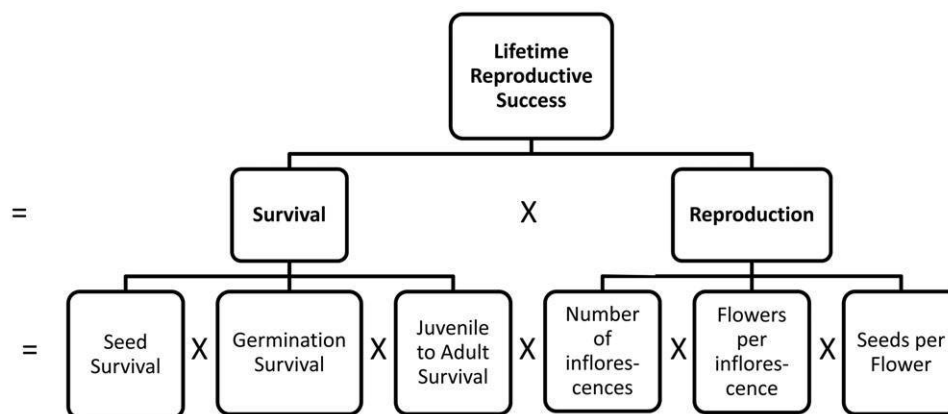


Fig. 1 Multiplicative fitness components. Total lifetime reproductive success is the product of survival and reproduction of the survivors. Each component can be further decomposed into multiplicative components as appropriate to the goals of the study.

typic distribution has highest fitness and the other extreme has lowest fitness, such that the response is a change in the mean of the trait. This is the form selection takes when a population is in the process of adapting to a novel or changing environment. Given the ubiquity of environmental change, such directional selection is likely critical to the survival of populations in the past and in the face of current human activities (Lynch and Lande 1992; Kinnison and Hairston 2007). However, in those cases where organisms are already close to optimally adapted to their habitats, selection becomes stabilizing, whereby intermediate phenotypes lie closest to the optimum and have highest fitness. Both high and low extremes of the phenotypic distribution suffer low fitness. Stabilizing selection takes the form

$$W \propto (Z - Z^*)^2,$$

where Z^* is the optimal value for the trait, and a negative relationship implies selection towards an intermediate optimum (provided that the maximal fitness corresponds to a trait value that lies within the range of variation in the population; fig. 2A). The strength of stabilizing selection, γ , is inferred from a quadratic regression of relative fitness on the trait:

$$W = \beta Z + (1/2)\gamma Z^2. \quad (4)$$

Note that stabilizing selection is estimated as the second derivative of the quadratic regression (Lande and Arnold 1983) such that the quadratic regression coefficient is half the stabilizing selection (the second derivative of eq. [4] is γ).

Basic textbooks present directional and stabilizing selection as separate categories of selection (diversifying selection is typically the third; see below), but conceptually there is no demarcation between the two (fig. 2A). A population that starts out far from the optimal phenotype for the environment will experience directional selection. However, as the population adapts and the mean phenotype approaches the optimum, one extreme of the phenotype distribution will “overshoot” the optimum and experience reduced fitness. As this occurs, selection becomes more stabilizing until the mean corresponds to the optimum at which point selection is no longer directional but entirely stabilizing (Fairbairn and Reeve 2001).

One of the classic examples of stabilizing selection serves as a useful illustration as well as a segue into selection on multiple characters. Weis and Abrahamson (1986) showed that the size of galls induced in goldenrod *Solidago altissima* by the aphid *Eurosta solidaginis* experience stabilizing viability selection. Large galls are disproportionately subject to bird predation, while small galls are disproportionately subject to attack by parasitoid wasps. Thus, survival (of the aphid) is maximized at intermediate gall size, because the probability of surviving both wasp and bird attack is highest for this phenotype. This example illustrates the relationship between directional and stabilizing selection. A shift in the relative intensity of bird versus wasp predation would shift the optimum gall size and would place directional selection on the population. As the population approaches the new optimum, directional selection would relax and stabilizing selection resume.

The gall size example is easily understood as selection on a single character but can be reformulated as selection on two characters—susceptibility to bird predation and susceptibility to wasp parasitism (in this case, multiplicative fitness components; fig. [1]). These characters, considered in isolation, experience directional selection for higher survival, but they are negatively correlated because each is dependent on a common underlying trait—the size of the gall. In this case, the negative correlation between two directionally selected characters creates a trade-off such as occurs often among multiplicative fitness components (Stearns 1989; Roff 2002; Roff and Fairbairn 2007). Directional selection on correlated characters is easily accommodated within a multivariate extension of the breeder’s equation (Lande 1982; Charnov 1989):

$$\begin{aligned} \Delta \bar{Z}_1 &= \sigma_{a1}^2 \beta_1 + \text{Cov}_{a(1,2)} \beta_2, \\ \Delta \bar{Z}_2 &= \sigma_{a2}^2 \beta_2 + \text{Cov}_{a(1,2)} \beta_1, \end{aligned} \quad (5a)$$

or in matrix form

$$\Delta \bar{\mathbf{Z}} = \mathbf{G}\boldsymbol{\beta}, \quad (5b)$$

where \mathbf{G} is the additive genetic variance/covariance matrix. This modification recognizes that the change in one trait will be partly determined by selection on the other, mediated by the additive genetic covariance between them. Where two multiplicative fitness components are negatively correlated, the equilibrium solution ($\Delta \bar{Z}_1 = \Delta \bar{Z}_2 = 0$) gives selection toward intermediate values of both traits (Lande 1982; Charnov 1989).

Recasting selection on gall size as a multivariate selection example may seem pedantic, but it illustrates the use of multiplicative fitness components to study the mechanism of selection. Plotting total survival against gall size would have been sufficient to show that stabilizing selection was present—what makes Weiss and Abrahamson’s (1986) study so compelling is that the mechanism of that selection is so clearly demonstrated. The example also illustrates a coherent framework for understanding selection on organisms as integrated suites of traits rather than as atomistic collections of independent traits (as is sometimes charged). Many cases of stabilizing selection come from a trade-off of directional selection on multiple interrelated traits such that stabilizing selection often acts in multivariate space (fig. 2B; Walsh and Blows 2009).

This becomes even clearer when we consider diversifying selection. Diversifying selection is defined as the situation where intermediate trait values have low fitness and the extremes (both high and low) have high fitness—in this it is the inverse of stabilizing selection (positive γ , where stabilizing selection implies negative γ). There are few convincing examples of diversifying selection acting on a single character. But in multivariate space, diversifying selection becomes more plausible. Mauricio and Rausher (1997) give a convincing example of diversifying selection on two categories of anti-herbivore defences in *Arabidopsis*—physical defense in the form of trichomes and chemical defense in the form of glucosinolate concentration. The effectiveness of each level of defense was measured by comparing the performance of genotypes in the presence and absence of herbivores. The

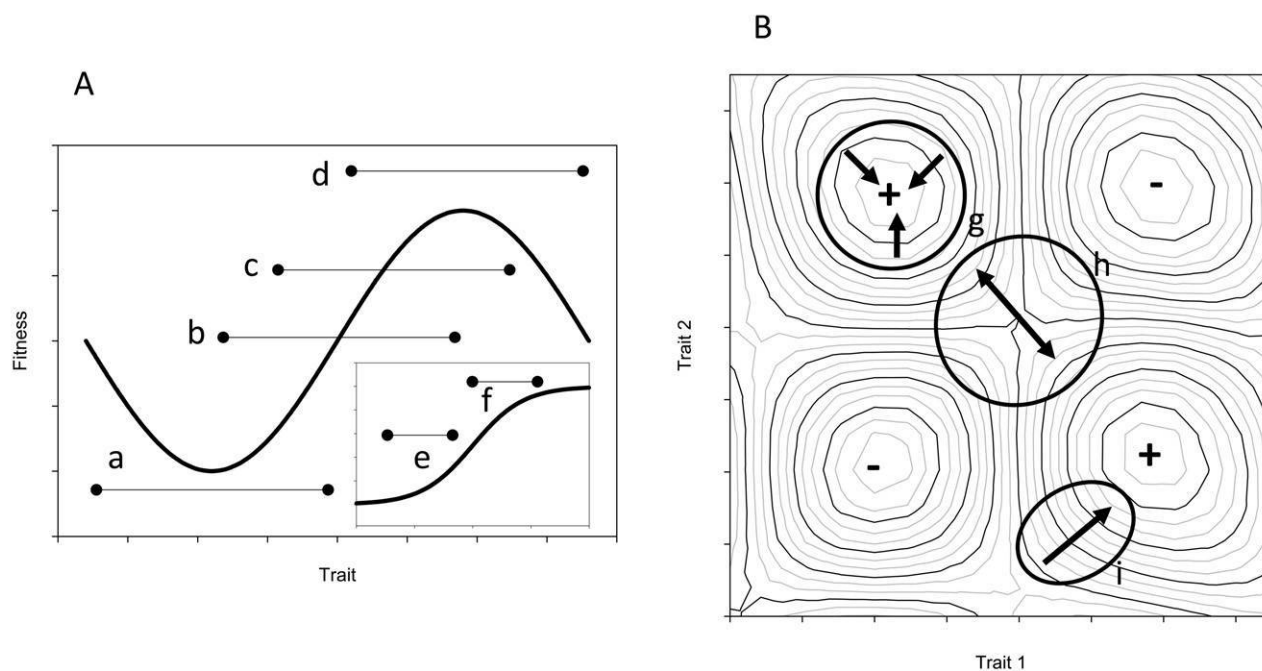


Fig. 2 Fitness functions in one and two dimensions. **A**, Hypothetical fitness function for a single trait. Horizontal lines indicate the phenotypic range of different hypothetical populations experiencing diversifying (population a, $\beta = 0$, $\gamma > 0$), directional (population b, $\beta > 0$, $\gamma = 0$), and stabilizing selection (population d, $\beta = 0$, $\gamma < 0$). Population c is approaching the optimum phenotype, and therefore, directional selection is giving way to stabilizing ($\beta > 0$, $\gamma < 0$). Inset: two populations experiencing nonlinear directional selection. The parameter γ is positive for population e and negative for population f, but for neither population does the phenotypic range include the maximum or minimum of the fitness function, and therefore neither is experiencing diversifying or stabilizing selection. **B**, Hypothetical fitness surface for two traits. Contours indicate trait combinations of equal fitness. The surface has two fitness peaks (+) and two hollows (-). Ovals indicate the phenotypic ranges of hypothetical populations under directional (population i), stabilizing (population g), and diversifying selection (population h; note that population h also exhibits correlational selection). Arrows indicate the direction of selection in each population.

most effective defence was obtained by genotypes exhibiting either a high concentration of glucosinolate with few trichomes or a high density of trichomes with little gluconsinolate. A partial mix of the two gave a valley of reduced defense. Selection on either trait in isolation was directional—herbivore resistance increased with either defense. But recognizing the organism as in integration of multiple traits revealed diversifying selection that favored either chemical or physical defense.

Mauricio and Rausher's (1997) study also provides an example of what is sometimes referred to as correlational selection. This term is not to be confused with the correlated response to selection described in the multivariate form of the breeders equation (eq. [5]). A correlated response to selection occurs when a trait changes because it is genetically correlated with a second trait under selection. Correlational selection occurs when selection acts on the cross product of two traits. In the *Arabidopsis* example (Mauricio and Rausher 1997), negative cross products were favored—high levels of one trait with low levels of another (see also Brodie 1992; population h in fig. 2). This is in effect an epistatic interaction for fitness, because fitness is not a linear function of the two traits. To the extent that the two traits have separate genetic bases, the genes for the two traits will interact epistatically in their effects on fitness.

Adaptation and Acclimation

Natural selection (if it acts on heritable variation) produces adaptations. Somewhat confusingly, “adaptation” refers to both a process and a character—the outcome of adaptation (the process) is an adaptation (a character that “fits” an organism to a particular environment or ecological circumstance). Formally defined, an adaptation is a trait that increases the fitness of individuals relative to individuals that lack the trait. The process of evolutionary adaptation is often (and quite reasonably) equated with the response to directional selection discussed above, although some include within the process of adaptation the origin of the variation on which selection acts (e.g., Barton and Keightley 2002). Adaptation is thus a process that acts on populations over multiple generations and not to individuals within a generation. The essence of adaptation is captured by Darwin's phrase “descent with modification”—that is, heredity across generations with changes brought about by the greater reproductive success of those individuals whose traits are better “fitted” to the environment. The result of this process is that the trait thus altered becomes an adaptation suiting the population to its environment.

In discussing adaptation by natural selection it is crucial to distinguish this meaning from the everyday English usage

which includes any modification in response to changing circumstances (cf. “an adaptable approach,” “adaptive management,” etc.). Many organisms have the ability to modify their morphology, physiology, or behavior in response to changing circumstances. These modifications frequently have the effect of increasing lifetime reproductive success, and (to make things more confusing) often mimic evolutionary changes that are recognized as adaptations! For example, Ehleringer and Mooney (1978) provide a demonstration of the value of leaf hairs to the desert shrub *Encelia farinosa*. In dry, hot conditions, the leaf hairs give the leaf a whitish hue (the species name *farinosa* refers to the fact that the leaves appear to have been dusted with flour), which reflects sunlight and serves to keep the internal temperature of the leaf below that of leaves that lack such reflection. In cooler, moister conditions during the winter, the plant benefits from absorbing rather than reflecting sunlight, both to warm the leaf and to drive photosynthesis (once moisture is available, allowing the stomata to open). It therefore produces leaves with much reduced pubescence and concomitantly greater light absorption (Ehleringer 1982). This phenotypic change mimics the differences in leaf reflectance that have evolved between different species of *Encelia* occurring along clines of aridity (Ehleringer et al. 1981).

The critical distinction to be drawn here is that descent with modification producing a heritable change across generations (and thus the difference between *Encelia* species) is a fundamentally different process from that which alters leaf reflectance of an individual *E. farinosa* plant in response to changes within its lifetime. To call them both adaptation would obscure this difference, and thus many biologists use the term “acclimation” to refer to those modifications that individuals make to changing circumstances to distinguish it from adaptation of populations. This distinction in no way diminishes the importance of the ability to acclimate to the survival of many species. It is simply that understanding is hampered if the same word is used to refer to two different processes.

Moreover, it is clear that the ability to acclimate is an adaptation (adaptive character), even if the process of acclimating is distinct from the process of adaptation. Acclimation is a form of phenotypic plasticity—the ability of a genotype to produce predictable changes of its phenotype in response to changes in the environment. The pattern of such a response is called a reaction norm (fig. 3). The *Encelia* leaves exhibit a reaction norm in which reflectance increases in more arid conditions while the internal leaf temperature remains constant. These reaction norms are presumably either close to optimal for the environment of *E. farinosa* or under directional selection to shift towards the optimal reaction norms. The evolution of reaction norms by natural selection has received a great deal of attention in recent years (Via and Lande 1985; Schlichting and Pigliucci 1998; Lande 2009). It is well established that reaction norms show heritable variation, as revealed by a significant genotype by environment interaction (Scheiner 1993; Schlichting and Pigliucci 1998). Thus genotypes differ in their reaction norms allowing the plastic response to respond to natural selection within the range of environments to which they are exposed (fig. 3D).

The Power of Natural Selection

Population geneticists (and most textbooks) recognize four evolutionary forces: mutation, migration, selection, and drift. Outside of evolutionary biology, selection receives by far the most attention, such that in the mind of the general public, natural selection is virtually synonymous with evolution. Within evolutionary biology, a vigorous debate has continued over the importance of natural selection relative to other mechanisms of evolutionary change. What can natural selection actually accomplish? Clearly, it can increase the frequency of those heritable variants that confer greater fitness. Yet many have questioned whether this simple action is enough to explain the full range of evolutionary phenomena (e.g., Eldredge 1985; Gould 2003; West-Eberhard 2005). Three questions have dominated: How fast can populations respond to selection? Are all traits adaptations? And can natural selection produce novel phenotypes?

The first question is empirical, and the answer appears to be “very fast” (Hendry and Kinnison 1999). Most debates over whether evolution by selection could explain observed rates of change seem to have been founded on a confusion over the scale of “fast” and “slow.” Changes in frequency of melanic peppered moths (Kettlewell 1956) are likely to be described as gradual by an ecologist, having taken 5–10 decades to reach the peak frequency of melanic morphs in the 1950s (Haldane 1956), but this would be instantaneous to a paleontologist. Paleontologists, noting periods of very rapid (on geological timescales) change in the fossil record, questioned whether such rapid change could be accounted for by a typically “gradualist” process of natural selection (Gould and Eldredge 1977). Two observations indicate that it can. First, theoretical calculations of the microevolutionary parameters implied to explain macroevolutionary transitions show that extremely weak selection pressures are sufficient to explain the evolution of changes that are considered “rapid” by paleontologists (Lande 1976). Second, comparing evolutionary rates of contemporary populations with those of the fossil record using the same units (Gingerich 1983, 2009) shows a striking pattern that the fastest rates are observed over the shortest timescales. Thus, contemporary evolutionary rates are clearly more than adequate to explain paleontological changes. Indeed, the current focus on contemporary evolution notes that selection can change populations dramatically within human lifetimes (Hendry and Kinnison 1999), often as the result of human activities (Hendry et al. 2008) and with demonstrable effects on ecological processes (Hairston et al. 2007).

The second question has engaged evolutionary biologists for decades, and the answer is almost certainly no—not all traits of an organism are adaptations. Many are adaptively neutral (especially at the molecular level; Kimura 1983), and stochastic processes can influence the evolutionary trajectory in multiple ways (LeNormand et al. 2009). To conclude that a trait is an adaptation requires detailed empirical evidence of its association with fitness. One can (for example) alter the trait and determine whether fitness is reduced (Endler 1986). If the trait is an adaptation—fits the organism to its environment—then removing that fit should reduce the organism’s ability to survive and reproduce. Galen and Stanton

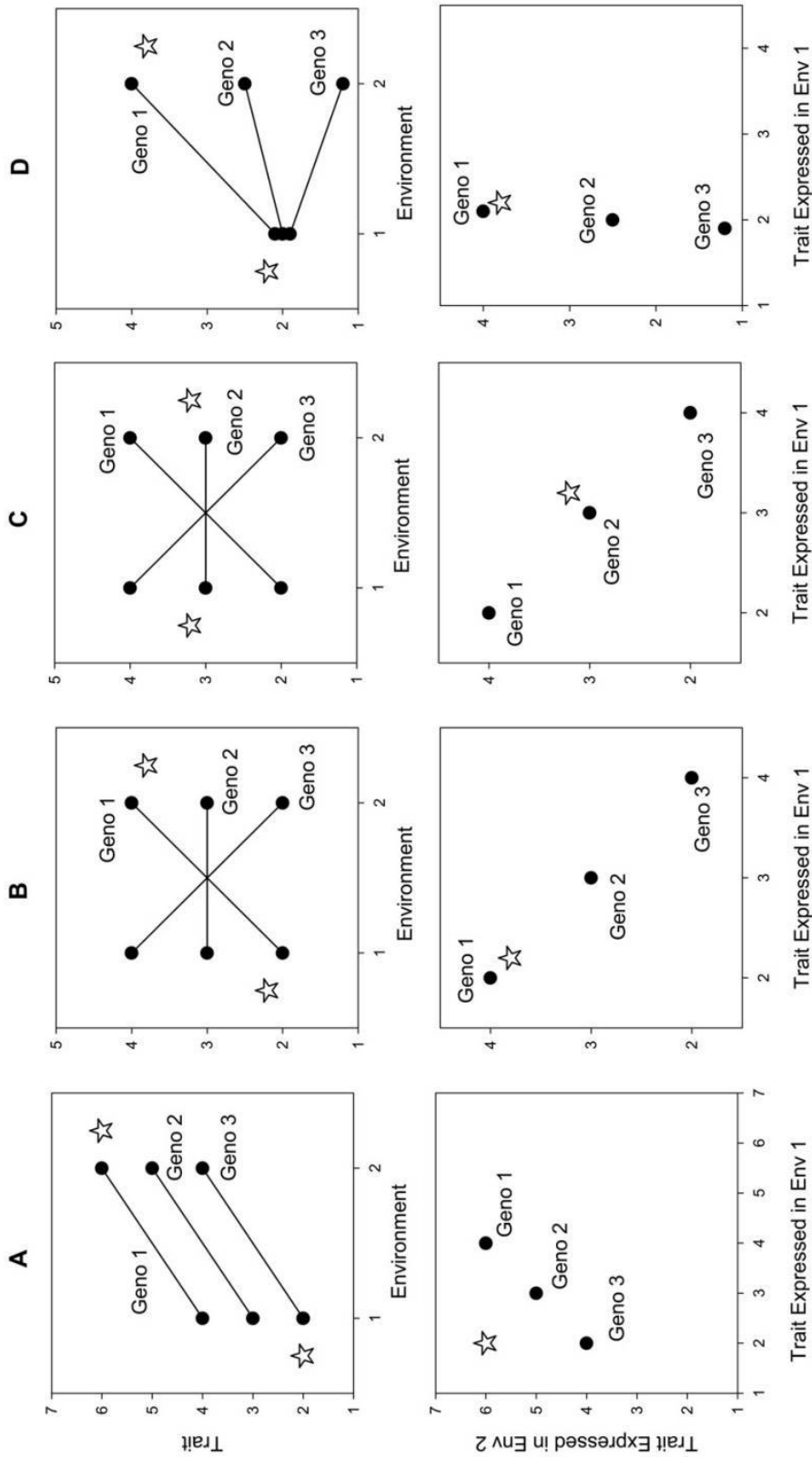


Fig. 3 Selection on reaction norms. Four hypothetical scenarios of selection in two environments are presented as reaction norms (top row) and as genetic correlations following Via and Lande (1985; bottom row). Stars indicate the optimal phenotype in each environment. *A*, Phenotypic plasticity is present, but all three genotypes have the same response such that there is no genetic variation in the plastic response. If both environments are equally frequent, selection will favor genotype 2, which is closest to the joint optimum phenotype. *B*, Genotypes differ in their response to the environment (there is a genotype \times environment interaction). A higher trait value is favored in environment 2 than in environment 1. Thus, genotype 1 is closest to the joint optimum across environments. *C*, As in *B*, but with very similar optima favored in both environments. Consequently the least plastic genotype (most homostatic) is closest to the joint optimum across environments. *D*, Conditionally neutral variation in reaction norms. Selection towards the optimum in environment 1 gives high fitness for all three genotypes, and differences between the reaction norms are neutral conditional on the population occurring in environment 1. If subsequently exposed to environment 2, variation in the reaction norms becomes exposed to selection such that the genotype with the most appropriate plastic response is favored by selection.

(2003) examined the adaptive value of heliotropism in alpine buttercups *Ranunculus adoneus*. By the simple expedient of holding some of the flowers in place with a small wire cage, they showed that flowers that are able to track the sun provide warmer and more attractive landing places for pollinators and so receive more visits.

In many other cases of course, such experiments can be exceedingly difficult. When I have been asked why members of the Pinaceae show paternal chloroplast inheritance (Wagner 1992), my typical answer regarding the movement of the organelles as gametes fuse during fertilization (Mogensen 1996) is usually interrupted—the questioner is rarely interested in mechanism but rather in adaptive function. Such an unusual trait seems to cry out for some adaptive explanation. Theoretical arguments can be offered, but without evidence they remain hypotheses. To the best of my knowledge, no one has offered any empirical evidence that selection acts on which parent contributes organellar DNA to the embryo is under selection, much less why any such selection should differ between the Pinaceae and other groups.

This desire to seek adaptive explanations for the complex and surprising traits of living things is widespread. It has also been widely (and justly) criticized. As Williams (1966, p. v) famously noted, “Evolutionary adaptation is a special and onerous concept that should be used only where it is really necessary.” An a priori assumption that all organisms are perfectly adapted (the “Panglossian paradigm” of Gould and Lewontin 1979) can lead scientists (and the wider public) to pursue adaptive explanations in the absence of meaningful evidence. There is something about the explanatory power of adaptation by natural selection that causes it to be invoked in explanation of phenomena from chloroplast inheritance in pines to unethical behavior in humans (e.g., Thornhill and Palmer 2000; see Helmreich and Paxson 2005 for a convincing refutation). The former is idle speculation, while the latter can be downright destructive. Such invocations rarely consider the alternate hypotheses, and a lack of evidence can be glossed over with a good adaptive story, particularly in the mass media. Such stories can have egregious consequences, especially when tied to false assumptions of genetic determinism. Nielsen (2009) discusses a recent case where genomic data initially suggested strong selection on genes affecting brain size in humans, but these data can be shown to be consistent with neutral hypotheses (Currat et al. 2006) and thus provide no evidence of natural selection.

The Source(s) of Variation and of New Phenotypes

The third question regarding the power of natural selection asks whether natural selection can produce novel traits. In isolation of course, it cannot. As equations (1) and (3) make clear, selection is distinct from the heritable variation necessary for a response. Thus, selection can only filter those variations that arise by other means. Indeed few aspects of adaptation by natural selection have created more confusion and misconceptions than the source of the variation on which selection acts.

Mutation is the ultimate source of all (genetic) variation. If a consistent selection pressure is sustained, new mutations

will arise while selection is continued (e.g., Barton and Keightley 2002). This is the view classically taken in molecular evolution (Kimura 1983), whereby each novel mutation is filtered as it arises by an existing selection pressure. Both beneficial and deleterious mutations occur, but only the beneficial mutations spread to fixation. In many other cases, the response to a novel selection pressure comes from a pool of “standing genetic variation” that has accumulated before the novel selection regime being imposed (Barrett and Schluter 2008). Indeed, since standing genetic variation is critical raw material for an adaptive response to changing environments, its preservation is a key component of conservation programs (Falk and Holsinger 1991; Frankel et al. 1995). But regardless of whether genetic variation arises before or during the process of adaptation, mutations occur randomly with respect to their effects on fitness.

This random (undirected, “blind”) nature of mutation has led many to erroneously assume that evolution is entirely a matter of random chance and, thus, hardly a satisfying explanation of the diversity and adaptedness of life. This view was most famously summarized by Sir Fred Hoyle’s assertion (*Nature* 294:105) that life was no more likely to have evolved by natural selection than that “a tornado sweeping through a junkyard could assemble a Boeing 747 jet.” But random mutation does not imply random evolution. Mutation is indeed random but need only generate the heritable variation that selection then filters. Selection is emphatically not a random process (Mills and Beatty 1979), and adaptive evolution consists of the filtering of random variation based on those variants most suitable to the environment(s) they encounter having consistently higher lifetime reproductive success.

Hoyle’s misconception also overlooks the fact that traits evolve incrementally. Complex traits have evolved by the successive modification of preexisting traits. Such traits might have originally evolved to serve some other purpose (called exaptations by Gould and Vrba 1982) as, for example, the evolution of seeds and flowers by progressive modification of leaves (Willis and McElwain 2002; Specht and Bartlett 2009). They may even have arisen for neutral reasons (Gould and Vrba 1982; see Lynch 2007 for an intriguing example). Gene duplications provide a rich opportunity for duplicated genes to evolve either novel or specialized functions by modification of their original function, as appears to have been important in such major evolutionary transitions as the origin of floral morphology (Irish and Litt 2005) and of C4 photosynthesis (Monson 2003).

While mutation must provide the original variants, a focus on mutation masks the diversity of ways that heritable changes in the DNA are translated into phenotypic variation on which selection might act. Recombination can greatly accelerate adaptation by combining advantageous mutations from different lineages rather than waiting for all the beneficial mutations to occur in the same lineage (Crow and Kimura 1965). Five biallelic loci (five mutation events) can produce 32 different gametes (2^5) and nearly 250 (3^5) different diploid genotypes. It is this multiplier effect that accounts for the burst of variation that is revealed following hybridization. By Mendel’s law of independent assortment, a hybrid swarm is a rich pool of selectable variation which will in-

clude many variants not seen in either parental strain. The hybrid recombination of existing variation has likely been a creative force in evolution (Anderson and Stebbins 1954; Arnold 1997; Mallet 2007), allowing colonization of novel habitat (Rieseberg et al. 2003), and/or the creation of high-fitness genotypes that displace both of the parents (Hegde et al. 2006). For example, the desert sunflowers *Helianthus anomalus*, *paradoxus*, and *deserticola* all appear to have adapted to their unique niches (sand dunes, salt marshes, and desert floors, respectively) by recombining the alleles of their parental species (*H. annuus* and *H. petiolaris*) in novel ways that could respond to selection in the novel environments (Rieseberg et al. 2003).

Two other mechanisms producing a burst of novel variants occur through the interactions of genes either with other genes or with environments. Both mechanisms can allow conditionally neutral mutations to accumulate into a large pool of standing variation before being exposed to selection. Many genes (e.g., Hsp90) function to buffer the organism against environmental variation, and such genes can also buffer the phenotype against certain mutations as well. These buffered mutations are thus conditionally neutral in that they are hidden from selection (neutral) conditional on the presence of functioning alleles at the buffering loci. Such mutations are free to accumulate as a pool of standing genetic variation until the buffering loci themselves become defective due either to mutation or to a stress sufficient to overwhelm the buffering. At this point the accumulated mutations are expressed releasing abundant novel, heritable phenotypic variation that is exposed to natural selection (Rutherford and Lundquist 1998; Sangster et al. 2008).

Such conditionally neutral variation can also arise through genotype by environment interactions, that is, genetic variation in the reaction norms (fig. 3D). Natural selection can act on the reaction norm but only on that part of the reaction norm that is expressed (fig. 3D); sections of the reaction norm that would be expressed in another environment are effectively neutral until the population encounters that novel environment (de Jong 2005). A standing pool of such variation can therefore accumulate by mutation and/or recombination until a novel environment is encountered, whereupon this variation becomes expressed and subject to selection in the novel conditions. If the new environment is strongly different from the old and favors a strongly different phenotype, then selection is likely to favor a relatively “steep” reaction norm, such that the favored genotypes will be highly plastic (Lande 2009). However, if the population remains in this new habitat, then selection may come to favor canalization of the reaction norm (possibly involving the buffering loci described above) so that the favored phenotype (in the new habitat) is constitutively produced (Waddington 1942; West-Eberhard 2005; Lande 2009).

Each of the above processes tends to occur following some disruption (hybridization, loss of canalization, introduction to new environments) and thus tends to produce a burst of novel variation at a time when selection pressures are also likely to be altered. It is tempting therefore to infer some additional mechanism of evolution over and above natural selection. This mechanism would seem to provide novel phenotypic variation to meet novel adaptive challenges, and such a mecha-

nism has been described by some as neo-Lamarckian (e.g., Gilbert and Epel 2009). In part this is because of the apparent inheritance of acquired characters—new phenotypes that have emerged during an individual’s lifetime not as a result of change in the DNA sequence. Some have gone so far as to offer the dramatic claim that the “the phenotype leads and the genotype follows”—that major evolutionary change occurs through environmentally induced adaptive phenotypic change (West-Eberhard 2005). This hypothesis posits that genetic variation occurs only after a plastic response to novel environments has already produced the adaptive phenotype. Selection then plays a reduced role by favoring those mutations that canalize the new adaptive phenotype.

Potential mechanisms for such inheritance of acquired traits are being suggested in the discovery, for example, that epialleles (such as variable states of DNA methylation) can be induced and then stably inherited, for at least a few generations. By altering patterns of gene expression, such epialleles have selectable phenotypic effects (Kalisz and Purugganan 2004), and to the extent that the methylation states are replicated, they can respond to that selection. However, it is unclear how epialleles can simultaneously be both environmentally altered and stably inherited—to the degree that they are one, the less they can logically be the other.

Moreover, describing these processes as “Lamarckian” evolution conflates two separate concepts. In addition to the inheritance of acquired characters, Lamarckian evolution also posits directed variation in those characters the organism “uses” to survive (Gould 1980; Burkhardt 1984). For Lamarckian inheritance to produce an adaptation, plastic responses must be adaptive a priori—the acquired characters (at least the ones that are inherited) must be beneficial. In one famous illustration, the blacksmith develops a muscular forearm in response to continued use, and a Lamarckian view posits that he passes this trait to his son. Yet rarely is it suggested that the blacksmith’s tendinitis would also be passed to the son despite this also being a phenotype that develops from continued use. To my knowledge, no mechanism has yet been offered that would create such differential heritability (whereby only those acquired characters that are adaptive are passed to offspring). The well-documented fact that plasticity itself evolves by natural selection such that plastic responses are typically adaptive implies that the induced phenotype is not in fact novel but rather the result of conventional natural selection acting on random variation during some prior exposure to the “new” environment. For a detailed critique of directed mutation, see Sniegowski and Lenski (1995—though written in the context of microbial evolution, the arguments apply broadly).

While hypotheses of inheritance of acquired characters may not intend to invoke the directed mutations of Lamarck, an incautious description of neo-Lamarckian ideas gives a false impression that evidence now supports a hypothesis of directed evolution. Since bursts of novel variation often accompany an environmental challenge, it is easy to unintentionally imply a directed response to the challenge. Yet most of the key examples of these processes can be explained by the standard evolutionary mechanism of selection acting on random genetic mutations. Conditionally neutral mutations are hidden by Hsp90 (e.g., Sangster et al. 2008) or by expos-

ing only limited sections of the reaction norm (de Jong 2005), creating a burst of novel variation when exposed. Induced polyphenisms (e.g., Suzuki and Nihout 2006) are consistent with existing theory on threshold traits (Roff 1997). Plasticity can indeed play a role in adaptation to novel environments (de Jong 2005; Lande 2009), but this occurs because Darwinian natural selection acts to favor the genotypes with the most adaptive plastic response over those with less adaptive reaction norms (fig. 3D). Ghalambor et al. (2007) provide a useful translation between neo-Lamarckian terminology and the standard evolutionary framework.

From So Simple a Beginning . . . One Long Argument

If you think the 20th Century sycophants have all the answers, don't burst your bubble with this course! (Poster advertising a graduate reading course on alternatives to the Modern Synthesis, ca. 1990)

Despite its multiple successes, mainstream evolutionary theory (sometimes called the Modern Synthesis, which refers to the unification of natural selection with Mendelian genetics) has come under frequent attack (frequently overstated, as in the quote above). At the heart of evolutionary theory is natural selection, which is the systematic variation in lifetime reproductive success among phenotypic variants in the population (Mills and Beatty 1979). The origin and heritability of these variants are independent of selection. Separating the response to selection (adaptation) into these two factors captures this central process of evolution. Variation occurs independently of its effects on the suitability of an organism to a given environment (i.e., independent of fitness). The mechanism by which adaptation (appropriateness to the ecological circumstances) increases is the filtering of these random variants by differential lifetime reproductive success, that is, by natural selection.

My theme in this article has been that the simplest expression of natural selection (eq. [1]) can be and has been extended to describe much more complex forms of natural selection that occur in natural populations (eqq. 5; fig. 2). The breeder's equation was derived for the very restricted conditions applicable to domestic species. It is taught to undergraduates because it is a simple beginning, not the final word. Consequently I have argued that challenges to the centrality of natural selection in evolution are less forceful than their proponents might claim, because they often seem to challenge the simplifications, rather than the core of natural selection.

It may be reasonable to ask whether an extended evolutionary synthesis is needed (Pigliucci 2007), but we must recognize that the question has been raised many times before (Stebbins and Ayala 1981; Eldredge 1985) and that past challenges to evolutionary theory eventually gave way to reconciliation with it. In the 1970s and 1980s, the Modern Synthesis seemed ready to topple in the face of Neutral Theory and Punctuated

Equilibrium. Neutralists showed that most mutations were rapidly eliminated by selection such that the majority of the genetic variation segregating in populations was neutral. Today, molecular evolution provides abundant evidence for positive natural selection adapting organisms to their environments (Kreitman and Akashi 1995; Nielsen 2005; Nachman 2006). Meanwhile, Gould and Eldredge (1977) argued that the gradualist view of microevolutionists was inappropriate to explain the bursts of "rapid" changes seen in the fossil record, whereas today we have realized that microevolution is sufficiently fast that humans can change its course, and a new journal has been devoted to applying evolution to practical problems (Tseng and Bernatchez 2009).

The most recent challenges to have taken up the torch against the orthodoxy invoke neo-Lamarckian mechanisms, suggesting something more than the "blind" mutations that so troubled Hoyle. For example, Gilbert and Epel (2009, p. 289) claim "a new interpretation of evolutionary biology" asserting (p. 319) that "in addition to allelic variation in the structural genes, there are two other sources of variation that can be acted on by selection: allelic variation in the regulatory regions of genes and developmentally plastic variation." Yet both of these sources of variation operate entirely within the "classical Darwinian" framework of random heritable variation being filtered by differential lifetime reproductive success. Plasticity has long been incorporated within this framework (Via and Lande 1985; de Jong 2005), and regulatory sequences are subject to exactly the same evolutionary forces as are structural genes.

What I hope to have accomplished here is to explain to colleagues in other fields that the study of natural selection already encompasses many of the subtleties to which its critics point. Thus, the challenges do not overturn the broader, more complete theory of natural selection that has been derived from the simple starting point encapsulated in the breeder's equation. Details of molecular biology, ecology, development, or paleontology have added rich detail to this basic mechanism. These findings enrich our understanding of evolutionary principles while they delight us with their intricacy. What they have not done is overturn our central understanding that adaptation results from natural selection acting on blind random mutations.

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