Can Evolution Supply What Ecology Demands?

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Review

Can Evolution Supply What Ecology Demands?

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A simplistic view of the adaptive process pictures a hillside along which a population can climb: when ecological 'demands' change, evolution 'supplies' the variation needed for the population to climb to a new peak. Evolutionary ecologists point out that this simplistic view can be incomplete because the fitness landscape changes dynamically as the population evolves. Geneticists meanwhile have identified complexities relating to the nature of genetic variation and its architecture, and the importance of epigenetic variation is under debate. In this review, we highlight how complexity in both ecological 'demands' and the evolutionary 'supply' influences organisms' ability to climb fitness landscapes that themselves change dynamically as evolution proceeds, and encourage new synthetic effort across research disciplines towards ecologically realistic studies of adaptation.

Trends

Adaptation to a changing environment is far from simple.

Ecological demands on populations can vary temporally and spatially.

Likewise, the supply of genetic and epigenetic variation is inherently complex.

Supply and demands can interact and alter evolutionary trajectories.

To track and predict adaptation, we need better integration across disciplines.

Supply and Demand in a Changing World

Evolution leads to better adaptation, and organisms evolve to match the demands of their ecology. True or false? If this was an examination question, an educated biologist might hesitate more than someone only superficially familiar with evolutionary theory. While the statement is a good first approximation of evolutionary theory, it comes with caveats that make the study of adaptation vastly more interesting than either steady progress that stops upon reaching an adaptive peak, or steady progression along an ever-higher performance curve would represent (e.g., Box 1, [1–4]). Analyses of genomes have made it possible to study how precisely an evolutionary process may succeed or fail to supply those genetic (or epigenetic) changes that the current environment 'demands' from its inhabitants. Meanwhile, evolutionary ecologists point out the importance of understanding the dynamic nature of the 'demands'. They are rarely constant over time and space [5,6] and because evolutionary process can feedback into ecology [7], it is possible to identify cascades between the ecological demands and genetic supplies. Using recent examples, we highlight how spatial and temporal complexity in ecological 'demands' and evolutionary 'supply' can influence the ability of species to respond to a changing environment (Figure 1).

Complex and Changing Demands

Classic population genetic theory is based on predicting responses to a predefined selection pressure (Box 2). Selection certainly occurs in the wild: general patterns emerging from meta-analyses of selection studies [review: [5]] include (i) directional selection generally favouring increased body size and earlier phenology, (ii) stabilizing selection, which is theoretically predicted to be common, is not often observed, (iii) selection on mating success is typically stronger than selection on viability, and (iv) there is a lot of spatial and temporal variation in strength and direction of selection, though it is often difficult to distinguish such patterns from sampling variation [8].

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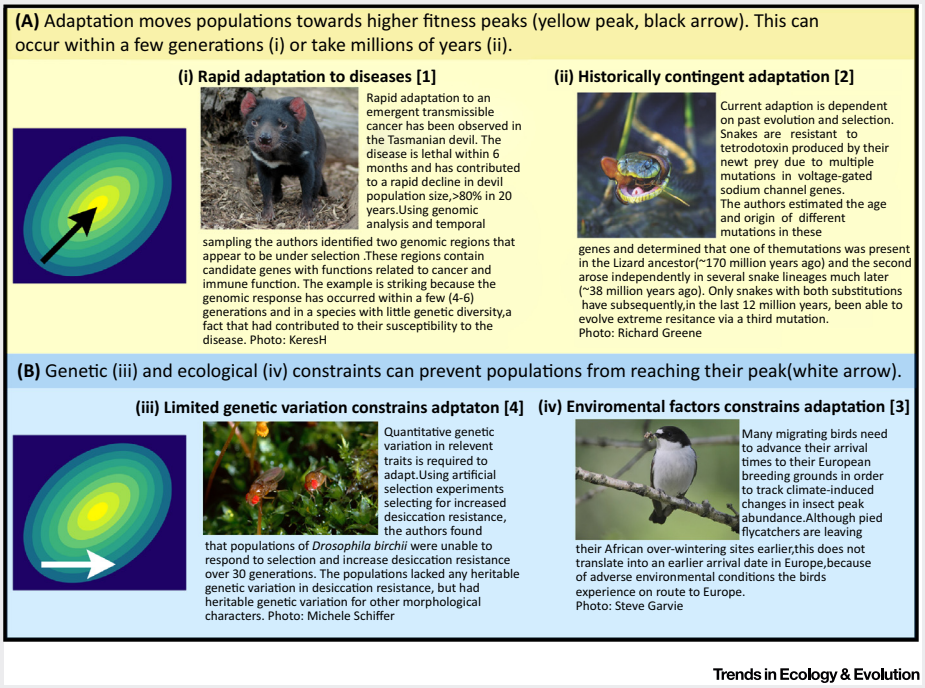
Box 1. Climbing Adaptive Mountains



By contrast, evolutionary ecology takes a step back and asks explicitly which circumstances and processes make selection positive or negative, stabilizing or divergent, constant over time or varying – the latter either due to external influences or due to positive or negative feedback (frequency dependence). This may require taking into account population dynamics, multiple traits, species interactions, and possibly spatial considerations. The need to weigh the pros and cons of a trait in a dynamic setting is practically done by employing economic arguments that weigh outcomes with their relative probabilities, and asking how these change during the course of evolution and ecological change.

Integrating evolutionary theory and ecological dynamics enables us to understand eco-evolutionary feedback, which occur when an organism modifies some feature of its environment and, by doing so, changes the nature of selection it experiences [7,9]. Often the most relevant feature of a species' environment is its own population density and adaptation to ecological demands will often alter density, which can alter the course of evolution. In great tits *Parus major*, increasing density shifts the emphasis of selection from high growth to high competitive ability, such that lower densities favour birds that lay large clutches, and vice versa [10]. Similar shifts between the importance of growth and ability to cope with competing conspecifics can happen in host–pathogen systems with added complications, because not all host individuals encounter the pathogen: prevalence of infection depends on, and simultaneously impacts the evolution of, the traits of hosts and pathogens [11]. At high pathogen density (shown in [11] for phages infecting bacterial hosts), a single host may become infected with multiple phages; thus, there is competition between multiple phage genomes. This not only makes selection 'backpedal' to favour competitive ability at the expense of fast growth, but also enables the spread of 'parasitic' incomplete viral genome particles that can only complete their life cycle when in the presence of a complete virus genome [11].

The study of real-life eco-evolutionary dynamics is challenging as possible genetic change nearly always combines with density dependence and environmental stochasticity: for

Glossary

Additive genetic variation: component of trait variation that is the result of the additive effects of genes.
Bottlenecks: severe reduction in population size.
Cryptic genetic variation: standing genetic variation that has little or no effect on phenotypic variation under normal conditions, but generates heritable phenotypic variation under changed environmental or genetic conditions.
Epiallele: a pair (or group) of identical genes that differ in their methylation.
Epimutation: a heritable change in gene activity not associated with a change in the DNA sequence but with modification of, for example, methylation status or modification of chromatin.
Evolvability: the ability of a population to undergo adaptive evolution.
Genetic drift: changes in allele frequencies due to random sampling from one generation to the next.
Genetic hitchhiking: allele frequency change of probable neutral locus that is genetically linked to a locus under selection.
Genetic variation: differences in DNA sequence between individuals.
Hill–Robertson effect: the probability of fixation of a beneficial mutation can be limited because it finds itself in linkage disequilibrium with a deleterious mutation.
Linkage disequilibrium: nonrandom association of alleles at two or more loci.
Linked selection: change of the allele frequency of loci genetically linked to a locus under selection. Includes allele frequency change due to any action of selection – positive selection or negative/purifying selection (also referred to as background selection).
Locus/Loci: a position in the genome, could be a single nucleotide position or 1000 s of base pairs of DNA sequence, it can correspond to a gene or many 100s of genes.
Mutation: a permanent change in the DNA sequence of an individual.
Mutational load: reduction in fitness due to deleterious mutations carried by a population.

Box 2. Predicting Responses to Selection

The one-generation change (R) in a trait can be predicted by the selection differential (S) and the heritability of the trait (h^2) according to the breeder's equation [88]

$$R = h^2 S$$

where S is a measure of the (phenotypic) covariance between the trait and relative fitness; h^2 is the proportion of observed phenotypic variation in a population that is due to the additive effects of genes ($h^2 = V_A/V_P$). The equation is useful because all the complexities of multilocus inheritance are captured by h^2, which can be estimated from measuring the resemblance of relatives (from similarity of parents and offspring, or genomic data). The predictive power of the breeder's equation is limited however, because it assumes that the focal trait is the singular cause of covariance between fitness and the phenotype, and ignores the influence of other phenotypic traits and the effect of environmental variation on trait–fitness covariance [89]. This assumption is likely to be violated in natural systems. A better estimate of R for a trait under 'natural' selection is provided by the Robertson–Price equation

$$R = \mathrm{cov}_A(trait, fitness)$$

where cov_A is the additive genetic covariance of trait with relative fitness in the population.

Multivariate Generalization of the Breeder's Equation

Lande ([90]) developed an extension of the breeder's equation to predict response to selection on multiple traits. This takes into consideration the additive genetic covariance between traits in the G-matrix. The G-matrix summarizes that information about trait covariation and provides a means by which genetic constraints among traits can be evaluated using a battery of metrics aimed at understanding the evolvability of complex phenotypes [91,92].

$$R = GP^{-1}S$$

where GP^{-1} is the inverted additive genetic and phenotypic variance–covariance matrix. The breeder's equation assumes that the breeding values (genotype) and environmental variables are normally distributed. It is important to note that h^2 and G are not properties of an individual, but of a population and they are not fixed, they will change with variation in allele frequencies due to linkage [93], drift [94,95], migration [96], and selection [97], and environmental variation can alter G [98]. The G can be stabilized if the underlying loci are strongly pleiotropic [97]. Long-term stability of G is expected among traits forming integrated evolutionary units, or modules [99]. Both the breeder's equation and the multivariate extension are useful to predict short-term responses to selection when h^2 or G remain relatively stable (see the supplemental information online).

Robustness: the ability of a phenotype to resist perturbation by mutations (or the environment).

example, if a yeast infection of *Daphnia* comes to a halt, a researcher can ask whether this should be attributed to evolving resistance of the host or seasonality affecting the transmission rate. Sophisticated modelling can then state that the observed halts of the epidemics are better explained if the model includes genetic change than if it does not [12,13]. Interspecific variation in density regulation could consequently be key to understanding why realized phenotypic change is slower in some species than others.

Understanding the role of population dynamics in shaping selection pressures is particularly important when populations face novel challenges. Anthropogenic activities often have the ecological effect of lowering population sizes, which then can impact evolutionary responses directly (for theory of how the rate of evolution links to population size, see [14]), or via changed ecological feedback and species interactions (e.g., [15]). Theoretical work has identified scenarios where the feedback effects range all the way to being counterintuitive. One example involves migrating birds that often fail to advance their arrival dates as much as climate change would demand [16]. Although constraints on the 'supply' side can be a cause, the ecology of territoriality itself may be a cause: breeding success is a function of relative timing (earlier arriving individuals gain better territories) as well as absolute timing (early arrival ensures a match between peak food availability and nestlings need). Climate change intensifies the component of selection that is causally related to absolute timing, but the component that is based on relative timing can diminish if climate change reduces the number of conspecifics that compete for territories – if this effect is strong enough, climate change can reduce selection to arrive early [17].

Another example is offered by increasing aridity (or grazing pressure) in ecosystems where plants face severe abiotic challenges, and local interactions are facilitative: sessile plants have

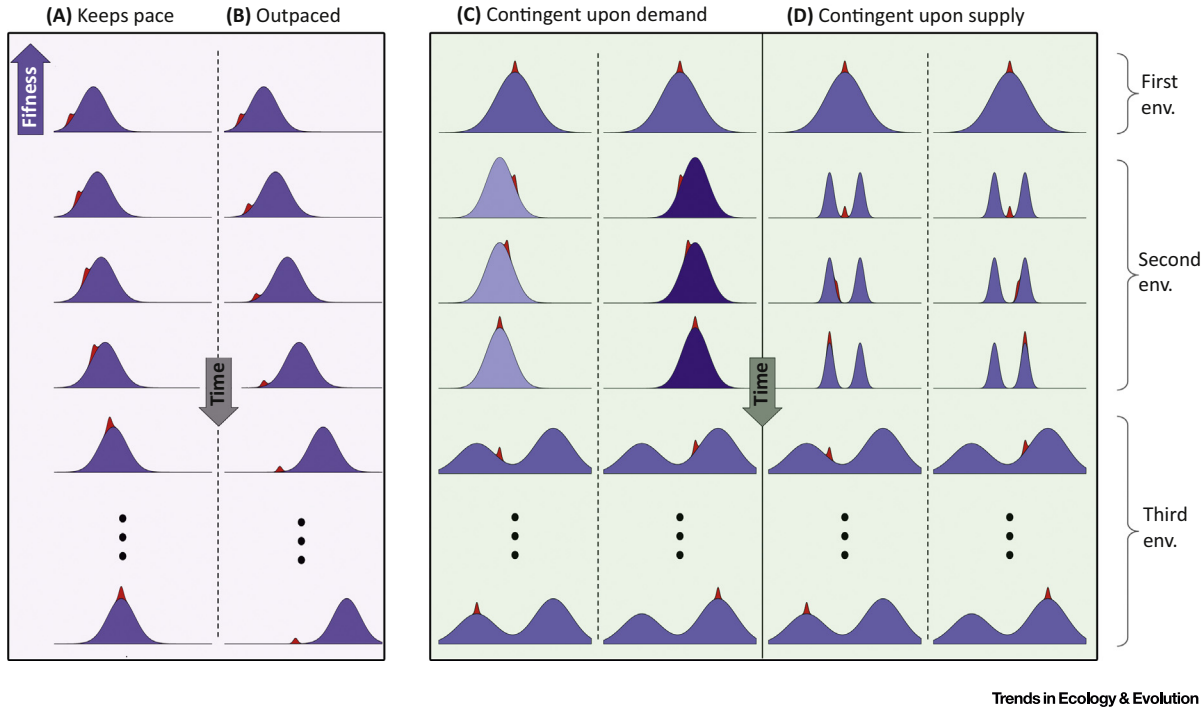


Figure 1. Adaptation through Time. A two-dimensional fitness landscape (blue curves) is shown as a function of a phenotypic trait. The distribution of the trait in the population is shown as a red peak. The left panel (pale purple) shows how supply can tract demand when the rate of change is slow (A), but when the rate of change is fast, the demand can outpace supply (B). The right panel (green) shows how evolution can be contingent on past experience and the sequence of demands. (C) In the second environment, the fitness landscape is different between the left and right panels; subsequent adaptation to these different environments leads to different evolutionary outcomes in the third (rugged) landscape, such that in the left panel the population moves to a lower fitness peak and in the right it moves to the higher fitness peak. (D) Here the final phenotypes in the population depend on the mutational sequence. All three landscapes are identical, however different mutations arising in the second environment lead to different evolutionary outcomes in the third. env., environment.

physical structures that prevent or reverse the degradation of immediately adjacent sites. This explains why desert environments often show striped or spotted spatial patterning of vegetation. If the harshness of the environment increases, the predicted response depends on seed dispersal: if dispersal distances are short, plants are selected to increase their investment in traits that maintain local facilitation; if long, the profitability of maintaining recolonizable neighbouring sites drops and 'cheater' types can exploit the system until its collapse, leading to a desert void of plants even though evolution of stronger facilitation would have allowed a population to deal with the new level of aridity [18].

The inherent coupling of supply and demand dynamics between species may also manifest itself in multispecies ecological cascades. Take the prominent example of climate-induced mistiming between four tropic levels: vernal budburst, herbivorous insects, insectivorous songbirds, and their avian predators (see [19]). In these cascades, climate change triggers an ever-earlier onset of vernal budburst that translates into the demand for insects to hatch earlier, which in turn demands that insectivorous birds lay earlier clutches, and demands concomitant changes in lay date for their avian predators [19]. The necessity for each resource or consumer (plants, insects, and birds) to shift phenology to keep up with climate change requires that the supply of variation upstream in the cascade will directly translate into a demand downstream in the cascade that can drive an adaptive response. Often the interacting partners differ in the generation time, as trees and insects do, but also for example in symbiosis [20], or geographic scale of gene flow (e.g., plant–pathogen coevolution [21]), with the possibility that some species end up with larger coevolutionary lags or poorer local adaptation.

The Supply Side: When and How Does It Work?

As stated earlier, ecologists can identify situations where evolution is not necessarily predicted to lead to improved population fitness. Even in cases where the ecological demands *per se* were simple enough to justify an expectation that selection pushes a population uphill on a fitness landscape, the response to selection may remain weak or absent. The reason why strong selection is not enough to guarantee an appropriate evolutionary response is that heritable or **additive genetic variation** (see Glossary) in the direction of selection is also required (Box 2). Artificial selection experiments show that **additive genetic variation** exists and we can observe a response to selection for many traits [22], but not all [4]. In the following section, we consider the genetic factors that impact how readily the population moves to a new adaptive peak following environmental change, with a focus on available genetic variation and genetic architecture, and then consider other factors including epigenetics and plasticity.

Genetic Variation

In the absence of a sufficient supply of adaptive variation, populations faced with a changing environment may fail to respond and, if the failure has drastic enough population dynamic consequences, also go extinct [23]. **Mutation** is the ultimate origin of genetic variation, and most mutations are the result of DNA repair errors (see [24]). The maintenance of genetic variation represents a balance between the rate at which mutations appear (*de novo* or through gene flow) and are lost or fixed, either via selection or **genetic drift**. Appearance of *de novo* mutations varies enormously: between species, individuals, and sexes, they relate to ecological and environmental factors, in particular stress [25,26], and covary with genomic features such as transposable elements [27], heterozygosity, and recombination [28]. Although *de novo* mutation is a source of potentially adaptive genetic variation, most variation is deleterious, generating a **mutational load** in populations, which may strongly affect population dynamics (for recent discussions, see [29,30]). Natural selection is generally expected to work on keeping mutation rates down, with the so-called drift barrier setting a lower limit of what can be achieved [30]. With respect to the loss of mutations, both drift and selection have a role. Under neutral models larger, more stable populations are less impacted by genetic drift and thus harbour greater neutral genetic variation compared to small ones, but this does not adequately explain variation in genetic diversity [31]. Progress in the genomics era has revealed many additional factors governing genetic variation. For example, environmental heterogeneity and gene flow can maintain higher genetic diversity [32], strong selection can reduce genetic variation across the genome, via **linked selection** [33], asexual species can accumulate more mildly deleterious mutations and thus greater genetic variation [34], and species that invest more heavily in offspring quality rather than quantity have lower genetic diversity [35].

Predicting the extent to which the loss of genetic variation will impact a population's persistence in the face of a changing environment has been challenging for several reasons. First, reductions in population size may lead to demographic as well as genetic problems that impact persistence; typically, researchers have more expertise in one and may overlook the other effects operating. Second, estimates of genetic variation in wild populations often measure neutral molecular genetic variation, but the rate of evolutionary change is governed by the additive genetic variation for ecologically relevant traits and their covariation with fitness (Box 2). Although neutral and additive diversity are expected to be correlated, this relationship is often weak [36] and these measures cannot be considered equivalent when considering ecological structure and community function [37]. The recovery and expansion of populations that have lost molecular genetic variation during severe **bottlenecks** provide further evidence that the relationship between bottlenecks, genetic diversity, and adaptive potential is not a simple one (e.g., Box 1) [1,38,39]. Under experimental circumstances, additive genetic variation has increased following a bottleneck [40,41] and changes in the conditions that a population experiences (ecological, environmental, or demographic) can release **cryptic genetic**

variation [42]. Whether this cryptic variation is the cause of a population's recovery is unclear, but it is overly simplistic to assume that a loss of neutral genetic variation equates to a loss of additive genetic variation and adaptive potential. Determining if a lack of adaptive potential is the reason why small populations fail to adapt is difficult because other confounding factors, such as loss of fitness from inbreeding or environmental stress and increased influence of drift, which can reduce the efficacy of selection and increase allele surfing, are acting simultaneously and these can also interfere with adaptation [14,39]. To date, few studies disentangle these multiple co-occurring effects [43,44] and more are needed to better understand evolutionary processes impacting small populations.

Genetic Architecture

Genetic architecture is a collective term for the number of **loci** controlling a trait, the loci's effects (additive, dominant, epistatic) and effect sizes, and the degree to which the action of these loci are independent and free to evolve in any direction. As organisms are forced to adapt to a new environment, the genetic architecture of a trait under selection can play an important role in facilitating or hindering adaptive evolution. Recent empirical studies based on genomics and theoretical work have greatly expanded our understanding of loci controlling complex traits and the interplay of genetic architecture and **evolvability** (Figure 2). The simplest genetic architectures are composed of single genes, for example, the case of industrial melanism in the pepper moth [45] or sexual maturation in salmons [46]. These classic examples of traits with a simple genetic architecture, however, are unlikely to be representative of the majority of ecologically relevant traits as most have a complex or polygenic architecture, controlled by many loci or genes, each having a small phenotypic effect [47–49].

The degree of nonindependence between multiple loci of polygenic traits has a great influence on the adaptive capacity of a species and population. Adaptation at polygenic traits necessitates the joint action of alleles at multiple loci, and will thus generate allelic covariation among them and **linkage disequilibrium** in a population [50,51]. Covariation between alleles can be achieved by the clustering of multiple loci into close physical linkage and a suppression of recombination, which acts to break apart allelic associations. The clustering is especially relevant in the context of adaptation to heterogeneous environments in species with high gene flow as it provides opportunities for locally favoured alleles to be coupled with reproductive isolating mechanisms. Genetic clusters can range in size; from a few megabases creating islands of divergence [52] to chromosomal arms [53] and all the way up the evolution of entire sex-specific chromosomes that allow male- and female-based genes to be inherited together [54]. Recent compelling examples of adaption involve genetic clusters, referred to as 'Supergenes', that incorporate a diverse range of traits, from social behaviour in ants [55,56], ornamentation in birds [57,58], mimicry in butterflies [59] to heavy metal tolerance in *Mimulus guttatus* [60].

Genetic nonindependence due to linkage does not always facilitate adaptation. When selection changes the allele frequency of a selected locus, it also changes the allele frequencies of neighbouring loci via **genetic hitchhiking** [61]. When advantageous mutations are in linkage with deleterious mutations, antagonistic selection on these loci can limit adaptive responses, known as the **Hill–Robertson effect** [62]. This effect can influence genomic divergence (e.g., [63]), patterns of genetic variation [29], and it has also been implicated in limiting adaptive evolution [64]. An interesting example of the latter comes from Soay sheep where the proportion of dark sheep, which have a fitness advantage related to larger size, has decreased over time counter to expectations. Gratten and co-authors [65] demonstrated that the coat colour mutation is linked to another locus that has antagonistic effects on size and fitness: sheep that are large and homozygous for dark coat colour have reduced fitness compared with heterozygous and light-coloured sheep. This could explain why light coloured sheep are

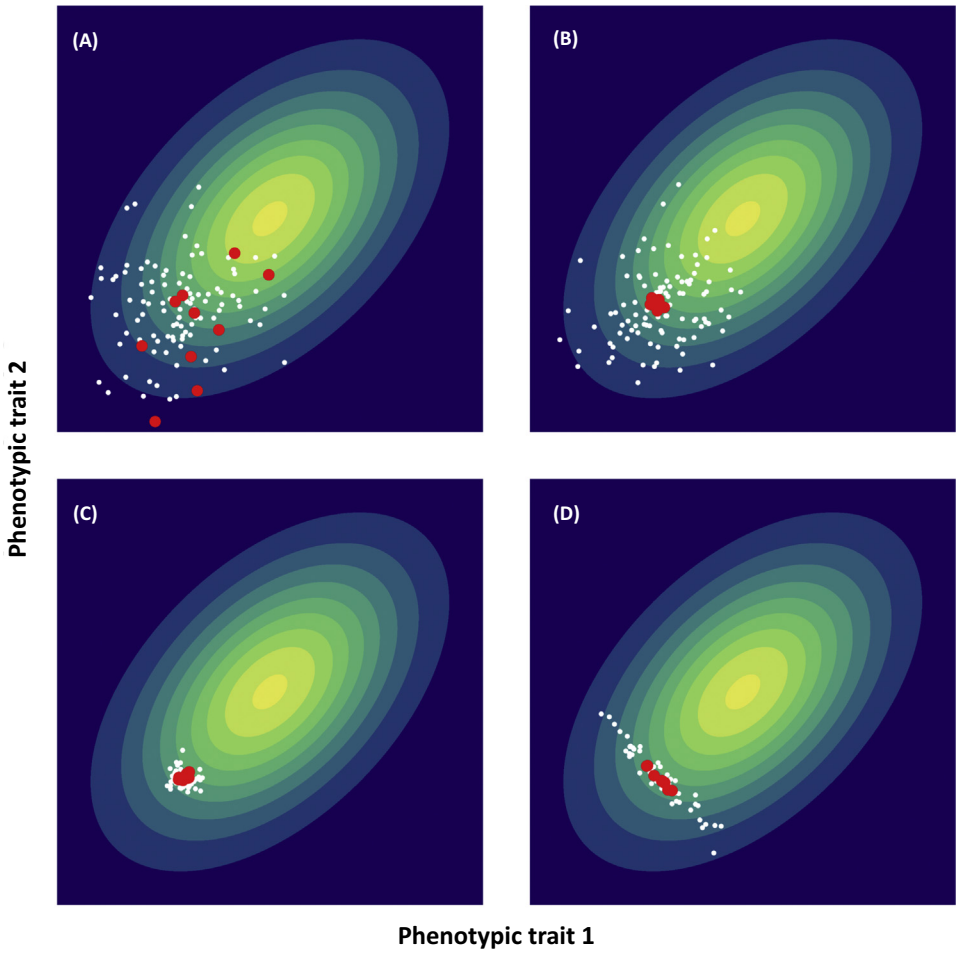


Figure 2. Constraints on Evolvability. Fitness gradient from low (blue) to high (yellow) is plotted as a function of two phenotypic traits. Red points correspond to the phenotypic values of individuals currently present in the population and white points refer to the phenotypic values that could result from mutation and recombination. (A) Available and potential supply allows for adaptation. (B) Available supply (red points) is restricted, and adaptation relies on generating new individuals with different trait values (white points), which in turn is dependent on population size and structure, mutation, and recombination rate. (C) Available and potential supply is restricted, for example, due to genetic architecture or antagonistic selection acting on traits, and thus adaptation is constrained. (D) Variation in current and potential phenotypes exist, but not in the direction required for adaptation.

increasing in frequency counter to expectations based only on phenotype–fitness associations [65].

In addition to genetic linkage, nonindependence between traits can develop when loci or traits are developmentally and functionally linked by pleiotropic gene action, which can create evolutionary units or modules [66,67]. Genetic modules can be identified by the clustering of pleiotropic gene effects among sets of phenotypic traits (e.g., [67,68]). Architectures involving modularity have been argued to combine flexibility with **robustness** in a manner that improves the evolvability of organisms [66,67]. Modularity itself may evolve to match new demands from the environment and create new independent trait modules by breaking trait genetic dependencies. This necessitates variation of the pleiotropy of underlying genes. Here epistasis has been shown to play an important role in providing such variation (e.g., in the mouse [69]; in HIV [68]).

Adaptation involving polygenic architectures that involve many nonindependent genes is somewhat analogous to scenarios involving multiple covarying phenotypic traits. It would be intriguing to link new findings on genetic clusters and modularity to recent findings that consider the number of traits under selection, their relationships, and the impact of gene flow. Similar to the number of genes, the number of traits under selection can also influence adaptive potential, however whether this helps or hinders adaptation remains unclear. For example, local adaptation in the face of gene flow can become easier to achieve if the adaptive task is multidimensional [70]; alternatively, when traits are highly correlated, this can reduce fitness and act as a correlational load on a population, constraining local adaptation [71]. It is important to understand the genetic and phenotypic architecture underlying adaptation and continue to develop multivariate, as well as univariate models.

Epigenetics, Plasticity, and Parental Effects

Interactions between genotype and environment can produce phenotypic, genetic, and epigenetic changes that influence adaptation. These interactions (genotype by environment, or G × E) can occur within a generation (i.e., phenotypic plasticity) or between generations (parental effects, transgenerational plasticity). The environment can induce changes to gene expression via epigenetic mechanisms: DNA methylation and histone modification [72,73], or parental effects, imprinting, and physiological and behavioural manipulation of offspring traits [74–78]. Epigenetic change is a mechanism that can underlie plasticity and parental effects [73,79], which could provide faster phenotypic responses to environmental change compared with 'traditional' genetic changes.

There are, however, logical challenges to overcome once one realizes that adaptation based on epigenetic variation shares a problem with 'traditional' adaptation. It is clearly easier to make **epimutations** selectively favoured if we assume they tend to move organisms towards the currently optimal state than if we do not make this assumption [80]. Given the lack of foresight of any evolutionary process, one should not generally assume **epialleles** to be automatically operating in a beneficial direction any more than we do this for genetic mutations. Much interest has been focused on maternal effects and here there is an obvious link to theories of phenotypic plasticity [73,81]. The potential advantage of maternal effects versus 'traditional' plasticity is that the mother has sampled the environment for longer and hence perhaps more reliably [77], which is beneficial if the information provided by the mother does not become outdated. The frequencies of temporal change are therefore, in several models (e.g., [76,78,81,82]), found to dictate whether incomplete resetting of epi-marks produces positive parental effects, or whether – a possibility only allowed in some models [81–83] – negative parental effects evolve.

Models vary in whether they consider temporal and/or spatial variation (e.g., temporal: [80], analytical model of [78], spatiotemporal: simulation model of [77,78,82]), and perhaps more importantly, whether they examine parental effects' success when the genome also offers competing ways to adapt, such as genetic local adaptation [77], offspring measuring the environment themselves (traditional plasticity; [77,81]), or bet-hedging without attention to cues [73]. This makes it somewhat premature to list the conditions most favourable for the evolution of epigenetic inheritance, but several models suggest a case where the shifts in environmental conditions are severe but rare [78,81,83], and – in case of spatial variation – where types of environments are roughly equally common [82,84].

It is also intriguing to comment on the case where epigenetic inheritance evolves without either parent transmitting information from environmental cues to offspring (e.g., [82]), as here the process appears to be different in mechanism but logically similar to the way traditional natural selection works. The fact that one's mother has survived to reproduce can be used to infer that

her phenotype is a well-performing one [85], and this effect – operating purely via demography – can also predict that an offspring should more faithfully copy the less dispersing parents' phenotype [86], for example, when pollen travel further than seeds [73,78,87]. In certain situations, with suitable levels of temporal unpredictability, it may also be beneficial to diversify as a form of bet-hedging [73], though again the availability of other options matters: if cues are available, non-cue-based diversification might become an inferior option [76].

Concluding Remarks

When do populations fail to adapt to novel challenges? Combining the expertise of several authors with different research backgrounds reveals intriguing differences of emphasis: a nonexhaustive list states that the demographic process operating during an eco-evolutionary feedback loop may work in a counterproductive direction for improved persistence, there may be too little additive genetic variation for selection to work on (or the 'task' may simply require too much evolutionary novelty), the population size may have become so low that drift overrides selection, the genetic architecture may show an unfortunate arrangement for the current task, or the appropriate plastic responses may never have evolved. A clear message from our synthesis is that genomic approaches can help, but a world where genomic work is routinely interpreted in the appropriate ecological context is still lacking, leaving many outstanding questions (see Outstanding Questions). But maybe, if we express a moment of optimism, it could be just around the corner, as we have all the tools in place – what is needed now is more collaboration between individuals who mostly focus on the 'demands' side and those whose research how the 'supply' works.

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Supplementary Information

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.tree.2016.12.005.

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Outstanding Questions

Are genetic constraints commonly the cause of adaptive failures?

Useful systems to study this include laboratory experiments, but also field studies of failed invasions or comparisons between multiple populations following disease outbreak – identifying why some populations went extinct when others did not.

How important is a lack of genetic variation for adaptation in small populations?

Work should also consider potential confounding factors such as inbreeding depression, stochastic extinction, and habitat deterioration.

What governs adaptive potential (evolvability)?

For example, is it genetic (genetic variation, genetic architecture, drift, epistasis), is it developmental (limited novelty possible), or is it ecological (population size, habitat deterioration, impaired gene flow)?

How does spatial and temporal variation in ecological factors affect trait evolution?

Consider the evolution of traits across variable ecological settings – how stable is the G-matrix, how stable is selection over time and across space, and how substantial is gene flow?

How important is eco-evolutionary feedback to adaptation?

Appreciating the complexity of the ecological processes is important: do not claim you have understood adaptation until the ecological dynamics of selection has also been described.

How does past selection influence future adaptive potential?

Such work includes determining the age and origin of mutations underlying ecological traits, and examining how past selection influences adaptive paths.

To what extent do constraints on short-term evolution influence long-term evolution?

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Microevolutionary change can be predicted based on methods involving the breeder's equation and the G-matrix; long-term evolution can also change the genetic architecture. What is the relationship between these, and what are the ecological settings that lead to organisms remaining 'stuck' at one equilibrium among multiple stable states (phylogenetic inertia)?

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