

## Opinion

## The Missing Response to Selection in the Wild

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**Although there are many examples of contemporary directional selection, evidence for responses to selection that match predictions are often missing in quantitative genetic studies of wild populations. This is despite the presence of genetic variation and selection pressures – theoretical prerequisites for the response to selection. This conundrum can be explained by statistical issues with accurate parameter estimation, and by biological mechanisms that interfere with the response to selection. These biological mechanisms can accelerate or constrain this response. These mechanisms are generally studied independently but might act simultaneously. We therefore integrated these mechanisms to explore their potential combined effect. This has implications for explaining the apparent evolutionary stasis of wild populations and the conservation of wildlife.**

### Our Ability to Predict the Response to Selection in the Wild Is Limited

Evidence for contemporary microevolution by natural selection has repeatedly been found in both plant and animal populations [1]. However, responses to selection often do not match quantitative genetic expectations in long-term surveys of wild populations (hereafter referred to as surveyed populations) [2], even with 10–70 years of phenotypic and pedigree data [3]. A lack of observed response to selection ('evolutionary stasis') appears to be the norm rather than the exception in these studies [4,5]. This is puzzling because the response to selection was often missing, even though there was evidence for genetic variation and selection pressures on a trait – theoretical prerequisites for evolution by natural selection. These findings reveal a conundrum in which the general expectation of an absence of response to selection derived from studies of these surveyed populations conflicts with the expectation of the presence of a response based on evidence from other types of studies.

We first summarize statistical explanations for this conundrum, namely that the measures of selection and quantitative genetic parameters are imprecise. In our opinion, the statistical issues that we highlight below should be taken into account more often in studies of surveyed populations. Our focus is on issues that are most relevant to a mixed modeling approach because this is the most widely used method for estimating quantitative genetic parameters [e.g., additive genetic(co)variance] in surveyed populations. We then briefly discuss the alternative, but not mutually exclusive, role of biological explanations in explaining this conundrum. Biological mechanisms have the potential to impede selection and thereby avoid the erosion of genetic variability by selection. We also introduce mechanisms that we argue need to be taken into account in surveyed populations.

### Highlights

Recent discoveries at the intersection of quantitative genetics and evolutionary ecology are challenging our views on the potential of wild populations to respond to selection.

Multiple biological mechanisms can disconnect genetic variation from the response to selection in the wild. We highlight areas for future research.

We provide an integrative framework that can be used to qualitatively assess the combined influence of these mechanisms on the response to selection.

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Although some mechanisms limit the amount of heritable variation that selection can act upon over short time-scales, other mechanisms promote the response to selection [6]. An issue with the biological explanations is that these mechanisms have generally been studied separately. As a result, their cumulative and interactive effects remain unexplored. We qualitatively combined the effects of several mechanisms in an imaginary population of interest characterized by three 'known' mechanisms to illustrate how to predict a corrected baseline expectation for the response to selection. We also discuss that, to some extent, the sum of the qualitative effects of all the mechanisms that we report below corroborates an expectation of evolutionary stasis in surveyed populations. We conclude that, although it does not make the statistical explanation less plausible, the biological explanation for evolutionary stasis is supported by our integrative approach. These explanations and the integrative framework that we present here can improve predictive scenarios of adaptive evolution in the wild [7], with implications for biological conservation, climate change mitigation, and the management of genetic resources in agronomy (Box 1).

## The Statistical Explanations

### Predicting the Response to Selection, in Theory

The additive genetic variance ( $V_A$ ) of traits is central to quantitative genetic studies of surveyed populations because  $V_A$  is used as a predictor of the evolutionary potential for trait change under directional selection. From  $V_A$ , we derive other parameters for comparing genetic variabilities [8]. Evolvability, for example, can be estimated by the coefficient of variation ( $CV_A = \sqrt{V_A}/\bar{z}$ ) to evaluate the magnitude of the expected directional response to selection. Narrow-sense heritability ( $h^2 = V_A/V_P$ , where  $V_P$  is the phenotypic variance) is also frequently used, and evaluates the potential of a population to respond to selection. In theory, the univariate and multivariate breeder equations predict the expected change in mean trait value ( $s$ ) as a result of selection ( $\Delta\bar{z}$ ). In the univariate case, this change is proportional to trait

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### Box 1. Applications of Measuring Genetic Variation in Traits

#### Mitigating the Impact of Climate Change

Climate change can increase ecophysiological demands on organisms. Rising mean temperatures, seasonality changes, and drought events leave many organisms with few options: migrate, adapt, or die. The ability of natural populations to respond to selection often depends on those genetic variants that can cope with the changes [45], which gives the potential for rescue in particular species. However, when a response to climate change is observed, this response can also be adaptive plasticity [46]. Uncovering which mechanisms contribute to adaptive changes [35] can inform policies that aim to mitigate the negative impacts of climate change [47].

#### Intraspecific Biodiversity Conservation

Biodiversity is declining because of anthropic environmental impacts that threaten the existence of species and the ecosystem services they provide. There is debate over the criteria for determining which populations or species will benefit from protection [48]. Conservation agencies have established priorities on the basis of taxonomic and intraspecific genetic diversity [49]. However, we are missing the complete link between intraspecific genetic diversity and the long-term ability of natural populations to persist [50]. The biological mechanisms discussed in this review are likely to affect intraspecific biodiversity and the evolutionary response to selection of threatened wild populations.

#### Managing Genetic Resources in Agronomy

In parallel with the threats posed to global food security by climate change [51], there is a need for increased agronomic yield as a result of human population growth and changes in societal practices [52]. The current rates of genetic improvement in yield do not meet demands [53]. The biological mechanisms synthesized in this review can contribute to this issue, but might also be used to alleviate it in the future. Global food security can be improved by integrating these mechanisms to monitor and improve crop yield.

heritability ( $h^2$ ) and the strength of selection (or selection differential,  $S$ ), i.e.,  $\Delta\bar{z} = h^2S$  [9]. These approaches have been successfully applied in plant and animal breeding. However, precisely measuring  $V_A$  and its derived statistics, and measuring selection, is challenging, as detailed below.

#### Oversimplifying Genetic and Environmental Effects Might Bias Predictions

Quantitative genetic studies in surveyed populations often assume an oversimplified view of the genetic basis of traits [10]. Non-additive sources of genetic variation (e.g., epistasis [11] and dominance [12]) are rarely accounted for, and can be confounded with  $V_A$ , which leads to inflated estimates. Environmental causes of phenotypic similarity between related individuals have similar effects, but they are more frequently approximated. This is generally done by including environmental factors [13] or indirect genetic effects (e.g., maternal effects [14]) in the mixed model. Failing to account for these effects can bias estimates of  $V_A$  and therefore the prediction of the response to selection [15].

#### Owing to Precision Issues with Statistical Estimates, Qualitative Predictions Might Be More Realistic

The precision of quantitative genetic and selection estimates are reduced by low statistical power. For example,  $V_A$  estimates often have large confidence intervals [7]. Consequently, these approaches provide an approximate magnitude for the genetic basis of traits (e.g., small, medium, and large  $h^2$ ), and caution must therefore be taken with quantitative predictions or comparisons. The same caution must be taken when measuring selection. In addition, skewed phenotypic distributions can generate false estimates of directional selection [16] because the estimation of selection gradients assumes normality. Nevertheless, our opinion is that discriminating between evolutionary scenarios on the basis of qualitative differences remains possible (e.g., low vs high  $h^2$ ) provided that confidence intervals are discussed and power analyses are conducted.

#### Extrapolating Predictions Can Be Misleading as a Result of Covarying Factors

Extrapolating findings based on  $V_A$  and selection to different contexts (e.g., multiple populations, years, similar environments) is unreliable [7]. This is because these parameters are not independent from covarying factors such as environmental conditions and trait means [8]. They also vary with the method being used (e.g., sibship similarity vs animal models for  $V_A$ ). Estimates are therefore associated with a unique set of conditions and cannot be generalized to other populations. Comparisons between multiple populations can still be achieved but require pedigree connections to quantify the effect of shared genes in different environments [17], or controlled conditions to homogenize environmental effects [18].

#### The Problem of Assigning Causality Between Selection and Genetic Change

Another limit to evaluating the expected response to selection by using  $h^2$  is the rarely verified assumption that selection acts on the underlying genetic variation of traits [19,20]. Any environmental source of covariance between trait and fitness violates this assumption [20], causing biased predictions [19]. An alternative method which relaxes the assumption of causation is the Robertson–Price equation, also known as the secondary theorem of natural selection (STS [19]),  $\Delta\bar{z} = \sigma_A(w, z)$ , where phenotypic change  $\Delta\bar{z}$  is equal to the additive genetic covariance between the trait ( $z$ ) and relative fitness ( $w$ ). Although theoretically correct, its application is nevertheless subject to two limitations. First, examples of genetic variance for fitness are rare (but see [21]). Second, the STS might not always quantify a change caused by selection [19]. Using the STS to estimate phenotypic change caused by selection is misleading because the change in a trait of interest that is not under selection can be driven by its genetic

correlation with any unmeasured trait under selection. This will cause genetic covariance between measured traits and fitness [19]. A possible solution is to adopt a multivariate approach to selection (genotypic selection gradient [21]). Such an approach might lead to a more accurate prediction of evolutionary change by selection on phenotypic traits.

Our interpretation of evolutionary processes and biological mechanisms is based on statistical measures (e.g.,  $V_A$ ). The limitations of these measures imply that it might be statistical failures, rather than biological mechanisms, that cause a discrepancy between predictions and observations of microevolutionary change. However, qualitative predictions for evolutionary scenarios are still possible. The conclusion that there is a missing response to selection might therefore be misleading if the expected response to selection is misestimated.

## The Biological Explanations

### The Mechanisms We Know, and the Mechanisms We Do Not Know

The hypothesis that biological mechanisms might be responsible for the missing response to selection in surveyed populations is widely acknowledged [2]. Many biological mechanisms, such as phenotypic plasticity, genetic correlations, indirect genetic effects, and age-specific responses, are well known to interfere with the connection between genetic variation and the response to selection, but are not always accounted for (Box 2). In addition, fluctuating selection affects the spatial and temporal scale at which we detect selection (Box 2). Figure 1 graphically depicts the impact of each of these mechanisms on the response to selection that was predicted from the breeder's equation ( $R$ , represented on the horizontal axis). The vertical axis depicts the departure from the baseline expectation that  $V_A$  will decrease following selection. For example, in the case of negative genetic correlations between traits under positive selection, the response to selection is expected to be constrained, and genetic variation maintained (Figure 1Bii). We present below other mechanisms to incorporate into the list of biological explanations.

### Demography and Connectivity Shape the Potential of Populations to Respond to Selection

Population demography can affect  $V_A$  and selection, but this is neglected in most quantitative genetic studies of surveyed populations. For instance, a sharp decrease in population size (e.g., population founding event or collapse) reduces genetic diversity (genetic bottleneck [22]),  $V_A$ , and the response to selection (Figure 2A) [23,24]. Conversely, genetic connectivity between populations with different adaptive optima can limit local adaptation but increase or restore genetic variability and adaptive potential [25,26]. In addition, a negative correlation is expected between the population growth rate and the variation in relative fitness (the opportunity for selection) [27]. Thus, declining populations are expected to have a higher opportunity for selection, and might therefore experience stronger selection [28]. Although there have been some empirical studies on the effects of demography and connectivity, these effects have mostly been explored through theoretical work. Their applicability and prevalence in the wild remain to be addressed.

### The Unknown Impact of Coevolution on Evolutionary Predictions

Interacting species that exert reciprocal selective pressures upon one another are widely documented in host–pathogen, predator–prey, and mutualistic interactions [29]. However, coevolution is largely ignored in studies of surveyed populations (but see [30,31]). We argue that neglecting these aspects can lead to the misestimation of  $V_A$  and the response to selection (Figure 2B). The classic illustration of this is the promotion of genetic variation in vertebrate major histocompatibility genes through coevolution with pathogens [32]. In addition, pathogens can maintain genetic variation in other traits of host species [33]. However, these studies

**Box 2. Widely Acknowledged Biological Mechanisms**

## Phenotypic Plasticity

Phenotypic changes in wild populations often result from plasticity rather than from microevolution [5,46]. This shortcutting of selection can result in genetic variation being maintained. When plasticity varies among the genotypes (genotype-by-environment interactions) under selection [54], the magnitude and/or the direction of selection can be altered and less predictable [55]. Genetic assimilation, whereby phenotypic plasticity is lost and the trait becomes canalized by genetic variation [56], is a particular case of response to selection [57], although its prevalence in wild populations remains unclear.

## Genetic Correlations among Traits

Traits that are genetically correlated do not evolve independently [58,59]. When this correlation is antagonistic to the direction of selection, the response to selection is constrained [60] because a change in one trait reduces fitness through the effect on the other trait. Consequently, genetic variation can be maintained. Conversely, when selection and genetic correlations are aligned, this favors the response to selection [6], and depletes genetic variation, compared to baseline expectations. Globally, genetic correlations were found to have both effects on selection [61].

## Indirect Genetic Effects (IGEs)

IGEs arise when the phenotype of a given individual is affected by the transmissible traits of others with whom it interacts [62], which can influence the response to selection [63]. This is clearly demonstrated by parental effects [64,65]. Furthermore, antagonistic selection between parental and offspring traits could constrain the response to selection [66]. Many studies neglect parental effects, potentially biasing heritability estimates and baseline expectations [67].

## Age-Specific Responses

Quantitative genetic evidence for senescence, usually defined as the decrease of survival and reproductive prospects with age, is common in wild animal populations [68], but is far from ubiquitous in plants [69]. This age-specific decline in selection often results in higher heritable variation in older age classes [70]. Age structures of natural populations are rarely accounted for in quantitative genetic studies [71], and this can misleadingly affect the predictors of evolutionary potential.

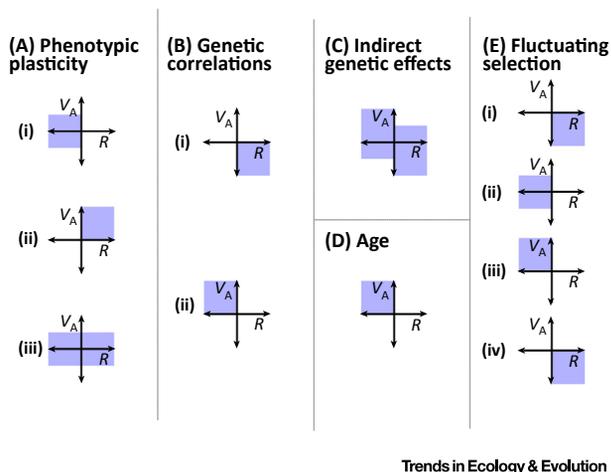
## Fluctuating Selection

Fluctuations in selection strength and/or direction exist over short spatial and time scales [72,73], but their evolutionary significance remains uncertain in nature [74]. Strong fluctuations occurring faster than the production of variation can reduce genetic variation more than expected, although intermediate slow fluctuations maintain higher genetic variation. Neglecting fluctuations can lead to erroneous estimates of the strength and direction of selection, particularly if selection changes sign at unmeasured times and/or locations.

generally estimate parameters in only one of the interacting species, while a full demonstration of coevolution requires that these are shown in both partner species [33]. Such simultaneous estimates remain virtually absent. Filling this gap should confirm predictions that host–pathogen coevolution promotes  $V_A$  [34]. Taking into account coevolution would improve the detection of selection pressures and responses that otherwise would not have been detected, or would have been overestimated.

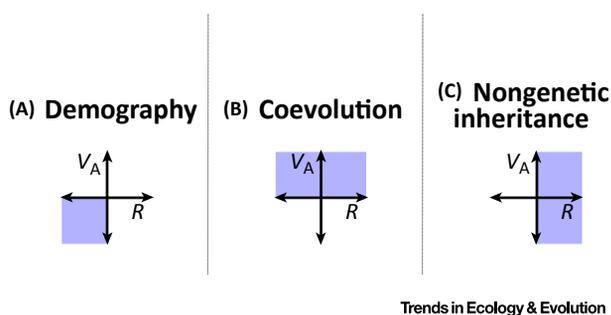
**The Potential Significance of Nongenetic Inheritance**

Where adaptive phenotypic changes were detected in surveyed populations, only one third of these were associated with genetic change in response to selection [35]. This raises questions about the mechanisms underlying the response to selection, in particular whether there is a contribution of nongenetic inheritance. Theory predicts that inherited nongenetic variation can respond to natural selection [36,37] (Figure 2C). Nongenetic inheritance might therefore account for a missing fraction of the genetic response to selection in the wild [39,40]. Equally,



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**Figure 1. Departure from Baseline Evolutionary Expectations: Widely Acknowledged Mechanisms.** Quadrant plots illustrate the effect of widely acknowledged mechanisms on the response to selection ( $R$ ) and the change in additive genetic variation ( $V_A$ ) relative to baseline predictions. Mechanisms increasing the response to selection lie to the right of the  $y$  axis, and those decreasing it lie to the left. Mechanisms causing the maintenance of additive genetic variation lie above the  $x$  axis, those eroding it lie below. Where expectations are close to baseline predictions, the effect remains centered around the axis. These mechanisms are (A) phenotypic plasticity, (B) genetic correlations, (C) indirect genetic effects (maternal genetic effect), (D) age effects, where old age classes are often associated with increased additive genetic variation, and (E) fluctuating selection. Phenotypic plasticity (A) was split into (i) the effect of plasticity itself, (ii) the effect of the canalization of plastic trait variation that becomes constitutively expressed, as in the case of genetic assimilation, and (iii) genotype-by-environment interactions. Genetic correlations (B) were split into (i) the effect of genetic correlations aligned with the direction of selection, and (ii) the effect of genetic correlations that are antagonistic with the direction of selection. Fluctuating selection (E) was split into (i) the effect of a low-frequency fluctuation with sign changes, (ii) effect of fast frequency fluctuation with sign changes, (iii) low-amplitude fluctuation with sign changes, and (iv) low-amplitude fluctuation with consistent sign.



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**Figure 2. Departure from Baseline Evolutionary Expectations: New Mechanisms.** In this figure we present in quadrant plots the effect of mechanisms that in our opinion should also be considered to affect the response to selection ( $R$ ) and the change in additive genetic variation ( $V_A$ ) relative to baseline predictions. Mechanisms increasing the response to selection lie to the right of the  $y$  axis, and those decreasing it lie to the left. Mechanisms causing the maintenance of additive genetic variation lie above the  $x$  axis, those eroding it lie below. Where expectations are close to baseline predictions, the effect remains centered around the axis. The mechanisms outlined here are (A) demography; we show here the effect of founding events, or long-term small population size associated with genetic drift, (B) coevolution, and (C) nongenetic inheritance.

nongenetic inheritance can lead to inflated estimates of  $h^2$ , contributing to the mismatch between the predicted and observed phenotypic response to selection. Apart from parental environmental effects, nongenetic components of heritability cannot yet be estimated in surveyed populations. Nevertheless, two studies disentangled genetic and nongenetic components of phenotypic similarity [41,42], which is an important first step [43]. There is a need to

improve both theoretical and empirical understanding of nongenetic inheritance, particularly to (i) assess the long-term stability of nongenetic inheritance mechanisms, (ii) build a theoretical framework taking into account their key properties, and (iii) test for their role in the response to selection.

### Towards an Integrative View of Biological Mechanisms

Biological mechanisms acting in isolation have been found to influence the response to selection in surveyed populations [2,44], but an integrative perspective of their combined action is lacking. As a result, two important questions remain unanswered. First, can we refine our expectations of the response to selection if we identify the biological mechanisms acting in a wild population of interest? Second, do most mechanisms constrain the response to selection and maintain  $V_A$ ? If the majority of biological mechanisms constrain the response to selection, this would support a biological explanation for evolutionary stasis. Until these questions are addressed, the standing evolutionary potential of wild populations cannot be properly evaluated. We can improve our predictive capabilities by integrating predictions from well-studied mechanisms, but there is currently no framework for assessing their combined impact. We build here a qualitative framework as a first version of an integrative theory of their impact on the evolutionary potential of wild populations to respond to directional selection.

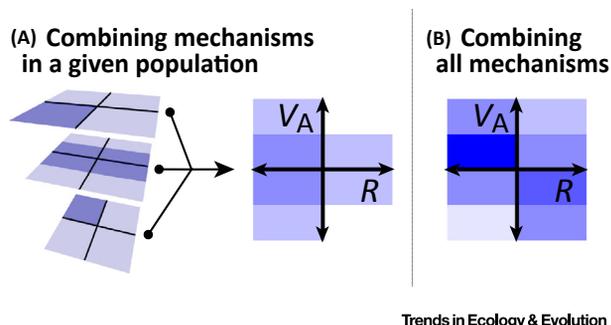
### How to Refine Our Baseline Expectation of the Response to Selection

The basis of this approach takes the predicted response to selection ( $R$ ) from the breeder's equation, and the consequent reduction in  $V_A$  as a baseline expectation. Figures 1,2 illustrate the deviation from these baseline expectations caused by individual mechanisms (indicated by the shaded regions). Assuming an equal weighting of these mechanisms, we can qualitatively combine the effects of individual mechanisms the most likely evolutionary scenario in a given population. It is then possible to adjust baseline expectations for a particular population.

Implementing this graphical tool should be possible in any population, and give testable predictions to refine our expectations of evolutionary change. Figure 3A illustrates how the impact of three mechanisms can be qualitatively combined for an imaginary population. In our example, an imaginary population is introduced to a new habitat, leading to a founding event that increases the loss of  $V_A$  and decelerates the response to selection (Figure 2A). In this new environment, different genotypes have different levels of plasticity, which does not affect the baseline predictions for  $V_A$  but can lead to a more uncertain prediction of  $R$  (genotype-by-environment interactions; Figure 1Aiii). Furthermore, two traits under positive selection in this population have a negative genetic correlation that is not aligned with the direction of selection, thereby constraining the response to selection and maintaining  $V_A$  (Figure 1Bii). Thus, combining these mechanisms gives us the most likely evolutionary scenario in this imaginary population (illustrated by the darkest sector on the combined graph in Figure 3A): a reduced response to selection compared to baseline prediction, and a change in  $V_A$  that is likely to conform with the baseline prediction. An emergent property of this framework is that it can identify when disparate species and populations are expected to respond similarly to selection owing to parallels in the effects of biological mechanisms.

### Taking Steps Towards a Global Understanding

This integrative approach could ultimately be applied to many mechanisms, populations, and species to refine the predictions of the response to selection and changes in genetic variation. Figure 3B illustrates the result of an integration of all the mechanisms outlined here. The darkest sector in the combined plot indicates a reduced response to selection compared to our baseline expectation, and the maintenance of a slightly higher than expected level of genetic



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**Figure 3. An Integrative Framework for Predicting Microevolutionary Change.** We show here how to integrate multiple mechanisms into our framework. The quadrant plots illustrate the effect of mechanisms that affect the response to selection ( $R$ ) and the change in additive genetic variation ( $V_A$ ) relative to baseline predictions. Mechanisms increasing the response to selection lie to the right of the  $y$  axis, and those decreasing it lie to the left. Mechanisms causing the maintenance of additive genetic variation lie above the  $x$  axis, those eroding it lie below. Where expectations are close to baseline predictions, the effect remains centered around the axis. Mechanisms are combined by superimposing the quadrant plots of their effects, which reveals a darkened area that corresponds to the most likely predictions about the response to selection and changes in genetic variation, relative to baseline expectations. Plot (A) illustrates how a sample of mechanisms known to characterize a particular population can be integrated. This illustrates predictions for an imaginary population, where founding effects, gene-by-environment interactions, and negative genetic correlations between traits under positive selection are combined to give predictions of reduced response to selection, accompanied by relatively negligible changes in genetic variation compared to the baseline expectation. Plot (B) illustrates the combined effect of all the mechanisms listed in the main text.

variation. This integrative perspective therefore appears to match the general observation of evolutionary stasis, thereby corroborating the biological explanation for stasis. However, a range of scenarios other than evolutionary stasis remains possible, indicated by the broad shading across Figure 3B, although empirical evidence for these scenarios is scarce.

It is important to note that the statistical explanations are not refuted by this approach, and the imprecision of quantitative genetic estimates therefore still needs to be taken into account. There is also the caveat that our list of mechanisms is not exhaustive, but other mechanisms might be of interest, such as sexual selection and those that are yet to be documented, and can be incorporated into this framework. The frequency with which different mechanisms occur in natural populations remains unclear because that reported in published studies might be an artefact of research motivated to find explanations for evolutionary stasis. This could be clarified by broadening the range of study systems. Future work could integrate additional mechanisms and weight all mechanisms by their frequency of occurrence and the relative strength of their effects.

### Concluding Remarks

The past 20 years of quantitative genetic studies in surveyed populations have confirmed that genetic variation for traits is not always synonymous with evolutionary potential, and that the expected response to selection is often missing. Although initially paradoxical, multiple explanations have been found for these frequent cases of stasis. The widely acknowledged statistical explanations point to the imprecision of empirical measures of quantitative genetic variation and selection as a likely explanation. However, biological mechanisms studied in isolation can cause a mismatch between the predicted and observed response to selection. The unified framework that we propose implies that the combined action of these mechanisms is likely to limit the response to selection of wild populations in general. However, quantitative genetic studies of surveyed populations are restricted to a relatively small number of cases. Their extension to other systems might add further mechanisms to this framework and contribute to our understanding of microevolution.

### Outstanding Questions

What is the relative strength of impact of each biological mechanism affecting the response to selection? We provide a framework where equal weighting is given to each mechanism, but this might not always be the case. In addition, these mechanisms might have non-additive interactions. Comparing our qualitative predictions to what is observed will inform us about the relative weighting the quantitative effects of these mechanisms.

What is the biological foundation for the observation that expected responses to selection are missing in the wild? The predictions from isolated mechanisms affecting the response to selection cover a range of evolutionary scenarios (response to selection and changes in genetic variation) that have not been observed in the wild. Whether this is an artefact of current research or reflects biological reality will become clearer with an increase in the number of quantitative genetic studies in diverse study systems.

Why might some species or populations share similar suites of mechanisms? Essentially, are there shared selection syndromes in the wild? Similar evolutionary changes could be a result of parallels between populations in the mechanisms influencing genetic variation and the response to selection. Using the integrative framework in multiple populations will reveal patterns across disparate species and populations.

From a fundamental perspective, we highlight that there is a mixture of statistical and biological explanations for evolutionary stasis. These two explanations are best considered in tandem because statistical uncertainty can have a biological origin, and the consequences of biological mechanisms are often discovered by revealing a statistical deviation from a prediction. From an applied perspective, this approach could be valuable to conservation biologists and agronomists by providing a tool for predicting the ability of a particular population to respond to selection. Biological mechanisms that are identified in managed populations could be manipulated to improve the prospects of population persistence. Although there is a general focus on the constraints imposed by biological mechanisms, in our opinion these mechanisms could also be considered as a force maintaining a transient form of evolutionary potential through conserving genetic variation. When the environment, genetic background, or selection pressures change, this potential may be released. The broader picture painted by our integrative approach is timely because it informs our expectations of the response to selection induced by anthropic environmental change, and could be used to mitigate its overall impact.

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