careful appraisal of its strengths and weaknesses is warranted. One concern is that experimental studies of microevolution can be difficult to compare owing to the diverse techniques employed. A consideration of the two studies highlighted by Svensson (Reznick's work on guppies[2] and Losos's work on *Anolis* lizards[3]) illustrates this problem. Reznick calculated rates of evolution in his introduced populations after capturing individuals from them and rearing their offspring for two generations in the laboratory (to ensure that he was quantifying genetic change)[2]. In contrast, Losos measured divergence among wild-caught lizards whose ancestors had been introduced to different islands[3], a comparison that would likely include a combination of environmental and genetic effects. Clearly the works of Reznick and Losos, both worthy of emulation, should not be considered equivalently in any calculation of evolutionary rates. Interestingly, any changes in the fossil record would reflect a combination of both genetic and environmental effects.

A second cautionary note arises from the use of two endpoints to assume a trend. Losos *et al.* apparently calculated evolutionary rates based on differences between an ancestral population and derived island populations that were sampled in a single year[3]. Reznick *et al.*'s calculation of evolutionary rates was based on a one-time comparison after 11 years in the Aripo River and two comparisons (after four and 7.5 years) in the El Cedro River[2]. Although both of these studies provided strong evidence for rapid evolution, two or three data points are rarely sufficient to accurately describe a relationship between two variables. In fact, estimates of change using only endpoints often mislead when extrapolated or interpolated to other time frames.

As has been suggested by others[4,5], studies based on time-series in introduced populations may provide a better way to evaluate the potential link between microevolutionary and macroevolutionary change. One example of such a time-series is the migratory timing of American shad introduced to the Columbia River system in the late 1800s (Ref. 6). From 1938 to 1993, the average date of migration for shad past Bonneville Dam has shifted 40 days earlier, equivalent to a rate of 0.73 days/year (predicted from a regression through the 55 years of data)[6]. If, however, the migratory timing of shad was known for only two of the years, say 1973 and 1984, we would have erroneously concluded that the rate of change was in the opposite direction at 1.6 days/year. Other random choices of any two years from the data set would yield spectacularly varying interpretations. Long-term data sets on introduced populations have the potential to determine if observed microevolutionary rates are sustained over time, or if they fluctuate or level-off to such a degree that they would be unlikely to result in macroevolutionary responses.

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Costs and limits of phenotypic plasticity

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The costs and limits of phenotypic plasticity are thought to have important ecological and evolutionary consequences, yet they are not as well understood as the benefits of plasticity. At least nine ideas exist regarding how plasticity may be costly or limited, but these have rarely been discussed together. The most commonly discussed cost is that of maintaining the sensory and regulatory machinery needed for plasticity, which may require energy and material expenses. A frequently considered limit to the benefit of plasticity is that the environmental cues guiding plastic development can be unreliable. Such costs and limits have recently been included in theoretical models and, perhaps more importantly, relevant empirical studies now have emerged. Despite the current interest in costs and limits of plasticity, several lines of reasoning suggest that they might be difficult to demonstrate.

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Adaptive phenotypic plasticity – the potential for an organism to produce a range of different, relatively fit phenotypes in multiple environments – seems like the pinnacle of evolution. We now recognize that plasticity has many ecological benefits[1–3], but what constrains its evolution? One widely acknowledged answer is that there is often a lack of sufficient genetic variation in plasticity to allow evolution[4,5]. Given sufficient genetic variation, however, plasticity may still not evolve because of inherent costs or problems that limit the efficacy of plasticity. These costs and limits have recently become an important focus of ecological and evolutionary thought. Costs of plasticity, in particular, are now frequently cited as an important subject needing further study[1,3,6–10]. However, the breadth of ideas on costs and other constraints on plasticity may be underappreciated; until now, these ideas had not been presented and distinguished in a single paper.

Constraints on plasticity have several ecological and evolutionary implications. For example, costs may have the evolutionary consequence of reducing the degree of plasticity that evolves[5,11] or of maintaining genetic variation for plasticity[12]. Constraints on plasticity may have the ecological consequence of shifting the competitive advantage between fixed and plastic taxa[5,13] or of promoting coexistence[14,15]. Given the potential importance of constraints, it is unfortunate that they remain an abstraction with little empirical information on their existence and magnitude in nature[6,16].

In this article, we review current ideas regarding constraints on plasticity and the distinctions among various costs and limits of plasticity. We review recent empirical tests for the constraints and discuss why evidence for them may remain elusive. Finally, we present the methods that have emerged for measuring costs of plasticity.

Costs versus limits of plasticity

The benefit of plasticity is the ability to produce a better phenotype–environment match across more environments than

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Box 1. Nine potential costs and limits of phenotypic plasticity

Costs of plasticity

- *Maintenance costs*: Energetic costs of the sensory and regulatory mechanisms of plasticity.
- *Production costs*: The production cost of inducible structures has been viewed by some as a cost of plasticity. Other authors disagree because production costs are also paid by fixed genotypes. In some cases, the production costs that plastic genotypes pay will exceed those paid by fixed genotypes; the excess is a true cost of plasticity.
- *Information acquisition cost*: The process of acquiring information about the environment may be risky, involve energy for sampling, or reduce foraging or mating efficiency.
- *Developmental instability*: Phenotypic imprecision may be inherent for environmentally contingent development. Imprecision can result in reduced fitness under stabilizing selection.
- *Genetic costs*: (1) Linkage – genes promoting plasticity may be linked with genes conferring low fitness. (2) Pleiotropy – plasticity genes may have negative pleiotropic effects on traits other than the plastic trait. (3) Epistasis – regulatory loci producing plasticity may modify expression of other genes.

Limits to the benefit of plasticity

- *Information reliability limit*: Plastic organisms can produce maladapted phenotypes when they are wrong about the environment, or, when correct initially but the environment changes.
- *Lag-time limit*: A plastic strategy must invoke development to alter phenotypes. The lag-time between an environmental change and a phenotypic response can reduce fitness.
- *Developmental range limit*: Fixed development may be more capable of producing adaptive, extreme phenotypes than facultative development.
- *Epiphenotype problem*: Plastic add-on phenotypes may be ineffective compared with the same phenotypic element that is integrated during early development.

would be possible by producing a single phenotype in all environments[17]. If constraints did not exist, organisms should exhibit 'perfect' or 'infinite' plasticity, expressing the best trait value in every environment with no cost for having that ability. Generally, plastic organisms fail this ideal because of an inability to consistently produce the optimum (i.e. a limitation) or because they pay a cost merely for the ability to be plastic[1,3,9–11,13,18]. Specifically, we can distinguish between costs and limits to the benefit of plasticity as follows. A cost of plasticity is indicated in a focal environment when a plastic organism exhibits lower fitness while producing the same mean trait value as a fixed organism.

In contrast, a limit of plasticity is evident when facultative development cannot produce a trait mean as near the optimum as can fixed development. For example, compare the phenotypes and associated fitnesses of two rotifer clones, one that is plastic and the other fixed. If the fixed and plastic clones produce the same spine length in a focal environment, yet the one that is plastic across environments exhibits lower fecundity, then that indicates a cost of plasticity. If the more plastic rotifer did not produce as long a spine as the fixed rotifer (and if longer spines are favored by selection), then that indicates a limit of plasticity.

The failure of plastic organisms to produce optima is not a cost of plasticity. Inappropriate phenotypes can be exhibited by both fixed and plastic organisms. In fact, the worst phenotype–environment mismatches are made by fixed organisms that are specialized to one environment but find themselves in an alternative environment. Phenotype mismatches by plastic genotypes are therefore best regarded as limits to the benefit of plasticity. Both costs and limits constrain evolution because each reduces the net value of realized plasticity compared with perfect plasticity.

Constraints on plasticity seem likely to occur because of differences that can exist between fixed and plastic development. Fixed development requires only production machinery (structural genes, polymerases, ribosomes, etc.), which leads to an expected phenotypic outcome (i.e. a mean phenotype, $\bar{z}$, typically with some variance, σ^2):

$$\text{production machinery} \rightarrow \bar{z} \pm \sigma^2$$

Plastic development can require this step alone if the mechanism of plasticity is allelic sensitivity[8,9], that is, the direct response of structural genes or their products to the environment. However, plasticity might additionally involve sensing cues about the environment, processing that information and invoking regulatory mechanisms before the production machinery is engaged:

$$\text{detect environment} \rightarrow \text{process information} \rightarrow \text{regulatory mechanism} \rightarrow \text{production machinery} \rightarrow \bar{z} \pm \sigma^2$$

The accessory steps in producing phenotypes through plastic development offer additional opportunities for costs or limits to manifest relative to fixed development. Costs of plasticity could arise during development even when the end-products of fixed and plastic development are identical. Limits to plasticity may arise that involve the phenotypes produced, even when the energetic demands of fixed and plastic development are similar.

Nine basic ideas exist on constraints of plasticity. Our definitions of these mechanisms are presented below and summarized in Box 1.

Costs of plasticity

Maintenance costs

Maintenance costs could be incurred by plastic organisms if facultative development requires the maintenance of sensory and regulatory machinery that fixed development does not require[5,11,13,18]. For example, the ethylene growth response in plants requires the response of an ethylene receptor protein on the cell membranes of plants[19]. If development were insensitive to ethylene, the energetic and material cost of producing the receptor protein (i.e. the sensory machinery) could be saved.

Production costs

Mixed views have appeared regarding whether production costs of induced structures are truly costs of plasticity. Many authors have demonstrated costs of environmentally induced character states, and have cited these character production costs as costs of plasticity (for animals see Refs 20–22; for plants see review in Ref. 7). For example, *Daphnia* with predator-induced spines can exhibit reduced reproductive capacity compared to those lacking spines[22]. However, the production cost of spines should not be considered a cost of plasticity, because organisms fixed for spines could pay the same spine-production cost. If organisms with fixed and plastic development pay the same production cost for a trait, then production costs cannot be viewed as costs unique to and inherent for plastic genotypes[6,16]. Data on character production costs are still important to obtain, however, because they are needed to assess the net value of plasticity. Production costs measure the cost saved by not producing the character in a non-inducing environment (i.e. one benefit of plasticity). Viewed another way, character production costs are also the cost of mistakenly producing a character when it is not necessary (i.e. the cost of a phenotype error).

By our definition, production costs should only be considered costs of plasticity if the cost of production is greater for plastic genotypes than for fixed genotypes producing the same phenotype. Scheiner (cited in Ref. 16) suggested that production costs of plasticity may exist in some cases under the strict definition. Consider a lineage of zooplankton with fixed spine production during a stage of ontogeny in which the production cost of the spine is low. A second lineage that employed facultative development to produce the spine, conditional upon sensing a chemical cue, might often produce the spine during an ontogenetic stage in which spine production is expensive. In this example, a

plastic strategist pays a greater production cost for the same phenotype as its fixed counterpart.

Information acquisition costs

The process of being plastic could entail an information acquisition cost – the cost of acquiring information about the environment[5,23,24]. For example, to express the optimal antipredator phenotype, prey must gather information on the local predation regime. To detect the presence, type or motivational state of predators, prey often must perform predator inspection behavior, which can be risky[25]. The process of sampling the environment can also have energetic costs associated with locomotion, or can require temporary reductions in foraging[26] or mating efficiency[27].

Developmental instability

Several authors have suggested that there may be an intrinsic connection between plasticity and developmental instability (see review in Ref. 28). Developmental instability is either measured as the within-environment phenotypic variance for a given genotype or as fluctuating asymmetry[28]. Both of these types of developmental instability can produce low fitness. For example, asymmetry can reduce performance traits[29] and broad phenotype distributions have lower fitness than narrow distributions under stabilizing selection[30]. If plasticity is associated with imprecise development, then this can result in reduced fitness that represents a cost of plasticity[16].

Despite the common supposition that plasticity and developmental instability could theoretically be connected, empirical evidence suggests that the two developmental parameters are generally unrelated phenomena[16,28]. When correlations are detected between developmental noise and plasticity, the relationship tends to be trait- or environment-specific. For example, Scheiner *et al.*[28] found that plasticity was correlated with developmental instability in a subset of the traits he studied, but only in one environment.

Genetic costs

Little is known in detail about the genetics of plasticity[3,8–10,31], although research in this area is increasing (see review in Ref. 10). We generally expect that plasticity manifests either because structural genes or their products are directly affected by the external environment ('allelic sensitivity') or because regulatory genes are affected by the environment and in turn affect the expression of structural genes[9]. Potential costs associated with either form of plasticity gene include: (1) linkage, where genes conferring adaptive benefits of plasticity are linked to costly genes for other traits[6,32]; (2) pleiotropy, where genes conferring adaptive plasticity on a trait also confer negative direct effects on other traits[13]; and (3) epistasis, where plasticity genes alter the expression of other genes and hence indirectly affect other traits.

Some genetic correlations may reflect the basis underlying one of the other mechanisms of plasticity cost. For example, there could be a genetic correlation between plasticity and a costly sensory mechanism that is necessary for the process of plasticity.

Limits to the benefit of plasticity

Information reliability limits

As noted earlier, producing a plastic response may require information about the environment. In addition to the cost of information acquisition, however, there can be problems associated with poor reliability of cues used to assess the environment[11,13,14,17,23,24,33]. Plastic organisms with unreliable information express poor phenotype–environment matching.

Lag-time limits

Temporary problems with poor phenotype–environment matching can also be generated by a lag-time between sensing and responding to environmental cues[2,13,17,34]. For traits that are flexible on a short time-scale (e.g. behavior or physiology), lag-time is minimal. In contrast, lag-time can be substantial for induced morphological traits[2,34] (e.g. spine or trichome growth). Lag-time problems can be mitigated if organisms use reliable indirect cues that predict an impending environmental shift. For example, copepod zooplankton use photoperiod as a cue to induce diapause and avoid the seasonal onset of heavy predation (see review in Ref. 35). The indirect nature of such cues may solve the lag-time problem; however, they can impose information-reliability problems.

Developmental range limits

Following the familiar saying that 'a jack-of-all-trades is a master of none', we might expect that fixed development would be more able than plastic development to produce extreme phenotypes. In other words, there may be a trade-off between the developmental range that can be expressed across habitats and the magnitude of expression that can be achieved within environments[4,13,15,16]. An example would be if plants fixed for elongate stems were able to produce longer stems than inducible plants could produce (when longer stems were favored).

Epiphenotype problem

A phenotype built as an add-on device may not be as good as one that is integrated during early development (i.e. the 'epiphenotype' problem)[16]. For example, a zooplankter growing a spine on an existing carapace may produce a weaker spine than if it had produced the carapace and spine together during development. Another example involves aperture shape in snails; the accretionary growth of shells implies that a transition in shell phenotype due to plasticity could create a weak point (a fault) that is a point of vulnerability.

What are the odds of detecting costs?

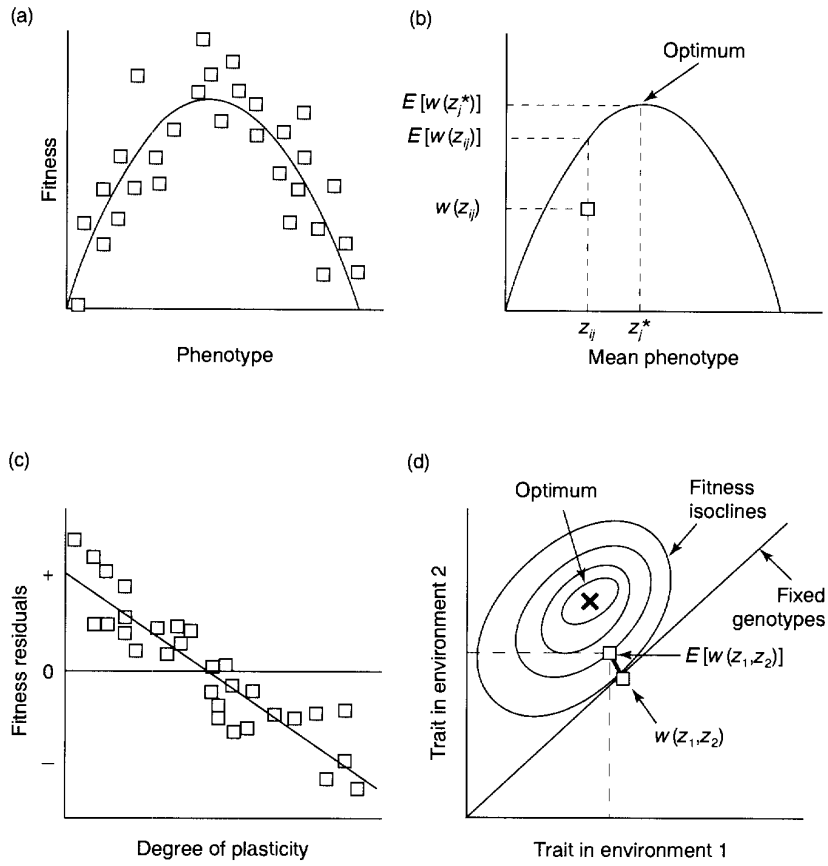
Here we question three expectations that underlie the search for costs of plasticity: (1) the expectation that plasticity is necessarily costly, (2) the expectation that costly but nonadaptive plasticity should persist in nature, and (3) the expectation that plasticity will remain costly over evolutionary time.

First, whether we expect plasticity or constancy to be more costly depends on whether the focal trait exhibits plasticity as an 'active' or 'passive' (*sensu* Ref. 10) response to environmental variation (i.e. Schmalhausen's 'autoregulatory' versus 'dependent morphogenesis', respectively[30]; see also Stearns[32] for a semantic discussion). For example, ectothermic animals usually show low metabolic rates, and low feeding, growth and developmental rates as a passive response to cool temperatures. If the normal effect of the environment is to alter the phenotype, then phenotypic constancy over a range of environments may be more costly than plasticity. Constancy in this case would require a regulatory apparatus or alternative structural alleles to resist environmental influences[5,30,36]. Constancy in passively plastic traits might require active plasticity at a more mechanistic level[37]. For example, endothermic animals use a variety of plastic thermoregulatory mechanisms to produce relatively constant basal metabolic rates despite variations in environmental temperature. Thus, whether we expect plasticity or constancy to be more costly depends on the type of plasticity[5].

Second, detecting a cost of plasticity may only be plausible for adaptive plasticity, rather than for arbitrary environmental effects on the phenotype[16]. Costly but maladaptive or neutral phenotypic responses are expected to go extinct. Thus we only expect costly forms of plasticity to persist if they are compensated for by benefits. Again, the benefit of plasticity is that it can mitigate a functional trade-off imposed by alternative environments[17]. If there is no prior knowledge that plasticity in a trait is adaptive, then tests for costs can be premature.

A problem arises even when examining adaptive plasticity. In theory, adaptive

Box 2. Measuring the costs and limits of plasticity



Recent papers have suggested a quantitative method for assessing the costs and limits of plasticity[5,16]. The key first step in detecting costs and limits of plasticity involves quantifying the relationship between phenotypic values and fitness in each environment using regression analyses or cubic splines (see review in Ref. 40). This allows us to calculate the expected fitness for a given genotype in each environment based on its mean phenotype (= genotypic value) in that environment. It also identifies the optimal phenotype and the maximum fitness possible in each environment [see (a) for a hypothetical example, where each point represents a genotype]. A genotype has made a phenotypic error if its mean phenotype differs from the optimal phenotype. The cost of a phenotypic error is the difference between the maximum fitness, $w(z_j^*)$, and the expected fitness for a given genotype based on its mean phenotype, $E[w(z_{ij})]$, where z_{ij} is the phenotype of genotype i in environment j, and w is fitness (b). Both plastic and fixed genotypes can make phenotypic errors. If the errors are intrinsic to plastic development, then this constitutes evidence for a limit to plasticity.

Actual fitness for a particular genotype, $w(z_{ij})$, will often not be identical to its expected fitness based on its mean phenotype (b). Some genotypes will have a higher than expected fitness and others a lower than expected fitness. A cost of plasticity is indicated if there is a negative relationship (across genotypes) between the genotypes' degree of plasticity and the fitness residuals (calculated from the regression of fitness on mean phenotype). That is, a cost of plasticity exists if fixed genotypes tend to have higher than expected fitness, while more plastic genotypes tend to have lower than expected fitness for a given phenotype (c). In statistical terms, the cost of plasticity is measured by the partial regression of fitness on plasticity after accounting for effects of mean phenotype on fitness.

The basic logic and method can be extended to any number of environments. Expected and actual fitnesses across multiple environments should account for the relative frequency of the different environments. As in the simple, one-environment analysis, a cost of plasticity is indicated if fitness residuals, $E[w(z_1,z_2)] - w(z_1,z_2)$, are negatively related to mean plasticity. In (d) a graphical example for the two-environment case is shown, where fitness isoclines are lines of equal fitness.

forms of plasticity should often go to fixation within populations, erasing the variation necessary to make comparisons between plastic and fixed strategies[16]. Theoretical models demonstrate that it is possible for a polymorphism to exist for the degree of plasticity in a population[14,15] and many empirical studies have demonstrated genotype–environment variance. However, the empirical studies have not always taken a functional approach, so the relative frequency of genotype–environment variance for adaptive versus nonadaptive plasticity is unclear. A potential solution to the problem of adaptive plasticity becoming fixed within populations is to perform multiple population comparisons[16]. Even if little variance in plasticity exists within populations, sufficient variation may exist among populations (owing to stochastic processes such as founder events and genetic drift, or because of different selection pressures among populations).

Finally, allelic variants producing plasticity at a cost should gradually be replaced by genetic mechanisms promoting the plasticity without cost or with reduced cost. Thus, natural selection should reduce costs over evolutionary time so that they disappear or become small and difficult to detect empirically.

The implications of the above comments are that we can expect the appropriate variation in adaptive plasticity to exist only for some traits, in some populations and for some species. Such a sparse distribution of plasticity costs among different organisms and traits means that many empirical tests will need to be conducted for costs before we can make generalizations about which costs exist, how common they are, and under what circumstances they occur[16].

Measuring plasticity costs

Although plasticity is easily documented without quantifying genetic relationships among organisms (e.g. Refs 38 and 39), the parameters needed to detect costs of plasticity require controlled quantitative genetic experimental designs[5,12]. Generally, related groups of individuals (full or half sibships, clones, etc.) are raised in two or more environments to estimate components of phenotypic variation due to genetic affiliation, environment and genotype–environment interactions. Each genotype is assigned a measure of its degree of plasticity (usually calculated as the difference between family means in alternative environments[1]). Plasticity can then be correlated with cost- or limit-indicating variables such as growth and fecundity (e.g. to test for maintenance costs). The general procedure is outlined in Box 2.

A recent empirical study[16] explicitly tested for costs and limits of predator-induced plasticity in shell morphology and growth rate in a freshwater snail (*Physa heterostropha*) that has two major predators (crayfish, *Orconectes obscurus*, and sunfish, *Lepomis gibbosus*). DeWitt[16] tested for energetic costs of plasticity, developmental instability and impaired developmental range for plastic genotypes relative to more-fixed genotypes. Twenty-nine snail families from a single population were raised in the following three environments: with fish, with crayfish and without predators. The snails exhibited adaptive

induced responses based on the capture techniques of the predators. Fish are shell-crushing predators that prefer large snails, whereas crayfish are shell-entry predators that selectively eat small snails. Snails responded to fish by producing crush-resistant (rotund) shells and by slowing growth relative to their sibs raised with crayfish. Snails raised with crayfish produced entry-resistant (elongate) shells and increased growth by delaying egg production. One cost of plasticity was indicated – snail families having the greatest morphological plasticity exhibited reduced growth relative to less plastic families. The mechanism underlying the growth reduction was thought to involve a connection between morphological plasticity and an anti-predator behavior that restricts feeding. However, more empirical work on this and other systems is called for to gain a complete picture of the mechanisms that produce costs and limits of plasticity.

Future directions

Models have already demonstrated several ecological and evolutionary consequences of various constraints on plasticity. Modeling these constraints may continue to be useful, but without good empirical data it is difficult to know which constraints are most likely or what magnitude each assumes. For example, as costs are documented empirically, we will have data to use in existing models, so that predictions about particular empirical systems can be made. The first researchers to document costs are likely to be met with both enthusiasm and intense scrutiny. Thus, it is important to be clear about semantics and discuss rigorous methodology at this early stage of inquiry.

Perhaps the best way to begin is to re-examine existing studies for evidence that plastic organisms pay costs or face limits from which fixed organisms are free. As detailed above, such tests will ideally focus on adaptive plasticity of well-understood traits. When plasticity has progressed to fixation within populations, studying multiple populations may provide the necessary variance to test for constraints on plasticity, with the caveat that populations vary in many ways, and the differences being studied do not always reflect the causes believed to be under investigation[18]. The methods we reviewed to test for constraints on plasticity will be useful for past and future data. The key in such analyses is that they control for simple production costs of inducible characters and costs inherent for particular environments.

Overall, much empirical work remains to be done before a full picture will emerge on the importance and frequency of various costs and limits of plasticity.

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