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SYMPOSIUM

Introduction to the Symposium—Uniting Evolutionary and Physiological Approaches to Understanding Phenotypic Plasticity

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From the symposium "Adaptation or Developmental Constraint? Uniting Evolutionary Theory and Empirical Studies of Phenotypic Plasticity" presented at the annual meeting of the Society for Integrative and Comparative Biology, January 3–7, 2014 at Austin, Texas.

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Synopsis Diverse subfields of biology have addressed phenotypic plasticity, but have emphasized different aspects of the definition, thereby shaping the questions that are asked and the methodological approaches that are employed. A key difference between studies of plasticity in the fields of evolutionary biology and physiology is the degree of focus upon the contribution of genetic variance to plastic traits. Although evolutionary biology is generally focused on the heritability and adaptive value of plastic traits and therefore the potential for plasticity to impact changes in traits across generations, physiological studies have historically focused on the timing and reversibility of plastic change across seasons or ages and the mechanisms underlying traits' plasticity. In this review and the symposium from which it emerged, we aimed to highlight ways that integrative biologists can better communicate about their research and design better studies to address phenotypic plasticity. Evolutionary theory clarifies the need to assess fitness using reliable measures, such as survival and reproductive success, and to consider the heritability and genetic variance underlying plasticity. Reciprocally, physiological research demonstrates that understanding the mechanisms that permit, or limit, plasticity, whether through pleiotropic effects, developmental, or functional linkages between traits, or epigenetic modifications, will shed light on limitations to phenotypic plasticity. Uniting the fields of evolution and physiology to address all aspects of phenotypic plasticity will be increasingly important as the rate of anthropogenic environmental change increases and biologists must predict the responses of wild populations to novel environments, as well as determine the most effective conservation interventions.

Introduction

Diverse subfields of biology have addressed phenotypic plasticity, yet emphasized different questions and employed different methodological approaches. Integrative biologists are uniquely positioned to develop a more holistic approach to studying phenotypic plasticity by building upon the approaches used across subfields, but this first requires clarifying definitions and terminology. A widely accepted definition of phenotypic plasticity is "the capacity of a single genotype to exhibit a range of phenotypes in response to variation in the environment" (Fordyce 2006). This definition, often used by evolutionary biologists, clearly includes evaluation of genetic contributions to plastic responses. Another definition of phenotypic plasticity is that it encompasses any phenotypic sensitivity to environmental factors (Whitman and Agrawal 2009). This second definition often is used in physiological studies and does not explicitly include genetic factors underlying plasticity. Thus, a key difference between studies of plasticity in the fields of evolutionary biology and physiology is the explicit inclusion or exclusion of genotypes. Otherwise, these definitions are intentionally broad and encompass a wide range of plastic traits that are continuous or discontinuous, adaptive or non-adaptive, compulsory or facultative, and reversible or irreversible (Morange 2009). Further, plasticity may rely upon specific or general environmental cues and phenotypic change may be anticipatory or responsive (Whitman and Agrawal 2009). For instance, a population of grasshoppers, *Schistocerca*

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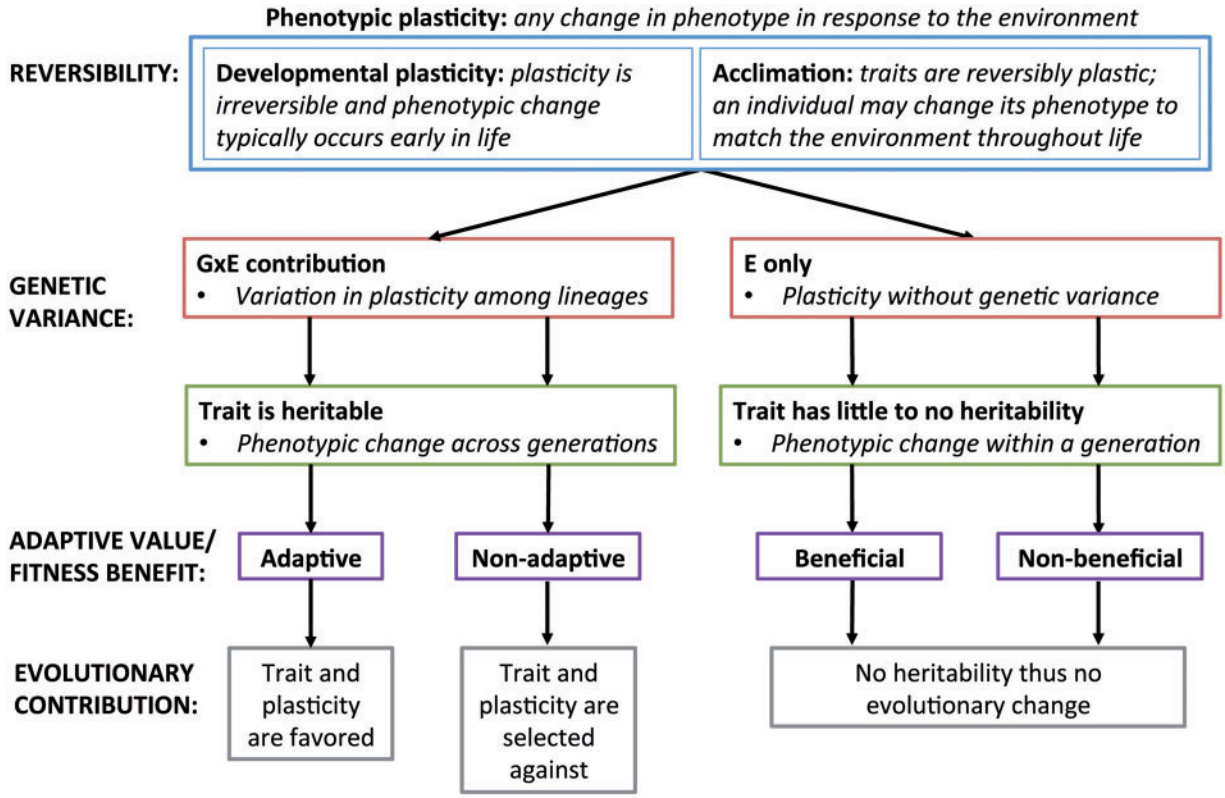


Fig. 1 Sub-categories of phenotypic plasticity and the roles genetic variance and fitness benefits play in microevolution of a trait.

emarginata, which is unpalatable to predators due to consumption of a toxic plant, displays cryptic green coloration when raised at low density but conspicuous yellow and black coloration when raised at high density (Sword 1999). The effect of rearing environment on coloration is reduced in a palatable population of the species, reflecting selection on the magnitude of phenotypic plasticity (Sword 2002). This classic example illustrates a characteristic of plasticity that is a focus of evolutionary biology: The role of plasticity in the evolution of a phenotype (e.g., warning coloration). In this review, we identify several sub-categories of plasticity that the fields of evolutionary biology and physiology independently focus upon and suggest how we can apply important contributions from both fields in an integrative approach to understanding phenotypic plasticity.

Sub-categories of plasticity

Physiological studies are typically interested in the timing and reversibility of plasticity and thus the effectiveness with which phenotypic outcomes match environmental conditions, as well as the proximate mechanisms that mediate plastic response (Nijhout 2003; Frankino and Raff 2004). Reciprocally, evolutionary studies often address the adaptive value of plasticity to understand its role in trait change within populations and are concerned with whether the plastic response is under current selection or whether it has emerged through past selective pressure. To make the features of plasticity addressed by each field of study especially clear, we suggest several definitions for sub-categories of plasticity (Fig. 1). First, we define plasticity as any change in phenotype as a result of the environment (Schlichting and Smith 2002), even if the genotype is unknown (both V_E and V_{GxE}, Fig. 2). Thus, plastic traits could include behavior, as well as changes in hormone levels, in addition to traits known to be heritable, irreversible, and adaptive, such as the timing of metamorphosis in amphibians (Gotthard and Nylin 1995). When traits are reversibly plastic such that an individual may change its phenotype to match the environment throughout its lifetime, we use the term acclimation, also sometimes termed phenotypic flexibility or, in the wild, acclimatization (Piersma and Lindström 1997; Piersma and Drent 2003; Garland and Kelly 2006). For example, the expansion and shrinkage of intestines in pythons to match metabolic demand are a form of acclimation (Starck and Beese 2001). Reciprocally, when plasticity

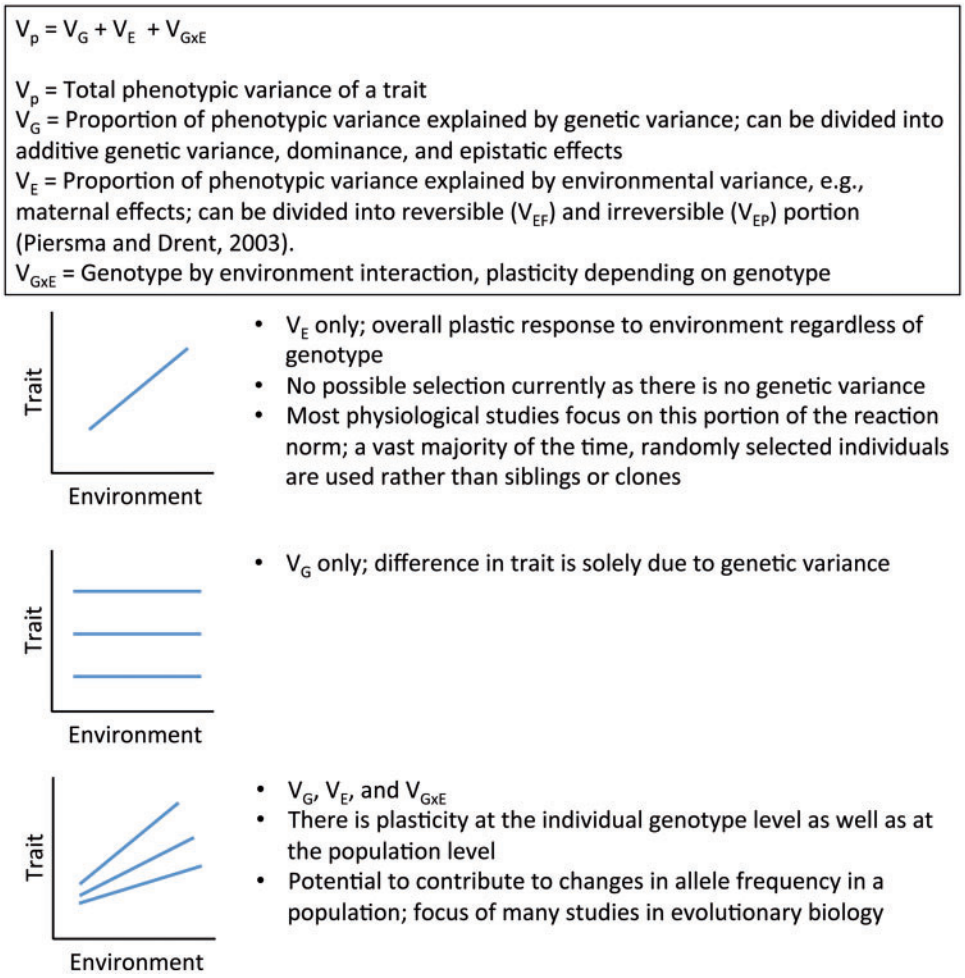


Fig. 2 Genetic and environmental portions of phenotypic plasticity, represented as an equation and reaction-norm plots.

is irreversible and occurs early in life, as is the case in many classical studies of phenotypic plasticity, we use the term developmental plasticity (Piersma and Drent 2003). Acclimation and developmental plasticity are subsets of plasticity by our categorization, and may or may not be demonstrated to be heritable or to be adaptive. Finally, when different genotypes vary in plasticity, regardless of whether it is known to be adaptive or reversible, we will refer to there being a G × E interaction. This is in opposition to having evaluated only an environmental contribution to plasticity—E only. Finally, as we discuss below, adaptive phenotypic plasticity might be considered as a subset of cases of phenotypic plasticity that are known to confer a fitness benefit (Pigliucci 2005) and, in some instances, to also be demonstrated to have arisen from past selection. Specifying the type of plasticity in which we are interested by using these categorizations will improve communication among researchers and help identify discrepancies in our thinking about phenotypic plasticity.

The reaction-norm approach

One instructive way to consider the differences in approach of physiology and evolutionary biology to the study of phenotypic plasticity is to consider the components of a reaction-norm plot upon which each subfield typically focuses. A reaction-norm plot graphically represents plastic and non-plastic portions of phenotypic variance. Phenotypic plasticity generally results both from genetic variation among individuals and from response to environmental change ($V_{G\times E}$, Fig. 2). However, variation in the expressed trait can also result solely from the plastic response of an individual to environmental change (V_E, no genetic variation; Fig. 2) such that phenotype will differ across environments but all individuals will respond in a similar way. Alternatively, variation in traits can be due to genetic difference alone (V_G, no phenotypic plasticity; Fig. 2) such that individuals differ in phenotype but no variation is attributed to the contributions of the environment. Studies of phenotypic plasticity in evolutionary

biology often center around $V_{G\times E}$ because the evolvability of a trait is in question and genetic variation is necessary for the trait to contribute to evolutionary processes. In contrast, studies of phenotypic plasticity in physiology generally focus on V_E as many studies quantify and compare the mean values of the traits of groups of individuals in two or more environments to understand the mechanisms underlying plasticity (Williams 2008).

Applying the reaction-norm approach

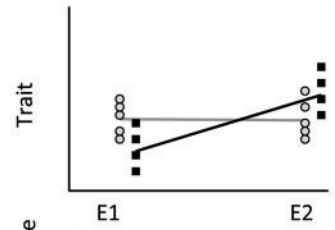
Although integrative biologists are generally well-aware of the reaction-norm approach, applying it to animal systems, particularly in the field, can be extremely challenging. We suggest several strategies for moving toward a reaction-norm approach in systems that are not especially amendable to this experimental design. First, exposing siblings or other related individuals to two or more environments, either as two treatments or in a repeated-measures design is an experimental improvement to traditional physiological approaches because it will distinguish $V_{G\times E}$ from V_E (Figs. 2 and 3). That is, this experimental approach will determine whether or not the plastic response to an environment depends on heritable, genetic differences among individuals. For example, testing the stress response of siblings in two different environments, rather than averaging the response of many unrelated individuals, would determine if there are genetic limitations to plasticity in this trait. A first step toward this approach is to simply include family lineage as a factor in statistical models. Thus, even if there is insufficient sample size to complete a balanced experimental design and explicitly test the genetic variance for a plastic trait, we can account for genetic variance as a random factor and the environmental contribution of plasticity can be described without subsuming all genetic contributions. As the need to address questions about how animals respond to anthropogenic environmental change and climatic change increases, incorporation of genetic variance for plasticity allows us to predict how populations will cope with a changing environment across generations. At the same time, additional experimental approaches are necessary to address questions about the physiological limitation, timing, or fitness consequences of plasticity (Nijhout 2003; Frankino and Raff 2004), which are also essential for predicting responses to a changing environment. In the following sections, we describe the focal questions and some experimental approaches used in evolutionary and physiological research on phenotypic plasticity and address how integrative biologists may build upon the foundation laid by both subfields.

Additional approaches to studying phenotypic plasticity

Measures of fitness

Physiological studies often address the mechanisms that underpin phenotypic plasticity and do not focus on fitness benefits. However, understanding the benefits of plasticity is essential to determining whether traits are stable and will spread within a population, or whether there will be selection against them. Determining the fitness benefit of a plastic trait is best achieved by quantifying direct measures such as reproductive success and survival.

From an evolutionary perspective, phenotypic plasticity is often considered as an evolved trait (DeWitt and Scheiner 2004); there can be genetic variation for plasticity (Newman 1994; Stinchcombe et al. 2004; Pigliucci 2005), plasticity as a trait can be heritable (Scheiner 1993, 2002; Nussey et al. 2005), and the ability of an individual to adjust its phenotype to match the environment often may be under selection (de Jong 2005). Thus, plasticity that confers a fitness benefit within the scope of environmental variation a species is likely to encounter, could be considered adaptive, that is, a trait that contributes to the survival and reproduction of an individual (Reeve and Sherman 1993). In contrast, within the field of physiology, the consequences of a plastic trait in individuals are sometimes estimated based on a single metric of performance, such as body size, rather than a fitness outcome such as number of offspring. The relatively new suite of measures of cellular function, some of which are correlated with survival, such as resistance to oxidative stress and length of telomere, offer additional physiological measures that may be good estimates of fitness. Nonetheless, fitness benefits are best understood by making direct and comprehensive measures of fitness, such as number of offspring recruited to the population, or lifetime reproductive success, and studies of evolutionary biology underscore the importance of using these direct measures whenever possible. To distinguish traits that are suggested to be under selection from ones that are shown to be, we use the term beneficial to refer to traits or phenotypes that increase performance of an individual in one dimension (such as increasing body mass) while reserving ''fitness benefit'' and ''adaptive'' for traits shown to directly increase lifetime reproduction and/or survival.



TO DETERMINE THE ROLE OF A PLASTIC TRAIT IN EVOLUTION

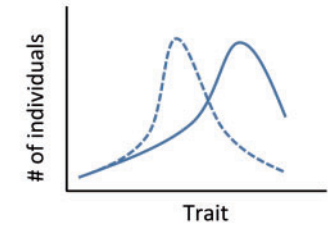
Reaction norm approach: phenotypic traits of individuals from different lineages are measured under different environmental conditions. Points reflect different individuals, symbols reflect different populations or families. V_{GxE}, V_E and V_G are captured.

- *Best approach for distinguishing GxE interaction from E or G only, determining genetic limitations on phenotypic response to environment and genetic variation in plasticity.*
- *To determine whether the trait is favored or selected against, a study assessing fitness benefits in a trait with GxE interaction should follow.*

TO DETERMINE THE FITNESS BENEFIT OF PLASTIC TRAITS

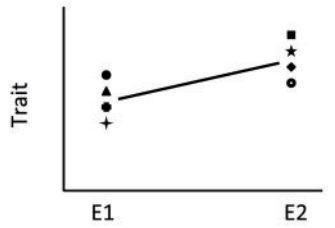
1. Measure fitness proxies in environments within the natural scope for the species

2. Use measures that directly affect fitness whenever possible; lifetime reproductive success and number of offspring recruited into the population are more informative than reproductive success within a season or performance measures such as body condition



Fitness benefit: difference in reproductive success and survival across a range of trait values (first graph). For traits that are heritable and have a positive linear regression with reproductive success and/or survival, the mean trait value is expected to increase after selection (second graph). Dotted and solid lines in the second graph represent a frequency of individuals before and after selection, respectively.

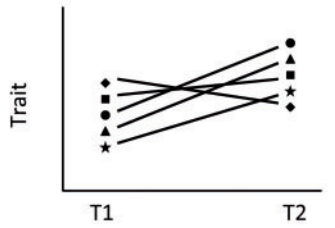
- *Determines the fitness benefit of a particular trait and whether the trait is favored or selected against. When measured in traits that are shown to be heritable, changes in population mean trait value and frequency overtime can be estimated.*



TO DETERMINE THE PROXIMATE MECHANISMS UNDERLYING PLASTIC TRAITS

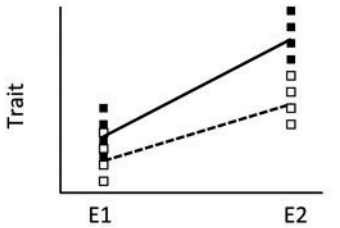
1. Treatment group approach: groups of individuals are exposed to two or more environments. Typically, genotype is unknown so phenotypic traits of different unrelated individuals are averaged under each environmental condition. Each point and symbol reflects a different individual. V_E is captured but V_G is subsumed in this estimate.

- *Determines average plastic response to the environment but cannot address genetic contributions to phenotypic plasticity. Useful for determining physiological limitations on phenotypic response to environment and physiological mechanisms underlying plastic traits. Extreme or novel environments are sometimes used.*
- *If clones or siblings are equally distributed among the treatment groups, this approach can also inform us about GxE interaction and genetic limitation on phenotypic plasticity.*



2. Repeated measures approach: phenotypic traits of the same individuals are measured under different environmental conditions, at different times (time (T) 1 and 2). Only relevant to traits with life-long plasticity (not developmental plasticity). Each symbol reflects a different individual at two time points. V_E is captured but V_G is subsumed in this estimate.

- *Determines inter-individual differences in plasticity and captures both genetic and non-genetic differences among individuals (e.g., inter-individual differences in developmental conditions and epigenetic alteration)*



TO DETERMINE THE REVERSIBILITY OF PLASTIC TRAITS

Age specific repeated measures: phenotypic traits of individuals from different age groups are measured under different environmental conditions. Points reflect different individuals, color differences reflect juveniles and adults.

- *Approach for determining life history stage limitations on phenotypic response to environment and reversibility of plasticity.*

Fig. 3 Summary of experimental approaches and recommendations for developing integrative studies of phenotypic plasticity.

Environmental context

Related to estimating the fitness benefits of plastic traits, the environmental conditions under which fitness proxies are measured can influence inferences about the benefit of plastic traits. When asking questions about plasticity, integrative biologists should use environmental manipulations within the scope of those that a species normally experiences and employ extreme manipulations of environment only when mechanisms underlying plasticity or

predictions about response to novel environments are the focus of research. The need to consider the environment in which fitness is estimated is illustrated by the water flea, *Daphnia*, which develops a protective helmet, or spines on the neck and tail, when raised with chemical cues emanating from predators (Tollrian 1995; Agrawal et al. 1999; Laforsch and Tollrian 2004). In the presence of predators, these defense mechanisms increase survival (Laforsch and Tollrian 2004); however, in the absence of predators, defensive morphology can lower competitive ability of the individual (Tollrian and Dobson 1999). Thus, the benefits of phenotypic modification change with the environment because the balance between benefits and costs of plastic morphology depends on the match between the phenotype and the environment.

As evolutionary studies focus on the adaptive value and evolvability of phenotypic plasticity, they generally expose individuals of known genotype to environments within the scope of those that naturally occur for the species. In contrast, physiological studies focused on the mechanisms underlying plasticity sometimes replicate extremes of ecological conditions or introduce novel conditions, and may measure fitness or physiological performance under common garden conditions to permit control over specific environmental factors. Exposing organisms to extreme conditions has been important for identifying the physiological basis of phenotypic plasticity, inter-individual variation in plastic response, and the magnitude of response to environmental change. However, the results of such studies should be interpreted with caution in regards to understanding the fitness benefits of phenotypic plasticity. First, fitness within the context of wild populations is a relative measure and thus should be reported as the survival or reproductive success of an individual or genotype relative to the rest of the population. Therefore, finding that a physiological impairment is associated with a particular environment does not necessarily mean that an individual or phenotype also has lower fitness, if that genotype still outcompetes other lineages. Second, experimental conditions that are novel, or overly harsh, may make it impossible for phenotypic plasticity to mitigate compromises in fitness, resulting in deficits and detrimental phenotypes (Garland and Kelly 2006; Monaghan 2008). That is, when extreme environmental manipulation is employed we may effectively be studying physiological limitations on a phenotype that may not vary among individuals, as plasticity can no longer mitigate environmental change. Such experimental designs may be relevant to understanding invasion biology and rapid ecological change, but we need to be aware that exposure to novel or extreme conditions is more relevant to examination of the mechanisms of plasticity than to its adaptive value.

Heritability

In cases when plasticity is demonstrated to be beneficial or adaptive, it is tempting to infer that natural selection will lead to change in the frequency of the trait's expression within the population over time. However, response to selection (R) depends both on heritability (h^2) and on the strength of selection (S; $R = h^2S$). Thus, even though a certain trait bestows greater reproductive success or survival compared with others, if heritability is low or none, then the frequency with which that trait is expressed within the population is not likely to change across generations (Fig. 1). It is particularly important to consider the heritability of a plastic trait when predicting the health and survival of a population facing ecological change. If plasticity is based entirely on V_E and there is no genetic variance for the trait, the trait will not evolve or even necessarily be maintained within a population facing environmental variability (Conner and Hartl 2004). An exception to this is when environmental conditions cause epigenetic reprogramming in the germline that can be passed vertically across generations (Chong and Whitelaw 2004; Allegrucci et al. 2005; Hajkova 2011). In other words, if environmental change causes changes in gene expression that are imprinted in the germlines, the epigenetic modification can be heritable and thus subject to natural selection and adaptive evolution. Since epigenetic modifications can occur faster, but are less stable, than changes in DNA sequence, induced epigenetic variation may accelerate the spread of a novel phenotype across a population (Richards et al. 2010). Determining the heritability of a plastic trait is essential for understanding whether plasticity will buffer populations from ecological change. Thus, integrative biologists addressing questions of how organisms cope with a changing environment need to consider the heritability of the mechanisms that permit plasticity, as well as the magnitude and physiological underpinnings of that plasticity.

Demonstrating selective pressure

In addition to quantifying the fitness benefits of plasticity, evolutionary biology sometimes also aims to demonstrate that a plastic trait was under historical selection for its current function, which is best

achieved through comparative studies. Using the comparative approach to determine the adaptive value of plastic traits could inform predictions about how selection will act upon the mechanisms underlying plasticity and whether plasticity in a trait is likely to persist within a species or population. To illustrate how assessing the adaptive value of a plastic trait could inform our understanding of physiological mechanisms consider the glucocorticoid stress–response. Species or populations that have a dampened stress–response (lower glucocorticoid levels in response to a standardized stressor) are hypothesized to tolerate a wider range of habitat types and to be superior colonizers. The comparative approach can be employed to determine whether a dampened stress–response is common to lineages that are colonizers, consistent with reduced plastic response being an adaptation; or whether particular clades and the ancestral outgroup show a dampened stress–response independent of colonization history, consistent with plastic response being ancestral but not presently adaptive (e.g., Bonier et al. 2007). That is, the comparative approach can be applied as another means of determining whether a plastic trait is likely to be under selection and to persist in particular populations.

Reversibility of plasticity

One of the greatest differences between approaches used in evolutionary and physiological research on phenotypic plasticity is the time-frame of study. As alluded to in the classic example of *Daphnia* above, when the environment changes, the match between an organism's phenotype and surrounding environment is disrupted (Whitman and Agrawal 2009). In response to environmental change, organisms alter their phenotype within, and across, generations (Meyers and Bull 2002; DeWitt and Langerhans 2004). The benefit of plasticity in traits depends upon the relative rate of change between phenotype and the environment. Evolutionary biology primarily focuses on across-generational shifts in phenotype, which are mediated by heritable genetic changes in response to natural selection (Whitman and Agrawal 2009). This type of phenotypic change is considered adaptive at the population level and does not necessarily consider the benefit of plasticity to an individual. The field of physiology, on the other hand, historically focused on within-generational change in phenotype at the individual level, which is mostly non-genetic (Whitman and Agrawal 2009) but presumably beneficial to the individual. This phenotypic change may be reversible acclimation or irreversible developmental plasticity, depending on the timing of the environmental change and the nature of the phenotypic change. It is important to address the reversibility of plasticity because plasticity is only beneficial for the individual if the rate of phenotypic change can keep pace with the environmental change on temporal and spatial scales (Whitman and Agrawal 2009; Fusco and Minelli 2010). An important contribution that the field of physiology has made to the study of phenotypic plasticity is the careful description of variation in the timing of phenotypic plasticity during an individual's lifetime and an understanding of how the timing of phenotypic change is beneficial, or detrimental, to individuals. Specifically, plasticity can have negative consequences for the individual if developmental mechanisms make animals vulnerable to environmental perturbation (Fusco and Minelli 2010); empirical studies demonstrate that poor environmental conditions during the period of phenotypic organization and developmental plasticity can generate detrimental phenotypes in adulthood (Monaghan 2008). For instance, deprivation of food, administration of glucocorticoid, and malarial infection during development in male songbirds resulted in simpler, shorter duration song in adulthood, which is associated with low reproductive success (Spencer et al. 2003, 2004, 2005; Schmidt et al. 2013). Additionally, there is evidence that developmental plasticity or limited acclimation later in life can generate phenotypes mismatched to the environment when ecological conditions are unpredictable (DeWitt et al. 1998). Thus, phenotypic plasticity may be beneficial or detrimental to individuals, depending on the degree of spatial heterogeneity in the environment, the speed of the environmental change relative to the organism's mobility and lifespan, the predictability of the change, costs of plasticity, developmental limitation on plasticity, and pleiotropic effects among traits (DeWitt et al. 1998; Schlichting and Smith 2002; Garland and Kelly 2006). Evolutionary biology historically focused on irreversible developmental plasticity (e.g., Pigliucci 2005). Given the importance of reversible plasticity to species' survival and fitness in the face of rapidly changing environmental conditions, a step forward would be to consider more complex cases of reversible plasticity. One experimental approach to assessing the reversibility of plasticity is to expose the same individuals to multiple environments, including across various life-history stages (such as both in juveniles and adults), that is, the repeated-measures design (Fig. 3).

Uniting concepts and future directions

The review of the different research approaches traditionally employed by the fields of evolution and physiology highlights the benefits of promoting interaction between the two historically separated fields. Key recommendations are summarized in Fig. 3. Evolutionary theory clarifies the need for proximate studies to assess fitness using reliable measures, such as survival, reproductive success, and competitive ability (Wilson and Franklin 2002), and to consider the heritability and genetic variance underlying plasticity. Using clones or siblings in environmental manipulations to tease apart phenotypic change in relation to the environment (E only) from gene by environment interactions (G × E) will improve physiological studies by distinguishing plasticity with the potential to contribute to allelic change within a population overtime, from plasticity that will only affect the individual (Conner and Hartl 2004). This distinction is especially important to this field when the studies aim to predict persistent and across-generational phenotypic response to environmental change in a population. Additionally, the traditional evolutionary approach encourages consideration of the types of environmental manipulations imposed, as a means of clarifying whether studies address plasticity or the limits of physiological capacity. Importantly, proximate studies of phenotype must consider that altered physiology does not always reflect compromised fitness; fitness benefits likely depend on the matching of phenotype with environment. Reciprocally, evolutionary theory does not always consider the difference between irreversible plasticity and life-long phenotypic acclimation, which is important for considering the consequences of plasticity to individuals and populations experiencing rapid environmental change (Whitman and Agrawal 2009); when the timing of phenotypic plasticity is mismatched with the rate of environmental change, plasticity may be disadvantageous. Physiological studies emphasize the importance of inter-individual variation in plastic traits, and evolutionary studies will benefit from considering both genetic and non-genetic individual differences in plasticity to estimate the likelihood of plasticity driving microevolution (Nussey et al. 2007). Most importantly, uncovering the physiological mechanisms that permit, or limit, plasticity, whether through pleiotropic effects, developmental, or functional linkages between traits, or epigenetic modifications, will shed light on the limitations of phenotypic plasticity and its capacity to mediate adaptive responses to environmental variation and change (Whitman and Agrawal 2009).

Phenotypic plasticity plays a vital role in organismal diversity, as well as in the survival of individuals and the health of populations, making work on this topic relevant to ecology and conservation. Carefully assessing the benefits, costs, and consequences of plasticity (DeWitt et al. 1998), and understanding the physiological mechanisms underlying plasticity, including genetic processes, will advance this area of integrative research (Whitman and Agrawal 2009). One of the greatest potential contributions of this work is likely to be in the field of anthropogenic environmental change: Predicting the response of wild populations to changing environments requires understanding both the proximate mechanisms underlying specific plastic traits and the heritability of those traits.

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