DOI: 10.1002/ajb2.16068

COMMENTARY

Botany

# Phenotypic plasticity made simple, but not too simple

**Richard Gomulkiewicz**[1] | **John R. Stinchcombe**[2]

[1]School of Biological Sciences, Washington State University, Pullman, Washington 99164, USA

[2]Department of Ecology and Evolutionary Biology, University of Toronto, Toronto, Ontario M5S3B2, Canada

**Correspondence**
Richard Gomulkiewicz, School of Biological Sciences, PO Box 644236, Washington State University, Pullman, WA 99164, USA.
Email: gomulki@wsu.edu

Phenotypic plasticity refers to environment-dependent trait expression (Dewitt and Scheiner, 2004).[1] Knowledge of phenotypic plasticity is important in virtually all areas of basic and applied biology. Researchers in applied fields (such as agriculture, medicine, public health, wildlife management, and conservation biology) have a vested interest in knowing how traits are or will be expressed under specific conditions. Ecologists are interested in how the expression of traits in different environmental conditions and habitats might affect population and community dynamics. And evolutionary biologists are interested in how traits with environmentally-conditional expression have and will evolve. The widespread interest in phenotypic plasticity has made it a prominent focus of biological research.

Phenotypic plasticity is an especially active research area in ecology and evolution with a brimming literature that has advanced the understanding of organismal variation, adaptation, and speciation (Sarkar, 2004; Pfennig, 2021). Most advances, especially recently, are based on highly simplified biological scenarios such as dichotomous environments or linear environmental gradients. Here we advocate a path for taking modern plasticity research in a far more biologically relevant direction.

Phenotypic plasticity, like any trait, can be heritable and respond to any evolutionary force. What makes plasticity unique is that it manifests *only* in a variable environment and is thus automatically complex. The key to addressing plasticity's ineluctable complexity, we contend, is a simple but *comprehensive* conceptual framework that can be used to address questions about phenotypic plasticity (including connections among areas of development, behavior, genetics, ecology, and evolution) with far more depth and realism than current literature.

The framework (Figure 1) involves four independent components: (1) patterns of plasticity; (2) environment encounters; (3) fitness consequences; and (4) inheritance. The first two components are needed to predict realized patterns of expression, the first three determine population dynamics, and all four contribute to evolution. Below, we describe each component in turn, highlighting key concepts and practices that enable researchers to enrich the understanding of phenotypic plasticity and its evolution in nature. While none of these four components is new, we have not seen them presented together in a systematic way, as here. We contend that widespread use of this structured quartet of concepts would drive modern studies of phenotypic plasticity in a much more productive, profound, connected, and comprehensible direction.

## PATTERNS OF PLASTICITY

The most complete and universal description of environment-dependent phenotypic expression, i.e., phenotypic plasticity, is the *reaction norm* (Woltereck, 1909; Johannsen, 1911; Schmalhausen, 1949), which refers to the set of phenotypes a genotype expresses in different environments. "Environments" can be quantitative or qualitative, simple or multicomponent, discrete or continuous, physical or biotic (including social), external or internal to an organism. They can encompass ancestral environments if phenotypic expression is impacted by

[1]Plasticity typically refers to the *consistent* expression of phenotypes in different environments. Traits that change unpredictably in different environments are usually said to be 'noisy' rather than plastic. Various terms are used to describe traits with the same phenotype in all environments, including 'aplastic', 'non-plastic', 'fixed', 'constant', 'canalized', and 'environmentally insensitive'.

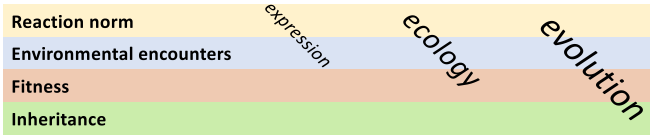


**FIGURE 1** The four fundamental elements of phenotypic plasticity and their roles in determining patterns of phenotypic expression realized in nature, ecology (population or community dynamics), and evolution.

trans-generational (epigenetic) effects (Bonduriansky, 2021) or internal environments (e.g., age, metabolic rate, body condition).

All reaction norms can be described as either a multivariate trait—an ordered list or vector—over discrete environments (Via and Lande, 1985) or as a function-valued trait—a curve or surface—over continuous environments (Stinchcombe et al., 2012; Kingsolver et al., 2015). Standard multivariate methods can be used for estimation, modeling, and inference; unobserved components of reaction norms can be imputed or interpolated; Gomulkiewicz et al. (2018) describes a number of function-valued methods, most of which require no information about genetics or relatedness.

Why do we encourage use of reaction norms to describe environment-dependent phenotypic expression over metrics expressly designed to quantify plasticity, especially given the simplicity and intuitive appeal of the latter? Though plasticity measures are easy to conjure, no single quantity pertains to all situations, particularly when there are more than two environments. Consequently, no scale exists to compare the plasticities of different genotypes, not even one that preserves rank orders. For example, Figure 2 depicts the reaction norms expressed by genotypes $G_1$ and $G_2$ over three environments ($E_1$, $E_2$, $E_3$). Were plasticity measured as phenotypic variance over environments, as is common, genotype $G_1$ would rank as more plastic than genotype $G_2$. However, were plasticity measured by the range of phenotypic responses—also commonplace—the ranking would be reversed. Finally, measuring plasticity as a mean difference between environments—also very common—requires specifying one environment as the reference point, and results in at least as many plasticities as there are pairs of environments (e.g., $E_1$-$E_2$, $E_1$-$E_3$, and $E_2$-$E_3$, and all the reverse orders). Absent an order-preserving scale, comparative statements like "this genotype is more plastic than that one" become effectively meaningless over realistically complex environments. Nonetheless, countless studies (uncritically) assume plasticity can be rank-ordered, likely because most consider just two environments or only linear reaction norms.

It can be highly tempting to fit reaction norms using linear functions (an approach that one of us has used himself), i.e., if there are two experimental environments, a plasticity metric such as a mean difference is mathematically equivalent to a slope, which seems like it would characterize a reaction norm. Likewise, if only linear functions are used, the slopes and intercepts appear to characterize the reaction

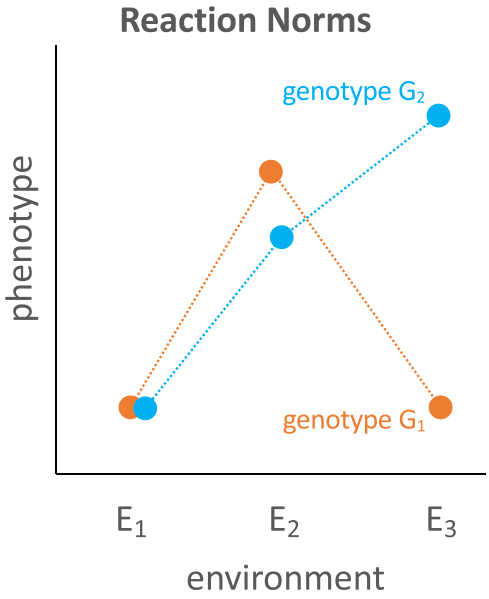


**FIGURE 2** Counterexample proving that there is no universal rank-preserving metric of phenotypic plasticity over more than two environments. Shown are hypothetical reaction norms for two genotypes ($G_1$, $G_2$) over three environments ($E_1$, $E_2$, $E_3$). If plasticity is measured by overall variation, genotype $G_1$ is more plastic than $G_2$. However, were plasticity measured by a genotype's maximal between-environment difference in expression, genotype $G_2$ ranks above $G_1$.

norm. While intuitively and analytically appealing, these scenarios (two environments, linear reaction norms) are in fact special situations in which plasticities can be ordered consistently (by, say, variance or slope) but not always (e.g., when using nonlinear transformations of pairs of phenotypic values; Wang et al., 2022). Thus, one should be skeptical that conclusions from studies confined to two environments or linear reaction norms extend to more realistic scenarios. Focus on these special cases perpetuates a situation in which a *general understanding* and synthesis remains beyond our grasp despite an accumulation of plasticity studies. If organisms typically experience more than two types of environments or if it is common for reaction norms to be non-linear, studies ignoring these realities are analogous to taking out-of-focus pictures with a camera, i.e., simply snapping more out-of-focus photos is not going to improve the quality of the image just as doing more oversimplified studies will not sharpen our picture of plasticity.

Reaction norms encompassing multiple environments and potentially non-linear changes in phenotypes have, unlike plasticity metrics, a standard representation depending on the environment of interest (see above). Reaction norms can also be used to calculate any plasticity measure, which makes them superior for studying any aspect of plasticity. The reverse is not true, i.e., a particular value of a plasticity metric such as a mean difference, range, or variance will almost always correspond to multiple reaction norms. In other words, the reaction norm, and not the (human-invented) metric, captures the biology.

Importantly, even in the event that reaction norms are linear, nothing is lost by adopting the reaction norm framework to study plasticity over either discrete or continuous environments. When population variation is described in terms of reaction norms, those that lack plasticity are not unique, but instead are merely part of a (multivariate) distribution. Indeed, the evolution and consequences of aplastic reaction norms involve the exact same mechanisms as plastic ones (Sultan, 2015).

Plasticity per se is too nonspecific of a concept and it lacks a universal measure to address anything but rudimentary questions about its evolution. In contrast, reaction norms have no such limitations. We thus recommend that plasticity be employed only as a category label and, in particular, it should not be quantified. Reaction norms are the proper quantitative platform to study environment-dependent phenotypic expression.

## ENVIRONMENTAL ENCOUNTERS

Plasticity itself can only be expressed if genotypes are exposed to more than one environment, and realized patterns of plasticity in any setting, natural or not, depends as much on the reaction norm as the frequencies of environmental exposures. Indeed, the distribution of environmental encounters is as crucial to the evolutionary and ecological consequences of plasticity as the reaction norm itself (Gomulkiewicz and Kirkpatrick, 1992). Yet studies rarely consider or attempt to measure environmental distributions that species encounter in nature (Arnold and Peterson, 2002).

There are innumerable ways populations experience environmental variability. "Fine" and "coarse" grained scales of environmental variation can be encountered through time or across space. Different distributions of exposure generally lead to different realized patterns of phenotypic expression and fitnesses (see section below), even for a genetically uniform population. To predict these realizations, one needs both a description/estimate of reaction norms found in a population and a description/estimate of the distribution of environments encountered (Figure 1).

Studies of phenotypic plasticity oftentimes assume—usually implicitly—that environments are encountered equally often. In an experimental context, the equal replication of different treatments differs—dramatically—from the natural distribution of these environments. If, say, an organism or genotype encounters an environment 50% of the time in an experiment (i.e., one with two treatments), but only 10% of the time in the wild, such a balanced design would disproportionately overweigh that component of the reaction norm and underweigh others compared to nature. Although using balanced experiments[2] or assuming a uniform distribution of environments in theoretical studies greatly simplifies comparisons of different patterns of plasticity, such comparisons will not represent nature if environments are encountered at all unevenly in the wild. Empirical estimates of environmental encounter frequencies are the ultimate means to test this speculation, which suggests a straightforward research agenda, i.e., measure the frequencies of environments an organism actually encounters. Fortunately, many environmental variables ($CO_2$, temperature, salinity, humidity, freezing days, precipitation, etc.) can be measured remotely with data loggers, ibuttons, and other instruments. Other, more biotic environments (e.g., competitor or mutualist densities) will require old-fashioned ecological field work. Moreover, documented patterns of environmental encounters will enable biologists to assess the proportionate importance of different environments for the evolutionary and ecological causes and consequences[3] of phenotypic plasticity (e.g., Kingsolver et al., 2001; Kingsolver and Buckley, 2017).

A number of theories that invoke plasticity, such as plasticity-led evolution, genetic assimilation, the Baldwin effect, and "buying time" for persistence (Crispo, 2007; Diamond and Martin, 2021) imagine a single, abrupt change from an ancestral environment to a novel one. If the novel environment is constant, as is usually implied, the only possible role for plasticity is phenotypic expression in the novel condition. This is the only moment one reaction norm could be favored *directly* over another. Post-shift, the novel environment becomes the "new normal." Consequently, any subsequent evolution of plasticity must be nonadaptive (see section below). Were the novel environment truly unprecedented in the history of the species then, akin to a new mutation, the phenotype expressed could be adaptive or nonadaptive in the new setting (Ghalambor et al., 2007).

Early models of phenotypic plasticity assumed passive environmental encounters (Via and Lande, 1985; Gomulkiewicz and Kirkpatrick, 1992; Gavrilets and Scheiner, 1993), but recent ones consider organisms that actively determine encounters either through habitat choice/preferences or by changing their local environment directly (niche construction; e.g., Sultan, 2015; Scheiner et al., 2021). Yet other models consider "internal" environments like age or individual condition itself (e.g., Matthey-Doret et al., 2020). With habitat-dependent dispersal (Edelaar and Bolnick, 2012), migration itself is a plastic trait that determines environmental encounters, potentially resulting in different exposures for different genotypes. Clearly more work is needed to understand how dynamic distributions of encounters might influence the expression and evolution of reaction norms… and vice versa.

[2]The issue of balance is additional to the artificiality of the experimentally-controlled environmental conditions themselves.

[3]It is crucial to distinguish ecological from evolutionary effects since, for example, an extreme environment could easily cause all genotypes to have the same, albeit low absolute fitness. This would completely preclude natural selection (because of the lack of variation in relative fitness) but the prospect of extinction—an ecological outcome—could be catastrophically permanent even were the extreme condition rare.

## FITNESS CONSEQUENCES

Trait expression can affect an organism's fitness in environments it encounters; individual fitnesses collectively determine population dynamics; and if the trait's expression is heritable, evolution. These truisms apply to plastic and non-plastic traits alike. Since plasticity manifests only in a variable environment, this too is required for plasticity itself to evolve *adaptively*. While this is a seemingly obvious point, many studies that consider adaptive phenotypic plasticity refer only to its evolution in a single environment, such as a novel one (see section above). Plasticity *can* evolve in a single environment but only non-adaptively via indirect selection due to associated "plasticity costs", as a correlated response, or by random genetic drift.

Not only can expression of a phenotype change in response to a change in environment but the fitness consequences of a particular expressed phenotype may also vary from one environment to the next. The realized fitness of an individual in a given environment or set of environments must reflect both considerations (e.g., Chevin et al., 2010) as well as any constitutive or environment-specific costs paid to enable plastic expression. In addition, the environment that determines trait expression during a "sensitive period" can, because of developmental or other delays, differ from the environment that determines fitness.

The relevant measure of fitness will depend on an organism's life history and how that relates to environmental variability. For example, an individual could experience multiple environments within its lifetime (e.g., daily thermal variation). Individual fitness would integrate over these fine-grained distributions (e.g., Kingsolver et al., 2007). At the other, coarse-grained extreme, an individual experiences a single environment in its lifetime but its descendants could develop in different environments because of *in situ* temporal change or dispersal. The fitness consequences of plasticity for both demography and adaptive evolution must then reflect these among-generational changes.

Many studies over continuous environments assume optimizing selection such that the optimum phenotype changes linearly (e.g., Chevin et al., 2010). This assumption is mathematically convenient with a bonus feature, viz., the optimal reaction norm is necessarily linear. Consequently, studies often consider *only* linear reaction norms, which lends itself to the further, conceptual perk that slope directly reflects plasticity. In reality, neither linearity assumption is empirically justified. Future studies should consider non-linear versions of optimizing selection and distributions that include nonlinear reaction norms.

Finally, plastic phenotypes may in fact have no differential effect on fitness, that is, different reaction norms may have equivalent consequences for total fitness. In these cases, phenotypic plasticity is a neutral trait and its evolution is best understood in terms of non-adaptive evolutionary processes including random genetic drift (Lande, 1976; Kimura, 1983).

## INHERITANCE

Like any trait, the heritable basis of a reaction norm could range from a major gene to many loci of individually small effect; it can be inherited in organisms that are asexual, sexual, self-fertile, self-incompatible, diploid, polyploid, or even via non-Mendelian mechanisms (extra-nuclear or

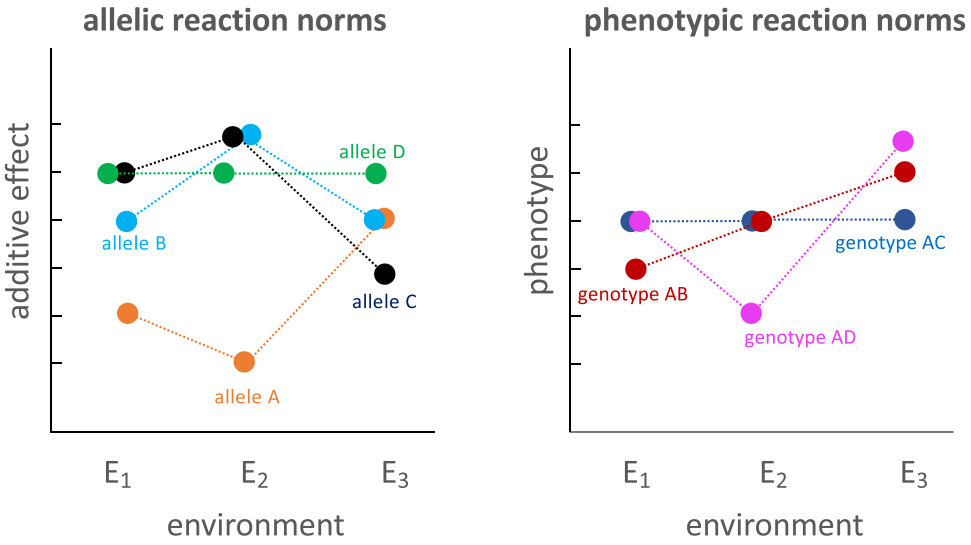


**FIGURE 3** Gene expression profiles (allelic reaction norms) and resulting phenotypic reaction norms. Left panel: Additive effects of four alleles (A, B, C, D) in each of three environments ($E_1$, $E_2$, $E_3$). Note that allele D has the same effect in all environments, i.e., D is not plastic. Right panel: Phenotypic reaction norms of three diploid genotypes with different combinations of alleles shown in the left panel. The phenotype expressed in each environment is determined by adding the allelic effects. Note that diploid genotype AC is not plastic even though both alleles are individually plastic whereas genotype AD is plastic despite allele D being aplastic.

transgenerational epigenetic; Auge et al., 2017). Any responses to selection (i.e., adaptation) can be described using standard population and quantitative genetics, as can other evolutionary processes that might affect their evolution such as mutation, recombination, and random genetic drift (e.g., Charlesworth and Charlesworth, 2010). Describing the spatial structure of genetic variation is of particular importance for species whose local populations encounter coarse-grained environmental variation via migration.

Many explicit multi-locus models of phenotypic plasticity posit the existence of "plastic" and "non-plastic" gene expression profiles across environments. While convenient, these gene classes are neither biologically necessary nor justified. Indeed, two genes with opposite reaction norms would additively produce an aplastic phenotype (Figure 3). A better approach for future studies is to consider gene-level reaction norms—a generalization of "mutation reaction norm" (Ogbunugafor, 2022)—that, when combined, produces overall reaction norms, whether plastic or not (Figure 3). Conceivably, gene-level reaction norms could prove valuable for detailed prediction of evolutionary responses to selection (see earlier section on Fitness consequences) or for describing the expected course of random genetic drift in study systems where reaction norm variation depends on just a few segregating genes or genotypes.

Studies often emphasize genotype-by-environment interaction ("G × E"), as it is necessary for plasticity to evolve (Saltz et al., 2018). The absence of G × E (parallel reaction norms) implies absence of genetic variation in plasticity. However, the absence of G × E does not imply the absence of plasticity per se, nor does the presence of G × E ensure the evolution of plastic genotypes. Consequently, G × E is necessary but not sufficient for plasticity to evolve. Although estimates of G × E variances can sometimes reveal how much fitness variation across environments is maintained by rank changes versus changes in variance (Vaidya and Stinchcombe, 2020) it is unknown if those inferences apply to other phenotypic measures of G × E variation. Regardless, one can always use a reaction norm approach to dissect root causes of G × E variation if not necessarily the reverse (Saltz et al., 2018).

## CONCLUSIONS

We urge that future studies of phenotypic plasticity organize around our four-component framework of reaction norms, environmental encounters, fitness consequences, and inheritance (Figure 1). Box 1 lists some best practices and compelling future research directions suggested by our framework. A complete understanding of phenotypic plasticity requires all four components (Via et al., 1995; Sultan, 2021) and, while we might pine for studies that consider the full foursome, this emphatically does not imply that studies must include all of the components to make valuable contributions. Rather, a key advantage of our framework is to provide a simple, but not too simple conceptual "wrapper" for investigations that address one or more of the components we describe, collectively providing a clear and consistent context for how each study contributes to our holistic understanding of phenotypic plasticity and its evolution.

**BOX 1. Future Advances and Best Practices**

We strongly encourage future studies of phenotypic plasticity to involve the following advances and best conceptual practices:

- Treat plasticity as a category, not a quantity; use reaction norms to study plasticity instead.
- Consider reaction norms over more than two environments.
- Don't limit studies of plasticity over continuous environments to linear reaction norms or linear gene expression profiles.
- Resist the temptation to fit reaction norms using only linear functions; embrace non-linearity.
- Non-plastic reaction norms are nothing special, biologically speaking. Though they are distinctly easy to describe, they are *a priori* no more important biologically than any other reaction norm shape.
- Give greater attention to the distribution of environmental encounters (including ancestral) and examine the implications of organism-mediated encounters (niche construction; habitat choice).
- Avoid automatically assuming that plasticity is adaptive, particularly in novel environments. Indeed, we need a "neutral theory" of plasticity evolution to enable more rigorous analyses and inferences of adaptive plasticity patterns in nature.
- Don't stop with detection of G × E interactions when studying the evolution of phenotypic plasticity. Use reaction norms to unpack causes of G × E variation.

## ACKNOWLEDGMENTS

We thank Jeremiah Busch, Carl Schlichting, and an anonymous reviewer for helpful feedback that improved this commentary as well as numerous colleagues for discussions about plasticity over the years. Our work was supported by the U.S. National Science Foundation (grant number DEB-2217559 to R.G.) and an NSERC Discovery grant (to J.R.S.).

## ORCID

*Richard Gomulkiewicz* http://orcid.org/0000-0001-6346-9576
*John R. Stinchcombe* https://orcid.org/0000-0003-3349-2964

## REFERENCES

Arnold, S. J., and C. R. Peterson. 2002. A model for optimal reaction norms: The case of the pregnant garter snake and her temperature-sensitive embryos. *American Naturalist* 160: 306–316.

Auge, G. A., L. D. Leverett, B. R. Edwards, and K. Donohue. 2017. Adjusting phenotypes via within- and across-generational plasticity. *New Phytologist* 216: 343–349.

Bonduriansky, R. 2021. Plasticity across generations. *In* D. W. Pfennig [ed.], Phenotypic plasticity and evolution: Causes, consequences, and controversies, 327–348. CRC Press, Boca Raton, Florida, USA.

Charlesworth, B., and D. Charlesworth, 2010. Elements of evolutionary genetics. Roberts and Co., Greenwood Village, Colorado, USA.

Chevin, L. M., R. Lande, and G. M. Mace. 2010. Adaptation, plasticity, and extinction in a changing environment: Towards a predictive theory. *PLoS Biology* 8: e1000357.

Crispo, E. 2007. The Baldwin effect and genetic assimilation: Revisiting two mechanisms of evolutionary change mediated by phenotypic plasticity. *Evolution* 61: 2469–2479.

Dewitt, T. J., and S. M. Scheiner. 2004. Phenotypic variation from single genotypes. *In* T. J. DeWitt and S. M. Scheiner [eds.], Phenotypic plasticity: Functional and conceptual approaches, 1–9. Oxford University Press, New York, New York, USA.

Diamond, S. E., and R. A. Martin. 2021. Buying time: Plasticity and population persistence. *In* D. W. Pfennig [ed.], Phenotypic plasticity and evolution: Causes, consequences, and controversies, 185–209. CRC Press, Boca Raton, Florida, USA.

Edelaar, P., and D. I. Bolnick, 2012. Non-random gene flow: An underappreciated force in evolution and ecology. *Trends in Ecology and Evolution* 27: 659–665.

Gavrilets, S., and S. M. Scheiner. 1993. The genetics of phenotypic plasticity. V. Evolution of reaction norm shape. *Journal of Evolutionary Biology* 6: 31–48.

Ghalambor, C. K., J. K. McKay, S. P. Carroll, and D. N. Reznick. 2007. Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Functional Ecology* 21: 394–407.

Gomulkiewicz, R., J. G. Kingsolver, P. A. Carter, and N. Heckman. 2018. Variation and evolution of function-valued traits. *Annual Review of Ecology, Evolution, and Systematics* 49: 139–164.

Gomulkiewicz, R., and M. Kirkpatrick. 1992. Quantitative genetics and the evolution of reaction norms. *Evolution* 46: 390–411.

Johannsen, W. 1911. The genotype conception of heredity. *American Naturalist* 45: 129–159.

Kimura, M. 1983. The neutral theory of molecular evolution. Cambridge University Press, Cambridge, UK.

Kingsolver, J. G., and L. B. Buckley. 2017. Quantifying thermal extremes and biological variation to predict evolutionary responses to changing climate. *Philosophical Transactions of the Royal Society B: Biological Sciences* 372: 20160147.

Kingsolver, J. G., S. Diamond, and R. Gomulkiewicz. 2015. Curve-thinking: Understanding reaction norms and developmental trajectories as traits. *In* L. B. Martin, C. K. Ghalambor, and A. Woods [eds.], Integrative organismal biology, 39–53. Wiley, Hoboken, New Jersey, USA.

Kingsolver, J. G., R. Gomulkiewicz, and P. A. Carter. 2001. Variation, selection, and evolution of function-valued traits. *Genetica* 112–113: 87–104.

Kingsolver, J. G., K. R. Massie, J. G. Shlichta, M. H. Smith, G. J. Ragland, and R. Gomulkiewicz. 2007. Relating environmental variation to selection on reaction norms: An experimental test. *American Naturalist* 169: 163–174.

Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. *Evolution* 30: 314–334.

Matthey-Doret, R., J. A. Draghi, and M. C. Whitlock. 2020. Plasticity via feedback reduces the cost of developmental instability. *Evolution Letters* 4: 570–580.

Ogbunugafor, C. B. 2022. The mutation effect reaction norm (mu-rn) highlights environmentally dependent mutation effects and epistatic interactions. *Evolution* 76: 37–48.

Pfennig, D. W. [ed.]. 2021. Phenotypic plasticity and evolution: Causes, consequences, and controversies. CRC Press, Boca Raton, Florida, USA.

Saltz, J. B., A. M. Bell, J. Flint, R. Gomulkiewicz, K. A. Hughes, and J. Keagy. 2018. Why does the magnitude of genotype-by-environment interaction vary? *Ecology and Evolution* 8: 6342–6353.

Sarkar, S. 2004. From the *reaktionsnorm* to the evolution of adaptive plasticity. A historical sketch, 1909–1999. *In* T. J. DeWitt and S. M. Scheiner [eds.], Phenotypic plasticity: Functional and conceptual approaches, 10–30. Oxford University Press, New York, New York, USA.

Scheiner, S. M., M. Barfield, and R. D. Holt. 2021. The evolution of habitat construction with and without phenotypic plasticity. *Evolution* 75: 1650–1664.

Schmalhausen, I. I. 1949. Factors of evolution: The theory of stabilizing selection. University of Chicago Press, Chicago, Illinois, USA.

Stinchcombe, J. R., J. H. Beder, P. A. Carter, G. W. Gilchrist, D. Gervini, R. Gomulkiewicz, B. Hallgrimsson, et al. 2012. Genetics and evolution of function-valued traits: Understanding environmentally responsive phenotypes. *Trends in Ecology and Evolution* 27: 637–647.

Sultan, S. E. 2015. Organism and environment: Ecological development, niche construction, and adaptation. Oxford University Press, New York, New York, USA.

Sultan, S. E. (2021). Phenotypic plasticity as an intrinsic property of organisms. *In* D. W. Pfennig [ed.], Phenotypic plasticity and evolution: Causes, consequences, and controversies, 3–24. CRC Press, Boca Raton, Florida, USA.

Vaidya, P., and J. R. Stinchcombe. 2020. The potential for genotype-by-environment interactions to maintain genetic variation in a model legume–Rhizobia mutualism. *Plant Communications* 1: 100114.

Via, S., R. Gomulkiewicz, G. de Jong, S. M. Scheiner, C. D. Schlichting, and P. H. van Tienderen. 1995. Adaptive phenotypic plasticity: Consensus and controversy. *Trends in Ecology and Evolution* 10: 212–217.

Via, S., and R. Lande. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. *Evolution* 39: 505–522.

Wang, S., W.-W. Feng, M.-C. Liu, K. Huang, P. A. Arnold, A. B. Nicotra, and Y.-L. Feng. 2022. Inherent conflicts between reaction norm slope and plasticity indices when comparing plasticity: A conceptual framework and empirical test. *Oecologia* 198: 593–603.

Woltereck, R. 1909. Weitere experimentelle Untersuchungen über Artveränderung, speziell über das Wesen quantitativer Artunterschiede bei Daphniden [sic]. *Verhandlungen der Deutschen Zoologischen Gesellschaft* 19: 110–173.

**How to cite this article:** Gomulkiewicz, R., and J. R. Stinchcombe. 2022. Phenotypic plasticity made simple, but not too simple. *American Journal of Botany* 109(10): 1519–1524. https://doi.org/10.1002/ajb2.16068