PHENOTYPIC PLASTICITY MADE SIMPLE, BUT NOT TOO SIMPLE

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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 conversation 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 our 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

[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'.

of development, behavior, genetics, ecology, and evolution) with far more depth and realism than current literature.

The framework (Fig. 1) involves four independent components: patterns of plasticity; environment encounters; fitness consequences; and 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 our 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 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 ourselves): 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 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: 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: 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 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 (Fig. 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 overweight that component of the reaction norm and underweight 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: measure the frequencies of environments an organism actually encounters. Fortunately, many environmental variables (CO2, temperature, salinity, humidity, freezing days, precipitation, etc.) can be measured remotely with data loggers, ibuttons, and other

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

153 instruments. Other, more biotic environments (e.g., competitor or mutualist densities)
154 will require old-fashioned ecological field work. Moreover, documented patterns of
155 environmental encounters will enable researchers biologists to assess the proportionate
156 importance of different environments for the evolutionary and ecological causes and
157 consequences[3] of phenotypic plasticity (e.g., Kingsolver et al. 2001, Kingsolver and
158 Buckley 2017).

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

169 Early models of phenotypic plasticity assumed passive environmental encounters
170 (Via and Lande 1985, Gomulkiewicz and Kirkpatrick 1992, Gavrilets and Scheiner
171 1993), but recent ones consider organisms that actively determine encounters either
172 through habitat choice/preferences or by changing their local environment directly (niche

[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.

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 & 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.

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 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

196 "sensitive period" can, because of developmental or other delays, differ from the
197 environment that determines fitness.
198 The relevant measure of fitness will depend on an organism's life history and how
199 that relates to environmental variability. For example, an individual could experience
200 multiple environments within its lifetime (e.g., daily thermal variation). Individual fitness
201 would integrate over these fine-grained distributions (e.g., Kingsolver et al. 2007). At the
202 other, coarse-grained extreme, an individual experiences a single environment in its
203 lifetime but its descendants could develop in different environments because of *in situ*
204 temporal change or dispersal. The fitness consequences of plasticity for both demography
205 and adaptive evolution must then reflect these among-generational changes.
206 Many studies over continuous environments assume optimizing selection such that
207 the optimum phenotype changes linearly (e.g., Chevin et al. 2010). This assumption is
208 mathematically convenient with a bonus feature: the optimal reaction norm is necessarily
209 linear. Consequently, studies often consider *only* linear reaction norms, which lends itself
210 to the further, conceptual perk that slope directly reflects plasticity. In reality, neither
211 linearity assumption is empirically justified. Future studies should consider nonlinear
212 versions of optimizing selection and distributions that include nonlinear reaction norms.
213 Finally, plastic phenotypes may in fact have no differential effect on fitness, that is,
214 different reaction norms may have equivalent consequences for total fitness. In these
215 cases, phenotypic plasticity is a neutral trait and its evolution is best understood in terms
216 of non-adaptive evolutionary processes including random genetic drift (Lande 1976,
217 Kimura 1983).
218

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 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 (Fig. 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 (Fig. 3). Conceivably, gene-level reaction norms could prove valuable for detailed prediction of evolutionary responses to selection (see previous subsection) 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 ("GxE"), as it is necessary for plasticity to evolve (Saltz et al. 2018). The absence of GxE (parallel

242 reaction norms) implies absence of genetic variation in plasticity. However, the absence
243 of GxE does not imply the absence of plasticity *per se,* nor does the presence of GxE
244 ensure the evolution of plastic genotypes. Consequently, GxE is necessary but not
245 sufficient for plasticity to evolve. Although estimates of GxE variances can sometimes
246 reveal how much fitness variation across environments is maintained by rank changes
247 versus changes in variance (Vaidya and Stinchcombe 2020) it is unknown if those
248 inferences apply to other phenotypic measures of GxE variation. Regardless, one can
249 always use a reaction norm approach to dissect root causes of GxE variation if not
250 necessarily the reverse (Saltz et al. 2018).
251
252 **Conclusion** We urge that future studies of phenotypic plasticity organize around our
253 four-component framework of reaction norms, environmental encounters, fitness
254 consequences, and inheritance (Fig. 1). Box 1 lists some best practices and compelling
255 future research directions suggested by our framework. A complete understanding of
256 phenotypic plasticity requires all four components (Via et al. 1995, Sultan 2021) and,
257 while we might pine for studies that consider the full foursome, this emphatically does
258 not imply that studies must include all of the components to make valuable contributions.
259 Rather, a key advantage of our framework is to provide a simple, but not too simple
260 conceptual "wrapper" for investigations that address one or more of the components we
261 describe, collectively providing a clear and consistent context for how each study
262 contributes to our holistic understanding of phenotypic plasticity and its evolution.
263
264

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 RG) and an NSERC Discovery grant (to JRS).

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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 GxE interactions when studying the evolution of phenotypic plasticity. Use reaction norms to unpack causes of GxE variation.

Figure Captions

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.

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.

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.

Figure 1

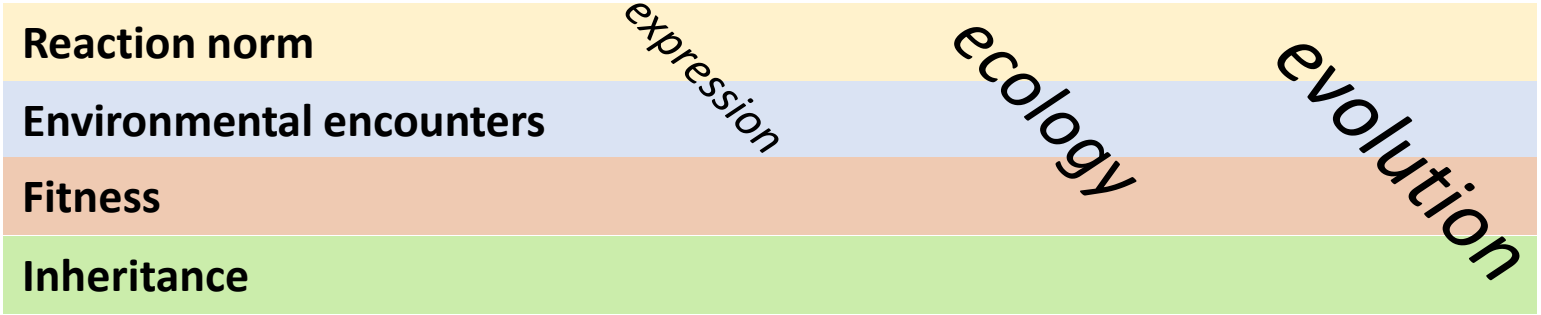
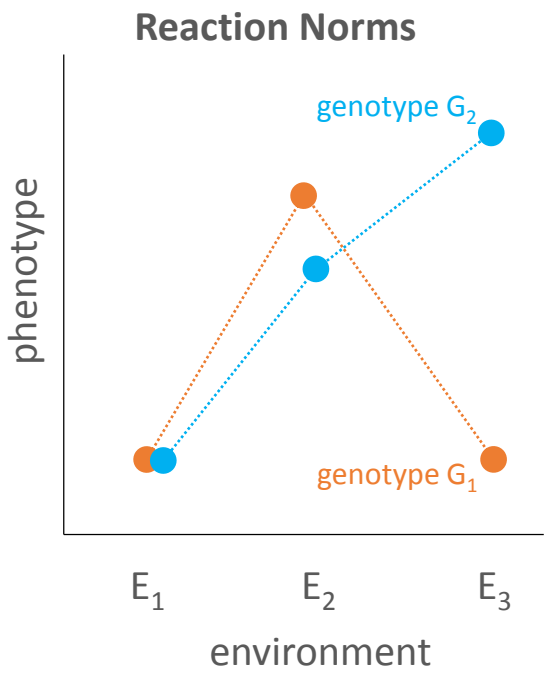


Figure 2


Reaction Norms
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phenotype
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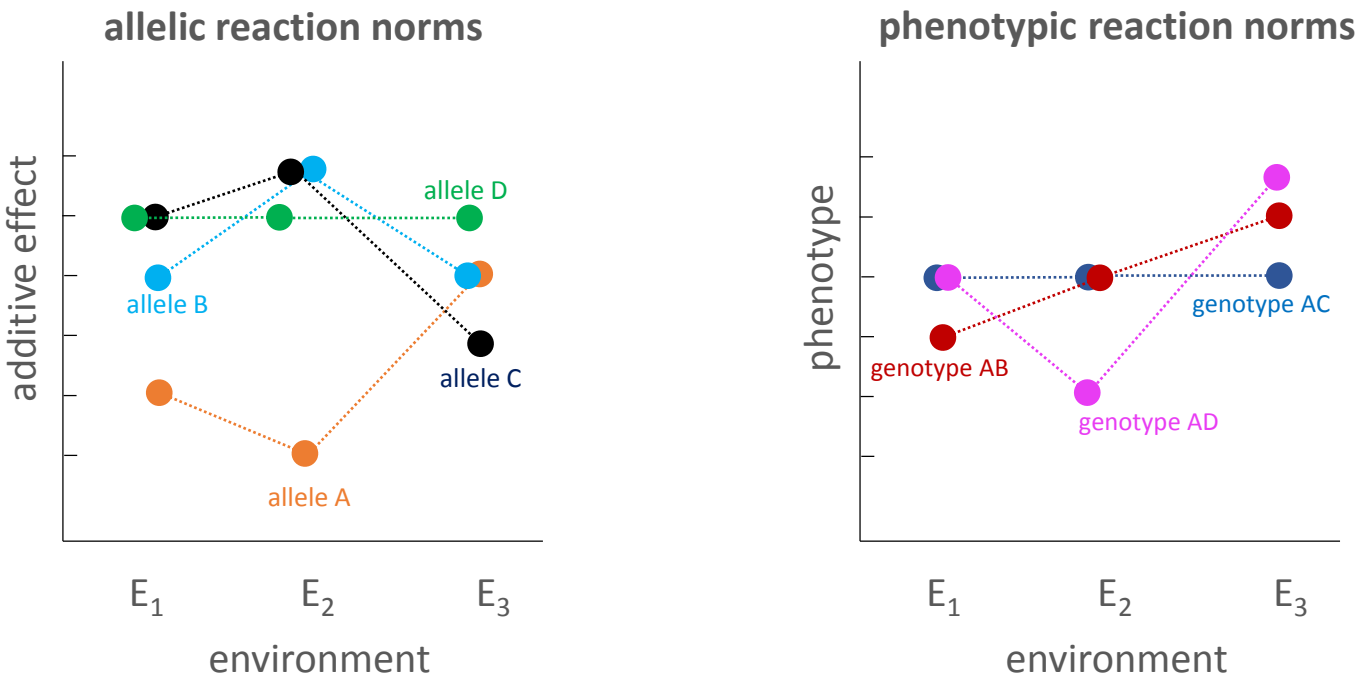
E_2
E_3
environment

Figure 3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