Developmental Plasticity and Evolution

Annalise B. Paaby and Nicholas D. Testa

Contents

Abstract

The environment plays a crucial role in the developing organism, first in defining the developmental trajectory from genotype to phenotype, then by modifying that trajectory by natural selection. Nearly all traits exhibit some degree of phenotypic plasticity: the capacity to change, or to develop in response to, the environment. The plasticity of a trait can itself evolve, and some of the most specialized adaptations include evolved responses to environmental variation. Plasticity has long been theorized to potentiate adaptive evolution, by environmental induction of phenotypes that boosts the potential for subsequent genetic evolution or by revealing cryptic alleles in new environments that in turn generate new adaptive phenotypes. A plastic trait may vary continuously, which can be described by norms of reaction, or it may produce discrete types as a polyphenism, a codified adaptive response to specific environmental signals. The concept of plasticity can also be applied to variation in phenotype associated with a single genotype in a

A. B. Paaby (✉) · N. D. Testa
School of Biological Sciences, Georgia Institute of Technology, Atlanta, GA, USA
e-mail: paaby@gatech.edu; ntesta3@gatech.edu

L. Nuño de la Rosa, G.B. Müller (eds.), *Evolutionary Developmental Biology*,
https://doi.org/10.1007/978-3-319-33038-9_110-1

single environment. Such microenvironmental plasticity defines in part the robustness of a trait. In the evolution of complex traits, tension between plasticity and its opposite, canalization, may be crucial for rapid evolution, adaptation, and the emergence of novelty.

Keywords
Reaction norm · Polyphenism · GxE · Genetic accommodation · Genetic assimilation · Canalization · Evo-devo

Introduction

In natural systems, the environment plays two roles: first, it mediates how genotypes are translated into phenotypes; second, it imposes selection (West-Eberhard 2003; see also the chapter on ▶ "Eco-Evo-Devo"). Together, these functions drive trait evolution, but the role of the environment in the first function can be overlooked even as its dominance is assumed in the second. This oversight may arise due to the explanatory power of genetics and its amenability to controlled laboratory experimentation. However, the ability of organisms to respond plastically to the environment is so ubiquitous that it is an essential component of the living world (Ehrenreich and Pfennig 2016). In many ways, we intuitively understand this: our expectation that the environment will impose influence is folded into many decisions we make, both scientifically and in our everyday lives. Nevertheless, in both mainstream culture and in scientific research, we often turn to genes first for biological explanations. In fact, genetic determinism – which does indeed play a profound role in human health, applied efforts in agriculture, and the evolution of natural populations – cannot be separated from environmental influence. The outcome of a genotype is undefined without specifying the environment. As genotype translates into phenotype, it travels along developmental trajectories that may be labile or robust and may be defined by generations of adaptive evolution or vulnerable to new influences (see the chapter on the ▶ "Genotype-Phenotype Map"). In turn, environmental selection pressures shape not only the phenotypes of an organism but the interactions between development and the environment that produces them.

Definitions and Related Concepts

Phenotypic plasticity is the ability of an individual to alter its phenotype in response to the environment, or the potential of an individual genotype to develop into alternative phenotypes in different environments (Fusco and Minelli 2010; Levis and Pfennig 2017). The first scenario can occur when a phenotype is labile over the course of an individual's lifetime: behavior, for example, or body size or composition, gene expression, or aspects of physiology. Even sex can change, in the case of

some trees, polychaetes, gastropods, and fish. The latter scenario occurs when a trait is expressed only once in a lifetime, like the age of reproductive maturity or the shape of a developmentally irreversible bony appendage. For such fixed traits, the critical aspect is that a single genotype holds the potential for different phenotypes, which are determined by the environment during development. One of the most well-studied examples is the formation of defensive "helmets" in *Daphnia* in the presence of predators (Agrawal et al. 1999).

When a plastic trait is discrete, the distinct phenotypes represent a *polyphenism* (Fig. 1a). Polyphenism is pervasive in eusocial insects as a mechanism for producing different castes, such as workers or queens, and may be controlled by diet, including feeding on royal jelly or pheromones (Simpson et al. 2011). Some moths and butterflies exhibit striking polyphenisms according to seasonal diet, including the development of spring *Nemoria arizonaria* caterpillars into morphs that mimic catkins, or oak tree flowers, versus leaf-eating summer caterpillars into mimics of oak twigs, and the development of prominent eyespots on the wings of adult *Bicyclus anynana* during the wet season versus the duller, camouflaged pattern during the dry season. Some locusts can develop into an antisocial "solitarious" phenotype or a swarming "gregarious" phenotype according to sight, smell, or tactile cues mediated by population density. Other insects exhibit polyphenic morphs with distinct dispersal abilities, such as long- or short-winged crickets and winged or wingless aphids, which are typically induced by signals of resource availability (Simpson et al. 2011). The plasticity of the *Daphnia* helmet (Agrawal et al. 1999) is a classic example of prey species polyphenism triggered by the presence of predators in the environment. This also occurs in barnacles, which grow hunched over in the presence of carnivorous snails, and bryozoans, which develop spines in the presence of predatory nudibranchs (Stearns 1989). Plants can also exhibit defensive plasticity, such as the increased production of mustard oil glycosides by the wild radish following damage by herbivorous caterpillars (Agrawal et al. 1999). Note, however, that plasticity is generally considered a polyphenism only when the phenotypes are discrete.

Most traits are not expressed as discrete types, but instead vary continuously (Fig. 1b). When plotted against variation in the environment, a continuous plastic trait can be represented by a *reaction norm* (Fig. 1c) (Stearns 1989). (It is worth noting that the distinction between a polyphenism and a continuously variable plastic trait can nevertheless be vague; the concept of a reaction norm was first introduced by Richard Woltereck while working in the early 1900s on helmet length in *Daphnia* (Simpson et al. 2011).) Plasticity is extremely pervasive, as the environment influences the expression of most traits. Most traits also exhibit nonzero heritability, indicating that phenotypes are almost always affected by genotype as well. Within populations, these two determinants – environment and genotype – influence trait expression such that different genotypes are likely to exhibit different reaction norms (Fig. 1d). When these functions have different slopes and intersect, we observe a *crossing of reaction norms*. Such nonparallelism is evidence of *gene-by-environment interaction* (GxE), in which the environment influences trait determination nonadditively across genotypes. For example, a genotype conferring slow metabolism might grow only slightly faster in a high-glucose environment compared

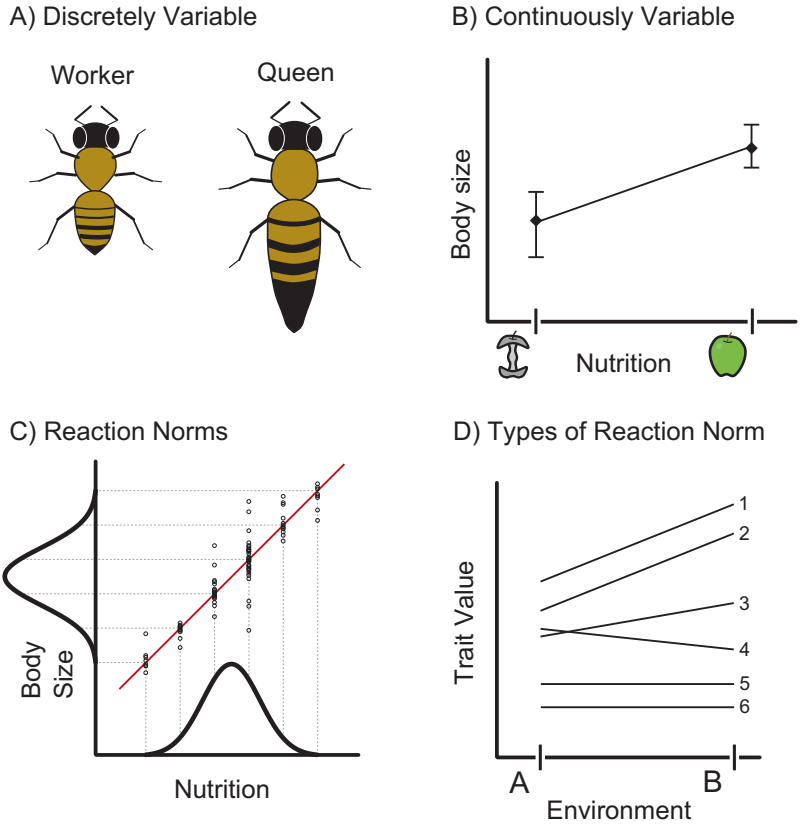

Fig. 1 A plastic trait that takes two or more discrete forms, like the castes of eusocial insects, is known as a polyphenism (**a**). Most traits vary continuously (**b**), and variation in the trait can be plotted against variation in the environment to produce a reaction norm (**c**). Typically, a reaction norm includes only individuals of the same genotype, to eliminate variation in phenotype contributed by the genetic component. Comparing the reaction norms of different genotypes captures the three most important components of phenotype variance: those arising from genetic effects, environmental effects, and the interaction between them (**d**). In this plot, reaction norms 1 and 2 both exhibit phenotypic plasticity, as genotypes 1 and 2 both produce different trait values in different environments. Here, genotype 1 has a higher average trait value than genotype 2, but the environment influences trait determination the same way in both genotypes. Norms 3 and 4 also exhibit plasticity, and the nonparallel slopes indicate a GxE interaction: the environment affects trait determination differently in genotype 3 than in genotype 4. In this comparison, the interaction is negative, as environment B raises the trait value of genotype 3 relative to environment A, but lowers it for genotype 4 (these two genotypes demonstrate interactions with every other genotype on the plot as well, though not all of these are negative). Norms 5 and 6 exhibit no plasticity, as the environment has no effect on trait value

to a low-glucose environment, whereas the difference might be dramatic for a genotype conferring fast metabolism. GxE is pervasive in natural populations (Morgante et al. 2015), and this is to be expected: if different genotypes produced

parallel reaction norms, one should be fittest in all environments and eventually fix in the population (Stearns 1989). Reaction norms themselves can evolve; this is discussed further in the section "Evolution of Plasticity."

The opposite of plasticity is *canalization* (see the chapter on ▶ "Canalization"), the production of an invariant phenotype even in a noisy environment (Stearns 1989). The term was coined by Waddington (1957), to illustrate the entrenched grooves or "canals" that developmental trajectories occupy in the formation of morphological features. Waddington conceived of canalization with regard to genetic variation, but now the term is used both ways, often specified as either "environmental canalization" or "genetic canalization" (Wagner et al. 1997). Although opposite in definition, the relationship between plasticity and canalization is intimate: a plastic trait can evolve into a canalized one via changes in the regulation of the trait's expression (Ehrenreich and Pfennig 2016), and plastic responses can themselves become canalized when responses to an environmental cue become stereotyped, as in a polyphenism. Trait lability and trait robustness may both evolve from the existence of conditionally functional variation, and a major motivation for studying plasticity and canalization is the hypothesis that dynamic tension between these phenomena might enable rapid evolution, adaptation, and the emergence of novel traits (Paaby and Gibson 2016).

The term *robustness* may be used as a synonym for canalization, though canalization is more often conceived in association with development, as in the ability of a developmental trajectory to withstand perturbations to produce an invariant phenotype (Masel and Siegal 2009; see also the chapter on ▶ "Robustness"). If the allelic or epigenetic state of a genic element is responsible for producing either an environmentally canalized phenotype or a plastic phenotype, then this element is considered a *phenotypic stabilizer* (Masel and Siegal 2009). A phenotypic stabilizer is analogous to a *phenotypic capacitor*, likewise a genic element with potential to store or release phenotypic variance, but in this case due to genetic, rather than environmental, variation. The switch of a phenotypic capacitor from one state to another, for example the disabling of the heat-shock protein HSP90, can lead to the release of *cryptic genetic variation* (Geiler-Samerotte et al. 2016; Rutherford and Lindquist 1998). Cryptic genetic variation is standing variation in a population that has little effect on phenotype until a perturbation induces expressivity, either through epistasis, like by the disruption of a phenotypic capacitor, or through GxE, if the perturbation is environmental (Paaby and Rockman 2014; see also the chapter on ▶ "Epistasis").

In a seminal series of experiments, Waddington (1953) used heat shock to release cryptic genetic variation for, and promote eventual *genetic assimilation* of, a "crossveinless" wing morphology in wild-type strains of *Drosophila melanogaster*, kicking off decades of investigation into the potential role of this mechanism in adaptive evolution (see the chapter on ▶ "Conrad Hal Waddington (1905–1975)"). Genetic assimilation occurs when cryptic mutations, neutral under normal conditions, penetrate to phenotype in a new environment, and selection eventually fixes the trait such that the environmental stimulus is no longer required (Fig. 2). Constitutive expression is made possible by new combinations of alleles that underlie the

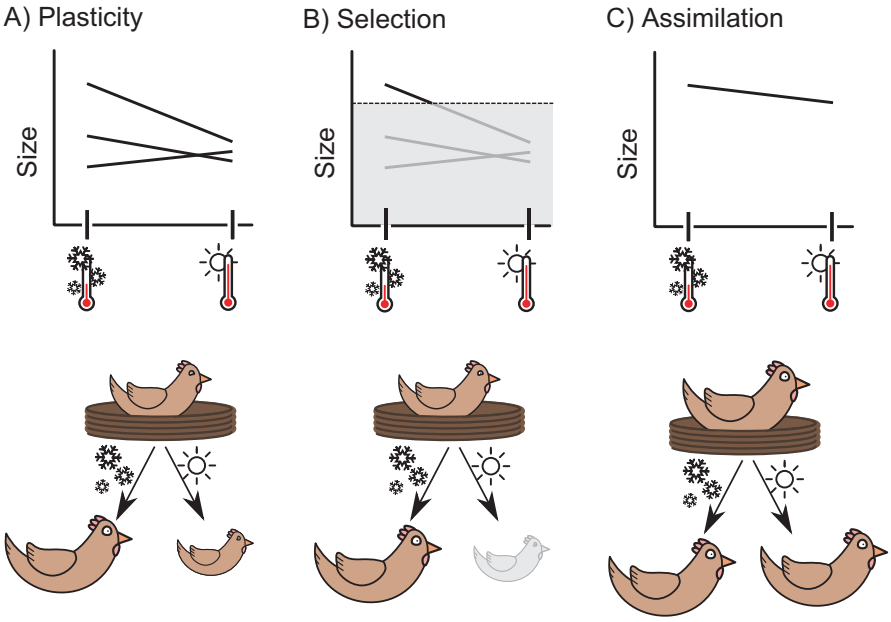

Fig. 2 Genetic assimilation can occur when phenotypic effects of genetic variation are revealed under a specific environment (**a**). The genetic variation is cryptic, such that the phenotype exhibits lower variance in the "normal" environment (here depicted as the warm condition) and higher variance in the new environment (the cold condition). Alleles that produce a novel phenotype (big birds) are now selected (**b**). In the beginning, the new phenotype is only produced in the new environment, but generations of selection produce allelic combinations that eventually fix trait expression in all environments (**c**). Most evidence for genetic assimilation comes from artificial selection experiments in the lab, so the extent of this phenomenon in natural systems is unknown

evolution of the plasticity of the trait, which is known as *genetic accommodation*; genetic assimilation is a dramatic form of genetic accommodation, as plasticity is completely lost in the evolved population (Ehrenreich and Pfennig 2016; see also the chapter on ▶ "Evo-Devo and Niche Construction" and the discussion of genetic accommodation therein). The case for genetic accommodation in trait evolution, including observations of plasticity in natural systems, has been comprehensively addressed in West-Eberhard's book on the topic (West-Eberhard 2003). However, Waddington's original experiments demonstrating genetic assimilation, and the contemporary investigations that succeeded them, provide proofs of principle but do not as yet clarify the extent to which plasticity, GxE, cryptic genetic variation, and canalization govern the evolution of natural populations.

Plasticity-First Evolution

A major question regarding plasticity in evolutionary biology is the relative importance of a "plasticity-first" mode of trait evolution, in which a plastic response to environmental change precedes and enables adaptive change, compared to a genes-first mode, in which selection acts on new genetic variants first and changes in plasticity are secondary. In other words, since the first step toward a new adaptive phenotype involves the production of a new developmental variant, the question is whether trait evolution more often starts with an environmentally induced variant or a variant induced by mutation (Levis and Pfennig 2016). Three factors support a plasticity-first mechanism: first, environmental change is likely to affect many or all individuals in a population, whereas de novo mutation, by definition, occurs in only one individual; second, since plastic responses are always linked to the environments that induce them, they may be primed to increase fitness; and third, environmental plasticity can promote the accumulation of cryptic alleles, a store of heritable variation that might potentiate rapid genetic adaptation (Levis and Pfennig 2016).

A crucial mechanism by which plasticity probably facilitates trait evolution relies on accumulation of cryptic genetic variation on which selection may eventually act, as exampled by Waddington's (1953) evolution of the *Drosophila* crossveinless wing phenotype. Accumulation of cryptic variation is an emergent property of any genetic system with epistasis or GxE, though it may also be facilitated by the evolved stability of developmental trajectories, which can shelter cryptic alleles (Hermisson and Wagner 2004). Once a sufficiently destabilizing environment reveals cryptic variation, selection can target the focal trait but also the environmental dependence of its expression, such that plasticity itself evolves, in the process of genetic accommodation. Waddington's genetic assimilation experiments demonstrate a complete loss of plasticity because the trait became constitutively expressed, but selection can also promote developmental sensitivity to environmental signals. The most extreme form of sensitization results in the evolution of polyphenism (Levis and Pfennig 2016). Evidence for genetic accommodation in natural systems is mostly indirect – since testing trait expression in a true ancestor is typically impossible – but observations of physiological and behavioral traits in house finches, behavior and morphology in stickleback fish, and melanin production in *Daphnia* provide compelling support (Moczek et al. 2011). Trait plasticity can theoretically promote evolvability, because the location of a decision point along the developmental path can evolve just as the terminal phenotype of the plastic trait is adaptively refined (see the chapters ▶ "Developmental Evolvability" and ▶ "Variational Approaches to Evolvability: Short- and Long-Term Perspectives"). This potentially dramatic lability of ontogenetic specification can increase the "evolutionary degrees of freedom" of the system (Moczek et al. 2011).

Despite the still-growing accumulation of observations elucidating how plasticity influences the evolution of natural systems (Levis and Pfennig 2017; West-Eberhard 2003), whether plasticity tends to jump-start evolutionary innovation remains an open question (Moczek et al. 2011). The best case study for this phenomenon involves investigation of spadefoot toads in the genus *Spea*, by Pfennig and

colleagues. *Spea* tadpoles develop into either small-jawed omnivores or large-jawed carnivores, depending on diet. Examination of *Scaphiopus couchii*, an omnivorous species used as a proxy for the non-plastic *Spea* ancestor, revealed slow growth and a release of cryptic genetic variation for size, development, and gut length when exposed to carnivorous conditions. Two *Spea* species also show evidence of genetic assimilation. Both exhibit intermediate ecomorph frequencies when they live alone in a single-species "ancestral" condition, but they show near fixation of one or the other ecomorph, independent of dietary resources, when they co-occur (Levis and Pfennig 2016). A macroevolutionary analysis of nematode evolution also provides evidence for the plasticity-first hypothesis. Here, plasticity appears to have promoted the diversification of mouthpart feeding mechanisms, including the evolution of a predatory and sometimes polyphenic morph with moveable teeth. The comparative analysis of 90 species showed that the historical appearance of mouthpart plasticity is associated with faster evolution, increased diversification, and subsequent independent losses of plasticity (Susoy et al. 2015). On the other hand, one analysis of gene expression and molecular evolution in the spadefoot toad system suggests that plasticity could be a consequence, rather than a cause, of rapid evolution. Genes with differential expression between the omnivore and carnivore ecomorphs were compared to a set of unbiased genes, both in the plastic *Spea* species and in four species that diverged before the evolution of plasticity. The biased genes, those presumed to be associated with plasticity, showed higher variance in expression and faster evolution than the unbiased genes, but the elevated rates predate the evolution of plasticity. With these findings, the authors speculate that plasticity may emerge when fast-evolving, dispensable genes become available for environment-dependent adaptation (Leichty et al. 2012).

Evolution of Plasticity

Given its inherent physical and chemical properties, by default development should respond plastically to environmental influence (Nijhout 2003). Evolved modifications to these biophysical responses to the environment include both the dampening of plasticity, via canalization, and codification of plasticity into a polyphenism. Under this expectation, Nijhout (2003) classifies plasticity two ways. Type 1 emerges as a function of the physical and chemical interface between the developing organism and the environment and is unlikely to increase fitness. Type 2 is an evolved adaptation to a particular environment. For continuous traits, evolution of plasticity means the evolution of reaction norms. Greater environmental heterogeneity speeds reaction norm evolution, though the extent to which environments are novel or rare may matter; cryptic genetic variation released in new environments will expose new reaction norms (Schlichting and Pigliucci 1998). The shape of reaction norms can vary dramatically by trait. Thermal traits often exhibit convex norms, with the lowest values at the temperature extremes; threshold-mediated traits are likely to be logistic; and morphological traits show a diversity of reaction norm shapes, including in different environments (Schlichting and Pigliucci 1998). As for possible genetic

mechanisms that permit plasticity, three have been proposed: pleiotropy, epistasis, and overdominance. The pleiotropic model states that one gene can pleiotropically affect fitness in different environments; the epistatic model expects the interaction of two or more genes, some affecting the height of the reaction norm and some determining its shape (see the chapters on ▶ "Pleiotropy and Its Evolution: Connecting Evo-Devo and Population Genetics" and ▶ "Epistasis"). The overdominance model predicts that plasticity should increase with homozygosity, as heterozygous loci buffer phenotypes from environmental perturbations (Pigliucci 2005).

Although polyphenisms are discrete, some may be the product of a discontinuous environment acting on the norm of reaction of what is otherwise a continuous trait (Nijhout 2003). Others are mediated by developmental switches, but even those may have evolved to direct the development of what was an ancestrally continuous trait. Hormone regulation, especially in insects, is a pervasive mechanism of polyphenism, wherein changes to hormone secretions or hormone sensitivity act on the process of metamorphosis. Notably, the environment that induces a polyphenic trait is often not the environment that imposes selection. For example, seasonal polymorphisms that promote fitness in either warm or cold environments often anticipate those conditions by photoperiod (Nijhout 2003).

In an unpredictable world, the ideal organism would have total plasticity: the ability to form or reform to every situation with maximum fitness. Obviously, total plasticity is not possible, and both costs and limits probably constrain its evolution (Murren et al. 2015; see also the chapter on ▶ "Developmental Constraints"). Costs to plasticity can be inferred from the evolution of specialist species; if a generalist could maximize fitness in multiple environments, then we would not expect specialists, with little ability to thrive outside their niches, to evolve in any of those environments (Kawecki 1994). And yet they do, across broad taxonomic and ecological ranges.

However, empirical studies often fail to detect any fitness costs to generalists. Identifying costs of plasticity can be tricky because they should not be confused with costs of the phenotypes themselves. If a generalist and a specialist both produce adaptive traits in a specific environment, but the traits are unequal and the specialist is fitter, this is not a cost of plasticity but a cost of phenotype. A cost of plasticity is the universal cost incurred to the generalist in all environments, such as the expense of larger genome to house additional genetic machinery. It may be that such universal costs are not so important in limiting the reach of plasticity and that instead insufficient selection is a more important constraint for the evolution of generalists (Murren et al. 2015). For example, generalist species may experience weaker selection against deleterious mutations that erode the fitness of environment-specific traits. Unlike specialists, in which fitness-compromising alleles will be exposed in all members of the population, in generalist populations only the subset of individuals within the specific environment will be subject to selection (Kawecki 1994). Variation in selection also affects the evolution of plasticity. Fluctuations in selection arising from environmental heterogeneity favor plasticity, over timescales occurring within individual lifespans as well as those spanning generations, especially when environmental cues are reliable. Limited genetic variation for plasticity has also been

proposed as a constraint for its evolution, though recent analyses have estimated greater evolution of norms of reaction than of the traits themselves, suggesting that this may not be an important limitation (Murren et al. 2015).

Micro-plasticity and the Quantitative Genetics Perspective

This chapter, and the literature on plasticity in general, has been focused at the level of the organism. However, plasticity can be observed and quantified at the population level, and indeed this is an important consideration in the evolution of populations. From a quantitative genetics perspective, if the mean of a trait within a population changes when the environment changes, the trait is plastic (Pigliucci 2005). The plasticity may include GxE (and it usually does), but it need not, if the environment pushes trait expression in the same direction and with the same magnitude for every genotype. Technically, plasticity may occur even if the mean and variance of the population remain unchanged; consider crossing reaction norms, as in Fig. 1d, but in a perfectly symmetrical hourglass arrangement with a balanced number of individuals for each genotype. In this scenario, plasticity is only at the level of the organism and not at the level of the population, so the plastic response does not change the ability of the population to evolve if a new phenotype is favored. However, population-level plasticity can occur without a change in trait mean so long as the variance changes. Here, the plastic response could enable faster phenotypic evolution, as outliers become targets of selection. This is the premise behind the theory that cryptic genetic variation can potentiate adaptive evolution (Paaby and Rockman 2014). Or, if stabilizing selection disfavors phenotypic outliers, evolution of allele frequencies can occur without phenotypic evolution. Both of these scenarios require that the higher trait variance is associated with the new environment (see the chapter on ▶ "Quantitative Genetics and Evo-Devo")

Usually, plasticity is discussed in terms of defined differences in the environment. However, even within controlled or static environments, individuals of the same genotype do not produce identical phenotypes. This variance in phenotype, due to unknown and uncontrolled variations in the developmental or external environment, is microenvironmental plasticity. This phenomenon has also been understood as developmental noise, stochastic or residual variation, and environmental sensitivity (Morgante et al. 2015).

The existence of microenvironmental plasticity implies that any individual genotype (reared in a constant environment) is associated not with one specific phenotype but with a distribution of possible phenotypes (Fig. 3). And just as genotype, the environment, or an interaction between them determines a phenotypic mean, so too will these factors influence phenotypic variance or the range of microenvironmental plasticity. The influence of genotype on microenvironmental plasticity can be strong, and sometimes its heritability is as large or larger than that of the trait mean. Consequently, microenvironmental plasticity itself can be a target of selection. For quantitative traits under stabilizing selection, microenvironmental plasticity in the trait should evolve toward zero, as the mean centers around the fitness optimum.

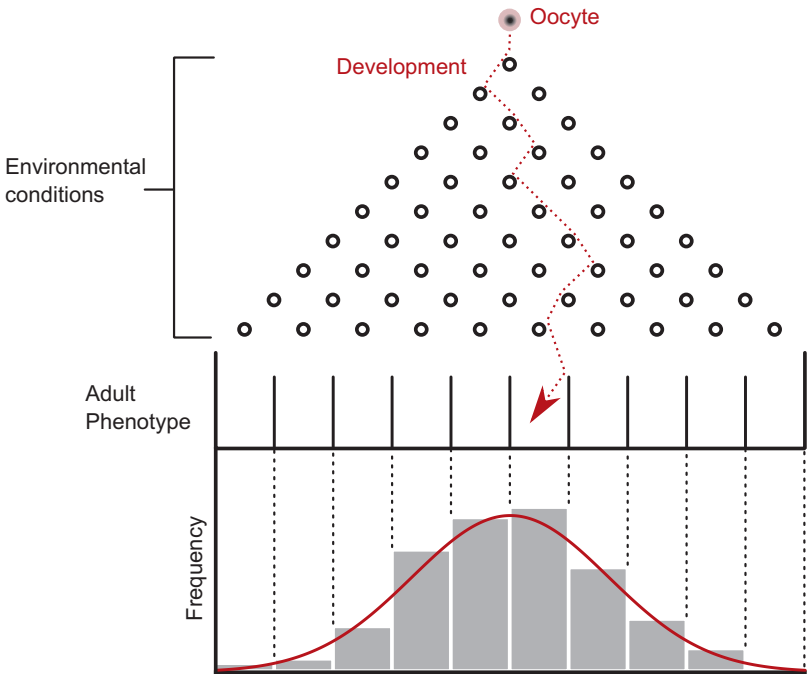

Fig. 3 The phenotype of any individual organism falls along a distribution of possible phenotypes, which is governed by both the individual's genotype and the environment. Plasticity occurs when different environments induce different distributions, but within a distribution, the different possible phenotypes may be considered products of "micro-plasticity" because they arise from minute and unknown variations in an otherwise controlled or consistent background. In this figure, we appropriate the concept of a "Galton board," wherein the pins represent micro-variations in the environment that, either stochastically or by unknown determinants, govern the developmental trajectory of the individual from oocyte to adult. In contrast to Waddington's canonical "epigenetic landscape" (Waddington 1957), here the ultimate phenotypic products are not discrete but continuous and are determined not by canalized developmental trajectories but assume, if we stick with the original conception by Galton, that each pin imposes a 50-50 left-right outcome for the falling ball and that the resulting distribution is approximately normal (Galton 1894). However, we make no assumptions that environmental micro-variations influence developmental outcomes with binomial probability or that phenotypic distributions are always normal

Within fluctuating environments, selection should favor nonzero microenvironmental plasticity, as a bet-hedging strategy increases the probability that some individuals in the next generation will be fit in their environment (Morgante et al. 2015).

Exploiting Plasticity in the Laboratory

Research into the evolution of development can take advantage of dramatic plastic responses to environmental perturbation. Related taxa often exhibit conservation of developmental processes at the morphological level but divergence in the genetic mechanisms that govern them, a phenomenon called developmental system drift (True and Haag 2001; see also the chapter on ▶ "Developmental System Drift"). Analyses of molecular evolution and comparative genomics can provide insight, but functional dissection of developmental processes is especially hard in non-model systems and systems that cannot produce viable interspecies crosses. However, environmental perturbations can decanalize development and induce morphological aberrations that may in turn provide clues to the cellular processes that connect the diverged genes to the conserved phenotype. This is analogous to using genetic perturbations to reveal hidden but functional differences in the developmental mechanisms that vary cryptically within populations (Paaby et al. 2015) or have diverged across species (Verster et al. 2014).

Unlike a gene-based perturbation, an environment-based perturbation might not provide a hypothesis-testing framework with mechanistic specificity regarding developmental variation across lineages. However, this may be compensated by the relative ease and consistency that an environment-based perturbation affords. For example, temperature stress, chemical exposure, and nutrition are easily controlled in the lab. There is no expectation that an environmental stress will necessarily reveal functional differences connected to the stress itself, for example, through a history of selection. Rather, the idea is that the environmental perturbation will destabilize developmental trajectories to reveal cellular or genetic differences between the tested lineages. For example, polymorphism in the candidate gene *Ultrabithorax* was shown to underlie variation of expression in, and eventual genetic assimilation of, the bithorax phenotype in *D. melanogaster* lineages exposed to ether (Gibson and Hogness 1996).

Like those that induced the bithorax (Gibson and Hogness 1996) and crossveinless (Waddington 1953) phenotypes, an informative perturbation is one that is sufficient to deform the developmental trajectory but not so effective that it kills the organism outright. Temperature stress has been used in multiple systems to induce heritable, intraspecific phenotypic variation, some of which recalls other naturally evolved phenotypes (Moczek et al. 2011). Across lineages, divergence in function but also the expression of recurrent phenotypes arises from the twin aspects of lability and robustness that characterize developmental processes (Paaby and Gibson 2016). For traits undergoing developmental system drift, natural selection will favor changes that stabilize the phenotype around the canonical type when de novo mutations or new environments cause deformations to the developmental trajectory. Consequently, a perturbation that unveils glimpses of these deformations will not be one that evokes type 2 plasticity, an evolved adaptation to a known environment, but one that evokes type 1, a physical and chemical response (Nijhout 2003) that can exaggerate differences in mechanism.

Cross-References

- ▶ Canalization
- ▶ Conrad Hal Waddington (1905–1975)
- ▶ Developmental Constraints
- ▶ Developmental Evolvability
- ▶ Developmental System Drift
- ▶ Eco-Evo-Devo
- ▶ Epistasis
- ▶ Evo-Devo and Niche Construction
- ▶ Genotype-Phenotype Map
- ▶ Pleiotropy and Its Evolution: Connecting Evo-Devo and Population Genetics
- ▶ Quantitative Genetics and Evo-Devo
- ▶ Robustness
- ▶ Variational Approaches to Evolvability: Short- and Long-Term Perspectives

References

Agrawal AA, Laforsch C, Tollrian R (1999) Transgenerational induction of defences in animals and plants. Nature 401:60–63

Ehrenreich IM, Pfennig DW (2016) Genetic assimilation: a review of its potential proximate causes and evolutionary consequences. Ann Bot 117:769–779

Fusco G, Minelli A (2010) Phenotypic plasticity in development and evolution: facts and concepts. Philos Trans R Soc Lond Ser B Biol Sci 365:547–556

Galton F (1894) Natural inheritance. Macmillan and Company, New York

Geiler-Samerotte KA, Zhu YO, Goulet BE, Hall DW, Siegal ML (2016) Selection transforms the landscape of genetic variation interacting with Hsp90. PLoS Biol 14:e2000465

Gibson G, Hogness DS (1996) Effect of polymorphism in the *Drosophila* regulatory gene Ultrabithorax on homeotic stability. Science 271:200–203

Hermisson J, Wagner GP (2004) The population genetic theory of hidden variation and genetic robustness. Genetics 168:2271–2284

Kawecki TJ (1994) Accumulation of deleterious mutations and the evolutionary cost of being a generalist. Am Nat 144:833–838

Leichty AR, Pfennig DW, Jones CD, Pfennig KS (2012) Relaxed genetic constraint is ancestral to the evolution of phenotypic plasticity. Integr Comp Biol 52:16–30

Levis NA, Pfennig DW (2016) Evaluating 'plasticity-first' evolution in nature: key criteria and empirical approaches. Trends Ecol Evol 31:563–574

Levis NA, Pfennig DW (2017) Phenotypic plasticity. In: Pfennig K (ed) Oxford bibliographies in evolutionary biology. Oxford University Press, New York

Masel J, Siegal ML (2009) Robustness: mechanisms and consequences. Trends Genet 25:395–403

Moczek AP et al (2011) The role of developmental plasticity in evolutionary innovation. Proc Biol Sci 278:2705–2713

Morgante F, Sorensen P, Sorensen DA, Maltecca C, Mackay TF (2015) Genetic architecture of micro-environmental plasticity in *Drosophila melanogaster*. Sci Rep 5:09785

Murren CJ et al (2015) Constraints on the evolution of phenotypic plasticity: limits and costs of phenotype and plasticity. Heredity 115:293–301

Nijhout HF (2003) Development and evolution of adaptive polyphenisms. Evol Dev 5:9–18

Paaby AB, Gibson G (2016) Cryptic genetic variation in evolutionary developmental genetics. Biology 5(2):28

Paaby AB, Rockman MV (2014) Cryptic genetic variation: evolution's hidden substrate. Nat Rev Genet 15:247–258

Paaby AB, White AG, Riccardi DD, Gunsalus KC, Piano F, Rockman MV (2015) Wild worm embryogenesis harbors ubiquitous polygenic modifier variation. eLife 4:e09178

Pigliucci M (2005) Evolution of phenotypic plasticity: where are we going now? Trends Ecol Evol 20:481–486

Rutherford SL, Lindquist S (1998) Hsp90 as a capacitor for morphological evolution. Nature 396:336–342

Schlichting CD, Pigliucci M (1998) Phenotypic evolution: a reaction norm perspective. Sinauer Associates Incorporated, Sunderland

Simpson SJ, Sword GA, Lo N (2011) Polyphenism in insects. Curr Biol 21:R738–R749

Stearns SC (1989) The evolutionary significance of phenotypic plasticity. Bioscience 39:436–445

Susoy V, Ragsdale EJ, Kanzaki N, Sommer RJ (2015) Rapid diversification associated with a macroevolutionary pulse of developmental plasticity. eLife 4:e05463

True JR, Haag ES (2001) Developmental system drift and flexibility in evolutionary trajectories. Evol Dev 3:109–119

Verster AJ, Ramani AK, McKay SJ, Fraser AG (2014) Comparative RNAi screens in *C. elegans* and *C. briggsae* reveal the impact of developmental system drift on gene function. PLoS Genet 10(2):e1004077

Waddington CH (1953) Genetic assimilation of an acquired character. Evolution 7:118–126

Waddington CH (1957) The strategy of the genes. George Allen and Unwin, London

Wagner GP, Booth G, Bagheri-Chaichian H (1997) A population genetic theory of canalization. Evolution 51:329–347

West-Eberhard MJ (2003) Developmental plasticity and evolution. Oxford University Press, Oxford