PHENOTYPIC PLASTICITY

Functional and Conceptual Approaches

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Phenotypic Variation from Single Genotypes

A Primer

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The Breadth of the Topic

A plant senses competitors for light and elongates its stem, accepting and elevating the challenge. A zooplankter senses diurnal predators and alters its daily vertical movements. A great breadth of ideas fall under the rubric of phenotypic plasticity, and this book is designed to express that breadth as a broad historical and conceptual review, bringing together a variety of (sometimes conflicting) viewpoints. In this chapter, we set the stage for the rest of the book by reviewing these diverse ideas under an intentionally broad definition of plasticity: *environment-dependent phenotype expression*. We emphasize the value of combining proximate, ultimate, and historical views. We hope to convey the need to understand how trait values are influenced by the environment, how individuals vary in this ability, and what such variations imply for how organisms live and reproduce.

Why Study Phenotypic Plasticity?

Phenotypic plasticity, as a paradigm, has broad significance and appeal because it unites perhaps all of biology. Phenotypic plasticity embraces genetics, development, ecology, and evolution and can include physics, physiology, and behavioral science. Although observations of environmentally induced phenotypes were once met blankly or with umbrage (chapter 2), the same observations today provoke biologists to ask how and why plasticity occurs. The change of interest reflects our new understanding that plasticity is a powerful means of adaptation. Alternative alleles or their products react dif-

ferently to the environment; those with favorable reactions persist while others go extinct. This mechanism produces flexible organisms that respond to environmental shifts with beneficial phenotypic changes (chapters 8–10).

Although the view of plasticity as provider of elaborate adaptations has stimulated a recent boom in research, plasticity also can be a liability for organisms. If a single phenotype is best in all circumstances, then environmentally induced deviation away from the best phenotype only reduces fitness. For example, *Eurosta solidaginis* larvae producing an intermediate gall size on goldenrod plants are least vulnerable to predators (Abrahamson and Weis 1999). Yet the gall size produced by the fly depends on the plant genotype, an aspect of the fly's environment (Weis and Gorman 1990). Thus, environmental influences interfere with a fly's ability to consistently produce the best gall size. Often genetic changes are required to compensate for maladaptive plastic responses to the environment (i.e., countergradient variation).

Even when plasticity can potentially help organisms solve the problem of alternative phenotypic optima in different environments, costs and limits of plasticity may make plasticity suboptimal compared with a compromise level of plasticity that is more economical (Van Tienderen 1991; reviewed in DeWitt et al. 1998). And once plasticity has evolved, it may obviate the need for alternative adaptations to environmental variation. So, as either adaptation or constraint, or as part of an integrated set of strategies, plasticity is a key element in the functioning of organisms in variable environments.

Defining Plasticity

A common definition of phenotypic plasticity is *the environmentally sensitive production of alternative phenotypes by given genotypes* (for a semantic review, see Stearns 1989). This definition leaves considerable flexibility in deciding what types of traits exhibit plasticity, because the word "phenotype" is left for individuals to define for themselves. Some scientists prefer to restrict the concept of phenotypic plasticity to developmental processes (chapter 5) rather than other labile means of expressing phenotypes, such as physiological or behavioral shifts (chapter 8). Another view holds plasticity to be any environment-dependent gene expression, which can include gene regulatory processes that may have no gross phenotypic effects.

The danger of too broad a definition is that all biological processes are to some extent influenced by the environment. Thus, everything falls in the realm of plasticity. We fail to see this as a problem, as long as the point of addressing these diverse phenomena as plasticity is to focus on the genotype–environment interaction. Such breadth of scope reinforces the idea that a particular trait value as observed in a given environment always is a special case of a potentially more complex relationship. That is, specific phenotype–environment observations are a fraction of a multidimensional space. This view promotes in our thinking the constant and useful caveat that given phenotype distributions may only apply for the environment in which observation is conducted. Extrapolation beyond given conditions must be justified rather than assumed.

Often there is resistance to use of the language and conceptual framework of plasticity to describe phenomena outside of development. Is suppression of vertical migration in plankton, based on chemical cues from predators, really plasticity? Yet the wealth of concepts and unique analytical tools in the field of plasticity research might inform tan-

gent fields of inquiry with different traditions. Likewise, these other fields can similarly inform mainstream studies in phenotypic plasticity research (e.g., chapters 5, 7, 8, and 11). Thus, each of the following examples, with increasingly liberal definitions of plasticity, can be considered plasticity: (1) development of alternative leaf types in high versus low light, (2) induced chemistry in response to herbivory, (3) production of lactase enzymes in bacteria triggered by the presence of lactose, (4) suppression of the vertical migration instinct in zooplankton by chemical cues from fish, (5) production of fever in endotherms upon infections, (6) buildup of muscles with use, and (7) animal learning. We suggest you draw the line where you wish, but be prepared to learn from those who draw their line elsewhere.

We also distinguish between this broad definition of plasticity as a trait, and a more narrow scope of evolutionary theory of plasticity (chapter 6). Theories of plasticity evolution all deal, in some fashion, with evolution and adaptation in uncertain environments, where uncertainty can be a function of a variety of ecological and biological mechanisms. Other aspects, such as costs of plasticity, are treated as constraints that are fixed by aspects of the organism's biology and are external to the evolutionary dynamic. Plasticity as a paradigm for evolutionary studies encompasses a much wider set of questions (chapter 13) than are covered by evolutionary models of plasticity.

Environmental or Genetic?

Although the definition of plasticity can expand or contract to accommodate various traits, it is important to keep the definition narrow with respect to which aspects of trait variation we refer to as plasticity. Biologists commonly partition total phenotypic variance (V_P) into that due to genetic effects (V_G) and that due to environmental effects (V_E). The equation $V_P = V_G + V_E$ can be found in most introductory biology textbooks. Typical in this gross view is that all deviations from genotype values (= breeding values) are deemed "environmental." But the environmental component is not accorded any functional or breeding value and is not distinguished from developmental or stochastic noise. The recognition of phenotypic plasticity, systematically induced variation attributable to specific environmental states, has allowed us to refine and go beyond the simple dichotomy (Via and Lande 1985).

With recognition of the importance of phenotypic plasticity, we expand the variance partition to $V_P = V_G + V_E + V_{G\times E} + V_{\text{errror}}$ (Scheiner and Goodnight 1984; Via and Lande 1986), which includes explicit recognition of a systematic environmental effect (V_E) and, perhaps more important, a genotype–environment interaction ($V_{G\times E}$). This interaction specifies that the environment's effect is different for some genotypes relative to others (figure 1.1). So persistence of one subset of genotypes over others can change the average effect of the environment. For example, if divergent natural selection favors some genotypes over others based on genotypic reactions to the environment, then adaptive evolution of plasticity will occur.

Finally, a typological dichotomy we must disintegrate involves the question, "Is variation plastic or genetic?" This question is enduring and perennially misleading. The query often reflects the incorrect view of environmental and genetic effects being exclusive entities. Besides the obvious fact that genes and environment can interact ($G\times E$ variation), plastic responses are underlain by genes even when plasticity exhibits no

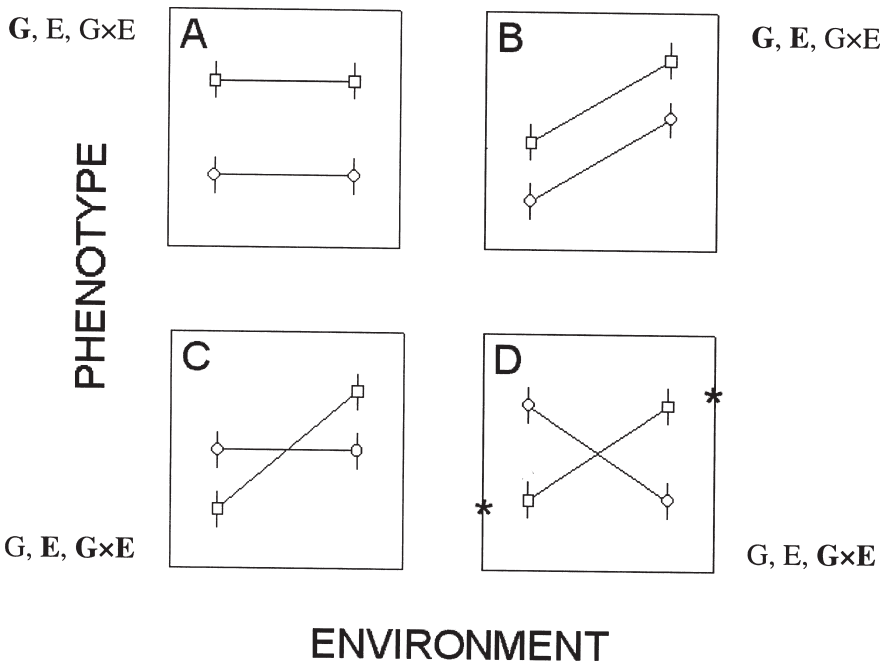

Figure 1.1. A diversity of reaction norms. Mean phenotype and variance in two environments for a family are denoted with circles, and those for another family are denoted with squares. Family means across environments are connected with a line, the *reaction norm*, to indicate familial identity. Boldface terms adjacent to the panels (*G*, *E*, *G*×*E*) indicate significant genetic, environmental, or gene–environment interaction variance, respectively. (A) Flat reaction norms (i.e., no phenotypic plasticity) with consistent genetic differences between families in both environments. (B) Sloped, parallel reaction norms, indicating plasticity and additive genetic variation for trait means but no interaction variance. That is, both genotypes are similarly plastic. (C) Differently sloped reaction norms, indicating genetic variation for plasticity (i.e., interaction variance). Because the overall slope is positive, there is also an effect of environment. Because the marginal mean phenotypes do not differ between families, there is no primary genetic effect (i.e., no genetic main effect). (D) As in C, family marginal means are the same for both families, but the families have opposite reactions to the environment. Therefore, only interaction variance is illustrated here. Asterisks are placed in D to illustrate adaptive optima. Because the family represented by squares would perform better than the other family, and families differ genetically in their degree of plasticity, plasticity would evolve.

additive genetic variance. Such responses are still genetic in the sense that they represent a range of reaction that could be subject to modification if suitable mutations arose and were favored by selection. That all organisms in a population have alleles responding similarly to an observed portion of an environmental gradient does not imply the reaction norm is nongenetic. [Conversely, just because a response is plastic still requires that we distinguish between active and passive plasticity (chapter 10).] The number of fingers on human hands is not heritable in the strict sense, yet it would be hard to contend that the trait is not genetic. These points may seem obvious, but we frequently see

plasticity cited as being "nongenetic," a tradition going back at least to Wright (1931). Put another way, such traits are actually *perfectly* heritable, in the sense that you will express the *exact* phenotype of your parents. Such traits are not heritable in the breeding-value (or evolutionary) sense that the inherited trait distinguishes you from random individuals in the population. Yet there are genetically fixed responses to the environment, genetically fixed plasticity.

Therefore, the answer to the question, "Is variation plastic or genetic?" is simple—it's genetic. Sometimes it is not heritable in the narrow sense (i.e., additive genetic variance), however.

Plasticity versus Developmental Noise

Phenotypic variation from single genotypes can be produced by phenotypic plasticity or developmental noise. Developmental noise consists of random fluctuations that arise during development that alter the phenotypic product of development (Lynch and Gabriel 1987; Scheiner et al. 1991). The effect of developmental noise on fitness could be good, bad, or neutral. For example, developmental noise is costly under stabilizing selection because organisms cannot consistently produce the optimal phenotype (Yoshimura and Shields 1992; DeWitt and Yoshimura 1998). Conversely, developmental noise can be good in fluctuating environments because genotypes with noisy development have broader environmental tolerance (*sensu* Lynch and Gabriel 1987), so the geometric mean fitness among generations is increased.

Scheiner et al. (1991) raised the question of whether phenotypically plastic genotypes are by necessity "developmentally noisy." Scheiner et al. (1991) found no clear relationship between developmental noise and plasticity in bristle number, wing length, or thorax length in *Drosophila melanogaster*. Likewise, DeWitt (1998) failed to find evidence that developmental noise is associated with plasticity for shell shape in a freshwater snail.

In the equation, $V_P = V_G + V_E + V_{G\times E} + V_{\text{error}}$, V_E is plasticity and V_{error} is developmental (or behavioral, etc.) noise. The designation of "noise" implies only that phenotypic deviations from a mean are random in direction but not necessarily random in magnitude. For example, in some environments it may be useful to hedge one's bets by producing variable offspring (Kaplan and Cooper 1984). In such environments, selection favors random phenotypic deviations from a mean, whereas other environments perhaps present strictly stabilizing selection for a mean with no variance. Therefore, it is reasonable to expect interesting evolutionary patterns where both trait means and variances, and potentially higher moments of phenotype distributions (e.g., skewness), vary across environments in an adaptive manner (chapter 7).

To illustrate this point, consider the freshwater snail *Physa*, in which chemical cues from fish induce crush-resistant shells. When fish cues are detected by snails it is certain that fish are present, yet the absence of cues does not always imply the absence of the predator. Thus, noninducing environments (absence of inducing cues) can be inscrutable (*sensu* Leon 1993). Inscrutable environments favor bet hedging, so we can expect snails in the noninducing environment to produce a moderate shell shape with greater variance than that produced in the inducing (fish) environment. Empirical data

match this prediction (figure 1.2; T.J.D., unpublished data). Imagine the simultaneous optimization that selection conducts for all these moments along an environmental gradient. It is perfectly reasonable to speak both of reaction norms for trait means and of reaction norms for developmental noise (DeWitt and Yoshimura 1998) and potentially higher moments (chapter 7).

Genetics or Ecology?

There is a point at which the mathematical abstractions about genetic correlations and phenotypic variance components lose intuitive value to ecologists. What do they tell us about the ecology of the organism? Similarly, geneticists may lose interest in studies

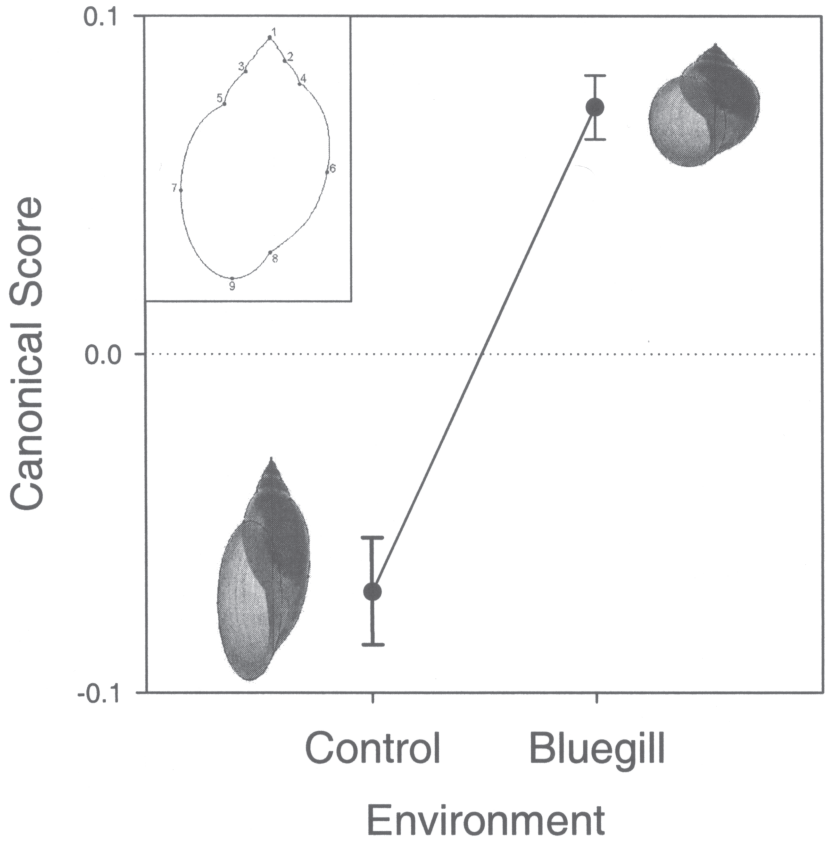

Figure 1.2. Reaction norm for shell morphology in the freshwater snail *Physa virgata*. The reaction norm depicts treatment means and variances of the first canonical axis (from a MANCOVA of partial warp scores; Bookstein 1991). The canonical y-axis describes elongate shells in a noninducing (control) environment and rotund shells in an inducing (bluegill sunfish) environment. Landmarks used in the analysis are indicated in the inset. Shell images illustrate shape differences between treatments magnified 10× using tpsSuper software (Rohlf 2000).

that do not report genetic variation for the traits. We sympathize with both views. It is unfortunate that genetic and functional aspects of plasticity are often studied in isolation of one another; quantitative genetic and functional ecology approaches should be complementary. Consider the traits a snail uses to avoid its many predators. One of the traits (growth rate) has high heritability within the environments, but little correlation (r_G) between environments (DeWitt 1996), which suggests that different genes contribute to the trait in alternative environments (Falconer and Mackay 1996). Although this suggestion is a mathematical deduction, the ecology of the animals indicates the likely cause. Behavior (hence, behavior genes) seems to determine growth rate in one environment—snails raised with fish perform antipredator behaviors that restrict feeding, so growth suffers (DeWitt 1998; Langerhans and DeWitt 2002). In an environment with an alternative predator, snails reduce allocation to reproduction (determined by a different suite of genes), seemingly to reduce time spent in a vulnerable size class (Crowl and Covich 1990; DeWitt 1998). Thus, we have a functional (and adaptive) basis for understanding the mathematical deduction. This example illustrates the sort of multiple-trait, multiple-environment approach that is needed to address plasticity evolution.

In many plasticity studies, fitness itself is frequently treated as a trait (e.g., Schmitt 1993; Stratton 1994). Environmental tolerance curves can be thought of as reaction norms for fitness over an environmental gradient. Conversely, reaction norms for fitness can be thought of as parts of environmental tolerance curves. Each view yields insight into the evolutionary ecology of organisms. However, we think that it is more fruitful to focus on individual traits and their reaction norms, not fitness *per se*, because this allows one to ask questions about plasticity as an adaptation.

Plasticity as Adaptation

The Problem

Consider the Olympic hopeful with two performance passions: to be a great sumo wrestler and to be a great pole-vaulter. He has an obvious problem. He needs a large body mass to increase performance in sumo, but large mass is a liability for pole vaulting. Where performance in one environment is inversely related to performance in another, a functional trade-off exists. Assuming that performance translates into fitness (Arnold 1992), then the functional trade-off creates divergent natural selection. The theoretical maximum fitness under divergent natural selection is achieved only by expressing the best phenotype in each environment (i.e., "perfect plasticity"). That is, there has to be perfect phenotype–environment matching for greatest adaptive value. Perfect plasticity is an insuperable strategy. Obviously, such perfection is never actually achieved in nature when the phenotypes diverge greatly. Yet a critical issue in plasticity studies is to define this maximum—to know what the best phenotype is in each environment. Knowing the functional ecology of an organism, we can adequately assess the degree to which an organism increases its fitness by facultative adjustments to the environment. Once the functional ecology and reaction norms are defined, we can think about the constraints that prevent perfection. Typically, however, this functional approach only proceeds about halfway, as detailed below.

The Benefits of Plasticity

The role of phenotypic plasticity in adapting to natural environments has been the focus of considerable work for decades. However, despite the large volume of work in this field (see figure 13.1), adaptive plasticity has not often been documented thoroughly (Scheiner 1993a; Gotthard and Nylin 1995)—but see chapters 8 and 9 for several examples. This problem persists because proof of adaptive plasticity requires analysis of fitness in multiple environments.

Consider the following example: Dodson (1988) showed that several cladoceran zooplankton (*Daphnia* spp.) produce small bodies when raised with fish, and that small size reduced predation risk in the presence of fish. Is the plasticity in body size adaptive plasticity? Probably. However, all that the preceding information tells us about adaptation is that small body size is favored in the presence of fish. We cannot make conclusions on the adaptive value of plasticity without further information. Three alternatives come to mind:

1. Perhaps small size is also favored in the absence of fish. In this case, nonplastic individuals strictly canalized for small bodies in all environments are more fit than are plastic types.
2. Perhaps there is no selection for body size in the absence of fish, but plasticity exhibits costs or limitations that make it a less suitable strategy than always producing small bodies. In this case, less plasticity might be the optimal strategy.
3. Perhaps plasticity in antipredator behavior is what is really adaptive, but performing the behaviors results in a correlated response in body size (DeWitt 1998). In this scenario, genotypes that are behaviorally responsive to predators survive better and their small body is merely a by-product.

Therefore, to show adaptive plasticity in the face of the first alternative, we have to show the induced phenotype to be adaptive in each environment being considered. Said another way, one must show that the canalized phenotype is inferior in the alternative environment. Examples of one-sided functional ecology are common, where authors demonstrate increased fitness of the induced character state in the inducing environment without testing for higher fitness of the alterative phenotype in other environments (e.g., Appleton and Palmer 1988; Dodson 1989; Parejko and Dodson 1991). Despite this rather stringent litmus test for demonstrating adaptive value, however, the cost of an induced defense is often easy to imagine, and so most cases of one-sided functional ecology are reasonably informative about adaptation. That said, documenting the absolute adaptive value of plasticity requires documentation of functional trade-offs, not merely selection within single environments.

As for alternative 2, we should be mindful of constraints and actively seek them out. The topic of costs of plasticity recently has been in vogue (reviewed in DeWitt et al. 1998). The current tests for costs (e.g., DeWitt 1998; Scheiner and Berrigan 1998; Donohue et al. 2000; Agrawal 2002; Johansson 2002; Relyea 2002) indicate that costs are certainly not pervasive, and probably are rare.

Thus, the major constraint on the evolution of plasticity likely involves limits: logistic constraints that prevent the evolution of perfect plasticity. Obviously, lack of genetic variation for plasticity may be an important constraint. Yet almost all studies find at least some genetic variation for plasticity (Scheiner 1993a). Other limits are probably more important, at least in the long term. Cue reliability is probably extremely

important. In the freshwater snails mentioned above, for example, a severe constraint is that the snails cannot tell predatory sunfish from nonpredatory sunfish (Langerhans and DeWitt 2002). The snails therefore end up responding inappropriately to nonpredatory fish. The responses (reduced growth and altered shell shape) are maladaptive because they make snails vulnerable to common alternative predators and limit fecundity. So, research emphases need to shift to include as much (or more) work on limits as is currently being directed to costs.

The third alternative, correlations among traits masking the true object of selection, illustrates a topic rarely addressed in plasticity studies. For more general studies of natural selection, this issue was brought to the fore 20 years ago (Lande and Arnold 1983). Recent advances show how these methods can be applied to trait plasticities (Scheiner and Callahan 1999; Scheiner et al. 2000, 2002b). We must consider the entirety of the organism in order to determine patterns of selection and parse functional adaptations.

Concluding Remarks

A central accomplishment of the Modern Synthesis (Provine 1971) has been the gain of a deep understanding about how natural selection shapes phenotypes. Up to now, that understanding has been confined almost entirely to fixed traits or traits in only a single environment. We are now ready to achieve a similar deep understanding divergent natural selection and the evolution of trait plasticity. To achieve this understanding, we need to understand the nature of plasticity, its evolution, and its effects on diversification. We need to know more about the mechanistic underpinnings of plasticity (chapters 3–5). We need to understand how plasticity is optimized and integrated with other strategies for dealing with variable environments (chapters 6 and 7). We need comprehensive (i.e., two-sided) studies of functional ecology, and we need to address constraints more often than has been common (chapters 9 and 10). We need to define the relevant trait space of plasticity (chapters 8 and 11). Finally, we need to discern how plasticity participates in evolutionary diversification at levels above populations (chapter 12).

Central to all these aspirations is not only that we expand among topics studied under the rubric of plasticity, but also that we integrate them. Our goals for this book are to present a diverse collection of ideas that embrace the breadth of concepts surrounding plasticity and to provide a pathway toward that integration.