Adaptive Plasticity in Amphibian Metamorphosis

What type of phenotypic variation is adaptive, and what are the costs of such plasticity?

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Amphibians, as a group, are geographically widespread, occurring on most major land masses and in a wide variety of habitats. The diversity of behavioral, physiological, and life history traits exhibited within the Amphibia (Duellman and Trueb 1986), as well as their experimental tractability, makes this group excellent for ecological and evolutionary studies.

Many amphibian species breed in temporary ponds that are occasionally filled by rain. These pools are variable in duration, depending on their initial depth and the frequency of rainfall (Newman 1989). Although the aquatic habitat provides an opportunity for growth before the terrestrial phase of life begins (Wilbur and Collins 1973), larvae are exposed to a high risk of mortality as ponds dry. In addition to differing in duration, ponds may vary in the density of predators, the density of conspecifics, food availability, and thermal stratification.

How do individual animals respond to variability in the aquatic larval environment? How does the environment influence individual fitness, and how are developmental responses to the environment molded by natural selection? More generally, what is the role of phenotypic plasticity—the specific phenotypic responses of the organism to specific environmental factors—in the ecology and evolution of organisms in variable environments? Amphibian studies, especially those of the larval stage, have proven to be an effective setting for addressing these questions (Wilbur 1990).

A plastic genotype might be the jack-of-all-ponds but the master of none

Phenotypic plasticity has recently received increased attention (e.g., Scharloo 1989, Scheiner and Lyman 1989, Schlichting 1986, 1989, Stearns 1989a, Travis in press, Via 1987, West-Eberhard 1989). These articles have reviewed the various definitions of phenotypic plasticity, the methods of measuring plasticity and genetic variation in plasticity, and the ecological and evolutionary consequences of plasticity. In this article, I review the evidence for adaptive phenotypic plasticity in larval amphibians to illustrate some of the difficulties in identifying adaptive plasticity and interpreting effects of the environment on the phenotype, and I suggest a general outline of the limits to the evolution of adaptive plasticity, focusing particularly on costs of plasticity.

Phenotypic plasticity

Stearns (1989a) reviewed the terminology associated with environmentally induced phenotypic variation and the definitions of phenotypic plasticity that have been used. Here, I consider *phenotypic plasticity* to mean a deterministic response to specific environmental factors that results in different phenotypes in different environments. This definition corresponds to the usage by Stearns (1989a) of plastic "norm of reaction." Stearns defines *norm of reaction* as a genotype-specific profile of phenotypes produced over some range of environmental conditions. A plastic norm of reaction is one for which the relationship between phenotype and environment has nonzero slope (i.e., different phenotypes are produced in different environments).

Not all such environmentally induced phenotypic variation is adaptive. I refer to a character state (e.g., a norm of reaction with nonzero slope) as adaptive when it confers higher fitness, in the current ecological context, than alternative character states (e.g., a nonplastic norm of reaction). The adaptive value of a particular norm of reaction depends on the distribution of environmental conditions and the net increment in relative fitness that is attributable to the different phenotypes that are produced (Via 1987).

The evolution of adaptive plasticity may be limited by a number of factors. Limits referred to as *constraints* are those that make evolution toward a certain character state (e.g., a particular norm of reaction) less likely. For example, a genetic constraint indicates a lack of genetic variation (Via 1987). A developmental constraint indicates that the current developmental processes cannot pro-

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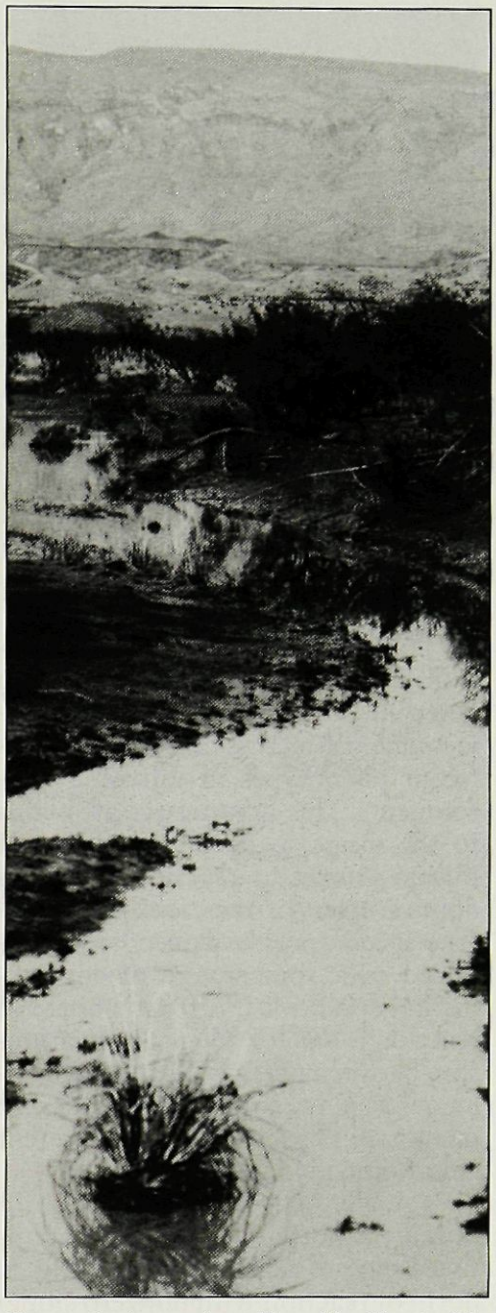

Figure 1. A pond on Tornillo Flat in Big Bend National Park, Texas, where spadefoot toads breed. The photo was taken the day after the pond filled. The pond lasted another ten days.

duce the character state. A cost is another type of limit that refers to fitness trade-offs between traits (Stearns 1989b). If there is a cost to plasticity, then the potential increase in fitness due to the ability to produce alternate phenotypes will be offset, to some extent, by a lower contribution to fitness of other traits.

Desert amphibian responses to pond drying

I have studied the ecology of larval spadefoot toads (*Scaphiopus couchii*, family Pelobatidae) to learn how environmental variation in ephemeral desert ponds affects natural selection on tadpole growth and development (Newman 1988a,b, 1989). Deserts are places of low, infrequent rainfall. Rain-filled pools, ranging from small puddles to large playas, have water in them for relatively short periods of time, yet they often provide the only opportunity for desert amphibians to breed (Figure 1). On my study site, spadefoot toads laid eggs in ponds that lasted anywhere from three days to several weeks, although durations greater than two weeks were uncommon (Newman 1989). Consequently, within a single population, tadpoles were faced with considerable variation in habitat quality.

For desert amphibians, rapid development improves the chances of survival by allowing metamorphosis before the pond dries (Figure 2). *S. couchii* tadpoles can develop from egg to metamorphosis in as little as eight days under some conditions (Newman 1989), and other desert species are nearly as fast (e.g., Bragg 1965, Forge and Barbault 1977, Low 1976, Pfennig 1990).

However, a genetically fixed, rapid rate of development may not confer the highest fitness if pond duration is variable, because additional growth in longer-lasting ponds is sacrificed (Figure 3). Larger size at metamorphosis may increase fitness by improving the chances of survival in the terrestrial environment or by resulting in earlier maturity or larger size at maturity (Berven 1990, Martof 1956, Semlitsch et al. 1988, Smith 1987). If tadpole development is plastic, such that metamorphosis occurs earlier in short-duration ponds but is delayed in longer-duration ponds, then plasticity may confer higher fitness than either a fixed fast or a fixed slow rate of development.

I observed such adaptive plasticity in an experiment I conducted in Big Bend National Park, Texas (Newman 1988a, 1989). *S. couchii* tadpoles developed faster and metamorphosed at a smaller size in short-duration experimental ponds than in ponds of longer duration. The larval period in natural ponds was also highly correlated with pond duration, indicating that flexibility in development is important under completely natural conditions (Newman 1989).

Nondesert species response to pond drying

Other species of amphibians have also been observed to respond to pond drying. In an experiment conducted in large outdoor tanks, *Bufo americanus*, which often breeds in temporary ponds, metamorphosed earlier in a rapidly drying experimental treatment (50 days) than they did in a slow drying treatment (100 days; Wilbur 1987). In the same experiment, the southern leopard frog *Rana utricularia*, which breeds in more permanent water, did not respond adaptively to the drying treatment. In another experiment, *Hyla pseudopuma*, a tropical species that typically breeds in temporary water, metamorphosed earlier under conditions of decreasing water volume than under constant water volume (Crump 1989).

Studies of the salamander, *Ambystoma talpoideum*, have found an even more dramatic effect of pond permanence. In semipermanent or permanent ponds, *A. talpoideum* larvae may not metamorphose at all, but they may become reproductively mature while retaining larval morphology (paedomorphosis; Semlitsch and Wilbur 1988). Larvae from six populations were plastic in the life-history pathway they followed: they metamorphosed more frequently from experimental drying ponds, but paedomorphosed more frequently in experimental constant ponds (Semlitsch et al. 1990). There were differences among populations in their average response to the experimental treatments as well, suggesting genetic differentiation among them in plasticity.

Developmental plasticity and other environmental factors

Pond drying is clearly a potential cause of mortality for amphibians, but it is not the only selective factor in the aquatic environment. Responses to two other aspects of habitat quality—predation and food level—have also received considerable attention in studies of tadpole ecology. In a laboratory experiment on *B. americanus* larvae, Skelly and Werner (1990) observed that tadpoles exposed to higher perceived predation risk (presence or absence of larvae of the dragonfly

Anax junius separated from the tadpoles by a mesh barrier) metamorphosed earlier and at a smaller size. Wilbur and Fauth (1990) also found that *B. americanus* tadpoles metamorphosed earlier, and at a smaller size, in the presence of a predator, in this case the newt *Notophthalmus viridescens*. Both of these experiments demonstrated that tadpoles can and do respond to risk of mortality in the aquatic habitat.

A great deal of work has focused on the effects of food and tadpole density on growth and development rates (e.g., Newman 1987, Smith 1983, Wilbur 1980). Much of the response to density and food may not represent adaptive plasticity. Tadpoles raised under crowded or low-food conditions typically develop slower and metamorphose at a smaller size than tadpoles raised under low-density and high-food conditions (reviewed in Wilbur 1980). The rates of growth and development are constrained by resource availability. However, adaptive plasticity in response to food level can still occur.

Wilbur and Collins (1973) suggested that it would be advantageous for timing of metamorphosis to depend on a tadpole's growth rate, because growth rate provides a direct indication of per capita food availability. In their model, for tadpoles above a minimal size, metamorphosis is initiated if a tadpole's growth rate dropped below some threshold level, indicating a deterioration in habitat quality. Alford and Harris (1988) experimentally altered food level available to tadpoles of *B. americanus* as they developed, and the results were consistent with Wilbur and Collins' predictions.

Wilbur and Collins did not include in their model a direct response to pond drying, although it is likely that conditions for growth deteriorate as a pond dries and gets more crowded. In other words, a decline in growth rate may be caused by pond drying, in which case their model could account for earlier metamorphosis in drying ponds. Semlitsch (1987) found that larval growth rate of *A. talpoideum* salamanders decreased as the experimental pond they were in dried, although most did not metamorphose. Failure to metamorphose was probably a result of insufficient time to complete development at the available level of resources. Interactions between environmental factors means that adaptive responses to a particular factor may be seen under some background conditions, but not others (Newman 1987, Wilbur 1987, Wilbur and Collins 1973).

In principle, any trait that affects fitness in a variable environment may be plastic. Two subspecies of tiger salamanders, *Ambystoma tigrinum*, develop elongate teeth and became cannibalistic in response to the density of conspecifics (Collins and Cheek 1983). In amphibian larvae, the development of a distinctive, carnivorous feeding apparatus, including larger mouth size, wider beak, and shorter gut in response to ingestion of fairy shrimp, has been found in the spadefoot toad *Scaphiopus multiplicatus* (Pfennig 1990, Pomeroy 1981). Carnivorous spadefoot tadpoles grew faster and metamorphosed earlier than omnivorous tadpoles, demonstrating that changes in morphology may also affect life history traits (Pfennig 1990, Pomeroy 1981).

Genetic variation in phenotypic plasticity in amphibians

For plasticity to evolve, there must be genetic variation for the norm of reaction within a population. Few amphibian studies have measured levels of genetic variation in plasticity.

My study of spadefoots documented variation within a population based on a small sample of families (Newman 1988a). Semlitsch et al. (1990) found that the propensity to metamorphose (versus paedomor-phose) varied among populations of *A. talpoideum*. Berven et al. (1979) found differences between populations of green frogs (*Rana clamitans*) in the response of larval development to temperature.

If nothing else, these results indicate that there is genetic variation in how environmental factors affect larval development. Much more work is required to assess levels of genetic variation in plasticity within populations.

Identifying adaptive plasticity

Much, if not most, of the within-population phenotypic variation in growth and development of amphibian larvae is the result of environmental influences. The studies I have reviewed were selected because they illustrate adaptive plasticity most unambiguously. However, many environmental factors can cause phenotypic variation, and not all (or even many) of the effects are unambiguously beneficial to the organism.

The general question, then, is to what extent does environmentally induced phenotypic variation reflect adaptive plasticity? Two questions must be answered: How does the organism respond to its environment (i.e., what is the norm of reaction), and what is the effect on fitness of a particular norm of reaction, relative to other norms of reaction (including a fixed phenotype, or norm of reaction with zero slope)?

Answering the first question involves raising individuals of known clones (or sibships or populations or however close you can get to genotype) in a range of environments (see Via 1987 for further discussion). To answer the second question, the relative fitness of phenotypes, or at least major components of fitness, must first be measured in each environment (see Endler 1986). Knowing the norm of reaction, the relative fitness of different phenotypes in each environment, and the frequency distribution of environments, the overall relative fitness associated with each norm of reaction can be estimated.

Some of the difficulties in recognizing adaptive plasticity can be illustrated with amphibian metamorphosis in temporary ponds. The first task is to determine the norm of reaction, or relationship between phenotype (development rate) and environment (pond duration). Recognition of a consistent relationship is complicated by a number of problems.

- The developmental response need not be directly to the disappearance of water but may be caused by any environmental factor or combination of factors that are highly correlated with pond drying.
- There may be nonadaptive effects of the environment superimposed on or interacting with adaptive responses. Some environmental factors may impose proximal constraints on the ability to respond, such as when low

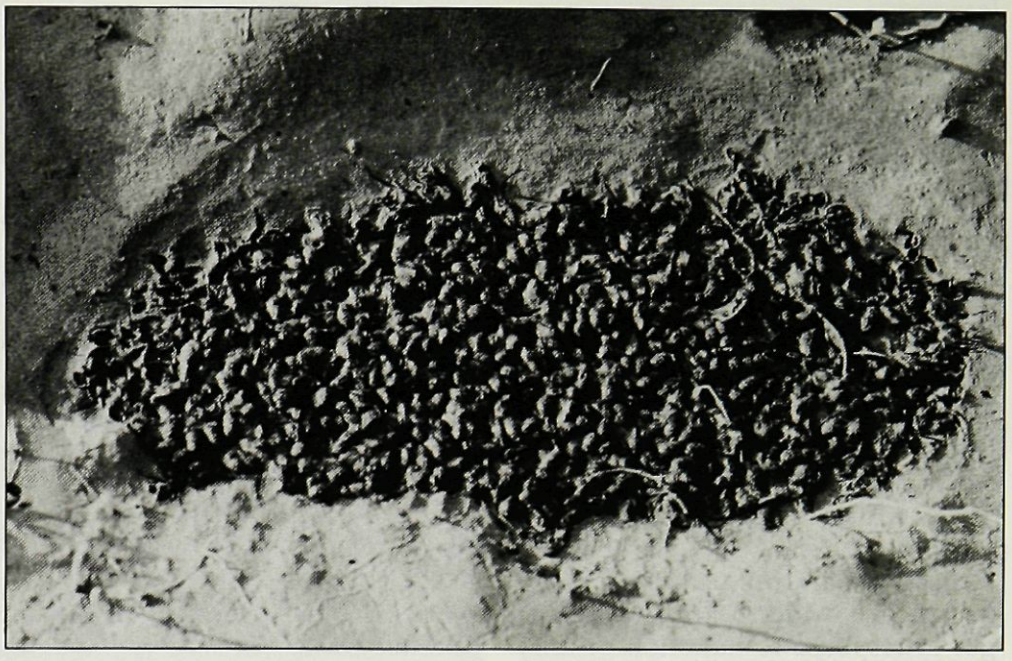

Figure 2. If development is too slow, tadpoles are unable to metamorphose before the pond dries. This photo shows the result: dead tadpoles.

resource availability resulting from intense intra- or interspecific competition prevents an adaptive response to pond drying (Newman 1989, Wilbur 1987).

- There may also be genetic variation within a population for phenotypic plasticity (a genotype-environment interaction). Indeed, this condition is necessary for natural selection to act on the norm of reaction. However, failure to recognize genetic differences may result in consistent responses within genotypes being masked by different responses of other genotypes (Via 1987).

Determining responses, therefore, requires carefully designed experiments based on an adequate knowledge of the organism's ecology and the experimental subject's pedigree.

Let us consider the first two of these problems in more detail. How can we interpret phenotypic plasticity when the proximal cues and mechanisms of the response do not directly involve the feature of the environment that makes the response necessary? For example, metamorphosing before a pond dries is necessary for survival, but development may not be accelerated by decreased water volume per se. Rather, some other factor that is correlated with decreased water volume, such as increased temperature or increased density, may provide the proximal cue or mechanism. In fact, the proximal mechanism of responses to pond drying or to decreasing food levels remains unknown.

However, even if pond drying does not directly cause accelerated development, the effects of correlated factors can still be adaptive, and consequently subject to selection. Genotypes that are particularly sensitive to these factors will have a selective advantage in a habitat where pond duration is variable, all else being equal. If there is genetic variation for sensitivity to such cues (and therefore genetic variation in norms of reaction), and differential fitness among genotypes because of their different norms of reaction, then an indirect response to pond drying can be favored by natural selection.

The primary difficulty in recognizing adaptive plasticity occurs when the response to an environmental factor could be fortuitously beneficial (at least under some circumstances) but not necessarily the result of natural selection. Plasticity would then be a byproduct of the developmental or physiological system and not an adaptation per se (a "spandrel," sensu Gould and Lewontin 1979).

For example, at one level, it would be difficult to consider plasticity in tadpole development an adaptation if accelerated development in drying ponds was the result of increased temperature. Accelerated development with higher temperature is a well-known phenomenon in amphibians and other ectotherms (e.g., Corbet 1962, Smith-Gill and Berven 1979). When the ancestors of spadefoot toads, or even the earliest amphibians, began to experience varying pond durations, their development was almost certainly already subject to temperature effects. However, sensitivity to temperature (or any other factor) can be modified by natural selection (Huey and Kingsolver 1989), so tadpoles from habitats with variable pond duration may be more sensitive to an increase in temperature than tadpoles from other habitats. Studies of other aquatic animals (dragonflies) support this idea (Corbet 1962). In this sense, the specific response of an organism to temperature can be considered an adaptation, even if the general temperature effect appears to be a "spandrel" (sensu Gould and Lewontin 1979).

Because of this possibility, claims about the adaptiveness of a response to environmental factors, especially those with nonadaptive alternative explanations, must be based on measurements of fitness components (see Endler 1986). However, comparisons of norms of reaction among populations in different habitats may provide a useful starting point for developing hypotheses about adaptive plasticity.

There are still other complications. A single environmental factor may influence the phenotype in qualitatively different ways. Increased temperature may provide a proximal cue to pond drying if there is a threshold temperature above which metamorphosis is initiated or if the temperature increase is abrupt. I found that temperature profiles of desert ponds changed dramatically a few days before drying (Newman 1989). This sudden change may trigger a developmental switch (sensu Smith-Gill 1983) that initiates metamorphosis, in addition to any direct effect that increased temperature may have. Alternatively, the adaptive response to pond drying may be the result of natural selection for increased sensitivity of development to temperature effects, as described previously.

In principle, the involvement of a particular environmental factor could be evaluated experimentally. Preventing temperature increases in drying ponds (e.g., by using shade cloth), for example, might reveal the role of temperature in the developmental response

to pond drying. If a response to pond drying is seen in ponds that do not get hotter as they dry, then temperature provided neither a cue to pond drying nor the direct mechanism. If a response to pond drying is no longer seen, then temperature was involved in some way. However, the experiment does not reveal the adaptive value of plasticity, only (maybe) the proximal mechanism.

Knowing the mechanism is not a necessary condition for measuring the adaptive value of a response. However, the more we know about proximal mechanisms, the better our chances of understanding the physical limits to plasticity. We also need to know the mechanism to compare responses among populations and species. If we see superficially similar responses, but know the mechanisms are different, we can infer that the responses evolved independently.

Limits on the evolution of adaptive plasticity

We have only begun to explore the role of adaptive plasticity in variable environments. We know even less about the proximal mechanisms of plasticity. We can, however, make some generalizations about the situations favoring the evolution of plasticity, or, conversely, limiting it. For plasticity to evolve by natural selection,

- the environment must be heterogeneous and genotypes likely to encounter more than one environment
- the phenotype conferring the highest relative fitness must vary with environmental conditions
- the benefits of plasticity to an individual's fitness must outweigh any costs of plasticity
- genetic variation in plasticity must exist in a population (Bradshaw 1965, Levins 1968, Schlichting 1986, Schmalhausen 1949, Travis in press, Via 1987).

Most organisms experience some amount of environmental heterogeneity, but it is not clear if most exhibit adaptive plasticity. To fully appreciate the adaptive role of phenotypic plasticity, we must also consider why the theoretically ideal norm of reaction might not evolve. In other words, when environmental conditions vary and the phenotype conferring the highest fitness varies with environmental conditions (the first two conditions), why would adaptive plasticity not evolve? Limits to the evolution of plasticity can be placed into four nonexclusive, and possibly nonexhaustive, categories: limitations based on an inability to sense the environment, developmental or physiological limits to the ability to respond to the environment (physical constraints), trade-offs between the ability to respond and other traits that affect fitness (in which case the third condition is not met), and lack of genetic variation for adaptive plasticity (the fourth condition is not met). In the remainder of this discussion, I explore in more detail each of these limits.

Deficient sensory capabilities. If the environment is truly unpredictable to the developing individual, then it cannot respond. There is nothing to respond to. If tadpoles, for example, could not perceive the qualities of their pond, especially its likely duration, then there would be no signal from the environment to be transduced into faster or slower development.

Environmental predictability is a function of the actual pattern of variability in the environment and the organism's ability to perceive the state of the environment. That is, environmental conditions such as pond drying may be unpredictable because there are no proximal cues (tadpoles probably cannot predict whether the pond will be refilled before it dries, or if the bank will cave in and fill in the pond). Alternatively, there may be many factors in the habitat that can be used to predict habitat quality, but if sensory capabilities are inadequate, then the habitat is unpredictable from the organism's perspective. If the environment is highly unpredictable, nonspecific phenotypic variation (variation that is not the result of an adaptive response to a specific factor) may still prove beneficial under some circumstances (Kaplan and Cooper 1984).

Inability to respond. Constraints on plasticity might also stem from physical limits on the range of phenotypes that can be produced or on the rates of developmental or physiological processes. Developmental mechanisms might not be sufficiently flexible to allow adaptive plasticity over a wide range of optimal phenotypes, and genotypes may exhibit adaptive plasticity only over a limited range of conditions. The maximal rate of development may also constrain adaptive plasticity, depending on when signals are received from the environment relative to the time when selection acts.

Costs of plasticity. The cost of plasticity is probably the least-understood limit to the evolution of plasticity. It is intuitively obvious that the benefits of plasticity to relative fitness must exceed the costs for plasticity to be favored by selection. It is not intuitively obvious what causes a cost of plasticity or how to measure it. Clearly, though, if there are costs of plasticity, they will be incurred by plastic genotypes and not by nonplastic genotypes. Therefore, to measure costs and benefits of plasticity, the relative fitness of genotypes with different norms of reaction (levels of plasticity) must be measured within all of the selective environments a population experiences.

The benefit of plasticity in a trait (call it X) is the ability to produce phenotypic values of that trait that confer relatively high fitness in all or some of the environments. Such an ability is beneficial because the environment is variable. The cost of plasticity in trait X is defined as a decreased contribution to fitness of other traits (Y) in all or some of the environments, resulting from a trade-off between plasticity in trait X and the average phenotypic value or level of plasticity of trait Y (and therefore the effect of trait Y on fitness). The trade-off is presumably caused by the functional integration of the traits (Schlichting 1989, Stearns 1989b), preventing them from evolving independently. Trait Y could be any trait that affects fitness, including the average value of a trait (X or some other trait) across environments, or plasticity in another trait, or plasticity in trait X over a different range of environments. Perhaps the main reason for our deficient knowledge of costs of plasticity is the difficulty in recognizing and measuring such trade-offs (Stearns 1989b).

To illustrate some of the subtleties in recognizing costs of plasticity, consider the following example. Black and Dodson (1990) observed that in *Daphnia pulex* the development of a structure called neckteeth, which is a morphological defense against predators, was associated with lower reproductive rates. In clones from ponds with varying predator densities, the production of neckteeth was inducible by high predator levels (Parejko and Dodson 1991).

These studies provide a good example of adaptive plasticity. Plasticity (facultative production of neckteeth) was beneficial, because the risk of predation varied and neckteeth reduced mortality rates when predators were present, whereas the cost of neckteeth (lower reproductive rates) was avoided in the absence of predators.

However, the cost of neckteeth is not a cost of plasticity (contra Black and Dodson 1990), because it would presumably be incurred regardless of whether the structure was inducible or obligate. A cost of plasticity, in this case, would involve a trade-off between the ability to respond to variation in predator density and other traits that affected fitness. If, for example, the developmental system that allowed neckteeth to be inducible required more energy than the systems that could not produce neckteeth, or that produced neckteeth obligately, then there would be an energetic cost of plasticity. Costs of plasticity could be measured, in this hypothetical scenario, by comparing, in the absence of predators, the fitness of clones with inducible defenses (plastic) to the fitness of clones that never produce neckteeth (nonplastic). The fitness of clones with inducible neckteeth and the fitness of clones with obligate neckteeth could be compared in the presence of predators. (Comparing the same pairs of clone types, but with environments reversed, was what we did to measure the benefits of plasticity; see preceding paragraph).

The same reasoning applies to growth and development of amphibian larvae in ponds of varying duration. The benefit of plasticity in development rate is larger size at metamorphosis in long-duration ponds than would be attained by a genotype with obligate fast development and lower mortality in short-duration ponds than would be experienced by a genotype with obligate slow development. The cost of metamorphosing at a small size in short-duration ponds, as with the cost of producing neckteeth, is not a cost of plasticity, unless genotypes with plastic development grow slower than tadpoles with fixed development in short-duration ponds.

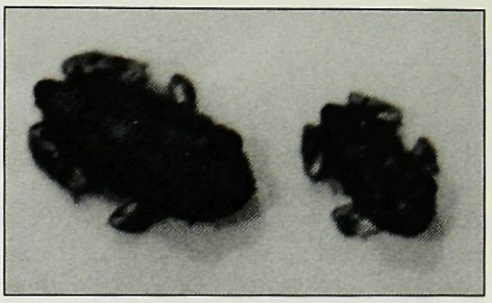

Figure 3. Variation in the size of toads at metamorphosis. The larger of the toadlets underwent metamorphosis the previous day and still has a tail stub. The smaller toadlet (9-millimeter snout-vent length) had metamorphosed two days earlier in a different pond.

Other possible costs of plasticity might include slower development (and higher mortality) than obligate fast-developing genotypes in short-duration ponds, or less growth (and smaller size at metamorphosis) than obligate slow-developing genotypes in long-duration ponds. In other words, the plastic genotype might be a jack-of-all-ponds but the master of none.

In one of my quantitative genetic experiments on *S. couchii* tadpoles (Newman 1988a), all families exhibited accelerated development in drying ponds, but the ones with the fastest average development rate were also the least plastic in growth, and the one with the slowest average development rate exhibited the greatest plasticity in growth. The apparent cost of plasticity in growth was a slower average development rate and higher mortality in short-duration ponds. If this trade-off is real, and not simply an artifact of inadequate sampling of norms of reaction in the population, then it is presumably a result of the way in which growth and development are functionally related, suggesting that a detailed knowledge of the developmental process would facilitate our ability to identify costs of plasticity.

Lack of genetic variation. Evolution of any trait by natural selection may be limited by the availability of genetic variation in the direction of the optimal phenotype. Via and Lande (1985) and Via (1987) presented a detailed model of genetic limitations on the evolution of phenotypic plasticity, based on quantitative genetic analyses.

Lack of genetic variation for plasticity means that all genotypes respond to the environment in similar ways (i.e., they have parallel norms of reaction). The norm of reaction of each genotype can be thought of as a combination of phenotypes (e.g., fast development in short-duration ponds, slow development in long-duration ponds). For plasticity to be favored by natural selection, genes must exist in a population that code for a combination of phenotypes that confer high relative fitness in the environments in which they are produced. If genotypes can produce adaptive phenotypes in only one environment, then plasticity will not be favored by selection (e.g., perhaps if there are developmental or physiological constraints on the range or combination of phenotypes a single genotype can produce, which may be more likely with a wider range or greater number of environments).

Lack of appropriate genetic variation in such a scenario is a superficial indicator of a deeper physical constraint. Until we know more about such physical constraints, however, this is a chicken-or-egg problem. Lack of a physical mechanism for plasticity could also mean that the mutations that would make the optimal norm of reaction physically possible simply have not occurred.

If all combinations of phenotypes are physically possible—that is, developmental or physiological systems can respond to the full range of environments the organism might experience and produce the range of phenotypes associated with the optimal norm of reaction—then lack of genetic variation for the combined optimal phenotypes is a temporary (but possibly long-term) constraint on the evolution of the optimal norm of reaction (Via 1987). Of course, the environment may not be at equilibrium, and the ideal norm of reaction may, thus, never be realized (Via and Lande 1985).

Additionally, if there are many possible environments, the appearance by mutations and recombination of a single genotype with appropriate (high-fitness) phenotypes in all of the environments may be unlikely, even if physically possible (Via 1987). One genotype may have an optimal norm of reaction for one set of environments, whereas another genotype has an optimal norm of reaction for some other set of environments, creating a trade-off in the ability to respond to the two sets of environments. If both sets of environments are common enough, the conditions for maintaining a genetic polymorphism in plasticity may be realized.

Clarifying the patterns

One potentially useful approach to clarifying patterns of adaptive plasticity is to compare norms of reaction among populations that differ in the levels of environmental variation they experience (e.g., Semlitsch et al. 1990). Although this method would require measuring norms of reaction and frequency distributions of environmental conditions in many populations, it would provide a direct assessment of the prevalence and magnitude of plasticity in specific traits and the contexts in which plasticity is found or not found. Ideally, this work would lead to a mechanistic analysis of the relationship between phenotypes and fitness.

Another approach, which might also reveal limits to adaptive plasticity is to generate and test models of optimal norms of reaction in a specific system. This method forces us to be explicit about the assumed relationships among environments, phenotypes, and fitness. Knowing the frequency distribution of environments and the relationship between phenotype and fitness, we can use optimality models (e.g., Ludwig and Rowe 1990, Werner 1986) to predict ideal norms of reaction, where the most fit phenotype is always produced. Differences between observed and predicted norms of reaction could be used to evaluate the existence and magnitude of limits to the evolution of ideal norms of reaction.

This data would not, however, reveal the nature of such limits. Correlations between norms of reaction of different traits (Schlichting 1989) could be experimentally measured and inspected for trade-offs involving plasticity.

These methods are complementary and require empirical studies of both the norms of reaction and the relationship between phenotypes and fitness as a function of the environment. With an expanded database on adaptive plasticity, the greatest need will be a better mechanistic understanding of the factors that might limit the evolution of plasticity, and particularly information about physical constraints and fitness trade-offs involving plasticity.

A better understanding of the mechanisms of phenotypic plasticity, both internally and at the interface between the organism and environment, and of the functional integration of traits would aid in identifying limits to plasticity. Therefore, collaborations among evolutionary ecologists, physiologists, and developmental biologists would be profitable.

Finally, we need more data on levels of genetic variation in plasticity within populations. Because genotypes with varying levels of plasticity could be compared, this information will not only indicate the potential for current selection on plasticity but also facilitate investigations of trade-offs and constraints involving plasticity. This goal is a more realistic view of the ecological and evolutionary consequences of environmental variability.

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