The Evolutionary Significance of Phenotypic Plasticity

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The Evolutionary Significance of Phenotypic Plasticity

Phenotypic sources of variation among organisms can be described by developmental switches and reaction norms

Stephen C. Stearns

Variation, the fuel that feeds evolutionary change, originates at the levels of both the genotype and the phenotype. Genetically identical organisms reared under different conditions may display quite distinct characteristics. Until recently, the types and sources of such phenotypic variation have been given little consideration in evolutionary theory. But a knowledge of the mechanisms and developmental patterns underlying phenotypic variation is crucial to the understanding of important evolutionary phenomena. Therefore some biologists are predicting an increasing focus on this variation leading to a Renaissance of the Phenotype (Scharloo page 465 this issue).

The processes generating phenotypic variation have not been a focus of the traditional evolutionary disciplines. Population genetics concentrates on changes in gene frequencies. Some other disciplines examine phenotypes, but not how they arise. For example, quantitative genetics attempts to connect genotype with phenotype. Life-history theory addresses the interactions of phenotypic traits that determine fitness.

To encourage biologists to consider how a developing organism may respond to a variable environment, in this issue of *BioScience* my colleagues and I describe one major type of phenotypic variation, which is described by a relationship known as a reaction norm.

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A reaction norm is a mirror that reflects environmental effects into phenotypes

Presenting one class of phenotypic variation, Stanley Dodson describes the ecology of predator-induced reaction norms, common in aquatic systems but rare elsewhere (page 447 this issue). Arie van Noordwijk summarizes the role of reaction norms in genetic ecology, where ecophysiology intersects with quantitative genetics (page 453 this issue). He pays special attention to the ecology of small birds. Next, Carl Schlichting examines how correlations among traits change across environmental gradients, an area where especially good information is available from plant population biology (page 460 this issue).

In the final article, Wim Scharloo shows how reaction norms can be analyzed as genotype-environment-mapping functions, an area where biochemistry interacts with developmental biology to influence gene expression (page 465 this issue). Thus the articles here span the range from genetics and biochemistry through developmental biology and physiology to ecology and evolution. Reaction norms is a theme that ties together many areas of biology.

Response to variation

Environmental variation provokes a variety of responses. The following definitions are intended to standardize word usage in this set of articles.

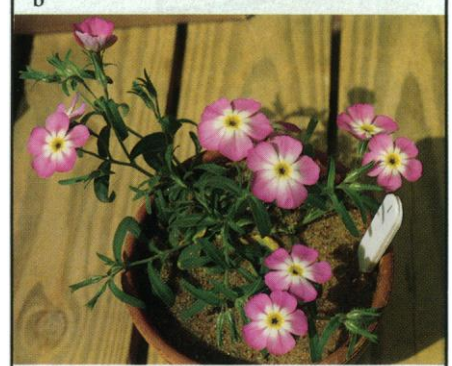
- If organisms always produce the same phenotype, regardless of variation in the environment, the relationship is described as *canalization*. In addition, Waddington (1942) used this term to describe the production of the same phenotype despite changes in genotype (due to mutation or recombination). I separate the canalization processes by referring to *environmental canalization* and *genetic canalization*. Schmalhausen (1949) used the term *autonomous development* for what I have called environmental canalization. Many traits that reflect ancestry and constraint are both environmentally and genetically canalized.
- *Phenotypic plasticity* is a general term that covers all types of environmentally induced phenotypic variation.
- When an organism produces a phenotype that varies as a continuous function of the environmental signal, the relationship is called a *reaction norm* (Woltereck 1909). A reaction norm is usually represented as a line or curve on a graph that plots a phenotype against an environmental factor (Figure 1). For example, a reaction norm could describe an increase in size that correlates with decreased environmental temperature, a relationship

Phenotypic variation in clutch size, timing of breeding cycle, and egg and body size of the great tit, *Parus major,* has been used in the combined study of ecophysiology and quantitative genetics (see page 453). Photo: © Basler Meisengruppe.

Developmental response to environment. Barnacles (*Chthamalus anisopoma*) react to presence of predatory snail (*Acanthina angelica*) during development. When the snail is present, the barnacle grows in a bent-over form that is resistant to predation (left, above and below) but has lowered reproductive capacity. When the snail is not present, the barnacles develop in a more typical form (right, above and below) with high reproductive output (Lively 1986a,b). Top view (above); side view (below). Photos: D. Stewart, Univ. of Canterbury, Christchurch, New Zealand.

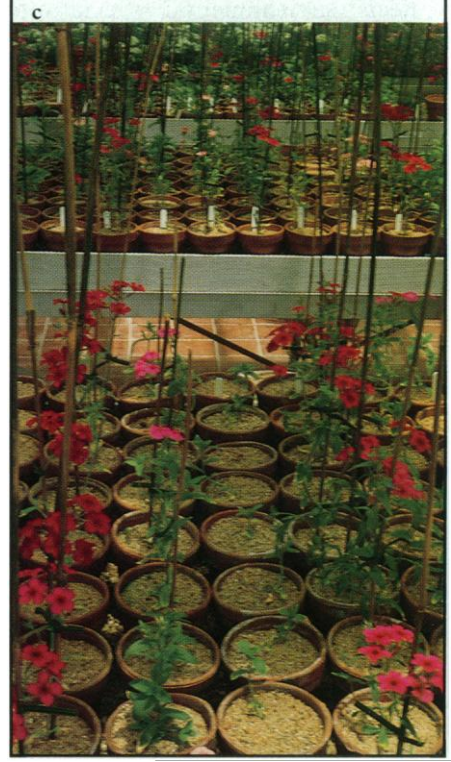
a

b



Plants of the genus *Phlox* have been analyzed to determine the effects of environment on the correlations among a variety of vegetative and reproductive traits (see page 460). (**a**) *Phlox cuspidata.* (**b**) *Phlox roemeriana.* (**c**) *Phlox drummondii* in a greenhouse experiment. Each row represents a different treatment. Note differences in size and time to flowering. Photo: C. D. Schlichting.

c



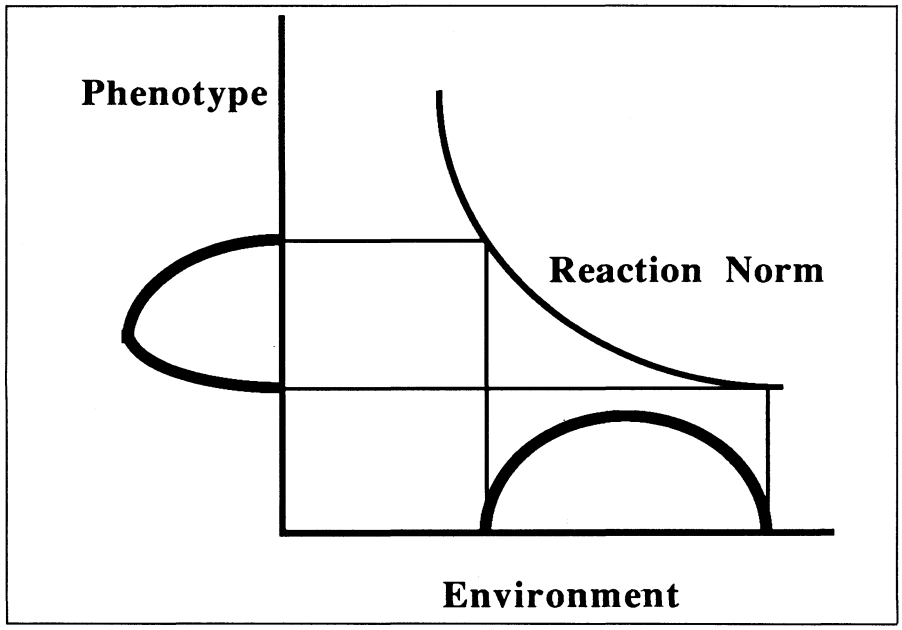


Figure 1. Reaction norms transform environmental variation into phenotypic variation. The differences between the two distributions can be considerable. From Suzuki et al. (1986).

common in insects, fish, amphibians, and reptiles. This restrictive definition distinguishes reaction norms from other types of phenotypic plasticity. Schmalhausen called this relationship *dependent development.* Some biologists find it helpful to think of the reaction norm as a curved mirror that reflects environmental effects into phenotypes.

- Reaction norms can be either *inflexible,* in which a characteristic once determined is never changed later in the organism's life, or they can be *flexible,* in which a characteristic can be altered more than once in the development of the same individual. Phenotypic variation in most life-history traits, such as growth rate or age at maturity, is described by irreversible reaction norms. Life history traits describe growth, reproduction, and survival and include, for example, age at maturity and birth rates.
- *Polyphenism* is the situation in which one genotype produces two or more discrete phenotypes in response to an environmental signal. The term *polyphenism* is the analog among phenotypes of the term *polymorphisms* among genotypes. Butterflies provide a visually striking example of polyphenism (see cover). Polyphenisms can also be found in other organisms, including rotifers, locusts, and barnacles. An alternation of phenotypes is usually associated with a seasonal alternation of backgrounds (for crypsis) or models (for mimics). For example, spring caterpillars of *Nemora arizonaria* feed on oak catkins low in tannin and mimic catkins; summer caterpillars feed on leaves high in tannin and mimic twigs. The switch between the two morphs is determined by tannin concentration in the food (Greene 1989).
- A hypothetical mechanism for phenotypic plasticity known as a *developmental switch* (Levins 1968) is thought to produce the polyphenisms. In this case, an organism assumes one of two or more possible phenotypes depending on the environment. For example, there may be one phenotype for values of an environmental variable below a threshold and another phenotype for values above that threshold. Schmalhausen (1949) called this condition *autonomous regulative development.* Developmental switches have been observed for such characteristics as environmental sex determination in crocodiles and turtles and caste determination in social insects.

History of the reaction-norm concept

Weismann (1885) viewed the organism as conceptually separable into two parts: one, the germline, associated with genetic transmission and the other, the soma, interacting with the environment. Weismann's dichotomy still fits neatly into the explanatory structure of microevolution (Figure 2), testimony to his pervasive influence on biology. In an influential text in 1909, Johanssen based his distinction between genotype and phenotype on Weismann's dichotomy. In that same year, Woltereck coined the term *reaction norm* to describe phenomena he first observed in *Daphnia.* Over the course of a season, within successive generations of a single clone, the body shape changes (cyclomorphosis). Woltereck's first description of reaction norms made several points that remain important.

> The recognition, that any quantitative trait can only be fully characterized through. . . a large number. . . of 'phenotype curves,'. . . we can denote as. . . the reaction norm of the quantitative trait being analyzed. . . The complete reaction norm with all its innumerable specific [environmental] relations is. . . inherited. The biotypes of *Daphnia* have originated through heritable changes in their reaction norms. . . (Woltereck, 1909, pp. 135–136).

Daphnia cyclomorphosis (see Figure 2 in Dodson, page 447 this issue) is a continuous phenotypic response (Jacobs 1987). Changes within a clone do not involve genetic differences, and the response differs among genotypes. Because this reaction norm is genetically variable, it is capable of evolving.

Between 1930 and 1970, many evolutionary biologists commented on the significance of reaction norms, but most did not make the idea a prominent element of their theories. Wright (1932, p. 147) said, "Individual adaptibility is. . . not only of the greatest significance as a factor of evolution in damping the effects of selection. . . but is itself perhaps the chief object of selection." Wright saw phenotypic plasticity as an agent that uncoupled phenotype from genotype: if the organism were adaptively plastic, it would produce superior pheno-

types across a range of environmental conditions and make genetic change less necessary.

Schmalhausen (1949, pp. 7–8) observed that part of plasticity is not adaptive: ". . . every genotype is characterized by its own specific norm of reaction, which includes adaptive modifications of the organism to different environments. . . Nonadaptive- . . . morphoses arise as new reactions which have not yet attained a historical basis. Usually such morphoses are extremely unstable."

Dobzhansky (1951, pp. 21–22) proposed that reaction norms provide the essential changes during evolution: ". . . what counts in evolution are the phenotypes. . . the genes act through the developmental patterns which the organism shows in each environment. What changes in evolution is the norm of reaction of the organism to the environment."

Waddington (1953, 1957) suggested that phenotypic plasticity extends the ecological range of a species, exposing it to selection pressures that it would otherwise not encounter and creating the opportunity for genetic assimilation. In contrast to Wright's idea that plasticity reduces the amount of genetic change in evolution, Waddington saw it as creating the opportunity for more genetic change.

The two views are not mutually exclusive; they apply to different time scales and different ecological situations. Wright's notion applies to adaptive plasticity evoked in the normal range of environments that the population has historically encountered. Waddington's applies to new situations, in which plasticity allows the organisms to survive but has the character of Schmalhausen's unstable morphoses, "which have not yet attained a historical basis."

Mayr (1963, pp. 146–148) said that reaction norms deserved more attention: "The genotype is not a mold into which the characters are cast, but rather a 'reaction norm' interacting with the environment in the production of the phenotype. . . Phenotypic modifiability, phenotypic stability,. . . and behavioral stability- . . . deserve much greater attention by evolutionists than they have so far received." But he did not mention reaction norms again in his book on animal evolution.

Most recent genetics texts do not mention the concept, with the outstanding exception of the book by Suzuki et al. (1986). Futuyma's (1986) text on evolutionary biology devotes one paragraph and one figure to reaction norms, then it mentions the concept again only once.

Until recently, the place given to reaction norms in evolutionary thought was mostly ceremonial. Notable exceptions were Schmalhausen (1949), Bradshaw (1965), and Levins (1968). These biologists placed phenotypic plasticity and reaction norms at the center of their thought and have strongly influenced the direction of current research. As Bradshaw noted, "Plasticity of a character appears to be (a) specific for that character, (b) specific in relation to particular environmental influences, (c) specific in direction, (d) under genetic control not necessarily related to heterozygosity, and (e) able to be radically altered by selection" (pp. 149–150).

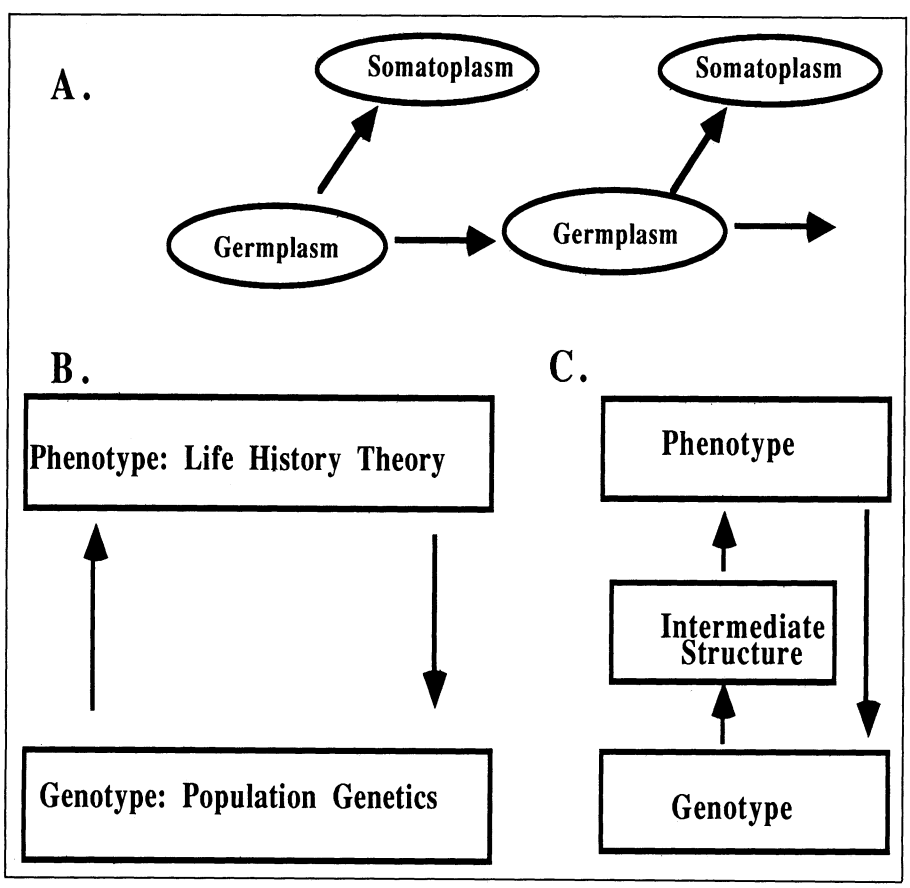


Figure 2. Weismann's distinction between germ plasm and soma (a) fits neatly into the explanatory framework of evolutionary biology (b). Population genetics explains changes in gene frequencies given fitness differences among genotypes. Life history theory explains the phenotypic sources of those fitness differences. Development is not included. The mechanisms producing reaction norms are one type of intermediate structure (c) required in evolutionary explanation.

Evolutionary studies

Reaction norms have important implications for genetics and life-history evolution. These relationships can be nonadaptive, maladaptive, or adaptive.

Nonadaptive and maladaptive reaction norms. The reaction norm can represent a nonadaptive or maladaptive response to unusual environmental conditions. Schmalhausen (1949) considered such relationships to be not yet incorporated by evolution into the developmental repertoire. Nonadaptive responses also are an inevitable result of the physicochemical nature of organisms. Although much of the information required to construct an organism resides in

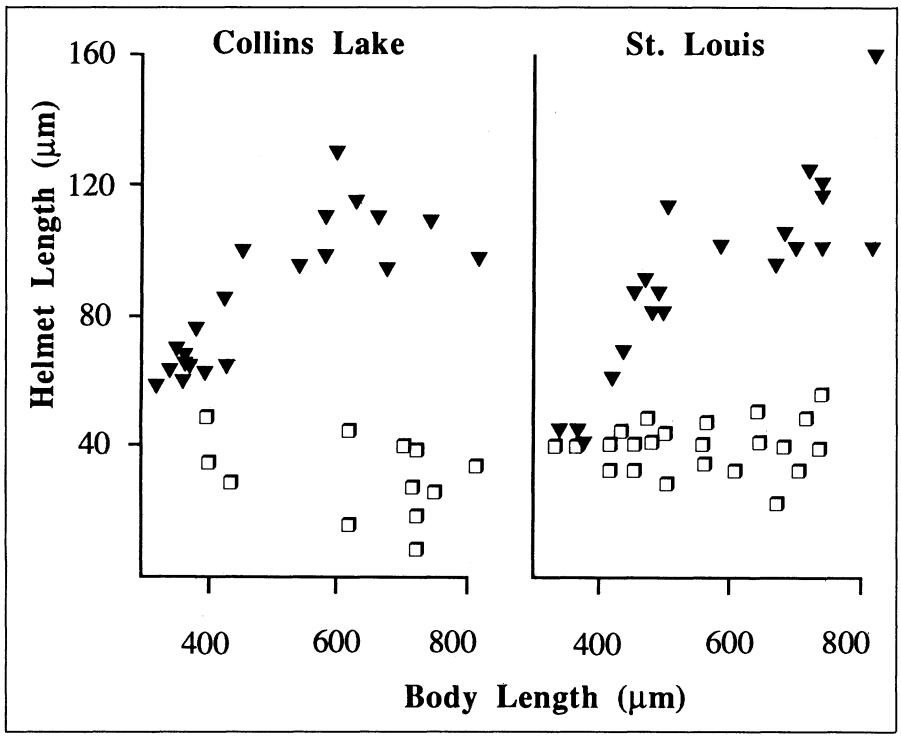


Figure 3. Representative responses from two of six *Daphnia* populations in which helmet formation can be induced by exposure to the *Chaoborus* factor. ▼, Animals exposed to *Chaoborus* factor; □, control animals. From Hebert and Grewe (1985).

DNA, development is constrained by the properties of the materials that make up the organism. All chemical systems, living or dead, are sensitive to temperature, pressure, pH, and substrate concentration. Any organism not buffered against those environmental changes to which chemical reactions are sensitive will inevitably be phenotypically plastic. There is no genetic control of adaptation involved in this component of plasticity, which is as much an encumbrace as an opportunity. Any adaptation in plasticity is layered upon and limited by the organism's chemistry (Stearns 1982).

Gene flow also can maladapt a reaction norm. Neighboring populations of mosquitofish (*Gambusia affinis*) at Clear Lake, Texas, differ significantly in their life-history traits in the field. The fish from the lower part of a freshwater stream produce fewer, larger offspring than those from the brackish estuary into which the stream drains. When the fish from both populations are reared in fresh and brackish water in the laboratory, however, neither population does well in fresh water; mortality rates are higher and growth rates are slower than in brackish water. Mosquitofish from an isolated freshwater population in Hawaii do equally well in both fresh and brackish water. Thus, not all fish grow poorly in laboratory fresh water.

Electrophoretic analysis of the two Texas populations indicates considerable gene flow between the two habitats. Gene flow from the large brackish population is holding the small freshwater population in a state of physiological maladaptation. There is a phenotypic response to the freshwater environment, a shift along a reaction norm, but it is a reaction norm determined by genes that have evolved primarily to deal with the brackish environment. Consequently, the differences in growth rate and mortality seen in fresh water are maladaptive (Stearns and Sage 1980).

Adaptive reaction norms. The criterion for identifying an adaptation is a change in phenotype that occurs in response to a specific environmental signal and that has a clear functional relationship to that signal. The relationship must result in an improvement in growth, survival, or reproduction. Among the adaptive reaction norms that have been identified are responses to changes in diet (prey-induced) and in risk (predator- or parasite-induced).

PREY-INDUCED REACTION NORMS. The changes in jaw morphology of cichlid fishes fed different diets are continuous and can be described as a reversible prey-induced reaction norm. Meyer (1987) divided a two-week-old brood of *Cichlasoma managuense*, assigning fry at random to one of two groups. For eight months one group was fed brine shrimp (*Artemia*) nauplii (a larval stage), and the other group was fed commercial flakes and lab-reared nematodes. Then the *Artemia* group and half the flake group were given adult *Artemia*, which are larger and more evasive than nauplii, and maintained for another eight months. The other half of the flake group remained on the flakes.

Standard length, head length, snout length, eye length, lower jaw length, cheek depth, and snout shape were measured at the ages of 2 weeks, 8.5 months, and 16.5 months. Fish that were eating *Artemia* developed a more pointed jaw; those eating flakes developed a rounder one. The direction of this prey-induced continuous variation in jaw shape was reversible by a change in diet.

In this case, however, fish whose jaw morphology was not adapted to the diet did not grow more slowly than fish that had adapted. When the flake-fed fish were switched to an *Artemia* diet, their growth accelerated, and they actually grew faster in body length than the fish that had been fed *Artemia* all along. More detailed growth experiments with induced and noninduced fish grown on both diets are needed to decide whether this reaction is an adaptation.

Grasshoppers provide another example of adaptation. They must feed efficiently to grow rapidly, and they must grow rapidly to achieve higher fecundities and to mate successfully before the end of the season. Some grasshoppers develop differently shaped mandibles on different diets; the differences between mandibles influenced by soft leaves (such as lettuce) and hard leaves (such as silicate-rich grasses) are especially striking. Growth rate measurements demon-

strate that having the mandibles appropriate to a given diet provides a distinct reproductive advantage (Thompson 1988).

Perhaps the simplest and most completely analyzed case of a prey-induced polyphenism, and one in which the mechanism of the developmental switch is known in detail, describes gut bacteria (*Escherichia coli*) and their prey—water-soluble molecules. These bacteria can replace their membrane proteins that admit small molecules to the cell. The replacement occurs in response to osmotic changes in the environment, and it is mediated by two-step control of gene expression. The first step changes a receptor molecule in the membrane. This change activates a control molecule in the cell that binds directly to the bacterial DNA and thus alters the expression of the genes that code for membrane proteins (Forst and Inouye 1988, Hall and Silhavy 1981). This system could serve as a starting point for the investigation of developmental switches in higher organisms.

Other prey-induced phenotypic reactions are discontinuous. For example, the rotifer *Asplanchna* (Gilbert 1980) switches among three morphs of different shapes and sizes to adapt to shifts in the types of prey available. The tiger salamander shifts from a plankton-feeding to a cannibalistic morph (Collins and Cheek 1983). These developmental switches do not produce reaction norms.

PREDATOR-INDUCED REACTION NORMS. Reaction norms for life-history traits appear in the literature on predator-induced defenses (Harvell 1986, Havel 1987, Lively 1986c). Phenotypic changes in fecundity and mortality may be byproducts of the cost of producing an alternate morph. However, they can also be adaptive in themselves. Harvell says the evolution of inducible defenses should be favored when prey are not killed in initial encounters with predators, attacks cannot be predicted from cues other than the presence of a predator, and the cost of defense is substantial.

In *Daphnia*, longer helmets and growth of spines during summer lower the efficiency of fish (Jacobs 1967) and invertebrate predators feeding on these organisms. In the first experimental demonstrations of these predator-induced responses, midge (*Chaoborus*) larvae and notonectids were the predators. Kreuger and Dodson (1981) showed that a substance associated with midge larvae stimulates the formation of small

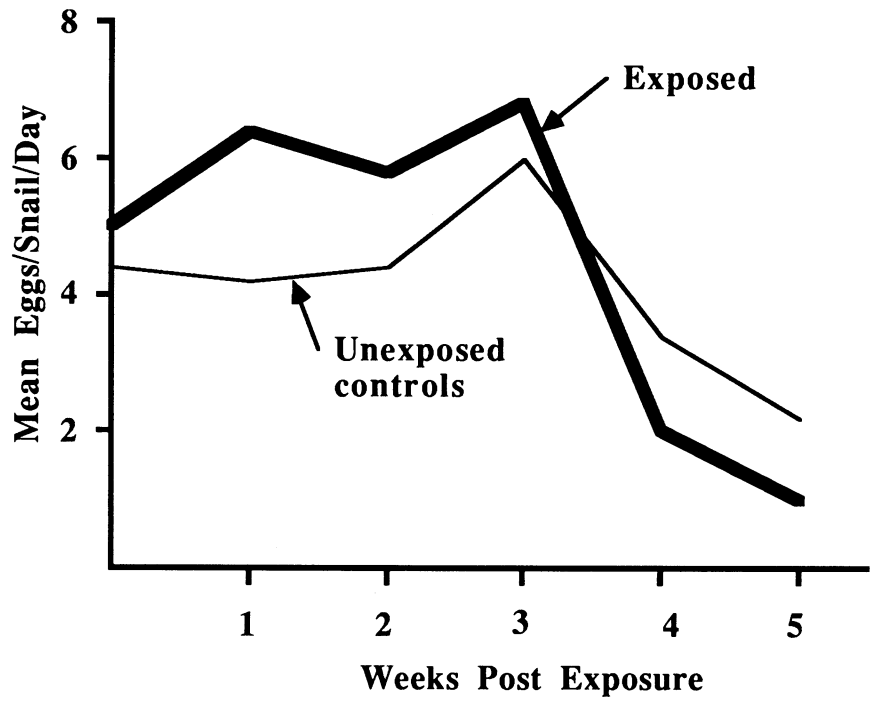


Figure 4. A snail's reaction to exposure to a trematode. From Minchella and Loverde (1981).

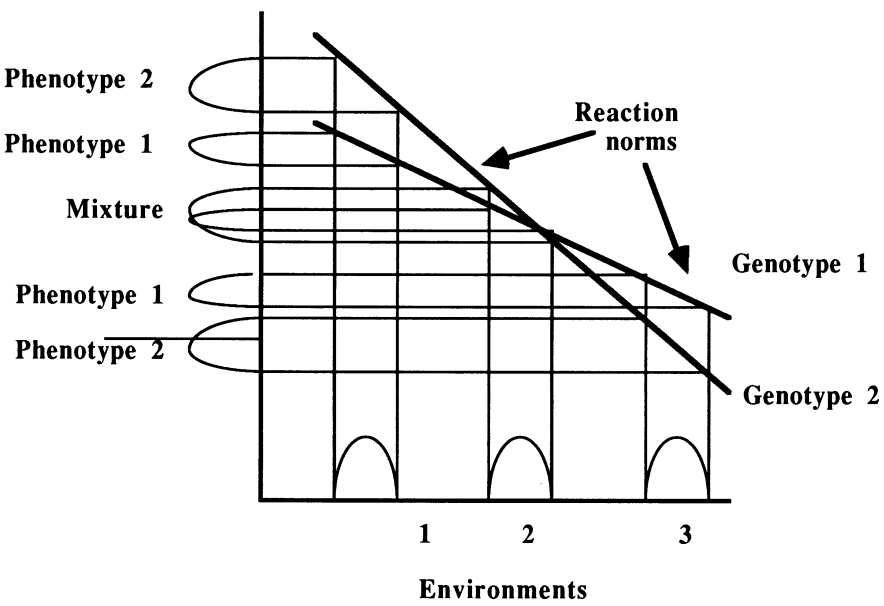


Figure 5. Crossing reaction norms convert three different environmental distributions into three qualitatively different phenotypic distributions. In environments 1 and 3, the genotypes can be distinguished in the phenotypes and heritability is significantly different from zero. In environment 2, near the crossing point of the norms, the genotypes are indistiguishable in the phenotypic mixture and heritability does not differ from zero. Between environments 1 and 3, the phenotypic ranking of the genotypes reverses. From Suzuki et al. (1986).

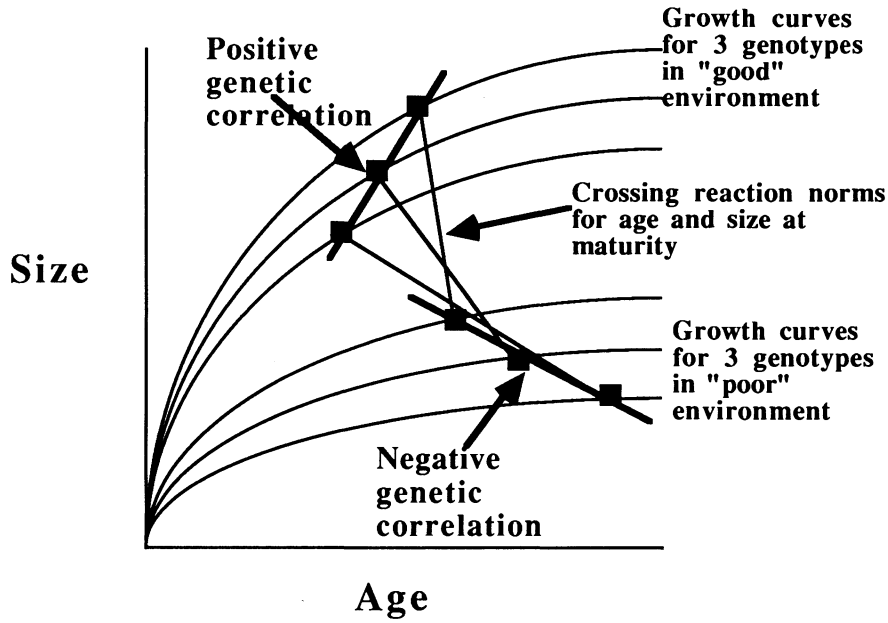


Figure 6. The first mechanism for producing a change in sign of genetic covariance: the reaction norms of the genotypes cross. Hypothetical growth curves for three genotypes are drawn for two environments: rapid and slow growth. The heavy dots represent maturation events.

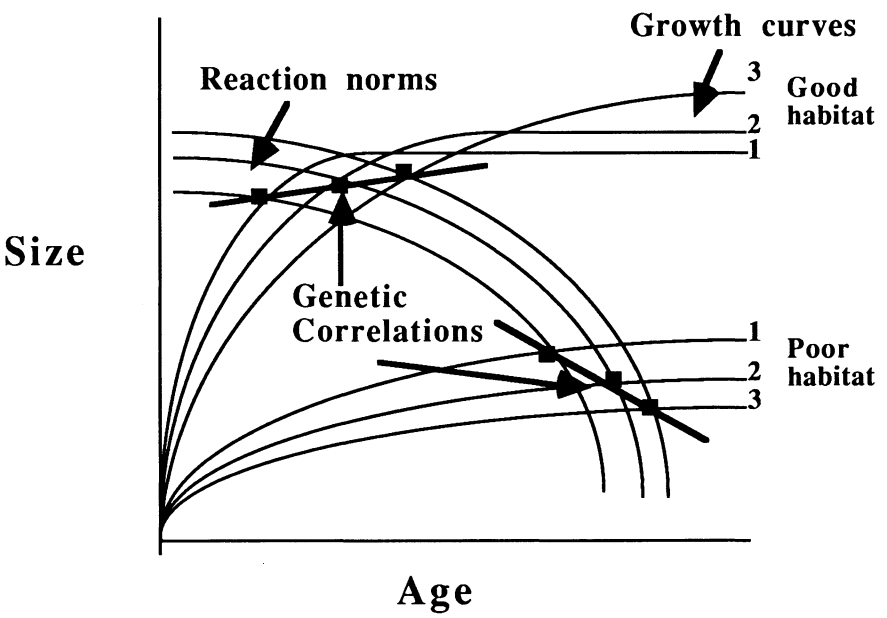


Figure 7. The second mechanism for producing a change in sign of genetic covariance: the growth curves cross (growth rate and asymptotic size have negative genetic correlations). Hypothetical growth curves for three genotypes—fast (1), intermediate (2), and slow (3) growth—are plotted for two environments. The heavy dots represent the maturation events of individuals; the heavy lines represent the genetic correlations in the two environments.

spines on the dorsal surface of the carapace in *Daphnia pulex*, and Grant and Bayly (1981) demonstrated that notonectids induce similar reactions in *Daphnia carinata*. In *Daphnia ambigua*, both helmet shape and the shape and number of spines on the carapace vary seasonally, and temperature is known to influence the helmet shape of neonates. Hebert and Grewe (1985) raised *D. ambigua* clones from six North American sites in water in which *Chaoborus* larvae had or had not been held, and the researchers also found changes in carapace morphology in response to the *Chaoborus* factor (Figure 3).

Predator-induced responses are not limited to planktonic crustacea living in freshwater ponds and lakes. They can also be found in the rocky intertidal zone. Palmer (1985) has shown that thin-shelled snails (*Thais lamellosa*) are more vulnerable to crab (*Cancer productus*) predation than are snails with thick shells. Recent work shows that the presence of crabs induces thicker shells and that the diversion of energy into shell growth correlates negatively with growth and reproduction.[1]

Such responses are likely to be widespread for two reasons. First, crab predation on benthic invertebrates is ancient and ubiquitous in the shallow waters of the earth's oceans (Vermeij 1987). Second, many marine invertebrates disperse widely as larvae and cannot predict whether they will encounter crab-dense or crab-free habitats. It makes sense to leave the choice of a shell morphology, and with it an entire trajectory of growth and reproduction, until after the adult habitat has been reached.

Working in the subtidal zone on bryozoans grazed on by nudibranchs, Harvell (1986) showed that trophically specialized nudibranch predators induce spine formation in bryozoan colonies. The spine formation entails a cost in growth rate, and smaller individuals produce fewer sexual propagules.

PARASITE-INDUCED REACTION NORMS. Parasites usually shorten the expected lifespan of their hosts, and many parasites, particularly trematodes, castrate their hosts. Infection by a parasite is a dependable cue that the host's survival and fecundity schedules will be altered for the worse. An adaptive response of the host would be to mature and start reproduction, if still juvenile, and to increase reproductive effort, if adult, to increase the chances of successful

[1]A. R. Palmer, 1989, unpublished data. Department of Zoology, University of Alberta, Edmonton, Canada.

reproduction before either castration or death (Minchella 1985).

The reaction of the snail *Biomphalaria glabrata* to a trematode parasite is apparently adaptive (Minchella and Loverde 1981; Figure 4): when exposed to parasites, the snail increases reproduction early in life. Infection is not necessary. It suffices to expose the snails to water in which the parasites have been held, making the experimental demonstration of the reaction especially elegant (Bell and Koufopanou 1986).

Significance for quantitative genetics

Reaction norms can be considered to be mechanisms that receive a distribution of environmental variation and transform it into a distribution of phenotypic variation (Suzuki et al. 1986). The shape of the distribution is compressed by a steep reaction norm and flattened by a more gradual reaction norm, and it can also be skewed. If only a single genotype, say a clone of *Daphnia*, is involved, the transformation is straightforward and can change the shape of the phenotypic distribution considerably (Figure 1).

Crossing of reaction norms. When two or more genotypes are present in nature—and many will always be present in a sexually reproducing species—the reaction norms of different genotypes are usually expected to have different slopes and shapes, therefore crossing on a graph (Figure 5). For traits closely associated with fitness, if the reaction norms did not cross, the phenotypic value of one reaction norm would be better in all the environmental conditions, so it would be expected to be fixed by selection.

The crossing of reaction norms is described in quantitative genetics as genotype × environment (G × E) interactions. These G × E interactions are expected to be pervasive. Almost every study that has looked for them has found them (Bell 1987, Gebhardt and Stearns 1988).[2]

Reaction norms that cross have two important effects on phenotypic distributions (Figure 6). First, they determine whether or not one can see heritable variation in the phenotype. In the region where the reaction norms cross, one cannot distinguish the genotypes from the phenotypes. Outside that region, the genotypic variation becomes clear. This observation is the basis for a fundamental tenet of quantitative genetics (Falconer 1981): heritabilities vary with the environments in which they are measured.

Second, reaction norms determine the ranking of the phenotypes. To the right of the crossing region, the genotypes have a phenotypic ranking opposite to the one they have to the left (Figure 5). For example, selection for higher phenotypic values would favor one genotype to the left of the crossing point and the other one to the right.

Genetic covariation. Reaction norms also influence the expression of genetic covariance. Traits show positive covariance when selection for an increase in one trait causes an increase in the other. They show negative covariance when selection that increases one produces decreases in the other. Negative genetic covariances are the basis of trade-offs among life-history traits. The underlying mechanisms of genetic correlation are that the same gene or genes influence both traits (pleiotropy) or that genes coding for the two traits are closely linked on a chromosome. Expression of covariance limits the rate and direction of microevolution.

Genetic correlation depends both on the population and on the environment (Falconer 1981). Gene frequencies differ among populations; expression of the different genotypes changes with the environment. Correlation between traits can change from negative to positive when examined in different environments.

A developmental mechanism can modulate the expression of genetic covariance. Correlation may change from negative to positive (or the reverse direction) in many traits involved in allometric relationships and in most life-history traits. In these cases, the correlation is positive in some environments and negative in others. In intermediate environments, selection for one trait will not correlate with any response in the other.

So far in this article, I have plotted reaction norms for single traits as functions of environmental variables. To see how reaction norms can modulate genetic correlation, it is convenient to plot two traits against each other—one on the abscissa and the other on the ordinate (Figure 6). Environmental conditions then can vary along the reaction norms, rather than along one axis.

Consider (in a population living in an environment heterogeneous for growth conditions) the organisms' age and size at maturity, two life-history traits strongly associated with growth patterns. The organisms should evolve a reaction norm for these characteristics of maturity. The commonest type of reaction norm describes organisms maturing early at a large size (when growth is rapid) and late at a small size (when growth is slow) (Stearns and Koella 1986).

I have suggested two relationships among reaction norms for age and size at maturity for different genotypes that can produce a change of sign of genetic correlation (Stearns in press). First, the largest and latest-maturing genotype under good growth conditions is the largest and earliest-maturing genotype under poor growth conditions. The other genotypes are simularly ranked (i.e., the second-largest and second-latest maturing genotype under good growth conditions is the second-earliest maturing under poor growth conditions, and so forth for the other genotypes). The reaction norms may or may not cross.

Second, growth rates have negative genetic correlations with maximal size, so that the fastest-growing genotypes produce the smallest adults. In this case, the extrapolated growth curves of the different genotypes would eventually intersect. Figure 7 plots growth curves and reaction norms for maturation in three genotypes. The growth curves cross, but the reaction norms do not. The black rectangles indicate maturation events for each genotype under each growth condition. The genetic correlation between age and size at maturity changes from positive under good growth conditions (at left) to negative under poor growth conditions (at right).

[2]R. G. Baker, 1989, personal communication. Crop Development Centre, University of Saskatchewan, Saskatoon, Canada.

Therefore, genetic correlation may change from positive to negative either when the growth curves of the genotypes cross, or when their reaction norms have different slopes and ranges. When growth conditions vary, genetic covariation can be expected to vary from positive to negative as well.

If natural selection molds the evolution of growth such that the growth curves or reaction norms of different genotypes normally cross, then the conditions under which the expression of genetic covariation changes from positive to negative would be quite general. That view could be extended to claim that the developmental mechanisms that modulate the expression of genetic variation and covariation themselves have evolved. The conclusion would be that organisms contain mechanisms that can make decisions to release or conceal genetic variation based on information about the environment and, if the decision is to release it, then the particular pattern of covariation among traits might also be controlled.

An alternative, nonadaptationist view is that these effects are byproducts of developmental systems. These characteristics would then exist for reasons having nothing to do with genetic variation or microevolutionary adaptation. Further work is required to distinguish between these alternatives.

If the genetic correlation between two traits changes regularly in space and time from positive to negative, then additional genetic variation in both traits will be maintained. This hypothesis for the maintenance of genetic variability relies heavily on mechanisms internal to the organism. No fluctuating selection pressures of symmetrical distribution are required, as in some other hypotheses. This hypothesis requires only a heterogeneity of growth conditions large enough to change frequently, but not necessarily symmetrically, the expression of genetic covariance from positive to negative.

Discrete phenotypic variation. In contrast to reaction norms, phenotypic variants, produced when developmental switches are triggered by environmental cues, express the capacity of a single genotype to encode two or more discrete phenotypes. Within each phenotype, the morphology, life history, and physiology are integrated to function in a specific ecological or social role. Examples include:

- Environmental sex determination, in which each genotype contains the potential to become either a male or a female (Bull 1983, 1987);
- The life cycles of aphids, rotifers and cladocerans, in which individuals switch between sexual and asexual reproduction;
- The polyphenisms found in some female rotifers, in which the switch is among three morphological types that feed with different efficiencies on different prey (Gilbert 1980);
- The antipredator shell polyphenisms of some barnacles (see photo page 437) (Lively 1986a,b);
- The diapause mechanisms of annual fish, in which several switches between diapausing and nondiapausing stages may follow in a succession that determines a broad distribution of hatching or germination times within a single clutch (Wourms 1972);
- The seasonal polyphenisms of butterflies, in which the switch is often between a spring and a fall morph or between a wet- and a dry-season morph, one of which is more active and brightly colored and the other of which is more crytic (Shapiro 1976);
- The phase polyphenism of locusts, in which the switch is between an isolated, sedentary phase and a gregarious migratory phase (Uvarov 1966);
- The fighting castes of some polyembroyonic wasp larvae (Cruz 1986);
- The castes of social insects, in which the switch may be between queen, various types of workers, and various types of soldiers (Brian 1965, Wilson 1971).

The significance of discrete phenotypic variation for evolutionary biology is at least fourfold. First, discrete phenotypic variation indicates that within each population, and within each sex, there may be a further subdivision of phenotypic types—each of which has its own life history and morphology. To lump them together would produce a meaningless average and obscure most of the interesting biological differences.

Second, these systems provide models in which the evolutionary significance of G × E interactions can be seen especially clearly. Third, within each discrete phenotypic variant, one can encounter a different reaction norm. Thus the discrete variation imposes a level of control that stands between the genotype and the phenotype and it structures the set of reaction norms that the phenotype may exhibit. Fourth, these cases make clear the way that phenotypic plasticity uncouples the phenotype from the genotype and reduces the pressure for further genetic change (Wright 1931).

Conclusions

Reaction norms describe continuous phenotypic plasticity; they are a mapping of the genotype onto the phenotype as a function of the environment. Reaction norms can be reversible or irreversible; many life-history traits have irreversible reaction norms. Every reaction norm is an inescapable physicochemical response modified to a greater or lesser extent by genetic changes; thus all reaction norms are mixtures of adaptations and constraints. They can be maladaptive, particularly in marginal populations living in heterogeneous environments. Reaction norms can be part of the organism's defense against predators and parasites and of its adaptation to diets of different prey species.

Reaction norms modulate the expression of genetic variation. For a single trait, reaction norms with different slopes can transform a symmetrical distribution of environmental values into a broader, narrower, or skewed distribution of phenotypic values. Crossing reaction norms indicate a particularly strong form of G × E interaction.

For a single trait, the crossing of reaction norms for different genotypes has two implications. First, near the crossing points it is not possible to distinguish the genotypic classes by their phenotypes, and measurements of heritabilities are near zero. Far from the crossing points the genotypes can be distinguished and heritabilities can be significant. Second, the

rank order of phenotypic values of genotypes reverses from one side of the crossing point to the other.

For two or more traits, crossing reaction norms imply that in some environments genetic correlations will be positive, but in others they will be negative. The same effect can be produced by crossing growth curves for any traits correlated with size, without the reaction norms crossing for the traits. Crossing reaction norms also generate the type of variation critical for the maintenance of sexual reproduction, according to one hypothesis (Bell 1987).

Developmental mechanisms generate much of the variation seen within populations. Genetics and demography are not sufficient to explain evolution; they must be combined with descriptions of phenotypic plasticity—reaction norms and developmental switches—before the sources of variation can be understood.

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