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Development and evolution of adaptive polyphenisms

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SUMMARY Phenotypic plasticity is the primitive character state for most if not all traits. Insofar as developmental and physiological processes obey the laws of chemistry and physics, they will be sensitive to such environmental variables as temperature, nutrient supply, ionic environment, and the availability of various macro- and micronutrients. Depending on the effect this phenotypic plasticity has on fitness, evolution may proceed to select either for mechanisms that buffer or canalize the phenotype against relevant environmental variation or for a modified plastic response in which some ranges of the phenotypic variation are adaptive to particular environments. Phenotypic plasticity can be continuous, in which case it is called a reaction norm, or discontinuous, in which case it is called a polyphenism. Although the morphological discontinuity of some polyphenisms is produced by discrete developmental switches, most polyphenisms are due to discontinuities in the environment that induce only portions of what is in reality a continuous reaction norm. In insect polyphenisms, the environmental variable that induces the alternative phenotype is a token stimulus that serves as a predictor of, but is not itself, the environment to which the polyphenism is an adaptation. In all cases studied so far, the environmental stimulus alters the endocrine mechanism of metamorphosis by altering either the pattern of hormone secretion or the pattern of hormone sensitivity in different tissues. Such changes in the patterns of endocrine interactions result in the execution of alternative developmental pathways. The spatial and temporal compartmentalization of endocrine interactions has produced a developmental mechanism that enables substantial localized changes in morphology that remain well integrated into the structure and function of the organism.

INTRODUCTION

In most organisms a genotype can produce many different phenotypes. The exact phenotype that is expressed depends on the environment in which the organism develops. Phenotypic plasticity can be gradual or discrete. Phenotypes that change with small gradual changes in an environmental variable are called reaction norms. In addition to these continuously variable phenotypes, some organisms can develop two or more discrete alternative phenotypes, without intermediate forms. This phenomenon is called polyphenism and can come about in two ways: either when different members of a species experience discretely different environments (as in the case of a bivoltine insect with discrete generations in different seasons) or when a continuously variable environment induces a discrete threshold-like switch from one developmental pathway to another.

The mechanisms that mediate these two types of phenotypic plasticity are beginning to be understood. As we will see below, the development of alternative phenotypes in reaction norms and polyphenisms can be caused by specially evolved mechanisms that are regulated by variation in the patterns of hormone secretion. Reaction norms can also result from the fact that the rates and timing of developmental processes are affected by such environmental variables as temperature, nutrition, photoperiod, and so on. These environmental variables affect the underlying chemical and metabolic processes of development directly, without the intervention of a specially evolved mechanism.

ORIGIN AND EVOLUTION OF PHENOTYPIC PLASTICITY

All phenotypes are believed to be primitively plastic. This is because developmental, physiological, and metabolic processes are normally sensitive to environmental variables such as temperature, pH, ionic strength, and nutrients. Such phenotypic sensitivity to environmental variables leads to what is called a reaction norm: the range of phenotypes produced by a given genotype when exposed to a range of values of a single environmental variable. Given that phenotypic plasticity is obtained gratis, as a by-product of the physics and chemistry of development, evolution of this plasticity can occur in two directions: One results in stabilization of the phenotype, effectively eliminating the plasticity, whereas the other results in the exploitation of the plasticity. If this phenotypic sensitivity to environmental variation reduces fitness, then it will be eliminated by the evolution of mechanisms that somehow reduce the sensitivity of the phenotype

Table 1. Examples of differences between the selective environment to which a polyphenism is an adaptation, and the inductive environment that actually triggers the polyphenic developmental switch

Polyphenism	Selective agent (to which the polyphenism is an adaptation)	Inducing stimulus
Seasonal	Lethal temperature, food scarcity	Photoperiod, nonlethal temperature
Phase (aphids)	Food quantity/quality	Crowding, temperature, photoperiod
Phase (locusts)	Food quantity/quality	Crowding
Phase (caterpillars)	Predation	Food quality
Wing length	Food quantity/quality	Crowding, photoperiod
Horn length	Mating success	Food quantity/quality
Caste (ants, soldiers)	Food quantity/quality, predators	Food quality, pheromones
Caste (ants, gynes)	Reproduction	Pheromones, overwintering
Caste (bees)	Reproduction	Nutrition, pheromones
Diapause	Lethal temperature	Photoperiod, nonlethal temperature

to a particular environmental variable. A broad diversity of homeostatic mechanisms have evolved that buffer physiological and metabolic systems in the face of environmental variation. Homeostatic mechanisms serve to maintain a constant internal environment by means of feedback mechanisms that return the system to a set point. Homeostatic mechanisms in physiology and metabolism have been studied for a long time and are now well understood. In development there has also been an evolution of mechanisms that buffer the emergent phenotype against environmental and genetic variation. Depending on one's perspective, the evolved insensitivity to variation is called canalization (the ability to return to a developmental trajectory after a disturbance) or robustness (the simple ability to tolerate or be insensitive to environmental or genetic variation) (Nijhout and Davidowitz 2002). Unlike physiological homeostasis, the mechanisms that confer robustness in development are not well understood at present.

Evolution has not invariably proceeded to stabilize the phenotype in the face of environmental variation. Instead, in many cases phenotypic plasticity has been exploited as a mechanism that enables an organism to develop different phenotypes that may be adapted to two or more environments, without requiring the evolution of a genetic polymorphism. Thus, rather than having a single phenotype that is well adapted to a single niche or function, organisms can develop alternative phenotypes, each of which may be optimized for fitness in a different environment. Phenotypic plasticity can therefore be divided into two kinds: type 1, which comes gratis in the absence of a homeostatic mechanism that buffers the phenotype against environmental variation during development but that is unlikely to be adaptive, and type 2, which is an adaptation to a particular set of environments.

Both type 1 and type 2 plasticity can be manifested as a reaction norm, but their underlying developmental physiol-

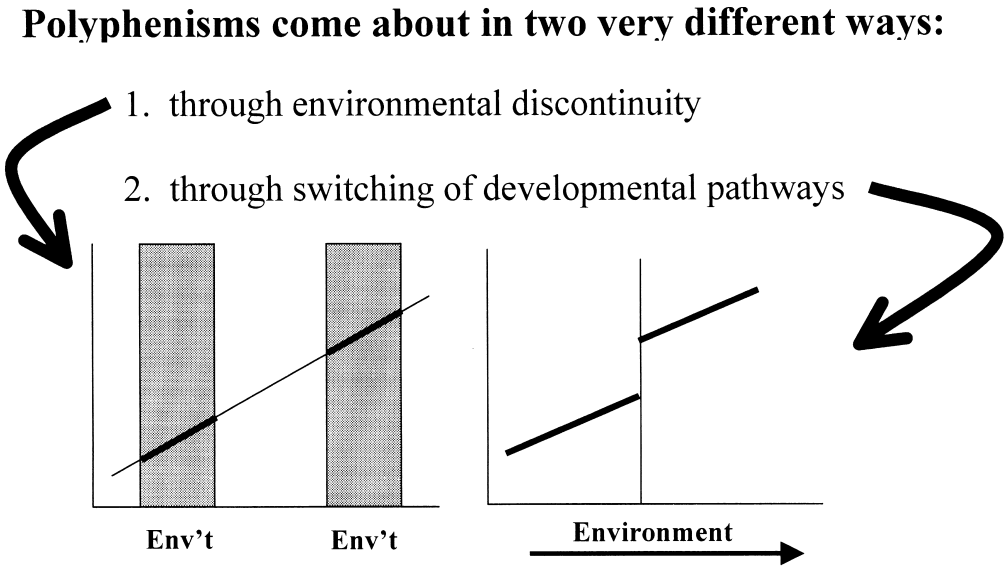

Fig. 1. Polyphenisms can come about in two different ways: (1) from a reaction norm when the environment is either discontinuous or is only sampled at discrete times or places so that the environment is effectively discontinuous; or (2) from switches in developmental pathways that produce a discontinuous reaction norm.

ogy and evolutionary history are likely to be very different. It is unlikely that type 1 plasticity can produce an array of phenotypes, each of which just happens, by chance, to have the highest fitness in the particular range of environments that induce them. The evolution of adaptive phenotypic plasticity must involve changes in development that alter the type 1 reaction norm. Reaction norm evolution and the evolution of adaptive phenotypic plasticity have been the subject of numerous theoretical and empirical studies (Via and Lande 1985; Lively 1986; West-Eberhard 1989; Gomulkiewicz and Kirkpatrick 1992; Via et al. 1995; Schlichting and Pigliucci 1998), and a great deal is known about the conditions under which the shape of the reaction norm can evolve under selection in different environments. By contrast, relatively little is known about the developmental mechanisms that produce a particular reaction norm nor about the way those developmental mechanisms change when reaction norms evolve.

Our understanding of the developmental mechanisms that underlie phenotypic plasticity comes almost entirely from studies on the development of polyphenisms. Polyphenisms are discrete alternative phenotypes that develop in response to environmental variation. Among the best known polyphenisms are the castes of social insects, the alternative seasonal forms of insects, heterophylly in plants, predator-induced polyphenisms in cladocerans, male mating polyphenisms in beetles, phase polyphenisms of migratory locusts, and the long- and short-winged dispersal polyphenisms of many insects (Cook 1968; Wheeler 1986; Harvell 1990; Nijhout 1994; Dingle and Winchell 1997; Schlichting and Pigliucci 1998).

Polyphenisms are adaptations to reliable and predictable variations in the environment. From an evolutionary perspective, perhaps the most interesting thing about a polyphenism is that *the inducing environment is not the same as the selective*

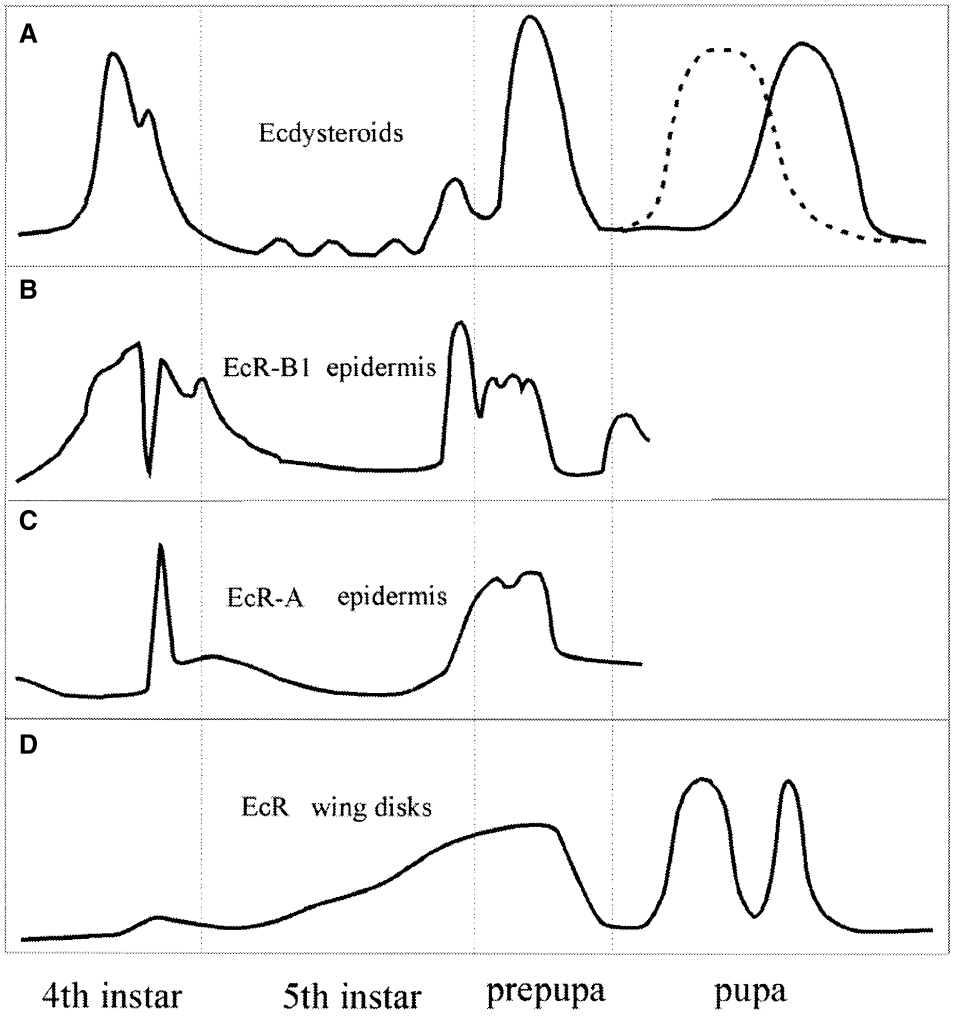

Fig. 2. (A–D) Temporal pattern of variation of ecdysteroid titers in the hemolymph and of ecdysone receptor (EcR) isoform expression in different tissues of larvae of the tobacco hornworm, *Manduca sexta* (Nijhout 1999).

environment. This is to say, the environment to which the alternative phenotype is an adaptation is not the same as the environment that induces the development of that phenotype (Table 1). For instance, a seasonal polyphenisms may be an adaptation to cold, or nutrient stress, but is typically induced by a change in photoperiod. A change in photoperiod (the relative lengths of day and night) does not in itself constitute an unfavorable environment, but it is an excellent predictor of a seasonal change and is thus a good predictor of future temperature or nutrient stress. Because the inducing environment is a token, or predictor, there is usually a considerable time lag between the inducing environment and the selective environment. The time interval between the sensitive period for induction and the actual development of the alternative phenotype was initially probably a simple consequence of the fact that a finite period of time is needed to develop a particular phenotype. In modern polyphenisms the time delay is likely to be part of the adaptation that ensures that a predictive environmental stimulus is sampled for a long enough period and at a time when its predictive power is strong.

Many insects have evolved a well-defined critical period in development when the individual is sensitive to inducing stimuli, and this critical period occurs long before the alternative phenotype actually develops. Evolution of the use of a token stimulus requires a correlation between the token and the selective environment and that the organism is somehow able to use the token to accurately predict the future environment and match its phenotype accordingly. The general conditions required for the evolutionary maintenance of a polyphenism, once it is established, have been studied by Moran

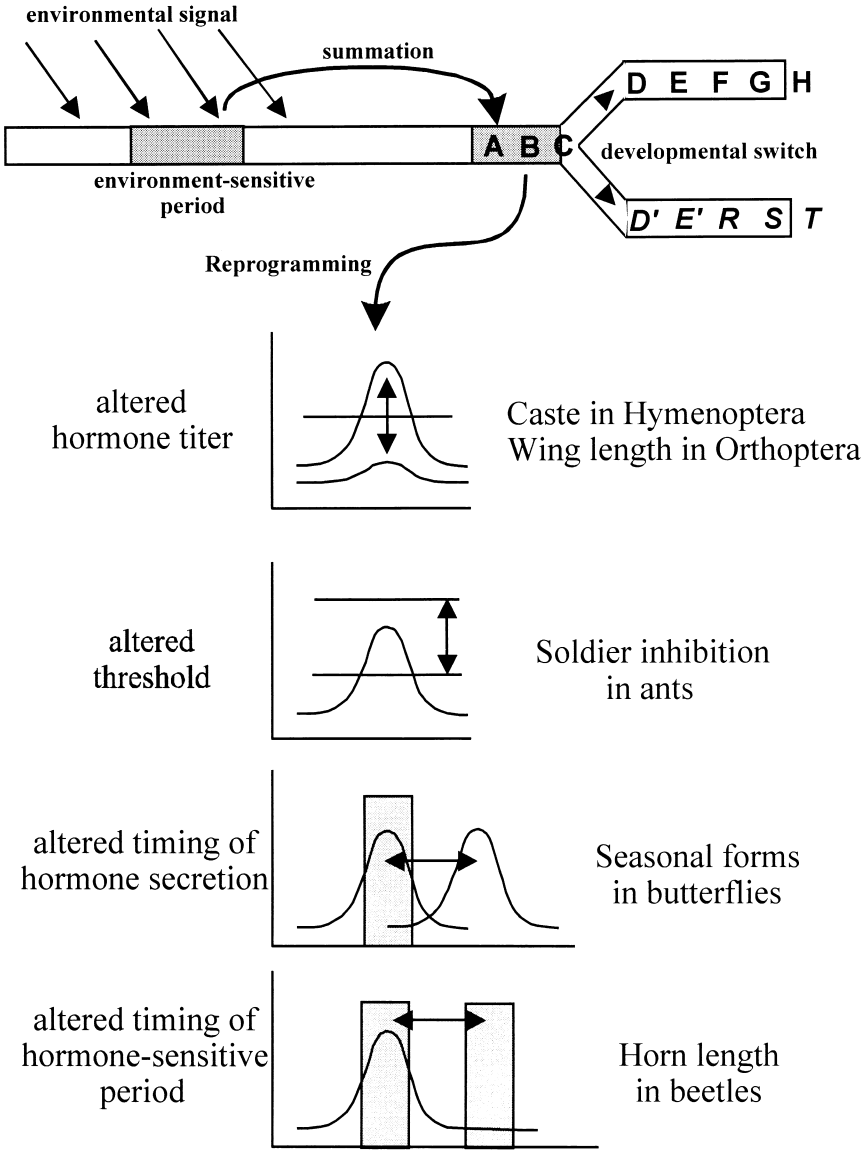

Fig. 3. Endocrine mechanisms underlying the polyphenic switch in insects. The upper diagram depicts the developmental period. At some time during larval life there is a sensitive period during which specific environmental stimuli such as temperature, photoperiod, or pheromones (Table 1) are integrated. These result in a reprogramming of an endocrine mechanism just before or during metamorphosis and leads to the initiation of one of two alternative developmental pathways that result in different adult phenotypes at metamorphosis. Four kinds of hormonally controlled developmental switching mechanisms have been identified to date, illustrated in the bottom four panels, with examples of the polyphenisms in which they have been documented. In all cases, hormones act during tissue-specific sensitive periods. Alternative developmental pathways ensue depending on whether the hormone is above or below a threshold value during such a period (Nijhout 1999).

(1992). Little or no research has been done on the evolutionary origins of polyphenisms.

It is likely that polyphenisms originate from continuously plastic phenotypes. Most modern polyphenisms are, in fact, reaction norms, although this is not always easy to detect in nature. In many and perhaps most polyphenisms, the discrete alternative phenotypes develop either because the environment is discontinuous or because the environment-sensing physiology has a threshold (Fig. 1). A discontinuous environment is one that would be experienced by a bivoltine insect (an insect that has two generations per year). In such animals, each generation develops in a different season and thus experiences a different combination of photoperiod, temperature, nutrition, and population density. In many cases when such polyphenic insects are exposed to intermediate environmental conditions, they develop a range of intermediate phenotypes not normally seen in nature (Nijhout 1994).

Examples of threshold environmental sensitivity can be found in the seasonal polyphenisms of multivoltine insects that have a critical photoperiod for the induction of the polyphenism (Nijhout 1994). Such insects develop alternative phenotypes depending on whether the photoperiod they experience is longer or shorter than the critical day length. Even with such thresholds it is possible to obtain a range of intermediate phenotypes (see Fig. 4), either by manipulating the environment or by manipulating the underlying developmental physiological mechanisms that produce the threshold (Windig 1994). It is therefore worth examining the mechanism by which development can be made sensitive to an environmental gradient so that a threshold phenotypic response results.

DEVELOPMENTAL SWITCHES

In all cases where the developmental mechanism of polyphenic regulation has been elucidated, the developmental switch that leads to alternative phenotypes is regulated by hormones. Virtually all of our knowledge of these mechanisms comes from the studies on the regulation of postembryonic development in insects. Because the external characteristics of insects are part of their (nonliving) cuticle, the expression of polyphenism typically requires a molt, and the alternative phenotype is expressed in the new cuticle that is synthesized at that time. Thus, depending on the prior environment, a polyphenic insect can molt into one of several alternative forms. Insects express both larval and adult polyphenisms. The latter are associated with the metamorphic molt, and such insects can facultatively metamorphose into alternative adult forms. In adult polyphenisms, the environment-sensitive period occurs sometime during larval life. The exact timing

Fig. 4. Phenotypic plasticity in the seasonally polyphenic butterfly *Araschnia levana.* In nature, different generations of this species develop in discretely different environments, and this results in a purely diphenic polyphenism represented by the two extreme forms outlined by boxes. The intermediate forms can be produced by timed ecdysone injections or by intermediate environments and illustrate that a reaction norm lies at the base of this polyphenism.

and duration of environment sensitivity can be early, middle or late in larval life, depending on the species.

The developmental switch mechanism operates sometime during the instar that precedes the molt to the polyphenic form and has the following general structure. During the relevant larval instar there are one or more relatively brief periods during which hormones can alter the course of subsequent development. These are called critical periods or hormone-sensitive periods, and they act essentially as binary switches, with alternative developmental pathways being selected depending on whether a hormone is above or below a threshold value. Interestingly, the principal hormones that control polyphenic development are the same ones that control molting and metamorphosis, namely, ecdysone and juvenile hormone. In addition several neuroendocrine hormones have evolved specialized associations with polyphenic regulation in several species of insects (Nijhout 1994).

The way in which developmental hormones regulate alternative patterns of gene expression is best understood for the hormone ecdysone. The ecdysone receptor is a heterodimer (Talbot et al. 1993) that acts as a transcription factor, and different combinations of the alternative subunits of the dimer regulate different patterns of gene expression (Truman et al. 1994). Different isoforms of the ecdysone receptor are expressed in tissue-specific temporal patterns (Fig. 2), and the tissue-specific periods of sensitivity to a particular effect of the hormone is determined by when and where these receptors are expressed.

When a peak of hormone secretion coincides with receptor expression in only one tissue, then only that tissue will respond to the hormone, and the remaining tissues will continue in their current state, as if no hormone had been present at all. Receptor expression, and therefore the hormone-sensitive periods, are regulated independently from fluctuations in hormone secretion. All known cases of polyphenic switching of developmental pathways occur by environmentally induced changes in either the timing of hormone secretion, the timing of a hormone sensitive period, or the threshold of hormone sensitivity (Nijhout 1999). The array of possibilities, together with examples of each type of regulation, is illustrated in Figure 3. Different patterns of gene expression result, depending on whether or not a peak of hormone secretion coincides with a period of hormone sensitivity, and this leads to the development of alternative phenotypes.

The origin of polyphenisms can be understood in developmental terms as being due to the origin or loss of a coincidence between a peak of hormone secretion and a hormone-sensitive period. Variation in the timing of hormone secretion or receptor expression (or of the threshold of hormone sensitivity) could produce an occasional partial or full mismatch, which then results in new phenotypes. Genetic stabilization of the mismatch in response to some environmental signals (but not others) could then fix the polyphenism. Evolutionary adaptation of the alternative morphs of a polyphenism is most likely facilitated by the fact that hormone-sensitive periods are time and tissue specific, so that developmental regulation is effectively compartmentalized in both time and space. Hence, different parts of an organism could vary and respond to selection independently. The genetic correlation structure of polyphenic phenotypes, however, has yet to be studied in detail.

REACTION NORMS

Perhaps the most interesting thing about having a hormonal regulation of development is that development comes under the control of the central nervous system. This is because the developmental hormones are directly regulated by neurosecretory factors or are themselves neurosecretory hormones. The central nervous system can integrate information about

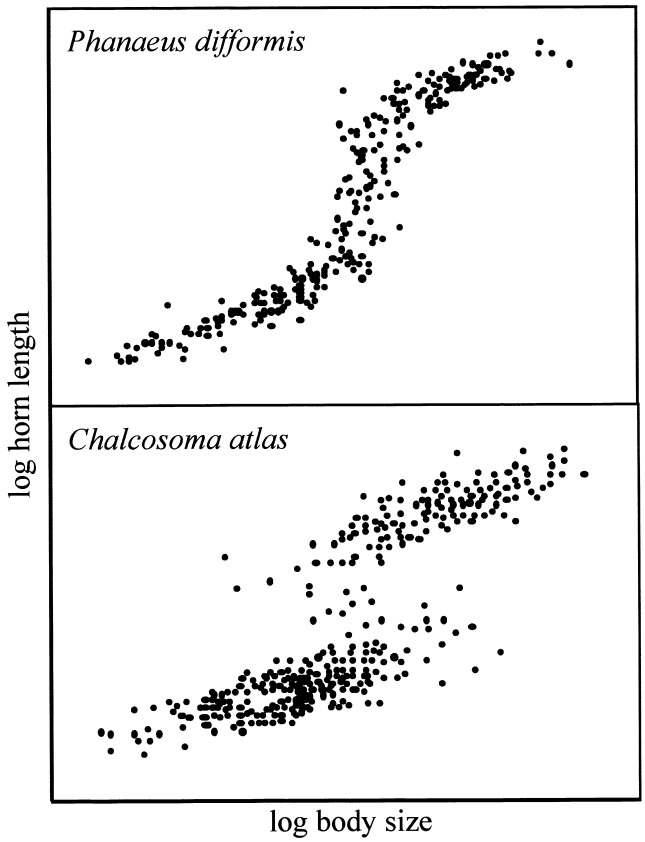

Fig. 5. Horn size allometries in two rhinoceros beetles. The allometry of Phanaeus is a continuous sigmoid curve and suggests an underlying continuous size-dependent generative mechanism. The allometry of *Chalcosoma* appears discontinuous. Discontinuous allometries come about through reprogramming of development and are not reducible to a smooth reaction norm (Emlen and Nijhout 2000).

the animal's internal and external environment and use this information to regulate the secretion of hormones. In this way, development can become responsive to a wide diversity of environmental signals, without the need to have developmental processes themselves be sensitive to the environment.

All known polyphenisms have this kind of indirect environmental sensitivity, and this is also the case for many phenotypes that exhibit continuous phenotypic plasticity, or reaction norms. As noted above, many polyphenisms are nothing more than reaction norms that are sparsely sampled. It would be interesting to find out whether there are any cases of adaptive phenotypic plasticity in animals in which development of the relevant trait is directly sensitive to the environmental variable or whether all cases are mediated by evolved integrated systemic processes, as in the case of polyphenisms.

The discrete alternative morphs of a polyphenisms arise if there is an unambiguous hit or miss of hormone secretion and hormone-sensitive period in different individuals. When there is partial overlap between a hormone pulse and a sensitive period, it is possible to get an intermediate phenotype. Such intermediates can be artificially produced by manipulating the timing of a hormone pulse, as illustrated in Figure 4. In this way it is possible to generate a range of intermediate phenotypes from what in nature is an invariant diphenism. In almost all polyphenisms it is possible to obtain a smoothly continuous range of intermediates either by environmental or physiological manipulation. This continuity suggests that the developmental mechanisms that give rise to the alternative forms of a polyphenism differ only in quantitative and not in qualitative ways.

In a few known cases, there is a discontinuity in the transition between one polyphenic morph and another (Fig. 5). In such cases the alternative morphs appear to have different allometric relationships, and it is unlikely that this can be achieved without a qualitative switch-like change in the developmental processes that give rise to the phenotypes (Nijhout and Wheeler 1982 1996).

A CASE STUDY: *ONTHOPHAGUS TAURUS*

Here I present a case study of the development and evolution of a polyphenism that illustrates many of the features outlined above. Males of the dung beetle *Onthophagus taurus* have a horn length polyphenism. Small males are essentially hornless, whereas large males have well-developed cephalic horns (Fig. 6). Horn length varies allometrically with body size, but the allometry is highly nonlinear. This results in a bimodal distribution of horn sizes, even though body size is normally distributed (Fig. 7). Each of the two horn morphs is an adaptation to differences in body size. Males defend tunnels dug by females and use their horns to combat other males for access to females (Emlen 1997a). Males with large body and horn sizes inevitably win contests when matched against males with smaller body and horn sizes (Moczek and Emlen 2000). One might believe that this should result in an

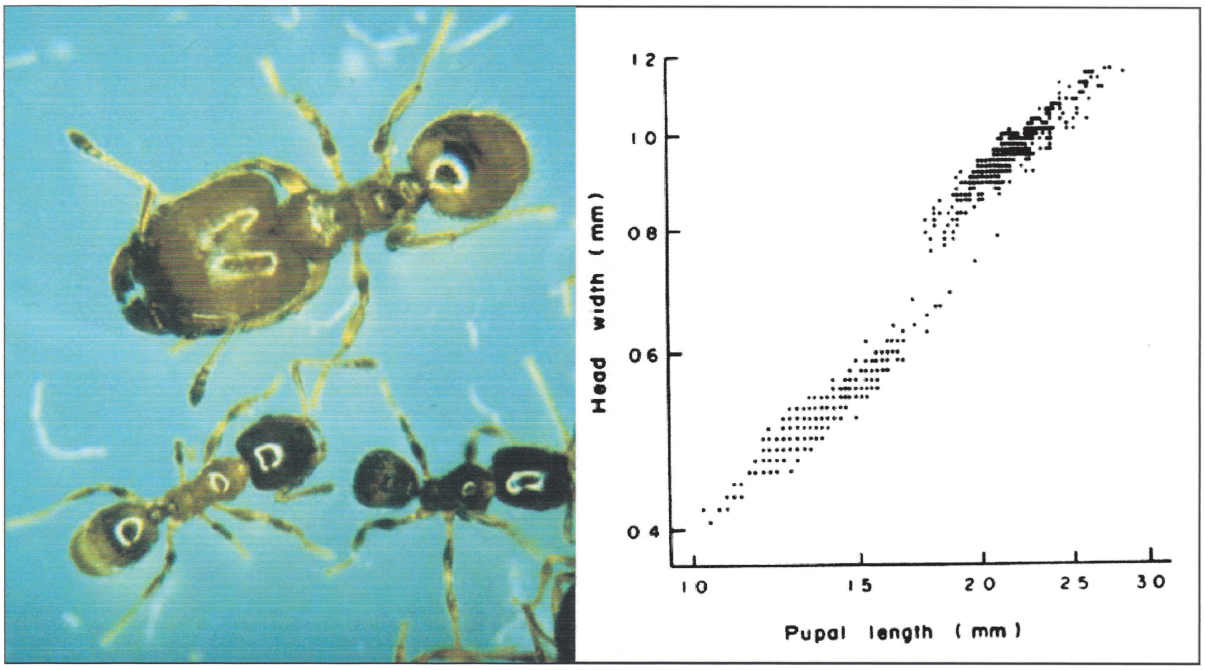

Fig. 6. Head width allometry in *Pheidole bicarinata.* Allometries of soldiers and workers are not continuous, so that it is possible to have two distinct morphologies in animals of the same body size (Wheeler and Nijhout 1983).

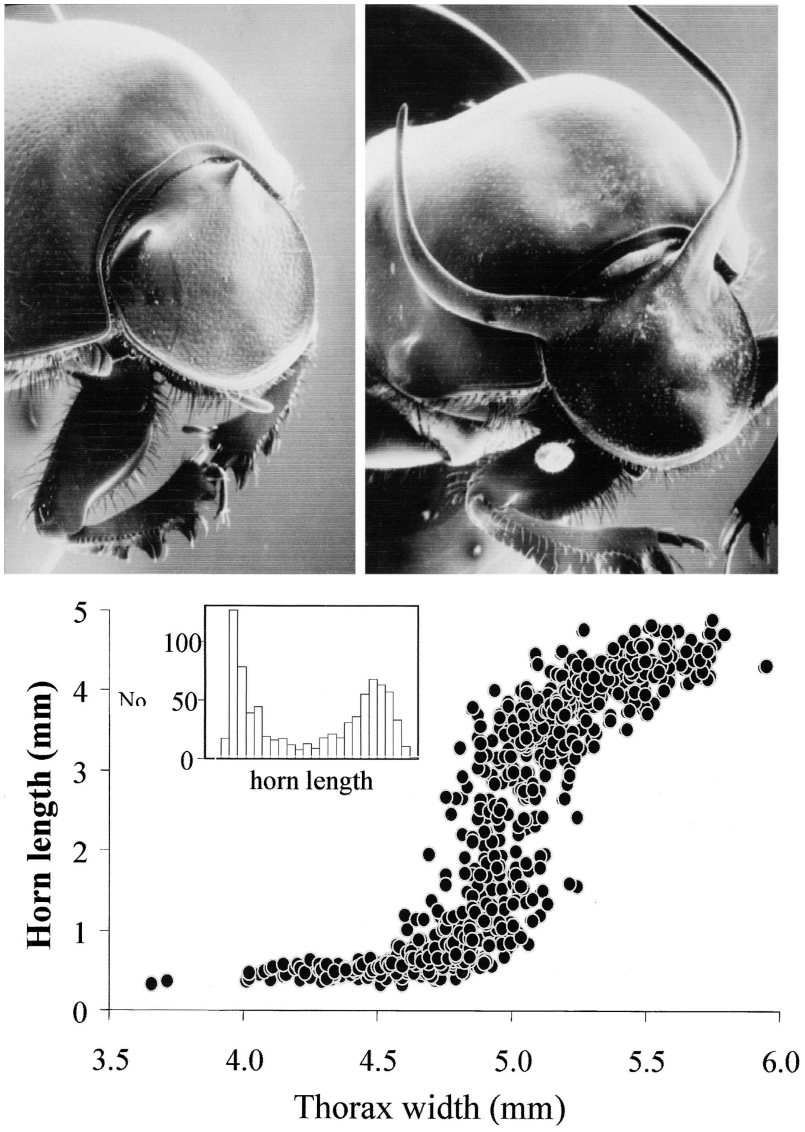

Fig. 7. Horn polyphenism in males of the beetle *Onthophagus taurus.* Horns are sigmoidally allometric with body size. As a result, horn size has a bimodal frequency distribution (inset). Beetles that metamorphose at a large body size develop large cephalic horns, whereas those that metamorphose at small body sizes are virtually hornless.

evolutionary escalation of body size, but this is not the case because body size in this beetle is not heritable (Emlen 1994; Moczek 1998; Moczek and Emlen 1999). Body size is determined entirely by the size and quality of the food supply with which a mother provisions a given egg. When a larva runs out of food, it almost immediately begins metamorphosis to the adult (Shafiei et al. 2001). Variation in food supply and in the nutritive quality of that food are the environmental variables that determine the range and distribution of body sizes.

Males that happen to be small are at a competitive disadvantage with large males, and this has led to the evolution of an alternative mating tactic in small males (Emlen 1997a; Moczek and Emlen 2000). These small males do not attempt to fight but either attempt to sneak past a defending male or actually dig their own tunnels that intersect defended tunnels at some distance below the defending male and thus gain access to the female. The alternative mating tactics of large- and small-bodied males result in a divergent selection on horn size. Large males benefit from having the largest possible horns, because these help them win combats. Small males benefit from having the smallest possible horns, because horns get in the way of their sneaking tactic. This divergent selection on relative horn size has resulted in a sharply sigmoidal body size–horn size allometry. Horn size in this beetle is a good example of adaptive phenotypic plasticity with two adapted extremes. The allometry of horns is effectively a reaction norm of horn size on nutrition.

The threshold body size at which the transition from hornless to horned males occurs is itself a plastic trait. When larvae are fed on food of low nutritive quality, mean body size is smaller and the transition to horn development occurs at a

smaller body size than it does in larvae fed on a nutrient-rich diet (Emlen 1997b).

The developmental regulation of horn expression has several components. Larvae of *Onthophagus* must somehow assess the presumptive body size of the adult, so that horn development can be suppressed or initiated, as appropriate. This assessment occurs in about the middle of the last larval instar (Emlen and Nijhout 1999) and coincides with a brief peak of ecdysone secretion that occurs in females and presumptive hornless males, but not in presumptive horned males. Treatment of presumptive hornless animals with juvenile hormone during this critical period induces subsequent horn development.

Horns develop during the prepupal stage from small imaginal disk-like clusters of cells on the head. In horned males these cells proliferate extensively during a very brief period of time, forming an elongated projection that will develop into the adult horn during metamorphosis. Similarly focused cell divisions do not occur in presumptive hornless males. There is a second period of hormone sensitivity in the prepupal stage, immediately before the time of horn differentiation. If prepupae are treated with juvenile hormone during this time, cell proliferation in the presumptive horns is suppressed (Emlen and Nijhout 2001). The presence of juvenile hormone during this second sensitive period thus inhibits horn development, which implies that normal horn development requires that juvenile hormone is absent at this time. Although the pathway of horn regulation has not yet been fully elucidated, it appears at present that it depends on two hormone-sensitive periods. A brief period of ecdysone secretion during the first hormone-sensitive period is regulated by a size assessment mechanism and may interact with juvenile hormone to program horn development in the prepupal stage. Horn development in the prepupa is, in turn, regulated during a second hormone-sensitive period during which juvenile hormone controls whether or not epidermal cells in the horn-forming region of the head will undergo cell divisions. Cell division in the insect epidermis is stimulated by ecdysone, so the simplest way in which juvenile hormone can exert its inhibiting effect is by suppressing the expression of ecdysone receptors in the appropriate epidermal cells.

The evolutionary origin of horn polypenism in *Onthophagus* thus involved two events: the origin of cephalic horns in males and the suppression of horn development in males that are smaller than a threshold size. Both events are regulated by hormones. Epidermal growth requires ecdysone, and the localized epidermal proliferation that results in horn development requires the localized expression of ecdysone receptors. In addition, a period of ecdysone secretion is required to induce cell proliferation. Insofar as ecdysone is used to regulate cell division and morphogenesis in many other tissues during metamorphosis, it is possible that horn development captured a preexisting peak of ecdysone secretion. This would represent a physiological co-option of a preexisting regulatory mechanism. The subsequent suppression of horns in small males required the evolution of a size-sensing mechanism in the final larval instar. Some insects monitor their body size by means of abdominal stretch reception (Nijhout 1994), but it is not known whether this is the case here. Our current hypothesis is that this size-sensing mechanism during the larval stage regulates the program of juvenile hormone level during the prepupal stage. Juvenile hormone normally declines just before the prepupal stage and is absent at the time that ecdysone-stimulated cell division occurs in the horns. It is possible that in small males the decline of juvenile hormone is delayed, so that it is still above threshold and is able to inhibit ecdysone-stimulated horn growth.

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