Adaptive phenotypic plasticity: consensus and controversy

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Phenotypic plasticity is an environmentally based change in the phenotype. Understanding the evolution of adaptive phenotypic plasticity has been hampered by dissenting opinions on the merits of different methods of description, on the underlying genetic mechanisms, and on the way that plasticity is affected by natural selection in a heterogeneous environment. During much of this debate, the authors of this article have held opposing views. Here, we attempt to lay out current issues and summarize the areas of consensus and controversy surrounding the evolution of plasticity and the reaction norm (the set of phenotypes produced by a genotype over a range of environments).

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When environments within the range of a species differ, it may be unlikely that any single phenotype will confer high fitness in all situations. In such a case, a change in the phenotype that depends on the environment (phenotypic plasticity) can provide increased environmental tolerance. Phenotypic plasticity is thus one solution to the problem of adaptation to heterogeneous environments. Because of the importance of heterogeneous environments in the ecology and evolution of most species, phenotypic plasticity has been of great interest to ecologists and evolutionary biologists for many years[1–4].

The literature on phenotypic plasticity has never been easy. Empirical studies have revealed phenotypic changes across environments for a wide variety of different characters in plants and animals, in natural and agricultural systems, and over both temporal and spatial variation in the environment[1–5]. Given the breadth of the empirical examples, a general picture of the issues and concepts involved in the evolution of phenotypic plasticity has been difficult to develop.

On certain issues, however, there is widespread agreement. It is clear that the degree of phenotypic change across environments can differ among characters, and that the amount and type of phenotypic change observed depends on the sort of environment under consideration[5]. The plastic changes in the phenotype may be either adaptive or not, but given appropriate genetic variability, adaptive phenotypic responses to the environment are thought to be able to evolve in populations that encounter predictable environmental change[1–4].

The theoretical literature on the evolution of phenotypic plasticity is also diverse and can be quite technical, making it difficult to sort out the biologically relevant areas of similarity and difference among models. Areas of clear consensus are elusive, partly because of differences among ways of conceptualizing phenotypic plasticity and the related concept – the norm of reaction (see Box 1). Two classes of model have recently been debated[6–12]. They are the 'character state' approach[13–15] and the 'polynomial'[16–18] (or 'reaction norm'[11,12]) approach. In the character state approach, the reaction norm for a particular character is modelled as the set of phenotypic values that would be expressed in each environment by a given genotype[2,13–15], and evolutionary models are based on population means and genetic (co)variances of these character states. In the polynomial approach, the reaction norm is described by a polynomial function of the phenotypic values expressed by a genotype across a range of environments[4,11,16–18], and evolutionary models are based on the population means and genetic (co)variances of coefficients of the polynomial.

Although these approaches are mathematically interchangeable under some circumstances[11,12], conceptual differences between modelling approaches have produced controversy about reaction norm evolution, particularly concerning the genetic basis of plasticity and the way that natural selection influences reaction norms[6–12]. The debate on the different modelling approaches pivots on the idea that phenotypic plasticity is a character in its own right, separate from the mean value of a character over all environments and under its own genetic control. The view that the mean and plasticity of a trait are separate has led to discussions of possible roles for 'genes for plasticity' and suggestions that selection might act on plasticity itself[6–8,19]. The alternative view is that phenotypic plasticity evolves as a by-product of natural selection on the phenotypic values of the character states expressed within environments[9,10], and that the same loci that affect the mean phenotype in each environment also determine the plasticity[9–11].

Three main issues must be addressed to resolve the present debate:

(1) How has our understanding of the evolution of phenotypic plasticity been influenced by the structure and assumptions of particular models?

(2) What is the genetic basis of plasticity? Are there 'genes for plasticity', and if so, what is their function and are they different from other loci that influence characters? Would including details of the underlying genetic system alter the predictions of models of reaction norm evolution? Do we need evolutionary models of the underlying genetic mechanisms to complement our models of the evolution of the phenotype?

(3) How does natural selection act on characters expressed in different environments, and under what conditions might natural selection act on the entire reaction norm rather than independently on phenotypic values within each environment?

Models of reaction norms and phenotypic plasticity

Discrete environments

Discrete environments arise both naturally (as in different host species for polyphagous insects) and as approximations to continuous environments (as in different light or temperature regimes). In either case, with n environments, the reaction norm for a genotype is described by the n values of the character states (the character state approach, Box 1). If environments are ordered along a single axis defined by the mean trait value[20], a polynomial function of the environment could also be used to model reaction norms in discrete environments (the polynomial approach, Box 1).

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Fortunately, for discrete environments, the character state and polynomial approaches are mathematically equivalent descriptions of the same biological pattern[11,12]. An empirical description of variability in reaction norms based on the means, variances and covariances of character states in different environments can be translated into the means, variances and covariances of the coefficients associated with the polynomial approach, and vice versa (Box 2).

If we consider reaction norm evolution as equivalent to the evolution of a set of correlated characters (either the character states or the polynomial coefficients), then genetic constraints on that evolution correspond to singularities in the genetic covariance matrices of character states or coefficients. Evolutionary constraints occur when the genetic variance of one or more character state (or coefficient) is zero, when any pairwise genetic correlation between character states (coefficients) is ±1, or when there are multiple correlations among the character states (coefficients) that cause the covariance matrix to be singular[2,13,15].

Importantly, because the models are meant to describe the same underlying biology, the same genetic constraints should be contained in the matrix of genetic variances and covariances of character states as in any of the several possible genetic covariance matrices of polynomial coefficients. However, because the actual matrices may look very different, the interpretation of the biological meaning of these constraints might easily differ[12]. Moreover, because sampling variance may have different effects on the estimates of genetic (co)variances of character states and those of coefficients, transforming between approaches (as outlined in Box 2) can sometimes produce a slightly different pair of matrices than if both matrices had been estimated directly from data.

Continuous environments

Gomulkiewicz and Kirkpatrick[15] extended the character state approach to continuous environments by replacing the vector of phenotypic means in different environments[13,14] with a mean function defined over a continuous environment. In their model, a covariance function that describes genetic variances within, and covariances of character states among, different environments on a continuum replaces the genetic covariance matrix of the discrete environment models.

Polynomial models describe the reaction norm in continuous environments in the same way as in the discrete environment case except that the environmental factor or index may assume any of a continuum of values[11,12,16–18]. The polynomial may represent an actual reaction norm, or it may approximate one by omitting higher order terms[11,12]. As in the discrete case, genetic constraints on the evolution of reaction norms in continuous environments may express themselves in the genetic covariance functions of character states[15] or in the covariance matrix of polynomial coefficients[4,17,18].

Although it is always possible to translate a polynomial model of the reaction norm in a continuous environment into a character state model, it is not generally possible to do the reverse (see Box 2). Thus, in continuous environments, the two approaches are not fully mathematically interchangeable. For both discrete and continuous environments, different insights may be obtained by considering the character state and polynomial models.

Box 1. Reaction norms and phenotypic plasticity

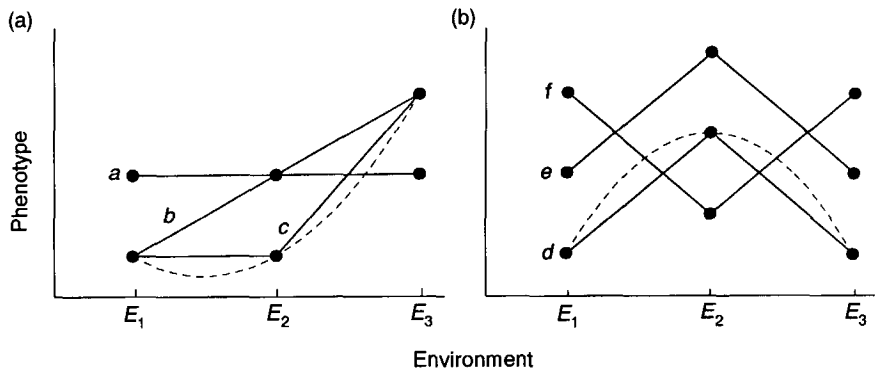
A reaction norm is the set of phenotypes that would be produced if a genotype were exposed to a defined set of environments. Although every individual inherits the genetic capability to produce each of the different phenotypes in the various environments, its actual phenotype depends on the particular environment(s) it experiences. The figure shows six hypothetical reaction norms for a trait in three environments (e.g. development time of a polyphagous insect on three different host plants, or plant size at three moisture levels).

This general view of the reaction norm as the potential phenotypic response to different environments applies to discrete and continuous environments; it is the definition used in this paper. A narrower definition is favoured by some[11,12] who apply the term reaction norm only to phenotypic responses to continuous environments that are modelled using the polynomial approach discussed below.

Two different mathematical descriptions of phenotypic responses to the environment are considered here:

The character state approach. In this view[13–15], the reaction norm is described by the set of phenotypic means expressed by a genotype over a range of environments. These are the character states. Graphically, means are plotted in arbitrary order on the *x*-axis for environments such as different host plants; they are ordered by the mean phenotype in each environment[20], or in the natural order for continuous environmental variables, such as temperature. A reaction norm can take on any shape in this visualization, and may be a simple set of lines connecting the phenotypes in discrete environments (solid lines in the figure below; Refs 13,14), or a function in a continuous environment[15]. The evolution of the reaction norm is described by changes in the mean phenotype expressed in each environment.

The polynomial (or reaction norm) approach. The responses are seen as a function of an environmental factor, for instance a polynomial[4,12,17,18]. For example, a straight line could be fitted to a set of means such as *a* or *b* in the figure, but a quadratic would be needed for norms such as *c*–*f* (examples are illustrated in dotted lines for *c* and *d*). In this approach, evolution of the reaction norm is described by changes in the coefficients of the polynomial.



Phenotypic plasticity is defined most generally as a change in the phenotype expressed in different environments (see Ref. 11 for a discussion of plasticity as a trait). In two environments, the change can simply be measured as a difference in the means (the character state approach) or as the slope (the polynomial approach). In multiple environments, measurement is more difficult[1,2]. Plasticity is sometimes collapsed into a single number, which quantifies some aspect of change, independent of the average phenotypic value. Such simple measures of plasticity may contain insufficient information to distinguish between reaction norms. For example, in part (b) of the figure, the reaction norms are very different, yet they show the same amount of change across environments. Thus, we propose to retain and analyse the entire set of parameters (such as the character states[2,13–15] or the vector of polynomial coefficients[4,16–18]) that describes the reaction norm.

Which model to choose for empirical studies?

How are empiricists to choose between the different approaches when planning experiments or analysing data on reaction norms? Since the two approaches are different parameterizations of the same general model, at least for the discrete environment case, the choice should be made based on the type of environment, the data type and the kinds of issues that one wishes to address.

Discrete or continuous environment?

De Jong[11] has stated that the polynomial model (also called the 'reaction norm' model[11,12]) is most appropriate for the study of graded responses in continuous environments, whereas the character state model is most appropriate to model

Box 2. Models of plasticity: character states or polynomial functions?

Using a multilocus quantitative genetic model, De Jong[11] showed that character state and polynomial models can be mathematically equivalent. A gaussian model[11,12] yielded the same result, and Van Tienderen and Koeliwijn[12] suggested statistical methods to switch from one approach to the other. The equivalence between the two approaches holds in discrete and in continuous environments when the reaction norm can be described by a polynomial or other Taylor expandable function.

The relation between the vector of character states in n environments, $\mathbf{z}^T=(z_1 \ldots z_n)$, and the coefficients of a polynomial function of order m (intercept, slope, etc.), $\mathbf{g}^T=(g_0 \ldots g_m)$, for a genotype can be written in matrix form as:

$$\mathbf{z}=\mathbf{X}\mathbf{g} \quad (1)$$

Each row of the matrix $\mathbf{X}$ has one in the first position, plus a polynomial series of a variable x that measures some aspect of the environment (the value of this variable in environment i being x_i). Using eqn (1), the character state in environment i is written as $z_i=g_0+g_1x_i^1+g_2x_i^2 \ldots g_mx_i^m=(1, x_i^1, x_i^2, \ldots, x_i^m)(g_0, g_1, g_2, \ldots, g_m)^T$. For a population, eqn (1) also holds, using the mean values of $\mathbf{z}$ and $\mathbf{g}$.

When the transformation in eqn (1) applies, the genetic (co)variance matrices of character states ($\mathbf{G}_z$) and of polynomial coefficients ($\mathbf{G}_g$) for a given population are related as:

$$\mathbf{G}_z=\mathbf{X}\mathbf{G}_g\mathbf{X}^T \quad (2a)$$

and:

$$\mathbf{G}_g=\mathbf{U}\mathbf{G}_z\mathbf{U}^T \quad (2b)$$

where $\mathbf{U}=(\mathbf{X}^T\mathbf{X})^{-1}\mathbf{X}^T$. Note that $\mathbf{U}$ exists only when $n>m$, that is, when the number of distinct environments is higher than the order of the polynomial.

Given eqn (1), the relationship between the selection gradients[26] on character states ($\boldsymbol{\beta}_z$) and on polynomial coefficients ($\boldsymbol{\beta}_g$) can also be found[11,12], yielding:

$$\boldsymbol{\beta}_g=\mathbf{X}^T\boldsymbol{\beta}_z \quad (3)$$

where $\boldsymbol{\beta}_z$, the selection gradient on character states, contains the within-environment selection gradients weighted by their environment's frequency[11,12]. The selection gradient on polynomial coefficients ($\boldsymbol{\beta}_g$) contains the covariances between the within-environment selection gradient and the appropriate powers of the environmental variable, x (Ref. 11). Thus, the selection gradient on the value in the mean environment (g_0) becomes the mean of the within-environment selection gradients [$\mathrm{cov}(x^0, \boldsymbol{\beta}_z)$], the selection gradient on the slope (g_1) becomes the covariance between the value of the environmental variable and $\boldsymbol{\beta}_z$ [that is, $\mathrm{cov}(x, \boldsymbol{\beta}_z)$], the selection gradient on the quadratic term is the covariance between the square of the environmental variable and the elements of $\boldsymbol{\beta}_z$ [that is, $\mathrm{cov}(x^2, \boldsymbol{\beta}_z)$], and so on.

For a multilocus model without epistasis[11], and a gaussian model[11–13], the selection responses in the vectors of the population mean values of character states ($\Delta\bar{\mathbf{z}}$) and polynomial coefficients ($\Delta\bar{\mathbf{g}}$) can be independently derived from eqns (1), (2a) and (3) as:

$$\Delta\bar{\mathbf{z}}=\mathbf{G}_z\boldsymbol{\beta}_z \quad (4a)$$

$$\Delta\bar{\mathbf{g}}=\mathbf{G}_g\boldsymbol{\beta}_g \quad (4b)$$

Also, from eqn (1), $\Delta\bar{\mathbf{z}}=\mathbf{X}\Delta\bar{\mathbf{g}}$

discrete responses to discrete environments. This is consistent with the view that the coefficients of the polynomial may be useful for understanding the biological phenomena underlying a response to a continuous environment. For example, the quadratic term may describe a metabolic response to temperature that is concave downward. However, the character state model was originally formulated to describe the evolution of graded responses to environments that are either actually discrete or simply points along a continuous gradient[13]. It has since been expanded to describe reaction norms in truly continuous environments[15]. Because organisms may perceive apparently discrete environments on a continuous scale that is determined by the quantity of some chemical cue (or nutritional suitability), or perceive continuous environments as being discrete (due to thresholds for particular stimuli), Van Tienderen and Koelewijn[12] argue that 'the distribution and nature of the environmental factor is not necessarily a useful guideline for a choice for one approach or another'.

In discrete environments, the character state and polynomial approaches are interchangeable: genetic (co)variances for polynomial coefficients can be calculated from those for character states, and vice versa[11,12]. However, in continuous environments, estimates of genetic (co)variances of character states cannot generally be transformed into estimates of genetic (co)variances of polynomial coefficients. Thus, the crucial evolutionary parameters for each approach must be estimated independently from the data. This makes the data type and the questions to be addressed paramount in deciding between methods. In some cases, it may be useful to examine a particular data set using both approaches.

Data types

To estimate genetic parameters of reaction norms, designed experiments are required in which several progeny from each family or clone are exposed to a discrete number of environmental treatments[12,21–25]. In continuous environments, both methods can allow extrapolation of the reaction norm between testing points to environments in which no observations were made (see Ref. 15 for an example of this for the character state approach). In the character state approach, estimation of within-environment means and genetic variances requires many observations in each environmental treatment. If it is impractical to test many individuals per family, or if conditions are such that few individuals experience exactly the same environment, the polynomial approach may be useful because all of the data for each family are used to estimate each of the parameters (although a measure of the environment must be obtained for each individual).

Certain types of characters (such as metabolic rate) that are expected to have a reaction norm over a continuous environment of a particular shape may perhaps be best understood using the polynomial approach[11]. In contrast, the character state approach may be more useful when most individuals occur in only a few different environments[2,13] or when the expected shape of the reaction norm over a continuous environment is not expected to be well fit with a polynomial[15]. Finally, in coarse-grained environments in which an individual spends its entire life within one environmental patch, it makes sense to estimate genetic parameters and individual selection on the various character states, rather than on polynomial coefficients, which are not expressed by individuals[9].

Estimation of selection on reaction norms

In the character state approach, selection is estimated by regressing individual relative fitness on phenotypic values within environments[26]. The selection gradients combined over all environments yield a vector with elements that quantify the forces of directional selection on the character state expressed in each environment (this vector becomes a function in the character state approach to continuous environments[15]). The biological meaning of the selection gradient on character states is straightforward because it is based on traits that can be observed in individuals, and concerns individual selection within environments.

Because no single individual is likely to experience all environments, phenotypic selection on the polynomial coefficients cannot be estimated empirically, although it can be calculated from the regressions of relative fitness on character state values within environments[11,12] (see Box 2). However, selection on the polynomial coefficients can be estimated at the genotypic level if different individuals of each genotype are spread across environments[11,27]. To do this, one would first fit a polynomial to observations for each genotype. Then, by weighting fitnesses within each environment with the environmental frequencies, the mean relative fitness of each genotype can be calculated and regressed on the polynomial coefficients for each genotype to obtain genotypic selection gradients.

Selection on the intercept and slope in the overall population may be readily biologically interpretable in some situations[12,27]. Depending on its sign, a significant quadratic term may either indicate selection for an intermediate optimum

value over a continuous environment or selection to reduce the curvature of a reaction norm. However, significant selection on the higher order coefficients is more difficult to interpret. For example, what does it mean in terms of the ecology of the organism to say that selection favours an increase or decrease in the coefficients of the cubic or higher terms? Perhaps a graphical comparison of the mean polynomial before and after selection would prove to be useful for interpreting selection on all coefficients simultaneously.

Estimation and interpretation of genetic constraints on reaction norm evolution

Constraints on the evolution of plastic responses may show up as singularities in the covariance matrix of either character states or polynomial coefficients (Box 2). However, documenting constraints empirically may be difficult because of the large sampling variances of genetic (co)variances[28]. The biological interpretation of constraints can be straightforward in the character state approach because they correspond to a lack of genetic variance within one or more environments or a pairwise or joint pattern of genetic correlations of ±1 among two or more character states[2]. In contrast, constraints in the covariance matrix of polynomial coefficients reflect absence of genetic variance in one or more coefficients or unfavourable correlations among coefficients. These constraints may be difficult to interpret biologically unless the character is a simple product of some biophysical law (such as enzyme K_m curves, which are constrained to a particular shape). Even though the biology is the same, the interpretation of constraints is almost certain to differ between reaction norms described with the two approaches[12]. Future work to determine the situations for which each method is best suited would be very useful.

Genetic mechanisms of plasticity

Little is known about the genetic basis of variation in any quantitative trait[29]; our knowledge of the genetic basis of plastic reactions is even scantier. Substantial progress has been made in conceptualizing the genetic basis of plasticity since the recent reviews by Schlichting[1],Via[2,9] and Scheiner[4]. Two classes of genetic effects that influence plastic responses to the environment can now be distinguished: (1) some alleles may be expressed in several different environments with varying effects on the phenotype ('allelic sensitivity'), and (2) regulatory loci may cause other genes to be turned on or off in particular environments ('gene regulation'). These classes may blur to the extent that regulatory loci influence the amount of gene product in different environments, thus potentially mediating allelic sensitivity.

Because both types of genetic mechanisms can cause plastic changes in the phenotype, either a locus showing allelic sensitivity or a regulatory locus with environment-specific action could be regarded as a plasticity gene. However, it is important to note that we are not defining loci that produce change that is in some way separate from the mean phenotype[6–8,19]. Both plasticity and the mean trait value are determined by 'the genes that determine the function joining the character states'[11]. This definition of plasticity genes is broader than that advanced by Schlichting and Pigliucci[8,19], who defined them as 'loci that exert environmentally dependent control over structural gene expression', thus focusing only on regulatory genes and excluding loci that exhibit allelic sensitivity.

Historically, two main types of plasticity have been recognized: graded responses and discrete or switched responses. Schmalhausen[30] defined a graded response to the environment as 'dependent development' and Smith-Gill called this 'phenotypic modulation'[31]. In contrast, Schmalhausen used the term 'autoregulatory morphogenesis' when distinct phenotypes are produced in different environments in a way that is apparently uncoupled from small environmental variation, and Smith-Gill termed this switch to a distinctly different phenotype 'developmental conversion'.

Recently, De Jong[11] and Schlichting and Pigliucci[19] have discussed how the two genetic mechanisms of plasticity may map onto these two major types of plastic responses. Both identified allelic sensitivity as the basis of graded responses (phenotypic modulation), and gene regulation as the basis of discrete responses (e.g. developmental conversion). However, distinct phenotypes in different environments could be produced either if regulatory loci act as 'switch genes'[32] or if a threshold response is coupled to continuous variation in some underlying character (potentially mediated either by allelic sensitivity or gene regulation). Moreover, regulatory genes may also affect graded reaction norms by turning a subset of the loci involved on or off in different environments or by altering the amount of gene product produced in different environments. Thus, real reaction norms probably involve both types of loci to varying extents: many cases of apparent allelic sensitivity are potentially mediated by regulatory loci that influence the amount of gene product, and the major action of a switch gene could be modified by accompanying allelic sensitivity at other loci.

Perhaps the most controversial issue presently associated with the genetic basis of reaction norms is whether or not explicitly including the epistatic gene action of the regulatory loci will alter the dynamics of the evolution of plastic responses as predicted by current genetic models. Epistatic gene action between regulatory and structural genes is probably pervasive[8,33,34]. However, genetic variation in the phenotype is only contributed by structural loci that are actually expressed, and even when there is epistatic gene action among structural loci, most of the genetic variance from these loci is additive genetic variance[35,36]. Genes with epistatic effects can thus influence the response to individual selection through their contribution to the additive genetic variance, and this is implicit in quantitative genetic models such as those of Via and Lande[2,13], Van Tienderen[14,37], Gomulkiewicz and Kirkpatrick[15], and Gavrilets and Scheiner[17,18]. For short-term predictions of evolution of phenotypic responses to the environment, such models may suffice as a first approximation even when epistatic variance exists[38].

The long-term effects of epistatic gene action on the response to phenotypic selection have not yet been incorporated into any model of reaction norm evolution. Schlichting and Pigliucci[8] argue that epistasis between regulatory and structural loci may affect the way that genetic (co)variances change in the course of evolution, and thus the permanence of genetic constraints on reaction norm evolution. It is certainly possible that evolution at the regulatory level could alter the pattern of genetic (co)variances in ways that cannot be predicted from current estimates of covariance matrices. Evolutionary models of the genetic mechanisms that underlie plasticity might be useful to complement quantitative genetic models of phenotypic evolution.

As yet, there is no consensus on the relationship between the presence of switch genes, their importance in epistatic gene action, whether they generate epistatic variance in character states, and their consequences for the evolution of plasticity. Those who believe that the outcome of selection is determined mainly by the internal constraints that are reflected in genetic covariances may regard current quantitative genetic models as a useful baseline against which the effects of complicating factors such as epistasis can be measured. In contrast, those who believe that the evolution of plasticity is governed not as much by selective forces and genetic covariances as by the contingent occurrences of novel regulatory mechanisms and switch genes may see little use in any of the current genetic models.

How does selection act on reaction norms?

Describing natural selection on phenotypically plastic traits in variable environments is a relatively complex undertaking. This is because the environments experienced by a population determine the phenotypes that individuals express and also establish the fitness consequences of those phenotypes. For a particular trait, all analyses require specification of (1) the trait's responses to varying environmental cues, (2) the pattern of environmental heterogeneity, (3) the form of selection within each environment, and (4) possible biological carry-over of either character states expressed early in the life cycle, or the maternal environment on characters expressed later in life. Combined, these factors determine the consequences of selection.

Considerable controversy has been generated about whether the shape of a reaction norm can be directly affected by selection, or whether reaction norms respond to selection only indirectly, through the evolution of the mean phenotypes in each separate environment[6–10]. Part of this controversy stems from whether one views selection as a process acting only on individuals or also on genes (through group selection on related individuals in different environments).

Many examples of phenotypic plasticity have probably evolved as by-products of selection towards different trait values within different environments[9,11]. When within-generation variation in the environment is absent (as in models of between-generation temporal or coarse-grained spatial variation), or when traits take on only a single value during an individual's lifetime (such as age or size at maturity), an individual can express at most a single component phenotype of its reaction norm. In such a case, only the expressed character state is exposed to individual selection in a particular environment, although evolution can occur in the rest of the reaction norm through correlated responses[13–15].

However, under some circumstances, it may be appropriate to consider selection acting on the entire reaction norm[4]. If the environment changes within generations and characters are labile during an individual's lifetime, several phenotypic components of the reaction norm (character states) could be selected each generation even though only one character state is expressed at a time. The sequence in which environments are experienced may affect the outcome of selection for two reasons. There may be biological carry-over effects of character states expressed early in the lifetime on individual fitness later in the life cycle (such as effects of a poor environment during juvenile stages on fecundity in the adult environment). Also, the selection gradient[26] on a given character state depends upon the environments that have been encountered previously. With these caveats, one could say that selection acts on multiple elements of a reaction norm of a labile trait when populations experience a fine-grained environment (see Ref. 15 for an example).

Several character states may also be selected simultaneously if reaction norms differ in the costs that they require to be produced or maintained[14]. If plasticity (or its converse, constancy) is costly, reaction norm shapes that are less costly to maintain can be favoured by selection. This can cause reaction norms to be selected on components other than the one currently expressed.

Finally, selection acting at the group level on clones or families rather than on individuals could also affect several components of the reaction norm each generation. In this case, the reaction norm is expressed among individuals of the same genotype in different environments. However, models of plasticity evolution in such structured populations have not yet been developed, so the conditions necessary to promote group selection on reaction norms are presently unclear.

Conclusions and future directions

Recent work has shown that two favoured models for describing the reaction norm (as a set of character states in different environments or as a polynomial function) are mathematically equivalent for the discrete environment case. However, the biological interpretation of evolutionary constraints and measures of natural selection on reaction norms can greatly depend on whether or not the problem is formulated on the basis of character states or polynomial functions. In some cases, it may be fruitful to analyse the same data set both ways, in order to compare and contrast the interpretations resulting from the two approaches. In continuous environments, the two models are probably not interchangeable, but they can each provide valuable insight into reaction norm evolution.

Two types of loci, those with environmentally based allelic sensitivity and regulatory loci that alter gene expression across environments, can be regarded as 'plasticity genes'. However, controversy remains over the extent to which these two genetic mechanisms map onto the two main types of plastic reactions to the environment (graded or switched). In most cases, reaction norms probably evolve as by-products of selection on phenotypic values expressed within environments, but selection may sometimes directly change the form of a reaction norm.

Much remains to be done at empirical and at theoretical levels before we will truly understand the mechanisms by which adaptive reaction norms evolve.

Empirical work

- It will be very useful to analyse an accumulation of empirical studies on the genetic basis of reaction norms. Currently, the relative contributions to the reaction norm of allelic sensitivity and regulatory genes that act as switches are unclear. We look forward to evaluating whether clear patterns emerge for particular situations.
- When cases of epistatic gene action among loci expressed within environments are identified, we must learn to what extent these loci produce epistatic variance (rather than additive genetic variance) and how this affects the evolutionary outcome of selection for different phenotypes in different environments.
- We need to determine the frequency of genetic constraints on reaction norms and understand more about the nature of such constraints. For instance, are these constraints due to unavoidable energetic trade-offs, such as when internal machinery needs to be maintained to produce the plastic (or homeostatic) reaction? Or are they due to the absence of regulatory mechanisms that may or may not evolve in the long run? The answers to these questions will influence how genetic constraints and genetic covariances change during the course of evolution.
- Much more empirical information is needed about the strength and types of selection on reaction norms, including the differences in phenotypic optima in different environments, the extent of group selection on reaction norms and the cost of plastic responses.
- We are also largely ignorant about the actual patterns of environmental heterogeneity experienced by populations. It would be useful to know more about the frequency and pattern of temporal change in environments, the spatial scale of changes in selection, and the number and frequencies of different environments commonly experienced by a given population.

Theoretical work

It will be difficult to interpret the evolutionary consequences of different forms of gene action or genetic variance on reaction norm evolution without additional theoretical work. Standard quantitative genetic models do not preclude the presence of gene regulation or epistatic interactions, insofar as they contribute to the additive genetic (co)variance. However, epistatic interactions may play an unknown role in long-term evolution through contribution of epistatic variance and effects

on changes in the additive genetic covariance structure.

- How do epistatic variance and epistatic gene action affect the long-term evolutionary response to selection on reaction norms? How robust are predictions from the current models that are based on additive genetic variance?
- Can explicit genetic models ('gametic' models[4,8]) be formulated that will complement the genetically less-detailed quantitative genetic models, and can we learn enough about the underlying genetic basis of plasticity to adequately estimate the parameters of such models?
- Do we need models that specifically address the evolution of regulatory mechanisms?
- Can we improve the extent to which the character state and polynomial models can be interchanged for continuous environments? What is the role of the sampling variance in the transformations? When the approaches are not interchangeable, will it be useful to apply both approaches?

Our challenge will be to use future theoretical work to design experiments in which values for critical parameters can be estimated in a range of natural populations. This will permit us to refine, reformulate and test concrete hypotheses about the mechanisms of reaction norm evolution.

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Animal ecomorphology

Ecological Morphology: Integrative Organismal Biology

by P.C. Wainwright and S.M. Reilly

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Modern ecomorphological analysis, as typified by analyses of organismal function in an ecological context, is extending results from traditional comparative analyses of functional morphology. It has been a quiet revolution that has been heralded by two paradigmatic shifts in conceptual approaches to analysis of morphological evolution. First, the synthesis of modern phylogenetic analysis in a statistically rigorous framework[1] with ecomorphological analysis has served to consolidate cladistic studies of morphology with analyses of form and function. Second, causes and ecological mechanisms underlying evolutionary changes in morphology can be studied directly by analyzing the effects of morphology on performance and the cascading effects of performance on fitness[2]. These recent conceptual advances are laced throughout the edited chapters of *Ecological Morphology: Integrative Organismal Biology*. The first conceptual advance, analysis of phylogenetic distribution of morphological change and associated changes in ecology, allows us to study the pattern of evolution. The second conceptual advance, analysis of natural selection, allows us to study the ecological processes responsible for evolution of morphology. Analysis of pattern and process are fundamental to all evolutionary studies, and thus *Ecological Morphology* probably has a more widespread audience than just ecomorphologists.

A useful starting point in a review of *Ecological Morphology* is a comparison with its conceptual doppelgänger, *New Directions in Ecological Physiology*[3], published seven years ago. Indeed, in the introduction, Wainwright and Reilly (p. 2) make the important point that 'We visualize ecological morphology as a field with no current conceptual distinctions from physiological ecology...'. The only formal distinction is that ecological physiology focuses on the role of abiotic environments in shaping physiology, whereas ecological morphology focuses on the role of abiotic and biotic environments in shaping morphology. This distinction may be somewhat artificial, given that many physiological processes are in a large measure inextricably linked in some way to morphology. Bradley's chapter in *Ecological Morphology* on the evolution of saline tolerance in mosquitos illustrates this point in a lucid manner. At the heart of both fields is the study of organismal adaptation.

Ecological Morphology is an excellent companion volume to *Ecological Physiology*. Wainwright and Reilly's explicit goal (p. 7) was to demonstrate to 'graduate students and others who are developing research programs in this area...the value of an integrative approach and present both a conceptual framework and a practical guide so that individuals working on the mechanistic basis of organismal performance may link that performance to evolutionary and ecological processes'. The editors and authors of individual chapters have succeeded in pointing out areas where conceptual insights are currently being made and they have also succeeded in documenting 'limitations and shortcomings of research to date'.

Empirical advances summarized in this book demonstrate that our understanding of patterns of ecomorphology (e.g. comparative patterns based on phylogenetic analysis) is further advanced than is our