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Review

Research review

Evolution of phenotypic plasticity: patterns of plasticity and the emergence of ecotypes

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Summary

Phenotypic plasticity itself evolves, as does any other quantitative trait. A very different question is whether phenotypic plasticity causes evolution or is a major evolutionary mechanism. Existing models of the evolution of phenotypic plasticity cover many of the proposals in the literature about the role of phenotypic plasticity in evolution. I will extend existing models to cover adaptation to a novel environment, the appearance of ecotypes and possible covariation between phenotypic plasticity and mean trait value of ecotypes. Genetic assimilation does not sufficiently explain details of observed patterns. Phenotypic plasticity as a major mechanism for evolution – such as, invading new niches, speciation or macroevolution – has, at present, neither empirical nor model support.

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Introduction

Two different views about phenotypic plasticity can be found in the literature, but as both views use the term 'phenotypic plasticity' they are often confounded. In the first view, phenotypic plasticity is a quantitative trait subject to selection and evolution and a property of the genotype. In this view, phenotypic plasticity might be of huge ecological importance but is a trait as any other trait as regards evolution. In the second view, phenotypic plasticity is a developmental process facilitating evolution. Here, phenotypic plasticity is an inherent property of the developing phenotype. In this second view, the most important question is not how phenotypic plasticity itself evolves, but how phenotypic plasticity changes development and thereby causes evolution. To rephrase and possibly overstate the divergence, phenotypic plasticity might be a trait subject to selection, or a developmental mechanism as important as selection in evolution.

Unfortunately, the two views are often not sufficiently distinguished. In many introductions to articles about phenotypic plasticity one might find in an introductory list of citations Schlichting (1986), West-Eberhard (1989) and Scheiner (1993), to cover reviews about plants, animals and models. However, these authors do not have the same views on phenotypic plasticity. Scheiner takes the first view, West-Eberhard the second, and Schlichting might have shifted from the first view in 1986 to the second view in 2003/2004 (Schlichting, 2003, 2004). The books by DeWitt & Scheiner (2004a) and by West-Eberhard (2003) clearly show the contrast. The first book mentioned focuses mainly on phenotypic

plasticity as an evolvable and evolving trait and the second book mentioned focuses on the role of developmental plasticity as agent of evolution. In the book by DeWitt & Scheiner (2004a) a difference in approach exists between the introductory chapter by DeWitt & Scheiner, and Schlichting's chapter. DeWitt & Scheiner (2004b) deal with the evolution of phenotypic plasticity as a classical quantitative genetics problem in an ecological context, whereas Schlichting (2004) is concerned with the role of developmental phenotypic plasticity in the diversification of taxa and the facilitation of evolutionary change.

West-Eberhard (2003) emphasizes the role of phenotypic plasticity in evolution. She proposes that adaptive evolution involves four stages. '(i) *Trait origin* – the initial appearance of a qualitatively distinctive developmental variant, which occurs when some new input (e.g. a mutation or an environmental change) affects a pre-existing responsive phenotype, causing a phenotypic change or reorganization. (ii) *Phenotypic accommodation* by individual phenotypes – the immediate adjustment to a change, due to the multidimensional adaptive flexibility of the phenotype. (iii) *Recurrence, or initial spread* – due to the recurrence of the initiating factor, whether environmental or mutational. This produces a subpopulation of individuals that express the trait. (iv) *Genetic accommodation* – gene frequency (evolutionary) change due to selection on variation in the regulation, form or side-effects of the novel trait in the subpopulation of individuals that express the trait' (p. 140, italics as in original text). West-Eberhard (2003) proposes that environmentally induced phenotypic change can give rise to adaptive evolution as readily as mutationally induced change, and that environmental factors can initiate evolutionary novelties (p. 498). She regards genes as followers in evolution (pp. 157–158). 'Adaptive innovation begins with reorganization of an already highly adapted genotype, in which negative effects are ameliorated by adaptive developmental plasticity'. 'Gene frequency change follows, as a response to the developmental change. In this framework, most adaptive evolution is accommodation of developmental-phenotypic change. Genes are followers, not necessarily leaders, in phenotypic evolution' (p. 158).

Schlichting (2004) ascribes a major role in evolution to genetic assimilation of novel phenotypes, and Pigliucci & Murren (2003) propose to regard genetic assimilation as a central concept. Genetic assimilation implies the conversion to a fixed genetic trait of an initially totally environmentally induced phenotypic threshold response (Waddington, 1942, 1953); the environmentally induced response need not be adaptive. After genetic assimilation, the phenotype is no longer plastic. In genetic assimilation, phenotypic plasticity is not itself of importance, but only an intermediate stage to a new genetically fixed and phenotypically invariant state. These ideas tend to ignore most quantitative genetic models of the evolution of phenotypic plasticity (see Berrigan & Scheiner's chapter in DeWitt & Scheiner (2004a) or Pigliucci (2001) for an overview) but, more importantly, an emphasis on genetic assimilation denies the ecological importance of phenotypic plasticity. The quantitative genetic view regards phenotypic plasticity itself as an evolved ecological adaptation to an intrinsically varying environment. The genetic assimilation view emphasizes the invariant phenotype and absence of genetic variation at the end of selection and seems concerned with ecotypic differences in two separate constant environments (Pigliucci & Murren, 2003).

To state my own position, I regard phenotypic plasticity as a quantitative trait conceptually comparable to any other trait subject to selection, and not as a developmental mechanism or process that facilitates evolution. My interest is in how phenotypic plasticity evolves, and whether it leads to adaptation. I make quantitative genetic models that tend to take an ecological view. Here, I intend to explain quantitative genetic models of the evolution of phenotypic plasticity and some of their implications, especially with regard to novel environments and limited migration. I will show how phenotypic patterns as reviewed by Pigliucci & Murren (2003) arise as straightforward consequences of selection for phenotypic plasticity in a population with limited migration.

Selection

Endler (1986) description of natural selection fully conforms to the description of natural selection by Darwin; at the same time it translates immediately into the standard model of change in the mean of a quantitative trait under selection (Falconer & Mackay, 1996; Roff, 1997). Endler's three assumptions translate into the presence of the phenotypic variance $V_P(z)$ in trait z, the existence of a phenotypic covariance between trait value z and fitness value w as $\mathrm{cov}_P(z, w)$, and the presence of genetic variation in trait z, as the genetic variance $V_A(z)$. Endler's first conclusion can be translated into the selection differential and the selection gradient: the mean of the trait distribution changes within a generation due to selection from $\bar{z}$ to $\bar{z} + S$, where the selection differential $S = \mathrm{cov}_P(z, \mathrm{w})/\bar{w}$. The selection gradient β is defined as the slope of fitness with phenotype, $\beta = \mathrm{cov}_P(z, w)/V_P(z)$. Endler's second conclusion translates into the predicted change in the mean of the trait distribution over two generations as the selection response equals

$$\Delta\bar{z} = \frac{V_A(z)\ \mathrm{cov}_P(z, w)}{V_P(z)\ \bar{w}} \qquad \text{Eqn 1}$$

The selection response can be written in two ways: as the well known $\Delta\bar{z} = h^2 S$ (Falconer & Mackay, 1996), where the heritability $h^2 = V_A(z)/V_P(z)$ gives the proportion of the phenotypic variance that is genetic, and as $\Delta\bar{z} = V_A(z)\beta/\bar{w}$ involving the selection gradient. The selection gradient can almost equivalently be written as $\beta = \partial\bar{w}/\partial\bar{g}$. The form $\beta = \partial\bar{w}/\partial\bar{g}$; (the change in mean fitness relative to the change in mean genotypic value) connects with selection in

multilocus models and with simultaneous selection on more than one character, and is therefore the most frequently used form (Lande, 1979).

The selection response in several traits resulting from simultaneous selection can be predicted from a matrix multiplication involving the genetic variances in the traits, their genetic covariances and the selection gradients on all traits. For two traits, the predicted responses in the mean genotypic values and therefore in the phenotypic trait means are given by:

$$\begin{bmatrix}\Delta\bar{z}_1\\ \Delta\bar{z}_2\end{bmatrix}=\frac{1}{\bar{w}}\begin{bmatrix}V_A(z_1) & COV_A(z_1,z_2)\\ COV_A(z_1,z_2) & V_A(z_2)\end{bmatrix}\begin{bmatrix}\dfrac{\partial\bar{w}}{\partial\bar{g}_1}\\ \dfrac{\partial\bar{w}}{\partial\bar{g}_2}\end{bmatrix} \quad \text{Eqn 2a}$$

or, in matrix notation

$$\Delta\bar{\mathbf{z}}=\frac{1}{\bar{w}}\mathbf{G}\boldsymbol{\beta} \quad \text{Eqn 2b}$$

($\Delta\bar{\mathbf{z}}$, vector of selection responses in phenotypic means for all traits; $\mathbf{G}$, genetic variance covariance matrix; $\boldsymbol{\beta}$, vector of selection gradients (Lande, 1979)). The vector of changes in mean phenotype for each trait is equivalent to the vector of changes in mean genotypic values due to selection: $\Delta\bar{\mathbf{z}}=\Delta\bar{\mathbf{g}}$. Expression 2b can be used for any number of traits. Independent evolution of all traits under selection requires that traits are not fully correlated; in genetic terms, that the number of loci coding for the traits is at least as large as the number of traits. Expression 2 represents a direct mathematical representation of Darwin's theory.

Selection on phenotypic plasticity as selection on more than one trait

Selection on phenotypic plasticity is always selection on more than one character. The most direct formulation is to regard trait expression in each environment as separate characters, the character states for these environments (Via & Lande, 1985) (Fig. 1a,b). This gives a basic model for selection on phenotypic plasticity that can be used as a point of departure for subsequent models. In Via and Lande's formulation, the selection response vector gives the changes $\Delta\bar{g}(x)$ in mean trait value $\bar{g}(x)$ in each environment x. The genetic variance–covariance matrix contains the genetic variances within each environment and the genetic covariance between trait expression over environments. The condition for independent evolution can now be interpreted as a genetic correlation over environments that is less than 1 in absolute value; $r=|1|$, i.e. genetic identity of traits in different environments, prevents independent evolution. Overall mean fitness $\bar{w}$ over all environments is given by the arithmetic mean of per environment mean genotypic fitnesses $\bar{w}_i$ weighted by the frequency f_x of each environment:

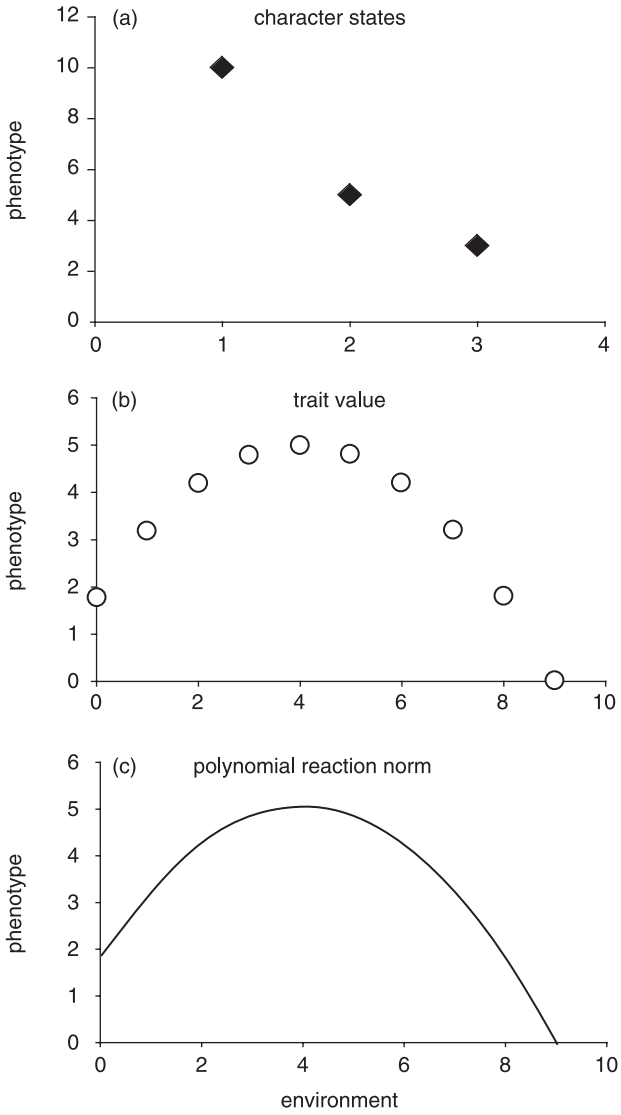


Fig. 1 Character states and reaction norms. (a) Individuals show very different traits in separate environments; no common physiological basis is present. (b) Individuals show different but related trait values in different environments. (c) The trait values shown in (b) can be regarded as function values at different environmental values given an underlying function, the reaction norm.

$$\bar{w}=\sum_x f_x\bar{w}_x$$

The selection gradient vector has the elements $\partial\bar{w}/\partial\bar{g}(x)$ for each environment x; given the expression for overall mean fitness, the selection gradient for each environment equals $f_x\partial\bar{w}_x/\partial\bar{g}(x)$. This simply means that each environment contributes to the overall selection in proportion to its frequency and its local selection strength (de Jong & Bijma, 2002).

We will be concerned with optimizing selection and here will use a quadratic fitness form that sits easily with multilocus selection. An analogous Gaussian fitness form is more convenient in modelling optimizing selection on a normally

distributed trait. In each environment x an optimum phenotypic value $\theta(x)$ is present. Fitness as a function of the individual's trait value in that environment decreases quadratically with distance from the optimum. Individual fitness is modelled as $w(x) = 1 - s(\theta(x) - z(x))^2$ if $1 - s(\theta(x) - z(x))^2 > 0$ and $w(x) = 0$ if $1 - s(\theta(x) - z(x))^2 > 0$. The selection gradient in environment x equals $\frac{\partial \bar{w}(x)}{\partial \bar{g}(x)} = 2s(\theta(x) - \bar{g}(x))$. Selection stops when all selection gradients in all environments are zero, that is, when the mean genotypic value in each environment equals the optimum phenotypic value in that environment: $\bar{g}(x) = \theta(x)$. Note that the result pertains to the mean genotypic value. Genetic variance is still present when the mean genotypic value and mean phenotypic value reach the optimum; if genetic variation were exhausted the optimum could not be reached. In the Via & Lande (1985) model, optimum plasticity is always reached, if the initial genetic variation allows it.

The model of Via & Lande (1985) gives the starting point for other models of the evolution of phenotypic plasticity. Specific model results follow from elaborating either the physiology of the trait or the life history over environments.

Trait continuity over environments

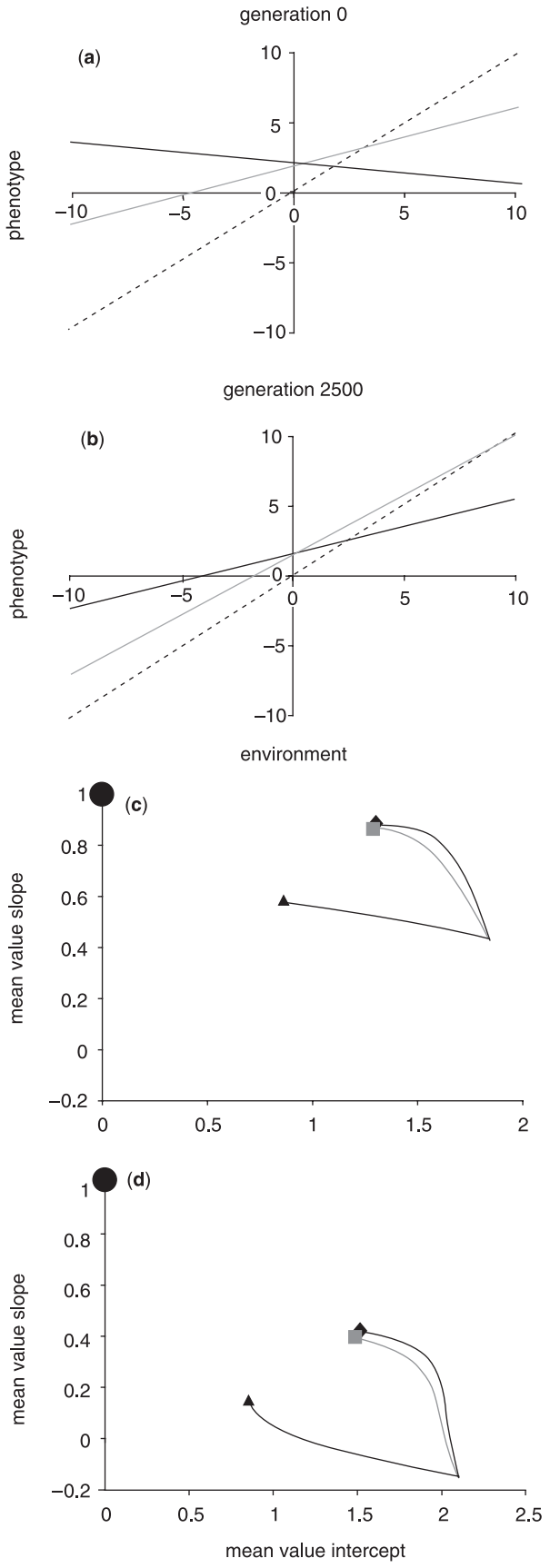
Phenotypically plastic traits are of two types. Discrete traits might be genetically and developmentally independent between environments, perhaps owing to an inducible switch. Inducible defences in plants might be an example (Agrawal *et al.*, 2002; Dicke & Hilker, 2003), as is the defence in the freshwater snail *Physa virgata* (DeWitt, 1998; DeWitt & Scheiner, 2004b). Alternatively, the trait might be physiologically continuous between environments, as the shade-avoidance response in plants (Schmitt & Wulff, 1993; Schmitt *et al.*, 2003; Huber *et al.*, 2004) or eyespot size in *Bicyclus anynana* (Windig, 1994). Both the shade-avoidance response and eyespot size in *B. anynana* had initially been treated as a polyphenism (here with two alternative phenotypes) but proved to be better represented by a continuous reaction norm after more detailed study. In cases of the discrete first type the trait can only be modelled by using separate trait values for each environment (Via & Lande, 1985) (Fig. 1a) and does not allow extrapolation to yet another environment. In cases of the continuous second type, the trait can be modelled both by separate trait values for each environment (Via & Lande, 1985) (Fig. 1b) and by a continuous function of the environment (de Jong, 1990, 1995; Gavrilets, 1993) (Fig. 1c). Knowledge of the trait values in a few environments allows predictions for trait values in neighbouring environments. Optimized phenotypically plastic traits of this type can easily adapt to novel environments, at least if the optimum phenotype over environments and the extant genotypes are similar functions.

The type of phenotypic plasticity in physiologically continuous traits is usually referred to as a reaction norm (Woltereck, 1909). An individual's genotype for the trait gives the function, and genotypic value in the individual's particular environment is given by the function value. To write a reaction norm as a polynomial function is an easy modelling strategy for a physiologically continuous trait: the genotypic value in any particular environment x is written as

$$g(x) = g_0 + g_1 x + g_2 x^2 \ldots + g_k x^k \qquad \text{Eqn 3}$$

In a polynomial reaction norm the coefficients g_i of the polynomial can be treated as quantitative traits that obey expression 2. A vector of selection responses $\Delta\bar{\mathbf{g}}$ contains the changes in mean 'height' $\bar{g}_0$, mean 'slope' $\bar{g}_1$, mean 'curvature' $\bar{g}_2$, etc. The matrix $\mathbf{G}$ is the genetic variance covariance matrix of the polynomial coefficients. The vector $\boldsymbol{\beta}$ of selection gradients now has elements $\partial\bar{w}/\partial\bar{g}$; mean fitness over all environments remains the same, $\bar{w} = \sum_x f_x \bar{w}_x$. Selection can be modelled straightforwardly if the optimum phenotype in each environment can also be written in polynomial form, as $\theta(x) = \theta_0 + \theta_1 x + \theta_2 x^2 \ldots + \theta_k x^k$. For linear reaction norm and linear optimum phenotype, quadratic optimizing selection takes the form $w(x) = 1 - s((\theta_0 + \theta_1 x) - (g_0 + g_1 x))^2$; for higher-order reaction norms the form of quadratic optimizing selection directly follows. Selection occurs within each environment, but can be rewritten in a form that involves quadratic selection for each polynomial coefficient; for example, for a linear reaction norm such as $w(x) = 1 - s((\theta_0 - g_0)^2 + 2(\theta_0 - g_0)(\theta_1 - g_1)x + x^2(\theta_1 - g_1)^2)$.

Both the polynomial reaction norm model and the character state model can be used for physiologically continuous traits. The polynomial reaction norm model and the character state model represent the same reaction norm (Fig. 1b,c) and are mathematically interchangeable (de Jong, 1995). The models represent equivalent modelling strategies for such physiologically continuous traits; which model is used depends on modelling convenience. Selection actually occurs within each environment. In Fig. 2a,b selection on a linear reaction norm is shown, comparing the original mean reaction norm and the mean reaction norm after 2500 generations selection. The two populations plotted are the two most extreme out of 10 randomly initiated runs. The mathematical conversion can be used to assess how strongly selection affects 'height' g_0, 'slope' g_1, 'curvature' g_2, and so on. The selection gradient on the reaction norm coefficients can be expressed in the moments of the distribution of the environment. The mean environment has by definition the value 0: $\bar{x} = 0$. If the occurrence of the environment x is given by the probability f_x, leading to a mean of the distribution of the environments of $\bar{x} = 0$ and a variance of the distribution of the environments given by σ_x^2, the selection gradient on the 'height' g_0 becomes $\beta_0 = 2s(\theta_0 - \bar{g}_0)$ and the selection gradient on the 'slope' g_1 works out to be $\beta_1 = 2s(\theta_1 - \bar{g}_1)\,\sigma_x^2$ for a linear reaction norm. The strength of selection on the slope of the linear reaction norm depends on the variance of the environmental variable,



and therefore on the width of the habitat and the frequency of the environments (Fig. 2c,d). For reaction norms that are higher polynomials, one goes on like this. If the reaction norm is a quadratic function of the environmental value, the third moment of the environmental variable appears in the formal selection gradient (de Jong, 1995, 1999).

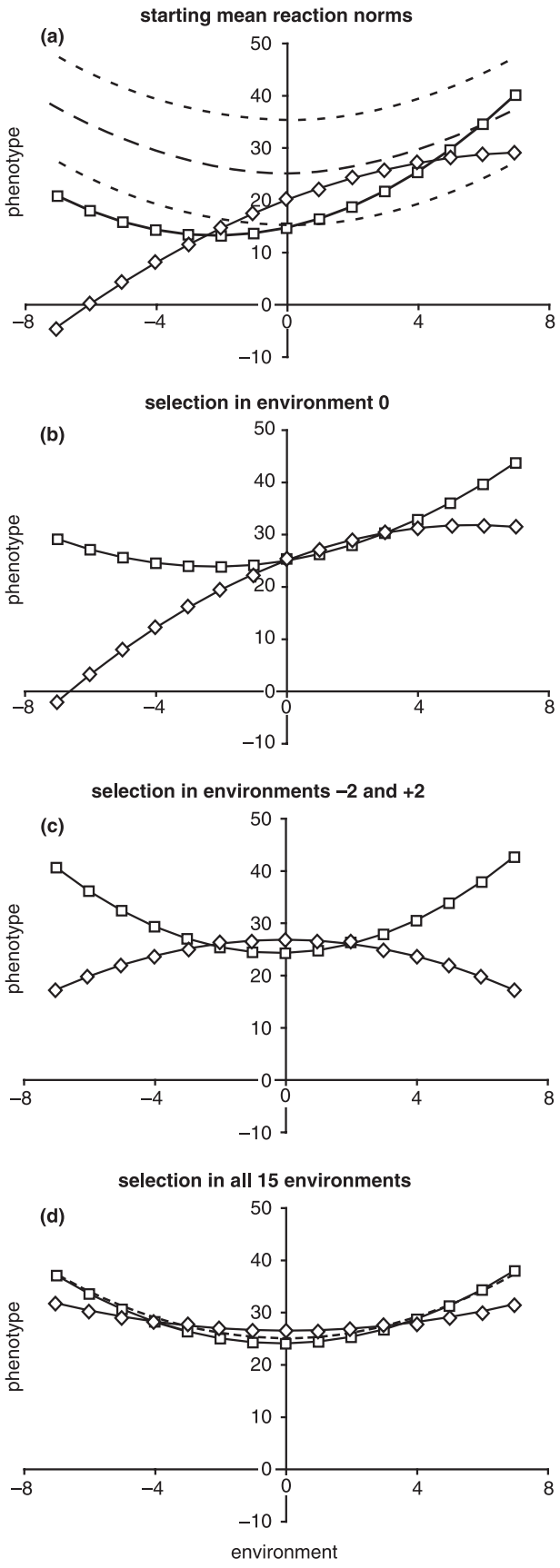
The number of environments

We all know that two points define a line and that three points are necessary to indicate curvature. Similarly, selection in a few environments only optimizes the value of the reaction norm in those environments, and might leave much residual genetic variation to be expressed in other environments. Selection in one environment fixes all polynomial reaction norms to go through one point. If such a population were exposed to other environments, much genetic variation would become visible. To eliminate genetic variation as much as possible, a linear reaction norm should be selected in at least two environments, a quadratic reaction norm should be selected in at least three environments, and so on.

In Fig. 3, selection is on reaction norms that are quadratic functions. The populations differ in mean 'height' $\bar{g}_0$, mean 'slope' $\bar{g}_1$, mean 'curvature' $\bar{g}_2$, and in genetic variation for

Fig. 2 Optimizing selection on a linear reaction norm and the influence of the variance in the environment on the selection response. (a,b) Selection over all 21 environments. (a) Initial mean reaction norms in two independent populations (black population 1, grey population 2) and the optimum reaction norm (dashed). (b) The reaction norms in the two populations after 2500 generations of selection in each population (black population 1, grey population 2) and the optimum reaction norm (dashed). (c,d) Selection over different numbers of environments: higher variance in the environment promotes selection on slope (diamonds, $\sigma_x^2 = 100.00$; squares, $\sigma_x^2 = 36.67$; triangles, $\sigma_x^2 = 1.00$); (c) higher variance in the environment promotes selection on slope (d) starting at a very deviant slope.

Multilocus selection is continued until genetic variation is exhausted or until generation 2500. Environments range from $x = -10$ to $x = +10$. Optimizing individual selection in environment x is given by $w(x) = 1 - s(\theta(x) - z(x))^2$ if $1 - s(\theta(x) - z(x))^2 > 0$, and $w(x) = 0$ if $1 - s(\theta(x) - z(x))^2 \leq 0$; here $\theta(x) = x$ and $s = 0.0044$. The value of s has been chosen to accommodate all initial reaction norm values over the entire environmental range of −10 to +10. Each reaction norm genotype $g = g_0 + g_1 x$ is specified by $n = 20$ independent loci that all code for both 'height' g_0 and 'slope' g_1. The value of g_0 for each allele is drawn from a normal distribution with mean and standard deviation $2.0/(2n)$. The value of g_1 for each allele is drawn from a normal distribution with mean $0.1/(2n)$ and standard deviation $1.0/(2n)$. The three selection regimes in (c,d) are: (1) selection in environments −10 and +10, both at probability 0.5; mean environmental value $\bar{x} = 0$, variance over the distribution of environments $\sigma_x^2 = 100.00$; (2) in all 21 environments from −10 to +10, all at probabilities $1/21 = 0.476$; mean environmental value $\bar{x} = 0$, variance over the distribution of environments $\sigma_x^2 = 36.67$; (3) selection in environments −1 and +1, both at probability 0.5; mean environmental value $\bar{x} = 0$, variance over the distribution of environments $\sigma_x^2 = 1$.



these parameters. Note that the *form* of the reaction norm function is given: mean curvature $\bar{g}_2 = 0$ is possible, but would still be accompanied by genetic variation in curvature, mean slope $\bar{g}_1 = 0$ is possible, but would still be accompanied by genetic variation in slope.

The initial mean reaction norms for two populations and the optimum reaction norm are shown in Fig. 3a for environments −7 to +7. If only one environment $x = 0$ is present, selection fixes the mean phenotypic value for that environment (Fig. 3b), and leaves many genetic differences both within and between populations in slope and curvature. If selection is in two environments, here at $x = -2$ and $x = +2$, the mean phenotypes in those two environments are fixed, but much variation in curvature remains (Fig. 3c). Selection in more than two environments affects directly all parameters, and the optimum reaction norm is approached (Fig. 3d). Of course, the mean slope and mean curvature also change under selection if only one environment is present, and the mean curvature also changes if two environments are present. But these changes are formally a correlated response to a direct selection gradient on height, or height and slope, respectively.

Physiological continuity in trait value therefore always leads to hidden variation in reaction norm values if selection is restricted to very few environments. The well-known genetic variation in transplant environments within and between populations of *Potentilla glandulosa* from alpine populations (Clausen *et al.*, 1940) might represent a good example of this type of reaction norm variation.

Novel environments

Phenotypically plastic traits might show phenotypic adaptation to a different environments, even if the environmental

Fig. 3 Optimizing selection on a quadratic reaction norm: the influence of the number of environments on not-expressed genetic variation. (a) Two initial mean reaction norms in two separate populations and the optimum reaction norm; (b) selection in environment $x = 0$; after 500 generations the optimum character state in $x = 0$ has been reached, but considerable genetic variation in 'slope' g_1 and 'curvature' g_2 remains; (c) selection in environments $x = -2$ and $x = +2$; after 500 generations the optimum character state in the selection environments been reached, but considerable genetic variation in 'curvature' g_2 remains. (d) Selection over all 15 environments. Note that some reaction norms were initially only selected over 8–10 environments, as in the other environments its fitness equalled zero.

Multilocus selection is continued until generation 500. Environments range from $x = 7$ to $x = -7$. Optimizing individual selection in environment x is given by $w(x) = 1 - s(\theta(x) - z(x))^2$ if $1 - s(\theta(x) - z(x))^2 > 0$, and $w(x) = 0$ if $1 - s(\theta(x) - z(x))^2 \leq 0$; here $\theta(x) = x$ and $s = 0.01$. Each reaction norm genotype $g = g_0 + g_1x + g_2x^2$ is specified by $n = 25$ independent loci that all code for both 'height' g_0, 'slope' g_1 and 'curvature' g_2. The value of g_0 for each locus is drawn from a normal distribution with mean $20/n$ and standard deviation $9/n$. The value of g_1 for each locus is drawn from a normal distribution with mean $1.0/n$ and standard deviation $4/n$. The value of g_2 for each locus is drawn from a normal distribution with mean 0.0 and standard deviation $1.0/n$.

change projects outside the range normally encountered. Such plastic adaptation to novel environments might occur, but not necessarily so. Upon environmental change, additional genotypic differentiation might, but need not, appear. The subalpine plant *Potentilla pulcherrima* did not show ecotypical differentiation in phenotypic plasticity in prefloration interval upon reciprocal transplantation between three altitudes (Stinson, 2004). Here, plants from three altitudes behaved identically, as if adaptive phenotypic plasticity could be extrapolated. However, plants of *Potamogeton pectinatus* from different substrate types revealed additional genetic variation upon transplantation to the other soil (Hangelbroek *et al.*, 2003). What will happen depends upon previous selection and the optimum phenotype in the new environment.

The simplest model case is environmental extension after the reaction norm has evolved to the optimum reaction norm. In Fig. 3d, the optimum reaction norm over environments −7 to +7 has been approached after selection in all environments for 500 generations. If the environment in Fig. 3d is then widened to the range −10 to +10, and the optimum reaction norm over the new range is an extension of the original quadratic optimum reaction norm, the populations would show themselves reasonably well adapted to their novel wider environmental range. Some minor residual genetic variation would become visible, and better visible in environments further away from the selection range. Since the reaction norms are physiologically continuous, no new developmental processes need be invoked. Previous selection has led to adaptation over an environmental range, and the environment is extended in a predictable fashion. If however, the environment would be extended to the range −10 to +10, but would there have optima totally different from the optimum over the range −7 to +7, the phenotypically plastic genotypes would do no better than genotypes without any possibility for plasticity. That is, phenotypic plasticity is adaptive over novel environments if both the plasticity and the environmental optima are continuous (Fig. 4a).

Phenotypically plastic traits might show some adaptation to environmental change even if the optimal reaction norm is not totally realized. In Fig. 3c, a quadratic reaction norm was selected in two environments; much genetic variation in curvature remained. If the environment changed the genetic variation would be expressed. Whether another environment was added or whether the environment collapsed to one new environment, genetic variation in phenotype would appear in this third environment. The newly expressed genetic variation in phenotype would be composed of phenotypic plasticity because of the continuity in physiology and genetic variation in reaction norm coefficients (Fig. 4b). The original optimum reaction norm would be selected if the optimum in the new environment conformed to the original optimum phenotype function, whether the old environments remained or disappeared. This as the new environment would be the third selective environment; two points of the phenotypic reaction

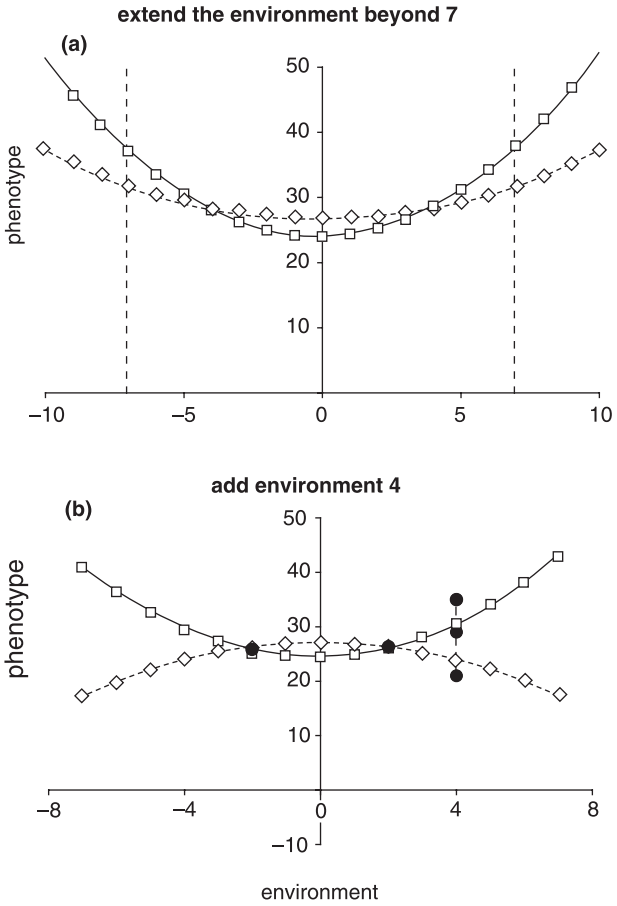


Fig. 4 Phenotypic plasticity and novel environments: newly expressed phenotypic plasticity. (a) extension of the environmental range of Fig. 3d from $x = -7$, $x = +7$ to $x = -10$, $x = +10$ yields direct phenotypic adaptation owing to continuity of both optimum (thick line) and evolved reaction norms; (b) a third environment, $x = 4$, additional to the two environments $x = -2$ and $x = +2$ present in Fig. 3c, leads to selection on the newly expressed phenotypic plasticity. Given any of the three new environments, selection for different reaction norms is still possible.

norm had already been selected, and any third environment leads to the full specification of the quadratic reaction norm to be selected. Appearance of any fourth environment would not lead to the expression of more genetic variation. In a fourth environment, the phenotype would be totally determined by the evolved phenotypic plasticity, as the reaction has been constrained to be quadratic.

Life history differences and population differentiation

The life history of the trait decides whether selection will lead to the optimal reaction norm or not. In the model of Via & Lande (1985), all zygotes were randomly mixed before entering an environment; otherwise the life history was treated much like a black box. One thing was clear: the phenotype developed

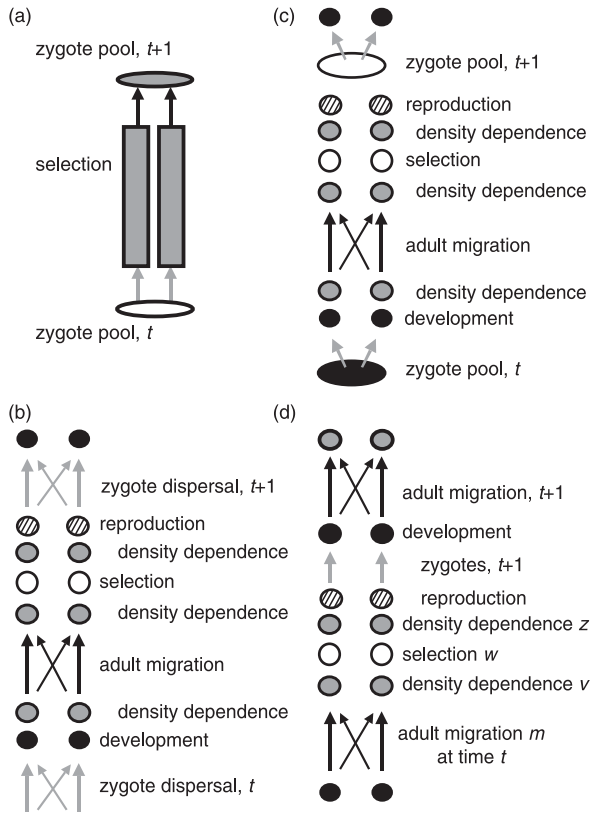


Fig. 5 Life histories assumed in the various models. (a) Zygotes are fully mixed in a zygote pool; selection takes place in the same environment as development; this is Via & Lande (1985). (b) Zygotes disperse from the parental environment, develop in environments x and after development migrate to environments y where selection takes place; density-dependent number regulation can take place at any of three stages in the life history; this model is detailed in de Jong & Behera (2002). (c) Zygote pool version de Jong & Behera (2002). (d) Zygote dispersal absent, adult migration present between development of the phenotype and selection; otherwise the life history of de Jong & Behera (2002), but two rather than three possible stages for density-dependent number regulation; notation added.

and was selected in the same environment (Fig. 5a). Optimal phenotypic plasticity evolved as a direct result. However, selection need not be predictable from the environment of development (Fig. 5b–d). Sasaki & de Jong (1999) and de Jong & Behera (2002) modelled the most detailed life history (Fig. 5c); fitness w that was a simple Gaussian curve representing optimizing selection with Via & Lande (1985) now absorbs migration probabilities and probabilities to survive in density dependent number regulation. de Jong & Behera's (2002) modelled life history involves: (1) two dispersal/migration stages, as zygotes and as adults; (2) selection in the environment of the adult; and (3) three periods of density-dependent number regulation (in the juvenile environment, in the adult environment before selection and in the adult environment after selection). The presence of zygote dispersal – before development and selection – and adult migration – between development and selection – in one model allows a decision on the relative importance of zygote dispersal and adult migration. The presence of a migration stage between developing the phenotype and selection on that phenotype is crucial for the potential outcome of the model (de Jong, 1999; Sasaki & de Jong, 1999; de Jong & Behera, 2002). Distinguishing between the density-dependent stages is important, as density dependence after selection introduces frequency-dependent selection and therefore a wider potential for polymorphism (Sasaki & de Jong, 1999; Burger & Gimelfarb, 2004).

Migration between development and selection might lower plasticity

Migration between development of the phenotype and selection is however, the determining feature of models that include life history components. Most migration will be modelled as from development environment to an identical selection environment, from development environment x to selection environment $y = x$, but an individual might go to environments $y = x + 1$ or $y = x - 1$, or any other environment. Distributing individuals around $y = x$ is an obvious modelling strategy, but in fact we have a large scope for choice in modelling here. An individual that develops in environment x might migrate to environments $y = x + a - 1$, $x = a$, $x = a + 1$, with a constant offset a. Alternatively, individuals from all environments $x < 0$ might experience environment y_1 as selective environment, but individuals from all environments $x > 0$ might experience environment y_2 as selective environment. In general, the probability for an individual that has developed in environment x to be selected in environment y is given by $f_{y|x}$. The selection gradient that decides how the phenotype in environment x evolves now involves averaging over the selection in all environments y that individuals developing in x reach. For quadratic optimizing selection, the selection gradient determining what optimum phenotype evolves in environment x becomes

$$f_x \sum_y 2 f_{y|x} s(\theta(y) - \bar{g}(x)) = 0 \qquad \text{Eqn 4}$$

The evolved optimum phenotype is therefore equal to

$$\bar{g}(x) = \sum_y f_{y|x} \theta(y) \qquad \text{Eqn 5}$$

The evolved phenotype equals the average optimum phenotype that the individuals leaving environment x actually encounter in the selection environments. The properties of their environment of development x do not play any role in the evolved developmental phenotype of environment x. This is a direct model consequence of the lack of constraints in environment x. The probability $f_{y|x}$ involves the migration probability $m_{y|x}$ but, depending upon the further details of the life history, it might contain many more components,

such as survival probabilities from density dependent number regulation, reproductive value, and a Gaussian form if optimizing selection is not quadratic but Gaussian. The components are specified in de Jong & Behera (2002).

The selection gradient in expression 4 is modelled according to the character state model of Via & Lande (1985); it can be rewritten in polynomial reaction norm shape (de Jong, 1999). Expression 5 also can be rewritten in reaction norm shape. A linear reaction norm evolves to mean coefficients for height $\bar{g}_0$ and slope $\bar{g}_1$ of

$$\bar{g}_0 = \theta_0 \quad \text{and} \quad \bar{g}_1 = \theta_1 \frac{\text{cov}(x,\, y)}{\sigma_x^2} = \theta_1 b_{yx} \qquad \text{Eqn 6}$$

Here θ_0 represents the optimum height and θ_1 the optimum slope of the reaction norm; b_{yx} is the slope of the regression of selection environments y on development environments x. Migration leads potentially to a lower than optimal reaction norm slope (Fig. 6a). The covariance between the values of the environment y of selection and the environment x of development has a maximum value of σ_x^2. The evolved value for the slope of the linear reaction norm is at most the optimal slope. If after development all individuals migrate randomly, and end up in an environment y independent of where they developed, the mean slope becomes zero. Full randomization of selection leads to development that is independent of the actual environmental conditions (Fig. 6b). The evolved reaction norm slope equals zero. As a consequence, individuals are not optimally adapted even though phenotypically plastic. A genetic load remains (de Jong, 1995).

The evolved reaction norm slope might equal zero, but we should be careful not to state that plasticity no longer exists. The model trait is still formally phenotypically plastic. That is, I am making a model difference between a trait description as $g(x) = g_0$, a fixed trait value physiologically independent of the environment, and $g(x) = g_0 + g_1 x$, where the trait is basically phenotypically plastic but might have a slope g_1 of zero. In the fixed case, selection in a populations with $\bar{g}(x) = \bar{g}_0$ will not yield plasticity; in the second case, selection in a population with $\bar{g}(x) = \bar{g}_0 + \bar{g}_1 x$ and $\bar{g}_1 = 0$ might well yield a phenotypically plastic population.

Migration only has an effect on evolved plasticity if the covariance between selection environments and development environments differs from the variance in development environments. A symmetric migration distribution around the development environment, together with similar selection gradients within those environments leads to a slope b_{yx} of value 1 between environments x and y. If so, migration has no effect: the average environment of selection is actually identical to the development environment. Migration only has effect if the migration distribution around the development environment is asymmetric. Asymmetry in the migration distribution can be reached in two ways: by limits to the environment and by density dependent number regulation.

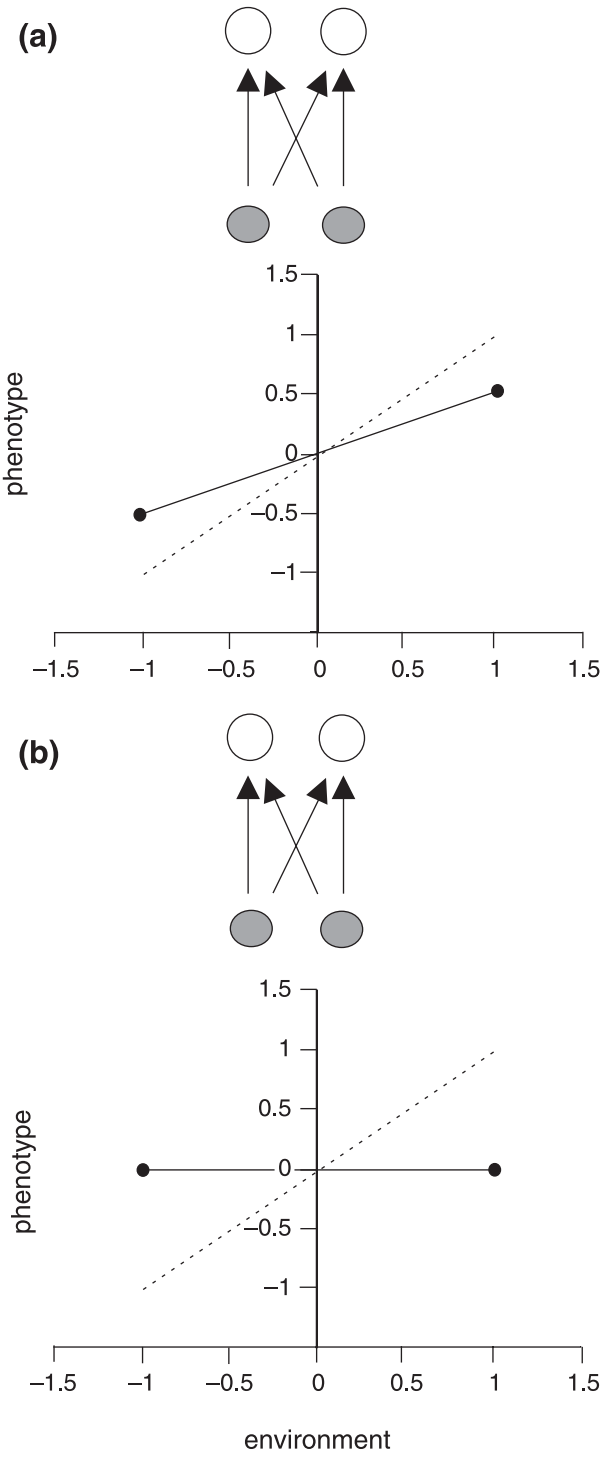


Fig. 6 Migration leads to a lower than optimal reaction norm slope. (a) Two environments, $\theta_1 = 1$, $\bar{g}_1 = 0.5$, due to $f_{-1|-1} = 0.75$, $f_{+1|-1} = 0.25$, $f_{-1|+1} = 0.25$, $f_{+1|+1} = 0.75$. (b) Two environments, $\theta_1 = 1$, $\bar{g}_1 = 0$, due to $f_{-1|-1} = 0.5$, $f_{+1|-1} = 0.5$, $f_{-1|+1} = 0.5$, $f_{+1|+1} = 0.5$.

Limits to the environment automatically arise if only few environments are present (as in Fig. 6). In habitats with many environments, two boundaries will always be present in a linear array of environments. In *Quercus ilex* in Italy, for

example, the boundaries are the Alps in the north and the Mediterranean in the south (Gratani *et al.*, 2003). Or such a boundary might be a diet on all *Daphnia* in open water vs a diet on snails along the shore for sticklebacks, *Gasterosteus aculeata* (Day *et al.*, 1994). Anyway, boundaries are a real biological feature. In the simulations, the boundaries are mirroring: if a migrating individual was bound for an environment a units to the left of the lower boundary, it ends up in the environment a units to the right of the lower boundary.

Asymmetry in the effective distribution of selection environments comes about when density-dependent number regulation takes place in the environment of selection and differs between environments. If density-dependent viability is lower in environment $y = x - 1$ but higher in environment $y = x + 1$ than in environment $y = x$, the effective migration distribution is skewed towards higher values of y. The optimum phenotype in environment $y = x + 1$ is found more often among surviving individuals than the optimum phenotype from selection environment $y = x - 1$. The covariance between environments of development and of selection is therefore affected. Phenotypic plasticity is limited and lower than the optimal plasticity when density-dependent viability is highest in the middle environments.

Ecotypes and plasticity

The presence of boundaries to the total habitat and effective asymmetry in migration due to differences in density dependent viability both redirect selection on a phenotypically plastic trait. Together, these environmental factors might cause the appearance of trait values and genotypes that seem specialized to one of the extreme environments of the habitat. However, the mean genotypic value might have been selected to a mean slope of zero (i.e. to $\bar{g}(x) = \bar{g}_0 + \bar{g}_1 x$ with $\bar{g}_1 = 0$). Plasticity has not disappeared as potential but is not expressed on average. We might see the emergence of ecotypes as a consequence of limited adult migration, density dependent viability and environmental boundaries, and selection on phenotypic plasticity.

Simulations will be used to provide examples. Theory is present in the literature (de Jong & Behera, 2002), as are simulations (Scheiner, 1998). We will consider a simple life history with migration of adults between development of the phenotype and selection but no zygote dispersal (Fig. 5d). Zygotes develop in their (asexual) parent's environment. In such a life history, limited adult migration provokes local genetic differentiation and therefore locally different reaction norms (Scheiner, 1998; de Jong, 1999). Density-dependent number regulation provides additional patterns.

The model of de Jong & Behera (2002) is used in the simulation; equations describing the model are found there. In the simulations, the optimum reaction norm is linear with optimum intercept $\theta_0 = 0$ and optimum slope $\theta_1 = 1$. Optimizing selection is of quadratic form, with $w(xy) = 1 - s(\theta(y) - z(x))^2$, where the selection intensity $s = 0.25$, the optimum in environment y equals $\theta(y) = \theta_0 + \theta_1 y$, and $z(x) = g_0 + g_1 x$ is the phenotype of an adult that has developed in environment x. Selection intensity $s = 0.25$ implies that fitness becomes zero when the difference between the trait value $z(x)$ and the optimum trait value $\theta(y) = y$ is more than 2.

Ten replicate populations were run for 15 000 generations in a habitat of 15 environments, for each case. Each of the 10 replicate populations was started with 14 individuals of optimal genotype $g(x) = x$. Organisms are individually represented as objects in the simulation; each individual has its environment of development, its environment of selection, its genetic reaction norm coefficients for height and slope and its status (alive or dead) as object properties. The genetic reaction norm coefficients can mutate independent of each other. Migration probabilities are uniformly distributed: an adult that develops in environment x can migrate to any environment y from $y = x - 4$ to $y = x + 4$ with probability $1/9 = 0.11$. Environmental boundaries mirror.

Density-dependent number regulation is within the selection patch in the simulation, either between migration and selection or between selection and reproduction. Density-dependent number regulation after selection introduces frequency-dependent selection (Prout, 1980). Density-dependent number regulation before selection but in the selection patch is the most direct frequency-independent comparison, and the only one such comparison possible in a model including adult migration but no zygote dispersal. The total amount of food over the environments is always the same (2500 units), but the distribution of food over environments varies. Food distribution follows a normal distribution, with most food in the middle environment $x = 7$; the width of the food distribution, corresponding to the standard deviation of a normal distribution, can be varied. In Figs 7 and 8, the food distributions and the mean number of adults present in generation 15 000 are shown.

Density-dependent number regulation depends on the total number irrespective of genotype, and is identical for all genotypes. Density-dependent number regulation follows from the distribution of food. The viability function that represents local density dependence is the same in all environments. However, density dependent number regulation can potentially occur before selection or after selection (Fig. 5d). This matters as density-dependent number regulation before selection works on the total number of migrant adults. If so, mortality might be high owing to this density dependence; selection additionally operates on the fraction survivors. However, if density-dependent number regulation occurs after selection, selection has already removed a number of animals and density dependence is less intense and partly compensates selective mortality. Because of this partial compensation of selective mortality, density dependent mortality after selection introduces frequency-dependent selection in the life cycle ((Prout, 1980; Sasaki & de Jong, 1999). Therefore, the genetic variation in the population might be

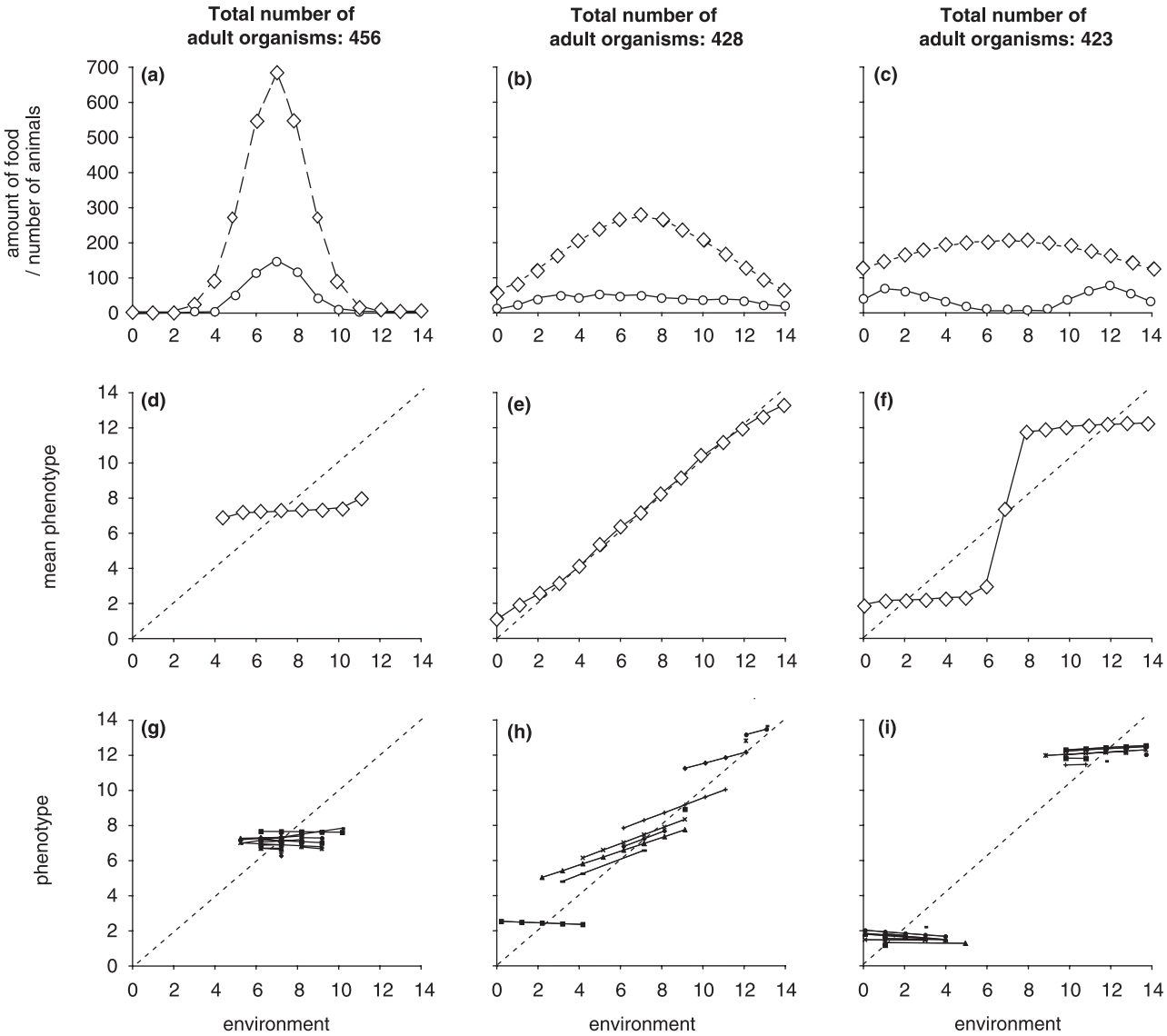


Fig. 7 With limited migration the evolved reaction norm depends upon the distribution of food over the environment, the total environmental width and the specifics of the life history; here, density-dependent number regulation before selection. (a–c) Amount of food per environment and mean adult number over 10 replicate populations; (d–f) evolved mean phenotype over 10 replicate populations; (g–i) genotypes present in run 1 The life history is given in Fig. 5d. Here, density dependence is present before selection, the density dependence denoted by *v* in Fig. 5d. Density-dependent viability is according to $v(y) = \exp(-3N_y/M_y)$, where N_y is the number of organism in environment y and M_y the amount of food available in that environment. Optimizing individual selection in environment y is given by $w(y) = 1 - s(\theta(y) - z(x))^2$ if $1 - s(\theta(y) - z(x))^2 > 0$, and $w(y) = 0$ if $1 - s(\theta(y) - z(x))^2 \leq 0$; here $\theta(y) = y$ and $s = 0.25$. Migration of adults is uniformly distributed; an adult that develops in environment x can migrate to any environment from $x - 4$ to $x + 4$ with probability $1/9 = 0.11$. Three different distributions of food over the total environment are given. For each food distribution, mean population number in generation 15 000 over 10 replicate populations is given. Each of 10 replicate populations is started with 14 individuals of optimal genotype, with optimum intercept $\theta_0 = 0$ and optimum slope $\theta_0 = 1$. Mutations in intercept and slope are independent of each other. The mutation rate equals $\mu = 0.01$ per individual at birth.

higher (Burger & Gimelfarb, 2004). Density dependence before or after selection therefore differs in two respects. Density dependence before selection leads to additivity of density-dependent and selective deaths, without any frequency effects. Density dependence after selection leads to numerical compensation of selective deaths, and frequency-dependent overall fitnesses.

Three widths of the food distribution are used, at widths 1.5, 4 and 7, narrower, similar and wider than the migration distribution, respectively. At food width 1.5 (Figs 7a,d,g and 8a,d,g), migration disperses the adults widely in comparison with the distribution of food. Survival is strongly skewed towards the middle environments (Figs 7a and 8a), and selection on the reaction norm becomes asymmetric because of

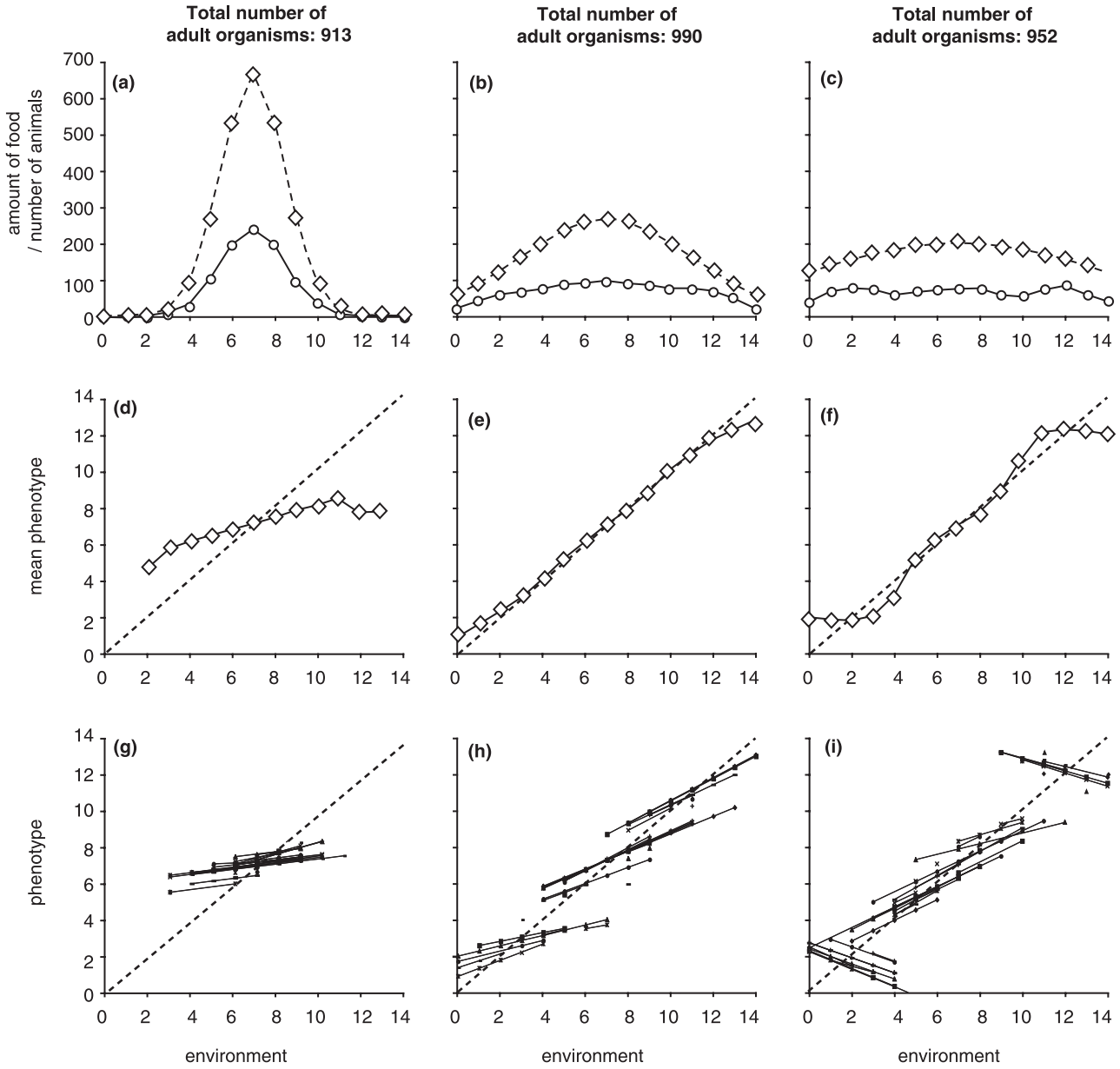


Fig. 8 With limited migration the evolved reaction norm depends upon the distribution of food over the environment, the total environmental width and the specifics of the life history; here, there is density-dependent number regulation after selection. (a–c) amount of food per environment and mean adult number 10 replicate populations; (d–f) evolved mean phenotype over 10 replicate populations; (g–i) genotypes present in run 1 The life history is given in Fig. 5d. Here, density dependence is present after selection, and is denoted by z in Fig. 5d. Density-dependent viability is according to $z(y) = \exp(-3N_y/M_y)$, where N_y is the number of organism in environment y and M_y the amount of food available in that environment. Optimizing individual selection in environment y is given by $w(y) = 1 - s(\theta(y) - z(x))^2$ if $1 - s(\theta(y) - z(x))^2 > 0$, and $w(y) = 0$ if $1 - s(\theta(y) - z(x))^2 \leq 0$; here $\theta(y) = y$ and $s = 0.25$. Migration of adults is uniformly distributed; an adult that develops in environment x can migrate to any environment from $x - 4$ to $x + 4$ with probability $1/9 = 0.11$. Three different distributions of food over the total environment are given. For each food distribution, mean population number in generation 15 000 over 10 replicate populations is given. Each of 10 replicate populations is started with 14 individuals of optimal genotype, with optimum intercept $\theta_0 = 0$ and optimum slope $\theta_0 = 1$. Mutations in intercept and slope are independent of each other. The mutation rate equals $\mu = 0.01$ per individual at birth.

density-dependent mortality. The phenotypic optima that belong to the environments with a high food supply are effectively selected almost independently of the development patch of the adult. The result is a mean reaction norm that shows little phenotypic plasticity and is near the optimum value for the middle and most productive environment (Figs 7d and 8d), as are the genotypic reaction norms (Figs 7g and 8g). Selection towards the optimum of the middle environment is somewhat stronger with density dependence before selection. This has three consequences: (1) the higher

mortality with density dependence before selection leads to a shorter viable range of environments (compare Fig. 7d with Fig. 8d); (2) the evolved slope of the genotypic reaction norms is even nearer zero (compare Fig. 7g with Fig. 8g); (3) the number of genotypes is lower (21 genotypes in Fig. 7g vs 33 genotypes in Fig. 8g).

The food distribution covers nearly all 15 environments wide habitat at width 4 (Figs 7b and 8b). Asymmetry in density-dependent viability is not noticeable, but some influence of the habitat boundaries is present. The result is an evolved mean population reaction norm that almost perfectly follows the optimal reaction norm, apart from some 'fraying at the edges' (Figs 7e and 8e). This mean population reaction norm is however, composed of genotypic reaction norms that deviate in intercept and slope from the optimum reaction norm (Figs 7h and 8h). These genotypic reaction norms cross the optimum reaction norm at the appropriate elevation (as had been argued from mathematical arguments; de Jong, 1999). Again, density-dependent number regulation before selection leads to a lower number of genotypes in the overall population (12 in Fig. 7h vs 36 in Fig. 8h).

A very wide distribution of food over environments leads to very different numerical patterns depending more upon the timing of density dependent number regulation in the life history. Limited adult migration causes disruptive selection. Individuals are concentrated towards the boundaries of the habitat at the end of the simulation (Fig. 7c). The boundary reflects migrating individuals, promoting selection for extreme trait values over selection for trait values that belong to middle environments. Individuals specifically adapted to low environments cannot survive in high environments. Selection on the phenotypically plastic trait leads to clearly separated ecotypes (Fig. 7f). The genotypes making up these ecotypes have evolved reaction norms with slopes near zero (Fig. 7i). The splitting into ecotypes despite the presence of sufficient food over the middle of the environment might have parallels in models of evolution of limiting similarity (Christiansen & Loeschcke, 1980; Loeschcke & Christiansen, 1984) and adaptive dynamics (Dieckmann, 1997; Dieckmann & Doebeli, 1999).

Density-dependent number regulation after selection does not remove all individuals from the middle part of the habitat range (Fig. 8c). The population mean reaction norm more or less follows the optimum reaction norm, but shows a pronounced effect near the habitat boundaries where individuals are turned back and selected more uniformly to low (high) optimum values of the trait (Fig. 8f). The genotypes that underlie the population mean here are of three types, which might be called ecotypes. In Fig. 8i we see a low environments specialist and a high environment specialist (both with evolved reaction norm slopes that are actually of opposite sign to the optimum slope!) and a middle environment generalist with a slightly lower slope than optimal. Note the much higher genetic variation in Fig. 8i than in Fig. 7i: 43 vs. 20 genotypes, respectively.

The simulations in Figs 7 and 8 give an impression of the diversity of patterns that is possible when selection on phenotypic plasticity is combined with limited migration. The simulation in Fig. 9 is aimed at a pattern of ecotypes and phenotypic plasticity as is shown by hayfield and pasture *Plantago lanceolata* (van Tienderen, 1989) or benthic and limnetic *Gasterosteus aculeatus* (Day *et al.*, 1994). The ecotypes in these species are phenotypically plastic in the direction of the 'other' ecotype if raised under its appropriate conditions, but do not reach the full phenotype of the 'other' ecotype in its own habitat. The simulation ecotypes are phenotypically plastic in the overall direction of the difference between the ecotypic means, but the plasticity within each ecotype is not so high as to abolish the difference between the ecotypes. The optimal reaction norm corresponds to the difference between the phenotypes in the extreme environments. The slopes of the evolved reaction norms are less steep than the optimal slope.

The emergence of ecotypes is in full accordance with the quantitative genetic models for selection on reaction norms. The only additional point to take into account is the life history and the ecology of the species.

Discussion

Classical quantitative genetic models of selection on phenotypically plastic traits can explain cases of phenotypic adaptation to varying environments, ready phenotypic adaptation to novel environments, genetic variation in novel environments, limits to plasticity and the emergence of ecotypes with nonoptimal reaction norms. Many patterns that are observed empirically are easily explained once standard models are applied. The adaptive role of phenotypic plasticity can be predicted from these models, not as a consequence of a developmental plasticity that is inherent to life and on a par with selection, but as a consequence of previous selection and evolving plasticity. Considerations of life history detail and physiological continuity have been added to the models of Via & Lande (1985) and Gavrilets (1993), but the genetics and logic remain the same.

The question has been asked whether phenotypic plasticity promotes evolution. In general, there is no answer if the question is phrased that way. A phenotypically plastic genotype might well have a wider niche than a nonplastic genotype. Environmental change would then cause selection and genotypic change in the nonplastic species but not as readily in the phenotypically plastic species. In this sense, phenotypic plasticity might delay evolution. Larger environmental change might perhaps be easier bridged by a phenotypically plastic species. But all this depends on the actual change in the environment, the optimum phenotypes and the actual range of phenotypic plasticity.

Ecotypic differentiation need not be accompanied by phenotypic plasticity, as in the classical studies of copper tolerance in *Agrostis tenuis* (Walley *et al.*, 1974). Ecological differentiation

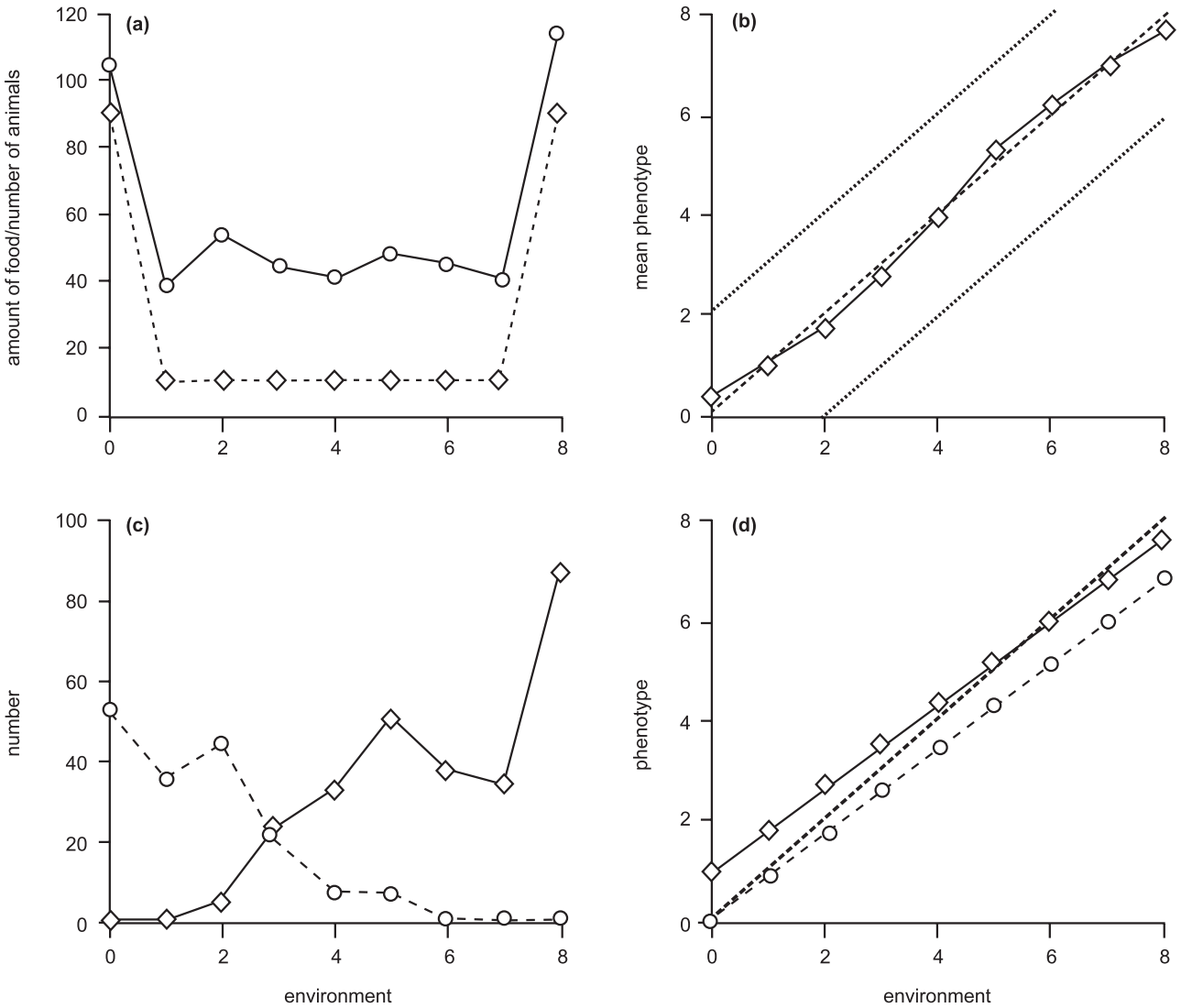


Fig. 9 Simulation aimed at a pattern of ecotypes and phenotypic plasticity as shown by benthic and limnetic *Gasterosteus aculeatus*. (a) Amount of food per environment and mean adult number over 10 populations. (b) Mean population reaction norm over 10 populations, and optimum reaction norm including fitness boundaries. (c) Ecotype number in run 1: left ecotype (round symbols, dashed line), mean over four genotypes; right ecotype (diamond symbols drawn line) mean over six genotypes (all genotypes present in the populations with more than 10 individuals). (d) Reaction norms of the two ecotypes in run 1: left ecotype (round symbols, dashed line), mean over four genotypes; right ecotype (diamond symbols drawn line), mean over six genotypes (all genotypes present in the populations with more than 10 individuals). Density dependence is present after selection (Fig. 5d). Density dependent viability is according to $z(y) = \exp(-3N_y/M_y)$, where N_y is the number of organism in environment y and M_y the amount of food available in that environment; the amount of food is much higher at the environmental boundaries than in the middle of the environments. Optimizing individual selection in environment y is given by $w(y) = 1 - s(\theta(y) - z(x))^2$ if $1 - s(\theta(y) - z(x))^2 > 0$, and $w(y) = 0$ if $1 - s(\theta(y) - z(x))^2 \leq 0$; here $\theta(y) = y$ and $s = 0.25$. Migration of adults is uniformly distributed; an adult that develops in environment x can migrate to any environment from $x - 2$ to $x + 2$ with probability $1/5 = 0.20$. Each of 10 replicate populations is started with 8 individuals of optimal genotype, with optimum intercept $\theta_0 = 0$ and optimum slope $\theta_1 = 1$. Mutations in intercept and slope are independent of each other. The mutation rate equals $\mu = 0.01$ per individual at birth.

might be totally due to phenotypic plasticity, as in heterophylly within a single floating individual plant between leaves within the water and in the air. Ecological adaptation mediated by phenotypic plasticity might be identical in all plant individuals, as in the flooding response of *Rumex palustris* (Voesenek *et al.*, 2003). In *Rumex palustris*, rosette leaves assume a horizontal position close to the substrate if not submerged. If submerged, the orientation of leaves changes quickly into an almost vertical position as a consequence of growth of specialize cells at the base of the petiole, and the petioles stretch, resulting in emergence of the leaf blade above the water (Voesenek *et al.*, 2004). However, here interest centres on ecotypic differentiation accompanied by phenotypic plasticity, especially where phenotypic plasticity within

each ecotype is in the direction of the other ecotype. Such ecotypes where the phenotypic plasticity covaries with ecotypic differentiation have been described in *Ranunculus flammula* (Cook & Johnson, 1968), *Plantago lanceolata* (van Tienderen, 1989) and *Quercus ilex* (Gratani *et al.*, 2003).

Quercus ilex populations were sampled in northern, middle and southern Italic and grown in an experimental garden near Rome (Gratani *et al.*, 2003). Morphology and physiology indicated adaptation to drought stress for the southern population and adaptation to winter stress at the northern distribution limit, the northern and southern populations showing specialization. The population from the middle of Italy possessed highest plasticity. On the face of it, the pattern in Fig. 8j resembles the data on *Q. ilex*.

Ranunculus flammula is flexible in its leaf shape; an individual plant develops terrestrial or aquatic leaves depending upon the immediate environment, possibly leading to different type leaves on the same plant (Cook & Johnson, 1968). Plants originating from several lakeshores in Oregon were grown first 4 months aquatically and afterwards for 3 months terrestrially. Plants differed highly in leaf shape by lake of origin, although aquatic leaves are always narrower than terrestrial leaves. Plants showed varying degrees of heterophylly, depending upon origin. In three localities, plants were sampled across a lakeshore, in order to investigate any potential differentiation between more terrestrial plants far up the shore and more aquatic plants farther down. Cook & Johnson (1968) summarized their findings as a tendency towards three kinds of plants, from wet terrestrial conditions with broad and not very plastic leaves, from amphibious conditions with medium but plastic leaves and from dry terrestrial conditions with narrow and not very plastic leaves. From survival experiments, Cook & Johnson (1968) showed that the most highly heterophyllous population possessed the greatest ecological amplitude. More specialized aquatic or terrestrial populations proved less heterophyllous. Population differentiation proved to exist on a local scale. At any one lakeshore, higher plants proved more terrestrial and lower-growing plants more aquatic in morphology. Plants from intermediate height at the shore had not been sampled. Cook & Johnson (1968) painted a picture of evolution in relation to succession. Colonization of a new lakeshore by the most plastic genotype would be followed by disruptive selection due to spatial differentiation under competitive conditions (Cook & Johnson, 1968, pp. 511–512).

The observations of Cook & Johnson (1968) on *Ranunculus flammula* can be modelled by the model specified above. In Figs 7i and 8i, the end result of a simulation that started with an optimally plastic genotype is shown. The verbal picture of the relation between plasticity and ecotypic differentiation as painted by Cook & Johnson (1968) is very near to model description and results here. West-Eberhard (2003, p. 414) regarded the heterophyllic *R. flammula* as a possible example of developmental character release (p. 406).

Phenotypic plasticity has recently been implicated in divergence within species, in speciation and macroevolution (Pigliucci & Murren, 2003; West-Eberhard, 2003; Schlichting, 2004). West-Eberhard (2003, chapter 27) outlined the 'developmental plasticity hypothesis of speciation' as a possible route to speciation among developmentally plastic organisms. 'The developmental-plasticity hypothesis of speciation proposes that developmental plasticity in trait expression within a parent population can predispose descendent sister populations to speciation by facilitating the intraspecific evolution of contrasting specializations' (p. 528). *Ranunculus flammula* is regarded as a possible example (p. 531).

Pigliucci & Murren (2003) review recent field studies for evidence that they regard as consistent with the hypothesis of genetic assimilation. These field studies typically contrast two ecotypes and examine the ecotypes for phenotypic plasticity by raising both ecotypes in their own and the other's environment. The ecotypes prove phenotypically plastic; their phenotype in the 'other' environment diverges from their phenotype in their 'own' environment in the direction of the phenotype of the other ecotype. Plasticity is in the same direction as the difference between the ecotypes mean trait values, but not as strong. Examples are hayfield and pasture *Plantago lanceolata* (van Tienderen, 1989) in pasture and hayfield, and the benthic and limnetic three-spine sticklebacks on their own and the opposite diet (Day *et al.*, 1994). Pigliucci & Murren (2003) regard this and similar field studies as lending indirect support to the hypothesis of genetic assimilation. Direct support for a hypothesis of genetic assimilation is not quoted and seemed not to be available. No attempt was made to explain the data starting from the quantitative genetic models of the evolution of phenotypic plasticity (i.e. the quantitative genetic models reviewed by Berrigan & Scheiner (2004), by Pigliucci (2001), and presented earlier).

Pigliucci & Murren (2003) achieve the link between the empirical data and genetic assimilation by changing the definition of genetic assimilation. Pigliucci & Murren (2003) define genetic assimilation as: genetic assimilation is a process that turns a plastic response into a genetically invariant one through continued selection for stable expression of the trait under new environmental conditions. This definition fits selection to a reaction norm slope of zero but does not represent Waddington's genetic assimilation. Waddington (1956) described the appearance of a deviant nonadaptive phenotype due to an environmental stimulus; selection for the occurrence of the deviant phenotype led to a line in which the deviation always appeared, even without the eliciting environmental stimulus. The point is that the stimulus is no longer necessary and the deviant phenotype appears in the original environment because alleles for its occurrence have become fixed; the genetically assimilated phenotype is not phenotypically plastic. In this way it is possible to select for expression of the *bithorax* phenotype starting with application of ether to pupae as a stressor. Falconer (1981) and Falconer & Mackay (1996)

states that genetic assimilation can be regarded as selection involving two thresholds, one for expression in the standard environment and one for expression in the inducing environment. (Remember that Falconer worked in the same Department as Waddington.) Two thresholds is a far cry from selection on reaction norm slope. Comparison of Waddington's experiment, as recounted by Pigliucci & Murren (2003), with the cases they call 'modern empirical evidence consistent with genetic assimilation' shows that none of these cases shows anything like genetic assimilation in the sense Waddington used it. Reaction norms are present, not threshold characters. Plastic phenotypes are expressed in the original environment. Pigliucci & Murren's (2003) scenariogram (their Fig. 1) lacks any relation to genetic assimilation. The cited empirical cases represent selection on the slope of the reaction norm in partly separated populations, and so does the scenariogram. Genetic assimilation is just a misapplied label to the reported findings.

Pigliucci & Murren (2003) deplore that genetic assimilation keeps failing to capture the imagination of empirical scientists. There is a good reason for that lack of attention: the lack of convincing examples. Waddington showed genetic assimilation could possibly work, but most of his examples were phenotypic deviants far removed from potential adaptation in nature. The only trait Waddington used where adaptation might potentially play a role was the change in size of the anal papillae in *Drosophila melanogaster* in response to salt (NaCl) in larval food. te Velde (te Velde, 1985; te Velde *et al.*, 1988a,b; te Velde & Scharloo, 1988) showed how anal papillae function, and how selection on anal papillae size as a quantitative trait induced adaptation to a salty environment. Noticeably, the work of te Velde showed how this trait behaved like a good quantitative genetic reaction norm.

The simulations reported in Figs 7 and 8 connect selection on phenotypic plasticity with the emergence of ecotypes. Genetic assimilation is absent. The evolved ecotypes do not possess the optimum reaction norm, even if phenotypically plastic. Optimizing selection on a phenotypically plastic trait need not yield optimum plasticity in the model. It does yield an optimum mean phenotype for the ecotype.

Do the simulations reported in Figs 7–9 show that developmental plasticity might facilitate intraspecific macroevolution or might contribute to the evolution of reproductive isolation (West-Eberhard, 2003 chapters 27 and 29)? Not at all. The simulations demonstrate the possible emergence of ecotypes as a consequence of limited migration and selection to local optima and, most importantly, show that phenotypic plasticity does not prevent ecotypes emerging. Evolved phenotypes depend upon the width of the distribution of food, and since selection on the slope of the reaction norm is assumed possible, the reaction norm slopes respond to selection. In a very similar model the genotype can be assumed fixed, that is, have an invariant reaction norm slope of zero. The results of such simulations are not shown here, but are very similar. Corresponding ecotypes emerge at the widest food distribution. The major difference is found for the intermediate environmental food width of 4 (Figs 7b,e,g and 8b,e,g). In the figures, phenotypic plasticity allows genotypes to follow the optimal reaction norm. With genotypes of fixed slope zero, phenotypic plasticity is replaced by genetic variation. The average phenotype in the population looks much the same for each environment, whether phenotypic plasticity or genetic variation is present. The difference is in the genetic variance within the populations, and in the deviation from the optimum that remains present. Phenotypic plasticity decreases 'genetic load'.

The conclusion is that in the model a similar mean phenotype is reached, and approaches the optimum phenotype, either by genetic variation or by phenotypic plasticity. Niche width is potentially higher for the individual phenotypically plastic genotypes. Selection therefore favours plasticity, if it is physiologically possible. However, plasticity is itself not an evolutionary mechanism and does not promote different evolutionary solutions. Phenotypic plasticity does not constitute a major alternative view of evolutionary biology, but takes its legitimate place in the neo-Darwinian modern synthesis.

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